Sunshine Act Meetings

Federal Register
Vol. 53, No. 109
Tuesday, June 7, 1988

This section of the FEDERAL REGISTER contains notices of meetings published under the "Government in the Sunshine Act" (Pub. L. 94-409) 5 U.S.C. 552b(e)(3).

EQUAL EMPLOYMENT OPPORTUNITY COMMISSION

"FEDERAL REGISTER" CITATION OF PREVIOUS ANNOUNCEMENT: 53 FR 20213, Thursday, June 2, 1988.

PREVIOUSLY ANNOUNCED TIME AND DATE OF MEETING: 2:00 p.m. (eastern time) Tuesday, June 7, 1988.

CHANGE IN THE MEETING: An item has been added to the agenda under the Open Session: Request for Approval of a Non-Competitive Requirement for Herman Miller's Vaughan Wall Demountable Wall Systems.

CONTACT PERSON FOR MORE INFORMATION: Hilda D. Rodriquez, Executive Officer (Acting), Executive Secretariat, (202) 634–6748.

Date: June 1, 1988.

Susan Daniel,

Acting Executive Officer Executive Secretariat.

[FR Doc. 88–12898 Filed 6–3–88; 4:03 pm]

BILLING CODE 6750-06-M

EQUAL EMPLOYMENT OPPORTUNITY COMMISSION

DATE AND TIME: 2:00 p.m. (eastern time) Monday, June 13, 1988.

PLACE: Clarence M. Mitchell, Jr., Conference Room, No. 200–C on the Second Floor of the Columbia Plaza Office Building, 2401 "E" Street, NW., Washington, DC 20507.

STATUS: Part of the Meeting will be Open to the Public and Part will be Closed to the Public.

MATTERS TO BE CONSIDERED:

Open Session

1. Announcement of Notation Vote(s)
2. A Report on Commission Operations (Optional)

Closed Session

Litigation Authorization: General Counsel Recommendation

Note.—Any matter not discussed or concluded may be carried over to a later meeting. (In addition to publishing notices on the EEOC Commission meetings in the Federal Register, the Commission also provides a recorded announcement a full week in advance on future Commission sessions. Please telephone (202) 634–6748 at all times for information on these meetings.)

CONTACT PERSON FOR MORE INFORMATION: Hilda D. Rodriguez, Executive Officer (Acting) on (202) 634–6748.

Date: June 2, 1988.

Susan Daniel,

Acting Executive Officer, Executive Secretariat.

[FR Doc. 88–12899 Filed 6–3–88; 4:03 pm]

BILLING CODE 6750-06-M

NUCLEAR REGULATORY COMMISSION

DATE: Weeks of June 6, 13, 20, and 27, 1988.

PLACE: Commissioners' Conference Room, 11555 Rockville Pike, Rockville, Maryland.

STATUS: Open and Closed.

MATTERS TO BE CONSIDERED:

Week of June 6

Thursday, June 9

10:00 a.m.
Briefing on Status of Pilgrim (Public Meeting)
11:30 a.m.
Affirmation/Discussion and Vote (Public Meeting)

Week of June 13—Tentative

Thursday, June 16

2:00 p.m.
Briefing on Advanced Light Water Reactors by EPRI (Public Meeting)
3:30 p.m.
Affirmation/Discussion and Vote (Public Meeting) (if needed)

Week of June 20—Tentative

Monday, June 20

1:00 p.m.
Discussion of Management-Organization and Internal Personnel Matters (Closed—Ex. 4)
2:30 p.m.
Briefing on Technical Specification Revisions (Public Meeting)

Tuesday, June 21

2:00 p.m.
Briefing on Proposed Rule on Fitness for Duty (Public Meeting)

Friday, June 24

11:00 a.m.
Affirmation/Discussion and Vote (Public Meeting) (if needed)

Week of June 27—Tentative

Monday, June 27

10:00 a.m.
Briefing on Proposed Rule on Early Site Permits; Standard Design Certification; and Combined Licenses for Nuclear Power Reactors (Public Meeting)

Wednesday, June 29

10:00 a.m.
Initial Briefing by the Advisory Committee on Nuclear Waste (Public Meeting)
11:30 a.m.
Affirmation/Discussion and Vote (Public Meeting) (if needed)

ADDITIONAL INFORMATION: Briefing on Master Plan for Integrating All Severe Accident Issues (Public Meeting) was held on June 2.

Note.—Affirmation sessions are initially scheduled and announced to the public on a time-reserved basis. Supplementary notice is provided in accordance with the Sunshine Act as specific items are identified and added to the meeting agenda. If there is no specific subject listed for affirmation, this means that no item has as yet been identified as requiring any Commission vote on this date.

TO VERIFY THE STATUS OF MEETINGS CALL (RECORDING): (301) 492–0292.

CONTACT PERSON FOR MORE INFORMATION: William Hill (301) 492–1661.

William M. Hill, Jr.,

Office of the Secretary.

June 2, 1988.

[FR Doc. 88–12900 Filed 6–3–88; 4:02 pm]

BILLING CODE 7590-01-M

SECURITIES AND EXCHANGE COMMISSION

"FEDERAL REGISTER" CITATION OF PREVIOUS ANNOUNCEMENT: (53 FR 19367 May 27, 1988).

STATUS: Closed meeting.

PLACE: 450 5th Street, NW., Washington, DC.

DATE PREVIOUSLY ANNOUNCED: Tuesday, May 24, 1988.

CHANGES IN THE MEETING: Cancellation.

The closed meeting to be held on Wednesday, June 1, 1988, after the 10:00 a.m. open meeting has been cancelled.

Commissioner Peters, as duty officer, determined that Commission business required the above change.

At times changes in Commission priorities require alterations in the scheduling of meeting items. For further information and to ascertain what, if any, matters have been added, deleted

or postponed, please contact: Kevin Fogarty at (202) 272–3195.

Jonathan G. Katz,

Secretary.

June 1, 1988.

[FR Doc. 88–12906 Filed 6–3–88; 4:03 pm]

BILLING CODE 8010–01–M

TENNESSEE VALLEY AUTHORITY

(Meeting No. 1403)

TIME AND DATE: 10 a.m. (E.D.T.), June 8, 1988.

PLACE: A.C. Reynolds High School Auditorium, Reynolds School Road, Asheville, North Carolina.

STATUS: Open.

Agenda

Approval of minutes of meeting held on May 18, 1988.

Action Items

A—Budget and Financing

A1. Modification of the Capital Budget Financed from Power Proceeds and Borrowings for Fiscal Year 1988—Rehabilitation of Precast Concrete Floor Slabs at all Fossil Plants.

B—Purchase Awards

B1. Invitation SA–17354A—Indefinite Quantity Term Agreement for Paper to be used by Office Support Services Branch in Chattanooga, Knoxville, and Muscle Shoals.

B2. Invitation HE–38057B—Instrument and Control Systms for Allen and John Sevier Fossil Plants.

B3. Negotiation GL–06298A—Dry Fly Ash Collection Facility for Colbert Fossil Plant.

C—Power Items

C1. Renewal Power Contract with Knoxville, Tennessee.

D—Personnel Items

*D1. Proposed Increase in Expenditures Under Personal Services Contract with Bishop, Cook, Purcell & Reynolds.

E—Real Property Transactions

E1. Grant of Permanent Easement for a Road Right of Way Affecting Approximately 3.3 Acres of Tims Ford Reservoir Land Located in Franklin County, Tennessee; and Modification of a Deed to a 33-Acre Track of Tims Ford Reservoir Land to Permit Subdivision and Use for Residential Development.

F—Unclassified

F1. Modification of Interagency Agreement Between TVA and the Department of Energy Providing for the Continuation of TVA's Assistance in the Disposal of Residual Materials from Vicinity Properties in Edgemont, South Dakota.

F2. Supplement to Contract No. TV–72077A with U.S. Department of Agriculture, Forest Service, Northeastern Forest Experiment Station, Providing for Conduct of Exposure Experiments at Whitetop Mountain to Determine Cause of Red Spruce Decline in High Elevation of Southern Appalachians.

F3. Memorandum of Agreement No. TV–74203A with U.S. Geological Survey (USGS), Department of the Interior, Covering Arrangements for the Coordination of Mapping Activities Within the Tennessee Valley Region and Cooperative Mapping Projects with the USGS.

F4. Supplements to Memorandum of Understanding (TV–71249A) Between TVA and Directorate of Engineering and Housing for TVA Support to the U.S. Army Forces Command, in Connection with Energy Resources Management.

F5. Supplement to Interagency Agreement No. TV–61655A with the U.S. Department of Energy Covering Arrangements for a Southeastern Regional Biomass Energy Program.

*F6. Proposed Changes to the Rules and Regulations of the Retirement System.

*Items approved by individual Board members. This would give formal ratification to the Board's action.

CONTACT PERSON FOR MORE INFORMATION: Alan Carmichael, Director of Information, or a member of his staff can respond to requests for information about this meeting. Call (615) 632–8000, Knoxville, Tennessee. Information is also available at TVA's Washington Office (202) 245–0101.

Dated: June 1, 1988.

W.F. Willis,

General Manager.

[FR Doc. 88–12873 Filed 6–3–88; 1:17 pm]

BILLING CODE 8120–01–M

Corrections

Federal Register
Vol. 53, No. 109
Tuesday, June 7, 1988

This section of the FEDERAL REGISTER contains editorial corrections of previously published Presidential, Rule, Proposed Rule, and Notice documents and volumes of the Code of Federal Regulations. These corrections are prepared by the Office of the Federal Register. Agency prepared corrections are issued as signed documents and appear in the appropriate document categories elsewhere in the issue.

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Food and Drug Administration

21 CFR Parts 184 and 186

[Docket No. 79N-0269]

Iron and Iron Salts; Affirmation of GRAS Status as Direct and Indirect Human Food Ingredients

Correction

In rule document 88-10582 beginning on page 16862 in the issue of Thursday, May 12, 1988, make the following corrections:

S 184.1304 [Corrected]

1. On page 16865, in the second column, in S 184.1304(a), in the second line, "H_{20}" should read "H_2O".

2. In the same column, in S 184.1304(d), in the last line, "waived" was misspelled.

S 184.1308 [Corrected]

3. On page 16866, in the second column, in S 184.1308(b), in the ninth line, "1200" should read "1100".

S 186.1300 [Corrected]

4. On page 16867, in the second column, in S 186.1300(b)(2), in the first line, "as" should read "at".

S 186.1374 [Corrected]

5. On the same page, in the second column, in S 168.1374(b)(2), in the first line, "as" should read "at".

BILLING CODE 1505-01-D

NUCLEAR REGULATORY COMMISSION

[Docket No. 50-249]

Commonwealth Edison Co.; Environmental Assessment and Finding of No Significant Impact

Correction

In notice document 88-11463 beginning on page 18361 in the issue of Monday, May 23, 1988, make the following corrections:

1. On page 18362, in the second column, in the first complete paragraph, in the 13th line, after "will" insert "not".

2. On the same page, in the same column, in the fourth complete paragraph, in the second line, "Mary" should read "March".

BILLING CODE 1505-01-D

Federal Register

Tuesday
June 7, 1988

Part II

Department of Labor

Occupational Safety and Health Administration

29 CFR Part 1910
Air Contaminants; Proposed Rule

DEPARTMENT OF LABOR

Occupational Safety and Health Administration

29 CFR Part 1910

[Docket No. H-020]

Air Contaminants

AGENCY: Occupational Safety and Health Administration (OSHA), Department of Labor.

ACTION: Proposed rule.

SUMMARY: The Occupational Safety and Health Administration (OSHA) proposes to amend its existing air contaminant standards § 1910.1000, Tables Z-1, Z-2, Z-3, and add a new Table Z-4. The amendments reduce permissible exposure limits for approximately 100 substances now listed in the "Z-tables," raise the permissible exposure limit for 1 substance, set permissible exposure limits for 205 substances currently not regulated by OSHA, add or change STEL's for 70 substances, and, as appropriate, set skin, short-term or ceiling limits.

OSHA has reviewed health evidence for all these substances and has determined that the new limits substantially reduce a risk of deleterious health effects among American workers, including cancers, central and peripheral neuropathies, lung disease, liver and kidney damage and other systemic effects. The health evidence forms a reasonable basis for proposing revisions to these levels. In the final rule, after review of all the evidence in the record OSHA will establish new levels which it determines will substantially reduce significant risks.

It has also preliminary concluded, based on a review of many data bases and an extensive survey, that the new limits are feasible. To assist in its analysis, OSHA has utilized the National Institute for Occupational Safety and Health-Recommended Exposure Limits (NIOSH-REL's) and the American Conference of Governmental Industrial Hygienists-Threshold Limit Values (ACGIH-TLV) published in 1987-88, as the starting points in its review.

OSHA will continue its practice of rulemaking for individual substances when regulations of that type are necessary and appropriate.

DATES: Written comments on the proposed standard must be postmarked on or before July 8, 1988. Notices of Intention to appear at the informal rulemaking hearings on the proposed standard must be postmarked on or before July 1, 1988. Individuals who wish to comment or appear during the public hearings must see Section VIII of this document for specific requirements.

Parties who request more than 10 minutes for their presentations at the informal public hearing and parties who will submit documentary evidence at the hearing must submit the full text of their testimony and all documentary evidence, postmakred on or before July 8, 1988. The informal rulemaking hearing is scheduled to begin on July 20, 1988.

ADDRESSES: Written comments should be submitted to the Docket Officer, Docket No. H-020, Room N-3670, U.S. Department of Labor 200 Constitution Avenue NW., Washington, DC, 20210, telephone (202) 523-7894.

Notice of intention to appear, testimony and documentary evidence to be submitted at the hearing are to be sent to Mr. Tom Hall, OSHA Division of Consumer Affairs, Docket No. H-020, Room N-3647, U.S. Department of Labor, 200 Constitution Avenue NW, Washington, DC 20210, telephone (202) 523-8615.

The hearing will be held in Washington, DC, in the Auditorium, Frances Perkins Department of Labor Building, Third and Constitution Avenue NW. The informal public hearing will begin at 9:30 a.m.

FOR FURTHER INFORMATION CONTACT: Mr. James F. Foster, Director, Office of Information and Consumer Affairs, OSHA, U.S. Department of Labor, Room N-3649, 200 Constitution Avenue NW., Washington, DC 20210, Telephone (202) 523-8151.

SUPPLEMENTARY INFORMATION:

Organization of this Document

This **Federal Register** notice discusses policy and legal issues, and includes the proposed amendments to 29 CFR 1910.1000 Tables Z-1, Z-2, and Z-3, and the proposed new Table Z-4. It includes a discussion of the generic health effects for 15 individual groupings (e.g., neuropathic, ocular, cardiovascular, etc.) as well as a review of the health effects for all of the individual substances. It also includes a summary of the preliminary regulatory analysis with feasibility determinations organized by industry sector.

The Docket (H-020) includes considerable additional data, including many health studies, the complete preliminary regulatory and feasibility analysis with appendices and additional feasibility information. This includes the final results of a large scale industry survey. Also included are several computerized data tapes and a master tape summarizing the available information. A four-volume printed version of this information, organized by substance, is also in the Docket. All this information is available for inspection and copying at the Docket Office. Copies will be available at the cost of reproduction.

OSHA is continuing to gather additional individual substance information. This includes health effects and feasibility information. A number of site visits reports will be placed in the docket.

The discussion is organized in the following manner:

I. Background
- A. Questions Solicited for NPRM
- B. History and Need for Revision of the PEL's
- C. Approach
- D. Basis for Identifying Replacement PEL's
- E. Substances Included in the Update of 1910.1000 Z-Tables
- F. Identification of Substances Requiring Special Attention
- G. Alternate Procedures for Dealing with Substances Requiring Special Attention
- H. Construction, Maritime and Agriculture Segments

II. Pertinent Legal Authority

III. Glossary

IV. Substances to be Regulated
- A. General Principles of Toxicology and Dose-Response
- B. Historical Development of Occupational Exposure Limits
- C. Description of the Substances for Which Limits Are Being Proposed
  1. Substances for Which Proposed Limits are Based on Avoidance of Neuropathic Effects
  2. Substances for Which Proposed Limits are Based on Avoidance of Narcosis
  3. Substances for Which Proposed Limits are Based on Avoidance of Sensory Irritation
  4. Substances for Which Proposed Limits are Based on Avoidance of Liver or Kidney Effects
  5. Substances for Which Proposed Limits are Based on Avoidance of Ocular Effects
  6. Substances for Which Proposed Limits are Based on Avoidance of Respiratory Effects
  7. Substances for Which Proposed Limits are Based on Avoidance of Cardiovascular Effects
  8. Substances for Which Proposed Limits are Based on Avoidance of Systemic Toxicity
  9. Substances for Which Proposed Limits are Based on Observed No-Effects Levels
  10. Substances for Which Proposed Limits are Based on Avoidance of Adverse Nuisance Effects
  11. Substances for Which Proposed Limits are Based on Avoidance of Odor and Taste Effects
  12. Substances for Which Proposed Limits are Based on Avoidance of Adverse Health Effects caused by Exposure to Analogous Substances

13. Substances for Which Proposed Limits are Based on Avoidance of Biochemical/Metabolic Effects
14. Substances for Which Proposed Limits are Based on Avoidance of Sensitization Effects
15. Substances for Which Proposed Limits are Based on Avoidance of Cancer
16. Substances for Which Current ACGIH-TLVs are Less Stringent than Existing OSHA PELs
17. Substances for Which OSHA is Proposing Short Term Exposure Limits
18. Substances for Which OSHA is Proposing to Add Skin Notations
D. References For Section IV
V. Summary of Preliminary Feasibility, Regulatory Impact, Regulatory Flexibility and Environmental Impact Analyses
VI. Clearance of Information Collection Requirements
VII. Summary and Explanation of the Proposed Standard
VIII. Public Participation Public Hearings
IX. Authority
X. Standard
XI. Appendices
Appendix A—Sampling and Analytical Methods
Appendix B—Preliminary Regulatory Impact, Regulatory Flexibility Analysis, and Feasibility Analysis

I. Background

A. Questions Solicited for NPRM

OSHA requests comments on all issues raised by this proposal including health effects, feasibility, risk and policy issues. The following are some specific questions which may assist commenters in their review.

1. Are substances included which should be excluded from this rulemaking?

2. Is additional health and feasibility documentation available relative to the proposed PEL's, beyond that described in the preamble?

3. Are substances included in this rulemaking used in industries other than those described in the preamble?

4. Are substances included in this rulemaking used for purposes other than those described in the preamble?

5. Do alternative unpublished exposure guidelines exist, such as those used in private workplaces, which may be suitable for general usage?

6. Is there information regarding laboratory analytical procedures which may be used in lieu of those suggested by OSHA (See Appendix A) to determine exposure to air contaminants?

7. Are the proposed exposure limits for each substance appropriate?

8. Is additional information available for those substances for which ACGIH proposed a higher TLV which might affect OSHA's decision that such a change was not justified?

9. Should the implementation dates for some substances be delayed because of sampling/analytical limitations or short term feasibility impact considerations?

10. Is there additional information relative to the OSHA plans to adopt some recommended 10 hour TWA REL's as an 8-hour TWA PEL?

11. Does the most current scientific information generally support acceptance of the hypothesis that all C-5-8-Alkanes are *not* equally toxic because a metabolite of n-Hexane exhibits unique neurotoxic properties?

12. OSHA has proposed to use exposure limits from two well-established sets of guidelines as a source of values to update the PEL's. Is information available about alternative sources which OSHA might consider for this purpose?

13. OSHA has outlined its criteria for identifying special situations. Are alternative criteria available which might be used in lieu of these, or in addition to them?

14. OSHA has outlined three alternative procedures for dealing with substances requiring special attention. Are additional approaches available which might be used in lieu of these, or in addition to them?

15. OSHA has performed feasibility analysis for the following substances, based on limited available information:
Acetonitrile
Carbon disulfide
Carbon monoxide
Carbon tetrachloride
Chloroform
Ethylene dichloride
Ethylene glycol dinitrate
Fibrous glass dust
Hydrogen cyanide
Isophorone diisocyanate
Nitrogen dioxide
Nitroglycerin
Trichloroethylene

Is further information available which might be used to supplement the present findings regarding the feasibility of achieving these levels in the workplaces?

16. OSHA has made a preliminary assessment of the proposed rulemakings' impact on large and small establishments. The Act requires OSHA to determine whether a regulation will have a significant impact on a substantial number of small entities, pursuant to the Regulatory Flexibility Act of 1980, 5 U.S.C. 601 *et seq.* Is there additional information regarding implementation of this rule for small businesses and entities which OSHA should consider?

17. OSHA has proposed PEL's for some substances, where the basis for this proposal also includes a carcinogenicity designation (e.g., TLV with a A1 or A2 designation; REL with a Ca designation). Should OSHA include a similar carcinogen designation in the Z–4 Table in this rulemaking?

18. OSHA has preliminarily decided that for substances where the ACGIH, TLV is a TWA and the NIOSH, REL is a Ceiling Value which is the same or one half of the TWA, OSHA will propose that the TWA be adopted as the PEL. Should this approach be modified in the final rulemaking? What approach should be used when the converse of this situation (TLV, Ceiling-REL, TWA) exists?

19. OSHA preliminarily plans to adopt a phased start-up schedule. This would include an initial start-up requirement permitting the use of alternate control methods for revised PEL's, followed at a later date by the required use of control methods fully consistent with the methods of compliance priorities in effect at that time. OSHA will shortly be requesting comments on the hierarchy of controls. An alternate approach is to set compliance date for engineering controls based on final determinations of that rulemaking. OSHA solicits comments on those approaches and suggestions regarding appropriate times for the two proposed start-up dates.

20. OSHA requests comment on whether the establishment of margins of safety below lowest observed or no effect levels is consistent with the concept of "significant risk," and on whether the specific margins of safety proposed for specific chemicals are appropriate.

21. OSHA has identified sensory irritation, which causes rhinitis, cough, sputum production, chest pain, wheezing and dyspnea as material impairment of health. OSHA invites comments on this understanding.

22. The question also arises of whether odorants present material impairment of health. That issue also might arise in the context of other substances. Based on the evidence in the final record concerning this issue, OSHA will determine if the criteria detailed in section IV–C–16 have been met, and take appropriate action. OSHA requests comment on this issue.

23. Is there exposure information available which can be supplied which will refine OSHA's estimates of employee exposures and overexposures to the substances being regulated?

24. Is there information available which can be supplied to improve or supplement the engineering controls identified as necessary in order to reduce exposure levels? Is there additional cost data which can be

supplied to refine the annual costs associated with these controls?

25. Under what conditions, involving which industrial processes, will respirators be needed during the start up period, for maintenance operations, or where other controls are infeasible in order to protect employees at the proposed exposure levels? Are respirators currently being used under the conditions identified, or would they need to be purchased? Please describe the type of respirator currently in use or needed.

26. As a result of simultaneously regulating many substances, what cost savings will be realized in purchasing new engineering controls? Are alternate engineering controls available to achieve the lower permissible exposure limits being proposed?

27. What is the current state of technology control and financing in firms which would need to comply with reduced exposure limits to wood dust?

B. History and Need for Revision of the PEL's

One of the principal reasons, if not the single most important basis, for Congress passing the Occupational Safety and Health Act of 1970, was Congress' recognition of the need to protect workers from occupational health hazards. In the preamble to the Act, Congress stated one of the purposes was to protect employees by "exploring ways to discover latent diseases, establishing causal connections between diseases and work in environmental conditions, and conduct other research relating to health problems, in recognition of the fact the that *occupations health standards present problems often different from those involved in occupational safety.*" (emphasis added).

The legislative history indicates Congressional concern for reduction in health risk from both the recognized hazards and from the many newly utilized chemicals. Congress stated in 1970,

> In the field of occupational health the view is particularly bleak, and due to the lack of information and records, may well be considerably worse than we currently know. Occupational diseases which first commanded attention at the beginning of the Industrial Revolution are still undermining the health of workers. Substantial numbers, even today, fall victim to to ancient industrial poisons such as lead and mercury. Workers in the dusty trades still contract various respiratory diseases. Other materials in industrial use are only now being discovered to have toxic effects. In addition, technological advances and new processes in American industry have brought numerous new hazards to the workplace. Carcinogenic chemicals, lasers, ultrasonic energy, beryllium metal, epoxy resins, pesticides, among others, all present incipient threats to the health of workers. Indeed, new materials and processes are being introduced into industry at a much faster rate than the present meager resources of occupational health can keep up with. It is estimated that every 20 minutes a new and potentially toxic chemical is introduced into industry. New processes and new resources of energy present occupational health problems of unprecedented complexity. (Senate Report 91–1282, p. 2)

To accomplish the goal of protecting workers from occupationally related disease Congress created a three-pronged approach in the OSH Act.

First, Congress desired that OSHA, as soon as possible after it was established, have in existence a set of basic, minimum health and safety standards. To accomplish this it provided in section 6(a) of the OSH Act that OSHA should adopt within its first two years, without hearing or public comment, established federal standards and national consensus standards.

At that time, under the Walsh-Healey Act, The Department of Labor had adopted for government contractors approximately 400 health standards based on the Threshold Limit Value (TLV) recommendations of the American Conference of Governmental Industrial Hygientists (ACGIH). Those were adopted as established federal standards. In addition about 25 additional exposure limits had been recommended by the American Standards Association (presently called the American National Standards Institute). Those were adopted as national consensus standards. OSHA adopted these initial exposure limits in May 1971. They are for the most part the maximum air contaminant levels set forth in Tables Z–1, Z–2, and Z–3 of 29 CFR 1910.1000.

Congress recognized the need to update these standards. It created two mechanisms for updating health standards: Regular or "6(b)" standards and emergency or "6(c)" standards.

Congress specified the procedures for regular standards in sections 6(b) (1)–(4) and 6(f). They provide that: The public may petition for new standards; OSHA may set up an advisory committee; and, before issuing a standard, OSHA must publish a proposal with an explanatory preamble, request public comments and publish an explanatory preamble with the final standard. In addition to these general requirements of informal rulemaking, Congress specified that OSHA must hold an oral hearing if requested and support its determination with substantial evidence in the rulemaking record.

Congress set forth the criteria for health standards in section 6(b)(5) of its Act. This stated:

> The Secretary, in promulgating standards dealing with toxic materials or harmful physical agents under this subsection, shall set the standard which most adequately assures, to the extent feasible, on the basis of the best available evidence, that no employee will suffer material impairment of health or functional capacity even if such employee has regular exposures to the hazard dealt with by such standard for the period of his working life. Development of standards under this subsection shall be based upon research, demonstrations, experiments, and such other information as may be appropriate. In addition to the attainment of the highest degree of health and safety protection for the employee, other considerations shall be the latest available scientific data in the field, the feasibility of the standards, and experience gained under this and other health and safety laws. Whenever practicable, the standard promulgated shall be expressed in terms of objective criteria and of the performance desired.

Congress also provided in section 6(c) for the issuance of Emergency Temporary Standards (ETS) to take immediate effect without rulemaking. However, OSHA is to issue a proposal and complete a section 6(b) rulemaking within 6 months. The criteria for issuing an ETS is that "employers are exposed to grave danger from exposure to substances or agents determined to be toxic or physically harmful or from new hazards, and that such emergency standard is necessary to protect employees from that danger." OSHA has found that section 6(c) procedures have not generally accelerated the regulatory process. Most ETS's have been litigated, and judicial stays have been issued either on procedural or substantive grounds.

Since the passage of the Act in 1970, OSHA has made substantial progress improving the occupational health of workers for some priority health hazards. Asbestos and arsenic exposures have been dramatically reduced, substantially reducing cancer risk to employees. Lead exposures have been reduced and we are now seeing a major reduction in employee blood lead levels, and lead related diseases. Cotton dust exposures have been reduced and byssinosis has been nearly eliminated from the textile work force. OSHA has also substantially reduced significant health risk from some of the newer chemicals such as ethylene oxide and vinyl chloride.

Through the hazard communication and access to employee exposure and medical records standards, OSHA has greatly expanded the ability of

employees to learn about and protect themselves from health hazards.

OSHA's standards have proven to be feasible, often costing less than estimated. The vinyl chloride standard cost one-tenth OSHA's contractor's estimate. The cotton dust standard has been credited with improving the industry's competitiveness and productivity while costing one-half OSHA's estimate.

The preambles to OSHA standards have been lengthy, detailed and sophisticated. They have thoroughly analyzed health studies and controversial scientific issues about carcinogencity and risk assessment. Extensive analyses of feasibility have been made.

OSHA has issued only 24 substance-specific health regulations since its creation. It has not been able to review the many thousands of currently unregulated chemicals in the workplace nor to keep up with reviewing the several thousand new chemicals introduced since its creation. It has not been able to fully review the literature to determine if lower limits are needed for many of the approximately 400 substances it now regulates.

Using past approaches and practices, OSHA could continue to regulate a small number of the high priority substances and those of greatest public interest. However, it would take decades to review currently used chemicals and OSHA would never be able to keep up with the many chemicals which will be newly introduced in the future.

OSHA believes it is a major priority to update its existing PEL's and to make a substantial effort to control exposure to chemicals newly used in the workplace for which no exposure limits exist. The existing health literature and expert judgment indicate that such new or lower limits are needed to protect against many types of deleterious health effects. These include kidney and liver diseases, respiratory diseases, reductions in lung function, nerve disorders and reduction in nerve function, carcinogenicity, irritation to the eyes, throat, skin and other organs which prevent working safely and many other disorders and dysfunctions.

As the preliminary regulatory analysis indicates, millions of employees in total are exposed to levels of these chemicals which, the literature or expert opinion indicates, do or may create deleterious health effects. Clearly, it is a most important occupational health priority to reduce or eliminate such disease and material impairments of health.

Congress clearly indicated that it was a major Congressional priority to consider and control, when needed, the many thousands of unregulated chemicals, and update the existing Z-Table chemicals. For example, the previous quotation indicated Congress' concern with the thousands of newly introduced chemicals. Congress also stated.

> Accordingly, it is essential that such standards (Table Z chemicals) be constantly improved and replaced as new knowledge and techniques are developed. In addition there are occupational hazards, particularly those affecting health—which are not covered by any standards at all. (Senate Report 91-1282, p. 6).

Government agencies and professional organizations have also recommended that OSHA lower exposures for many Table-Z substances and add limits for currently unregulated substances. The National Institute for Occupational Safety and Health has recommended new or lower exposure limits for approximately 160 chemicals (REL's) in its Recommendations for Occupational Safety and Health Standards, September 1986.

The American Conference of Governmental Industrial Hygienists (ACGIH) 1987–88 Threshold Limit Values (TLV's) adopted new exposure limits for approximately 200 substances not regulated by OSHA, and lower limits, short-term exposure limits, ceiling or skin limits for approximately 200 substances now regulated by OSHA.

In light of its priority to address the many unregulated health hazards and improve the existing Table Z limits, OSHA commenced a review process to determine the best way to achieve this goal. It has reviewed its past history and set up an internal task force to consider the matter. OSHA requested the Administrative Conference of the United States to study the issue and make recommendations.

In other rulemakings, important factors as discussed below led OSHA to perform detailed analyses, consider all possible issues, write lengthy preambles and have extensive administrative procedures. However, in OSHA's view, to review and regulate many substances in a reasonable period requires some narrowing of the issues, focus of analysis, and reducing the length of the discussions in the preamble.

The factors that led to OSHA's detailed approach to analysis are clear from OSHA's history: when OSHA issued a health standard it would be sued both by industry arguing its health standards were too strict and by unions arguing tis standards were not strict enough. For example, in 1974, OSHA issued standards for 14 carcinogens (39 FR 3756; Jan. 29, 1974). Industry sued, claiming that OSHA's procedures were inadequate and various studies were poorly done. The unions sued arguing that OSHA had not set a no detectable exposure level, had not set up a permit system and had not set detailed enough requirements for medical examinations. See *Synthetic Organic Chemical Mfgs. v. Brennan; Oil Chemical & Atomic Workers* v. *Brennan*, 503 F.2d 1155, 506 F2d 385 (3rd Cir. 1974).

More recently industry challenged the ethylene oxide standard as being too low and the unions challenged the same standard for not including a short term exposure limit (STEL). See *Public Citizen Health Research Group et al v. Tyson; Assoc. of Ethylene Oxide Users v. Tyson*, 796 F. 2d 1479 (D.C. Cir. 1986).

OSHA also found that lengthy discussions of what it perceived were major issues and less detailed discussion of what it perceived to be minor issues or issues which could wait until another day sometimes resulted in a court remand. For example, the preambles to the final lead standard were over 100 **Federal Register** pages (43 FR 52952, Nov. 14, 1978; 43 FR 54354, Nov. 21, 1978) and included extensive health and feasibility discussions. However, the feasibility discussions were brief for those segments of industry which OSHA believed had fewer problems. The court generally upheld the standard but remanded for reconsideration some of those segments. (See OSHA's similar experience for *Asbestos, Building and Construction Trades vs. Brock*, 86–1359 (D.C. Circ., Feb. 2, 1988)) *United States Workers* v. *Marshall* 647 F. 2d 1189 (D.C. Cir. 1980). Energetic industry challenges, difficulties in gathering detailed data and some lengthy agency reviews have resulted in a major commitment of OSHA resources, numerous hearings and several **Federal Register** notices, in attempting to complete this remand. In another example, a court did not accept the agency's decision to intially limit the hazard communication regulation to the manufacturing sector though the standard as issued created major employee protection benefits. *United Steelworkers* v. *Auchter*, 763 F. 2d 728 (3rd Cir. 1985.).

The success of a project to regulate a large backlog of chemicals for which there is a generally recognized need for new or improved employee protection requires some recognition of the need for agency flexibility in several areas. One is in narrowing the scope of the issues to be covered. A second is in less detailed discussion for each substance. Third is in the flexibility not to cover some issues at the present time when

administrative resources are not available to consider them. In the future when resources are available, the Agency will decide whether the issues have priority.

OSHA, in its first 17 years has also had to address difficult scientific, feasibility and policy issues. These include extrapolation from animal data to humans, (ETO supra), epidemiology risk assessment and significant risk analysis (Arsenic, 48 FR 1864, Jan. 14, 1983; Asbestos, 51 FR 22612, June 20, 1986), feasibility for industries with aging facilities (lead, arsenic, supra), lowest feasible level (Benzene, 52 FR 34460, Sept. 11, 1987, for example) and others.

In response to both the court challenges and the need to face difficult issues, OSHA has engaged in detailed and extensive analyses. These have resulted in lengthier preamble discussions and in-depth analyses for all issues. For example, the 1986 asbestos final standard as published in the **Federal Register** had a 100 page explanatory preamble for general industry. The original asbestos Sec. 6(b) standard had a 2 page preamble (37 FR 11318, June 7, 1972).

Now that OSHA has reviewed these issues in depth several times, has experience "gained under this * * * law" (sec. 6(b)(5)) on these issues, and has had its analysis upheld in the Courts, somewhat less detailed chemical-by-chemical analyses should be appropriate. The accumulated judicial guidance and agency experience reduces the need for as extensive a discussion of some of the issues.

Overall, this preamble is lengthy and both the scientific and feasibility analyses are longer than in previous OSHA rulemaking. OSHA is fully meeting the requirements to analyze significant risk and feasibility. However, the analyses for each chemical are briefer than that provided as part of individual rulemaking.

OSHA has also followed the more extensive administrative procedure of what is called hybrid rulemaking rather than the minimum requirements of informal rulemaking. (See the OSHA procedural rules in 29 CFR Part 1911.) This includes oral hearings, questioning by the public in hearings, extensive right to comment, decision based on an identified record and an Administrative Law Judge presiding at hearings.

As discussed above, in part this is a requirement of the OSHA Act. However, it also reflects relevant legal doctrine. See *International Harvester v. Ruckleshouse.* 478F 2d 615 (D.C. Cir. 1973). This approach has served OSHA well by increasing the Agency's knowledge, and gaining commendation from the courts. See *Industrial Union Dept. v. Hodgson,* 499 F. 2d 467 (D.C. Circ. 1974). It has also been commended in the academic community.

OSHA is not short-cutting its administrative procedures in this matter. However, it is, of course, covering more territory in a single proposal for exposure limits than it has previously. (OSHA's hazard communication regulation, which also has very wide scope, was covered in a single proceeding and was upheld.) This method seems reasonable to OSHA in light of the nature of its proposal and its past experience. Also the Supreme Court in *Vermont Yankee* v. *N.R.D.C.,* 435, U.S. 519, 543.44 (1798) has indicated that agencies have broad flexibility in devising appropriate administrative procedures. It stated that: "administrative agencies should be free to fashion their own rules of procedure, and to pursue methods of inquiry capable of permitting them to discharge their multitudinous duties.

Additional factors which have affected the number of health standards issued by OSHA include various policies of Congress and the President. OSHA develops Environmental Impact Statements as required by the National Environmental Policy Act, conducts Regulatory Flexibility Analyses as required by the Regulatory Flexibility Act and conducts detailed analysis required by the Paperwork Reduction Act. All of the Presidents during OSHA's existence have stressed the need to reduce inflation and improve the cost effectiveness of regulations. Under various Executive Orders (E.O. 12044, 12291) OSHA has been required to perform extensive economic analyses. OSHA, of course, must continue to carry out these important goals of Congress and the President.

As mentioned, OSHA consulted with the Administrative Conference of the United States on appropriate procedures to face the issue of the large number of chemicals which needed new exposure limits. The Conference issued two lengthy reports of a study by two professors of administrative law. After extensive consideration, it made two sets of recommendations to OSHA, Recommendation 87–1, 52 FR 23629 (1987) and 87–10, 92 FR 40147 (Dec. 30, 1987).

The Administrative Conference specifically recommended:

1. Updating the 1971 Consensus Standards. The Occupational Safety and Health Administration, as an interim step should continue to update the Table Z national consensus standards adopted in 1971 if updating can be accomplished by expedited rulemaking procedure (e.g., including more concise preambles) appropriate to the nature of the revised Table. OSHA should update the 1971 standards on a generic basis (i.e., include multiple standards in one proceeding) when consensus recommendations are available, which are generally accepted by employers and workers in the affected industries, and when the new standards can be evaluated on the basis of risk and feasibility information reasonably available to the Agency. This interim step should not interfere with OSHA's continuing responsibility to promulgate and modify safety and health standards.

As this discussion indicates there is clear and generally recognized need to improve occupational health protection of workers from a substantial number of chemicals which are present in the workplace. Clearly an improved approach to regulation is needed to solve this problem in a reasonable time period. OSHA has reviewed the law, Congressional intent, its history, and the recommendations of experts. Based on this review, OSHA has adopted the approach reflected in this proposal and described in greater detail in the approach section below.

OSHA believes that the proposal will lead to a major improvement in occupational health, probably the greatest improvement it can achieve in a short period of time. Consequently, it believes it is justified to make, as one of its first priorities, reducing exposure to approximately 400 substances. It believes it is appropriate to leave for later standards and a second stage, of somewhat lower priority, more detailed analyses of some substances, where lower limits may be appropriate, and review of the need for medical, monitoring and industrial hygiene provisions is required.

C. Approach

This proposed rulemaking represents a different OSHA approach to the problem of setting permissible exposure limits for the wide variety of potentially hazardous substances present in the work place. Under typical section 6(b) rulemaking, OSHA has developed detailed standards for individual substances based on an extensive review of all available information. This is an extremely time and resource-intensive process. In the proposed rulemaking, OSHA will rely to a large extent on the evaluation of other existing exposure limits which have been developed and widely accepted as health protection guidelines, recommendations, or regulations together with an evaluation of the more important studies for each substance. These sources have been developed by

various organizations within and outside the United States.

Analysis of all these guidelines clearly documented the inadequacies of the existing Permissible Exposure Limits (PEL), defined in the § 1910.1000 Z-Tables, which were all developed prior to 1968. Further analysis identified two sources, the American Conference of Governmental Industrial Hygienists, Threshold Limit Values (ACGIH/TLV) and the National Institute for Occupational Safety and Health, Recommended Exposure Limits (NIOSH/REL's), which could be used in this rulemaking. The TLV's were used to identify those substances to be considered in this rulemaking. Both the REL's and TLV's were used to permit OSHA to determine which limits provide a more appropriate permissible exposure limit in light of the evidence and the statutory requirements OSHA must follow. A detailed explanation of this analysis is provided in a separate section of this preamble. The justifications for limiting this proposed rulemaking to the question of allowable air concentrations, without considering ancillary requirements, such as control methods, personal protective equipment, training, etc., are described in a separate section of this preamble.

While OSHA has relied extensively on the guidance provided by the documentation for the ACGIH TLV's and NIOSH REL's, OSHA has also reviewed other health effects data to determine, as the OSHA Act requires, if significant risks exist; if the new exposure limits would substantially reduce that risk; and if technological and economic feasibility exists for the proposed PEL's. OSHA has concentrated its efforts on reviewing summaries of the major studies, with emphasis on the literature used to support the exposure limits proposed by NIOSH and ACGIH for the several hundred substances being considered in this proposed rulemaking. With this approach, it is not necessary to analyze in depth all available studies for each substance. When the studies support the recommended level within the context of OSHA's legal requirements, OSHA is proposing to adopt either the TLV or REL, rather than attempt a detailed analysis. The details of the approach used to resolve differences between individual permissible exposure limits are provided in another section of the preamble. This preamble will discuss the health effects and risk for each substance being considered.

OSHA is also engaging in an extensive analysis of technological and economic feasibility. OSHA is utilizing: A review of the literature; expert professional judgement by a selected group of certified industrial hygienists and professional engineers; a large scale survey of past experiences in the work place; and input from several computer-processable data bases to determine whether the proposed levels are technologically and economically feasible. The survey will be focused by using existing exposure sampling data which indicate usage and the level of compliance already achieved. OSHA is not attempting to analyze the feasibility of alternative proposed exposure levels except where the TLV and REL differ significantly. In those instances only the REL and TLV are considered. However, the absence of adequate feasibility information for a given TLV or REL will eliminate that exposure level from consideration as a possible OSHA PEL.

To attain the objectives of the proposed rulemaking, it has been necessary to limit the discussion of health effects for each individual substance. To use this approach, and satisfy the technical and legal requirements for section 6(b) rulemaking, OSHA has identified 15 health effect categories to include all the substances considered in the rulemaking. While some substances have health effects which fall into more than one category, for purposes of organization, OSHA has grouped the substances according to the ACGIH rationale for identifying the primary health effect. For each of these groups there is a generic discussion of the general health effects considerations which lead to the development of permissible air concentrations for those substances falling within that category. This is then supplemented by a limited discussion of: health effects, significance of risk, and risk reduction for each substance, primarily based on the TLV and REL documentation. In each instance, OSHA reaches its own conclusion regarding which (if either) of these limits are appropriate. OSHA is also considering other relevant guidelines for allowable air concentrations to identify any specific substances which require special attention.

This preamble will include the generic discussions for all 15 health effects groups, and an evaluation of the specific substances.

OSHA anticipates that there are three possible outcomes for the proposed PEL's in this proposal. These are:

(1) The final rule adopts the proposed limits.

(2) The final rule adopts limits different from the existing or proposed limits.

(3) The final rule adopts no changes to the existing limits.

To make a decision between these three alternatives, OSHA will consider three types of information.

(a) Facts, objectively defined on the basis of information now available.

(b) Policy judgment consistent with the statutory requirements the Agency must follow.

(c) Assessment of the record presented during or as a result of the public hearing scheduled for this rulemaking. This assessment of the record will consider both factual and policy information.

Specific evidence which OSHA will consider includes the following:

Number of exposed employees at various exposure levels, number of manufacturing processes involved, quantity of material involved, cost of compliance, type of effect (e.g., acute, chronic), severity of health effects, absence of an existing PEL, magnitude of proposed change to PEL, general acceptance of proposed PEL, and quality of the evidence.

OSHA will use all these criteria in reaching its final decisions based on statutory requirements for each of the substances currently included in the proposal.

In most previous health rulemakings subsequent to the benzene decision OSHA has regulated health hazards which have posed risks above 1 in a thousand over a working lifetime. These are clearly in the range of risks which the agency generally considers "significant." These risks have been quantified through risk assessments using accepted statistical techniques. In most of these rulemakings, the limits of the agency's action have been determined by the constraint of feasibility, meaning that significant risks were likely to remain at the new levels, according to the quantitative risk assessments, but that further reductions in exposures were feasible. In the formaldehyde final rulemaking OSHA stated that regarding the question of significant risk, it "believes that figures of 0.6 per 100,000 (i.e., 6 in one million) predicted by the lower end of the range may be approaching a level that can be viewed as safe in the context of the workplace environment.

Most of OSHA's prior standards have involved chronic health hazards where studies have identified risks at relatively high levels of exposure. OSHA utilized standard risk assessment models to estimate risks at lower levels of

exposure. It used these estimates as the basis for significant risk determinations at existing and proposed levels.

Many of the substances OSHA is regulating in this proposed rulemaking are acute hazards. Studies indicate that workers may suffer health effect at a level close to the current exposure limit, or if not exposure limit exists, at a specific level. With data close to the level of interest, extensive extrapolation is not necessary and the primary question is what level will substantially reduce or eliminate the significant risk identified.

OSHA is relying on guidelines already available in the literature, which use a variety of adjustments to account for the uncertainties inherent in translating animal or human health studies to the actual work situation. Adjustments are required due to inter-species and intra-species variations, sex and age differences, and the limited statistical power of typical studies. Statistical power is frequently limited because of the small number of test subjects, and the time constraints regarding single or repeated exposures. For the same reason the available studies may not support precise quantitative estimates of the probability of adverse health effects. In these situations, even where the presence of risk is well-established at the current level, it may not be possible to reach a conclusion concerning the precise point at which that risk ceases to be significant, in other words, how far exposure limits should be reduced. However, the evidence may be sufficient to justify a specific level as substantially reducing or eliminating significant risk.

The Supreme Court has indicated that the decision about whether a particular level of risk is significant will be based on largely on policy considerations. OSHA will include in the final rule only those limits which the record supports as being consistent with the statutory requirement that OSHA must follow. In carrying out this responsibility OSHA will consider the quality of the evidence, the type of risk, the reasonableness of the risk assessment or evidence of risk, the substantial reduction or elimination of significant risk, and the appropriateness of the final limit, taking into account statutory requirements, the cost and compliance factors, and policy considerations. Where more information is required to support a new final rule, the agency will collect it before revising existing limits or establishing new limits. Policy discretion is particularly appropriate where risk appears to be in the range where the question of its insignificance is presented.

OSHA is proposing to regulate 428 chemicals in this proceeding. In developing new PELs for some of these chemicals, questions may arise about the point at which the risks associated with them become insignificant. (See the discussion about formaldehyde quoted above.) EPA and FDA have recently considered analogous issues under other statutory authorities, specifically, what levels of risk are sufficiently low so that no further regulatory action is necessary or where there is more discretion whether to take action or not.

The FDA has indicated that in its view, risks of less than one in one-million from a lifetime of exposure are *de minimus*. See 51 FR 28344 at 362 August 7, 1986 and *Public Citizen* v. *Young*, 831 F. 2d 1108 (D.C. Cir., 1987). "FDA's proposed one-in-one million dividing point has been used by EPA to distinguish acceptable and unacceptable risks. 119 FR 46294 (1984) (general guideline); 51 FR 1602, 1635 (1986) (hazardous wastes). FDA has used the same break point to determine whether the general safety clause of the Act applies. 47 FR 14 138 (1982) "*Public Citizen*, Note 4 ibid. Public comment is invited on how OSHA should, as a matter of policy, address these issues.

Policy discretion concerning the appropriate extent of regulation if any may be particularly appropriate where risk appears to be in the range between clearly significant and *de minimus*, when the question of its insignificance is presented. One area of such discretion may be whether ancillary provisions are appropriate when risk is in this middle range. For example, medical screening tests may not be as effective, or the side effects may outweigh their value when the predicted risk is relatively low, and it is of value to focus medical resources where these have greater value. See, for example, discussion of medical surveillance for OSHA's arsenic standard, 43 FR 19620–1 (May 5, 1978) and benzene standard, 52 FR 34547–54 (September 11, 1987).

On the other hand, the Supreme Court stated in the benzene decision about medical surveillance that, "It should also be noted that, in setting a permissible exposure level in reliance on less-than-perfect methods, OSHA would have the benefit of a backstop in the form of monitoring the medical testing.

Thus, if OSHA properly determined that the permissible exposure limit should be set at 5 ppm, it could still require monitoring and medical testing for employees exposed to lower levels. By doing so, it could keep a constant check on the validity of the assumptions made in developing the permissible exposure limit, giving it a sound evidentiary basis for decreasing the limit if it was initially set too high. Moreover, in this way it could ensure that workers who were unusually susceptible to benzene could be removed from exposure before they had suffered any permanent damage.

* * * This is precisely the type of information-gathering function that Congress had in mind when in enacted section (6)(b)(7), which empowers the Secretary to require medical examinations to be furnished to employees exposed to certain hazards and potential hazards in order to most effectively determine whether the health of such employees is adversely affected by such exposure. See Legis. Hist., p. 147 (Emphasis added)" (448 U.S. 658). *Accord, National Cottonseed Prods. Assoc.* v. *Brock*, 829 F. 2d 482 (D.C. Cir. 1987), Cert. denied 56 U.S.L.W. 3733 (4/26/88).

OSHA is not raising the issue of the appropriateness of ancillary provisions in this rulemaking for reasons discussed above. However, this question may be of relevance to any comments on the issue of insignificant risk and regulatory discretion.

D. Basis for Identifying Replacement PEL's

OSHA's PEL's were based originally on established Federal standards and consensus standards, as provided by the Occupational Safety and Health Act. Those standards, in turn, were based largely upon air contaminant standards (Threshold Limit Values or TLV's) promulgated by the American Conference of Governmental Industrial Hygienists (ACGIH). Originally, ACGIH–TLV's were adopted for employees of government contractors, pursuant to the Walsh-Healey Act. At the time, they represented the most comprehensive set of workplace exposure guidelines available. They were then adopted under the OSHAct as established federal standards.

ACGIH updates its TLV's annually, adjusting exposure limits and adding new substances to its list as evidence of health effects is accumulated. During the past 20 years, a gap has developed between the OSHA PEL's and the more current ACGIH–TLV's because OSHA has approached standards-setting on an item-by-item basis and has not re-evaluated the PEL's listed in the 1910.1000 Z Tables as a group.

In the present rulemaking, OSHA had many more sources of standards from which to consider exposure limits, and a comparison of several of these sources was made early in the review process. In the review it became clear that two sorts of decisions, based on alternative

sources of standards, would be made. First, it was necessary to determine which substances would be included in the updating, since the health effects of many more substances are now known. Second, it was necessary to propose new limits for each substance which will provide protection to exposed workers.

OSHA examined lists of recommended exposure limits compiled by a variety of professional organizations, international bodies, governments and government agencies, using these criteria:

1. Comprehensiveness: What is the breadth (number of substances covered) and depth (inclusion of STEL's and ceilings as well as 8-hour TWA's) of the guidelines as a whole?
2. Currentness: How recent are the limits and how often are they updated?
3. Review Process: What procedures does he limit-setting body use to update its list?
4. Feasibility: To what extent is feasibility considered when limits are decided upon?
5. Documentation: What reasons are provided when limits are set and how well-documented are those decisions?
6. Applicability: Are the limits suitable for application in U.S. workplaces?

OSHA considered exposure limits set by professional organizations. (The American National Standards Institute, ANSI; The American Industrial Hygiene Association, AIHA; The American Conference of Governmental Industrial Hygienists, ACGIH), by NIOSH, by several countries (United Kingdom, Sweden, Federal Republic of Germany, and Japan), and three international bodies (European Economic Community, EEC; International Labor Organization, ILO; and the World Health Organization, WHO).

Not all of these limits could be evaluated against all of the criteria, since information was unavailable in some cases. Some organizations do not explain their reasons for setting guidelines, and others do not publish descriptions of their limit-setting and review procedures. OSHA nonetheless identified a group of exposure limit lists that warranted additional study, including the NIOSH Recommended Exposure Limits (REL's), WHO limits, British and West German limits and the TLV's established by ACGIH.

For the purpose of determining which substance would be included in the rulemaking, OSHA determined that the 1987–88 ACGIH–TLV list was most suitable. This TLV list included all of the substances in the present Z-tables, plus some two hundred more recently enrolled common substances. None of the other lists had equal depth and breadth. The number of hazardous substances included in other lists ranged from three (the EEC) to about 400 (United Kingdom), compared to over 600 substances in the ACGIH list. The NIOSH REL's covered a total of approximately 160 substances with numerical values.

OSHA then began its analysis of the particular exposure limits on the ACGIH list, comparing them with values suggested by other sources. All of the sources cited above indicated the need for updating, but inconsistencies in documentation of the recommended limits reduced the utility of most sources. For example, while the United Kingdom has many recommended limits, documentation by means of specific studies and description of methodology is often missing or inadequate for OSHA's purposes. In many instances, West German limits were based on the same 1968 documentation used by OSHA. Although West Germany has amended more than one hundred of the standards, documentation of feasibility and of risk has not been available to support the limits. OSHA believes that such documentation of feasibility would be especially necessary if German standards were to be adopted for application in U.S. workplaces, given the differing social, economic and political environments in the two countries.

NIOSH's recommended exposure limits (REL's) are well-documented and they are set with U.S. workplaces in mind. NIOSH explains the basis for its limits in criteria documents or in Current Intelligence Bulletins. OSHA has considered each of the available REL's in preparing this proposal, and has used the NIOSH-recommended exposure limit wherever it is shown to be both feasible and more appropriate than the corresponding ACGIH TLV. In each instance where there is a "significant difference" between a TLV and REL for an individual substance, OSHA has independently evaluated which value should be adopted as the PEL. In those few instances where REL's indicated limits for substances which were not on the ACGIH list, no new limit was proposed, because it was necessary to set a boundary on the number of substances to be evaluated. It was considered a higher priority to devote time to completing the regulation of 428 substances, rather than attempting to determine if additional substances, in other listings, should be considered. Where the REL and TLV differed in matters such as minor variations in TWA, lack of a STEL or ceiling, or use of a ceiling in lieu of a STEL, simplified procedures (described elsewhere) were adopted. These procedures considered the availability of feasibility data, practical sampling considerations, and the time available to complete this rulemaking. The criteria for defining significant differences between REL's and TLV's is discussed in section I–F.

Thus, the proposed modifications of OSHA's permissible exposure limits are based on the best information available to the agency at this time, regarding both health effects and feasibility, making use of the guidelines and recommendations developed by other organizations, primarily those of NIOSH and ACGIH. OSHA requests that the public provide any further relevant information regarding both health effects and feasibility which will supplement the information upon which this proposal is based.

E. Substances Included in the Update of § 1910.1000 Z Tables

Table I–E lists those 420 substances which are addressed by this proposal. This includes substances listed by the ACGIH in its publication "Threshold Limit Values and Biological Exposure Indices for 1987–88", excluding: (a) 24 substances for which comprehensive OSHA standards exist; (b) 9 substances for which OSHA standard development is in active progress; (c) and 160 substances from the existing Z-Tables where there is no difference between the OSHA PEL and the adopted 1987–88 ACGIH-TLV. This rulemaking therefore includes all substances which have been changed or added to the ACGIH TLV list since 1968. Changes or additions are considered to exist when: (1) A TWA or STEL has been modified; (2) a STEL or ceiling has been added (where none existed before); (3) where skin notation has been added; or (4) where some type of exposure limit (TWA, STEL or ceiling) exists where no OSHA limit existed in the § 1910.1000 Z-Tables.

Use of the TLV list as a reference point to define the bounds of this rulemaking is necessary to limit the number of substances under consideration to manageable size. Since the TLV list is the most extensive available source, and is the basis for the existing OSHA PEL's in the § 1910.1000 Z-Tables, it represents the best choice to serve this limited purpose.

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TABLE I-E. List of Substances for Which ACGIH Recommendation Differs From OSHA's Current Standard

H.S. Number/Chemical Name		CAS Number
1001	ACETALDEHYDE	75-07-0
1002	ACETIC ACID	64-19-7
1003	ACETIC ANHYDRIDE	108-24-7
1004	ACETONE	67-64-1
1005	ACETONITRILE	75-05-8
1006	ACETYLSALICYLIC ACID (ASPIRIN)	50-78-2
1007	ACROLEIN	107-02-8
1008	ACRYLAMIDE	79-06-1
1009	ACRYLIC ACID	79-10-7
1010	ALLYL ALCOHOL	107-18-6
1011	ALLYL CHLORIDE	107-05-1
1012	ALLYL GLYCIDYL ETHER (AGE)	106-92-3
1013	ALLYL PROPYL DISULFIDE	2179-59-1
1014	ALPHA-ALUMINA	1344-28-1
1015	ALUMINUM (ALKYLS)	7429-90-5
1016	ALUMINUM (METAL)	7429-90-5
1017	ALUMINUM (PYRO POWDERS)	7429-90-5
1018	ALUMINUM (SOLUBLE SALTS)	7429-90-5
1019	ALUMINUM (WELDING FUMES)	7429-90-5
1020	AMITROLE (3-AMINO-1,2,4-TRIAZOLE)	61-82-5
1021	AMMONIA	7664-41-7
1022	AMMONIUM CHLORIDE (FUME)	12125-02-9
1024	AMMONIUM SULFAMATE (AMMATE)	7773-06-0
1025	ANILINE	62-53-3
1028	ASPHALT FUMES	8052-42-4
1029	ATRAZINE	1912-24-9

TABLE I-E. List of Substances for Which ACGIH Recommendation Differs From OSHA's Current Standard (continued)

H.S. Number/Chemical Name		CAS Number
1031	BARIUM SULFATE	7727-43-7
1032	BENOMYL	17804-35-2
1033	BERYLLIUM & COMPOUNDS	7440-41-7
1034	BISMUTH TELLURIDE (SE-DOPED)	1304-82-1
1035	BISMUTH TELLURIDE (UNDOPED)	1304-82-1
1036	BORATES, TETRA, SODIUM (ANHYDROUS)	1303-96-4
1037	BORATES, TETRA, SODIUM (DECAHYDRATE)	1303-96-4
1038	BORATES, TETRA, SODIUM (PENTAHYDRATE)	1303-96-4
1039	BORON OXIDE	1303-86-2
1040	BORON TRIBROMIDE	10294-33-4
1041	BROMACIL	314-40-9
1042	BROMINE	7726-95-6
1043	BROMINE PENTAFLUORIDE	7789-30-2
1044	BUTANE	106-97-8
1045	2-BUTANONE (MEK)	78-93-3
1046	2-BUTOXY ETHANOL	111-76-2
1047	N-BUTYL ACETATE	123-86-4
1048	BUTYL ACRYLATE	141-32-2
1049	SEC-BUTYL ALCOHOL	78-92-2
1050	TERT-BUTYL ALCOHOL	75-65-0
1051	N-BUTYL ALCOHOL	71-36-3
1052	N-BUTYL GLYCIDYL ETHER	2426-08-6
1053	N-BUTYL LACTATE	138-22-7
1054	BUTYL MERCAPTAN	109-79-5
1055	O SEC-BUTYL PHENOL	89-72-5
1056	P-TERT-BUTYLTOLUENE	98-51-1

TABLE I-E. List of Substances for Which ACGIH Recommendation Differs From OSHA's Current Standard (continued)

H.S. Number/Chemical Name	CAS Number
1057 CALCIUM CARBONATE	1317-65-3
1058 CALCIUM CYANAMIDE	156-62-7
1059 CALCIUM HYDROXIDE	1305-62-0
1060 CALCIUM OXIDE	1305-78-8
1061 CALCIUM SILICATE, TOTAL DUST	NONE
1062 CALCIUM SULFATE	7778-18-9
1063 CAMPHOR (SYNTHETIC)	76-22-2
1064 CAPROLACTAM (DUST)	105-60-2
1065 CAPROLACTAM (VAPOR)	105-60-2
1066 CAPTAFOL (DIFOLATAN)	2425-06-1
1067 CAPTAN	133-06-2
1068 CARBOFURAN (FURADAN)	1563-66-2
1069 CARBON DIOXIDE	124-38-9
1070 CARBON DISULFIDE	75-15-0
1071 CARBON MONOXIDE	630-08-0
1072 CARBON TETRABROMIDE	558-13-4
1073 CARBON TETRACHLORIDE	56-23-5
1074 CARBONYL FLUORIDE	353-50-4
1075 CATECHOL (PYROCATECHOL)	120-80-9
1076 CELLULOSE	9004-34-6
1077 CESIUM HYDROXIDE	21351-79-1
1078 CHLORINATED CAMPHENE	8001-35-2
1079 CHLORINE	7782-50-5
1080 CHLORINE DIOXIDE	10049-04-4
1081 1 CHLORO-1 NITROPROPANE	600-25-9
1082 2 CHLORO-6-TRICHLOROMETHYL PYRIDINE (NITRAPYRIN)	1929-82-4

TABLE I-E. List of Substances for Which ACGIH Recommendation Differs From OSHA's Current Standard (continued)

H.S. Number/Chemical Name	CAS Number
1083 CHLOROACETYL CHLORIDE	79-04-9
1084 O-CHLOROBENZYLIDENE MALONONITRILE	2698-41-1
1085 CHLORODIFLUOROMETHANE	75-45-6
1086 CHLOROFORM	67-66-3
1087 CHLOROPENTAFLUOROETHANE	76-15-3
1088 CHLOROPRENE	126-99-8
1089 O-CHLOROSTYRENE	1331-28-8
1090 O-CHLOROTOLUENE	95-49-8
1091 CHLORPYRIFOS	2921-88-2
1092 CHROMIC ACID & CHROMATES	7440-47-3
1093 CHROMIUM, METAL	7440-47-3
1094 CHROMYL CHLORIDE	14977-61-8
1095 CLOPIDOL(COYDEN)	2971-90-6
1096 COAL DUST, < 5% QUARTZ	NONE
1097 COAL DUST, > 5% QUARTZ	NONE
1098 COBALT CARBONYL	10210-68-1
1099 COBALT HYDROCARBONYL	16842-03-8
1100 COBALT, METAL, FUME, DUST	7440-48-4
1101 COPPER (FUME)	7440-50-8
1102 CRAG HERBICIDE (SESONE)	136-78-7
1103 CRUFOMATE	299-86-5
1104 CYANAMIDE	420-04-2
1105 CYANOGEN	460-19-5
1106 CYANOGEN CHLORIDE	506-77-4
1107 CYCLOHEXANOL	108-93-0
1108 CYCLOHEXANONE	108-94-1

TABLE I-E. List of Substances for Which ACGIH Recommendation Differs From OSHA's Current Standard (continued)

H.S. Number	Chemical Name	CAS Number
1109	CYCLOHEXYLAMINE	108-91-8
1110	CYCLONITE	121-82-4
1111	CYCLOPENTANE	287-92-3
1112	CYHEXATIN	13121-70-5
1113	DDT	50-29-3
1114	DECABORANE	17702-41-9
1116	DI-SEC-OCTYL-PHTHALATE	117-81-7
1117	2,6-DI-TERT-BUTYL-P-CRESOL	128-37-0
1118	DIAZINON	333-41-5
1119	DIBUTYL PHOSPHATE	107-66-4
1120	2-N-DIBUTYLAMINOETHANOL	102-81-8
1121	1,1-DICHLORO-1-NITROETHANE	594-72-9
1122	1,3-DICHLORO-5,5-DIMETHYLHYDANTOIN	118-52-5
1123	DICHLOROACETYLENE	7572-29-4
1125	P-DICHLOROBENZENE	106-46-7
1126	1,1-DICHLOROETHANE	75-34-3
1127	DICHLOROETHYL ETHER	111-44-4
1128	DICHLOROMONOFLUOROMETHANE	75-43-4
1129	1,3-DICHLOROPROPENE	542-75-6
1130	2,2-DICHLOROPROPIONIC ACID	75-99-0
1131	DICROTOPHOS (BIDRIN)	141-66-2
1132	DICYCLOPENTADIENE	77-73-6
1133	DICYCLOPENTADIENYL IRON	102-54-5
1134	DIETHANOLAMINE	111-42-2
1135	DIETHYL KETONE	96-22-0
1136	DIETHYL PHTHALATE	84-66-2

TABLE I-E. List of Substances for Which ACGIH Recommendation Differs From OSHA's Current Standard (continued)

H.S. Number	Chemical Name	CAS Number
1137	DIETHYLAMINE	109-89-7
1138	DIETHYLENE TRIAMINE	111-40-0
1139	DIGLYCIDYL ETHER (DGE)	2238-07-5
1140	DIISOBUTYL KETONE	108-83-8
1141	DIMETHYL 1,2-DIBROMO-2,2-DICHLOROETHYL PHOSPHATE	300-76-5
1142	DIMETHYL SULFATE	77-78-1
1143	DIMETHYLANILINE	121-69-7
1144	DINITOLMIDE (3,5-DINITRO-O-TOLUAMIDE)	148-01-6
1145	DIOXANE (DIETHYLENE DIOXIDE)	123-91-1
1146	DIOXATHION (DELNAV)	78-34-2
1147	DIPHENYLAMINE	122-39-4
1148	DIPROPYL KETONE	123-19-3
1149	DIPROPYLENE GLYCOL METHYL ETHER	34590-94-8
1150	DIQUAT	85-00-7
1151	DISULFIRAM	97-77-8
1152	DISULFOTON	298-04-4
1153	DIURON	330-54-1
1154	DIVINYL BENZENE	108-57-6
1155	EMERY	112-62-9
1156	ENDOSULFAN	115-29-7
1158	EPICHLOROHYDRIN	106-89-8
1159	ETHANOLAMINE	141-43-5
1160	ETHION (NIALATE)	563-12-2
1161	ETHYL ACRYLATE	140-88-5
1162	ETHYL BENZENE	100-41-4
1163	ETHYL BROMIDE	74-96-4

TABLE I-E. List of Substances for Which ACGIH Recommendation Differs From OSHA's Current Standard (continued)

H.S. Number/Chemical Name		CAS Number
1164	ETHYL ETHER	60-29-7
1165	ETHYL MERCAPTAN	75-08-1
1166	ETHYL SILICATE	78-10-4
1167	ETHYLENE CHLOROHYDRIN	107-07-3
1168	ETHYLENE DICHLORIDE (1,2-DICHLOROETHANE)	107-06-2
1169	ETHYLENE GLYCOL	107-21-1
1170	ETHYLENE GLYCOL DINITRATE	628-96-6
1171	ETHYLIDENE NORBORNENE	16219-75-3
1172	N-ETHYLMORPHOLINE	100-74-3
1173	FENAMIPHOS	22224-92-6
1174	FENSULFOTHION (DASANIT)	115-90-2
1175	FENTHION	55-38-9
1176	FERBAM	14484-64-1
1177	FERROVANADIUM DUST	12604-58-9
1178	FIBROUS GLASS DUST	NONE
1179	FLUORINE	7782-41-4
1180	FLUOROTRICHLOROMETHANE	75-69-4
1181	FONOFOS	944-22-9
1182	FORMAMIDE	75-12-7
1183	FURFURAL	98-01-1
1184	FURFURYL ALCOHOL	98-00-0
1185	GASOLINE	8006-61-9
1186	GERMANIUM TETRAHYDRIDE	7782-65-2
1187	GLUTARALDEHYDE	111-30-8
1188	GLYCERIN (MIST)	56-81-5
1189	GLYCIDOL (2,3-EPOXY-1-PROPANOL)	556-52-5

TABLE I-E. List of Substances for Which ACGIH Recommendation Differs From OSHA's Current Standard (continued)

H.S. Number/Chemical Name		CAS Number
1190	GRAIN DUST	NONE
1191	GRAPHITE, NATURAL, RESPIRABLE	7782-42-5
1191A	GRAPHITE, SYNTHETIC	NONE
1192	GYPSUM, TOTAL DUST	NONE
1194	N-HEPTANE	142-82-5
1195	HEXACHLOROBUTADIENE	87-68-3
1196	HEXACHLOROCYCLOPENTADIENE	77-47-4
1197	HEXACHLOROETHANE	67-72-1
1198	HEXAFLUOROACETONE	684-16-2
1200	N-HEXANE	110-54-3
1201	HEXANE ISOMERS	NONE
1202	2-HEXANONE	591-78-6
1203	HEXONE (METHYL ISOBUTYL KETONE)	108-10-1
1204	HEXYLENE GLYCOL	107-41-5
1205	HYDRAZINE	302-01-1
1206	HYDROGEN BROMIDE	10035-10-6
1207	HYDROGEN CYANIDE	74-90-8
1208	HYDROGEN FLUORIDE	7664-39-3
1209	HYDROGEN SULFIDE	7783-06-4
1210	HYDROGENATED TERPHENYLS	61788-32-7
1211	2-HYDROXYPROPYL ACRYLATE	999-61-1
1212	INDENE	95-13-6
1213	INDIUM & COMPOUNDS	7440-74-6
1214	IODOFORM	75-47-8
1215	IRON OXIDE (DUST AND FUME)	1309-37-1
1216	IRON PENTACARBONYL	13463-40-6

TABLE I-E. List of Substances for Which ACGIH Recommendation Differs From OSHA's Current Standard (continued)

H.S. Number/Chemical Name	CAS Number
1243 MESITYL OXIDE	141-79-7
1244 METHACRYLIC ACID	79-41-4
1245 METHOMYL (LANNATE)	16752-77-5
1246 METHOXYCHLOR	72-43-5
1247 4-METHOXYPHENOL	150-76-5
1248 METHYL 2-CYANOACRYLATE	137-05-3
1249 METHYL ACETATE	79-20-9
1250 METHYL ACETYLENE/PROPADIENE MIXTURE	74-99-7
1251 METHYL ACRYLONITRILE	126-98-7
1252 METHYL ALCOHOL	67-56-1
1253 METHYL BROMIDE	74-83-9
1254 METHYL CHLORIDE	74-87-3
1255 METHYL CHLOROFORM (1,1,1-TRICHLOROETHANE)	71-55-6
1256 METHYL DEMETON	8022-00-2
1257 METHYL ETHYL KETONE PEROXIDE	1338-23-4
1258 METHYL FORMATE	107-31-3
1259 METHYL IODIDE	74-88-4
1260 METHYL ISOAMYL KETONE	110-12-3
1261 METHYL ISOBUTYL CARBINOL	105-30-6
1262 METHYL ISOPROPYL KETONE	563-80-4
1263 METHYL MERCAPTAN	74-93-1
1264 METHYL N-AMYL KETONE	110-43-0
1265 METHYL PARATHION	298-00-0
1266 METHYL SILICATE	681-84-5
1267 ALPHA METHYL STYRENE	98-83 9
1268 METHYLCYCLOHEXANE	108-87-2

TABLE I-F. List of Substances for Which ACGIH Recommendation Differs From OSHA's Current Standard (continued)

H.S. Number/Chemical Name	CAS Number
1217 IRON SALTS (SOLUBLE)	NONE
1218 ISOAMYL ALCOHOL	123-51-3
1219 ISOBUTYL ALCOHOL	78-83-1
1220 ISOOCTYL ALCOHOL	26952-21-6
1221 ISOPHORONE	78-59-1
1222 ISOPHORONE DIISOCYANATE	4098-71-9
1223 2-ISOPROPOXYETHANOL	109-59-1
1224 ISOPROPYL ACETATE	108-21-4
1225 ISOPROPYL ALCOHOL	67-63-0
1226 ISOPROPYL ETHER	108-20-3
1227 ISOPROPYL GLYCIDYL ETHER	4016-14-2
1228 ISOPROPYLAMINE	75-31-0
1229 N-ISOPROPYLANILINE	643-28-7
1230 KAOLIN, TOTAL DUST	NONE
1231 KETENE	463-51-4
1232 LIMESTONE, TOTAL DUST	NONE
1233 MAGNESITE, TOTAL DUST	NONE
1234 MAGNESIUM OXIDE FUME	1309-48-4
1235 MALATHION	121-75-5
1236A MANGANESE, FUME	7439-96-5
1237 MANGANESE CYCLOPENTADIENYL TRICARBONYL	12079-65-1
1238 MANGANESE TETROXIDE	1317-35-7
1239 MARBLE, TOTAL DUST	1317-65-3
1240 MERCURY (ARYL AND INORGANIC COMPOUNDS)	7439-97-6
1241 MERCURY (VAPOR)	7439 97 6
1242 MERCURY, (ORGANO) ALKYL COMPOUNDS	7439-97-6

TABLE I-E. List of Substances for Which ACGIH Recommendation Differs From OSHA's Current Standard (continued)

H.S. Number/Chemical Name		CAS Number
1269	METHYLCYCLOHEXANOL	25639-42-3
1270	O-METHYLCYCLOHEXANONE	583-60-8
1271	METHYLCYCLOPENTADIENYL MN TRICARBONYL	12108-13-3
1272	METHYLENE BIS (4-CYCLOHEXYLISOCYANATE)	5124-30-1
1273	4,4'-METHYLENE BIS(2-CHLOROANILINE)	101-14-4
1275	METRIBUZIN	21087-64-9
1276	MICA	12003-38-2
1277	MINERAL WOOL FIBER	NONE
1278	MOLYBDENUM (INSOLUBLE COMPOUNDS)	7439-98-7
1279	MONOCROTOPHOS (AZODRIN)	6923-22-4
1280	MONOMETHYL ANILINE	100-61-8
1281	MORPHOLINE	110-91-8
1282	NAPHTHALENE	91-20-3
1283	NICKEL (SOLUBLE COMPOUNDS)	7440-02-0
1284	NICKEL CARBONYL	13463-39-3
1286	NITRIC ACID	7697-37-2
1287	P-NITROANILINE	100-01-6
1288	P-NITROCHLOROBENZENE	100-00-5
1289	NITROGEN DIOXIDE	10102-44-0
1290	NITROGLYCERIN	55-63-0
1291	2-NITROPROPANE	79-46-9
1292	NITROTOLUENE	1321-12-6
1293	NONANE	111-84-2
1294	NUISANCE PARTICULATES, TOTAL DUST	NONE
1295	OCTACHLORONAPHTHALENE	2234-13-1
1296	OCTANE	111-65-9

TABLE I-E. List of Substances for Which ACGIH Recommendation Differs From OSHA's Current Standard (continued)

H.S. Number/Chemical Name		CAS Number
1297	OIL MIST (MINERAL)	8012-95-1
1298	OSMIUM TETROXIDE	20816-12-0
1299	OXALIC ACID	144-62-7
1300	OXYGEN DIFLUORIDE	7783-41-7
1301	OZONE	10028-15-6
1302	PARAFFIN WAX FUME	8002-74-2
1303	PARAQUAT, RESPIRABLE DUST	4685-14-7
1304	PENTABORANE	19624-22-7
1305	PENTAERYTHRITOL, TOTAL DUST	115-77-5
1306	PENTANE	109-66-0
1307	2-PENTANONE (METHYL PROPYL KETONE)	107-87-9
1308	PERCHLOROETHYLENE	127-18-4
1309	PERCHLORYL FLUORIDE	7616-94-6
1310	PERLITE	NONE
1312	PETROLEUM DISTILLATES (NAPHTHA)	8002-05-9
1313	PHENOTHIAZINE	92-84-2
1314	PHENYL ETHER (VAPOR)	101-84-8
1315	PHENYL GLYCIDYL ETHER	122-60-1
1316	PHENYL MERCAPTAN	108-98-5
1317	PHENYLHYDRAZINE	100-63-0
1318	PHENYLPHOSPHINE	638-21-1
1319	PHORATE (THIMET)	298-02-2
1320	PHOSDRIN (MEVINPHOS)	7786-34-7
1321	PHOSPHINE	7803-51-2
1322	PHOSPHORIC ACID	7664-38-2
1323	PHOSPHORUS OXYCHLORIDE	10025-87-3

TABLE I-E. List of Substances for Which ACGIH Recommendation Differs From OSHA's Current Standard (continued)

H.S. Number/Chemical Name	CAS Number
1324 PHOSPHORUS PENTASULFIDE	1314-80-3
1325 PHOSPHORUS TRICHLORIDE	7719-12-1
1326 PHTHALIC ANHYDRIDE	85-44-9
1327 M-PHTHALODINITRILE	626-17 5
1328 PICLORAM (TORDOM)	1918-02-1
1329 PICRIC ACID	88-89 1
1330 PIPERAZINE DIHYDROCHLORIDE	142-64-3
1331 PLASTER OF PARIS, TOTAL DUST	NONE
1332 PLATINUM, METAL	7440-06-4
1333 PORTLAND CEMENT	65997-15-1
1334 POTASSIUM HYDROXIDE	1310-58-3
1335 PROPARGYL ALCOHOL	107-19-7
1336 PROPIONIC ACID	79-09-4
1337 PROPOXUR (BAYGON)	114-26-1
1338 N-PROPYL ACETATE	109-60-4
1339 PROPYL ALCOHOL	71-23-8
1340 N-PROPYL NITRATE	627-13-4
1341 PROPYLENE DICHLORIDE	78-87-5
1342 1,2-PROPYLENE GLYCOL DINITRATE	6423-43-4
1343 PROPYLENE GLYCOL MONOMETHYL ETHER	107-98-2
1344 PROPYLENE OXIDE	75-56-9
1346 RESORCINOL	108-43-3
1347 RHODIUM (METAL FUME & INSOLUBLE COMPOUNDS)	7440-16 6
1348 RHODIUM (SOLUBLE SALTS)	7440-16-6
1349 RONNEL	299 84 3
1350 ROSIN CORE SOLDER PYROLYSIS PRODUCT (AS HCHO)	NONE

TABLE I-E. List of Substances for Which ACGIH Recommendation Differs From OSHA's Current Standard (continued)

H.S. Number/Chemical Name	CAS Number
1351 ROUGE, TOTAL DUST	NONE
1352 SILICA, AMORPHOUS, DIATOMACEOUS EARTH	68855-54-9
1353 SILICA, AMORPHOUS, PRECIPITATED OR GEL	NONE
1354 SILICA, CRYSTALLINE-CRISTOBALITE	14464-46-1
1355 SILICA, CRYSTALLINE QUARTZ, RESPIRABLE	14808-60-7
1356 SILICA, CRYSTALLINE TRIDYMITE	15468-32-3
1357 SILICA, CRYSTALLINE TRIPOLI (AS QUARTZ DUST)	1317-95-9
1358 SILICA, FUSED	60676-86-0
1359 SILICON	7440-21-3
1360 SILICON CARBIDE	409-21-2
1361 SILICON TETRAHYDRIDE	7803-62-5
1362 SILVER, METAL, DUST, AND FUME	7440-22-4
1363 SOAPSTONE, TOTAL DUST	NONE
1363A SOAPSTONE, RESPIRABLE DUST	NONE
1364 SODIUM AZIDE	26628-22-8
1365 SODIUM BISULFITE	7631-90-5
1366 SODIUM FLUOROACETATE	62-74-8
1367 SODIUM HYDROXIDE	1310-73-2
1368 SODIUM METABISULFITE	7681-57-4
1369 STARCH, TOTAL DUST	NONE
1371 STODDARD SOLVENT	8052-41-3
1372 STYRENE (PHENYLETHYLENE)	100-42-5
1373 SUBTILISINS (PROTEOLYTIC ENZYMES)	9014-01-1
1374 SUCROSE, TOTAL DUST	57-50-1
1375 SULFUR DIOXIDE	7446 09 5
1376 SULFUR MONOCHLORIDE	10025-67 9

TABLE I-E. List of Substances for Which ACGIH Recommendation Differs From OSHA's Current Standard (continued)

H.S Number/Chemical Name		CAS Number
1377	SULFUR PENTAFLUORIDE	5714-22-7
1378	SULFUR TETRAFLUORIDE	7783-60-0
1379	SULFURYL FLUORIDE	2699-79-8
1380	SULPROFOS	35400-43-2
1381	TALC (NON-ASBESTIFORM)	14807-96-6
1382	TANTALUM	7440-25-7
1383	TEMEPHOS	3383-96-8
1384	TERPHENYLS	26140-60-3
1385	1,1,2,2-TETRACHLOROETHANE	79-34-5
1386	TETRAETHYL LEAD	78-00-2
1387	TETRAHYDROFURAN	109-99-9
1388	TETRAMETHYL LEAD	75-74-1
1389	TETRASODIUM PYROPHOSPHATE	7722-88-5
1391	4,4'-THIOBIS (6-TERT-BUTYL-M-CRESOL)	96-69-5
1392	THIOGLYCOLIC ACID	68-11-1
1393	THIONYL CHLORIDE	7719-09-7
1394	TIN (ORGANIC COMPOUNDS)	7440-31-5
1395	TIN OXIDE	18282-10-5
1396	TITANIUM DIOXIDE	13463-67-7
1397	TOLUENE	108-88-3
1398	TOLUENE-2,4-DIISOCYANATE	584-84-9
1399	O-TOLUIDINE	95-53-4
1400	P-TOLUIDINE	106-49-0
1401	M-TOLUIDINE	108-44-1
1402	TRIBUTYL PHOSPHATE	126-73-8
1403	1,1,2 TRICHLORO-1,2,2 TRIFLUOROETHANE	76-13-1

TABLE I-E. List of Substances for Which ACGIH Recommendation Differs From OSHA's Current Standard (continued)

H.S. Number/Chemical Name		CAS Number
1404	TRICHLOROACETIC ACID	76-03-9
1405	1,2,4-TRICHLOROBENZENE	120-82-1
1406	TRICHLOROETHYLENE	79-01-6
1407	1,2,3-TRICHLOROPROPANE	96-18-4
1408	TRIETHYLAMINE	121-44-8
1409	TRIMELLITIC ANHYDRIDE	552-30-7
1410	TRIMETHYL PHOSPHITE	121-45-9
1411	TRIMETHYLAMINE	75-50-3
1412	TRIMETHYLBENZENE	25551-13-7
1413	2,4,6-TRINITROTOLUENE (TNT)	118-96-7
1414	TRIORTHOCRESYL PHOSPHATE	78-30-8
1415	TRIPHENYL AMINE	603-34-9
1416	TUNGSTEN & COMPOUNDS (INSOLUBLE)	7440-33-7
1417	TUNGSTEN & COMPOUNDS (SOLUBLE)	7440-33-7
1418	URANIUM (INSOLUBLE COMPOUNDS)	7440-61-1
1419	URANIUM (SOLUBLE COMPOUNDS)	7440-61-1
1420	N-VALERALDEHYDE	110-62-3
1421	VANADIUM (V_2O_5, DUST)	7440-62-2
1422	VANADIUM (V_2O_5, FUME)	7440-62-2
1423	VEGETABLE OIL MIST	NONE
1424	VINYL ACETATE	108-05-4
1425	VINYL BROMIDE	593-60-2
1426	VINYL CYCLOHEXENE DIOXIDE	106-87-6
1427	VINYL TOLUENE	25013-15-4
1428	VINYLIDENE CHLORIDE	75-35-4
1429	VM & P NAPHTHA	8032-32-4

TABLE I-E. List of Substances for Which ACGIH Recommendation Differs From OSHA's Current Standard (continued)

H.S. Number/Chemical Name	CAS Number
1430 WELDING FUMES (TOTAL PARTICULATE)	NONE
1430a WOOD DUST, HARD WOOD	NONE
1430b WOOD DUST, SOFT WOOD	NONE
1431 XYLENE (O,M,P-ISOMERS)	1330-20-7
1432 M-XYLENE-ALPHA,ALPHA'-DIAMINE	1477-55-0
1433 XYLIDINE	1300-73 8
1434 ZINC STEARATE	557-05-1
1435 ZINC CHLORIDE FUME	7646-85-7
1436 ZINC CHROMATES (CrVI)	13530-65-9
1437 ZINC OXIDE (FUME)	1314-13-2
1438 ZINC OXIDE, TOTAL DUST	1314-13-2
1439 ZIRCONIUM COMPOUNDS	7440-67-7

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F. Identification of Substances Requiring Special Attention

As previously noted, the substances to be considered in this proposed rulemaking are defined by the aodpted 1987–88 ACGIH TLV's. The use of some boundary for this rulemaking is necessary to avoid the impossible task of considering over 100,000 chemicals which exist in U.S. workplaces. Use of a single source to identify such a boundary is necessary to provide a clearly defined reference point for additional analyses. Since the 1968 ACGIH TLV's were used (through the Walsh-Healey Act) as the primary basis for the existing Z-Tables adopted in 1971, and since the 1987–88 TLV's represent the most extensive data base available (as described in a previous section of the preamble), selection of the TLV's to satisfy the objective of setting a boundary, is appropriate.

While the adopted 1987–88 ACGIH TLV's define the boundaries for this proposed rulemaking, the OSHA analysis (previously described) will consider both the 1987–88 ACGIH TLV's and NIOSH REL's as the potential alternatives for selecting replacements for the existing OSHA PEL's noted in the § 1910.1000 Z-Tables, or expansion of these Z-Tables.

Twenty or more years have elapsed since the TLV's forming the basis for OSHA's PEL values were set. During the interim, a great deal of knowledge has been gained regarding the hazards of these and other substances generally found in the workplace. Control technologies have evolved during this same time, altering previous standards of feasibility for workplace exposures.

OSHA realizes that while this proposal represents a major improvement in health protection, it is also necessary that OSHA evaluate the appropriateness of the individual TLV's or REL's OSHA should also identify any individual substances which will require: (1) Special attention as part of this rulemaking, or (2) follow-up rulemaking activities.

Of the 428 substances considered in this proposed rulemaking, approximately 75 involved changes to the existing OSHA TWA; approximately 70 involve the addition of, or changes to, a STEL in conjunction with an existing PEL; approximately 25 involve changes to both the TWA and STEL; and 205 involve establishment of some allowable exposure limit where none existed before. These four categories cover most of the 428 substances to be covered by this proposal.

In most instances, proposed new PELs establish lower permissible exposure limits or establish exposure limits where none existed before. In a very limited number of instances, however, the permissible exposure limits could be increased in keeping with the best current judgment regarding toxicity, hazard, and risk for the specific substance in question. In those instances, OSHA will ascertain that the legal requirements for increasing an established PEL are satisfied. The details of this requirement are noted in that part of section IV of this preamble dealing with substances falling within this category.

Since two independent sources will be used to develop new PEL's it is necessary to indicate what criteria will be used to identify substances where the TLV and REL "differ significantly." These substances will undergo additional analyses to resolve the different recommendations. For this rulemaking a "significant difference" is presumed *NOT* to exist when:

(a) The TLV and REL values are the same.

(b) TLV and REL values differ by less than 10%.

(c) The TLV and REL Time Weighted Averages (TWA) are the same, but there are differences in the Short Term Exposure Limit (STEL) or Ceiling (C).

(d) The TWA in one data base is the same, or one-half, the STEL/C in the other data base.

OSHA realizes that (c) and (d) represent simplification of the standard setting analysis. For both (b) and (c) situations, the TLV values will be used since they represent a logical extension of the existing Z-Tables, which are based on the 1968 TLV's. This approach is intended to facilitate the proposed changes for a large number of substances. Evidence may be introduced into the record which will permit further analysis of these situations. OSHA will consider comments on this procedure prior to final rulemaking. Based on comments, priorities and administrative resources, OSHA will consider if proposing further refinements in the future is appropriate.

For situation (d), the data base providing a TWA value will be used, since this type of limit represents a more typical exposure monitoring situation. This will result in the adoption of PEL's based either on a TLV or REL.

On the basis of these criteria, Table I–F–A lists 35 substances where there is a "significant difference" between the TLV and the REL, and the REL is lower than the TLV. Table I–F–B lists 9 substances for which there is a "significant difference", and the REL is higher than the TLV. Individual evaluations will be performed by OSHA for each of the substances listed in these Tables.

The approach of using primarily exposure limits from two well-established sets of guidelines carries with it the responsibility for OSHA to determine also which of these limits is appropriate in light of the statutory and legal requirements that OSHA must satisfy. OSHA describes in the following text these circumstances where consideration of other factors may be appropriate. OSHA requests comments on these proposed circumstances and/or the alternate procedures for handling these situations (described in section I–G).

To satisfy these requirements, and complete the objectives of revision and expansion for a large number of exposure limits in a reasonable time period, OSHA will identify two special situations by comparing the ACGIH TLV's with exposure limit guidelines developed by: (1) United Kingdom 1987 Occupational Exposure Limits (OEL); (2) West German—1985 Maximum Allowable Concentrations (MAK); (3) Japanese Permissible Exposure Limits—1983; (4) Swedish-Allowable Workplace Air Concentrations—1984. OSHA will consider the significance of differences among these sources, and take appropriate action in each case. Use of the TLV's as a reference point for this comparison is dictated by the fact that the TLV's define the boundary for the substances to be considered in this proposed rulemaking, and any significant differences between the TLV's and REL's are considered in the evaluation previously noted for substances listed in Tables I–F–A and I–F–B.

OSHA proposes to identify these two special situations as follows:

(a) At least three of the four alternate data bases recommend allowable exposure limits that are less than the 1987–88 ACGIH-TVL's. Five substances fall into this category, and are identified in Table I–F–C. This criterion avoids undue consideration of any single low proposed exposure limit, and identifies those instances where there is reasonable uncertainty regarding the proper levels which will improve worker protection. One of these substances noted in Table I–F–C is also identified in Table I–F–B.

(b) At least three of the four alternate data bases recommend allowable exposure limits which are *greater* than the 1987–88 ACGIH-TLV's. Ten substances fall into this category, and these are identified in Table I–F–D. Two of the substances noted in Table I–F–D are also noted in Table I–F–B. and four

of these substances are listed in Table I–F–A.

Special situations are also indicated for the following three circumstances:

(c) The 1987–88 TLV exceeds the existing PEL. When considering these substances, OSHA will ascertain that the legal requirements detailed in the cotton dust preamble (50 FR 511 32–3, Dec. 13, 1985) for increasing or eliminating an existing PEL are satisfied. Analyses of these situations are detailed in that part of section IV which discusses substances for which current ACGIH TLV's are less stringent than current OSHA limits.

(d) The available analytical and sampling methods are not adequate to measure the air concentrations of a specific chemical. OSHA has reviewed this concern and has identified 7 substances for which adequate sampling and analytical methods are not available. These are listed in Table I-F-E and details regarding analytical procedures are provided in Appendix A. OSHA will consider if rulemaking can proceed for such substances, with final implementation being deferred until OSHA or others develop the necessary sampling and analytical procedures.

(e) Substances for which recent information suggests that the 1987–88 TLVs or REL's may not be appropriate. OSHA will consider this aspect prior to final rulemaking if the hearing record provides relevant additional information. This category may include substances such as carcinogens, which have implicit or explicit recommendations to maintain exposures to levels which are: As low as feasible; as low as detectable; minimal; as low as practical; etc. Clearly, the use of detection limits as a PEL does not fully consider the feasibility issue. OSHA preliminarily believes that such substances cannot be considered in this rulemaking without greatly extending the decision process, and inordinately delaying changing the PEL's for 428 substances. Therefore, it would be more appropriate to proceed only on the basis of available, quantitative information provided by the TLV's or REL's. OSHA intends future consideration, using individual rulemaking, for such substances.

OSHA invites public comments on the five special situations identified. OSHA also is interested in learning of any chemicals which should be added to, or deleted from the listing in Tables I-F-A through I-F-E. Such suggestions should clearly indicate the basis and supporting data for any requested changes.

Table I-F-A

Significant Difference between REL and TLV REL lower than TLV

1. Acetone
2. Acretonitrile
3. Beryllium and its compounds
4. n-Butyl glycidyl ether
5. Carbon disulfide
6. Carbon monoxide
7. Carbon tetrachloride
8. Chlorine
9. Chloroform
10. Chloroprene
11. Chromic acid and chromates, non-carcinogenic
12. Dioxane
13. Ethylene dichloride
14. Ethylene glycol dinitrate
15. Fibrous glass dust
16. n-Heptane
17. Hexane Isomers
18. 2-Hexanone (methyl n-butyl ketone)
19. Hydrazine
20. Hydrogen cyanide
21. Mercury (aryl and inorganic compounds)
22. Mesityl oxide
23. Methyl propyl ketone
24. Nickel (soluble compounds)
25. Nitroglycerin
26. Nitrogen dioxide
27. Octane
28. Pentane
29. Petroleum distillates (naphtha)
30. Phenylhydrazine
31. Silica, crystalline quartz, respirable
32. Sulfur dioxide
33. Trichloroethylene
34. Vinyl acetate
35. Zinc chromate

Table I–F–B

Significant Difference Between REL and TLV REL Higher than TLV

1. Acrylamide
2. Ammonia
3. Carbon dioxide
4. Furfuryl alcohol
5. n–Hexane
6. Isophorone diisocyanate
7. Malathion
8. Methyl n-amyl ketone
9. Stoddard solvent

Table I–F–C

Substances Requiring Special Attention

At least three data base exposure limits are less than the ACGIH TLV's.

1. Furfuryl alcohol
2. Methyl chloroform
3. Selenium
4. Tetraethyl lead
5. Tetramethyl lead

Table I–F–D

Substances Requiring Special Attention

At least three data base exposure limits are greater than the ACGIH TLV's.

1. Acrylamide
2. Carbon tetrachloride
3. Cobalt
4. Ethylene glycol dinitrate
5. Furfural
6. n–Hexane
7. Methylene chloride
8. Nitroglycerin
9. Toluene di-isocyanate
10. Zinc chromate

Table I–F–E

Substances Requiring Special Attention

Inadequate Analytical or Sampling Methods

1. Aluminum alkyls
2. Cyanamide
3. Ethylidene norbornene
4. Hexafluoracetone
5. Mercury (alkyl compounds)
6. Subtilisins
7. Sulfur pentafluoride

G. Alternate Procedures for Dealing with Substances Requiring Special Attention

In previous sections of this preamble, OSHA has indicated an approach where different recommendations exist for an individual TLV or REL. For these substances an extended analysis of the health and feasibility data was performed to determine which level will be proposed. Evaluation of feasibility and significance of risk are statutory requirements which OSHA must follow. The absence of any evidence indicating these requirements are satisfied would preclude OSHA from adopting a PEL. Therefore, permissible exposure levels cannot be adopted if they are primarily based on limits of detection, or similar concepts, unless OSHA's legal requirements are satisfied.

OSHA is also considering three possible alternative procedures for the substances categorized according to the criteria previously described, as requiring special attention (Tables I–F–C, and I–F–D). Any additional substances which may be identified during the rulemaking process as requiring special attention may receive similar treatment.

The first alternative would be to review in depth all the available health effects data for each of these substances, the data and reasoning which lead to the different allowable exposure limits, the criteria of the organization providing each

recommendation, relative dates of adoption, and the applicability of supporting data to typical workplaces in the United States. Based on this analysis, OSHA could adopt either the REL, TLV or some other recommended or other allowable exposure limit. This would require preparation of numerous mini-standards, a major time commitment by OSHA, and would probably result in a significant delay in the adoption of 428 new or revised permissible exposure limits proposed in this rulemaking.

A second approach would be to identify the substances noted in Section I–F–, Tables C, and D, (plus any additional substances identified during the rulemaking process) as requiring possible individual follow-up evaluation and possible separate rulemaking but adopt the proposed PEL or another level if clear evidence is presented to the record. This approach would: permit prompt updating and expansion of all the PEL's noted in the Z–Tables on the basis of either the TLV or REL; use the current rulemaking analyses to identify chemicals requiring priority consideration regarding their PEL; and reduce conflicts among the several allowable exposure guidelines which currently face many industrial health protection organizations. Future rulemaking based on OSHA priorities could consider the necessary and appropriate refinements based on all the available evidence.

A third approach would be to maintain the current OSHA PEL's for the substances noted in Section I–F, Tables C, and D (plus any additional substances identified during the rulemaking process), while revising the remaining substances on the basis of TLV or REL guidance. These unchanged substances would then be identified for future follow-up evaluations and possible separate rulemaking under 6(b) provisions. This is similar to the second approach but without benefit of the updating process.

OSHA invites comments on these three options and others, so they can be considered in the final rulemaking. OSHA initially believes that the second approach is the best option since it promptly provides health protection for over 400 substances of concern, when the evidence indicates it is needed.

The substances identified in Table I–F–E, where OSHA is not aware of an adequate analytical method, present a different situation. OSHA is considering issuing the proposed new PEL's if supported by the record on health and feasibility grounds. OSHA would also consider staying these PEL's until an analytical procedure becomes available. OSHA requests comments on this, or alternate approaches.

H. Construction, Maritime and Agriculture Segments

Currently the exposure limits which apply to construction are the ACGIH Threshold Limit Values of Air Contaminants for 1970 and certain substance specific section 6(b) standards. See 29 CFR 1926.55, 58 and 29 CFR 1910.19. OSHA is to consult with the Advisory Committee on Construction Safety and Health prior to proposing new standards that have a major impact on construction. See 29 CFR 1911.10(a). OSHA is in the process of consulting with the Construction Advisory Committee. After receiving their recommendations, OSHA intends to propose amendments covering exposures to toxic substances in construction reflecting the facts in this proposal and the views of the Construction Advisory Committee.

Parts 1916, 1917 and 1918 of 29 CFR cover, respectively, employment in shipyards, marine terminals and longshoring. Part 1916 for shipyards references the 1970 TLV's of the ACGIH. See 29 CFR 1915.5 and 1915.12 (b)(3). Part 1917 for marine terminals references the current Z–Tables. See 29 CFR 1917.2(p), and 1917.23. Part 1918 for longshoring refers to "dangerous gaseous contaminants not immediately dangerous to life" and "heavy concentrations of dusts." See 29 CFR 1918.93 (e) and (f). Certain substance specific section 6(b) standards also cover these industries. See 29 CFR 1910.19.

OSHA, as part of the rulemaking covering construction, intends to consider applying this proposal to the maritime sectors.

Subpart Z of 29 CFR Part 1910, and the included Z–Tables specifically do not apply to Agriculture. See 29 CFR 1928.21(b). In addition, many of the chemicals which affect agriculture are pesticides regulated by the EPA over which OSHA may not have jurisdiction, pursuant to section 4(b)(1) of the OSHA Act. In the future OSHA will consider based on relevance, priorities and administrative resources if it is appropriate to consider coverage for agriculture.

II. Pertinent Legal Authority

The publication of a final standard is authorized by sections 6 and 8 of the Occupational Safety and Health Act of 1970 (the Act), 29 U.S.C. 655 and 657. Section 6(b)(5) governs the issuance of occupational safety and health standards dealing with toxic materials or harmful physical agents. It states:

> The Secretary in promulgating standards dealing with toxic materials or harmful physical agents under this subsection shall set the standard which most adequately assures, to the extent feasible, on the basis of the best available evidence, that no employee will suffer material impairment of health or functional capacity even if such employee has regular exposure to the hazard dealt with by such standard for the period of his working life. Development of standards under this subsection shall be based upon research, demonstrations, experiments, and such other information as may be appropriate. In addition to the attainment of the highest degree of health and safety protection for the employee, other considerations shall be the latest available scientific data in the field, the feasibility of standards, and experience gained under this and other health and safety laws. Whenever practicable, the standard promulgated shall be expressed in terms of objective criteria and of the performance desired.

Section 3 (8) defines an occupational safety and health standard as "a standard which requires conditions, or the adoption or use of one or more practices, means, methods, operations or processes, reasonably necessary or appropriate to provide safe or healthful employment and places of employment."

The Supreme Court has held under the Act that the Secretary, before issuing any new standard, must determine that it is reasonably necessary and appropriate to remedy a significant risk of material health impairment. *Industrial Union Department* v. *American Petroleum Institute*, 448 U.S. 607 (1980). The Court stated that "* * * before he can promulgate any permanent health or safety standard, the Secretary is required to make a threshold finding that a place of employment is unsafe in the sense that significant risks are present and can be eliminated or lessened by a change in practices" (448 U.S. at 642). The Court also stated "that the Act does limit the Secretary's power to require the elimination of significant risk" (448 U.S. 644, n. 19).

The Court indicated, however, that the significant risk determination is "not a mathematical straitjacket," and that "OSHA is not required to support its finding that a significant risk exists with anything approaching scientific certainty." The Court ruled that "a reviewing court [is] to give OSHA some leeway where its findings must be made on the frontiers of scientific knowledge (and that) * * * the Agency is free to use conservative assumptions in interpreting the data with respect to carcinogens, risking error on the side of over protection rather than under protection" (448 U.S. at 655, 655).

The Court also stated that "while the Agency must support its finding that a certain level of risk exists with substantial evidence, we recognize that its determination that a particular level of risk is 'significant' will be based largely on policy considerations." (488 U.S. at 655, n. 62).

After OSHA determines that a significant risk exists and that such risk can be reduced or eliminated by the proposed standard, it must set a standard which is technologically and economically feasible. In *American Textile Manufacturers Institute* v. *Donovan*, 452 U.S. 490 (1981) the Supreme Court held that "cost-benefit analysis is not required by statute because feasibility analysis is" (452 U.S. 531, n. 32). For non-threshold toxic substances, the aim is to set the lowest feasible level necessary to eliminate significant risk.

As stated above in the history and approach sections of the preamble, OSHA has concluded that updating the Z-tables to reflect recent information is the highest priority for the Agency. This will reduce exposure limits for approximately 200 substances regulated currently by the Z-tables and add exposure limits for approximately 200 substances. The health; literature indicates this must be accomplished to improve worker health: it is one of Congress's concurrent goals and will greatly increase occupational health protection for a very large number of workers.

In order to accomplish this high priority task in a reasonable time in the light of limited administrative resources, it is necessary to narrow somewhat the issues to be faced by the agency in this proceeding. Consequently, it is necessary to delay other worthwhile goals and concurrent Congressional purposes to a later stage.

This approach is consistent with general principles of administrative law. An agency may set priorities within the framework of its statutory authority. Secondly, an Agency may take substantial steps towards its statutory goals without having to achieve them completely at the first stage, when agency resources are not sufficient to complete all aspects initially.

Section 6(g) of the OSHA Act clearly indicates that OSHA has authority to set priorities among competing occupational health and safety goals. It states:

> In determining the priority for establishing standards under this section, the Secretary shall give due regard to the urgency of the need for mandatory safety and health standards for particular industries, trades, crafts occupations, businesses, workplaces or work environments. The Secretary shall also give due regard to the recommendations of the Secretary of Health, Education, and Welfare regarding the need for mandatory standards in determining the priority for establishing such standards.

Several Court decisions recognize that OSHA has some discretion in setting priorities. For example, the District of Columbia Circuit Court stated,

> The Act has built in flexibilities that the Secretary may use, such as his right to initially determine whether or not there will be a standard; what the standard will be; the priorities between the various occupations that may require standards; the altering and changing of those priorities even though once set; the forgiving of inaction where the Secretary makes a contemporaneous statement of reasons; the right to delay hearings; to process higher priority standards more quickly than initiated ones . . . Since the Congress left such open-ended discretion in the Secretary at many key points in the Act, including Section 6(g) we find implicit acknowledgement that traditional agency discretion to alter priorities and defer action due to legitimate statutory considerations was preserved. *National Congress of Hispanic Cit.* v. *Usery* 554 F. 2d 1196, 1199–1200, (1977). See also *National Cong.* v., *Marshall*, 626 F. 2d 882, 889 (1979).

Similarly, the Court stated, in regard to delaying a relevant decision to a later stage, in *Natural Resources Defense Council* v. *S.E.C.*, 606 F. 2d 1031 (1979) that,

> Deference to the SEC's decision to consider environmental disclosure in another proceeding is in our view, appropriate. Our discussion of the scope of review of agency rulemaking shows that the quasi-legislative nature of rulemaking requires even greater agency freedom to manage and structure decisionmaking than is required in licensing or adjudication. (1056)

It also stated.

> the Agency alone is cognizant of the many demands on its, its limited resources, and the most effective structuring and timing of proceedings to resolve those competing demands. An agency is allowed to be master of its own house, lest effective agency decisionmaking not occur in any proceeding; and judicial review awaits the agency's conclusion of its proceedings. See Myers v. Bethlehem Shipbuilding Corp., 303 U.LS. 41, 58 S.CT 459, 82L.ED. 638 (1938). (p. 1056)

Within this context the ACGIH's Threshold Limit Values and NIOSH Recommended Exposures Limits have been used in determining the substances to give priority to in this proceeding. They have also been used as a starting point for OSHA's significant risk and feasibility analysis. See also the other criteria used which are discussed in the "History" and "Additional Factors" sections.

Secondly, OHSA has concluded that setting exposure limits for these chemicals has priority at this stage over exploring the need for accompanying medical surveillance, monitoring and industrial hygiene provisions. Section 6(b)(7) of the Act, of course, indicates that "where appropriate" such provisions are to be included. That was a concurrent goal of Congress as was Congress' goal to lower exposure for the many unregulated or inadequately regulated substances when scientific data indicates lower exposures are needed.

OSHA has inadequate time to accomplish both goals at this time. Lowering exposures is a higher priority because it is more effective in reducing diseases and material impairments of health.

OSHA has already addressed some of section 6(b)(7)'s goals, labels and warnings, in the Hazard Communication Regulation, 29 CFR 1910.1200 for all substances. It is working on a standard to improve respirator use for all chemicals (47 FR 20803). It is considering generic regulation for monitoring and medical surveillance. OSHA does not have the resources to conclude this rulemaking in any reasonable time, and also consider these issues.

OSHA will meet its statutory requirements to determine whether the proposed new levels substantially reduce significant risk and are feasible. OSHA's risk assessments are briefer than has been the case for rulemakings limited to single sustances. Elaborate and detailed risk assessments for these 428 substances which are generally recognized as needing new limits, would make it impossible for OSHA to address these hazards in any reasonable time period.

Many of the exposure limits are based on moderate safety factors below levels which human or animal studies report "no observed effect" (sometimes called a "threshold"). Incorporation of a moderate safety factor does not result in setting a limit below which significant risk is eliminated. Many of the studies are of small scale and do not have the scientific quality (statistical power) to demonstrate that a "no observed effect level" has been shown. In many instances interpretation of health effects studies must account for inter-species and intra-species variability, and the lack of statistical power.

Finally, when the reported no-effect level is based on animal date, consideration must be given to the fact that humans may be more or less sensitive than the animals. All experts in this situation recommend setting an exposure limit which includes a safety factor from the reported no observed effect level in animals. Therefore, a

moderate safety factor below the level which is hypothesized, or reported as demonstrating no observed effect, does not put OSHA in the position of proposing a limit below the level where there is insignificant risk.

In this rulemaking, OSHA is also proposing new exposure limits for some possible carcinogens and some other chemicals for which there may be a remaining significant risk. It is possible that much more detailed analysis or new data might find that a lower limit than OSHA proposed would both reduce remaining significant risk and be feasible.

However, OSHA preliminarily believes that to address those questions in this rulemaking would significantly delay completion of this rulemaking which substantially improves health protection for many millions of workers.

OSHA's experience in ethylene oxide, benzene, formaldehyde, asbestos and other such substances, indicates that these issues take a large amount of administrative and scientific resources for each substance. Consequently, OSHA preliminarily concludes that it is appropriate to complete this action as first priority. In the future, based on priorities, OSHA will explore the issue that for some substances lower limits might be determined to be appropriate after more extensive analysis.

III. Glossary

The following terms and acronyms appear in the proposed standard and the preamble supporting it. This glossary is provided as a convenience to the reader.

ACGIH—American Conference of Governmental Industrial Hygienists.

CHRIS—Chemical Hazard Response Information System, a data base developed for use by the U.S. Coast Guard.

HSDB—Hazardous Substances Data Bank, a data base developed by the National Library of Medicine.

IARC—International Agency for Research on Cancer.

ILO—The International Labor Organization, and agency of the United Nations.

IMIS—The Integrated Management Information System, a source of enforcement data developed by and for OSHA.

MSDS—Material Safety Data Sheet, a means of disseminating risk information and safe handling information to workers; it is required by OSHA's Hazard Communication Standard (29 CFR 1910.1200).

NIOSH—The National Institute for Occupational Safety and Health, a research agency located within the Department of Health and Human Services.

NOES—National Occupational Exposure Survey, a compilation of data about the prevalence of certain chemicals in workplaces based on a survey of some 4000 workplaces.

OCIS—OSHA Computerized Information System, a data base containing records of laboratory analyses of air contaminant samples, gathered during OSHA inspections.

OSHA HS Number—OSHA has identified each substance considered in their rulemaking with a unique 4-digit number, referred to as the "Health Standard (HS) number." This step was taken for ease of reference and convenience of readers.

PEL—Permissible Exposure Limit, the exposure limits contained in OSHA's air contaminant standard. (See "Z-Tables")

REL—Recommended Exposure Limit, produced by NIOSH and publicized through Criteria Documents and Current Intelligence Bulletins.

RTECS—Registry of Toxic Effects of Chemical Substances, a NIOSH publication.

TLV—Threshold Limit Value, a recommended exposure limit produced by the ACGIH.

TSCA—Toxic Substances Control Act, administered by the Environmental Protection Agency, EPA.

WHO—World Health Organization, an agency of the United Nations.

IV. Substances to be Regulated

A. General Principles of Toxicology and Dose-Response

As long ago as the 16th century, people recognized that there is no such thing as an absolutely safe chemical. The Swiss physician Paracelsus, who live from 1493 to 1541, said:

> All substances are poisons; there is none which is not a poison. The right dose differentiates a poison and a remedy.

On the other hand, methods have been devised to permit any chemical, no matter how poisonous, to be handled safely; this is done either by limiting the dose or controlling the exposure. However, before the necessary degree of control can be determined for a particular exposure or situation, the toxicity of the substance in question must be known. The paragraphs that follow describe the methods used by scientists to measure the relative toxicity of substances and to select exposure limits that will prevent exposed individuals from suffering adverse effects from such exposures. As this discussion demonstrates, methods of choosing exposure limits must, because of the lack or inadequacy of dose-response information for most chemicals, rely heavily on experience in the use of these substances and on scientific and professional judgment.[1]

Chemicals range in inherent toxicity from those that are relatively harmless even after large doses have been administered to others that cause death if encountered even in small quantities. Toxicologists rank chemicals by categories that range from practically non-toxic (an adult human would have to consume a quart) to supertoxic (fewer than 7 drops would be lethal for most people).

In the occupational setting, it is the risk associated with a particular use of a chemical rather than its inherent toxicity that is important. *Risk* can be defined as the probability that a substance will produce harm under certain conditions of use. The converse of risk is *safety*, which is the probability that no harm will occur under specific circumstances.

The degree of hazard associated with exposure to a specific substance depends on the manner in which it is handled in a particular situation: A supertoxic chemical that is processed in a closed, isolated system may be less hazardous in actual use than a relatively low-toxicity compound handled in an open batch process. Another factor affecting the ability of a chemical to elicit a toxic response is the susceptibility of the biological system or individual. For the relative degree of hazard to be known in a particular instance, this requires knowledge about the chemical agent, the exposure situation, and the exposed subject. In addition, the route of administration and the duration and frequency of exposure must be known.

Route of Exposure

There are four principal routes of exposure by which toxic substances can invade humans or animals. These are inhalation, ingestion, dermal absorption, and parenteral administration (i.e., administration through routes other than the intestinal canal). The route of administration of a toxin also affects the relative toxicity of the agent. For example, a chemical that can be detoxified in the liver will be less toxic if it is administered orally than if it is given systemically (i.e., inhaled). Studies that provide information about the relative toxicity of an agent via different routes of exposure can provide a considerable amount of information

[1] The material in this section derives principally from the following sources: Klaasen, Amdur, and Doull, 1986; National Research Council, 1956; and Cohen, 1986a, b.

about the absorbability of the agent. For example, if exposure to a certain dose of a chemical via all routes of administration causes death within the same time period, it can be assumed that the substance in question is easily and rapidly absorbed. On the other hand, if the dermal dose of a chemical that is required to kill a subject is much higher than the dose required to produce the same effect when the chemical is ingested, one can deduce that the skin provides, to some degree, a barrier against that agent's toxicity. Other, less important elements affecting the response to a toxic substance include the relative concentration of the substance, the volume of the vehicle used to administer the chemical, the chemical and physical properties of the vehicle, and the dose rate, i.e., the period of time over which the dose is administered.

Duration and Frequency of Exposure

Scientists conduct animal experiments that involve four different types of exposure: acute, subacute, chronic, and subchronic. Acute exposures are limited to periods of less than 24 hours and can involve either single or repeated exposures within that period. Subacute exposures are repeated exposures that last for one month or less, while subchronic exposures have a duration of one to three months. When a research project having a chronic regimen is conducted, the test animals are dosed repeatedly for a period lasting more than three months. Animals exposed acutely can have both immediate and delayed-onset responses. Similarly, chronic exposures can cause immediate reactions as well as long-term effects.

The frequency of dosing also has an important influence on the magnitude of the toxic effect: A large single dose of an acute toxin will usually have more than three times the effect of one-third the dose given at three different times, and the same dose administered in 10 or 15 applications might have no effect whatsoever. The pattern of dosing is important because it is possible for some of the substance to be excreted between successive administrations or because the lesion caused by the toxin has a chance to be partially or completely repaired between applications. Thus a chronic effect is said to occur: (1) If a toxic substance accumulates in the system of an exposed person or animal because the dose absorbed is greater than the body's ability to transform or eliminate the substance; (2) if it produces adverse effects that are not reversible; or (3) if it is administered in a manner that permits inadequate time for repair or recovery.

Variation in Response

Responses to toxic insults vary in a number of ways. For example, some toxicants have immediate effects, while other are associated with delayed symptom onset. The latency period for carcinogenic agents may be as long as 40 years for some types of cancer, and even some acute agents, such as some chemicals that have adverse ocular effects, may not cause overt symptoms until hours after exposure.

Another difference in type of response concerns the reversibility or irreversibility of the effect. Reversibility depends on the site of action as well as the magnitude of the insult. That is, some tissues of the body, such as the liver, have considerable ability to regenerate; others, like the kidney or central nervous system, do not.

The site of action associated with toxic substances also varies widely. Local effects are those lesions caused at the site of first contact between the agent and the organisms. Examples of localized effects are skin burns caused by contact with a caustic substance and site-of-contact tumors that develop at the locus of the injection of the carcinogen.

In contrast to localized effects, systemic effects involve the absorption and distribution of the toxic agent from the point of entry to a distant site; the toxic response is manifested at this distant point. An example of a systemic poison is mercury, which produces its toxic effect on the central nervous system. Often, the site of deposition for a chemical is not the organ system most affected by the toxin. For example, although lead is deposited and concentrated in the bone, it affects the central nervous system. Any sites that are adversely affected by the toxic effects of exposure to a substance, whether they are sites of contact or distal sites, are called the target organs of toxicity.

In cases of systemic poisoning, the system most often affected is the central nervous system (CNS); it is common for the CNS to be involved even when another target, such as the liver, is the primary target organ of toxicity. In descending order of frequency, the systems or organs most often involved in cases of systemic poisoning are the central nervous system, the circulatory system, the blood and hematopoietic system, the visceral organs (liver, kidney, lung), and the skin.

Dose-Response

The relationship that associates the dose of a chemical with the effects it causes is called the dose-response relationship. A single data point relating a dose to a response is sufficient to establish a dose-response relationship. As additional data become available, it is possible to expand our understanding of the dose-response relationship to cover a range of doses or exposures. Dose-response is the most important single principle in toxicology, and an understanding of dose-response is important in establishing occupational or other exposure limits. Knowing how toxic substances act makes it easier to predict the potential effects of exposure. (It is, of course, generally true that lowering dose reduces response, and data are often available to demonstrate that lower doses reduce responses, at least on the grossly observable level. However, data showing that more subtle responses, e.g., those at the subcellular level, have been reduced are rarely available.)

To apply dose-response relationships, it is helpful if several types of data are available. First, it must be possible to relate a response to a particular chemical. Although basic data pointing toward causality may be available, it is often difficult to refine the dose-response relationship further. For example, epidemiological studies often identify an association between a disease and one or more causative agents. However, since information on the precise identity of the etiologic agent, the actual dose received, and the true site of the response is usually not available, it is often impossible to use data from epidemiological studies to establish a precise dose-response relation between a specific dose of a toxin and an effect.

The second condition to be met before dose-response can be established is that is must be possible to relate the response to the dose. It is relatively easy to determine that a large dose causes an obvious response. Refining the relationship, however, involves three other requirements: (1) That there be a receptor site; (2) that the response and the intensity of the response be related to the concentration of the toxin at the receptor site; and (3) that the concentration of the toxin at the site be related to the dose given.

The third principle underlying the concept of dose-response is that there be a quantifiable means of measuring the toxicity of a substance and a precise method of expressing this measured toxicity. Although lethality in test animals is often used to measure toxicity, the best form of measurement would involve quantification of the sequence of molecular events occurring during the toxic response. In the

absence of such endpoints, other good methods are available. For example, it is common to measure an effect believed to be related to the substance in question. The level of activity of an enzyme in the blood is often used as a measure of effect, e.g., serum glutamic-oxaloacetic transaminase (SGOT) levels are used to measure liver damage. Many different endpoints can be used to measure toxic effects, such as changes in muscle tone, heart rate, blood pressure, electrical activity of the brain, motor functioning, and behavior.

The most widely used endpoint, especially when a new substance is involved, is lethality in an animal test system. Lethality studies allow scientists to make comparative assessments of a chemical's toxicity as it relates to that of many other substances. Research of this type also permits the gathering of essential information on dose, duration, route of administration, site of action, and the target organ of toxicity.

Form of the Response

The classic form of dose-response is sigmoidal (Figure 1). This form characterizes the relationship between the amount of a toxin administered and the degree of response to that dose. The response is measured on the ordinate, and the dose is represented on the abscissa.

Dose-response can be thought of in two ways:

- As exposure increases, the proportion of the population that manifests the response increases;
- As exposure increases, the intensity of the response increases.

A relatively flat dose-response curve means that a large change in dose is required before there is a significant change in response. A step curve, on the other hand, means that a small change in dose will elicit a large increase in response. Although it is sometimes possible to generate a curve of the type shown in Figure 1, it is not necessary to do so to demonstrate that exposure at a given level is associated with a particular response. That is, it is not necessary to have sufficient data to define, in mathematical terms, the dose-response relationship to know that exposure at a given level is associated with adverse consequences.

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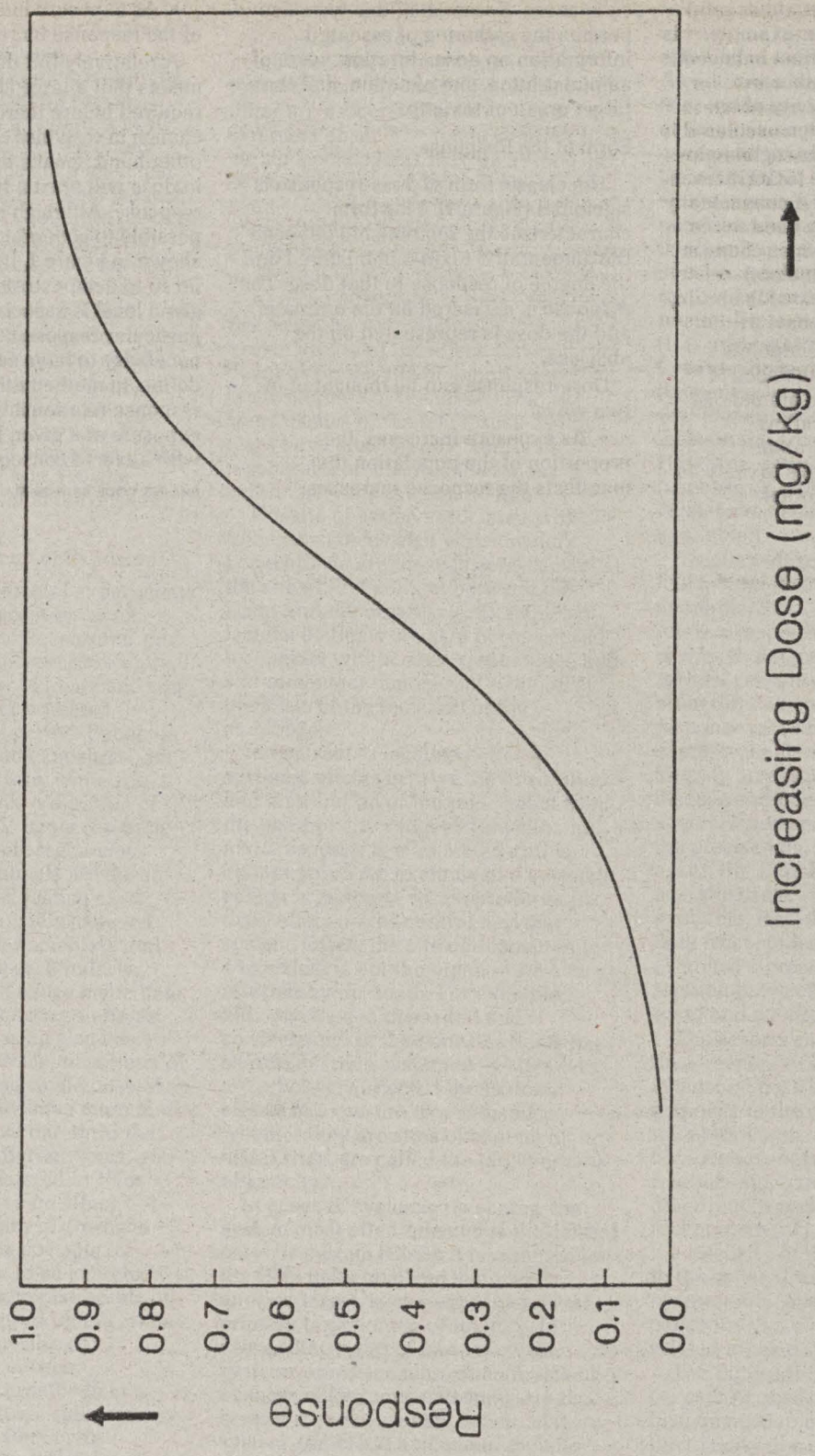
Figure 1

Diagram of dose-response relationship



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In the regulatory context, it is most common to express dose-response relations in terms of the percentage of the population responding. However, this information alone is meaningless unless the endpoint being considered is known. For every substance, there are several dose-response relationships, depending on endpoint: A substance that produces irritation at low doses may cause more severe symptoms or even death at high doses and in other conditions. For example, many substances that are mucosal irritants at low doses will produce pulmonary edema and nervous system effects at high doses.

Plotting the cumulative percentage of individuals responding against dose produces the typical sigmoid curve. Such a curve reflects the fact that at the lowest dose, zero percent of the population responds, while 100 percent of the population will respond at the highest dose. However, if the percentage responding is plotted against incremental rather than total dose, the curve produced is abnormal distribution (Figure 2). This curve says that a relatively small percentage of the population will manifest the response at the lowest dose and that a similarly small percentage of the population will exhibit the effect at the highest dose. What this normal distribution of response reflects is individual and species variation in exposed populations. A wide degree of variation occurs even in inbred, homogeneous laboratory animals, and such variability increases dramatically when a heterogeneous population, such as workers, is involved. Individuals responding at the left end of the curve shown in Figure 2 are hypersusceptible, while those at the right end could be termed resistant.

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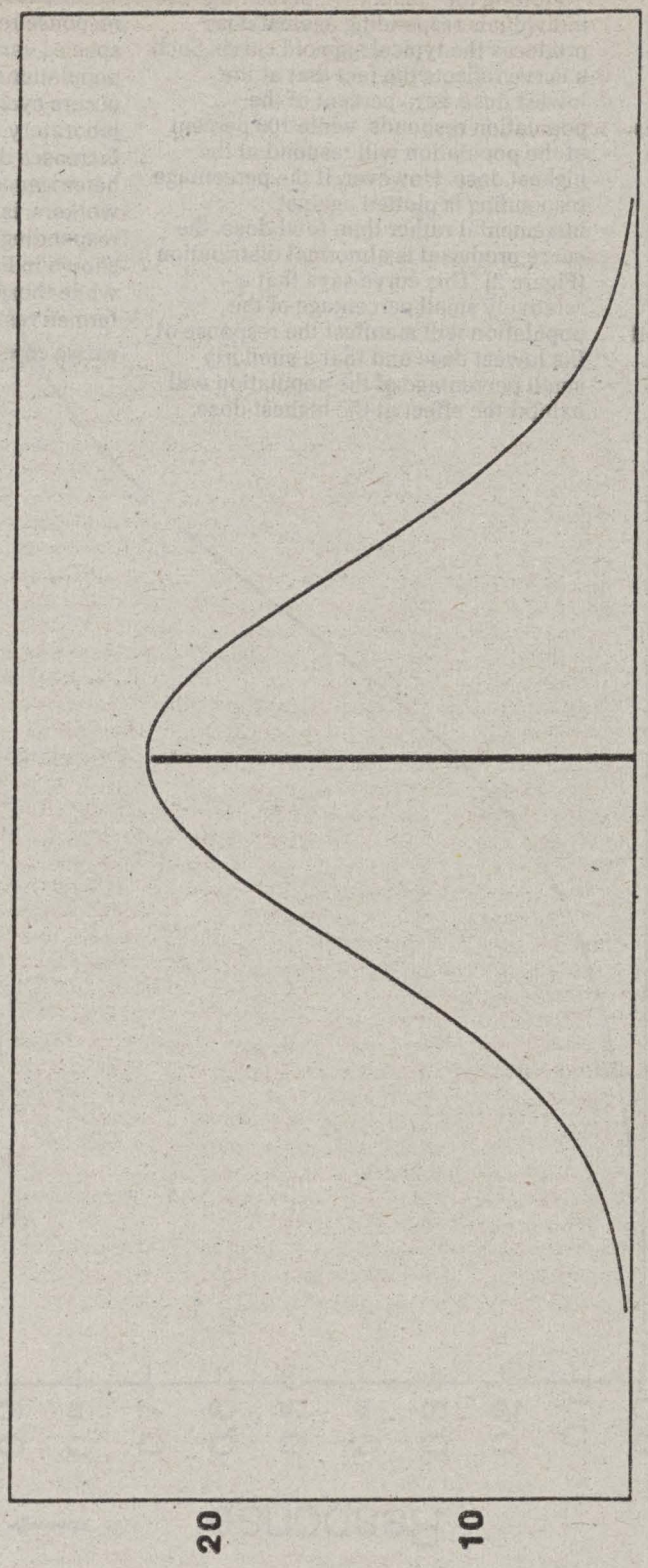
Figure 2
Diagram of quantal dose-response relationship



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Because the relationship between dose and response is sigmoidal, response approaches zero as dose approaches zero. However, because of the mathematical form used to express this relationship, a true zero response can never be achieved. In the strictest sense, therefore, a true threshold dose level (i.e., the dose with which a zero response is associated) can never be established on the basis of experimental research. Instead, scientists attempt to define the minimum dose associated with a specific endpoint, which is customarily termed the "threshold" dose for that particular endpoint. However, unless a specific endpoint, such as respiratory irritation, cholinesterase inhibition, the development of a tumor, or death, is specified, the concept of a threshold is essentially meaningless. In fact, a separate threshold could be said to exist for each of these endpoints.

The extent to which an experimentally derived "threshold" actually reflects the true threshold for a substance (i.e., the level above which a response will occur and below which no response will occur) depends on several factors, such as the number of animals used to determine the experimental threshold, the number of dose levels tested, and the degree of variation represented in the test subjects. For example, to determine an LD_{50} (the lethal dose that will kill 50 percent of the animals tested) with a high degree of precision requires the use of a minimum of 50 test animals and 5 dose groups (10 animals in each group). Other factors that can influence the magnitude of the median lethal dose include the sources involved, the sex and age of the animals, the environmental conditions prevailing during the test conditions, diet, the health status of the subjects being tested, and their past exposure to other toxic substances.

In toxicological research, the experimentally observed "threshold" dose is called the low observed effect level (LOEL) or the low observed adverse effect level (LOAEL). Alternatively, the threshold may be expressed as the highest no observed effect level (NOEL), i.e., the highest dose administered and found not to produce a given response. Determination of an accurate NOEL requires both a careful interpretation of the toxicological data and the use of an adequate number of test animals. The National Academy of Sciences (1985) has concluded that the chance of finding a no adverse effect level (that is, of missing an adverse effect) at a given dose is statistically greater in experiments having a small number of animals than in studies involving a large number of animals. Thus, the degree of confidence one has that NOEL actually represents a "safe" dose, rather than a research design artifact, increases with the number of animals tested. The greatest degree of confidence is associated with studies involving a large number of animals that were tested at several doses that were administered at close intervals.

Safety Factors

Because of the uncertainties surrounding the use of NOELs and LOELs, the difficulties of extrapolating from animals to humans, and the problems involved in trying to account for biological variability (both across species and within the same species), regulators and others have used safety factors to aid them in setting permissible exposure limits. Safety factors are margins applied to the NOEL or LOEL to determine a level believed to be "safe." Safety margins are necessary because studies designed to establish NOELs and LOELs do not generally pick up other-than-gross-organ responses or include hypersusceptible individuals. Regulatory agencies and limit-setting organizations have traditionally used safety factors ranging from 2 to 1,000.

These factors are designed to account for:

- Sensitive members of a population;
- Extrapolation from animals to human; and
- Administration of a substance via a different route.

Additional safety factors may be applied if the toxicological endpoint assessed by the NOEL or LOEL is an insensitive one, e.g., lethality. This is necessary because the dose that is "safe" for an insensitive endpoint will not really be safe in terms of any adverse effects occurring before that endpoint is reached. That is, the LOEL associated with convulsions caused by a neuropathic agent will not be safe or protective against the decrement in mental capacity observed in animals or people long before convulsions occur; to give another example, a dose that is "safe" in terms of lethality is often sufficient to cause serious damage to various organ systems.

Types of Toxicological Evidence

The evidence available to scientists wishing to evaluate the toxicity of a substance can be derived from studies in laboratory animals, in-vitro studies in cell or tissue systems, reports of clinical observations, studies of exposed human populations, or from intervention studies conducted with human volunteers. The preceding paragraphs have described animal studies (or "bioassays"). The following section discusses the two most common types of human evidence: Data derived from clinical observations and information from epidemiological studies.

Clinical observations. Much of the data on the toxic effects associated with human exposures have come from industrial accidents, fatal poisonings, or other such tragedies. This information is generally more useful in delineating broad categories of pathological effects than in refining a specific dose-response relationship, because the exposure levels causing the accident are known to be high but cannot be quantified with precision.

Epidemiological studies. Studies conducted by epidemiologists are designed to reveal the patterns of disease or mortality prevailing in certain groups of people (usually workers) exposed to a single toxin or to a group of substances. One of the advantages of epidemiological studies is that they involve humans and their responses to actual situations. The interpretation of the results of epidemiological studies is complicated by the inevitable presence of confounding variables that occur whenever human populations are involved. Ideally, the populations being studies (i.e., the study population and the control population) should be fully comparable with regard to every variable except the single characteristic under study. Because it is rarely possible to achieve this degree of comparability, statistical techniques are often used to attempt to adjust for this lack of comparability. In addition, if the measured effect is relatively large, it is unlikely that confounding factors will obscure the true picture.

Broadly speaking, epidemiological studies can have two possible outcomes: They can report an effect or they can report no effect; in the former case, the study is termed a positive study, and in the latter, a negative one. Within each of these categories, it is possible for the study to be correct (that is, to give a true-positive or true-negative result) or to be incorrect (that is, to give a false-positive or a false-negative result). A false-positive result reports that there is an increased risk when in fact there is not, and a false-negative study reports that there is no increased risk when in fact there is.

The probability that a study will detect a statistically significant effect if that effect is actually present is called the power of the study. As the power of a study increases, the likelihood of producing a false-negative error decreases. Power is dependent on two factors: The level of relative risk being

evaluated and the number of cases of the effect (i.e., disease) that are expected in the population being studied. The number of expected cases depends both on the sample size and the expected disease frequency in the comparison population. For example, a study involving a small population and a common disease can have the same power as a study of a rare disease in a large population. Consequently, studies of larger samples have sufficient power to detect smaller increases in risk, and studies of smaller samples will be able only to detect large increases in relative risk.

Because epidemiological studies have limitations, it is essential that the power of such studies, particularly of negative studies, be examined to ensure that their sample sizes are adequate to detect the absence of increased risk with validity. When the power of a study is not adequate, negative studies cannot be said either to contradict or to support the conclusion that increased risk exists. However, a study with a positive result may indicate a relationship if the excess risk is high, even if the study's sample size is small and the effects of some factors are not controlled for.

Quality of Evidence

Dose-response models have often been used in the quantitative assessment of the risks associated with exposures to carcinogenic substances. However, less scientific effort has been devoted to models to be used with non-carcinogenic substances. Mathematically precise methods to establish the true no-effect level or to define the dose-response curves have not been developed for most of the more than 400 substances involved in this rulemaking.

Most of the scientific work that has been done was designed to identify lowest observed effect or no-effect levels for a variety of acute effects. As described above, experts in industrial hygiene and occupational health have developed factors to be used to offset, at least to some extent, the insensitivity of NOELs and LOELs to such factors as subcellular effects, sensitive individuals, and chronic effects. It is possible to use these data, combined with professional judgment and OSHA's expertise and experience, to determine that significant risk exists at current levels of exposure and that a reduction in these levels will substantially reduce that risk. OSHA is also confident that it is not attempting in this rulemaking to reduce exposures to insignificant levels. However, additional analysis may well reveal that the levels being proposed at this interim stage can be refined further in the future.

B. Historical Development of Occupational Exposure Limits

Early Limits

Until the development of occupational health standards, the occurrence of adverse health effects resulting from exposures to hazardous substances or conditions in the workplace could only be determined *ex post facto*—after impairment had already occurred to the health and welfare of exposed employees. In her 1910 studies of lead poisoning, Dr. Alice Hamilton was forced to rely on "personal observations of working conditions and the illness and deaths of workers to demonstrate the existence of harmful exposures" (Paull 1984). The concept of occupational exposure limits thus represents a dramatic breakthrough in the battle against occupational disease and remains "one of the most useful and indispensable tools yet devised for safeguarding the health and well-being of industrial workers" (Thomas 1979).

Occupational exposure limits are air quality values that apply in workplaces, and they are derived by studying the correlation between the amount of a toxic substance absorbed by the body and its effects on health. Within the context of occupational exposure, knowledge of this relationship permits quantification of the etiology "of a large number of occupational health impairments, [evaluation of] the risk of such impairments and, if necessary, [consideration of] the effectiveness of preventive meaures" (Parmeggiani 1983). More specifically, an understanding of the levels at which disease or other health effects occur can be used to establish limits of occupational exposure below which health hazards are unlikely to occur in most workers.

The historical development of occupational exposure limits began with the published reports of a German scientist whose investigations in 1883 into the effects of experimental animals (and on himself) of carbon monoxide in known air concentrations caused him to conclude that "the boundary of injurious action of carbon monoxide lies at a concentration in all probability of 500 parts per million, but certainly [not less than] 200 parts per million" (Cook 1987). Shortly after the appearance of this first documented dose-response value, another German researcher, K. N. Lehmann, published a series of reports on a number of chemical substances under the title "Experimental Studies on the Effect of Technically and Hygienically Important Gases and Vapors on the Organism." This series culminated in 1936 with a comprehensive paper on chlorinated hydrocarbons, published as Volume 116 of *Archiv fuer Hygiene*.

In 1912, Rudolf Kobert published a table of exposure limits, based on animal studies, for 20 compounds. One of the first tables of hazardous air concentrations to originate in the United States was a technical paper published in 1921 by the U.S. Bureau of Mines. The 33 substances included in this table were those frequently encountered in the workplace. In addition to limits based on acute toxic effects, this table provided some information on the least detectable odor concentration and the lowest airborne concentration required to cause irritation (Paull 1984; Cook 1987).

Throughout the 1920s and 1930s, data became available that correlated concentrations of harmful substances with observed effects on worker health for such materials as lead and mercury compounds, benzene, and granite dusts. These early occupational health studies, which were based on animal experiments and on findings in exposed workers, provided the kind of data needed to link human exposures "to concentrations that were capable of producing not only acute, but chronic health effects" (Paull 1984).

After 1935, the emphasis of researchers had shifted, for the most part, from the reporting of a series of values for a range of acute effects to results that yielded a single limit based on studies of repeated exposures. Over the years, a sizable amount of data about the levels of exposure that would not produce injurious effects had been amassed for a considerable number of substances. "By the early 1940s, control of the occupational environment to prevent the harmful absorption of toxic materials was becoming an accepted principle, and the practical problem of defining what was 'harmful' was beginning to be met by employing maximum allowable concentrations" (Paull 1984). In 1943, Sterner explained the meaning of the term maximum allowable concentrations as "the upper limit of concentration of an atmospheric contaminant which will not cause injury to an individual exposed continuously during his working day and for indefinite periods of time" (Paull 1984).

The first lists of maximum allowable concentrations of airborne toxic substances were issued between 1933 and 1938. The Union of Soviet Socialist Republics (U.S.S.R.) was the first country to make occupational exposure limits a statutory obligation; in 1933 it published a list that included 14 substances (although health standards for some air pollutants apparently were

used in the Soviet Union during the 1920s). The first American list was published four years later by the State of Massachusetts, and in 1938 Germany issued occupational health standards for a number of organic solvents (Holmberg and Winell 1977). Additionally, the United States "imposed limited occupational safety and health requirements on certain contractors with the Federal government" when the Walsh-Healey Act was passed in 1936 (Mintz 1984).

Standards Developed by Professional Organizations

During the 1940s, American organizations led in the development of occupational health standards, beginning with the American Standards Association (now the American National Standards Institute, or ANSI) list of "maximum acceptable concentrations" (MACs), which appeared in 1941. This list represented a consensus of opinion by the ASA and a number of industrial hygienists who had formed the American Conference of Governmental Industrial Hygienists (ACGIH) in 1938 (Baetjer 1980). Originally conceived of as a time-weighted concentration to be maintained as an average over the working shift, the MAC was redefined in 1957 to mean an upper level (ceiling level) that should never be exceeded (Turner 1976).

An important contribution to occupational health standard-setting was made in 1945 by Warren Cook, who published a list of maximum allowable concentrations for 132 industrial atmospheric contaminants. These limits had been developed by six states, the U.S. Public Health Service, and the American Standards Association, and included Cook's own list of "accepted or tentative values" based on industrial experience, animal experimentation, human sensory response, or a combination of these factors. This table was followed by:

> documentation supported by 187 specific references, indicating the basis and reliability of each value. Cook was the first investigator to codify all of the available data on MAC's and present it in one publication. His list of recommended values was incorporated, practically without changes, by the ACGIH in establishing the TLVs. In support of Cook's inferences, it should be noted that 50 of the * * * values that he recommended in 1945 were subsequently adopted as federal standards, and are still in use today (Paull 1984).

The American Conference of Governmental Industrial Hygienists Subcommittee on Threshold Limits presented its second report at the Eighth Annual Meeting of the ACGIH in 1946. The report included values for 131 gases, vapors, dusts, fumes, mists, and 13 mineral dusts "compiled from the list reported by this subcommittee * * * in 1942, from the list published by Warren Cook in * * * 1945, and from published values of the Z-37 Committee of the American Standards Association" (Cook 1987). The Committee's report noted that:

> Considerable difficulty attends the fixing of satisfactory values for maximal allowable concentrations of chemicals in respirable atmospheres because of the lack of a uniform definition of the maximum allowable concentration concept. One concept is that the M.A.C. value should represent as accurately as possible that concentration at which a worker exposed for a sufficient period of time will just escape physiological or organic injury and occupational disease. A second concept is that the M.A.C. should represent some fraction of that concentration which will injure the worker in order to allow a margin of safety in the design of protective equipment and guard against possible synergistic effects in the case of multiple exposures. A third concept is that the M.A.C. should perform the functions of the former concepts and in addition provide a work environment free of objectionable but non-injurious concentrations of smokes, dusts, irritants and odors. Obviously all of these concepts cannot be fulfilled with the establishment of a single value. M.A.C. values in use at the present time represent examples of all of these concepts.
>
> The committee feels that the establishment of dual lists or a single definition is not possible at the present time.

The report concluded by stressing that the 1946 list of M.A.C. values was presented "with the definite understanding that it be subject to annual revision" (Report of the Sub-committee on Threshold Limits 1946).

Papers presented at both the Ninth International Congress on Industrial Medicine in London (1948) and at the Fifteenth International Congress of Occupational Health in Vienna (1966) also dealt with maximum acceptable concentrations. The first of these proposed that zones of toxicity be set up to facilitate an understanding of the relative hazards of substances, "since the boundarties of MAC values were not sharp lines of demarcation" (Cook 1987). At the 1966 meeting, discussion took place on the advantages of the concept of a "peak level" of exposure—an extension of the "ceiling level" notion inherent in the definition of a MAC since 1957. A "peak level" was defined as one "that can be applied to certain substances for brief designated periods and for a strictly limited number of times during the work shift, with a designated time interval between peaks. The 'peak' concept places a limit on the intermittent higher exposures that occur in many industrial operations. The time-weighted average exposure limit is of course to be observed [even when a peak has also been assigned to a substance]" (Cook, 1987).

Terminology and definitions throughout this early period were ambiguous and imprecise, reflecting uncertainty as to exactly what needed to be and could be done in the realm of occupational health standard setting. Initially, the ACGIH designated its recommended limits as "maximum allowable concentrations," although this term was often used interchangeably with "threshold limit values." Confusion about the meaning, interpretation, and relative significance of the terms being employed during this embryonic period was common. After 1953, the ACGIH defined the concept of threshold limit values in the preface to its annual published list of occupational health standards as "maximum average atmospheric concentrations * * * for an eight-hour day." This definition of the TLVs as average concentrations differed from the general understanding of the original term "maximum allowable concentrations," which were essentially ceiling values (Stokinger 1962).

By 1955, the ACGIH's philosophy of establishing threshold limits had begun to shift from concern solely with the prevention of acute stress and illness to consideration also of "more subtle effects on health, such as the effects of annoying or irritating agents * * *. Thus, whereas a threshold limit value of 3 or 4 ppm for chlorine would insure no impairment to workers' health, this value is reduced to 1 ppm in the interest of greater freedom from irritant effects" (Stokinger 1956). It should be noted that such analyses often did not take into account chronic effects or responses among sensitive workers.

Documentation for the 238 substances included in the TLV list for 1956 was provided by Smyth in a separate paper in which the author

> recommended that the TLV's include references to the underlying data, and that the concepts represented by the values be restated in more realistic toxicological terms. In his analysis of the TLVs, he [Smyth] concluded that nine categories of objectionable action were guarded against: chronic toxicity, acute toxicity, narcosis, irritation, asphyxiation, fume fever, eye pigmentation, allergic response, and cancer. (Paull 1984)

At about the same time, Stokinger stated that in his opinion, the Threshold Limits Committee had avoided grappling with the issue of developing a method for establishing limits for industrial

carcinogens and noted that, with the exception of nickel carbonyl, limits had not been assigned for potential carcinogens (Paull 1984). In 1962, however, the TLV Committee included three carcinogens as additions to the TLV list, although these were listed separately in an appendix and did not have assigned TLVs.

Despite the fact that the ACGIH had stressed early on that TLVs were intended as guides and not as rigidly enforceable limits, the American Standards Association's MAC values (or, where none was available, the TLV) were included as mandatory limits in the Safety and Health Standards for Federal Supply Contracts, which were published in 1960 under the Walsh-Healey Act. Following this action, the ACGIH issued a statement on the definitions and interpretations of TLVs and MACs (Stokinger 1962). At the same time, the ACGIH announced the production of the first edition of the *Documentation for Threshold Limit Values* (ACGIH 1962); this was followed by another paper in which the work and intentions of the Threshold Limits Committee were reviewed. Turner states that:

> [a]t this time the concept of ceiling values and excursion factors around the time-weighted average values was introduced in order to reduce conflict or confusion with the 'maximal' values in the American [ANSI] Standards. A 'C' (ceiling value) listing was to be given to those fast-acting substances thought likely to be injurious if the concentration exceeded the limit value by more than a designated factor for a relatively short period (about 15 min.). The factor varied between 3 and 1.25, depending inversely upon the magnitude of the TLV. A corollary was that the factor would also indicate the limit of permissive excursion of the concentration above the TLV for a substance not given a 'C' listing, always provided that the time-weighted average concentration did not exceed the TLV. This rule of thumb approach to limiting exposure is no doubt appropriate to certain substances when they are used routinely throughout the working day. It seems to have little relevance in other instances where exposure is irregular or where the basis for fixing the TLV is on grounds other than toxicity. (Turner 1976)

Limits in the Era of OSHA

The enactment of the Occupational Safety and Health Act of 1970 marked the first "comprehensive and serious attempt * * * to protect the health and safety of American workers" (Mintz 1984); it also greatly extended the use of MACs and TLVs by authorizing the newly established Occupational Safety and Health Administration (OSHA) to adopt as its own standards "national consensus standards" and established federal standards (29 U.S.C. 655(a)). Mintz notes that "in addition to the safety standards adopted under section 6(a), OSHA also adopted permissible exposure limits for approximately 400 toxic substances. These [start-up] health standards, now appearing in 29 CFR 1910.1000, * * * were derived from both national consensus and established federal standards. The national consensus standards had been issued by ANSI, while the established federal standards had been adopted under the Walsh-Healey Act from the TLVs * * * recommended by the * * * ACGIH" (Mintz 1984).

Since OSHA's large-scale adoption of the ANSI consensus standards and the 1968 ACGIH TLVs, the Agency has promulgated standards under section 6(b) of the OSH Act to regulate the industrial use of 24 substances, most of which have been identified as occupational carcinogens, but the ANSI and ACGIH start-up standards continue to comprise the major part of the Agency's occupational health and safety program.

In the interval since the establishment of OSHA and the adoption of the ACGIH and ANSI limits by the Agency, the ACGIH has continued to revise, update, and document the recommended limits that appear in its annual list of TLVs. Since 1968, annual revisions have been made to these limits by the ACGIH. During this time, the TLVs have been "accepted on an international basis as the best available guides for providing healthful occupational environments, and at least 18 countries, including the United States, have either adopted them as legal standards or as guides to legal action, thus verifying their efficacy in accomplishing this purpose" (Paull 1984).

The action OSHA takes today initiates the process of updating the Agency's Z table permissible exposure limits. That these limits are seriously out of date is attested to by the fact that the ACGIH has found it necessary to revise or add nearly 200 limits to its list in the 20 years since the limits that were later adopted by OSHA were initially published. Recognition that OSHA's Z table limits need updating to reflect recent developments in toxicology and new data on the health effects associated with exposure to these substances is widespread throughout industry: For example, OSHA requires and it is standard practice for organizations that develop Material Safety Data Sheets (MSDSs) to include on these MSDSs the ACGIH's current TLV values, as well as the OSHA limit.

The following section describes the methodology used by OSHA in selecting the limits it is proposing today. The Agency believes that promulgation of these limits will address a broad range of significant risks now prevalent in industry. As many industrial hygienists and occupational safety and health professionals have noted, the use of permissible exposure limits continues to be the single most efficacious way of protecting the health, functional capacity, and well-being of the American worker.

C. Description of the Substances For Which Limits Are Being Proposed

In this rulemaking, OSHA considered revising 428 substances and is proposing to revise existing or add new limits for several hundred (a total of 406) toxic substances currently being manufactured, used, or handled in workplaces throughout general industry. This section of the preamble identifies the proposed PELs, describes the available toxicological data for many of these substances, and explains the Agency's rationale for selecting the proposed limits for several of these substances.

The universe of substances included in this rulemaking is bounded by the substances for which the American Conference of Governmental Industrial Hygienists (ACGIH) has established a Threshold Limit Value (TLV) for exposures in the work environment. That is, OSHA is not proposing at this time to establish exposure limits for any hazardous substance that is not included in the ACGIH's 1987–88 List of TLVs. In addition, where the limit included in the current ACGIH list is identical to OSHA's existing Z-table limit for the same substance, OSHA is not proposing a change. OSHA has determined that the ACGIH's TLV is clearly the most comprehensive list for this purpose.

Although limits are not being proposed for chemicals excluded from the ACGIH's 1987–88 list, OSHA has not limited its consideration of appropriate limits to those levels established by the ACGIH. The Agency has also carefully evaluated the exposure limits recommended by the National Institute for Occupational Safety and Health, OSHA's sister agency. In instances where both NIOSH and the ACGIH have recommended substantially different limits for the same substance, OSHA has thoroughly analyzed the evidence presented by each organization before determining which of the two limits to propose. The proposed limits thus represent, in the Agency's professional judgment, those levels found to be most consistent with OSHA's mandate and the case law that

has subsequently developed to interpret that mandate. (For a discussion of the relevent legislative and judicial principles, see the section elsewhere in this preamble entitled History and Need for Revision of the PELs.

For ease of analysis and presentation, the substances included in the scope of this rulemaking have been grouped into 18 separate sub-sections. In general, these groupings reflect the principal mechanisms of action or target organ systems involved in the toxic responses that occur when workers are exposed to these substances.

In addition to target organs or effect groupings, separate sections dealing with limits set on the basis of no-effect levels or by analogy are included. Three additional sections cover substances for which the ACGIH has increased its limits, substances for which OSHA is proposing to add short-term limits, and those for which the Agency proposes to add skin notations.

The following sections are included:

1. Substances for Which Proposed Limits Are Based on Avoidance of Neuropathic Effects.
2. Substances for Which Proposed Limits Are Based on Avoidance of Narcotic Effects.
3. Substances for Which Proposed Limits Are Based on Avoidance of Sensory Irritation.
4. Substances for Which Proposed Limits Are Based on Avoidance of Liver or Kidney Effects.
5. Substances for Which Proposed Limits Are Based on Avoidance of Ocular Effects.
6. Substances for Which Proposed Limits Are Based on Avoidance of Respiratory Effects.
7. Substances for Which Proposed Limits Are Based on Avoidance of Cardiovascular Effects.
8. Substances for Which Proposed Limits Are Based on Avoidance of Systemic Effects.
9. Substances for Which Proposed Limits Are Based on Observed No-Effect Levels.
10. Substances for Which Proposed Limits Are Based on Avoidance of Adverse Nuisance Effects.
11. Substances for Which Proposed Limits Are Based on Avoidance of Taste and Odor Effects.
12. Substances for Which Proposed Limits Are Based on Avoidance of Adverse Health Effects Caused by Exposure to Analogous Substances.
13. Substances for Which Proposed Limits Are Based on Avoidance of Biochemical/Metabolic Effects.
14. Substances for Which Proposed Limits Are Based on Avoidance of Sensitization Effects.
15. Substances for Which Proposed Limits Are Based on Avoidance of Cancer.
16. Substances for Which Current ACGIH TLVs Are Less Stringent than Existing OSHA PELs.
17. Substances for Which OSHA Is Proposing Short-Term Exposure Limits.
18. Substances for Which OSHA Is Proposing To Add Skin Notations.

A list of the references that OSHA relied on in evaluating the toxicological evidence pertaining to these chemicals appears at the end of Section IV.

1. Substances for Which Proposed Limits Are Based on Avoidance of Neuropathic Effects

Introduction

Many industrial chemicals have been shown to cause severe neurological effects in exposed workers, and in many cases these effects are irreversible. There are 20 chemicals for which limits have been set on the basis of avoidance of neuropathic effects. Table C1–1 lists the current PEL, ACGIH TLV, NIOSH REL, CAS number, and OSHA identification number for each of these chemicals. The table shows time-weighted averages (TWAs), ceiling limits, and short-term exposure limits (STELs). For this group of 20 compounds, OSHA is proposing to lower its current TWA-PEL for two substances; add a STEL to an existing TWA for five substances; change an existing ceiling to a TWA and STEL for one substance; change an existing TWA-PEL to a ceiling value for one substance; amend existing short-term or ceiling limits for four substances; and establish new exposure limits for seven substances not currently regulated by OSHA. As Table C1–1 shows, most of the proposed limits are revisions of existing OSHA limits, rather than new additions to Table Z. In only five instances is there a difference between the quantitative limits set by the ACGIH and NIOSH for the same substance.

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TABLE C1-1. Substances for Which Limits Are Based on Avoidance of Neuropathic Effects

H.S. Number/ Chemical Name	CAS No.	CURRENT PEL*	ACGIH TLV**	NIOSH REL***
1051 n-Butyl alcohol	71-36-3	100 ppm TWA	50 ppm Ceiling, Skin	—
1078 Chlorinated camphene	8001-35-2	0.5 mg/m^3 TWA	0.5 mg/m^3 TWA, Skin 1 mg/m^3 STEL	—
1114 Decaborane	17702-41-9	0.05 ppm TWA, Skin	0.05 ppm TWA, Skin 0.15 ppm STEL	— —
1116 di-sec-octyl-Phthalate	117-81-7	5 mg/m^3 TWA	5 mg/m^3 TWA 10 mg/m^3 STEL	— —
1123 Dichloroacetylene	7572-29-4	—	0.1 ppm Ceiling	—
1149 Dipropylene glycol methyl ether	34590-94-8	100 ppm TWA, Skin	100 ppm TWA, Skin 150 ppm STEL	—
1200 n-Hexane	110-54-3	500 ppm TWA	50 ppm TWA	100 ppm TWA 510 ppm Ceiling (15 min)
1202 2-Hexanone	591-78-6	100 ppm TWA	5 ppm TWA	1 ppm TWA

TABLE C1-1. Substances for Which Limits Are Based on Avoidance of Neuropathic Effects (continued)

H.S. Number/ Chemical Name	CAS No.	CURRENT PEL*	ACGIH TLV**	NIOSH REL***
1216 Iron pentacarbonyl	13463-40-6	--	0.1 ppm TWA 0.2 ppm STEL	--
1236A Manganese, fume	7439-96-5	5 mg/m^3 Ceiling	1 mg/m^3 TWA 3 mg/m^3 STEL	--
1237 Manganese cyclopentadienyl tricarbonyl	12079-65-1	--	0.1 mg/m^3 TWA, Skin	--
1238 Manganese tetroxide	1317-35-7	--	1 mg/m^3 TWA	--
1240 Mercury (aryl and inorganic compounds)+	7439-97-6	0.1 mg/m^3 Ceiling	0.1 mg/m^3 TWA, Skin	0.05 mg/m^3 TWA (8-hr)
1241 Mercury, vapor	7439-97-6	0.1 mg/m^3 Ceiling	0.05 mg/m^3 TWA, Skin	--
1242 Mercury, (organo) alkyl compounds	7439-97-6	0.01 mg/m^3 TWA 0.04 mg/m^3 Ceiling	0.01 mg/m^3 TWA, Skin 0.03 mg/m^3 STEL	-- --
1251 Methylacrylonitrile	126-98-7	--	1 ppm TWA, Skin	--
1253 Methyl bromide	74-83-9	20 ppm Ceiling, Skin	5 ppm TWA, Skin	Lowest feasible level

TABLE C1-1. Substances for Which Limits Are Based on Avoidance of Neuropathic Effects (continued)

H.S. Number/ Chemical Name	CAS No.	CURRENT PEL*	ACGIH TLV**	NIOSH REL***
1304 Pentaborane	19624-22-7	0.005 ppm TWA	0.005 ppm TWA 0.015 ppm STEL	-- --
1316 Phenyl mercaptan	108-98-5	--	0.5 ppm TWA+	0.1 ppm Ceiling (15 min)
1342 1,2-Propylene glycol dinitrate	6423-43-4	--	0.05 ppm TWA, Skin	--

* OSHA's TWA limits are for 8-hour exposures; its STELs are for the durations specified; and its ceilings are peaks not to be exceeded for any period of time.

** The ACGIH TWA-TLV is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

*** NIOSH TWA limits are for 10-hour exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

\+ OSHA's current limit is retained.

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Description of the Health Effects

The human nervous system comprises the central nervous system (CNS) and peripheral nervous system (PNS). The CNS is made up of the brain and spinal cord, while the PNS consists of a network throughout the body of nerves that communicate with the CNS via connections to the spinal cord. The brain and spinal cord are bathed in cerebrospinal fluid, which supplies nutrients to the CNS and also acts as a barrier against some foreign substances. This barrier protects the central nervous system. In general, fat-soluble substances readily diffuse across this barrier and water-soluble substances do not.

Chemicals that affect the central nervous system may manifest their toxic effects peripherally. An example of this is the tremor associated with elemental and organic mercury poisoning. Exposure to some chemicals, such as n-hexane, is associated with axonal degeneration of the nerves in both the central and peripheral nervous systems. Baker (1983) refers to this dual-system effect as central-peripheral distal axonopathy.

Nervous system toxicants can affect motor function, sensory function, or integrative processes, and they can also cause changes in the behavior of exposed persons. Substances that cause demyelination or neuronal damage can produce motor dysfunction that is expressed as muscular weakness or unsteadiness of gait, while exposures to chemicals that are associated with loss of sensory function may result in alterations in touch, pain, or temperature sensation or damage to sight or hearing. Other neuropathic chemicals affect the way in which information is processed in the brain and can interfere with learning and memory.

Although mature neurons cannot divide and be replaced, the nervous system has considerable ability to restore function lost as a result of exposure to toxic chemicals. This capability to restore function even after neurons have been killed is achieved by two mechanisms: Plasticity of organization and redundancy of function. That is, when some neurons die, other cells that perform the same function may be able to maintain an adequate level of functioning, or other neurons may be able to "learn" how to perform the lost function. However, even when one of these mechanisms comes into play to compensate for neuronal damage, the overall reserve capacity of the nervous system will have been diminished. The loss of this reserve could be critical in a situation in which additional demands are placed on the nervous system. Thus, even so-called reversible neuropathic effects should be seen as toxic effects causing alterations in an impairment of the normal functioning of the nervous system.

The neurological effects potentially associated with chemical exposures are numerous, and it is not always easy to identify the precise target site. However, recent medical advances have made tests available that can detect neurological damage that was not detectable several years ago. For example, electrophysiological methods have been developed to measure damage to the visual pathway caused by such exposures. Because of the variation in individual responses to chemical exposures, exposure limits should be set with a view toward this range of susceptibility and the avoidance of any neuropathic effects.

Peripheral Nervous System Effects

The pathological mechanisms associated with peripheral neuropathies result from segmental demyelination or axonal degeneration. Segmental demyelination destroys the myelin sheath but leaves the axon intact; this causes a slowing in nerve conduction velocity. Muscle weakness is often the first sign of such segmental demyelination, and this effect can progress to a decline in motor function or paralysis. Although remyelination may occur within weeks after injury, even a temporary loss in motor or sensory function places the affected worker or others at risk of injury.

Axonal degeneration is a more serious effect in that recovery is often slow or incomplete. It causes demyelination secondary to the degeneration of the distal portion of the nerve. This effect occurs when a chemical interferes with the physiologic dynamics of the nerve, e.g., when it decreases the transport of nutrients to the nerve. The axon will degenerate (die-back) sufficiently to accommodate the cell's capacity to supply it with nutrients. Axonal degeneration can also occur as a result of biochemical or metabolic derangement of the central nervous system. Alkyl mercury and elemental mercury are examples of chemicals causing this type of effect (Cavanaugh 1985).

Central Nervous System Effects

The mechanism of action of central nervous system toxins is not well understood but is believed to be associated with neurochemical alteration in the brain. Seizures, Parkinsonism, intellectual impairment, narcosis, dementia, cranial neuropathy, and visual disturbances are all examples of effects that can occur after overexposures to neuropathic chemicals. The more serious CNS effects, such as Parkinsonism, dementia, intellectual impairment, and cranial neuropathy, are generally irreversible (Baker 1983). Before these effects are manifested, subtle changes in behavior may occur; if these subtle signs are interpreted correctly, exposure can be stopped before irreversible damage occurs.

Dose-Response Relationships and Neuropathic Effects

The development of chemically induced neurological effects is believed to follow a dose-response pattern. At an exposure intensity or duration below the no-effect level, detectable effects are unlikely to be evident. As exposure intensity/duration increases to and beyond this level, the toxin begins to interfere with the normal cellular processes of the neurological system. At this early stage, transient signs and symptoms may appear. Overt effects become more severe as exposure continues and finally progress to serious loss of neurological function and possible permanent damage to neural tissue. Increases in our ability to detect neurological changes at lower levels of exposure have shown that neurobehavioral changes or impairment may occur at levels previously thought to be innocuous. These early effects can be important indicators of potential functional impairment at exposure levels below those that produce either transient or permanent damage. Heavy metals, solvents, and pesticides are examples of chemicals that can cause symptoms that include nausea, sensory and motor function impairments, depression, sleep disturbances, cognitive impairment, and sexual dysfunction. Limits for substances in this group are generally designed to maintain worker exposures below the level associated with such symptoms. This approach ensures that employees will not be likely to suffer these adverse symptoms and provides a margin of safety against the risk of more severe or permanent neurological impairment.

The following discussions describe OSHA's preliminary findings for all of the substances in this group and illustrate the serious nature of the risk faced by workers exposed to these toxicants.

n-BUTYL ALCOHOL

CAS: 71-36-3; Chemical Formula: $CH_3CH_2CH_2CH_2OH$

H.S. No. 1051

OSHA's current PEL for n-butyl-alcohol is a 100 ppm 8-hour TWA; the ACGIH-recommended limit is a 50 ppm ceiling, with a skin notation.

n-Butyl alcohol is a colorless, highly refractive liquid with a mild vinous odor that has long been known to cause irritation of the eyes and headaches in occupational settings. Systemic exposure effects in the form of vestibular and auditory nerve injury have only been reported in workers in France and Mexico (Seitz 1972; Velasquez 1964; Velasquez et al. 1969).

The current OSHA limit of 100 ppm (TWA) is based on the studies of Tabershaw, Fahy, and Skinner (1944) and of Smyth (1956). These studies indicated that workers experienced no narcotic or systemic effects at levels lower than 100 ppm. Mild irritation was reported in humans exposed to 24 ppm; this irritation became uncomfortable and was followed by headaches at 50 ppm (Nelson, Ege, Ross et al. 1943).

More recent data reported by Seitz (1972), Velasquez (1964), and Velasquez et al. (1969) indicate serious long-term systemic effects on the auditory nerve and hearing loss (hypoacusia), the magnitude of hearing loss was related to length of exposure. Nine of 11 workers exposed without hearing protection to 80 ppm for periods of from 3 to 11 years displayed impaired hearing. This phenomenon was particularly evident in younger workers (Velasquez 1964; Velasquez et al. 1969).

OSHA believes that the current PEL of 100 ppm is not protective against the risk of hearing loss and vestibular injury associated with exposure to n-butyl alcohol, because these types of systemic injuries have been observed at levels below 100 ppm. A skin notation is also being proposed because of n-butyl alcohol's potential to cause skin irritation. The Agency preliminarily concludes that the proposed 50 ppm ceiling will reduce this risk substantially. This health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for n-butyl alcohol if the Agency determines that this limit will substantially reduce significant risk.

CHLORINATED CAMPHENE (60 Percent)
CAS: 8001-35-2; Chemical Formula: $C_{10}H_{10}Cl_8$
H.S. No. 1078

OSHA currently has a limit of 0.5 mg/m³, with a skin notation, for chlorinated camphene. The ACGIH recommends a limit of TLV-TWA 0.5 mg/m³ and a TLV-STEL of 1 mg/m³ for chlorinated camphene (60 percent), with a skin notation. This substance is an amber waxy solid with a pleasant, pine-like odor.

Chlorinated camphene has demonstrated a moderately high acute toxicity in animal studies (ACGIH 1986, p. 114). Toxic doses cause varied central nervous system effects, including nausea, muscle spasms, confusion, and convulsions (Hayes 1963). Data indicate that rats and guinea pigs show no significant effects at dietary levels of 800 ppm daily for a 6-month period (Alderson Reporting Co. 1972). Monkeys tolerate 10 ppm daily but show toxic symptoms after 2 weeks' feeding at the 60-ppm level (Sosinerz 1972). Although chlorinated camphene may accumulate in fatty tissues, it clears quickly when ingestion is terminated (Sosinerz 1972).

In humans, the acute lethal dose of chlorinated camphene is between 2 and 7 grams, and a dose of 10 mg/kg causes nonfatal convulsions in some exposed individuals. The ACGIH (1986, p. 115) concludes that the acute toxicity of chlorinated camphene is equivalent to that of chlordane, for which the fatal human dose is estimated to be around 6 grams; the ACGIH TLV-TWA for chlordane is 0.5 mg/m³. One study of 25 human volunteers failed to reveal toxic responses to a daily 30-minute exposure to 500 mg/m³ for 10 consecutive days, followed by similar exposures for 3 consecutive days 3 weeks later (Shelansky 1947, as cited in ACGIH 1986, p. 115). There are no reports of occupational poisonings, and a review of the medical records of employees engaged in the manufacture and handling of chlorinated camphene showed no ill effects in workers exposed for an average of 3.7 years (Hercules 1972).

OSHA is proposing a PEL of 0.5 mg/m³ TWA and a 15-minute STEL of 1.0 mg/m³ for this insecticide, with a skin notation. The Agency preliminarily concludes that both a TWA and a STEL are required to protect exposed workers against the risk of neuropathic and systemic effects. The health evidence forms a reasonable basis for proposing a new limit for chlorinated camphene. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

DECABORANE
CAS: 17702-41-9; Chemical Formula: $B_{10}H_{14}$
H.S. No. 1114

OSHA currently has an 8-hour TWA limit of 0.05 ppm TWA and a skin notation for decaborane. The ACGIH recommends a TLV-TWA of 0.05 ppm and a TLV-STEL of 0.15 ppm, also with a skin notation. Decaborane forms colorless crystals, which are stable at ordinary temperatures and have a pungent odor.

The acute toxicity of decaborane is extremely high for small laboratory animals. The 40-hour LC_{50}s for rats and mice are 46 and 12 ppm, respectively (Schecter 1958). Dermal LD_{50}s for rabbits and rats are 71 and 740 ppm, respectively (Svirbely 1955). Acute exposures to decaborane cause loss of coordination, convulsions, weakness, tremors, and hyperexcitability. Its primary effects are on the kidneys and liver. Studies of repeated exposures to this substance suggest that the toxicity of decaborane is intermediate between that of pentaborane and diborane. The ability of decaborane to penetrate the skin is particularly notable, as is its toxicity to the central nervous system in some species, e.g., rats and rabbits (Svirbely 1954 and 1955). Monkeys showed decreased ability for certain operant behaviors when injected with doses of 3 to 6 mg/kg decaborane (Reynolds et al. 1964). Central nervous system toxicity has been observed in humans exposed occupationally (Krackow 1953).

OSHA is proposing an 8-hour TWA PEL of 0.05 ppm TWA, a 15-minute STEL of 0.15 ppm, and a skin notation for decaborane. The Agency preliminarily concludes that this level will provide protection against the risk of neuropathies, kidney and liver damage, and percutaneous absorption possible in the absence of a short-term limit for decaborane. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for decaborane if the Agency determines that this limit will substantially reduce significant risk.

di-sec-OCTYL PHTHALATE
CAS: 117-81-7; Chemical formula: $C_{24}H_{38}O_4$
H.S. No. 1116

OSHA currently has a limit of 5 mg/m³ TWA for di-sec-octyl phthalate. The ACGIH recommends a TLV-TWA of 5 mg/m³ and a TLV-STEL of 10 mg/m³ for this light-colored, viscous, odorless, and combustible liquid.

Di-sec-octyl phthalate (DEHP) has an extremely low acute oral toxicity in small laboratory animals. The oral LD_{50} reported for the mouse is 26.3 g/kg; for the rat, it is 33.8 g/kg (Krauskopf et al. 1973). No skin irritation or sensitization potential has been demonstrated in either animals or humans, and the lethal dermal dose in rabbits is about 25 ml/kg (Singh, Lawrence, and Autian 1972). Shaffer, Carpenter and Smyth (1945) and Lawrence (unpublished data) have reported deaths in exposed rats as well

as chronic diffuse inflammation of the lung, resembling burn responses, after DEHP exposures in mice at unspecified levels.

Long-term dietary toxicity studies in rats, guinea pigs, and dogs have established a no-effect dose level of about 60 mg/kg/day, and no carcinogenic or histologic abnormalities were observed at this level (Gesler 1973). Higher doses were associated with growth retardation and increased liver and kidney weights but not histologic abnormalities. Metabolic studies have demonstrated that laboratory animals do not appreciably metabolize DEHP (Dillingham and Autian 1983). Teratogenicity studies in pregnant rats indicated that fertility is unaffected at doses of 0.1, 0.2, or 0.33 percent of the acute intraperitoneal LD_{50} dose for rats, although slight effects on embryonic and fetal development were observed in these animals; skeletal deformities were the most common teratogenic effects observed (Dillingham and Autian 1973). Mutagenic effects were observed at intravenous doses of one-third, one-half, and two-thirds of the acute LD_{50}; these effects were consistent with DEHP's ability to produce dominant lethal mutations (Dillingham and Autian 1973).

A study of exposures to a mixture of the vapors of diethyl phthalate, dibutyl phthalate, and di-2-ethylhexyl phthalate reported that exposures to 1 to 6 ppm caused no phthalates in the blood and no peripheral polyneuritis (Raleigh, as cited in ACGIH, p. 223). However, Russian investigators examined male and female workers exposed to between 1.7 and 66 mg/m^3 of various combinations of airborne phthalates and noted complaints of pain, numbness, and spasms in the upper and lower extremities after 6 to 7 years' exposure. Polyneuritis was observed in 32 percent of the workers studied, and 78 percent of these workers showed depression of vestibular receptors (Milkov et al. 1973).

OSHA proposes an 8-hour PEL of 5 mg/m^3 and a 15-minute STEL of 10 mg/m^3 for di-sec-octyl phthalate. The Agency preliminarily concludes that these limits together will protect workers from the risk of pain, numbness, spasms of the extremities, and polyneuritis possible at excursions above the existing 8-hour TWA PEL. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for di-sec-octyl phthalate if the Agency determines that this limit will substantially reduce significant risk.

DICHLOROACETYLENE

CAS: 7572-29-4; Chemical Formula: C1C≡CC1
H.S. No. 1123

OSHA currently has no limit for dichloroacetylene. The ACGIH recommends a TLV-ceiling of 0.1 ppm for this liquid, which explodes on boiling.

In preliminary inhalation exposure studies, guinea pigs demonstrated a 4-hour LC_{50} of 20 ppm; death occurred 2 or 3 days after exposure and was caused by pulmonary edema. In rats, similar exposures to dichloroacetylene in the presence of 330 ppm of trichloroethylene indicated an LC_{50} of 55 ppm (Siegel, as cited in ACGIH 1986, p. 177). When dichloroacetylene was mixed with 9 parts of ether, the 4-hour LC_{50} in rats was 219 ppm; in combination with 7 parts of trichloroethylene, the 4-hour LC_{50} in rats was 55 ppm; and exposure to dichloroacetylene with 10 parts trichloroethylene caused a 4-hour LC_{50} in guinea pigs of 15 ppm (Siegel et al. 1971).

In humans, dichloroacetylene exposure causes headache, loss of appetite, extreme nausea, and vomiting; it affects the trigeminal nerve and facial muscles and exacerbates facial herpes. Disabling nausea was experienced by approximately 85 percent of individuals exposed for prolonged periods of time (not further specified) at concentrations from 0.5 to 1 ppm (Saunders 1967). A number of occupational fatalities have been attributed to exposure to dichloroacetylene (Humphrey ad McClelland 1944; Firth and Stuckey 1945). Humphrey and McClelland (1944) reported on 13 instances of cranial nerve palsy, nine of which had labial herpes, following exposure to dichloroacetylene. These patients also had symptoms of nausea, headache, jaw pain, and vomiting. Autopsies of two of these fatalities revealed edema at the base of the brain (Humphrey and McClelland 1944).

OSHA is proposing a ceiling limit of 0.1 ppm for dichloroacetylene. The Agency preliminarily concludes that exposure to dichloroacetylene poses a risk of disabling nausea and serious systemic effects to workers exposed to the levels permitted by the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for dichloroacetylene. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

DIPROPYLENE GLYCOL METHYL ETHER

CAS: 34590-94-8; Chemical Formula: $CH_3OC_3H_6OC_3H_6OH$
H.S. No. 1149

OSHA currently has an 8-hour TWA limit of 100 ppm for dipropylene glycol methyl ether (DPGME), with a skin notation. The ACGIH recommends a TLV-TWA of 100 ppm and a TLV-STEL of 150 ppm for this colorless liquid with a mild, pleasant, ethereal odor and a bitter taste.

DPGME is a central nervous system and cardiac depressant. Dogs receiving intravenous injections of DPGME exhibited auricular fibrillation caused by auricular anoxia, depressed conduction, and heart block. Ventricular asystole accompanied increased intra-auricular pressure. Irritation and local pain preceded death from respiratory depression (Shideman and Procita 1951). Rowe and associates (1954) reported a single acute oral LD_{50} for rats of 5.4 ml/kg. Even at the highest levels tested (not further specified), no single application of DPGME to the skin of rabbits was lethal, although some narcosis and transient weight loss did occur. However, a significant number of deaths occurred in a group of rabbits treated with 65 repeated dermal applications of 3 ml/kg and higher during a 90-day period. Four animal species, including the monkey, were exposed repeatedly to 7-hour daily inhalation exposures of between 300 and 400 ppm DPGME; the animals exhibited narcosis and changes in the lung and liver (Rowe, McCollister, Spencer et al. 1954).

Humans inhaling levels of 300 to 400 ppm found them very disagreeable, but 100 ppm was tolerable and, in the opinion of the authors, was not likely to produce organic injury (Rowe, McCollister, Spencer et al. 1954). Patch tests on the skin of 250 human subjects produced neither irritation nor sensitization (ACGIH 1986, p. 221). Humans exposed to DPGME vapor concentrations at levels between 50 to 2000 ppm experienced eye, nose, and throat irritation before the onset of CNS impairment, which occurred at 1000 ppm in one of two subjects (Stewart, Baretta, Dodd, and Terkelson 1970).

OSHA is proposing a PEL of 100 ppm TWA and a STEL of 150 ppm for dipropylene glycol methyl ether. The Agency believes this combined limit will reduce the risk of central nervous system effects and irritation that may exist when workers are exposed for short periods above the 100 ppm PEL. In addition, OSHA is proposing a skin notation because of evidence in experimental animals that DPGME can be percutaneously absorbed. The health evidence forms a reasonable basis for proposing a revision of this level. At the time of the final rule, OSHA will establish a new limit for dipropylene

glycol methyl ether if the Agency determines that this limit will substantially reduce significant risk.

n-HEXANE
CAS: 110-54-3; Chemical Formula: $CH_3(CH_2)_4CH_3$
H.S. No. 1200

n-Hexane has been shown to produce distal axonopathy in both experimental animals and humans; it is metabolized to 2,5-hexanedione (2,5-HD), which is thought to be the causative agent of most of the adverse neurological effects observed after exposure to hexane (Schaumburg et al. 1983). OSHA's current PEL for n-hexane is 500 ppm. The ACGIH arrived at a TLV at 50 ppm for this substance, based primarily on studies (Miyagaki 1967; Inoue et al. 1970) showing peripheral neuropathies at exposure levels as low as 210 ppm. The REL for n-hexane recommended by NIOSH is 100 ppm (10-hour TWA). NIOSH based its recommendation on the same studies as those cited by the ACGIH (Miyagaki 1967; Inoue et al. 1970). NIOSH reasoned as follows:

> The absence of definitive epidemiologic or toxicologic evidence makes it difficult to determine how much lower the environmental limit should be. Professional judgment suggests a TWA concentration of 350 mg/m³ (100 ppm) offers a sufficient margin of safety to protect against the development of chronic nerve disorders in workers (NIOSH 1977a, p. 74).

The adverse neurological effects of hexane exposure are manifested as both sensory and motor dysfunction. Initially, there is a symmetric sensory numbness of the hands and feet, with loss of pain, touch, and heat sensation. Motor weakness of the toes and fingers is often present; as the neuropathy becomes more severe, weakness of the muscles of the arms and legs may also be observed (Schaumburg et al. 1983). There are no known conditions that predispose an individual to hexane neurotoxicity (Schaumburg et al. 1983). The onset of neurological symptoms may not be evident for several months to a year after the beginning of exposure. Recovery may be complete, but severely exposed individuals often retain some degree of sensorimotor deficit.

The dose-response relationship for n-hexane exposure in humans is not well defined, although it is clear that the severity of the resulting neuropathy increases as the exposure level of n-hexane increases. A number of studies have shown a consistent relationship between exposure levels of 500 ppm (OSHA's current exposure limit) to 2000 ppm and the development of characteristic peripheral neuropathies (Yamamura 1969; Yamada 1967). Neuropathic effects have also been shown to occur at levels between 210 and 500 ppm (Takeuchi et al. 1975).

Reports of effects occurring at levels of 210 to 500 ppm indicate that the current OSHA PEL of 500 ppm is not adequate to protect exposed workers from adverse sensorimotor neuropathic effects, and exposure at this level thus represents a risk to workers. The decreased sensitivity to pain, touch, and temperature associated with n-hexane exposure can also make a worker more susceptible to injuries and accidents. Further, the delayed onset of a clinical response, which is typical of hexane exposure, increases the probability that exposure will continue until irreversible effects occur.

The NIOSH REL is 100 ppm. However, both the presence of peripheral neurophathies at 210 ppm and the delay in onset of neurological symptoms indicate the need to adopt the 50 ppm TLV to prevent development of these symptoms. Sampling data from the IMIS data base show that 75 percent of the samples are below the 50 ppm level, and additional information confirms that attaining this level is feasible. OSHA therefore proposes a PEL of 50 ppm TWA. This health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for n-hexane if the Agency determines that this limit will substantially reduce significant risk.

2-HEXANONE (METHYL n-BUTYL KETONE)
CAS: 591-78-6; Chemical Formula: $CH_3CO-CH_2CH_2CH_2CH_3$
H.S. No. 1202

OSHA's current PEL for 2-hexanone is 100 ppm TWA; the NIOSH REL is 1 ppm (10-hour) TWA; and the ACGIH recommends a TLV-TWA of 5 ppm. Industrial exposure to 2-hexanone causes distal neuropathy manifesting as interference with motor and sensory function; even in cases characterized by minimal intensity, electrodiagnostic abnormalities were seen (ACGIH 1987). In animals, exposure to 2-hexanone causes axonal swelling and thinning of the myelin sheath. A metabolite of 2-hexanone, 2,5-hexanedione, appears to be responsible for the neural damage; this same metabolite is formed when n-hexane (discussed above) is metabolized.

The limit of 5 ppm TWA for 2-hexanone recommended by the ACGIH is based on the results of several different studies. These include a study showing decreases in sciatic-tibial nerve conduction in animals exposed to levels of 75 ppm for 9 months (Johnson et at. 1979); another study reporting neuropathy in animals after 6 months of exposure to 50 ppm (Streletz, Duckett, and Chambers 1976); and a study identifying 2,5-hexanedione in the serum of humans after a 1-day exposure to 50 ppm (diVincenzo, Kaplan, and Dedinas 1976).

The NIOSH REL for 2-hexanone of 1 ppm (10-hour TWA) is based on an epidemiologic study describing an outbreak of neurologic disease among workers in a plant that manufactures printed fabrics (Allen et al. 1975, as cited in NIOSH 1978). This study reported that a screening of 1,157 exposed workers revealed 86 verified cases of distal neuropathy. 2-Hexanone was suspected of being the neurotoxicant because it had only recently been introduced into the process (Allen et al. 1975). When recommending its limit, NIOSH relied on an industrial hygiene survey of the plant conducted by Billmaier et al. in 1974, which showed that 2-hexanone concentrations near the textile printing machines ranged from 1 to 156 ppm (10-minute area samples). After reviewing this evidence, NIOSH concluded that the 1 ppm level could not a considered a no-effect level for 2-hexanone-induced neuropathy.

The ACGIH (1987) stated that interpretation of the results of the Billmaier study was complicated because the exposure measurements reported in the study had been taken after the outbreak of neuropathic effects had occurred. In addition, the ACGIH pointed out that Billmaier et al. found poor work practices at the plant (gloves were rarely used, employees washed their hands with solvent, etc.), and thus dermal exposure may have contributed substantially to the outbreak.

Both human and animal studies show the development of disease at exposure levels below the existing 100 ppm PEL, clearly indicating the need to reduce this risk. There is disagreement regarding the interpretation of the reported epidemiology study to substantiate the 1 ppm REL. At this time OSHA has inadequate data to support feasibility at the 1 ppm level, while feasibility at 5 ppm has been demonstrated. OSHA therefore proposes adoption of a 5-ppm (TWA) PEL. The health evidence forms a reasonable basis for proposing a revision in this level. At the time of the final rule, OSHA will establish a new limit for 2-hexanone if the Agency determines that this limit will substantially reduce significant risk.

IRON PENTACARBONYL
CAS: 13463-40-6; Chemical Formula: $Fe(CO)_5$
H.S. No. 1216

OSHA currently has no limit for iron pentacarbonyl. The ACGIH recommends a TLV-TWA of 0.1 ppm with a TLV-STEL of 0.2 ppm, as iron, for this highly flammable, oily, colorless-to-yellow liquid.

In studies of rats, iron pentacarbonyl has been reported to have approximately one-third the acute toxicity of nickel carbonyl (for which ACGIH has recommended a TLV of 0.05 ppm TWA) (Sunderman, West, and Kincaid 1959). In 1970, Gage found that a 5.5-hour exposure at 33 ppm caused fatalities in three of eight rats; four of eight animals died after two 5.5-hour exposures at 18 ppm. At 7 ppm, no ill effects were observed in rats exposed 18 times for 5.5 hours (Gage 1970). There are no reports of long-term dose-response exposure studies in laboratory animals, and no evidence exists that iron pentacarbonyl is carcinogenic in either man or animals (ACGIH 1986, p. 327).

Immediate symptoms of acute exposure to high concentrations of iron pentacarbonyl include headache and dizziness, followed in 12 to 36 hours by fever, cyanosis, cough, and shortness of breath. The primary clinical effect is lung injury, although degenerative changes in the central nervous system have also been reported (ACGIH 1986, p. 327).

OSHA proposes a permissible exposure limit of 0.1 ppm TWA and a STEL of 0.2 ppm for iron pentacarbonyl. OSHA preliminarily concludes that these limits will protect exposed workers from the risk of headache, dizziness, fever, dyspnea, cyanosis, pulmonary injury, and central nervous system effects potentially associated with exposures at the levels permitted in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for iron pentacarbonyl. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

MERCURY (ARYL AND INORGANIC COMPOUNDS)
CAS: 7439-97-6: Chemical Formula: Hg
H.S. No. 1240

The current OSHA limit for all inorganic forms of mercury is 0.1 mg/m^3 Hg as a ceiling limit; this limit was adopted from ANSI standard Z37.8 (1943). ACGIH currently recommends a 0.1 mg/m^3 TLV-TWA for aryl and inorganic mercury compounds. NIOSH (1973b) has recommended a 0.05 mg/m^3 limit as an 8-hour TWA.

In 1971, the ACGIH recommended a 0.05 mg/m^3 TLV-TWA for all forms of mercury, including inorganic compounds. ANSI also reduced its standard to 0.05 mg/m^3 in 1972, and NIOSH recommended the same limit in 1973. The 0.05 mg/m^3 limit was based largely on the study of Smith et al. (1970) on workers exposed to mercury levels ranging from less than 0.1 to 0.27 mg/m^3 in chlor-alkali plants. The authors reported a significant dose-related increase in the incidence of weight loss, tremors, abnormal reflexes, nervousness, and insomnia among workers exposed to 0.1 mg/m^3 or more. There were slight increases in the incidence of insomnia and loss of appetite among workers exposed to 0.1 mg/m^3 or less. Smith et al. (1970) concluded that a limit of 0.1 mg/m^3 contained little or no margin of safety. Other studies (Bidstrup et al. 1951; Turrian et al. 1956) have also reported symptoms of mercury poisoning among workers exposed below 0.1 mg/m^3. The 0.05 mg/m^3 limit established by ACGIH, ANSI, and NIOSH also follows the 1968 recommendation of an international committee (Permanent Commission & International Association on Occupational Health 1968).

In 1980, the ACGIH revised its recommended TLV for aryl and inorganic mercury compounds to 0.1 mg/m^3 Hg. In revising this limit, the ACGIH cited discrepancies in the literature regarding the ratio of blood and urinary mercury levels to airborne concentrations of mercury (Bell et al. 1973; Stopford et al. 1978). These studies reported lower ratios of mercury body burden to airborne concentration when personal sampling is used rather than area sampling. According to Bell et al. (1973), the lower ratio results because mercury exposure measurements are generally found to be higher when personal sampling is conducted, presumably as a consequence of contamination of clothing. The ACGIH argued that the 0.05 mg/m^3 limit may be too stringent to apply when personal sampling is conducted. The ACGIH also stated that, unlike elemental or alkyl mercury, little mercury is deposited in the brain following exposure to aryl or inorganic mercury compounds. Based on this reasoning, the ACGIH adopted the higher 0.1 mg/m^3 TLV-TWA for aryl and inorganic compounds of mercury. However, the ACGIH (1986) also noted that, although central nervous system effects are less likely to occur from exposure to mercury salts than from other forms of mercury, the risk of renal and oral effects would "presumably be just as great." Therefore, they cautioned that the higher limit for mercury salts "may be subject to debate" (ACGIH 1986). The 0.05 mg/m^3 REL for the aryl and inorganic forms of mercury was established in 1973 and has been superseded by the 1980 ACGIH TLV of 0.1 mg/m^3 TWA, which is based on more recent studies. However, adopting the TLV would constitute a deregulation of OSHA's current standard for mercury. Because the health effects data available do not satisfy the requirements for deregulation, OSHA proposes to maintain its current 0.1 mg/m^3 ceiling PEL.

METHYLACRYLONITRILE
CAS: 126-98-7: Chemical Formula: $CH_2=C(CH_3)C=N$
H.S. No. 1251

There is no current OSHA standard for methylacrylonitrile, and NIOSH has no REL for this substance. The ACGIH recommends a 1-ppm TLV-TWA with a skin notation to protect workers who are occupationally exposed to methylacrylonitrile.

Methylacrylonitrile has been shown to be extremely toxic in animals, both by inhalation and dermal absorption. Beagles exposed for 90 days to 13.5 ppm convulsed and lost motor control in their hind limbs. Microscopic brain lesions were detected in one of the dogs. The level at which no effects were detected was determined to be between 3.2 ppm and 8.8 ppm (ACGIH 1986).

Neuropathic effects in dogs at levels of exposure below 8.8 ppm indicate that, in the absence of an exposure limit, occupational exposure to methyl acrylonitrile may be associated with a risk of neurological impairment. OSHA believes that establishing a 1 ppm PEL (which provides for a margin of safety below the observed no-effect level in dogs) and a skin notation will reduce the risk of neurological impairment that currently exists in the absence of an OSHA limit for this substance. The health evidence forms a reasonable basis for proposing a new limit for methylacrylonitrile. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce a significant risk.

METHYL BROMIDE
CAS: 74-83-9; Chemical Formula: CH_3Br
H.S. No. 1253

OSHA's current PEL for methyl bromide is a 20 ppm ceiling, while the ACGIH limit is 5 ppm as an 8-hour TWA, with a skin notation. NIOSH recommends that the REL for this substance be set at the lowest feasible level. Acute poisoning from methyl bromide is characterized by lung irritation, pulmonary edema, convulsions, and coma. Chronic exposure to low concentrations of methyl bromide generally produces

central nervous system effects, including muscle weakness and pain, incoordination, inability to focus one's eyes, and behavioral changes (ACGIH 1986; Craft 1983). The onset of neurological signs and symptoms is delayed for several hours to a few days after exposure.

Methyl bromide is a gas and an inhalation hazard, although it can also be absorbed through the skin. A report by Hine (1969) notes that methyl bromide has been responsible for more deaths among occupationally exposed workers in California than the organophosphates. It is hypothesized that methyl bromide has a greater potential for toxicity than other organic bromides, because its greater lipophilicity provides increased access to the brain.

Various studies demonstrate methyl bromide's toxicity in humans. Ingram (1951) reports ill effects (symptoms not specified) after exposure to methyl bromide at concentrations of 100 ppm. Similar exposure concentrations were also reported by Hine (1969) in a case study of two date packers in California. Johnson (1977) indicates that 34 packers became sick when exposed to an average methyl bromide concentration of 50 ppm, although concentrations in the packing room may have been as high as 100–150 ppm during the purging of a fumigation chamber.

Watrous (1942) described nausea, vomiting, and headache in 90 workers who were exposed for 2 weeks to concentrations "generally below" 35 ppm. These symptoms emphasized the need to create a TLV to protect workers from the nausea, vomiting, and headaches associated with lower levels of exposure. This need is strengthened by the fact that since these symptoms are usually delayed in onset, workers may not have sufficient warning of this substance's potential neurotoxicity.

The presence of neurologic symptoms at levels below 35 ppm indicates that a ceiling limit of 20 ppm is not adequate to protect workers from the effects of methyl bromide poisoning. OSHA proposes a PEL of 5 ppm TWA, with a skin notation, to more adequately protect workers against these incapacitating symptoms and to reduce this existing risk substantially. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for methyl bromide if the Agency determines that this limit will substantially reduce significant risk.

MANGANESE FUME
CAS: 7439-96-5; Chemical Formula: MnO
H.S. No. 1236a

OSHA currently has a ceiling limit of 5 mg/m³ for manganese fume. Because of its potential for damage to the lungs and central nervous system, the ACGIH recommends an 8-hour TWA of 1 mg/m³ and a 3-mg/m³ STEL for manganese fume.

Symptoms of manganese poisoning range from sleepiness and weakness in the legs (Fairhall 1957) to difficulty in walking and uncontrolled laughter (Fairhall and Neal 1943). Health surveys of employees exposed to manganese fume have demonstrated a high incidence of pneumonia in these workers (Davies 1946). Tanaka and Lieben (1969) found seven cases of pneumonia and 15 borderline cases of pneumonia among 144 workers exposed to manganese dust or fume concentrations greater than 5 mg/m³; three of these cases were associated with fume rather than dust exposure. Those workers exposed to fume levels below 5 mg/m³ exhibited no signs of pneumonia. In a separate study by Smyth, Ruhf, Whitman, and Dugan (1973), 3 cases of manganese poisoning were detected among 71 employees exposed to levels of 13.3 mg/m³ fume.

OSHA is proposing a 1-mg/m³ TWA and a 3-mg/m³ STEL for manganese fume. The Agency preliminarily concludes that both a TWA limit and STEL are required to protect exposed employees from the risk of manganese poisoning, lung damage, and pneumonia associated with exposure to these fumes. The health evidence forms a reasonable basis for proposing a new limit for manganese fume. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

MANGANESE CYCLOPENTADIENYL TRICARBONYL
CAS: 12079-65-1; Chemical Formula: C_5H_5-$Mn(CO)_3$
H.S. No. 1237

OSHA currently has no limit for exposure to manganese cyclopentadienyl tricarbonyl (MCT). The ACGIH recommends a TLV-TWA of 0.1 mg/m³ (as manganese), with a skin notation.

A Russian study reported that a single 2-hour exposure to MCT at 120 mg/m³ was fatal to 80 percent of albino rats, although rabbits, guinea pigs, and rats survived a single 2-hour exposure at 20 to 40 mg/m³. Chronic exposure of rats for 11 months at levels averaging 1 mg/g³ for 4 hours daily showed delayed effects (7 months from onset of exposure) on neuromuscular excitability, evidence of kidney damage, and decreased resistance to infection (Arkipova, Tolgskaya, and Kocketkova 1965). The tails of 10 white mice were dipped in a gasoline mixture containing 1 gram MCT per 100 ml; a second group of mice had their tails immersed in gasoline without MCT. An equal number of fatalities were observed in the gasoline plus MCT and gasoline only groups after four or five 2-hour applications, and all tails exhibited necrosis. The authors concluded that these effects were caused by the gasoline and not by the MCT (Arkipova, Tolgskaya, and Kocketkova 1965). Further studies in rabbits showed that MCT applied dermally as an oil emulsion caused irritation of the skin. These authors also investigated the dermal toxicity of tetrahydrofuran in MCT solutions versus tetrahydrofuran in oil. All animals whose tails had been dipped in the hydrofuran solution of MCT died within an hour, while animals whose tails had been dipped in pure tetrahydrofuran did not (Arkipova, Tolgskaya, and Kocketkova 1965). The same authors concluded that MCT is toxic at low concentrations, has cumulative properties, affects the nervous system, is irritating to the skin, and causes early histological changes in the respiratory tract.

More recent reports describe MCT-induced pulmonary edema and convulsions in the rat (Penney et al. 1985). The ED_{50}s for convulsions were 32 mg/kg orally and 20 mg/kg intraperitoneally; LD_{50}s were 24 mg/kg orally and 14 mg/kg intraperitoneally. Necrosis of the bronchiolar tissue and pulmonary parenchymal damage were seen in mice and rats given intraperitoneal doses (Haschek et al. 1982).

OSHA has preliminary concluded that occupational exposure to MCT poses a risk of neuropathic effects, kidney damage, skin irritation, pulmonary edema, and tissue damage. The Agency is therefore proposing an 8-hour TWA PEL of 0.1 mg/m³ for manganese cyclopentadienyl tricarbonyl, with a skin notation, to protect workers against the risk of these effects, which have been shown to occur at the levels permitted by the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for manganese cyclopentadienyl tricarbonyl. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

MANGANESE TETROXIDE
CAS: 1317-35-7; Chemical Formula: Mn_3O_4
H.S. No. 1238

OSHA currently has no limit for manganese tetroxide (compounds and fume). The ACGIH recommends a TLV-TWA of 1 mg/m³, as manganese, for this brownish-black powder and its fume. Ferromanganese fume has been determined by x-ray diffraction analysis to consist primarily of manganese tetroxide.

Findings from a Russian study indicated that intratracheal suspensions of manganese oxide, manganese dioxide, and manganese tetroxide particles (particle size less than 3um) produced pneumonitis and other similar pulmonary effects in rats (Levina and Robachevsky 1955). These investigators also determined that manganese tetroxide has a greater toxicity than the lower oxides of manganese and that freshly prepared oxides were more potent than those stored for 6 months to a year.

Two cases of manganese fume poisoning were reported in a plant where concentrations were between 2.7 and 4.7 mg/m³ (Whitlock, Kimuso, and Bittenbender 1966), but other investigators have questioned these air analysis data and believe that exposures to manganese tetroxide concentrations of 5 mg/m³ or less cause no harmful effects (Whitman and Brandt 1966). In a 7-year study, Smyth and co-workers investigated chronic manganese poisoning in workers exposed to both ferromanganese fumes and dust. Five of 71 employees suffered from chronic manganism; of these five cases, three resulted from fume exposure and two from dust exposure. Two of the three fume-exposure victims were exposed over a 5-year period to an estimated average ferromanganese concentration of 13.3 mg/m³; however, the third victim worked in an operation where air concentrations of manganese were less than 1 mg/m³, which suggests that certain individuals may be hypersusceptible to manganese poisoning. The dust-exposed victims worked in areas where air concentrations were in the range of 30 to 50 mg/m³ throughout the study period (Smyth, Ruhf, Whitman, and Dugan 1973).

Martonik (cited in ACGIH 1986, p. 357) reports that the fume has greater toxicity than the dust. During a 2-year period, at least one case of acute manganese poisoning was documented at a fume concentration level of 7.5 mg/m³ and another case at the same welding operation may have been manganism.

OSHA is proposing a 1 mg/m³ 8-hour TWA for manganese tetroxide (compounds and fume). The Agency preliminarily concludes that this limit will provide protection against the risk of chronic manganese poisoning, pneumonitis, and other respiratory effects associated with exposure to manganese tetroxide at the levels presently permitted by the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for manganese tetroxide. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

MERCURY (VAPOR)
CAS: 7439-97-6
H.S. No. 1241

OSHA currently has a ceiling limit of 0.1 mg/m³ for mercury (including vapor). The ACGIH recommends a TLV-TWA of 0.05 mg/m³ for mercury vapor, measured as mercury, and a skin notation. Elemental mercury is a silvery, odorless, heavy liquid.

Inhalation of high concentrations of mercury vapor for relatively brief periods can cause pneumonitis, bronchitis, chest pain, dyspnea, coughing, stomatitis, gingivitis, salivation, and diarrhea (NIOSH 1973; Ashe, Largent, Dutra et al. 1953). Chronic mercurialism is manifested by central nervous system effects, including tremor, a variety of neuropsychiatric disturbances, and loss of appetite (Kazantzis 1968; Smith, Vorwald, Patil, and Mooney 1970).

Severe organ damage occurred in rabbits exposed for 4 hours to an average vapor concentration of 28.8 mg/m³. Damage was observed in the kidneys, liver, brain, heart, lungs, and colon (Ashe, Largent, Dutra et al. 1953). The oral LD_{50} in rats for mercuric vapor is 18 mg/kg (NTIS PB 214-270, as cited in ACGIH 1986, p. 358). A study by Smith, Vorwald, Patil, and Mooney (1970) indicated that workers in the chlorine-producing industry who were exposed to mercury showed that chronic exposures to a 1-mg/m³ vapor concentration produced no effects in these workers. Six of 75 workers regularly exposed to 0.05 to 0.1 mg/m³ of mercury vapor in a glassware manufacturing plant reported insomnia, and one was found to have tremors (Danziger and Possick 1973). One of 11 workers employed in a mercury mine or refining plant and exposed at vapor concentrations below 0.1 mg/m³ had sore gums, loose teeth, or excess salivation (Rentos and Seligman 1968).

NIOSH (1973) has recommended a 10-hour TWA limit of 0.05 mg/m³ for inorganic mercury and concluded that hyperactivity, rather than tremor, may be the most typical symptom of chronic mercurialism. Two studies report no evidence of mercury vapor poisoning in industrial settings where characteristic exposures ranged between 0.05 and 0.1 mg/m³ (Danziger and Possick 1973; McGill et al. 1964).

In workers exposed at levels about 0.1 mg/m³, toxic symptoms were seen (Rentos and Seligman 1968). Turrian, Grandjean, and Turrian (1956) found that 33 percent of workers exposed to the vapor at levels above 0.05 mg/m³ had erethism, while only 8 percent of those exposed below this level manifested this symptom. About 20 percent of workers in both groups exhibited tremor. The ACGIH notes that, after exposure to the vapor, "a relatively high percentage of the absorbed mercury remains in the brain" compared with the amount deposited in the brain after exposure to the aryl and inorganic compounds (ACGIH 1986, p. 359). The ACGIH accordingly recommends a higher TLV-TWA for these compounds.

OSHA proposes a PEL of 0.05 mg/m³ TWA for mercury vapor, with a skin notation. The Agency preliminarily concludes that this limit will protect workers against the risk of acute and chronic mercury poisoning that exists from workplace exposures to this vapor at levels above 0.05 mg/m³. The skin notation is proposed because the vapors of elemental mercury can be readily absorbed through the skin. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for mercury (vapor) if the Agency determines that this limit will substantially reduce significant risk.

MERCURY, (ORGANO) ALKYL COMPOUNDS
CAS: 7439-97-6
H.S. No. 1242

OSHA has a current PEL of 0.01 mg/m³ TWA and a ceiling limit of 0.04 mg/m³ for the alkyl compounds of mercury. The ACGIH recommends a TLV-TWA of 0.01 mg/m³ and a TLV-STEL of 0.03 mg/m³ for these compounds, as mercury. A skin notation is also recommended by the ACGIH. Alkyl mercury compounds include volatile liquids, such as dimethyl and diethyl mercury, as well as many complex salts, which are usually solids.

Alkyl mercury compounds pose greater health hazards than do the inorganic compounds or mercury because they can penetrate the blood-brain barrier and the placenta very quickly. The primary toxic effects associated with exposure to the organic compounds of mercury are injuries to the central and peripheral nervous

systems and to the kidneys (Casarett and Doull 1975). In addition, data concerning mouse and rat exposures to alkyl mercury compounds have demonstrated toxicity to the gastrointestinal system, pancreas, liver, gonads, and cardiovascular system. Suppression of the immune system and impairment of the endocrine system have also been demonstrated (Shakbazyan et al. 1977). Fatalities in mice have been reported at exposures of 10 to 30 mg/m³ for 3 to 5 hours (Trakhtenberg 1950).

Methyl mercury is among the most damaging of the alkyl compounds to humans because it accumulates in the body and causes developmental effects (Wilson 1977). A 3-month exposure to approximately 1 mg/m³ diethyl mercury caused death in two individuals (Hill 1943). Another fatal case of alkyl mercury poisoning has also been described (Hook, Lundgren, and Swensson 1954). On the basis of his work with laboratory animals, Trakhtenberg (1950) stated that even a concentration as low as 0.00001 mg/m³ could not be tolerated by humans on a continuing basis. However, a later study reported no consistent, acute effects of mercury poisoning at air concentrations between 0.01 and 0.1 mg/m³, despite the fact that brief excursions considerably above this range occurred (Dinman, Evans, and Linch 1958).

OSHA is proposing a PEL of 0.01 mg/m³ TWA and a 15-minute STEL of 0.03 mg/m³ for the alkyl compounds of mercury, with a skin notation. The Agency preliminarily concludes that exposure to the alkyl mercury compounds poses a risk of severe neuropathic and other systemic injury. The Agency believes that both the short-term and 8-hour limits are necessary to reduce these risks and that the skin notation will protect against the dermal absorption possible in the absence of any OSHA limit or skin notation. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for mercury, (organo) alkyl compounds if the Agency determines that this limit will substantially reduce significant risk.

PENTABORANE
CAS: 19624-22-7; Chemical Formula: B_5H_9
H.S. No. 1304

OSHA's current limit for pentaborane is 0.005 ppm as an 8-hour TWA. The ACGIH has the same 8-hour TWA but additionally recommends a 15-minute STEL of 0.015 ppm. Pentaborane is a colorless liquid with a strong and penetrating odor.

In both humans and animals, inhalation of pentaborane vapor causes central nervous system effects (Svirbely 1954; Rozendaal 1951; Lowe and Freeman 1957; Cordasco, Cooper, Murphy, and Anderson 1962).

The 5-minute LC_{50} for rats and mice is 67 and 40 ppm, respectively; for 60 minutes, these values are 10 and 6 ppm for rats and mice, respectively (Weir, Bath, and Weeks 1961, as cited in ACGIH 1986, p. 459). Rats exposed repeatedly to 3-ppm pentaborane by inhalation exhibited tremors, hyperexcitability, belligerency, and weight loss (Svirbely 1954). Rats, rabbits, monkeys, and dogs exposed repeatedly to pentaborane vapor at concentrations of 1 ppm for 4 weeks or 0.2 ppm for 6 months lost weight (Levinkas, Paslian, and Bleckman 1958). In the same experiments, rats and rabbits exposed at 1 ppm showed reduced activity and impaired locomotor ability, respectively, and monkeys and dogs exhibited apathy, loss of appetite, insensitivity to pain, loss of mobility, tremor, and impaired coordination. The ACGIH (1986, p. 459) notes that the 0.2 ppm concentration reported in the Levinkas et al. study (1958) was a calculated rather than measured value and that the actual exposure level was probably closer to 0.01 ppm.

Humans accidentally overexposed to pentaborane experience tremors, convulsions, behavioral changes, loss of memory, impaired judgment, and other symptoms of central nervous system intoxication (Svirbely 1954; Rozendaal 1951; Lowe and Freeman 1957; Cordasco, Cooper, Murphy, and Anderson 1962).

OSHA is proposing an 8-hour TWA of 0.005 ppm for pentaborane and a STEL of 0.015 ppm. The Agency preliminarily concludes that these limits will protect workers against the risk of central nervous system effects, such as tremors and convulsions, behavioral changes, and loss of judgment, potentially associated with exposure to pentaborane at levels only slightly above those that would be premitted by the 8-hour TWA alone. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for pentaborane if the Agency determines that this limit will substantially reduce significant risk.

PHENYL MERCAPTAN
CAS: 108-98-5; Chemical Formula C_6H_5SH
H.S. No. 1316

OSHA has no current limit for phenyl mercaptan. The ACGIH recommends a TLV-TWA of 0.5 ppm. NIOSH recommends a 15-minute ceiling limit of 0.1 ppm for phenyl mercaptan (benzenethiol). Phenyl mercaptan is a colorless liquid with an offensive, garlic-like odor.

The primary acute hazards of exposure to phenyl mercaptan are central nervous system stimulation followed by post-convulsive CNS depression, severe eye and skin irritation, systemic toxicity to spleen, kidney, lung, and liver tissues, and narcotic effects (ACGIH 1986, p. 478).

Phenyl mercaptan has been reported to have 4-hour inhalation LC_{50} values of 33 and 28 ppm for rats and mice, respectively (Doull and Plzak 1962; Fairchild and Stokinger 1958). The oral LD_{50} for the rat is reported to be 46 mg/kg (McCord and Witheridge 1949; Robles 1975, as cited in ACGIH 1986, p. 478). For the rabbit and rat, the dermal LD_{50} values are 134 mg/kg and 300 mg/kg, respectively (Doull and Plzak 1962; Fairchild and Stokinger 1958; Schafer 1972). The responses of animals to exposure to phenyl mercaptan were uniform regardless of species and progressed from CNS stimulation to incoordination, skeletal and muscular paralysis, respiratory depression, followed at high concentrations by coma and death. High doses (not further specified) administered via inhalation produced lung, liver, and kidney changes in mice (Doull and Plzak 1962; Fairchild and Stokinger 1958; Schafer 1972). In rabbits, phenyl mercaptan is a severe eye and skin irritant (McCord and Witheridge 1949; Robles 1975; Schafer 1972).

In humans, phenyl mercaptan is a moderately toxic skin irritant and causes severe dermatitis, headaches, and dizziness at unspecified levels (Fairchild and Stokinger, 1958; McCord and Witheridge 1949).

OSHA is proposing an 8-hour TWA limit of 0.5 ppm for phenyl mercaptan. The Agency preliminarily concludes that this limit will protect exposed workers from the risk of CNS effects, skin irritation, and systemic injury potentially associated with occupational exposure to phenyl mercaptan at the uncontrolled levels permitted by the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for phenyl mercaptan. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

PROPYLENE GLYCOL DINITRATE
CAS: 6423-43-4; Chemical Formula: $C_3H_6N_2O_6$
H.S. No. 1342

OSHA currently has no limit for propylene glycol dinitrate. The ACGIH

recommends a TLV–TWA of 0.05 ppm, with a skin notation. When freshly prepared, propylene glycol dinitrate is a colorless liquid with a disagreeable odor.

Exposure to this substance affects blood pressure, causes methemoglobinuria and respiratory toxicity, injures liver and kidney tissues, and distorts vision. It can also cause headache and incoordination.

The oral LD_{50} value for the rat is between 480 and 250 mg/kg (Clark and Litchfield 1969; Andersen and Mehl 1973), and the subcutaneous LD_{50} is 530 mg/kg (Andersen and Mehl 1973). Mice are reported to be somewhat more resistant, with a subcutaneous LD_{50} of slightly more than 1200 mg/kg; however, cats appear to be even more susceptible to propylene glycol dinitrate with a subcutaneous LD_{50} of between 200 and 300 mg/kg (Clark and Litchfield 1969). In all species studied, death occurs by anoxia, which is caused by almost complete conversion of hemoglobin to methemoglobin (Clark and Litchfield 1969). Skin tests in albino rabbits did not produce irritation, but ocular instillation caused transient conjunctival redness (Jones, Strickland, and Siegel 1972). Twenty-day skin exposures in rabbits at 1 g/kg showed minor irritation, and at 2 g/kg, rabbits became weak and cyanotic; one of five rabbits died, and this animal's hemoglobin and hematocrit values had decreased. When the dose was increased to 4 g/kg, the rabbits methemoglobin values rose to 34.5 percent at death, an indication, along with elevated serum and urinary nitrate levels, that propylene glycol dinitrate is readily absorbed through the skin (Jones, Strickland, and Siegel 1972). Continuous 90-day inhalation exposures in dogs at 10 ppm showed kidney and liver changes; exposures at 35 ppm caused heavy iron deposits in the liver, spleen, and kidneys. Female (but not male) rats showed a drop in blood pressure within 30 minutes after injection of doses above 5 mg/kg. Rhesus monkeys displayed mydriasis in 90-day exposures at 35 ppm but no change in avoidance behavior during a visual discrimination and acuity threshold test (Jones, Strickland, and Siegel 1972).

In humans, 8-hour exposures to 0.2 ppm or higher propylene glycol dinitrate resulted in visual distortion and headache (Stewart et al. 1974). Subjects developed a tolerance for the headache response, but the visual effects were cumulative. Impaired balance occurred after 6.5 hours of exposure at 0.5 ppm, and a 40-minute exposure at 1.5 ppm caused eye irritation. Subjects exposed at 0.5 ppm for 8 hours experienced a consistent elevation in diastolic pressure but no pulmonary irritation. At concentrations of 0.03 to 1.5 ppm, no hematologic effects were observed (Stewart et al. 1974). Studies of human exposures to levels below 0.1 ppm do not report evidence of chronic neurotoxicity (Horvath et al. 1981).

OSHA proposes a TWA limit of 0.05 ppm, with a skin notation, for propylene glycol dinitrate. The Agency believes that this limit will protect workers against the risk of hepatotoxic, hematologic, and central nervous system effects that exists from workplace exposure in the absence of any OSHA PEL. The skin notation is proposed to protect against the risk of skin absorption. The health evidence forms a reasonable basis for proposing a new limit for propylene glycol dinitrate. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

Preliminary Conclusions

OSHA preliminary concludes that risks are associated with occupational exposure to the group of neuropathic toxicants shown in Table C1–1. The effects caused by such exposures include brain lesions, nausea, vomiting, general depression of the central nervous system, interference with sensory and motor functions, and alterations in the ability of the brain to process information. Affected workers may experience drowsiness, dizziness, loss of ability to concentrate, mood changes, reduced awareness, learning difficulty, unsteadiness, and auditory and visual disturbances. In addition, employees experiencing these effects are at risk and are likely to hurt themselves or others in accidents caused by their reduced functional capacity. OSHA believes that the health evidence for these neurotoxins forms a reasonable basis for proposing new or revised limits. At the time of the final rule, the Agency will establish these limits if it determines that significant risk will be substantially reduced thereby.

2. Substances for Which Proposed Limits are Based on Avoidance of Narcosis Effects

Introduction

Proposed limits for 19 substances are based primarily on evidence showing that occupational exposure to these substances causes narcosis. The narcotic effects of exposure to substances such as alcohols, aliphatic hydrocarbons, and chlorinated hydrocarbons have been recognized in industry as serious adverse effects for many years. Table C2–1 lists these chemicals, their CAS and HS numbers, and their OSHA, ACGIH, and NIOSH limits. For seven of these substances, the Agency proposes to lower the 8-hour limit and to revise or add a STEL. In five cases, the 8-hour limit will remain the same and a proposed STEL will be added. Eight-hour TWAs and/or STELs are being proposed for four previously unregulated substances; in two cases, OSHA is proposing the NIOSH REL, and, in the remaining case, an existing limit would be replaced by a new value.

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TABLE C2-1. Substances for Which Limits Are Based on Avoidance of Narcosis

H.S. Number/ Chemical Name	CAS No.	CURRENT PEL*	ACGIH TLV**	NIOSH REL***
1044 Butane	106-97-8	—	800 ppm TWA	—
1049 sec-Butyl alcohol	78-92-2	150 ppm TWA	100 ppm TWA 150 ppm STEL	—
1050 tert-Butyl alcohol	75-65-0	100 ppm TWA	100 ppm TWA 150 ppm STEL	—
1111 Cyclopentane	287-92-3	—	600 ppm TWA	—
1163 Ethyl bromide	74-96-4	200 ppm TWA	200 ppm TWA 250 ppm STEL	—
1185 Gasoline	8006-61-9	—	300 ppm TWA 500 ppm STEL	—
1194 Heptane	142-82-5	500 ppm TWA	400 ppm TWA 500 ppm STEL	85 ppm TWA 440 ppm Ceiling (15 min)
1201 Hexane isomers	—	—	500 ppm TWA 1000 ppm STEL	100 ppm TWA 510 ppm Ceiling (15 min)

TABLE C2-1. Substances for Which Limits Are Based on Avoidance of Narcosis (continued)

H.S. Number/ Chemical Name	CAS No.	CURRENT PEL*	ACGIH TLV**	NIOSH REL***
1218 Isoamyl alcohol	123-51-3	100 ppm TWA	100 ppm TWA 125 ppm STEL	--
1221 Isophorone+	78-59-1	25 ppm TWA	5 ppm Ceiling	4 ppm TWA
1254 Methyl chloride	74-87-3	100 ppm TWA 200 ppm STEL 300 ppm Ceiling	50 ppm TWA 100 ppm STEL	Lowest feasible level
1255 Methyl chloroform (1,1,1-Trichloroethane)	71-55-6	350 ppm TWA	350 ppm TWA 450 ppm STEL	350 ppm Ceiling (15 min)
1296 Octane	111-65-9	500 ppm TWA	300 ppm TWA 375 ppm STEL	75 ppm TWA 385 ppm Ceiling (15 min)
1306 Pentane	109-66-0	1000 ppm TWA	600 ppm TWA 750 ppm STEL	120 ppm TWA 610 ppm Ceiling (15 min)
1307 2-Pentanone (Methyl propyl ketone)	107-87-9	200 ppm TWA	200 ppm TWA 250 ppm STEL	150 ppm TWA
1308 Perchloroethylene (Tetrachloroethylene)	127-18-4	100 ppm TWA 200 ppm STEL 300 ppm Ceiling	50 ppm TWA 200 ppm STEL	Lowest feasible limit

TABLE C2-1. Substances for Which Limits Are Based on Avoidance of Narcosis (continued)

H.S. Number/ Chemical Name	CAS No.	CURRENT PEL*	ACGIH TLV**	NIOSH REL***
1371 Stoddard solvent	8052-41-3	500 ppm TWA	100 ppm TWA (525 mg/m^3)	350 mg/m^3 TWA 1800 mg/m^3 Ceiling (15 min)
1397 Toluene	108-88-3	200 ppm TWA 300 ppm STEL 500 ppm Ceiling	100 ppm TWA 150 ppm STEL	100 ppm TWA (8-hr) 200 ppm Ceiling (10 min)
1406 Trichloroethylene+	79-01-6	100 ppm TWA 200 ppm STEL 300 ppm Ceiling	50 ppm TWA 200 ppm STEL	25 ppm TWA

* OSHA's TWA limits are for 8-hour exposures; its STELs are for the durations specified; and its ceilings are peaks not to be exceeded for any period of time.

** The ACGIH TWA-TLV is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

*** NIOSH TWA limits are for 10-hour exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

+ Proposed PEL is based on NIOSH REL.

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Description of the Health Effects

Narcosis is the result of general depression of central nervous system (CNS) function. When the CNS becomes sufficiently depressed, the awareness or consciousness of exposed workers is affected. Initial symptoms of narcosis include drowsiness, difficulty in concentration, and mood changes; these may progress to slurred speech, dizziness, and loss of coordination, and, in more severe cases, loss of consciousness, coma and death. Except in more serious cases, CNS depression is reversible on removal from exposure and causes no permanent damage to the CNS. However, because narcosis adversely affects workers' concentration and coordination, there is an increased risk of injuries and accidents caused by mistakes and errors in judgment.

The mechanism by which substances induce narcosis is poorly understood. CNS depressants may have the same mechanism of action as general anesthetics, which appear to produce a reversible effect on electrically excitable neuronal membranes.

Dose-Response Relationship and Narcotic Effects

The induction of narcosis following exposure to narcotic agents is expected to follow the classical S-shaped (sigmoidal) dose-response relationship. As exposure level increases, both the percent of exposed persons affected and the severity of the effect will increase. Although it is not known whether a true threshold exists for the occurrence of the molecular events leading to narcosis (i.e., disruption of electrical impulses in neurons), there is usually a level at which most exposed individuals will manifest the onset of symptoms associated with narcosis. The no-effect level for a particular substance will be determined largely by individual susceptibility, the extent to which the material is absorbed, and the rate at which it is metabolized and eliminated.

The following discussion describes OSHA's preliminary findings for the substances in this group and illustrates the potentially serious consequences of workplace exposure to these substances.

BUTANE
CAS: 106–97–8; Chemical Formula: C_4H_{10}
H.S. No. 1044

OSHA has no current limit for butane. the ACGIH recommends a threshold limit value of 800 ppm TWA for this colorless, flammable gas.

The primary risk of exposure to butane is its ability to cause narcosis at high exposure levels. Exposure to 10,000 ppm for 10 minutes causes drowsiness, but there are no reports of systemic toxicity or irritation (Gerarde 1963).

In rats, the 4-hour LC_{50} for butane is 658 gm/m³, or about 280,000 ppm (NIOSH 1977). Humans exposed to 1000 ppm for a single 8-hour day, or to 500 ppm for 2-week periods of 8-hour workdays, showed no harmful subjective or abnormal physiological responses but did show a reduced visual evoked response (VER) wave amplitude during the second week (Stewart et al. 1977).

OSHA is proposing a permissible exposure limit of 800 ppm TWA for butane. The Agency preliminarily concludes that this limit will protect exposed workers against the risk of narcosis and drowsiness potentially associated with exposures at previously uncontrolled levels. The health evidence forms a reasonable basis for proposing a new limit for butane. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

sec-BUTYL ALCOHOL
CAS: 78–92–2; Chemical Formula: $CH_3CH_2CHOHCH_3$
H.S. No. 1049

OSHA's current limit for sec-butyl alcohol is 150 ppm as an 8-hour TWA. The ACGIH recommends a TLV–TWA of 100 ppm, with a 15-minute STEL of 150 ppm. sec-Butyl alcohol is a colorless liquid with a strong, wine-like odor.

The acute toxicity of sec-butyl alcohol is reported to be lower than that of n-butanol, for which OSHA is proposing a ceiling of 50 ppm. The oral LD_{50}s in rats for these two substances are 6.5 g/kg for sec-butyl alcohol and 4.4 g/kg for n-butanol, respectively (Smyth, Carpenter, and Weil 1951). Liquid sec-butyl alcohol is less injurious to the eyes than liquid n-butanol (ACGIH 1986, p. 77). Occupational exposures to sec-butyl alcohol at levels of about 100 ppm were reported not to be associated with difficulties (Bank, unpublished communication, as cited in ACGIH 1986, p. 77).

OSHA proposes to reduce the permissible exposure limit for sec-butyl alcohol to 100 ppm TWA, and to include a 150-ppm STEL, to afford adequate protection against the risk of narcosis and irritation caused by short-term elevated exposures. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for sec-butyl alcohol if the Agency determines that this limit will substantially reduce significant risk.

tert-BUTYL ALCOHOL
CASL 75–65–0; Chemical Formula: $(CH_3)_3COH$
H.S. No. 1050

OSHA currently has a limit of 100 ppm for tert-butyl alcohol. The ACGIH recommends a TLV–TWA of 100 ppm, with a TLV–STEL of 150 ppm. At ordinary temperatures and pressures, tert-butyl alcohol exists in the form of colorless hygroscopic crystals (ACGIH 1986).

Although similar to the other butyl alcohols in many respects, tert-butyl alcohol is more volatile and has a greater potential for narcotic effects (Weese 1928). Mice exposed to t-butyl alcohol exhibit a stronger narcotic response than they show when exposed to normal or iso-butyl alcohol (Weese 1928). Repeated daily doses of t-butyl alcohol that produced narcosis were not fatal in animals (Schaffarzick and Brown 1952). In humans, contact with t-butyl alcohol produces erythema and hyperemia (Oetel 1936).

OSHA proposes an 8-hour TWA PEL of 100 ppm for tert-butyl alcohol, with a 15-minute STEL of 150 ppm. The Agency preliminarily concludes that this combination of limits will protect against the risk of narcosis permitted at elevated short-term levels. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for tert-butyl alcohol if the Agency determines that this limit will subtantially reduce significant risk.

CYCLOPENTANE
CAS: 287–92–3; Chemical Formula: $CH_2CH_2CH_2CH_2CH_2$
H.S. No. 1111

OSHA currently has no limit for cyclopentane. The ACGIH recommends a TLV–TWA of 600 ppm for this flammable, mobile liquid.

The few existing animal data indicate the cyclopentane is a central nervous system depressant. As with other alicyclic hydrocarbons, high concentrations cause excitement, loss of equilibrium, stupor, coma, and, rarely, respiratory failure (Gerard 1963). No major toxicological animal studies on the effects of cyclopentane exposure have been reported, and evaluations of the toxic properties of this substance have therefore relied on the animal data for n-pentane. n-Pentane has been shown to cause narcosis in animals at exposures of 90,000 to 120,000 ppm for 5 to 60 minutes (Abritti et al. 1976). Swann (1974) reported that a concentration of 130,000 ppm is fatal. Almost no data are available concerning the chronic effects of cyclopentane exposure.

Abritti et al. (1976) report that petroleum solvents used in the Italian shoe industry contain up to 18 percent cyclopentane. Workers exposed to these solvents have been observed to suffer from polyneuropathy, and Oettel reported in 1936 that skin exposure to such solvents caused burning and skin blistering after 15 minutes of confined contact. It has not been determined whether the irritation was caused by cyclopentane or by other substances, such as n-hexane, in the solvent.

OSHA is proposing a PEL of 600 ppm TWA for this substance. OSHA has preliminarily concluded that occupational exposure to cyclopentane poses a risk of irritation and narcosis at the levels permitted in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for cyclopentane. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

ETHYL BROMIDE
CAS: 74-96-4; Chemical Formula: C_2H_5Br
H.S. No. 1163

OSHA currently has an 8-hour TWA limit of 200 ppm for ethyl bromide. The ACGIH also recommends a limit of 200 ppm as an 8-hour TWA, and in addition recommends a 15-minute STEL of 250 ppm. Ethyl bromide is a colorless, highly volatile, and flammable liquid with an ether-like odor; it becomes yellow when exposed to light and air.

The concentrations of ethyl bromide reported as lethal to guinea pigs are 3200 ppm for 9 hours and 1700 ppm for 12.5 hours (Sayers, Yant, Thomas, and Berger 1929). Von Oettingen (1955) reported the minimal lethal concentration of this substance for mice as 3500 ppm.

Ethyl bromide acts as a central nervous system depressant (narcotic); additionally, exposure irritates the lungs and causes congestion and fatty degeneration of the liver, intestinal hemorrhage, and kidney swelling. Several deaths have been reported from the use of ethyl bromide as general anesthetic (von Oettingen 1955).

OSHA is proposing a PEL of 200 ppm as an 8-hour TWA and a 15-minute STEL of 250 ppm for ethyl bromide. The Agency preliminarily concludes that these limits will work to reduce the risk of narcosis, kidney and liver damage, and respiratory irritation associated with occupational exposure to elevated levels of ethyl bromide. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for ethyl bromide if the Agency determines that this limit will substantially reduce significant risk.

GASOLINE
CAS: 8006-61-9; Chemical Formula: None
H.S. No. 1185

There is no current OSHA PEL for gasoline. The ACGIH has established a 300-ppm 8-hour TWA and a 500-ppm 15-minute STEL.

Studies have shown that exposure to 2000 ppm of gasoline for 30 minutes produces mile anesthesia, while exposure to concentrations between 500 and 900 ppm for 1 hour produces dizziness (Gerarde 1963; Runion 1975). However, these authors also found that people exposed to gasoline at concentrations 160 to 270 ppm for several hours do not experience any symptoms of narcosis.

OSHA is proposing an 8-hour TWA of 300 ppm, supplemented by a STEL of 500 ppm to ensure that levels do not exceed 300 ppm for any length of time; these limits are intended to protect against the early symptoms of narcosis. OSHA preliminarily concludes that uncontrolled exposure to gasoline place exposed employees at risk of experiencing drowsiness, headaches, dizziness, and loss of coordination. The health evidence forms a reasonable basis for proposing a new limit for gasoline. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

HEPTANE
CAS: 142-82-5; Chemical Formula: $CH_3(CH_2)CH_2CH_3$
H.S. No. 1194

The current OSHA limit for heptane is 500 ppm as an 8-hour TWA. The ACGIH TLVs for heptane are 400 ppm as a TWA and 500 ppm as a STEL. NIOSH (1977a) has recommended that workplace exposures not exceed 85 ppm as a 10-hour TWA or 440 ppm as a 15-minute ceiling limit.

Patty and Yant (1939) reported that exposure to 1,000 ppm for 6 minutes caused slight dizziness in humans; exposures to higher levels caused vertigo, incoordination, and hilarity. They also reported that a 4-minute exposure to 5,000 ppm produced complaints of nausea and loss of appetite. Based on this information, as well as animal data showing 10,000 to 15,000 ppm as being an effect level for narcosis (Fuhner 1921), the ACGIH concluded that heptane was more acutely toxic than hexane. They therefore recommended limits that are somewhat lower than for hexane isomers.

As discussed above in the discussions on pentane and hexane isomers, NIOSH has recommended the same occupational limits for all C_5–C_8 alkanes (i.e., 350 mg/m³ TWA and 1800 mg/m³ as a 15-minute ceiling). This recommendation considers that all C_5–C_8 alkanes possess potential neurotoxic capability similar to that of n-hexane. The ACGIH disagrees with this concept, believing that the neurotoxicity caused by exposure to n-hexane is the result of a unique metabolite, 2,5-hexanedione.

Because heptane is considered to be more acutely toxic than hexane, OSHA preliminarily concludes that it is appropriate to revise its limit for heptane to a level below that of hexane isomers and, thus, reduce the risk of narcosis. Therefore, OSHA proposes to revise its limit for heptane to 400 ppm TWA and 500 ppm STEL. As in the case of hexane isomers and pentane, OSHA solicits comment on the evidence that heptane is potentially neuropathic.

HEXANE ISOMERS
CAS: None; Chemical Formula: $(CH_3)_2C_2H_2$; $n(CH_3)_4C_2H_2$
H.S. No. 1201

OSHA has no current limit for the hexane isomers. The ACGIH TLVs for hexane isomers are 500 ppm TWA and 1000 ppm STEL. NIOSH has a recommended TWA limit for these isomers of 100 ppm, supplemented by a 510-ppm (15 min) ceiling.

A study by Drinker et al. (1943) shows that humans exposed to 1400 to 1500 ppm of hexane experienced nausea and headache. Patty and Yant (1929) found that a 10-minute exposure to 5000 ppm caused giddiness and dizziness in exposed subjects. A study by Nelson et al. (1943) showed no effects in unacclimated subjects exposed to hexane isomers in concentrations of 500 ppm, but narcotic effects have often been seen in people exposed at levels above 100 ppm (Elkins 1959). The ACGIH based its recommendation primarily on the Nelson et al. (1943) study.

NIOSH recommends limits for the hexane isomers of 100 ppm as a 10-hour TWA and 510 ppm as a 15-minute short-term limit. This recommendation is based on human and animal evidence that exposure to n-hexane below concentrations of 500 ppm is associated with the development of polyneuropathy (Inoue et al. 1970; Miyagaki 1967); NIOSH (1977a) did not distinguish between n-hexane and other hexane isomers when making its recommendation for an exposure limit. It should be noted that NIOSH concluded that all C_5–C_8 alkanes are potential neuropathic agents and cited a human study (Gaultier 1973) that reported

neuropathy among workers exposed to an alkane mixture containing less than 5 percent n-hexane.

The ACGIH (1986, p. 307) disagrees with NIOSH that all C_5–C_8 alkanes are potential neuropathic agents, citing evidence that a metabolite of n-hexane (2,5-hexanedione) is responsible for the unique neurotoxic properties of n-hexane (see discussion of n-hexane in Section IV–C1 of this Preamble). The ACGIH concluded that "it seems unlikely that all the hexanes would follow the same metabolic route in the body [as n-hexane], in view of the marked variations in structure of the molecule" (ACGIH 1986, p. 307).

After reviewing the evidence cited by ACGIH (1986) and NIOSH (1977a), OSHA preliminarily finds that workers exposed to hexane isomers are at significant risk of experiencing narcosis and of developing neuropathy in the absence of an OSHA limit. The NIOSH RELs for the hexane isomers (100 ppm TWA and 510 ppm STEL) are based on the hypothesis that all of the C_5–C_8 alkanes are potential neuropathic agents. OSHA tentatively accepts the analysis that a unique metabolite of n-hexane is responsible for such effects and therefore proposes at this time a 500-ppm TWA and a 1000-ppm STEL for the hexane isomers. Because of the scientific disagreement on this subject, OSHA specifically requests comments on the toxicity or absence of toxicity of the other C_5–C_8 alkanes. The health evidence forms a reasonable basis for proposing a new limit for hexane isomers. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

ISOAMYL ALCOHOL (PRIMARY AND SECONDARY)
CAS: 123–51–3; Chemical Formula: $(CH_3)_2CHCH_2CH_2OH$—Primary; $(CH_3H_6)_2CHOH$—Secondary
H.S. No. 1218

OSHA's current limit for the isoamyl alcohols is 100 ppm as an 8-hour TWA. The ACGIH has established an 8-hour TLV–TWA of 100 ppm and a 15-minute STEL of 125 ppm for these substances, which are colorless liquids that have pungent tastes and an alcoholic odor that causes coughing.

In rats, the oral LD_{50} for the primary isoamyl alcohol in 7.07 mg/kg (Smyth et al. 1969). Haggard, Miller, and Greenburg (1945) determined that isoamyl alcohol's anesthetic toxicity was approximately 12 times higher than that of ethyl alcohol; these authors believed that exposure to isoamyl alcohol would not cause chronic effects.

Smyth (1956) reported that the principal effect of inhalation exposure to this substance is narcosis, and this author believed that, by analogy with the narcotic effects of butyl alcohol, a 100-ppm level would protect exposed workers against narcosis but not against irritation. Nelson, Ege, Ross and co-workers (1943) stated that unacclimatized human volunteers reported throat irritation at a concentration of 100 ppm, and eye and mucous membrane irritation at higher levels.

OSHA is proposing an 8-hour TWA of 100 ppm and a 15-minute STEL of 125 ppm for the isoamyl alcohols (primary and secondary). The Agency preliminarily concludes that these limits will work together to ensure that workers are protected against the eye and mucous membrane irritation known to be associated with exposures above 100 ppm. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for isoamyl alcohol if the Agency determines that this limit will substantially reduce significant risk.

ISOPHORONE
CAS: 78–59–1; Chemical Formula: $C_9H_{14}O$
H.S. No. 1221

The current OSHA limit for isophorone is 25 pm as an 8-hour TWA. The ACGIH has established a 5 ppm TLV as a ceiling limit, and NIOSH recommends a workplace standard of 4 ppm as a 10-hour TWA for isophorone. Isophorone is a colorless liquid at room temperature, and it has a comphor-like order.

Studies in animals and with human volunteers indicate that exposures to high concentrations of isophorone cause nephrotoxic and other adverse effects. A paper by Smyth, Seaton, and Fischer (1942) reported that guinea pigs and rats exposed to 550 ppm isophorone for 6 weeks demonstrated degenerative changes in the kidneys and liver. At an exposure level of 25 ppm, no adverse effects were noted, but at 50 ppm, the liver of one animal and the kidneys of four others were damaged. The entire group of 20 animals exposed at 50 ppm survived, but 2 of 16 animals died after this level was raised to 100 ppm (Smyth, Seaton, and Fischer 1942). Volunteers exposed for a few minutes to isophorone vapor at concentrations between 40 and 400 ppm experienced eye, nose, and throat irritation; several subjects exposed at the 200-ppm level developed headache, nausea, faintness, dizziness, and a feeling of suffocation (Smyth and Seaton 1940). Silverman, Schulte, and First (1946) reported that volunteers exposed to 25 ppm isophorone, the current OSHA PEL, complained of irritation of the eyes, nose, and throat. Another study conducted by the Western Electric Company (Ware, personal communication, 1973) reported that workers exposed for a one-month period to levels of 5 to 8 ppm isophorone demonstrated fatigue and malaise. When the workplace level was reduced to between 1 and 4 ppm, there were no complaints of adverse effects. The NIOSH Criteria Document for the ketones (1978) notes that all of the ketones are central nervous system depressants, and that workplace exposures to more than one ketone may produce additive effects.

OSHA is proposing to reduce the current 8-hour TWA PEL of 25 ppm to an 8-hour TWA of 4 ppm, to protect workers against the risk of fatigue, nausea, and headaches demonstrated to occur in exposed workers at levels between 5 and 8 ppm. The Agency preliminarily concludes that this limit will substantially reduce this occupational risk. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for isophorone if the Agency determines that this limit will substantially reduce significant risk.

METHYL CHLORIDE
CAS: 74–87–3; CHEMICAL FORMULA: CH_3Cl
H.S. No. 1254

OSHA's current limits for methyl chloride are 100 ppm as an 8-hour TWA, 200 ppm as a 15-minute ceiling, and 300 ppm as a 5-minute peak in any 3-hour period. The ACGIH has established a 50-ppm 8-hour TLV–TWA limit and a 100-ppm 15-minute STEL for this substance. NIOSH recommends the lowest feasible limit for methyl chloride and considers it a carcinogen. Methyl chloride is a colorless, sweet-smelling gas.

There is considerable evidence in humans and some in animals demonstrating that exposure to methyl chloride by inhalation or dermal absorption produces narcosis and other central nervous system effects, including respiratory failure and death (ACGIH 1986, p. 380). In animals, repeated exposures to 500 ppm or to higher concentrations can be life-threatening, but exposures to 300 ppm for 64 weeks caused no apparent effects (Smith and von Oettingen 1947).

Reports in the earlier literature described by Fairhall (1969) indicate that moderate (not further specified) exposure causes ocular symptoms that

may persist for weeks, while serious (not further specified) exposure has severe effects in the central nervous system. Patty (1963) states that serious exposure causes central nervous system, liver and kidney, and bone marrow effects, with symptoms of ataxia, staggering gait, weakness, tremors, vertigo, speaking difficulty, and blurred vision. Symptoms may be of several weeks' duration or may even be permanent (Patty 1963).

The Dow Chemical Company (as cited in ACGIH 1986, p. 380) studied the methyl chloride exposures of employees in 54 job classifications over a 4-month period. Exposures ranged from 5 to 78 ppm methyl chloride (8-hour TWA), averaged 30 ppm over the work shift, and occasionally included peaks as high as 440 ppm. Medical examination of these workers revealed no detectable effects of methyl chloride exposure. However, average 8-hour exposures in the range of 195 to 475 ppm caused symptoms of weakness, drowsiness, staggering gait, thickness of the tongue, and memory lapses in some of the employees (Dow Chemical Company, as cited in ACGIH 1986, p. 380).

In a study of six cases of industrial methyl chloride poisoning, workers chronically exposed to levels between 200 and 400 ppm developed neurotoxic symptoms after 2 or more weeks of exposure (Scharen-Weber, Spears, Cowles 1974). Symptoms included drowsiness, dizziness, mental confusion, misty vision, staggering gait, and slurred speech, and symptoms sometimes recurred after apparent recovery and in the absence of renewed exposure.

Repko and co-workers (1976) found that workers exposed to concentrations of methyl chloride ranging from 7.4 to 70 ppm but averaging 33.6 ppm displayed a significant performance decrement, and that exposures below 100 ppm produced significant but transitory changes in functional capacity.

OSHA is proposing an 8-hour TWA of 50 ppm and a 15-minute STEL of 100 ppm for methyl chloride. The Agency preliminarily concludes that these two limits together will protect workers from the risk of neurotoxic effects, including functional impairment, performance decrements, headaches, dizziness, slurred speech, and staggering gait associated with exposure to this substance at the levels permitted by OSHA's current PEL. The Agency believes that the proposed PEL and STEL will work together to reduce this risk substantially. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for methyl chloride if the Agency determines that this limit will substantially reduce significant risk.

METHYL CHLOROFORM (1,1,1-TRICHLOROETHANE)

CAS: 71-55-6; Chemical Formula: CH_3CCl_3
H.S. No. 1255

OSHA currently has an 8-hour TWA limit of 350 ppm for methyl chloroform. The ACGIH has established the same TWA limit as well as a TLV-STEL of 450 ppm. NIOSH recommends a 15-minute ceiling limit of 350 ppm. Methyl chloroform is a clear, nonflammable liquid.

The primary health concerns associated with exposure to methyl chloroform are anesthesia and cardiac sensitization. The oral toxicity of methyl chloroform is low, with LD_{50} values ranging from 5.7 to 12.3 g/kg for rats, mice, rabbits, and guinea pigs. This substance does, however, defat the skin on contact causing redness and scaling (Torkelson et al. 1958). Skin absorption is relatively insignificant; the acute percutaneous LD_{50} in rabbits is greater than 16 g/kg; slight, reversible irritation was observed from applications of 0.5 g/kg to rabbit skin for 90 days (Torkelson et al. 1958). Repeated expsoure of animals to concentrations between 1000 and 10,000 ppm for 3 months produced anesthesia and lung and liver damage in some species, but exposure to 500 ppm of methyl chloroform vapor for 7 hours daily, 5 days/week for 6 months caused no toxic changes in guinea pigs, rabbits, or monkeys (Torkelson et al. 1958). Other animal studies (Gehring 1968; Plaa, Evans, and Hine 1958; Rowe et al. 1963) have reported that methyl chloroform has low hepatotoxicity, but cardiac sensitization has occurred at high doses (5000–10,000 ppm) (Rennick et al. 1949; Trochimowicz et al. 1976). Tests in rats and mice for teratogenicity and carcinogenicity have demonstrated negative results (Schwetz et al. 1975; NIOSH 1976; Weisberger 1977).

In humans, it has been reported that anesthetic effects may begin to occur at methyl chloroform concentrations approaching 500 ppm (Stewart et al. 1969). Deaths from anesthesia and/or cardiac sensitization have been noted in employees working in confined areas (Patty 1963). Kramer and co-workers (1978) conducted an epidemiological study of men and women exposed for periods ranging from several months to 6 years to methyl chloroform at levels that occasionally exceeded 200 ppm; when compared to matched-pair controls, no adverse exposure-related effects were found (Kramer, Ott, Fulerson et al. 1978).

OSHA is proposing a PEL of 350 ppm TWA and a STEL of 450 ppm for methyl chloroform The Agency preliminarily concludes that this combined PEL–STEL limit will protect workers against the risk of anesthetic and cardiac-sensitizing effects potentially associated with exposure at the elevated short-term levels permitted by an 8-hour limit alone. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for methyl chloroform if the Agency determines that this limit will substantially reduce significant risk.

OCTANE

CAS: 111-65-9; Chemical Formula: $CH_3(CH_2)_6CH_3$
H.S. No. 1296

OSHA's current limit for octane is 500 ppm as an 8-hour TWA. The ACGIH has established a 300-ppm TWA and a 375-ppm STEL NIOSH (1977a) recommends a 75-ppm 10 hour TWA and a 385-ppm 15-minute ceiling limit.

Mice exposed to octane concentrations of 6600 to 13,700 ppm developed narcosis in 30 to 90 minutes (Fuhner 1921). Flury and Zernik (1931) believed the narcotic concentration in humans to be 5000 ppm; Patty and Yant (1929) placed the narcotic concentration at 8000 ppm. Based on this information, the ACGIH concluded that octane was 1.2 to 2 times more toxic than heptane, and recommended TLVs of 300 ppm TWA and 375 ppm STEL.

As discussed in more detail above for the other C_5–C_8 alkanes, the NIOSH (1977a) recommended limits are based on their finding that all C_5–C_8 alkanes present a neurotoxic hazard similar to n-hexane. The ACGIH disagrees with this conclusion, believing the neurotoxic properties of n-hexane to be unique among the alkane series.

Because octane is known to be more toxic than heptane, OSHA believes a lower limit is warranted to protect against the risk of narcosis. At this time, OSHA is proposing to revise its limits for octane to 300 ppm TWA and 375 ppm STEL. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for octane if the Agency determines that this limit will substantially reduce significant risk. OSHA also requests comment on the strength of the evidence that exposure to octane may present a more serious neuropathic hazard.

PENTANE

CAS: 109-66-0; Chemical Formula: C_5H_{12}
H.S. No. 1306

OSHA's current limit for pentane is 1000 ppm TWA. In 1976, the ACGIH adopted a 600-ppm TLV-TWA and a

750-ppm TLV–STEL. NIOSH (1977a) has recommended that workplace exposures to pentane not exceed 120 ppm as a 10-hour TWA and 610 ppm as a 15-minute short-term limit. Pentane is usually encountered in volatile petroleum fractions, some of which are used as solvents. Pure pentane is used as a blowing agent for plastics, in solvent extraction, and in ice manufacture.

Fairhall (1957) stated that narcosis and mucous membrane irritation were the only reported toxic effects resulting from exposure to pentane. The reported lethal concentration in humans is 130,000 ppm. (Flury and Zernik 1931; Swann et al. 1974). According to Patty and Yant (1929), humans exposed for 10 minutes to 5000 ppm did not complain of any adverse symptoms.

In a report by Gaultier et al. (1973), five cases of polyneuropathy occurred among employees exposed to a solvent containing 80 percent pentane, 14 percent heptane, and 5 percent hexane. Based largely on this report, NIOSH (1977a) recommended the same occupational limit for all C_5–C_8 alkanes as for the neuropathic agent n-hexane (350-mg/m³ TWA and 1800-mg/m³ 15-minute short-term limits; these limits are equal to about 120-ppm TWA and 610-ppm 15-minute short-term limits for pentane).

The ACGIH (1986) points out that the rationale used by NIOSH in setting a limit for pentane ignores the theory that n-hexane is uniquely neuropathic via metabolism to 2,5-hexanedione, which is the same metabolite that is formed during exposure to another neuropathic agent, methyl butyl ketone. The ACGIH (1986) established its limits of 600 ppm TWA and 750 ppm as a STEL to "provide a substantial margin of safety against narcotic and irritative effects," but did not rule out the possibility that "chronic exposure to high concentrations may lead to neuropathy" (ACGIH 1986). The ACGIH believed that the manifestation of such effects would require heavy exposures to pentane and concluded that its recommended limits would be adequately protective. At this time, OSHA accepts the evaluation that all C_5–C_8 alkanes are not equally toxic because a metabolite of n-hexane exhibits unique neurotoxic properties. The Gaultier study does not provide specific isomer exposure data supporting the RELs of 120 ppm (TWA) and 610 ppm (STEL). The Agency therefore proposes a TWA of 600 ppm and a STEL of 750 ppm as the permissible exposure limits for pentane. Because of the disagreement regarding the subject of equal C_5–C_8 alkane toxicity, OSHA is specifically requesting comments on this scientific question.

2-PENTANONE (METHYL PROPYL KETONE)

CAS: 107-87-9; Chemical Formula: $CH_3COC_3H_7$

H.S. No. 1307

The current OSHA limit for 2-pentanone is 200 ppm as an 8-hour TWA. ACGIH recommends a 200-ppm TLV–TWA and a 250-ppm TLV–STEL. NIOSH (1978g) has recommended a 150-ppm limit as a 10-hour TWA.

Both the ACGIH- and NIOSH-recommended limits are based on a study by Specht et al. (1940), who found that guinea pigs exhibited irritation and weakness on exposure to 2500 ppm, and that exposure to 5000 ppm produced narcosis and coma. The authors concluded that 2-pentanone was considerably less toxic than methyl butyl ketone but is more toxic than methyl ethyl ketone, and is likely to be more irritating than methyl ethyl ketone or acetone. The ACGIH-recommended limits are apparently based on a judgment that the 200-ppm TLV–TWA and 250-ppm TLV–STEL are low enough to prevent narcosis and irritation.

NIOSH (1978) applied the findings of Specht et al. (1940) to the findings of Nelson et al. (1943), who reported that volunteers complained of slight irritation on exposure to 100 ppm methyl ethyl ketone. Because 2-pentanone was believed by Specht et al. (1940) to be at least as irritating as methyl ethyl ketone, NIOSH (1978) believed that a "slight reduction" in the OSHA standard was warranted. Therefore, NIOSH recommended a 150-ppm limit for 2-pentanone.

Both the ACGIH and NIOSH limits are designed to reduce the acute health effects observed at 100 ppm. While the 150-ppm REL appears to be more protective, OSHA has preliminarily concluded that the combination of a 200-ppm TWA and a 250-ppm STEL is more protective and is necessary to prevent the adverse health effects associated with exposure to this chemical. OSHA therefore proposes these limits for adoption as the PEL to reduce the risks associated with exposures at elevated short-term levels. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for 2-pentanone if the Agency determines that this limit will substantially reduce significant risk.

PERCHLOROETHYLENE (TETRACHLOROETHYLENE)

CAS: 127-18-4; Chemical Formula: $CCl_2=CCl_2$

H.S. No. 1308

OSHA's current limits for perchloroethylene are 100 ppm as an 8-hour TWA, 200 ppm as a 15-minute ceiling, and 300 ppm as a 5-minute peak not to be exceeded in any 3-hour period. The ACGIH has established an 8-hour TWA of 50 ppm and a 15-minute STEL of 200 ppm for perchloroethylene. NIOSH recommends maintaining exposures at the lowest feasible limit and classifies this chemical as a carcinogen. Perchloroethylene is a clear, colorless liquid with an ether-like odor.

The oral LD_{50} for mice is 8.85 mg/kg, and the lethal air concentration for this species is 6000 ppm for 4 hours (Handbook of Toxicology 1956). Rats were exposed 8 hours/day, 5 days/week for as long as 17 months to 70, 230, or 470 ppm (Carpenter 1937). At 70 ppm, all animals survived and no pathology was noted. Animals also survived the higher exposures, but changes were seen at postmortem in the kidneys and livers of these rats. Rats, guinea pigs, cats, rabbits, and monkeys were exposed to varying concentrations of perchloroethylene for 7 hours/day, 5 days/week for various durations (Rowe et al. 1952). Rats exposed at 1600 ppm developed drowsiness and depression after 1 week and liver and kidney changes after 4 weeks. Guinea pigs exposed to 400 ppm for 130 exposures developed heavier kidneys and livers and fatty degeneration of the liver. Rats, rabbits, and monkeys exposed for 130 7-hour exposures to 400 ppm showed no effects.

Mice exposed in another study to 200 ppm 4 hours/day for 1, 2, 4, or 8 weeks developed fatty degeneration of the liver (Kylin, Sumegi, and Yllner 1965). Exposure of pregnant rats to 300 ppm perchloroethylene on days 6 through 15 of gestation did not cause teratological effects but did produce developmental toxicity (Schuetz, Leong, and Gerhing 1975). This substance does not appear to be mutagenic (ACGIH 1986, p. 464).

In an NCI gavage study for carcinogenicity, perchloroethylene proved carcinogenic to the livers of mice but not to those of rats (NCI 1977). Application of perchloroethylene to the skin of rats and mice, with and without a promoter, did not induce cancer (Thiess et al. 1977).

In humans being treated for worms, perchloroethylene doses of 2.8 or 4 ml caused narcosis, exhilaration, and signs of inebriation; at doses of 1 to 8 ml, no changes were seen in liver function tests (Lambert 1933; Fernando et al. 1939). A worker exposed to an estimated concentration of 1470 ppm perchloroethylene and Stoddard solvent for 3.5 hours lost consciousness (Stewart

et al. 1961). Studies have shown that exposure to perchloroethylene at 2000 ppm for 1.5 minutes caused unconsciousness; exposure at 500 ppm for 50 minutes caused increased salivation, metallic taste, eye irritation, and other effects; exposure at 216 ppm for 2 hours caused eye burning, light-headedness, motor incoordination, and other effects (Carpenter 1973). A 4-hour exposure to 100 ppm caused eye irritation (Stewart et al. 1974).

Chronic exposure to 200 ppm perchloroethylene caused early signs of central nervous system depression, but exposure to 100 ppm on a regular basis apparently had no adverse effect (Stewart et al. 1974, 1977). Direct contact with the liquid causes erythema, skin burns, and dermal absorption (Morgan 1969; Gold 1969; Stewart and Dodd 1964).

Two epidemiological studies of occupationally exposed perchloroethylene workers have failed to show increases in the incidence of liver tumors among these workers (Blair, Decoufle, and Grauman 1979; Kaplan 1980, as cited in ACGIH 1986, p. 464).

OSHA is proposing an 8-hour TWA of 50 ppm and a 15-minute STEL of 200 ppm for perchloroethylene. The Agency preliminarily concludes that these limits, taken together, will protect workers against the risk of eye irritation, headaches, and other effects associated with exposure to perchloroethylene. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for perchloroethylene if the Agency determines that this limit will substantially reduce significant risk.

STODDARD SOLVENT
CAS: 8052–41–3; Chemical Formula: C_9H_{20}
H.S. No. 1371

OSHA's current limit for stoddard solvent is 500 ppm as an 8-hour TWA. The ACGIH has established a TLV-TWA of 100 ppm. NIOSH (1977h) recommends limits of 350 mg/m³ as a 10-hour TWA and 1800 mg/m³ as a 15-minute ceiling for all refined petroleum solvents; these limits correspond approximately to a 60 ppm TWA and 310 ppm STEL, respectively. Stoddard solvent is a refined petroleum solvent having a flash point in the range of 102 to 100 °F and containing 65 percent or more C_{10} and higher molecular weight hydrocarbons.

The current OSHA limit of 500 ppm (equivalent to the 1968 ACGIH TLV) was based on the toxicities of the major components of stoddard solvent (i.e., 80 to 85 percent nonane and isodecane and 15 to 20 percent trimethyl benezene); specifically, the recommended limit was calculated from the TLVs established for pentane and trimethyl benzene. The revised ACGIH limit of 100 ppm thus reflects a reduction in the TLVs for pentane and trimethyl benzene. The ACGIH (1986) noted that a report by Carpenter et al. (1978), who found slight kidney damage among rats exposed to 330 ppm for 65 days, provided additional evidence that the earlier limit was too high.

The NIOSH limit of 350 mg/m³ TWA and 1800 mg/m³ is derived from its recommended limits for C_5–C_8 alkanes, which are designed to protect against neuropathic effects. NIOSH recommended the same limit for stoddard solvent because of the lack of data on chronic effects and because of a report of polyneuropathy occurring among workers exposed to jet fuels containing mixtures of kerosene and gasoline. NIOSH reasoned that although C_5–C_8 alkanes present in jet fuel may have been responsible, it was possible that the heavier hydrocarbon components may also have been responsible. Thus, the recommended limits for stoddard solvent reflect a concern that higher molecular weight hydrocarbons may be neuropathic.

The NIOSH RELs for stoddard solvent are based on the hypothesis of equivalent neuropathic toxicity for all of the C_5–C_8 alkanes. OSHA has tentatively rejected this approach. OSHA therefore proposes that the 100 ppm TWA be adopted as the new PEL to reduce the risk; this limit is based on information developed for analogous solvents. The Agency requests comments on this issue, as discussed above.

TOLUENE
CAS: 108–88–3; Chemical Formula: $C_6H_5CH_3$
H.S. No. 1397

The current OSHA standard for toluene is 200 ppm as an 8-hour TWA limit, with a 300 ppm ceiling and a 500 ppm peak for a maximum of 10 minutes in 8 hours. The ACGIH has established an exposure limit for toluene at 100 ppm as an 8-hour TWA and 150 ppm as a 15-minute STEL. NIOSH recommends a 100 ppm 8-hour TWA and a 10-minute ceiling of 200 ppm. Toluene is a flammable, colorless liquid with an aromatic hydrocarbon odor.

The acute toxicity of toluene in animals is greater than that of benzene. Patty (1963) reports that the lethal doses of toluene and benzene in mice are 10,000 and 14,000 ppm, respectively. The oral LD_{50} in rats is 7.53 ml/kg (Smyth et al. 1969). Exposure of rats to 2500 or 5000 ppm of toluene caused a temporary decrease in white cell count but no evidence of damage to the blood-forming organs or the liver. Fairhall (1957) stated that severe toluene exposure can cause a marked drop in the red blood cell count and partial destruction of the blood-forming elements of the bone marrow, but other researchers report that numerous animal studies indicate that toluene is not a bone marrow toxin (Gerarde 1960). A study by Greenberg, Mayers, Heinmann, and Moskowitz (1942) reported that painters exposed to toluene levels of 100 to 1100 ppm exhibited enlarged livers, a moderate decrease in red blood cell counts, enlarged red blood cells, and absolute lymphocytosis, but no leukopenia. Wilson (1943) observed workers exposed to toluene at varying levels up to 1500 ppm. At levels less than 200 ppm, signs of headache, fatigue, and nausea were present. Those workers exposed to 200 to 500 ppm toluene experienced temporary amnesia, lack of coordination, and anorexia. Levels of exposure from 500 to 1500 ppm resulted in a marked loss of coordination, diminished reaction time, pronounced weakness, and heart palpitations. Red cell counts were also decreased, and two cases of aplastic anemia required lengthy hospital treatment; however, the author noted that he could not rule out the possibility that benzene contamination of the toluene was the cause of these effects. Incidences of aplastic anemia (one fatal) have been noted in six glue sniffers; toluene was the base solvent in the glue (Powers 1965). A man who had inhaled toluene regularly at unspecified levels for 14 years developed permanent encephalopathy (Knox and Nelson 1966). Von Oettingen, Neal, Donahue, et al. (1942) found that exposures of 200 ppm for an unspecified duration caused slight changes in muscle coordination in human volunteers. Later studies by Ogata, Tomokuni and Takatsuka (1970) showed an increase in reaction time, and a decrease in pulse rate, and a decrease in systolic blood pressure at exposures to 200 ppm for 7 hours.

OSHA is proposing an 8-hour TWA PEL of 100 ppm and a STEL of 150 ppm for toluene. The Agency preliminarily concludes that studies clearly indicate that a risk of hepatotoxicity, hematopoietic, and nervous system effects exists at levels substantially below the current 200-ppm 8-hour TWA PEL. OSHA believes that the new limits will protect exposed workers from the risk of these serious health effects, which have been demonstrated to occur even as a result of less than full-shift exposures.

TRICHLOROETHYLENE)
CAS: 79-01-6; Chemical Formula:
$CCl_2=CHCl$
H.S. No. 1406

OSHA's current limit for trichloroethylene, adopted from ANSI, is 100 ppm TWA, 200 ppm as a ceiling not to be exceeded for more than 5 minutes every 2 hours, and 300 ppm as a peak limit. The ACGIH has recommended a 50-ppm TLV-TWA and a 200-ppm TLV-STEL. NIOSH (1978h) considers trichloroethylene to be a potential human carcinogen and has recommended a 25-pm 10-hour TWA.

The ACGIH (1986) cited several studies establishing that trichloroethylene primarily affects the central nervous system and liver; some of these studies have indicated that chronic exposure to less than 100 ppm trichloroethylene is associated with a variety of nervous disturbances. Haas (1960) and Grandjean (1955) reported nervous symptoms among workers exposed for 5 years or more to concentrations ranging from 1 to 335 ppm; the frequency of complaints increased when average exposures exceeded 40 ppm. Bardodej and Vyskocil (1956) also reported symptoms of trichloroethylene poisoning, including tremors, giddiness, anxiety, and alcohol intolerance, among workers exposed above 40 ppm. In contrast, controlled laboratory experiments with human subjects exposed for up to several days to 100 or 200 ppm have generally reported no behavioral or subjective responses. The ACGIH concluded that although the symptoms reported by workers are subjective and commonly found among individuals with no chemical exposure, the consistency of the reports "suggests the possibility of some subjective complaints as concentrations exceed about 50 ppm" (ACGIH 1986). Therefore, the ACGIH recommended a TLV-TWA of 50 ppm and a TLV-STEL of 200 ppm to minimize complaints of headache, fatigue, and irritability.

The ACGIH (1986) also reviewed some of the carcinogenicity data on trichloroethylene. In an NCI bioassay (1976b), mice given trichloroethylene by gavage developed hepatocellular carcinomas, but rats did not. The species difference in response was attributed to a difference in the way trichloroethylene is metabolized between the mouse and rat (Stott et al. 1982). An inhalation study on mice, rats, and Syrian hamsters (Henschler et al. 1980) found only an increase in the occurrence of malignant lymphomas in mice, which the authors believed were peculiar to the strain of mouse used (NMRI). The ACGIH also cited a number of epidemiologic investigations of cohorts as large as 7,688 workers, in which no correlation between cancer mortality and exposure to trichloroethylene was found (Novotna et al. 1971; Axelson et al. 1978; Tola et al. 1980).

After reviewing some of the same data, NIOSH (1978h) concluded that the results of the NCI gavage study indicate trichloroethylene (TCE) to be a potential human carcinogen, although it was "not considered to be a potent carcinogen." In addition, NIOSH concluded that the current 100 ppm limit would not sufficiently protect against neuropathic symptoms caused by exposure to trichloroethylene. NIOSH's recommended limit of 25 ppm was based on an evaluation of several industrial hygiene reports showing that many degreasing operations, including those using open-top tanks, are maintaining exposures at less than 50 ppm TWA. NIOSH believed that a 25-ppm TWA level could be uniformly achieved by the use of engineering control technology.

Since publication of the 1978 NIOSH report, several recent bioassays on trichloroethylene have been published and are currently being reviewed by EPA. Fukuda et al. (1983) exposed female rats and mice to 50, 150, or 450 ppm trichloroethylene for 103 weeks and reported an increase incidence of lung tumors among mice only. Maltoni et al. (1986) exposed rats and mice to 100, 300, or 600 ppm trichloroethylene and reported a significant increase of renal adenocarcinomas and Leydig cell tumors in rats, and a signfiant increase in hepatomas and lung tumors in mice. In 1986, the NIP reported an increase in the incidence of kidney tumors in rats given trichloroethylene by gavage; however, the NTP considered the response to be weak (3 of 49 animals) and reported that the results were only statistically significant after corrections for high mortality were made.

The 50-ppm TWA and 200-ppm STEL established by the ACGIH were established to minimize subjective complaints of headache, fatigue, and irritability. However, NIOSH has concluded that TCE is a potential human carcinogen, although not a "potent" one. NIOSH recommended a REL of 25 ppm TWA because the current PEL does not sufficiently protect against nervous system effects. NIOSH believes this REL is feasible and can be achieved by engineering control technology. In light of the uncertainty of the carcinogenicity issue, OSHA proposes adoption of a 25-ppm TWA REL to substantially reduce occupational risk. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for trichloroethylene if the Agency determines that this limit will substantially reduce significant risk.

Preliminary Conclusions

OSHA preliminarily concludes that workers exposed to these narcosis-causing substances are at risk of loss of consciousness, uncoordinated movements, inability to concentrate, and drowsiness; these highly undesirable and serious health effects may additionally have the potential to cause serious workplace accidents and injuries. The new or reduced exposure limits being proposed by OSHA are intended to protect employees from experiencing these risks in their places of work and will create a substantial reduction in such risks. The health evidence for these substances forms a reasonable basis for proposing the revision or addition of limits at the proposed levels. At the time of the final rule, OSHA will establish limits for these narcotic substances if the Agency determines that these limits will reduce significant risk.

3. Substances for Which Proposed Limits Are Based on Avoidance of Sensory Irritation

Introduction

Exposure to many chemical agents is associated with the development of sensory irritation, which is initiated when these substances come into contact with mucous membranes or skin. Limits have been set for a large group of chemicals on the basis of their sensory irritant effects. These substances, which number 79, are shown in Table C3-1, along with their current OSHA limits, CAS numbers, NIOSH RELs, ACGIH TLVs, and OSHA HS numbers. For six of these chemicals, OSHA is proposing to reduce the 8-hour TWA, and for an additional nine, the Agency proposes both to reduce the 8-hour limit and to add a STEL. In 22 cases, the 8-hour limit would remain unchanged but a STEL would be added. In six instances, a ceiling is proposed for deletion, and this limit would be replaced by an 8-hour TWA and/or STEL limits. Thirty of these chemicals were previously unregulated by OSHA, and for these, OSHA is proposing 8-hour

limits, 8-hour limits supplemented by a STEL, or ceiling limits. For five substances, OSHA is proposing to replace an existing TWA limit with a ceiling limit. For 15 of these substances, the NIOSH and ACGIH limits are different (see Table C3–1). OSHA is proposing the NIOSH REL for three substances.

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TABLE C3-1. Substances for Which Limits Are Based on Avoidance of Irritant Effects

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1001 Acetaldehyde	75-07-0	200 ppm TWA	100 ppm TWA 150 ppm STEL	--
1002 Acetic acid	64-19-7	10 ppm TWA	10 ppm TWA 15 ppm STEL	--
1004 Acetone+	67-64-1	1000 ppm TWA	750 ppm TWA 1000 ppm STEL	250 ppm TWA
1007 Acrolein	107-02-8	0.1 ppm TWA	0.1 ppm TWA 0.3 ppm STEL	--
1010 Allyl alcohol	107-18-6	2 ppm TWA, Skin	2 ppm TWA, Skin 4 ppm STEL	--
1012 Allyl glycidyl ether	106-92-3	10 ppm Ceiling	5 ppm TWA, Skin 10 ppm STEL	9.6 ppm Ceiling (15 min)
1013 Allyl propyl disulfide	2179-59-1	2 ppm TWA	2 ppm TWA 3 ppm STEL	--
1021 Ammonia	7664-41-7	50 ppm TWA	25 ppm TWA 35 ppm STEL	50 ppm Ceiling (5 min)
1022 Ammonium chloride fume	12125-02-9	--	10 mg/m^3 TWA 20 mg/m^3 STEL	--

TABLE C3-1. Substances for Which Limits Are Based on Avoidance of Irritant Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1036 Borates, tetra, Na (anhydrous)	1303-96-4	—	1 mg/m^3 TWA	—
1037 Borates, tetra, Na (decahydrate)	1303-96-4	—	5 mg/m^3 TWA	—
1038 Borates, tetra, Na (pentahydrate)	1303-96-4	—	1 mg/m^3 TWA	—
1042 Bromine	7726-95-6	0.1 ppm TWA	0.1 ppm TWA 0.3 ppm STEL	—
1045 2-Butanone (MEK)	78-93-3	200 ppm TWA	200 ppm TWA 300 ppm STEL	200 ppm TWA
1047 n-Butyl acetate	123-86-4	150 ppm TWA	150 ppm TWA 200 ppm STEL	—
1053 n-Butyl lactate	138-22-7	—	5 ppm TWA	—
1054 n-Butyl mercaptan	109-79-5	10 ppm TWA	0.5 ppm TWA	0.5 ppm Ceiling (15 min)
1063 Camphor	76-22-2	2 ppm TWA	2 ppm TWA 3 ppm STEL	—

TABLE C3-1. Substances for Which Limits Are Based on Avoidance of Irritant Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1064 Caprolactam (dust)	105-60-2	—	1 mg/m^3 TWA 3 mg/m^3 STEL	—
1065 Caprolactam (vapor)	105-60-2	—	20 mg/m^3 TWA 40 mg/m^3 STEL	—
1077 Cesium hydroxide	21351-79-1	—	2 mg/m^3 TWA	—
1079 Chlorine+	7782-50-5	1 ppm Ceiling	1 ppm TWA 3 ppm STEL	0.5 ppm Ceiling (15 min)
1083 Chloroacetyl chloride	79-04-9	—	0.05 ppm TWA	—
1084 o-Chlorobenzylidene malononitrile	2698-41-1	0.05 ppm TWA	0.05 ppm Ceiling, Skin	—
1105 Cyanogen	460-19-5	—	10 ppm TWA	—
1106 Cyanogen chloride	506-77-4	—	0.3 ppm Ceiling	—
1119 Dibutyl phosphate	107-66-4	1 ppm TWA	1 ppm TWA 2 ppm STEL	—
1122 1,3-Dichloro-5,5-dimethylhydantoin	118-52-5	0.2 mg/m^3 TWA	0.2 mg/m^3 TWA 0.4 mg/m^3 STEL	—

TABLE C3-1. Substances for Which Limits Are Based on Avoidance of Irritant Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1127 Dichloroethyl ether	111-44-4	15 ppm Ceiling, Skin	5 ppm TWA 10 ppm STEL, Skin	—
1130 2,2-Dichloropropionic acid	75-99-0	—	1 ppm TWA	—
1137 Diethylamine	109-89-7	25 ppm TWA	10 ppm TWA 25 ppm STEL	—
1140 Diisobutyl ketone	108-83-8	50 ppm TWA	25 ppm TWA	25 ppm TWA
1158 Epichlorohydrin	106-89-8	5 ppm TWA, Skin	2 ppm TWA, Skin	—
1162 Ethyl benzene	100-41-4	100 ppm TWA	100 ppm TWA 125 ppm STEL	—
1164 Ethyl ether	60-29-7	400 ppm TWA	400 ppm TWA 500 ppm STEL	—
1165 Ethyl mercaptan	75-08-1	10 ppm Ceiling	0.5 ppm TWA	0.5 ppm Ceiling (15 min)
1169 Ethylene glycol	107-21-1	—	50 ppm Ceiling	—
1171 Ethylidene norbornene	16219-75-3	—	5 ppm Ceiling	—

TABLE C3-1. Substances for Which Limits Are Based on Avoidance of Irritant Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1183 Furfural	98-01-1	5 ppm TWA, Skin	2 ppm TWA, Skin	—
1184 Furfuryl alcohol	98-00-0	50 ppm TWA	10 ppm TWA 15 ppm STEL, Skin	50 ppm TWA
1187 Glutaraldehyde	111-30-8	—	0.2 ppm Ceiling	—
1196 Hexachlorocyclo-pentadiene	77-47-4	—	0.01 ppm TWA	—
1204 Hexylene glycol	107-41-5	—	25 ppm Ceiling	—
1206 Hydrogen bromide	10035-10-6	3 ppm TWA	3 ppm Ceiling	—
1208 Hydrogen fluoride+	7664-39-3	3 ppm TWA	3 ppm Ceiling	3 ppm TWA 6 ppm Ceiling (15 min)
1211 2-Hydroxypropyl acrylate	999-61-1	—	0.5 ppm TWA, Skin	
1217 Iron salts (soluble)	Varies with compound	—	1 mg/m^3 TWA	—
1224 Isopropyl acetate	108-21-4	250 ppm TWA	250 ppm TWA 310 ppm STEL	—

TABLE C3-1. Substances for Which Limits Are Based on Avoidance of Irritant Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1225 Isopropyl alcohol	67-63-0	400 ppm TWA	400 ppm TWA 500 ppm STEL	400 ppm TWA 800 ppm Ceiling (15 min)
1228 n-Isopropylamine	75-31-0	5 ppm TWA	5 ppm TWA 10 ppm STEL	—
1243 Mesityl oxide	141-79-7	25 ppm TWA	15 ppm TWA 25 ppm STEL	10 ppm TWA
1248 Methyl 2-cyano-acrylate	137-05-3	—	2 ppm TWA 4 ppm STEL	—
1261 Methyl isobutyl carbinol	105-30-6	25 ppm TWA, Skin	25 ppm TWA 40 ppm STEL, Skin	—
1263 Methyl mercaptan	74-93-1	10 ppm Ceiling	0.5 ppm TWA	0.5 ppm Ceiling (15 min)
1264 Methyl n-amyl ketone++	110-43-0	100 ppm TWA	50 ppm TWA	100 ppm TWA
1267 alpha-Methyl styrene	98-83-9	100 ppm Ceiling	50 ppm TWA 100 ppm STEL	—
1270 o-Methylcyclo-hexanone	583-60-80	100 ppm TWA, Skin	50 ppm TWA 75 ppm STEL, Skin	—

TABLE C3-1. Substances for Which Limits Are Based on Avoidance of Irritant Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1298 Osmium tetroxide	20816-12-0	0.002 mg/m^3 TWA	0.002 mg/m^3 TWA 0.006 mg/m^3 STEL	—
1302 Paraffin wax fume	8002-74-2	—	2 mg/m^3 TWA	—
1322 Phosphoric acid	7664-38-2	1 mg/m^3 TWA	1 mg/m^3 TWA 3 mg/m^3 STEL	—
1325 Phosphorous trichloride	7719-12-1	0.5 ppm TWA	0.2 ppm TWA 0.5 ppm STEL	—
1334 Potassium hydroxide	1310-58-3	—	2 mg/m^3 Ceiling	—
1350 Rosin core solder	—	—	0.1 mg/m^3 TWA	—
1365 Sodium bisulfite	7631-90-5	—	5 mg/m^3 TWA	—
1367 Sodium hydroxide	1310-73-2	2 mg/m^3 TWA	2 mg/m^3 Ceiling	2 mg/m^3 Ceiling (15 min)
1368 Sodium metabisulfite	7681-57-4	—	5 mg/m^3 TWA	—
1376 Sulfur monochloride	10025-67-9	1 ppm TWA	1 ppm Ceiling	—
1377 Sulfur pentafluoride	5714-22-7	0.025 ppm TWA	0.01 ppm Ceiling	—

TABLE C3-1. Substances for Which Limits Are Based on Avoidance of Irritant Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1387 Tetrahydrofuran	109-99-9	200 ppm TWA	200 ppm TWA 250 ppm STEL	—
1389 Tetrasodium pyrophosphate	7722-88-5	—	5 mg/m^3 TWA	—
1392 Thioglycolic acid	68-11-1	—	1 ppm TWA, Skin	—
1405 1,2,4-Trichloro-benzene	120-82-1	—	5 ppm Ceiling	—
1408 Triethylamine	121-44-8	25 ppm TWA	10 ppm TWA 15 ppm STEL	—
1421 Vanadium (V_2O_5, dust)	7440-62-2	0.5 mg/m^3 Ceiling	0.05 mg/m^3 TWA	0.05 mg/m^3 Ceiling (15 min)
1422 Vanadium (V_2O_5, fume)	7440-62-2	0.1 mg/m^3 Ceiling	0.05 mg/m^3 TWA	0.05 mg/m^3 Ceiling (15 min)
1424 Vinyl acetate	108-05-4	—	10 ppm TWA 20 ppm STEL	4 ppm Ceiling (15 min)
1429 VM & P Naphtha	8032-32-4	—	300 ppm TWA	75 ppm TWA 400 ppm Ceiling (15 min)

TABLE C3-1. Substances for Which Limits Are Based on Avoidance of Irritant Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1431 Xylene	1330-20-7	100 ppm TWA	100 ppm TWA 150 ppm STEL	100 ppm TWA 200 ppm Ceiling (10 min)
1435 Zinc chloride fume	7646-85-7	1 mg/m^3 TWA	1 mg/m^3 TWA 2 mg/m^3 STEL	—

* OSHA's TWA limits are for 8-hour exposures; its STELs are for the durations specified; and its ceilings are peaks not to be exceeded for any period of time.

** The ACGIH TWA-TLV is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

*** NIOSH TWA limits are for 10-hour exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

\+ Proposed limit is the NIOSH REL.

++ OSHA's current limit is retained.

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Description of the Health Effects

Irritant effects are readily perceived by affected individuals. The symptoms of sensory irritation include stinging, itching, and burning of the eyes, tearing (or lacrimation), a burning sensation in the nasal passages, rhinitis (nasal inflammation), cough, sputum production, chest pain, wheezing, and dyspnea (breathing difficulty). In the majority of cases, the onset of symptoms occurs rapidly upon exposure to the irritant; it is therefore easy to associate the causative agent with the irritant effect.

These effects cause severe discomfort and may be seriously disabling, as is the case with dyspnea or wheezing. The tearing and eye irritation associated with exposure to sensory irritants is often severe and can be as disabling as the weeping caused by exposure to tear gas. In addition to these primary effects, workers distracted by irritant effects are considerably more likely than non-exposed workers to have accidents and thus to endanger both themselves and others. (These adverse health effects also clearly have substantial productivity impacts.)

The eye irritation caused by exposure to irritants is believed to result from stimulation of the sensory nerve endings in the cornea. There is little information available on the relationship between the severity of the effect and the physical or chemical properties of the irritating substance. In addition, the mechanism of action underlying this irritant effect is not well understood. Mechanisms that have been suggested include physical action of the irritant on nerve endings, binding of the irritant to sulfhydryl groups of protein, inhibition of cellular respiration, and cholinesterase inhibition (Grant 1986). The symptoms of eye irritation are usually transient and do not generally persist after cessation of exposure; however, exposure to concentrations of lacrimators that exceed the levels associated with transient eye irritation may produce corneal or conjunctival injury that requires medical treatment (Grant 1986).

Sensory irritation of the pulmonary system primarily affects the upper respiratory tract and causes an increase in sputum production; inflammation of the nasal passages, trachea, and upper bronchial tree; and decreased cilial clearance. These effects produce a burning sensation in the nasal passages and throat; coughing; sneezing; and acute bronchitis. The development of bronchitis indicates that the cilial clearance mechanism has been compromised and the resulting mucus retention increases the risk of secondary bacterial infection. Wheezing may also be apparent, particularly if the affected individual has a history of hyper-reactive airways disease. If exposure is sufficiently intense, the irritant may reach the lower portion of the bronchial tree, causing a chemical burn of the parenchyma and the sudden collection of fluid in interstitial spaces and alveoli (pulmonary edema). Irritation-induced edema may have a delayed onset (12 hours or more) and can cause hypoxia and difficulty in breathing.

For the great majority of substances in this group, current limits are derived from human evidence that exposure to the chemical agent at a particular airborne concentration will be associated with sensory irritation. For a few substances in this group, animal evidence provided the basis for limit setting. Several general types of evidence may be used to lower existing limits:

- Consideration of new human evidence;
- Reinterpretation of human data that formed the basis for setting the 1968 TLV;
- Consideration of evidence from industrial experience showing that employees are not experiencing irritation; and
- Evaluation of new animal evidence.

The studies that provide the basis for the sensory irritant levels being proposed by OSHA are generally controlled-exposure experiments using human volunteers or reports of employee complaints arising in industrial settings. Almost all of these studies report either a NOE level for irritation (described either as the "complaint level" or the level that can be "tolerated" for 8 hours) or an exposure level below which there have been no reported complaints.

Dose-Response Relationships and Sensory Irritation

The onset of sensory irritation is considered a "threshold" or NOE level; that is, for any sensory irritant, there is an exposure level below which very few if any individuals will experience sensory irritation. As exposure increases above this level, a larger proportion of exposed individuals will notice the effect and the effect will become increasingly severe. At some level above this NOEL, all exposed persons will experience sensory irritation, although the intensity of the response may vary.

The risk of experiencing irritation that is associated with exposures below the NOEL will be minimal (except in the hypersensitive individual), while the risk of experiencing the irritant effect will increase directly as exposure increases. At some point above the NOE level, i.e., at some dose of the substance, the response will be 100 percent, and all exposed persons will experience irritation. According to general toxicologic principles, the shape of the curve that describes responses above the NOEL is sigmoidal, and the steepness of the curve is a function of the variability in individual responses to the particular irritant. For example, if nearly all persons exposed to the substance will experience a response at approximately the same concentration (dose), the curve will be steep; if, on the other hand, the percentage of people responding increases only slowly as concentration rises, the curve will be considerably flatter.

Analyses of the toxicologic data for the the substances in this group of chemicals follow. The following paragraphs describe OSHA's preliminary findings for the substances in this group of sensory irritants.

ACETALDEHYDE
CAS: 75-07-0; Chemical Formula: CH_3CHO
H.S. No. 1001

OSHA's current PEL for acetaldehyde is 200 ppm as an 8-hour TWA. The 200-ppm 1968 TLV established by the ACGIH for acetaldehyde was based on a sensory irritation study conducted by Silverman et al. (1946) that showed that unacclimatized individuals experienced eye irritation at 50 ppm, but that a level of 200 ppm was tolerable for an 8-hour day. The ACGIH has subsequently lowered its limit for acetaldehyde to 100 ppm as an 8-hour TWA and supplemented this with a STEL of 150 ppm. NIOSH has no REL for this substance.

Reexamination of the data reported by Silverman et al. (1946) reveals that, at 200 ppm of acetaldehyde, all exposed persons experienced inflammation of the conjunctivae of the eyes, which manifested as redness. OSHA therefore preliminarily concludes that the current PEL of 200 ppm places exposed employees at risk of conjunctivitis and other irritation and that a reduction to 100 ppm is necessary to reduce this risk. OSHA also finds that a STEL is necessary to supplement the 8-hour limit, because without a STEL, workers could be exposed at levels many times those that have been shown to cause corneal injury, sensitization, and respiratory tract irritation. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for acetaldehyde if

the Agency determines that this limit will substantially reduce significant risk.

ACETIC ACID
CAS: 64–19–17; Chemical Formula: CH_3COOH
H.S. No. 1002

The current OSHA PEL for acetic acid is a 10-ppm 8-hour TWA. The ACGIH recommends a TLV–TWA of 10 ppm and a TLV–STEL of 15 ppm. Acetic acid is a clear, colorless, flammable liquid with a pungent odor.

The 10-ppm TWA was established on the basis of work indicating that industrial exposure to acetic acid at 10 ppm was non-irritating (Sterner 1943). Patty (1949) reported that exposures to 800 to 1200 ppm cannot be tolerated by humans for longer than three minutes. One of six rats exposed to 16,000 ppm died (Smyth 1956), and guinea pigs exhibited minor changes in respiration after exposure at 5 ppm, with more pronounced effects at 100 ppm (Amdur 1961).

In humans, conjunctival irritation has been reported for exposures below 10 ppm (duration not specified) (Baldi 1963), and workers exposed to concentrations of 60 ppm during the workshift, plus one hour daily at 100 to 260 ppm, for 7 to 12 years developed respiratory irritation, conjunctivitis, bronchitis, pharyngitis, and erosion of exposed teeth (Parmeggiani and Sassi 1954). Vigliana and Zurlo (1956) observed respiratory, gastrointestinal, and skin irritation in the same group of workers.

To protect against these irritant effects, which were associated with short-term exposures, OSHA is proposing to supplement the existing 10-ppm 8-hour TWA with a STEL of 15 ppm.

The Agency preliminarily concludes that the combined TWA–STEL is necessary to protect exposed workers against the risk of respiratory, gastrointestinal, and skin irritation associated with industrial exposures to acetic acid. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for acetic acid if the Agency determines that this limit will substantially reduce significant risk.

ACETONE
CAS: 67–64–1; Chemical Formula: CH_3COCH_3
H.S. No. 1004

OSHA's current Z table limit for acetone is 1000 ppm as an 8-hour TWA. The ACGIH recommends a 750-ppm TLV and a 1000-ppm STEL for this substance, while the NIOSH REL is 250 ppm TWA.

The 1000-ppm limit for acetone was established primarily on the basis of information that indicated that workers experienced irritation at exposure levels ranging from 2500 to 3000 ppm (Oglesby et al. 1949). The 1000-ppm TLV represented the hygienic standard observed in the 1960s and earlier for that group of vapors considered by industrial hygienists to be relatively harmless. In the interval since the adoption of the 1968 TLVs by OSHA, additional information has been developed that shows that exposure to 1000 ppm of acetone causes sensory irritation in some workers. The ACGIH (1986) reports that a study by Vigliani and Zurlo (1955) found that acetone production workers exposed at the 700-ppm level for 3 hours daily for 7 to 15 years experienced inflammation of the respiratory tract, stomach, and duodenum, giddiness, and loss of strength; some of these effects go beyond irritation effects. In another study reported on by the ACGIH, 10 men exposed to various acetone concentrations for 3 to 5 minutes found a level of 200 ppm satisfactory and experienced "slight" irritation at 300 ppm, but could still "tolerate" exposure at 500 ppm (Nelson et al. 1943). The ACGIH concluded that a TLV of 750 ppm was appropriate because Nelson's results were based on "extremely short, 3 to 5 minute, exposures," while DiVincenzo's findings demonstrated "no effects from acetone at 500 ppm except an awareness of odor" (ACGIH 1986).

In recommending a 250-ppm 10-hour TWA limit, NIOSH relied on the Nelson et al. (1943) study, as well as a controlled human exposure study (Matsushita 1969) showing that subjects exposed to 500 ppm for 6 hours experienced mucosal irritation as well as general weakness the following day. Another study cited by NIOSH (Parmeggiani and Sassi 1954) indicated that employees exposed to acetone in the range of 307 to 918 ppm experienced mucosal irritation and CNS disturbances. NIOSH (1978j) concluded that adverse effects will occur upon exposure to acetone concentrations below 500 ppm, and therefore recommended a 250-ppm TWA limit.

OSHA preliminarily concludes that the 750-ppm TLV does not protect against deleterious health effects observed at 700 ppm, 500 ppm, and 300 ppm and that a 250-ppm 8-hour TWA limit is necessary to provide adequate protection against the risk of acetone-induced irritation at these levels. Feasibility is indicated by the fact that more than 95 percent of a very large number of air samples reported in the IMIS data base reveal exposures below 250 ppm. OSHA therefore proposes that the 250-ppm REL be adopted as the OSHA PEL to reduce the risk of irritation for exposed individuals. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for acetone if the Agency determines that this limit will substantially reduce significant risk.

ACROLEIN
CAS: 107–02–8; Chemical Formula: $CH_2=CHCHO$
H.S. No. 1007

OSHA currently has an 8-hour TWA PEL of 0.1 ppm (0.25 mg/m^3) for acrolein. The ACGIH recommends a 0.1-ppm TLV-TWA for this substances, as well as a STEL of 0.3 ppm for a 15-minute period. Acrolein is a colorless or yellowish flammable liquid with a disagreeable, choking odor.

In early inhalation studies of cats (Iwanoff 1911), exposure to 10 ppm acrolein for 3.5 hours was found to have only transient effects, including salivation, lacrimation, respiratory irritation, and mild narcosis. However, later studies reported that an exposure to 1 ppm of acrolein produced marked nose and eye irritation in 5 minutes or less (Cook 1945). Over longer periods, studies have demonstrated fatalities in 1 of 6 rats exposed for 4 hours to airborne concentrations of acrolein at 8 ppm; at 16 ppm, the mortality was 100 percent (Smyth 1956). Irritation of the upper respiratory tract is the primary symptom of acrolein inhalation, but lung edema can occur after exposure to high concentrations (Henderson and Haggard 1943). In addition, skin contact with acrolein causes skin burns and severe injury to the cornea.

OSHA is proposing 0.1 ppm as an 8-hour time-weighted average and a 15-minute short-term limit of 0.3 ppm to provide the necessary protection against the acutely irritating effects of short-term exposure to acrolein. The Agency preliminarily concludes that the combined TWA-STEL being proposed will reduce the risk of severe irritation, skin burns, and corneal damage to which workers could be exposed with an 8-hour TWA alone. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for acrolein if the Agency determines that this limit will substantially reduce significant risk.

ALLYL ALCOHOL
CAS: 107–18–6; Chemical Formula: $CH_2=CHCH_2OH$
H.S. No. 1010

OSHA has a current PEL of 2 ppm TWA for allyl alcohol with a skin notation. The ACGIH also has a TLV-TWA of 2 ppm, supplemented by a STEL of 4 ppm and a skin notation. Allyl alcohol is a colorless liquid with a pungent, mustard-like odor.

The most important adverse effects of occupational exposures to allyl alcohol are upper-respiratory-tract irritation and burns of the eyes. Severe eye irritation has been reported in humans at exposure levels of 25 ppm, and milder irritation has been reported at 5 ppm (Dunlap, Kodarma, Wellington et al. 1958; McCord 1932). Necrosis of the cornea and temporary blindness occurred in one individual exposed to allyl alcohol at a level irritating to the eyes and nose (Smyth 1956). Skin absorption may lead to serious systemic injury (visceral congestion, periportal congestion of the liver, hematuria, and nephritis); when evaporation is prevented or reduced, skin contact causes burns (ACGIH 1986, p. 18).

Exposure to airborne concentrations of allyl alcohol causes a series of characteristic effects, including lacrimation, photophobia, blurred vision, and retrobulbar pain (Dunlap, Kodarma, Wellington et al. 1958). Exposed individuals do not develop a tolerance for this substance, and they also do not become sensitized (Kodama and Hine 1958).

OSHA proposes a short-term limit of 4 ppm (15 minutes) and an 8-hour TWA PEL of 2 ppm to provide protection against the risk associated with severe eye irritation resulting from exposure to allyl alcohol. This short-term limit ensures that workers will be protected against the health effects associated with exposures at levels only somewhat above the current 8-hour TWA limit. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for allyl alcohol if the Agency determines that this limit will substantially reduce significant risk.

ALLYL GLYCIDYL ETHER
CAS: 106-92-3; Chemical Formula: $C_6H_{10}O_2$
H.S. No. 1012

OSHA's current PEL for allyl glycidyl ether (AGE) is 10 ppm (45 mg/m^3) as a ceiling. The ACGIH recommends a TLV-TWA of 5 ppm and a 15-minute STEL of 10 ppm and additionally has a skin notation. NIOSH has recommended a 15-minute ceiling of 9.6 ppm for this colorless liquid, which has a characteristic, but not unpleasant, odor.

In limited human exposure studies, AGE has been demonstrated to cause dermatitis and eye irritation; the substance produces moderate primary skin irritation and severe eye irritation in animals (Hine, Kodama, Wellington et al. 1956). At 260 ppm, animals experienced irritation of the eyes and respiratory distress; at high levels (e.g., 400, 600, and 900 ppm), corneal opacities and severe respiratory difficulties occurred (Hine, Kodama, Wellington et al. 1956). The percutaneous LD_{50} for rabbits is 2.55 g/kg. Intragastric administration of AGE in mice, rats, and rabbits has also been demonstrated to cause depression of the central nervous system (Hine, Kodama, Wellington et al. 1956).

In humans, skin sensitization occurs readily (Hine and Rowe 1962), and, based on animal studies, percutaneous absorption would appear likely. In addition to primary irritation and sensitization, the potential exists for cross-sensitization with other epoxy agents (ACGIH 1986, p. 20).

OSHA is proposing to establish a PEL of 5 ppm as an 8-hour TWA and to supplement this with a 15-minute STEL of 10 ppm, and with a skin notation, to ensure protection against primary irritation and to minimize sensitization effects. The Agency preliminarily concludes that this combination of full-shift and short-term limits, in addition to a skin notation, will reduce the risk of sensitization, primary irritation, and percutaneous absorption to which workers could otherwise be exposed at the current ceiling of 10 ppm. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for allyl glycidyl ether if the Agency determines that this limit will substantially reduce significant risk.

ALLYL PROPYL DISULFIDE
CAS: 2179-59-1; Chemical Formula: $CH_2=CHCH_2S_2C_3H_7$
H.S. No. 1013

The current OSHA PEL for allyl propyl disulfide is 2 ppm (12 mg/m^3) as an 8-hour TWA. The ACGIH recommends a TLV-TWA of 2 ppm and a TLV-STEL of 3 ppm (18 mg/m^3). Allyl propyl disulfide is a liquid with a pungent, irritating odor.

Nearly all occupational exposures to allyl propyl disulfide, the primary volatile constituent of onion oil, occur in the processing of onions and onion products. Allyl propyl disulfide's irritative effects on the human eye, nose, and upper respiratory tract are well recognized. The most severe irritation effects have occurred when workers were exposed to allyl propyl disulfide in the vicinity of onion slicing machines, where average concentrations of 3.4 ppm have been measured (Feiner, Burke, and Baliff 1946).

OSHA proposes a 2-ppm 8-hour TWA limit and a STEL of 3 ppm to protect against irritation and lacrimation, which can occur as a result of higher short-term exposures. OSHA preliminarily concludes that adding a STEL of 3 ppm will reduce the risk of lacrimation and upper respiratory tract irritation to which employees could otherwise be exposed. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for allyl propyl disulfide if the Agency determines that this limit will substantially reduce significant risk.

AMMONIA
CAS: 7664-41-7; Chemical Formula: NH_3
H.S. No. 1021

OSHA's current limit for ammonia is 50 ppm TWA. The ACGIH revised its TLV-TWA to 25 ppm in 1973, and added a 35-ppm STEL in 1976. NIOSH (1974a) has recommended a 5-minute short-term limit of 50 ppm for ammonia. Ammonia is principally used as a feedstock in the manufacture of fertilizers and other chemical substances, and is also used as a refrigerant.

The chief effect resulting from exposure to ammonia is eye and upper respiratory tract irritation. Vigliani and Zurlo (1956) reported respiratory tract irritation and irritation of the conjunctivae in workers exposed to 100 ppm; exposure to a concentration of 20 ppm caused complaints of discomfort among uninjured workers. An analysis of plant surveys (Bureau of Industrial Hygiene, Detroit Department of Health, 1965-1970) conducted by ACGIH (1986) showed that the "complaint level" was between 20 and 25 ppm. ACGIH (1986) also stated in their documentation for the ammonia TLVs that general field experience with a "large number of workers" exposed to ammonia from printing and copying machines indicated that concentrations of 20 to 25 ppm were the maximum concentrations not associated with complaints of irritation. The ACGIH selected a 25-ppm TLV-TWA and a 35-ppm TLV-STEL "to protect against irritation to eyes and respiratory tract and minimize discomfort among uninjured workers" (ACGIH 1986).

In recommending a 5-minute 50-ppm short-term limit, NIOSH relied on several reports that ammonia concentrations as low as 50 ppm are moderately irritating (Vigliani and Zurlo 1956; Industrial Biotest Laboratories 1973; MacEwen et al. 1970; Mangold 1971; Pagnotto 1973). NIOSH concluded

that the "irritating or annoying effects * * * [are] more dependent upon concentration than length of exposure," and that "a standard expressed as a time-weighted average is inappropriate since it would permit fluctuations to concentrations considerably higher than 50 ppm" (NIOSH 1974a, p. 69). Therefore, NIOSH recommended a 50-ppm ceiling limit to restrict 5-minute fluctuations in exposure levels and to "ensure that such possibly irritating exposures are brief" (NIOSH 1974a, p. 70).

The ACGIH (1986) disagreed that a short-term limit alone was more appropriate than a TWA limit, citing animal evidence (Stombaugh 1960) indicating that continuous, 24-hour exposure over several days produced effects not observed from high, short-term exposures. NIOSH also reviewed this and other evidence (Doig and Willoughby 1971; Coon et al. 1970), showing that laboratory animals exhibited chronic lung inflammation and marked thickening of the tracheal epithelium following continuous exposure to average concentrations exceeding 100 ppm. Continuous exposure to concentrations below 100 ppm did not generally result in microscopic lung abnormalities (Coon et al. 1970; Stombaugh et al. 1969).

OSHA believes that the human evidence discussed by the ACGIH (1986) and NIOSH (1974a) clearly indicates that the current limit of 50 ppm as an 8-hour TWA would permit short-term exposures to levels well above those reported to caused conjunctival and respiratory tract irritation, even among workers who are acclimated to the effects of ammonia. OSHA preliminarily concludes that there is a risk of mucosal irritation at the current PEL and that it is necessary to lower the PEL to reduce that risk. The 50-ppm 5-minute ceiling recommended by NIOSH is above exposure levels that are reported to be moderately irritating to the eyes and respiratory tract. Therefore, OSHA is proposing to revise its existing PEL for ammonia to 25 ppm as a TWA and 35 ppm as a STEL. Extensive air sampling data reported in the IMIS data base show that more than 90 percent of exposures are below 25 ppm TWA and thus demonstrate the feasibility of the proposed limits. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for ammonia if the Agency determines that this limit will substantially reduce significant risk.

AMMONIUM CHLORIDE (FUME)
CAS: 12125-02-8 Chemical Formula: NH_4Cl
H.S. No. 1022

No current OSHA PEL for ammonium chloride fume has been established. The ACGIH recommends a TLV-TWA of 10 mg/m³ and a 20-mg/m³ TLV-STEL. Ammonium chloride is a white crystalline solid, somewhat hygroscopic, with a cool, saline taste.

Ammonium chloride is an irritant to the skin and respiratory passages when inhaled and produces mild systemic toxicity when ingested (Sax 1968). Large amounts of fume can be generated during galvanizing operations, and these fumes should be controlled to prevent irritation of the respiratory tract (ACGIH 1986, p. 28).

OSHA proposes a permissible exposure limit of 10 mg/m³ TWA, with a 15-minute short-term limit of 20 mg/m³, to protect workers exposed to ammonium chloride fume in the numerous applications in general industry involving this substance. The Agency preliminarily concludes that this combination of TWA and STEL limits will protect workers against the risk of respiratory irritation and systemic effects, and will eliminate or reduce this risk. The health evidence forms a reasonable basis for proposing a new limit for ammonium chloride fume. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

BORATES, TETRA, SODIUM SALTS (ANHYDROUS AND PENTAHYDRATE)
CAS: 1303-96-4 Chemical Formula: $Na_2B_4O_7$ (anhydrous), $Na_2B_4O_7 \cdot 5H_2O$ (pentahydrate)
H.S. Nos. 1036 and 1038

OSHA currently has no limit for exposure to anhydrous or pentahydrate sodium tetraborate. The ACGIH recommends a TLV-TWA of 1.0 mg/m³. The anhydrous form is a light gray, odorless solid; the pentahydrate form is white, odorless, and crystalline.

In the workplace, the salient toxic effects of the tetraborates are acute irritation of the skin, eyes, nose, and upper respiratory tract. Dermatitis, cough, nasal irritation, shortness of breath, and nosebleeds resulting from visible (but unstated) concentrations of borate dust in mining and milling facilities have been reported (Birmingham and Key 1963). There is evidence that respiratory ill health may be associated with inhalation exposures to dehydrated sodium borate dust (Hogan 1965; Ury 1966).

To prevent these acute irritant effects and to protect against chronic respiratory effects, OSHA is proposing an 8-hour TWA PEL of 1.0 mg/m³ for the anhydrous and pentahydrate forms of sodium tetraborate. The Agency preliminarily concludes that this limit will protect exposed workers against the risk of acute irritation and chronic respiratory ill health potentially associated with these substances, which have not previously been regulated by OSHA. The health evidence forms a reasonable basis for proposing a new limit for borates. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

BORATES, TETRA, SODIUM (DECAHYDRATE)
CAS: 1303-96-4; Chemical Formula: $N_2B_4O_7 \cdot 10H_2O$
H.S. No. 1037

OSHA currently has no limit for decahydrate sodium tetraborate. The ACGIH recommends a TLV-TWA of 5.0 mg/m³ for this white, odorless, and crystalline substance.

In humans, exposure to more than 1 gram of borax ($N_2B_4O_7 \cdot 10H_2O$) through gastroenteric or percutaneous absorption can cause acute toxicity in the form of severe gastrointestinal irritation, kidney injury, and even death from central nervous system depression or vascular collapse. It is also known that chronic exposure to small amounts can cause mild gastroenteritis and dermatitis (Browning 1969; Deichmann and Gerarde 1969; Thienes and Haley 1972). However, neither of these types of systemic poisoning have been reported in the workplace (Browning 1969).

In the workplace, the salient toxic effects of the tetraborates are acute irritation of the skin, eyes, nose, and upper respiratory tract. Dermatitis, cough, nasal irritation, shortness of breath, and nosebleeds resulting from visible (but unstated) concentrations of borate dust in mining and milling facilities have been reported (Birmingham and Key 1963). There is evidence that respiratory ill health may be associated with inhalation exposures to dehydrated sodium borate dust (Hogan 1965; Ury 1966).

To prevent exposed workers against these acute irritant effects and the potential for chronic respiratory ill health, OSHA is proposing an 8-hour PEL of 5.0 mg/m³ for decahydrate sodium tetraborate. The Agency preliminarily concludes that this limit will protect workers exposed to this form of sodium tetraborate from the risk posed by these acute and chronic irritant and respiratory effects. The health evidence forms a reasonable basis for proposing a new limit for borax. At the time of the final rule, OSHA will promulgate a new limit if the

Agency determines that this limit will substantially reduce significant risk.

BROMINE
CAS: 7726-95-6; Chemical Formula: Br_2
H.S. No. 1042

OSHA's current limit for bromine is 0.1 ppm TWA. The ACGIH recommends a TLV-TWA of 0.1 ppm with a TLV-STEL of 0.3 ppm. Bromine is a dark, reddish-brown, non-combustible, diatomic liquid that has irritating vapors.

Early studies of bromine exposure indicated that workers exposed to 0.75 ppm for 6 hours exhibited no symptoms (Flury and Zernik 1931). Later studies reported physiological responses to different concentrations of bromine and used these findings to make the following recommendations: The maximal allowable concentration for prolonged exposure should be 0.1 to 0.15 ppm, and the maximal allowable concentration for short exposure (i.e., 30 minutes to 1 hour) should be 4 ppm (Henderson and Haggard 1943). These investigators found levels of 40 to 60 ppm dangerous for short-term exposures, and a level of 1000 ppm proved rapidly fatal even during short exposures. These authors reported that the effects of exposure to bromine include respiratory irritation and lung edema. Elkins (1951) reported that exposure at 1 ppm in a plant handling liquid bromine was excessively irritating.

OSHA proposes a PEL of 0.1 ppm (8-hour TWA) and a STEL of 0.3 ppm for a 15-minute period. The Agency believes that both the TWA and short-term limits are necessary to reduce the risk of respiratory irritation and lung damage that could occur in the absence of a short-term limit. The Agency believes that the combined TWA-STEL will substantially reduce this risk among occupationally exposed workers. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for bromine if the Agency determines that this limit will substantially reduce significant risk.

2-BUTANONE (METHYL ETHYL KETONE)
CAS: 78-93-3; Chemical Formula: $CH_3COCH_2CH_3$
H.S. No. 1045

OSHA's current limit for 2-butanone is 200 ppm TWA. The ACGIH's recommended limit is 200 ppm TWA, with a STEL of 300 ppm. NIOSH recommends a 200-ppm 10-hour TWA limit for 2-butanone. 2-Butanone is a colorless, flammable liquid and has an objectionable odor.

2-Butanone is an ocular and upper respiratory tract irritant. Some authors (Nelson, Nelson, Ross et al. 1943) report that exposures to 200 ppm caused mild eye irritation in some subjects and that others experienced these effects even at concentrations of 100 ppm. Studies conducted in the 1940s noted low-grade intoxication at 300 to 600 ppm (Smith and Mayers 1944), slight nose and throat irritation at 100 ppm, and mild eye irritation at 200 ppm (Nelson, Nelson, Ross et al. 1943). Later studies have shown that approximately 50 percent of exposed individuals experience eye and nose irritation at 200 ppm (as reported in ACGIH 1986, p. 395).

OSHA is proposing a 15-minute STEL of 300 ppm and an 8-hour TWA limit of 200 ppm to protect exposed individuals against the short-term exposures known to cause irritation. However, since a significant number of all exposed workers experience adverse irritant effects even at 200 ppm, the Agency realizes that the 300-ppm STEL may not be fully protective. Therefore OSHA specifically requests comments on the adequacy of the proposed limits to reduce the risk of such irritation in exposed employees. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for 2-butanone if the Agency determines that this limit will substantially reduce significant risk.

n-BUTYL ACETATE
CAS: 123-86-4; Chemical Formula: $CH_3COO(CH_2)_3CH_3$
H.S. No. 1047

The current OSHA limit for n-butyl acetate is 150 ppm, measured as an 8-hour TWA. The ACGIH (1986) also recommends an 8-hour TWA of 150 ppm with the addition of a 200-ppm 15-minute STEL. n-Butyl acetate is a colorless liquid with a fruity odor.

n-Butyl acetate is an irritant to the eyes, skin, and respiratory system. In a study involving cats exposed for 6 hours to 6100 ppm, slight narcotic effects were noted (Flury and Wirth 1933). When exposed to 4200 ppm n-butyl acetate for 6 days at 6 hours per day, cats experienced slight irritation of the respiratory passage; at 3100 ppm, changes in blood cell morphology were recorded. At exposures of 1600 ppm, these cats exhibited slight irritation of the eyes and salivation (Flury and Wirth 1933). Air concentrations of 10,000 ppm (n-butyl acetate proved fatal to rats after 8 hours; 4 hours of exposure at the same level produced no deaths (Smyth, et al. 1956). A paper by Sayers, Schrenk, and Patty (1936) reported that guinea pigs demonstrated eye irritation effects at 3300 ppm, became unconscious after 9 hours exposure to 7000 ppm, and died after 4 hours of exposure to 14,000 ppm.

Human volunteers complained that throat irritation, which began at an exposure level of 2000 ppm n-butyl acetate, worsened and became quite severe at 300 ppm (Nelson, Ege, Morwich et al. 1943).

OSHA is proposing an 8-hour TWA of 150 ppm and a 200-ppm STEL for n-butyl acetate. The Agency preliminarily concludes that these limits are necessary to protect workers from the risks of eye, skin and respiratory irritation, in addition to narcotic effects, potentially associated with exposures to this substance at the levels permitted by the 8-hour limit alone. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for n-butyl acetate if the Agency determines that this limit will substantially reduce significant risk.

n-BUTYL LACTATE
CAS: 138-22-7; Chemical Formula: $C_7H_{14}O_3$
H.S. No. 1053

OSHA currently has no limit for n-butyl lactate. The ACGIH-recommended limit is a TLV-TWA of 5 ppm. Butyl lactate is a colorless liquid ester of lactic acid.

In humans, prolonged exposures to n-butyl lactate at approximately 7 ppm, with brief peak excursions to 11 ppm, caused headache, irritation of the pharyngeal and laryngeal mucosa, and coughing in all workers, and occasional nausea, vomiting, and sleepiness in some (Zuidema and Pel 1969, as cited in ACGIH 1986, p. 82). Headache, coughing, and irritation of the pharynx were sometimes related to n-butyl lactate concentrations of 4 ppm; however, no adverse effects were observed at a concentration of 1.4 ppm. Studies employing improved sampling and analytic methods have subsequently concluded that, although the odor of n-butyl lactate is discernible at the 7-ppm level, this concentration does not produce objectionable or injurious effects (Turner 1972).

OSHA proposes a limit of 5 ppm TWA for n-butyl lactate to protect against the risk of irritation, headache, nausea, and coughing associated with occupational exposures to this substance. However, the Agency notes that studies show adverse effects at levels below the proposed 5-ppm limit. OSHA specifically requests data on the health effects associated with exposures to this substance and the airborne concentrations at which such effects occur. The health evidence forms a reasonable basis for proposing a new

limit for n-butyl lactate. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

n-BUTYL MERCAPTAN
CAS: 109-79-5; Chemical Formula: $CH_3CH_2CH_2CH_2SH$
H.S. No. 1054

This chemical is used as a solvent and chemical intermediate, and as an odorant for natural gas. OSHA's current limit for n-butyl mercaptan is 10 ppm as an 8-hour TWA. The ACGIH has recommended a TLV-TWA of 0.5 ppm. The NIOSH REL for n-butyl mercaptan is 0.5 as a 15-minute short-term exposure.

Humans exposed to concentrations of butyl mercaptan report that the "readily noticeable" odor level for this substance is between 0.1 and 1 ppm, although the odor threshold is significantly below this level (ranging from 0.001 to 0.0001 ppm). Gobbato and Terribile (1968) have reported that symptoms of CNS toxicity occurred in humans exposed for 1 hour to concentrations of n-butyl mercaptan believed to lie in the range of 50 to 500 ppm. These same authors reported that mucosal irritation occurred in human volunteers exposed to 4 ppm of ethyl mercaptan, a closely related substance. Irritation did not occur at exposures to 0.4 ppm. The ACGIH established the TLV at 0.5 ppm, which is approximately halfway between 0.1- to 1.0-ppm levels reported as being readily noticeable (ACGIH 1986). Thus, the ACGIH TLV was not set at a level designed to protect against detecting the odor of butyl mercaptan, but to avoid the intolerable odor effects of higher concentrations of this substance. NIOSH (1978) recommended a 15-minute short-term limit of 0.5 ppm because the toxic action of n-butyl mercaptan, like that of other thiol compounds, is expressed as an acute effect. NIOSH concluded that a short-term limit was more appropriate than a TWA limit, and that "use of a ceiling [i.e., short-term] concentration instead of a TWA has the effect of increasing the protection provided to the worker about twofold" (NIOSH 1978, p. 85).

OSHA preliminarily concludes that the current PEL for n-butyl mercaptan is insufficient to protect workers from experiencing the adverse acute effects caused by exposure to this substance. The current PEL of 10 ppm is between 10 and 100 times higher than the concentration of n-butyl mercaptan that is readily detected by smell and is more than twice the concentration reported as causing mucosal irritation for a closely related substance. OSHA finds that workers are at risk of these acute effects in the absence of a more stringent limit and is proposing to reduce its exposure limit for n-butyl mercaptan to 0.5 ppm TWA. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for n-butyl mercaptan if the Agency determines that this limit will substantially reduce significant risk.

CAMPHOR (SYNTHETIC)
CAS: 76-22-2; Chemical Formula: $C_{10}H_{16}O$
H.S. No. 1063.

OSHA currently has a limit of 2 ppm for camphor. The ACGIH recommends a TLV-TWA of 2 ppm and a TLV-STEL of 3 ppm. Synthetic camphor is a colorless or white crystalline substance with an aromatic odor.

Synthetic camphor is known to cause severe injuries in animals exposed for prolonged periods by inhalation to 6 mg/m^3. Exposure may cause convulsions, congestion and changes in the gastrointestinal tract, and damage to the kidneys and brain (Flury and Zernik 1931). Animal bioassays showed that camphor was not carcinogenic in rats injected subcutaneously; however, when the cancer promoter, croton oil, was concurrently applied to the skin of mice, two of 110 treated mice developed carcinomas (Graffi et al. 1953).

In humans, there are reports of industrial exposure to camphor that resulted in coma, dyspnea, and headache; one fatality from inhalation of the vapor has been noted (Flury and Zernik 1931). Exposures for up to 10 months in a synthetic camphor packing plant are reported not to have involved eye and nose irritation if concentrations were maintained at or below 2 ppm (Gronka, Bobkoski, Tomchick, and Rakow 1969). However, the same authors report that these packing plant workers did experience nose and throat inflammation.

OSHA proposes a TWA of 2 ppm and a STEL of 3 ppm for synthetic camphor to prevent the risk of irritation to the eyes, nose, and central nervous system potentially associated with elevated exposures to this substance. The Agency preliminarily concludes that the combined TWA-STEL is necessary to reduce this risk. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for synthetic camphor if the Agency determines that this limit will substantially reduce significant risk.

CAPROLACTAM (DUST)
CAS: 105-60-2; Chemical Formula: $C_6H_{11}NO$
H.S. No. 1064

OSHA has no current permissible exposure limit for caprolactam dust. The ACGIH recommends a 1-mg/m^3 TLV-TWA for this substance, with a short-term exposure limit of 3 mg/m^3. Caprolactam is a white crystalline solid with an unpleasant odor.

In humans, caprolactam has been shown to be a convulsant, a dermal and respiratory irritant, and a dermal sensitizer, but dosage levels in humans are ill-defined (Ferguson and Wheller 1973; Tumà, Fossela, and Waidhofer 1981). In animals, exposure to caprolactam by several routes can cause convulsions, tremors, mydriasis, opisthotonus (Elison, Lein, Zinger, et al. 1971; Lein, Lein, and Tong 1971) and salivation (Goldblatt et al. 1954). Cardiovascular and respiratory effects have been reported in rabbits and cats, with an initial increase in blood pressure followed by a decrease in blood pressure and an increased respiratory rate (Goldblatt, Farquharson, Bennett, and Askew 1954). Weight loss and initial growth depression occur in rates and mice (Morrison, Ross, and Ruth 1980).

One animal study observed that caprolactam's convulsant effects on rats, rabbits, and cats occur at injection doses above 100 mg/kg (Goldblatt et al. 1954). Results of studies in guinea pigs were consistent with these findings (Hohensee 1951). In a 90-day feeding study of dogs, Burdock, Kolwick, Alsakor, and Marshal (1984) reported that dogs given dietary dose levels of 0.1, 0.5, and 1.0 percent caprolactam showed weight losses at 1.0 percent and lesser losses at 0.5 percent. Hematologic and ophthalmologic changes did not occur. In a 2-year bioassay of rats and mice, caprolactam was not observed to be carcinogenic (NCI/NTP 1982). A Polish study in animals observed hematologic and systemic changes, increased mortality, kidney and liver damage, and growth inhibition in animals given daily doses of 50 or 100 mg/kg (Zwierzchowski et al. 1967).

The results of early studies of caprolactam's teratogenicity in rats and rabbits indicate that it is not teratogenic even at doses as high as 1000 mg/kg/day (Gas, Powers, Robinson et al. 1984).

Studies of industrial exposures to caprolactam dust in Germany report severe irritation on inhalation of 10 percent caprolactam in dust (Hohensee 1951). Workers experienced a bitter taste, nervousness, epistaxis, upper respiratory tract irritation, and dry and splitting skin on the lips and nose (Hohensee 1951). Ferguson and Wheller (1973) found that some workers exposed to caprolactam vapors experienced nose

and throat irritation at concentrations above 10 ppm, and others reported irritation at levels below 10 ppm. Direct contact with the solid form of caprolactam produces primary skin irritation (Ferguson, unpublished communication, as cited in ACGIH 1986, p. 96–2). Brief (unpublished communication, as cited in ACGIH 1986, p. 96–2) also reports that the dust produces skin irritation.

OSHA is proposing a permissible exposure limit of 1 mg/m³ TWA and a 3-mg/m³ STEL for caprolactam dust to prevent the risk of respiratory and skin irritation and of sensitization, to which workers can be subjected in the absence of any OSHA limit. The Agency preliminarily concludes that this combination of limits will substantially reduce this risk. The health evidence forms a reasonable basis for proposing a new limit for caprolactam (dust). At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

CAPROLACTAM (VAPOR)
CAS: 105–60–2; Chemical Formula: $C_6H_{11}NO$
H.S. No. 1065

OSHA has no current permissible exposure limit for caprolactam as vapor. The ACGIH recommends a TVL–TWA of 20 mg/m³ for the vapor, supplemented by a STEL of 40 mg/m³. Caprolactam is a white, crystalline solid at room temperature, and thus high vapor levels occur only at elevated temperatures.

The health effects of exposure to caprolactam vapor are identical to those described for caprolactam dust, except that contact with the vapor is reported to be even more irritating (Hohensee 1951). Workers exposed to the vapor at approximately 12 ppm complained of a bitter taste in the mouth, nervousness, epistaxis, upper respiratory tract congestion, and dry and splitting skin; other workers reported experiencing heartburn, flatulence, and a heavy feeling in the stomach (Hohensee 1951).

In another report of industrial exposure to the vapor, Ferguson and Wheeler (1973) reported that workers routinely exposed to unspecified levels and occasionally to concentrations as high as 100 ppm for 18 years reported severe discomfort from burning nose, throat, and eyes. This irritation response was dose-related, with no workers reporting effects at 7 ppm or below, some experiencing transient upper respiratory tract irritation at levels above that, and others reporting eye irritation at concentrations of 25 ppm and above (Ferguson and Wheeler 1973). Ferguson (private communication, 1972, as cited in ACGIH 1986, p. 96.1) noted that a group of 143 workers, some of whom were exposed for as long as 17 years to vapor concentrations of 5 to 10 ppm showed no evidence of adverse effects. At higher vapor exposures (53 to 521 mg/m³), all subjects experienced eye irritation (Ferguson, private communication, 1972). Human volunteers exposed at low relative humidities to concentrations of the vapor in the range of 10 to 100 ppm showed a dose-related response, but at higher relative humidities, no irritation was observed below a concentration of 14 ppm (Ferguson and Wheeler 1973).

OSHA is proposing to establish an 8-hour TWA exposure limit of 20 mg/m³ for caprolactam vapor and to supplement this with a STEL of 40 mg/m³. The Agency preliminarily concludes that this combination of limits will protect workers from the risk of eye, upper respiratory, and skin irritation to which they could otherwise be exposed in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for caprolactam vapor. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

CESIUM HYDROXIDE
CAS: 21351–79–1; Chemical Formula: CsOH
H.S. No. 1077

OHSA currently has no limit for cesium hydroxide. The ACGIH recommends a threshold limit vlaue of 2 mg/m³ as an 8-hour TWA. Cesium hydroxide is a colorless or yellowish fused crystalline mass; it is the strongest base known and is highly soluble in water and alcohol.

Animal studies indicate that cesium hydroxide has an acute oral toxicity of about one-third that of potassium hydroxide, which causes lesions of the nasal septum and irritation of the eyes and respiratory tract (Kanpov 1971, as cited in ACGIH 1986, p. 495). The oral LD_{50} for cesium hydroxide in rats is 1016 mg/kg. A concentration of 5 percent cesium hydroxide did not produce skin irritation; however, severe irritation of the eyes resulted from contact with a 5-percent concentration of cesium hydroxide. Cesium hydroxide does not cause skin sensitization (Johnson, Lewis, and Perone 1972).

OSHA proposes a TWA of 2 mg/m³ as an 8-hour permissible exposure limit for cesium hydroxide. The Agency preliminarily concludes that this limit will protect exposed workers from the risk of severe eye irritation associated with exposure to this substance at the levels permitted in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for cesium hydroxide. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

CHLORINE
CAS: 7782–50–5; Chemical Formula: Cl_2
H.S. No. 1079

The current OSHA limit for chlorine is a 1-ppm ceiling limit. The ACGIH has recommended a TLV–TWA of 1 ppm with a TLV–STEL of 3 ppm. NIOSH (1976e) has recommended a limit of 0.5 ppm over a 15-minute sampling duration.

Exposure to chlorine at concentrations around 5 ppm has been associated with respiratory symptoms, erosion of teeth, and inflammation of mucous membranes (Flury and Zernik 1931; Patty 1963). Ferris et al. (1964) reported slight effects on the respiratory system in workers exposed to chlorine ranging from negligible to 7 ppm. Rupp and Henschler (1967) reported burning of the eyes among human subjects exposed to 0.4 ppm; some of these subjects reported painful eyes after 15 minutes' exposure to this level. In a separate test, subjects reported respiratory irritation upon exposure to 0.5 ppm, and a concentration of 1 ppm was described as being uncomfortable.

After reviewing these reports, the ACGIH recommended the 1-ppm TLV–TWA and 3-ppm TLV–STEL to "minimize chronic changes in the lungs, accelerated aging, and erosion of the teeth" (ACGIH 1986, p. 117). NIOSH (1976e) reviewed these studies, as well as others (Matt 1889; Beck 1959) that reported ocular and respiratory irritation associated with exposure to chlorine levels of around 1 ppm for 30 minutes or less. NIOSH (1976e) recommended a 15-minute 0.5-ppm limit to prevent possible eye and respiratory tract irritation.

The current OSHA limit for chlorine (1 ppm ceiling) and the ACGIH TLVs (1 ppm TWA and 3 ppm STEL) are inadequate to protect against the risk to workers' health. Studies show that humans exposed to 0.4 ppm experienced burning of the eyes, and when exposed to 0.5 ppm they experienced respiratory irritation. The subjects were "uncomfortable" when exposed to 1 ppm of chlorine. OSHA therefore proposes adoption of the NIOSH REL (0.5 ppm ceiling) to prevent possible eye and respiratory irritation and thus substantially to reduce this risk. The Agency's preliminary feasibility analysis is based on limited data at this level; additional feasibility information is requested from the public. The health

evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for chlorine if the Agency determines that this limit will substantially reduce significant risk.

CHLOROACETYL CHLORIDE
CAS: 79-04-9; Chemical Formula: $ClCH_2COCl$
H.S. No. 1083

OSHA has no current limit for chloroacetyl chloride. The ACGIH recommends a TLV-TWA of 0.05 ppm. Chloroacetyl chloride is a colorless liquid with a pungent odor.

The oral LD_{50} in rats fed this substance is between 0.12 and 0.25 g/kg. Chloroacetyl chloride is corrosive to the skin and eyes, and skin absorption of this substance can be lethal. Inhalation of 4 ppm for 5 to 10 minutes caused respiratory problems in rats; however, no effect was observed in these animals when they inhaled 2.5 ppm for a period of 7 hours (Dow Chemical Company 1977, as cited in ACGIH 1986, p. 122). Thirty-day inhalation studies with rats, mice, and hamsters showed eye and respiratory irritation at 2.5 ppm and no effect at 0.5 ppm (Dow Chemical Company 1977, as cited in ACGIH 1986, p. 122).

Reports of the acute effects associated with exposure to chloracetyl chloride in humans include mild to moderate skin burns and erythema, eye burns and tearing, cough, dyspnea, and cyanosis, as well as mild gastrointestinal effects. Eye and respiratory irritation occurred in an industrial setting characterized by an exposure level of 0.009 to 0.017 ppm, with excursions as high as 0.140 ppm (Dow Chemical Company 1977, as cited in ACGIH 1986, p. 122). An accidental drenching with a mixture containing chloroacetyl chloride resulted in extensive first- and second-degree burns, pulmonary edema, and three episodes of cardiac arrest, followed by coma and anoxia-induced brain damage (Pagnotto 1978, as cited in ACGIH, p. 122). Other ingredients of the mixture involved in the accident included xylidine, benzene, and sodium carbonate. Rescuers of this victim experienced hand blisters, chest tightness, and nausea for 2 days.

OSHA proposes a PEL of 0.05 ppm TWA for chloroacetyl chloride. The Agency preliminary concludes that this limit is necessary to protect exposed employees from the risk of eye, skin, and respiratory irritation; gastrointestinal effects; and severe systemic effects, including life-threatening coma, cardiac arrest, and pulmonary edema, to which workers could presently be exposed in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for chloroacetyl chloride. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

o-CHLOROBENZYLIDENE MALONONITRILE
CAS: 2698-41-1; Chemical Formula: $ClC_6H_4CH=C(CN)_2$
H.S. No. 1084

OSHA's existing PEL for o-chlorobenzylidene malononitrile (OCBM) is 0.05 ppm as an 8-hour TWA. The ACGIH recommends a ceiling limit of 0.05 ppm, with a skin notation. o-chlorobenzylidene malononitrile is a white crystalline solid with a pepper-like odor.

This substance has extremely irritating properties. It causes intense eye and skin irritation, coughing, difficulty in breathing, chest tightness, running nose, dizziness, nausea, and vomiting. These effects are evident on exposure to concentrations between 12 and 20 mg/m^3, and they become incapacitating within 20 seconds of exposure; the effects persist for approximately 5 to 10 minutes after the victim has been removed to fresh air (*Military Chemistry and Chemical Agents* 1963).

OCBM is only slightly toxic to laboratory animals when they are exposed intravenously, subcutaneously, or through inhalation (Punte, Weimer, Ballard, and Wilding 1962). In animals, it has been demonstrated that OCBM is metabolized by the body to cyanide (Frankenberg and Sorbo 1973). Short-term exposures to high levels of OCBM did not cause carcinogenic, teratogenic, or embryolethal effects in animals (McNamara et al. 1973).

Three of four human volunteers exposed to a 1.5-mg/m^3 concentration of OCBM aerosol dispersed from a 10-percent solution in methylene chloride for 90 minutes developed headaches, and one showed mild eye and nose irritation. Headaches persisted for 24 hours in two subjects. At 4 to 5 mg/m^3, subjects' problem-solving abilities were affected and they showed eye irritation, conjunctivitis, lacrimation, and skin burning (Punte, Owens, and Gutentag 1963). Other researchers observed no persistent clinical abnormalities in seven subjects exposed to OCBM at concentrations ranging from 1 to 13 mg/m^3 over a 15-day period; however, none of these subjects developed a tolerance for the compound. Severe skin senitization has also been reported in workers handling OCBM (Schumes and Taylor 1973).

OSHA is proposing a ceiling limit of 0.05 ppm and a skin notation for o-chlorobenzylidene malononitrile. The Agency preliminarily concludes that these limits are necessary to protect workers from the risk of severe eye and upper respiratory tract irritation, skin sensitization, dyspnea, nausea, lacrimation, vomiting, and performance decrement associated with brief exposures to this substance. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for o-chlorobenzylidene malononitrile if the Agency determines that this limit will substantially reduce significant risk.

CYANOGEN
CAS: 460-19-5; Chemical Formula: $(CN)_2$
H.S. No. 1105

OSHA currently has no limit for cyanogen. The ACGIH recommends a TLV-TWA of 10 ppm for this colorless gas, which has a pungent, almond-like odor.

The acute toxicity for cyanogen in various animal species is high (Flury and Zernik 1931). One-hundred ppm was fatal to cats in 2 to 3 hours, and 400 ppm was fatal to rabbits in less than 2 hours. However, rabbits exposed to 100 ppm for 4 hours showed practically no effects. Cats exposed to 50 ppm were severely affected but recovered (Flury and Zernik 1931). Investigations in the rat suggest that cyanogen is approximately 10 times less acutely toxic than is hydrogen cyanide (McNerney and Schrenk 1960).

Human tests showed that subjects experienced almost immediate eye and nasal irritation at exposures of 16 ppm (McNerney and Schrenk 1960).

OSHA proposes a PEL of 10 ppm TWA for cyanogen. Because of the high acute toxicity of this substance in experimental animals, the Agency preliminarily concludes that this limit is necessary to protect against the risk of irritation and systematic effects associated with exposure at the levels permitted in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for cyanogen. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

CYANOGEN CHLORIDE
CAS: 506-77-4; Chemical Formula: ClCN
H.S. No. 1106

OSHA currently has no limit for cyanogen chloride. The ACGIH recommends a ceiling limit of 0.3 ppm

for this colorless liquid or gas, which has a pungent odor.

The chronic effects of cyanogen chloride, which include hoarseness, conjunctivitis, and edema of the eyelid, have long been recognized (Reed 1920).

Flury and Zernik (1931) observed the effects of exposure to cyanogen chloride in five animal species. In mice, a concentration of approximately 500 ppm was fatal within 3 minutes; in cats, 120 ppm was fatal in 3.5 minutes; 48 ppm was fatal to dogs in 6 hours; in goats, a 1000-ppm exposure for 3 minutes caused death after 70 hours; and 1200 ppm was fatal to the rabbit. Several other studies have demonstrated that animals exposed to cyanogen chloride exhibit pulmonary edema and interference with cellular metabolism (Jandorf and Bodansky 1946; Aldrich and Evans 1946).

Human data indicate that 1 ppm is the lowest irritant concentration for a 10-minute exposure; 2 ppm was intolerable for this time period, and 48 ppm was fatal in 30 minutes (Prentiss 1937). The Michigan Department of Public Health (1977) reported that a concentration of about 0.7 ppm caused severe eye and nasal irritation, forcing workers to quit the area.

OSHA is proposing a ceiling limit of 0.3 ppm for cyanogen chloride. The Agency preliminarily concludes that this ceiling is necessary to protect exposed workers from the risks of severe irritation, metabolic effects, and pulmonary edema associated with exposure to this substance at the levels permitted by the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for cyanogen chloride. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will subtantially reduce significant risk.

DIBUTYL PHOSPHATE
CAS: 107-66-4; Chemical Formula: $(n\text{-}C_4H_9O)_2(OH)PO$
H.S. No. 1119

OSHA currently has an 8-hour TWA PEL of 1 ppm for dibutyl phosphate. The ACGIH recommends a TLV-TWA of 1 ppm and a TLV-STEL of 2 ppm for this pale amber liquid.

There are no published reports of toxic reactions caused by exposure to dibutyl phosphate. However, in a personal communication to the ACGIH, Mastromatteo reported that workers exposed to relatively low levels of dibutyl phosphate developed respiratory tract irritation and headache (Mastromatteo, as cited in ACGIH 1986, p. 175).

OSHA is proposing an 8-hour TWA PEL of 1 ppm and a 15-minute STEL of 2 ppm for dibutyl phosphate. The Agency preliminarily concludes that both a TWA and a STEL are necessary to protect exposed workers from the risk of respiratory tract irritation and headaches reported at low levels of exposure and to substantially reduce this risk. This health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for dibutyl phosphate if the Agency determines that this limit will substantially reduce significant risk.

1,3-DICHLORO-5,5-DIMETHYLHYDANTOIN
CAS: 118-52-5; Chemical Formula: $C_5H_6Cl_2N_2O_2$
H.S. No. 1122

OSHA currently has a limit of 0.2 mg/m³ TWA for 1,3-dichloro-5,5-dimethylhydantoin (DCDMH). The ACGIH recommends a TLV-TWA of 0.2 mg/m³ TWA and a TLV-STEL OF 0.4 mg/m³ for this white powder, which has a mild odor similar to that of chlorine.

1,3-Dichloro-5,5-dimethylhydantoin produces systemic toxicity in laboratory animals. The acute oral LD_{50} in rats of both sexes is 542±84 mg/kg when administered as a 10-percent aqueous suspension. Rats dying within 48 hours of administration showed gastrointestinal hemorrhage at necropsy. The animals tolerated aqueous solutions of DCDMH maintained at 20-ppm available chlorine (Industrial Bio-Test Laboratories, as cited ACGIH 1986, p. 183).

Limited human exposure data have been provided by Baier, who reported that individuals experienced extreme respiratory irritation at an average level of 1.97 mg/m³, but that some experienced this degree of irritation even at 0.7 mg/m³ (Baier, as cited in ACGIH 1986, p. 183).

OSHA is proposing a PEL of 0.2 mg/m³ TWA and a STEL of 0.4 mg/m³ for DCDMH. These limits are based on evidence of systemic toxicity in laboratory animals and respiratory irritation at low exposure levels in human subjects. The Agency preliminarily concludes that both a TWA and STEL are required to protect exposed workers from the risk of respiratory irritation that has been shown to occur at levels only slightly above the level specified by the 8-hour TWA limit. OSHA believes that the two limits will reduce this risk substantially. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for 1,3-dichloro-5,5-dimethylhydantoin if the Agency determines that this limit will substantially reduce significant risk.

DICHLOROETHYL ETHER
CAS: 111-44-4; Chemical Formula: $(CH_2ClCH_2)_2O$
H.S. No. 1127

OSHA currently has a 15-ppm ceiling limit, with a skin notation, for dichloroethyl ether. The ACGIH recommends a TLV-TWA of 5 ppm with a TLV-STEL of 10 ppm, also with a skin notation. Dichloroethyl ether is a colorless, flammable liquid with a nauseating odor.

The primary health hazards associated with exposure to this substance are irritation of the eyes and respiratory system and pulmonary damage. Schrenk, Patty, and Yant (1933) report that guinea pigs exposed to the vapor of dichloroethyl ether at 500 ppm experienced immediate and severe eye and nose irritation, respiratory disturbances after 1.5 to 3 hours, and death after 5 to 8 hours. Lung, kidney, liver, and brain damage were also observed in these animals; exposure to a reduced level of 105 ppm caused eventual death after 10 hours of continuous exposure. A one-hour exposure to 105 ppm caused irritation only (Carpenter, Smyth, and Pozzani 1949). At 35 ppm, for an unspecified duration, irritation but no other adverse effects were observed (Schrenk, Patty, and Yant 1933). Rats responded similarly, with 4-hour exposures to 250 ppm proving lethal (Carpenter, Smyth, and Pozzani 1949).

Repeated exposures to 69 ppm (7 hours/day, 5 days/week for 130 days) caused no serious injury in rats or guinea pigs; only mild stress-related effects were noted (Kosyan 1969). However, other studies of guinea pigs have shown mild primary irritative effects on the skin, and fatalities occurred when 300 mg/kg was applied dermally as a pure liquid for 24 hours (Smyth, Jr. and Carpenter 1948). Direct contact of dichloroethyl ether with the eye causes moderate pain, conjunctival irritation, and transient corneal injury (Carpenter and Smyth 1946). A sufficient amount of dichloroethyl ether can be absorbed through the skin to be lethal (Carpenter and Smyth 1946). Mice have been reported to develop hepatomas after prolonged oral administration (80 weeks) at 300 mg/kg (Innes et al. 1969).

Humans exposed briefly to dichloroethyl ether at concentrations above 550 ppm experienced intolerable eye and nasal irritation, with coughing, nausea, and retching. Concentrations between 100 and 260 ppm were irritating but tolerable; however, the odor of

dichloroethyl ether was still nauseating at 35 ppm (Schrenk, Patty, and Yant 1933). Eye irritation has been reported from industrial exposure to a concentration of dichloroethyl ether of 2.5 ppm (Bell and Jones 1958). A single fatality, presumably from inhalation of the vapor, has been reported but not documented (Elkins 1959).

OSHA proposes an 8-hour TWA limit of 5 pm and a STEL of 10 ppm, with a skin notation, for dichloroethyl ether. The Agency preliminarily concludes that this combination of limits will protect workers against the risk of irritation, lung injury, and nausea associated with occupational exposure to elevated levels of dichloroethyl ether. The skin notation is proposed because dichloroethyl ether can cause systemic toxicity if percutaneously absorbed. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for dichloroethyl ether if the Agency determines that this limit will substantially reduce significant risk.

2,2-DICHLOROPROPIONIC ACID
CAS: 75-99-0; Chemical Formula: CH_3CCl_2COOH
H.S. No. 1130

OSHA currently has no limit for 2,2-dichlororopionic acid. The ACGIH recommends a TLV of 1 ppm TWA for this liquid.

In a communication to the ACGIH (1986, p. 190), the Dow Chemical Company (1977) reported that 2,2-dichloropropionic acid is corrosive to the skin and can cause permanent injury to the eye. The oral LD_{50} in rats is between 0.7 and 1 g/kg. Seven-hour exposures to a saturated atmosphere of the acid vapor caused no ill effects in rats, and a 120-day study of dietary exposure in rats showed a no-effect level of 15 mg/kg/day (Dow Chemical Company, as cited in ACGIH 1986, p. 190).

Acute human exposures have been reported to cause mild to moderate skin, eye, respiratory, and gastrointestinal irritation. Minimal respiratory irritation was observed in workers exposed at concentrations of between 2 and 7 ppm (ACGIH 1986, p. 190).

OSHA is proposing a PEL of 1 ppm TWA for 2,2-dichloropropionic acid. The Agency preliminarily concludes that this limit will protect exposed workers from the risk of eye, respiratory, and gastrointestinal irritation at the levels permitted in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for 2,2-dichloropropionic acid. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

DIETHYLAMINE
CAS: 109-89-7; CHEMICAL FORMULA: $(C_2H_5)_2NH$
H.S. No. 1137

OSHA's current limit for diethylamine is 25 ppm as an 8-hour TWA. The ACGIH has established an 8-hour TLV-TWA of 10 ppm and a 15-minute STEL of 25 ppm for this substance, which is a colorless liquid with an ammonia-like odor.

Diethylamine is a strong irritant of the eyes, skin, and mucous membranes, and chronic sublethal exposures cause tracheitis, bronchitis, pneumonitis, and pulmonary edema (ACGIH 1986, p. 197). In rabbits, the dermal LD_{50} is 0.82 ml/kg, and instillation of solutions of 1 percent or greater into the eyes of rabbits caused corneal opacity (Sutton 1963). Direct contact of the skin with diethylamine causes necrosis (ACGIH 1986, p. 197). Rabbits exposed 7 hours/day, 5 days/week for 6 weeks to 50 or 100 ppm diethylamine survived; those exposed to 50 ppm showed marked lung and corneal irritation, and, occasionally, degeneration of the heart muscle (Brieger and Hodes 1951). In the animals exposed to 100 ppm, these changes were more severe, and the parenchymatous degeneration of the heart muscle was marked (Brieger and Hodes 1951).

OSHA is proposing an 8-hour TWA of 10 ppm and a STEL of 25 ppm for diethylamine. The Agency preliminarily concludes that this combined limit will protect workers from the risk of skin burns, corneal injury, pulmonary irritation, and skin, eye, and upper respiratory tract irritation potentially associated with exposures to this substance at the levels permitted by an 8-hour limit alone. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for diethylamine if the Agency determines that this limit will substantially reduce significant risk.

DIISOBUTYL KETONE
CAS: 108-83-8; Chemical Formula: $[(CH_3)_2CHCH_2]_2CO$
H.S. No. 1140

OSHA currently has an 8-hour limit of 50 ppm TWA for diisobutyl ketone. NIOSH recommends a 10-hour TWA of 25 ppm, and the ACGIH has established a TWA limit of 25 ppm for this clear liquid with a mild ether-like odor.

The primary health effects associated with exposure to diisobutyl ketone are eye, nose, and throat irritation, although experimental animals have shown some systemic effects. Diisobutyl ketone has a uniformly low acute toxicity by all routes of exposure. Rats and guinea pigs survived single exposures of from 7.5 to 16 hours to essentially saturated vapor (McOmie and Anderson 1949). Smyth and co-workers (1949) reported that five of six rats died after exposure to 2000 ppm for 8 hours; they also reported a percutaneous LD_{50} for rabbits of greater than 20 ml/kg. Direct application of diisobutyl ketone to rabbit skin was only mildly irritating, and no eye irritation was reported after instillation into the rabbit eye. The oral toxicity for the rat was reported as 5.8 g/kg (Smyth et al. 1949). Carpenter and Smyth (1946) reported a no-effect level for diisobutyl ketone of 125 ppm in rats and guinea pigs given thirty 7-hour exposures. At 250 ppm, the liver and kidney weights of female rats increased, and the liver weights of male guinea pigs decreased; at levels of 530 and 920 ppm, rats showed increased liver and kidney weights; and at 1650 ppm, increased mortality was noted (Carpenter and Smyth 1946).

Silverman, Schulte, and First (1946) reported eye irritation and complaints of objectionable odor in volunteer human exposures to concentrations above 25 ppm. No worker illnesses have been linked to diisobutyl ketone exposure (ACGIH 1986, p. 203)

OSHA proposes a TWA limit of 25 ppm for diisobutyl ketone. The Agency preliminarily concludes that this limit will protect workers against the risk of irritation that is asociated with workplace exposures to diisobutyl ketone levels greater than 25 ppm. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for diisobutyl ketone if the Agency determines that this limit will substantially reduce significant risk.

EPICHLOROHYDRIN
CAS: 106-89-8; Chemical Formula: C_3H_5ClO
H.S. No. 1158

OSHA currently has a limit on 5 ppm TWA, with a skin notation, for epichlorohydrin. the ACGIH has established a limit of 2 ppm TWA, also with a skin notation. NIOSH recommends reducing employee exposure to the lowest feasible limit, and considers epichlorohydrin a carcinogen.

In animals, epichlorohydrin is irritating and systemically toxic by all routes of exposure (Shell Chemical Corporation 1958, as cited in ACGIH 1986, p. 233). Fatalities are caused and central nervous system and respiratory

tract effects resulting from exposure to high concentrations.

In mice, single 30-minute exposures to 8300 ppm of epichlorohydrin vapor caused muscular paralysis and death from respiratory failure; similar results have been reported for dermal application of the liquid at 0.5 ml/kg in rats, and repeated oral administration at 0.1 mg/kg in mice (Shell Chemical Corporation 1980, as cited in ACGIH 1986, p. 233). At 32 ppm (7 hours/day, 5 days/week) for 91 days, rats failed to show normal weight gain, and at 16 ppm they showed increased kidney size (ACGIH 1986, p. 233). Gage (1959) confirmed these findings and demonstrated lung, liver, and kidney injury in rats from repeated 6-hour exposures at concentrations ranging from 17 to 120 ppm. No effects were observed by this author at 9 ppm. The oral LD_{50} in rats is reported as 260 mg/kg, and the dermal LD_{50} in rabbits is reported as 755 mg/kg (Lawrence 1972). A 4-hour exposure at a level of 250 ppm was fatal to rats (Carpenter 1949).

There have also been reports of carcinogenicity in mice from dermal application and subcutaneous injection (Van Duuren et al. 1974), as well as reproductive effects from injestion; mutagenic effects were observed in microbial systems and in the fruit fly (NIOSH 1976).

In humans exposed to concentrations above 100 ppm for brief periods, lung edema and kidney lesions have been reported (NIOSH 1976). Exposure at 20 ppm caused burning of eyes and nasal mucosa (Wexler 1971). Another exposure to an unknown concentration caused eye and throat irritation, nausea, dyspnea, bronchitis, and an enlarged liver (Schultz 1964). Painful irritation of subcutaneous tissues follows skin contact in humans (ACGIH 1986, p. 233).

OSHA proposes an 8-hour TWA limit of 2 ppm, with a skin notation, for epichlorohydrin. The Agency preliminarily concludes that this limit will protect workers from the risk of dermal, respiratory, liver, and kidney effects that are potentially associated with exposure to epichlorohydrin at elevated concentrations. The skin notation is retained because of this substance's capacity to penetrate the skin and cause toxicity. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for ephclorohydrin if the Agency determines that this limit will substantially reduce significant risk.

ETHYL BENZENE
CAS: 100-41-4, Chemical Formula C_8H_{10}
H.S. No. 1162

OSHA currently has an 8-hour TWA limit of 100 ppm for ethyl benzene. The ACGIH recommends a limit of 100 ppm TWA and a 15-minute STEL of 125 ppm for this colorless, flammable liquid with an aromatic odor.

Ethyl benzene is a skin and mucous membrane irritant. It is reported to be the most severe irritant of the benzene series (Oettel 1936). In rabbits, repeated dermal application of the liquid causes reddening, exfoliation, and blistering of the skin (Wolf et al. 1956). The acute toxicity of ethyl benzene is low, with death reported to occur at 10,000 ppm in guinea pigs exposed for a few minutes; at 5000 ppm, ethyl benzene was described as dangerous to life for those exposed for 30 to 60 minutes. Dying animals suffered intense congestion and edema of the lungs, as well as generalized visceral hyperemia (Yant, Schrenk, Waite, and Patty 1930). The narcotic dose in laboratory animals is reported to be 10,000 ppm (reached within 18 minutes) and narcosis is preceded by vertigo, unsteadiness, and ataxia (Yant, Schrenk, Waite, and Patty 1930).

Chronic inhalation exposures of guinea pigs, monkeys, rabbits, and rats at concentrations of from 400 ppm to 2200 ppm, 7 to 8 hours/day, 5 days/week for as long as 6 months produced no effects in these species, except that liver damage occurred, on the average, in animals exposed to 400 ppm for 186 days (Wolf et al. 1956).

Observations in humans suggest that intolerable eye and nose irritation occurs at 5000 ppm; immediate and severe eye irritation and tearing and moderate nose irritation is exhibited at 2000 ppm; and irritation that reportedly can be tolerated occurs at 1000 ppm. At 200 ppm, the vapor produces transient eye irritation (Gerarde 1963).

OSHA is proposing a PEL of 100 ppm TWA and a 15-minute STEL of 125 ppm for ethyl benzene. The Agency preliminarily concludes that both of these limits are required to protect those exposed from the risk of irritation associated with occupational exposure to ethyl benzene above the 100-ppm level even for a brief period. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for ethyl benzene if the Agency determines that this limit will substantially reduce significant risk.

ETHYL ETHER
CAS: 60-29-7; Chemical Formula: $C_2H_5OC_2H_5$
H.S. No. 1164

OSHA currently has a limit of 400 ppm TWA for ethyl ether. The ACGIH recommends the same time-weighted average limit, with a STEL of 500 ppm for a 15-minute period. Ethyl ether is a colorless, volatile, mobile liquid with a distinct odor and a burning, sweet taste. It is extremely flammable and is a severe fire and explosion hazard when exposed to heat or flame.

Ethyl ether causes narcosis and general anesthesia. Concentrations of 3.6 to 6.5 volumes percent in air are anesthetic to humans; 7- to 10-percent concentrations cause respiratory arrest, and concentrations greater than 10 percent are fatal (ACGIH 1986, p. 259). Repeated workplace exposures deliberately induced to produce the so-called "ether jag" have caused narcosis, exhaustion, headache, dizziness, sleepiness, excitation, and other psychic disturbances (Hake and Rowe 1963). In women, albuminuria and polycythemia may result (Browning 1965). Repeated exposure may cause skin desiccation; irritation of the mucous membranes and eyes occurs on contact with the liquid or after exposure to high concentrations of the vapor (Hake and Rowe 1973). Nelson and co-workers (1943) reported that workers began to experience nasal irritation at 200 ppm (Nelson, Ege, Ross et al. 1943). Henderson and Haggard calculated that the amount of ether absorbed by a man of average height at a concentration of 400 ppm would not cause intoxication. Armor (1950) observed that exposure effects occur only at levels of 500 ppm and above.

OSHA is proposing a PEL of 400 ppm TWA and a 15-minute STEL of 500 ppm for ethyl ether. The Agency preliminarily concludes that both of these limits are necessary to protect against the risk of narcosis and irritation potentially associated with excursions above the 8-hour TWA level. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for ethyl ether if the Agency determines that this limit will substantially reduce significant risk.

ETHYL MERCAPTAN
CAS: 75-08-1; Chemical Formula: C_2H_5SH
H.S. No. 1165

OSHA currently has a ceiling limit of 10 ppm for ethyl mercaptan. The ACGIH recommends a TLV of 0.5 ppm TWA. The NIOSH recommended exposure limit for this substance is 0.5 ppm as a 15-minute ceiling. Ethyl mercaptan is a colorless liquid with a persistent and penetrating leek-like odor.

Acute animal toxicity data concerning ethyl mercaptan are taken from a single study that reports the following findings. The 4-hour inhalation LC_{50} values in rats and mice are 2770 ppm and 4420 ppm,

respectively. In the rat, the intraperitoneal LD_{50} is reported to be approximately 450 mg/kg. One drop applied to rabbit eyes caused only slight irritation, but high concentrations of vapor caused considerable irritation within 15 minutes. Maximal sublethal intraperitoneal doses have been reported to induce deep sedation, with higher exposures causing restlessness, muscular incoordination, skeletal muscular paralysis, cyanosis, respiratory depression, coma, and death. Although inhalation tests showed no noteworthy pathology in rats, intraperitoneal injection caused lymphotic infiltration of the liver with occasional necrosis (Fairchild and Stokinger 1958).

In chronic inhalation studies of rabbits, rats, and mice, a 5-month exposure to 40 ppm caused minimal cardiovascular and other systemic effects (Blivona 1965).

Studies of human volunteers, exposed at 4 ppm for 3 hours daily for 5 to 10 days, have reported minimal effects. At this level, all subjects experienced altered taste and olfactory reactions, periodic nausea, mucous membrane irritation, and fatigue. Exposure to 0.4 ppm produced no unpleasant symptoms (ACGIH 1986, p. 262).

OSHA is proposing to reduce its current ceiling limit of 10 ppm for ethyl mercaptan to 0.5 ppm as a time-weighted average, to protect workers against the risk of nausea, fatigue, and irritation associated with exposure to concentrations well below the current PEL. The Agency preliminary concludes that the revised limit will substantially reduce this risk. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for ethyl mercaptan if the Agency determines that this limit will substantially reduce significant risk.

ETHYLENE GLYCOL
CAS: 107-21-1; Chemical Formula: CH_2OHCH_2OH
H.S. No. 1169

OSHA currently has no limit for ethylene glycol. The ACGIH recommends a ceiling limit of 50 ppm (approximately 125 mg/m^3) for this clear, colorless, odorless, and hygroscopic liquid.

Ethylene glycol poses virtually no exposure risk at room temperature because of its low vapor pressure; at elevated temperatures, however, exposures are possible and adverse effects have been reported as a result of exposure to mists.

In studies of rats, guinea pigs, rabbits, dogs, and moneys, Coon and colleagues (1970) reported that animals exposed over a 30-day period to concentrations at 10 or 57 mg/m^3 for 8 hours daily, 5 days per week, showed no adverse effects. Moderate to severe eye irritation did occur in rats and rabbits exposed at 12 mg/m^3 for 24 hours per day for 90 days. (Coon et al. 1970). Wiley and co-workers reported no ill effects in animals exposed to approximately 350 to 400 mg/m^3, 8 hours per day, for 16 weeks (Wiley, Hueper, and von Oettingen 1936).

Rowe concluded that daily exposure to 100 ppm of the vapor did not cause systemic or eye injuries (1962), although Troisi described nystagmus in overexposed workers (concentrations not reported) (1950). In a human inhalation study, Wills and collegues (1974) reported that volunteers exposed to the aerosol from 20 to 22 hours per day for 4 weeks, at an average concentration of 12 ppm, complained of throat irritation, mild headache, and lower back pain. Complaints were more pronounced when the concentration was raised to 140 mg/m^3 (50 ppm) for part of a day. Average concentrations of 80 ppm were found intolerable by the subjects, who reported a burning sensation in the throat and respiratory passages; irritation was also common at 60 ppm (1974).

Based on evidence of an occupational risk of severe throat and respiratory irritation associated with exposure to the vapor and mist, OSHA proposes a ceiling limit of 50 ppm for ethylene glycol; this level is just below the level at which clinical symptons were noted. The Agency preliminarily concludes that this limit will substantially reduce the risk associated with the uncontrolled exposures at the currently uncontrolled level. The health evidence forms a reasonable basis for proposing a new limit for ethylene glycol. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

ETHYLIDENE NORBORNENE
CAS: 16219-25-3; Chemical Formula: C_9H_{12}
H.S. No. 1171

OSHA currently has no limit for ethylidene norbornene. The ACGIH recommends a ceiling limit of 5 ppm. This colorless liquid reacts with oxygen.

In a range-finding study, five of six rats died following a 4-hour exposure to 4000 ppm 5-ethylidene-2-norbornene (Smyth, Carpenter, Weil et al. 1967). Other studies of longer duration have reported that exposures to 237 ppm for 7 hours per day, 5 days per week for 88 days, resulted in death for 21 of 24 rats. No deaths resulted from repeated exposures at 90 ppm, but renal lesions and enlarged livers were observed, liver lesions, testicular atrophy, and hydrothorax occurred at the 237-ppm level (Kinkead, Pozzani, Geary, and Carpenter 1971). Beagle does similarly exposed to 93 ppm for 89 days survived, but exhibited such effects as testicular atrophy, hepatic lesions, and slight blood changes. Less pronounced effects were seen after exposure to 61 ppm, but no effects were seen at 22 ppm (Kinkead, Pozzani, Geary, and Carpenter 1971).

Human volunteers exposed for 30 minutes to ethylidene norbornene concentrations of 11 ppm experienced eye and nose irritation; at 6 ppm, transient eye irritation occurred (ACGIH 1986, p. 261).

OSHA proposes a ceiling limit of 5 ppm to minimize the risk of irritation that has been documented to occur in occupational exposures to concentrations as low as 6 ppm for 30-minute periods. The Agency preliminarily concludes that this limit will reduce this risk substantially. The health evidence forms a reasonable basis for proposing a new limit for ethylidene norbornene. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

FURFURAL
CAS: 98-01-1; Chemical Formula: $C_5H_4O_2$
H.S. No. 1183

OSHA currently has an 8-hour TWA limit of 5 ppm, with a skin notation, for furfural. The ACGIH recommends a TLV-TWA of 2 ppm, also with a skin notation. Furfural is a colorless, oily liquid that turns rust-colored when exposed to air and light.

An inhalation exposure to 260 ppm of furfural was fatal to rats but not to mice or rabbits. A 4-week exposure of dogs to 130 ppm for 6 hours a day caused liver damage, but no adverse effects were observed at 63 ppm (AIHA 1965).

Bugyi and Lepold (1952) describe numbness of the tongue and oral mucosa, absence of a sense of taste, and labored breathing in workers exposed to furfural (at unspecified levels) in a poorly ventilated facility. Koreman and Resnik (1930) state that inhalations of from 1.9 to 14 ppm furfural caused headaches, itching throat, and eye irritation; Kuhn (1944) reported that exposure to furfural damages the eyesight in some individuals. NIOSH (1975) describes widespread eye and respiratory tract irritation in workers at a griding wheel plant exposed to furfural vapor at levels ranging from 5 to 16 ppm.

However, Dunlop and Peters (1953) report that a 15-year study of furfural use in the synthetic resin industry revealed that this substance is not hazardous to health in facilities that are adequately ventilated, and that only occasional individual sensitivity was found.

OSHA proposes a PEL of 2 ppm TWA, with a skin notation, for furfural. The Agency preliminarily concludes that these limits will protect workers against the risk of headaches and eye and respiratory irritation associated with exposure to furfural at the levels permitted by OSHA's existing PEL. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for furfural if the Agency determines that this limit will substantially reduce significant risk.

FURFURYL ALCOHOL
CAS: 98-00-0; Chemical Formula: $C_5H_6O_2$
H.S. No. 1184

OSHA's current limit for furfuryl alcohol is 50 ppm TWA. NIOSH (1979) has also recommended a 50-ppm limit as a 10-hour TWA. The ACGIH recommends a 10-ppm TLV–TWA, a 15-ppm TLV–STEL and a skin notation.

The basis for the ACGIH-recommended limits are two foundry studies in which furfuryl alcohol was released during core preparation. Apol (1973) reported no discomfort among workers exposed to 10.8 ppm furfuryl alcohol, but severe lacrimation occurred at 15.8 ppm. Formaldehyde was also present at a concentration of 0.33 ppm. Burton and Rivera (1972) found no irritation, headache, or dizziness among workers exposed to 8-hour TWA concentrations of 5 and 6 ppm, with excursions up to 16 ppm. The ACGIH concluded that a TLV–TWA of 10 ppm with a 15-ppm TLV–STEL would protect workers against irritation effects.

NIOSH (1979) also reviewed these studies, but concluded that it was unknown whether the lacrimation reported by Apol (1973) was caused by furfuryl alcohol, formaldehyde, or both combined. They also noted that the current OSHA limit (50 ppm) is five times lower than the concentration reported to cause no adverse effects in monkeys (Woods and Seevers 1954–56). NIOSH (1979) concluded that the 50-ppm limit should remain, since no information exists showing that this limit offers inadequate protection.

The 50-ppm REL is based on the hypothesis that severe lacrimation noted in a foundry study was due to the presence of formaldehyde. More serious effects than severe lacrimation would occur at this formaldehyde level, and therefore OSHA believes that the health effects observed at 15.8 ppm are caused by exposure to furfuryl alcohol. OSHA thus finds that the REL does not provide adequate protection and proposes a 10-ppm (TWA), 15-ppm (STEL), and a skin notation for this substance to reduce this risk. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for furfuryl alcohol if the Agency determines that this limit will substantially reduce significant risk.

GLUTARALDEHYDE
CAS: 111-30-8; Chemical Formula: OCH $(CH_2)_3$CHO
H.S. No. 1187

OSHA currently has no limit for glutaraldehyde. The ACGIH recommends a ceiling limit of 0.2 ppm. Glutaraldehyde is an aliphatic dialdehyde which forms colorless crystals.

Glutaraldehyde is strongly irritating to the nose, eyes, and skin (*Human Sensory Irritation Threshold of Glutaraldehyde Vapor* 1976, as cited in ACGIH 1986, p. 285) and can cause allergic contact dermatitis from occasional or incidental occupational exposure (Jordan et al. 1972). The rat oral LD_{50} has been variously reported as 250, 820, and 2380 mg/kg (Stonehill et al. 1965; Smyth 1963, as cited in Fassett 1981; NIOSH 1975). The dermal LD_{50} in the rabbit is 2560 mg/kg, and the 4-hour inhalation LD_{50} in the rat is 5000 ppm (NIOSH 1975).

Mice exposed to alkalinized glutaraldehyde at 8 and 33 ppm for 24 hours have showed marked nervous behavior and panting and washing of the face and limbs; those exposed to 33 ppm exhibited signs of toxic hepatitis at autopsy (Varpela et al. 1971).

In a study of cold-sterilizing operation in which the operator was exposed for 12 minutes to an activated 2-percent aqueous solution, a measurement of 0.38 ppm glutaraldehyde was taken in the operator's breathing zone; the operator and the investigators experienced severe eye, nose, and throat irritation as well as sudden headache at the end of this procedure (Schneider and Blejer 1973). Another study employing very precise methods of airborne concentration measurement reported the irritation response level for glutaraldehyde to be 0.3 ppm and the odor recognition threshold to be 0.04 ppm (Colwell 1976, as cited in ACGIH 1986, p. 285).

OSHA proposes a ceiling level of 0.2 ppm for glutaraldehyde. The Agency preliminarily concludes that this ceiling limit will prevent the risk of irritation to the eyes, nose, and throat potentially associated with exposure to this substance at the levels permitted in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for glutaraldehyde. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

HEXACHLOROCYCLOPENTADIENE
CAS: 77-47-4; Chemical Formula: C_5Cl_6
H.S. No. 1196

OSHA has no current limit for hexachlorocyclopentadiene. The ACGIH recommends a TLV–TWA of 0.01 ppm. Hexachlorocyclopentadiene is a yellow to amber-colored, nonflammable liquid with a pungent odor.

Hexachlorocyclopentadiene has a high order of acute toxicity in laboratory animals. Rabbits, mice, rats, and guinea pigs died from inhaling 89.5 percent of the vapor in air (Treon, Cleveland, and Cappel 1955). In 150 daily exposures of 7 hours each, rabbits, rats, and guinea pigs survived concentrations of 0.15 ppm, but a similar exposure was fatal to four of five mice. At approximately twice this concentration, mice, rats, and most rabbits died by or before the 25th exposure, but guinea pigs survived 30 exposures. The vapors caused tearing, labored respiration, and, at high concentrations, tremors. Treon and associates observed degenerative changes in the brain, heart, liver, adrenal glands, and kidneys; pulmonary irritation occurred in all species, even at the lowest concentration of 0.15 ppm. At higher concentrations, pulmonary edema, hyperemia, necrotizing bronchitis, and bronchiolitis were observed.

In humans, there are few data concerning hexachlorocyclopentadiene's toxicity. Irritation is known to occur, but the intolerable odor and eye irritation associated with exposure to hexachlorocyclopentadiene have discouraged prolonged exposures (McGilvray 1971, as cited in ACGIH 1986, p. 300).

OSHA is proposing an 8-hour TWA of 0.01 ppm for this severely toxic substance. The Agency preliminarily concludes that this limit will protect exposed workers against the risks of exposure to this acute toxin at the levels permitted in the absence of any OSHA limit. These risks include intense eye and pulmonary irritation and multiple organ damage. The health evidence forms a reasonable basis for proposing a new limit for hexachlorocyclopentadiene. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines

that this limit will substantially reduce significant risk.

HEXYLENE GLYCOL
CAS: 107-41-5; Chemical Formula: $(CH_3)_2$-$COHCH_2$-$CHOH$-CH_3
H.S. No. 1204

OSHA currently has no limit for hexylene glycol. The ACGIH recommends a ceiling limit of 25 ppm for this liquid, which has a mild, sweetish odor.

In mice, the LD_{50} is reported to be 3.8 ml/kg, and it is reported to be 4.79 g/kg in rats. A single dose of 2.0 ml/kg induced hypnosis in mice. Undiluted hexylene glycol instilled into the rabbit eye caused irritation and corneal injury (Smyth and Carpenter 1948).

The Shell Chemical Corporation has reported that oral administration of hexylene glycol can cause nervous system depression that is manifested by an initial state of excitation, followed by deep depression (Shell Chemical Corporation, as cited in ACGIH 1986, p. 309). When the liquid is applied to the skin, mild to moderate irritation occurs, although skin absorption does not. At high concentrations, hexylene glycol vapors evoke a strong sensory response: a 5-minute exposure at 1000 ppm produced eye irritation and throat and respiratory discomfort. At concentrations of 50 ppm for 15 minutes, slight eye irritation is reported (ACGIH 1986, p. 309).

OSHA is proposing a ceiling limit of 25 ppm for hexylene glycol to minimize the risk of neuropathy and irritation that may occur as a result of even brief excursions at the high concentrations permitted in the absence of any OSHA limit. The Agency preliminarily concludes that this limit will substantially reduce this significant risk. The health evidence forms a reasonable basis for proposing a new limit for hexylene glycol. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

HYDROGEN BROMIDE

CAS: 10035-10-6; Chemical Formula: HBr
H.S. No. 1206

The current OSHA PEL for hydrogen bromide is 3 ppm as an 8-hour TWA. The ACGIH recommends the same 3 ppm value as a ceiling limit not to be exceeded at any time during the working day. Hydrogen bromide (HBr) is a colorless, corrosive, non-flammable gas with an acrid odor.

Animal studies have demonstrated that hydrogen bromide has a considerably higher acute toxicity than hydrogen chloride (HCl) in mice and a somewhat higher acute toxicity than this chemical in rats (NIOSH 1977). In mice, the LC_{50} is 800 ppm HBr in air for 60 minutes (and 2500 HCl in air for 30 minutes); in rats, the LC_{50} is 2800 ppm HBr in air for 60 minutes (and 5000 ppm HCl in air for 30 minutes).

The chief toxic effect of hydrogen bromide in humans is primary irritation of the nose and throat. Irritation begins within several minutes at levels between 3 and 6 ppm. At 2 ppm, the odor of HBr is detectable, but no irritation is experienced (Connecticut State Department of Health, as cited in ACGIH 1986, p. 312). No chronic effects have been associated with exposure to hydrogen bromide.

Based on evidence that hydrogen bromide is a primary irritant without known chronic toxicity, OSHA is proposing a 3-ppm ceiling for this substance. The Agency preliminarily concludes that this limit will protect against the risk of primary irritation to which workers can be exposed at the current PEL. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for hydrogen bromide if the Agency determines that this limit will substantially reduce significant risk.

HYDROGEN FLUORIDE
CAS: 7664-39-3; Chemical Formula: HF
H.S. No. 1208

The current OSHA standard for hydrogen fluoride is 3 ppm as an 8-hour TWA. The ACGIH has established a ceiling limit of 3 ppm for this substance, and NIOSH recommends 3 ppm as a 10-hour TWA and a 15-minute ceiling of 6 ppm for hydrogen fluoride. Hydrogen fluoride is a fuming, colorless liquid; at temperatues above 19°C (66°F), it becomes a colorless gas.

Guinea pigs and rabbits survived 40 ppm for 41 hours, but exposure to 300 ppm for 2 hours or more was fatal (Machle, Thamann, Kitzmiller, and Cholak 1934). Animals exposed to 3 ppm hydrogen fluoride for 30 days showed no adverse effects (Ronzani 1909). Stokinger and co-workers (1949) reported that animals repeatedly exposed to 7 ppm on a daily basis exhibited mild respiratory tract irritation. One study by Largent (1961) demonstrated kidney, liver, and lung damage in laboratory animals repeatedly exposed to 17 ppm hydrogen fluoride. At 8.6 ppm, the pathologic changes seen in exposed animals were minor, except for lung damage in one dog (Largent 1961).

In studies with humans, Largent (1960, 1961) reported that volunteers exposed repeatedly to concentrations of hydrogen fluoride as high as 4.7 ppm for 6 hours/day for 10 to 50 days experienced irritation and burning of the eyes and nose, in addition to reddening of the skin, at concentrations above 3 ppm. Industrial experience has shown that direct contact of the skin with hydrogen fluoride results in severe burns that may have delayed onset but later develop into ulcers that eventually scar (Patty 1981). A report by Eagers (1969) described several industrial accidents in which workers died in a matter of hours after accidential splashing from ruptured containers of hydrogen fluoride (the cause of death was respiratory failure and cardiac arrest). Kleinfeld (1965) reported a fatal case of hydrogen fluoride poisoning that caused death from pulmonary edema.

NIOSH (1976) cites numerous studies that consistently show that long-term occupational exposures to hydrogen fluoride lead to fluorosis in workers. The NIOSH limit is based in part on a study by Derryberry, Bartholomew, and Fleming (1963) showing that the threshold limit for minimal increases in bone density caused by fluoride (fluorosis) is below 4.3 ppm of hydrogen fluoride.

The Agency is proposing an 8-hour TWA limit of 3 ppm and a 6-ppm 15-minute ceiling for hydrogen fluoride. These are the current NIOSH-recommended limits for this substance. OSHA preliminarily concludes that both a TWA and a STEL are required to protect exposed workers from the risk of fluorosis, eye burning, and skin and upper respiratory tract irritation potentially associated with exposure at the levels permitted by the TWA alone. The Agency believes that these limits will substantially reduce these risks. The health evidence forms a reasonable basis for proposing a new limit for hydrogen fluoride. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

2-HYDROXYPROPYL ACRYLATE
CAS: 999-61-1; Chemical Formula: $CH_2CHCOOCH_2CHOHCH_3$
H.S. No. 1211

OSHA currently has no limit for 2-hydroxypropyl acrylate. The ACGIH recommends a TLV-TWA limit of 0.5 ppm and a skin notation. 2-hydroxypropyl acrylate (HPA) is a liquid at room temperature.

In experimental animals, 2-hydroxypropyl acrylate has a high acute toxicity. The Dow Chemical Company (1977), as cited in ACGIH 1986, p. 320) has reported an oral LD_{50} for the rat of 0.25 g/kg, and a dermal LD_{50} in the

rabbit of approximately 0.25 mg/kg. In guinea pigs, direct contact with HPA caused severe eye burns and skin corrosion and sensitized some of the experimental animals. Rats exposed to a concentration 650 ppm HPA in air for 7 hours did not die. Longer-term inhalation studies (30 days for 2 hours/day/6 days/week) in rats, dogs, rabbits, and mice resulted in some irritation at 5 ppm (Dow Chemical Company, as cited in ACGIH 1986, p. 320).

OSHA is proposing an 8-hour TWA Limit of 0.5 ppm and skin notation. The Agency preliminarily concludes that these limits will protect exposed workers from the risk of irritant effects and skin and eye burns associated with exposure to 2-hydroxypropyl acrylate at the previously uncontrolled level. OSHA believes these limits will substantially reduce these risks. The health evidence forms a reasonable basis for proposing a new limit for 2-hydroxy-propyl acrylate. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

IRON SALTS (SOLUBLE)
CAS: 7705–08–0 (ferric chloride); 10421–48–4 (ferric nitrate); 10028–22–5 (ferric sulfate); 7758–94–3 (ferrous chloride); 7720–78–7 (ferrous sulfate); Chemical Formula: Varies with compound
U.S. No. 1217

OSHA currently has no limit for soluble iron salts. The ACGIH recommends a limit of 1 mg/m^3, as iron, for these substances.

When injected into the bloodstream of experimental animals, iron salts (especially the ferric salts) are highly toxic (ACGIH 1986, p. 328). The acute intravenous dose of ferric chloride that is lethal to rabbits is about 7.2 mg/kg (Drinker, Warren, and Page 1935). The salts are also considered irritants to the respiratory tract when inhaled as dusts and mists (Drinker and Nelson 1944). Stewart and Faulds (1934) described the ferric salts as skin irritants. The oral toxicities of iron salts are considered to be moderate to low, and marked gastrointestinal irritation results from ingestion (U.S. Dept. of Labor 1941); 30 grams is the estimated fatal dose for humans (Smyth 1956).

OSHA is proposing an 8-hour TWA PEL of 1 mg/m^3, as iron, for the soluble salts of iron. The Agency preliminarily concludes that this limit will protect exposed workers from the risk of skin and mucous membrane irritation associated with exposure to these salts at the existing uncontrolled levels. The health evidence forms a reasonable basis for proposing a new limit for iron salts. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

ISOPROPYL ACETATE
CAS: 108–21–4; Chemical Formula: $CH_3COOCH(CH_3)_2$
H.S. No. 1224

OSHA currently has a 250 ppm TWA limit for isopropyl acetate. The ACGIH recommends a TLV-TWA of 250 ppm and a 15-minute STEL of 310 ppm for this colorless liquid, which has a fruity odor.

The oral LD_{50} for rats is reported to be 6.75 g/kg; five of six rats died after a 4-hour exposure to 32,000 ppm, and one of six rats died after a 4-hour exposure to 16,000 ppn (Smyth, Carpenter, West, and Pozzani 1954).

The primary problems in occupational exposures to isoproply acetate are eye and mucous membrane irritation. In humans, exposure to 200 ppm isoproply acetate caused eye irritation, with nose and throat irritation occurring at higher concentrations (Silverman, Schulte, and First 1946). Data show that isopropyl acetate is more similar to ethyl acetate than to n-propyl acetate in its toxic-effects (von Oettingen 1960).

OSHA proposes an 8-hour TWA PEL of 250 ppm and a STEL of 310 ppm for isopropyl acetate. The Agency preliminarily concludes that both a TWA and a STEL are necessary to protect exposed workers from the risk of eye and respiratory irritation associated with elevated exposure to this substance. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for isopropyl acetate if the Agency determines that this limit will substantially reduce significant risk.

ISOPROPYL ALCOHOL
CAS: 67–63–0; Chemical Formula: $CH_3CHOHCH_3$
H.S. No. 1225

OSHA currently has a limit of 400 ppm TWA for isopropyl alcohol. The ACGIH recommends a TLV-TWA limit of 400 ppm, with a 500-ppm TLV-STEL. NIOSH has recommended a limit of 400 ppm TWA, with a 15-minute ceiling of 800 ppm. Isopropyl alcohol is a colorless, flammable liquid with a slight odor resembling that of rubbing alcohol.

Rats exposed at concentrations of 12,000 ppm for 4 hours survived, but extending the duration of exposure to 8 hours killed the animals (Smyth, unpublished results, 1937–1955, as cited in ACGIH 1986, p. 337).

Isopropyl alcohol has been demonstrated to be mildly irritating to the eyes, nose, and throat in humans exposed to 400 ppm (Nelson et al. 1943); at 800 ppm, these symptoms were more intense. In addition, it has narcotic, irritative, and acute toxic effects at higher concentrations. Weil has reported that an excess of paranasal sinus cancers has been observed among workers manufacturing isopropyl alcohol (1952). It has been established that the cancers associated with isopropyl alcohol manufacture were caused by isopropyl oil and not by isopropyl alcohol itself (NIOSH 1976).

OSHA is proposing a PEL of 400 ppm TWA and a STEL of 500 ppm for isopropyl alcohol. The short-term limit is being added to reduce the risk of irritation and narcotic effects at the higher short-term concentrations permitted by the TWA alone. The Agency preliminarily concludes that the addition of the STEL will reduce this risk. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for isopropyl alcohol if the Agency determines that this limit will substantially reduce significant risk.

n-ISOPROPYLAMINE
CAS: 75–31–0; Chemical Formula: $(CH_3)_2CHNH_2$
H.S. No. 1228

OSHA currently has a limit of 5 ppm TWA for n-isopropylamine. The ACGIH recommends a TLV-TWA of 5 ppm and a TLV-STEL of 10 ppm for this flammable, volatile, colorless liquid, which has an odor similar to that of ammonia.

The most serious effect of n-isopropylamine in laboratory animals is respiratory tract irritation, which can be severe enough to cause lung edema. Rats survived a 4-hour inhalation at 4000 ppm, but an 8000-ppm exposure resulted in fatalities (Smyth, Carpenter, and Weil 1956).

Proctor and Hughes (1978) have reported that the odor of n-isopropylamine becomes strong and unpleasant at the 10-to 20-ppm level; nose and throat irritation is experienced even as a result of brief exposures.

OSHA is proposing a PEL of 5 ppm TWA and a 15-minute STEL of 10 ppm for this substance. The Agency preliminarily concludes that both a TWA and STEL are required to protect exposed workers from the risk of upper respiratory tract irritation known to occur even at brief excursions above the 8-hour PEL. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for n-isopropylamine if the Agency

determines that this limit will substantially reduce significant risk.

MESITYL OXIDE
CAS: 141–79–7; Chemical Formula: $(CH_3)_2C=CHCOCH_3$
H.S. No. 1243

OSHA's current limit for mesityl oxide is 25 ppm TWA. The ACGIH has recommended a 15-ppm TLV-TWA and a 25-ppm TLV-STEL. NIOSH (1978j) recommended a 10-ppm limit as a 10-hour TWA.

Silverman et al. (1946) found that a majority of test subjects experienced eye irritation upon exposure to 25 ppm mesityl oxide and nasal irritation at 50 ppm. A toxicity data sheet published by Shell Chemical Corporation (cited in ACGIH 1986, p. 361) confirmed 25 ppm as being the maximum comfort level. Smyth et al. (1942) reported liver and kidney damage among rats and guinea pigs exposed to 100 ppm mesityl oxide for 6 weeks; no adverse effects were reported for animals exposed to 50 ppm. After reviewing these data, the ACGIH (1986, p. 361) concluded that the former TLV of 25 ppm should be reduced to 15 ppm because of the greater systemic toxicity of mesityl oxide compared to other saturated ketones. NIOSH (1978j), relying on the same data, recommended a limit of 10 ppm as a 10-hour TWA and noted that the eye irritation caused by mesityl oxide may be more serious than that caused by lower ketones. This belief was based on the observation that eye irritation generally becomes more severe as the molecular weight of the ketone increases.

Studies indicate that eye irritation occurs following exposure to 25 ppm of mesityl oxide, and nasal irritation is experienced at the 50-ppm level. Animal studies show liver and kidney damage in experimental animals exposed to 100 ppm. OSHA proposes that a PEL of a 15-ppm TWA and a 25-ppm STEL be adopted as a necessary and more protective limit than the REL of 10 ppm TWA to provide protection against these occupational risks.

METHYL 2–CYANOACRYLATE
CAS: 137–05–3; Chemical Formula: $CH_2=C(C=N)COOCH_3$
H.S. No. 1248

OSHA has no current limit for methyl 2-cyanoacrylate. The ACGIH recommends a limit of 2 ppm TLV–TWA with a TLV–STEL of 4ppm. Methyl 2-cyanoacrylate is a colorless, viscous liquid.

In a personal communication to the ACGIH TLV Committee in 1985, Eastman Kodak reported on the toxicity of methyl 2-cyanoacrylate in experimental animals. The oral LD_{50} in rats is reported to be 1.6 to 3.2 g/kg, and the dermal LD_{50} in guinea pigs is 10 ml/kg. The adverse effects reported in laboratory animals are slight irritation of the skin and corneal damage. An inhalation LC_{50} or 101 ppm has been reported in rats exposed for 6 hours to methyl 2-cyanoacrylate. Repeated exposures (6 hours/day for 5 days/week) at 31.3 ppm for a total of 12 exposures caused only a slight decrease in the rate of weight gain in rats and no nasal or tracheal lesions or systemic toxicity. No changes were observed in rats similarly exposed at 3.1 ppm (Eastman Kodak, as cited in a CGIH 1986, p. 383).

In a simulated workbench exposure, McGee and co-workers reported nasal irritation in humans at 3 ppm and eye irritation at 5 ppm (McGee, Oglesby, Raleigh, and Fassett 1968). There are no reports of occupational poisonings.

OSHA proposes of PEL of 2 ppm TWA and a 4-ppm STEL for this previously unregulated substance. The Agency preliminarily concludes that both of these limits are necessary to protect against the risk of eye, skin, and upper respiratory tract irritation in workers exposed at the uncontrolled levels permitted by the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for methyl 2-cyanoacrylate. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit substantially reduce significant risk.

METHYL ISOBUTYL CARBINOL
CAS: 108–11–2; Chemical Formula: $CH_3CHOHCH_2CH(CH_3)_2$
H.S. No. 1261

OSHA currently has an 8-hour TWA limit of 25 ppm, with a skin notation, for methyl isobutyl carbinol. The ACGIH recommends a TLV–TWA of 25 ppm and a TLV–STEL of 40 ppm, also with a skin notation. Methyl isobutyl carbinol is a colorless, stable liquid.

In rabbits, a 24-hour skin application of 3.56 ml/kg was lethal to half the animals, indicating toxic absorption through the skin (Smyth, Carpenter, and Weil 1951). Rats exposed by inhalation to 2000 ppm of methyl isobutyl carbinol vapor died, and the same authors report that the oral LD_{50} for rats is 2.6 g/kg (Smyth, Carpenter, and Weil 1951).

Humans volunteers exposed to methyl isobutyl carbinol reported eye irritation at 50 ppm (Silverman, Schulte, and First 1946).

OSHA proposes an 8-hour TWA of 25 ppm and a 15-minute STEL of 40 ppm for methyl isobutyl carbinol. The Agency preliminarily concludes that these limits will together protect workers from the risk of eye irritation potentially associated with exposure to this substance in the absence of a STEL. OSHA is retaining the skin notation for methl isobutyl carbinol because of its demonstrated dermal toxicity. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for methyl isobutyl carbinol if the Agency determines that this limit will substantially reduce significant risk.

METHYL MERCAPTAN
CAS: 74–93–1; Chemical Formula: CH_3SH
H.S. No 1263

OSHA currently has a ceiling limit of 10 ppm for methyl mercaptan. The ACGIH recommends an 8-hour TLV–TWA of 0.5 ppm, and NIOSH recommends a 15-minute ceiling of 0.5 ppm. Methyl mercaptan is a flammable, water-soluble gas with a disagreeable odor like that of rotten cabbage.

Methyl mercaptan acts on the respiratory center, producing death by respiratory paralysis. DeRekowski (1893) and Frankel (1921) have reported that the acute toxicity of methyl mercaptan is similar to but somewhat lower than that of hydrogen sulfide; however, Ljunggren and Norberg (1943) have concluded that the two substances exhibit toxicities of the same magnitude. Pulmonary edema results from exposures to lower, less actue concentrations of methyl mercaptan (Fairchild, as cited in ACGIH 1986, p. 405).

Inhalation of (an unspecified concentration of) methyl mercaptan produced coma and death in one worker; acute hemolytic anemia and methemoglobinemia developed after this exposurer (Schultz, Fountain, and Lynch 1970).

OSHA is proposing a PEL of 0.5 ppm as an 8-hour TWA for methyl mercaptan. The Agency believes that this limit is necessary to reduce the risk of respiratory and pulmonary injury that exists for workers exposed to higher concentrations. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for methyl mercaptan if the agency determines that this limit will substantially reduce significant risk.

METHYL n-AMYL KETONE
CAS: 110–43–0; Chemical Formula: $CH_{23}COC_6H_{11}$
H.S. No. 1264

The current OSHA limit for methyl n-amyl ketone is 100 TWA. NIOSH (1978j) also recommends a limit of 100 ppm as a 10-hour TWA, and the ACGIH recommends a TLV–TWA of 50 ppm.

Johnson et al. (1978) found no neurologic impairment in rats and monkeys exposed to 131 ppm or 1025 ppm methyl n-amyl ketone for 9 months. No gross or histopathologic changes were found. Because of the absence of any human data indicating the concentration of methyl n-amyl ketone that produces sensory irritation, ACGIH (1986) believed in prudent to reduce the TLV-TWA from 100 ppm to 50 ppm. NIOSH (1978j) concluded that there was no basis for revising the 100-ppm OSHA limit; they cited evidence that methyl n-amyl ketone was likely to be as irritating as 2-pentanone (which has a recommended limit of 150 ppm), and therefore the 100-ppm limit was probably sufficient for methyl n-amyl ketone.

No neurological or histopathological effects were observed at 131 ppm. The ACGIH's 50-ppm TLV applies an additional factor of safety to this no-observed-effect level, while the NIOSH REL is based on a judgment hat such a reduction is unnecessary. The health evidence for methyl n-amyl ketone may not be sufficient to support a revision to the existing limit, and OSHA accordingly is retaining the Z table limit. However, there may be other information on the health effects of occupational exposure to methyl n-amyl ketone, and OSHA is specifically requesting such information from the public. At the time of the final rule, OSHA will make a final determination, based on the best available evidence, of whether to retain or revise the limit for this substance.

a-METHYL STYRENE
CAS: 98-83-9; Chemical Formula: $C_6H_5C(CH_3)=CH_2$
H.S. No. 1267

OSHA currently has a ceiling limit of 100 ppm for a-methyl styrene. The ACGIH recommends a limit of TLV-TWA 50 ppm with a TLV-STEL of 100 ppm a-Methyl styrene is a polymerizable, colorless liquid.

OSHA's existing ceiling limit of 100 ppm is based on data developed in 1955 by the Dow Chemical Company (as cited in ACGIH 1986, p. 410) and by Wolf, Rowe, McCollister et al (1956). These data demonstrated that 7 hours/day, 5 days/week exposures to a-methyl styrene for 6 months produced no ill effects in rats, guinea pigs, rabbits, or monkeys.

In humans, however, these authors reported that a 2-minute exposure to 200 ppm caused eye irritation and complaints about this substance's unpleasant odor.

OSHA is proposing an 8-hour TWA of 50 ppm and a 15-minute STEL of 100 ppm for a-methyl styrene. The Agency preliminarily concludes that these limits will protect workers from the risk of eye irritation potentially associated with exposure to this substance at the current OSHA PEL. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for a-methyl styrene if the Agency determines that this limit will substantially reduce significant risk.

o-METHYLCYCLOHEXANONE
CAS: 583-60-8; Chemical Formula: $CH_3C_6H_9CO$
H.S. No. 1270

OSHA currently has a limit of 100 ppm TWA for o-methylcyclohexanone with a skin notation. The ACGIH recommends lowering this limit to TLV-TWA 50 ppm and adding a TLV-STEL of 75 ppm; ACGIH also recommends a skin notation. ortho-Methylcyclohexanone is a somewhat viscous liquid with an acetone-like odor.

o-Methylcyclohexanone has both irritative and narcotic effects at relatively low concentrations. The commercial product contains a mixture of isomers; however, toxicity data describe effects of the ortho isomer only. Gross reported that 450 ppm had irritative effects on the eyes and respiratory systems of rabbits, and 2500 ppm produced narcotic effects (1943). Treor et al. reported the oral LD_{50} to be between 1 and 1.25 g/kg for rabbits. Eye problems were observed at about 500 ppm, but 182 ppm showed no adverse effects (1943).

Patty (1943) has reported that concentrations of 100 ppm have no narcotic effects in humans, but may cause irritation.

OSHA concludes that the current TWA of 100 ppm is insufficient to prevent eye and respiratory system irritation. A TWA limit of 50 ppm is proposed with a STEL of 75 ppm for o-methylcyclohexanone and a skin notation. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for o-methylcyclohexanone if the Agency determines that this limit will substantially reduce significant risk.

OSMIUM TETROXIDE
CASE: 20816-12-0; Chemical Formula: OsO_4
H.S. No. 1298

OSHA currently has a limit of 0.002 mg/m³ for osmium tetroxide. The ACGIH recommends a TLV-TWA of 0.0002 ppm (0.002 mg/m³) and a TLV-STEL of 0.0006 ppm (0.006 mg/m³). Osmium tetroxide is a noncombustible, colorless to pale yellow solid with a disagreeable, chlorine-like odor.

Exposure to osmium tetroxide is known to produce ocular effects and respiratory irritation. In 1933, Brunot reported that rabbits died from pulmonary edema four days after a 30-minute exposure to osmium tetroxide at 130 mg/m³ or higher. Visual problems (e.g. delayed lacrimation and "halo" effects) were reported by this investigator after a brief exposure to osmium tetroxide at a significantly lower concentration (Brunot 1933). A 4-hour LC_{50} value of 40 ppm has been reported in rats and mice (NIOSH 1977). Toxic effects to bone marrow have been reported in guinea pigs (Hardy 1974).

Industrial experience indicates that concentrations in a precious metal refining plant ranged from 0.1 to 0.6 mg/m³; intermittent exposures produced symptoms (sometimes delayed) of lacrimation, vision disturbances, headache, conjunctivitis, and cough (McLaughlin, Milton, and Perry 1946). Complaints of persistent and severe nose and throat irritation have been reported (Hardy and Hamilton 1974). Fairhall (1949) reported a human fatality resulting from inhalation exposure to OsO_4. Flury and Zernik (1931) reported that 0.001 mg/m³ is the highest concentration of osmium tetroxide that can be tolerated for 6 hours without harmful effects.

OSHA is proposing a TWA limit of 0.0002 ppm and a STEL of 0.0006 ppm for osmium tetroxide. The Agency preliminarily concludes that this combined limit will protect workers against the risk of ocular disturbances and respiratory irritation associated with exposure to this substance at the levels permitted in the absence of a short-term limit. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for osmium tetroxide if the Agency determines that this limit will substantially reduce significant risk.

PARAFFIN WAX FUME
CAS: 8002-74-2; Chemical Formula: C_nH_{2n+2}
H.S. No. 1302

OSHA currently has an 8-hour TWA limit for paraffin wax fume. The ACGIH recommends a TLV-TWA of 2 mg/m³. Paraffin is a white or slightly yellow odorless solid that is derived from petroleum.

Paraffin is considered nontoxic in its solid state, but fume generated when it is in the molten state may cause discomfort and nausea (Journal of American Medical Association, cited in ACGIH 1986, p. 455). In the most recent report of industrial exposure effects, paraffin fume is reported to cause no

discomfort in most cases when the concentration is maintained at or below 2 mg/m³, although one instance of mild discomfort was reported at concentrations between 0.6 and 1 mg/m³ (Massachusetts Division of Occupational Hygiene 1970, cited in ACGIH 1986, p. 455).

OSHA is proposing a PEL of 2 mg/m³ TWA for paraffin wax fume. The Agency preliminarily concludes that this limit will protect workers against the risk of nausea and irritation that exists in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for paraffin wax fume. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

PHOSPHORIC ACID
CAS: 7664-38-2; Chemical Formula: H_3PO_4
H.S. No. 1322

OSHA currently has a limit of 1 mg/m³ as an 8-hour TWA for phosphoric acid. The ACGIH recommends a TLV-TWA of 1 mg/m³ and a TLV-STEL of 3 mg/m³. Phosphoric acid is a colorless, odorless solid at temperatures below 21°C, but it becomes a viscous, clear liquid at higher temperatures.

In humans, there have been reports of respiratory irritation from exposure to phosphorus pentoxide fume at concentrations of between 3.6 and 11.3 mg/m³; concentrations of 100 mg/m³ were unendurable except to workers who had developed a tolerance to the fume over time (Rushing 1957, as cited in the ACGIH 1986, p. 483). The AIHA Hygiene Guide reports that phosphoric acid is less hazardous than nitric or sulfuric acid (1957).

To protect unacclimatized workers from the risk of throat irritation, OSHA proposes a TWA limit of 1 mg/m³, with a STEL of 3 mg/m³, for phosphoric acid. The Agency preliminarily concludes that the combined 8-hour TWA and STEL limit is necessary to reduce this risk of irritation, which has been shown to occur at levels only slightly above those permitted by the TWA alone. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for phosphoric acid if the Agency determines that this limit will substantially reduce sigificant risk.

PHOSPHORUS TRICHLORIDE
CAS: 7719-12-2; Chemical Formula: PCl_3.
H.S. No. 1325

OSHA currently has an 8-hour TWA limit of 0.5 ppm for phosphorus trichloride. The ACGIH recommends a TLV-TWA of 0.2 ppm and a TLV-STEL of 0.5 ppm for this fuming, colorless, noncombustible liquid.

The primary occupational hazards associated with exposure to phosphorus trichloride are respiratory irritation and intoxication involving cough, bronchitis, pneumonia, and conjunctivitis (Henderson and Haggard 1943; International Labour Office 1934; Sassi 1954).

Early studies indicate that severe symptoms did not occur in cats and guinea pigs until concentration levels reached 50 to 90 ppm for exposures lasting 1 hour, although slight illness was observed at 0.7 ppm after an exposure of 6 hours (Butjagin 1904). However, by 1934, the effects of phosphorus trichloride were considered to be 5 to 10 times as intense as those of hydrolyzed hydrochloric acid (International Labour Office 1934). More recently, Weeks and associates (1964) reported studies in which 4-hour LC_{50} values of 104 ppm for rats and 50 ppm for guinea pigs were obtained.

OSHA is proposing a PEL of 0.2 ppm TWA and a STEL of 0.5 ppm for phosphorus trichloride. The Agency preliminarily concludes that both a TWA and a STEL are required to reduce the risk of respiratory and eye irritation that exists for workers exposed to this substance. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for phosphorus trichloride if the Agency determines that this limit will substantially reduce significant risk.

POTASSIUM HYDROXIDE
CAS: 1310-58-3; Chemical Formula: KOH
H.S. No. 1334

OSHA has no current limit for potassium hydroxide. The ACGIH recommends a ceiling limit of 2 mg/m³. Potassium hydroxide is a white, deliquescent material that occurs in the form of pellets, sticks, lumps, or flakes.

Potassium hydroxide is corrosive to tissues. The health hazards of potassium hydroxide are similar to those of the other strong alkalies, such as sodium hydroxide. These substances gelatinize tissue on contact, causing deep, painful lesions. Dust or mist exposures may cause eye or respiratory system irritation and nasal septum lesions (Kanpov 1971).

OSHA is proposing an 8-hour TWA limit of 2 mg/m³ for this previously unregulated substance. The Agency preliminarily concludes that this limit will protect workers against the risk of respiratory irritation and severe dermal lesions that exists in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for potassium hydroxide. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

ROSIN CORE SOLDER PYROLYSIS PRODUCTS
CAS: None; Chemical Formula: None
H.S. No. 1350

OSHA currently has no limit for rosin core solder pyrolysis products. The ACGIH has established an 8-hour TLV-TWA of 0.1 mg/m³ for these compounds, measured as formaldehyde. This limit applies to the thermal decomposition products of gum rosin soldering flux (3 to 6 percent rosin and 30 to 700 percent tin-lead solder)(Lozano and Melvin, as cited in ACGIH 1986, p. 514).

A 2-week exposure of guinea pigs and rats to these products at average concentrations of 0.96 mg/m³ caused reduction in rate of weight gain in male guinea pigs, abnormal liver-to-body-weight ratios in guinea pigs of both sexes, and abnormal heart-to-body-weight ratios in male rats (Industrial Bio-test Lab, Inc., as cited in ACGIH 1986, p. 514). Lungs of the animals exposed in this same study were hyperemic.

In humans, slight bronchial irritation has been reported at 1 mg/m³ (Industrial Bio-test Lab, Inc., as cited in ACGIH 1986, p. 514). Several workers chronically exposed to levels as high as 0.15 mg/m³ had to be removed from exposure because of intractable upper respiratory tract irritation; when concentrations were kept below 0.1 mg/m³, such irritation was not reported (Christy 1965, as cited in ACGIH 1986, p. 514). In a study designed to quantify dose-response levels for irritation in human volunteers, subjects were exposed for 15 minutes to these products at aldehyde concentrations (as formaldehyde, which is the best indirect measure of rosin pyrolysis products) of 0.04 to 0.2 mg/m³ (U.S. Public Health Service 1965, as cited in ACGIH 1986, p. 514). Subjects detected the odor at 0.07 mg/m³, and 80 percent of subjects reported moderate to severe irritation of the eyes, nose, and throat at a concentration of 0.12 mg/m³ or above. At levels below 0.05 mg/m³, fewer than 10 percent of subjects experienced irritation. Mucous membrane irritation occurred in 30 percent of subjects exposed at 0.07 mg/m³. Some subjects reported severe irritation at 0.1 mg/m³ (U.S. Public Health Service 1965, as cited in ACGIH 1986, p. 514).

OSHA is proposing an 8-hour TWA limit of 0.1 mg/m³, measured as formaldehyde, for rosin core solder

pyrolysis products. The Agency preliminarily concludes that this limit will protect workers from the risk of eye, skin, and upper respiratory tract irritation demonstrated to occur at the higher levels permitted in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for rosin core solder pyrolysis products. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

SODIUM BISULFITE
CAS: 7631-90-5; Chemical Formula: $NaHSO_3$
H.S. No. 1365

OSHA's current Z tables have no limits for exposure to sodium bisulfite. The ACGHIH limit is 5 mg/m³ as an 8-hour TWA. Sodium bisulfite is a white crystalline powder, which has an odor like that of sulfur dioxide.

The oral LD_{50} in rats fed this substance is 2 g/kg (Dow Chemical Company 1977, as cited in ACGIH 1986, p. 534), and the intraperitoneal LD_{50} for rats is 115 mg/kg (Hoppe and Goble 1951). The ACGIH reports that sodium bisulfite is an eye, skin, and mucous membrane irritant; acute exposures have resulted in mild eye and respiratory effects (ACGIH 1986, p. 534).

OSHA is proposing an 8-hour TWA limit for sodium bisulfite, a level half that for the inert dusts, because the Agency has preliminarily concluded that this limit will protect exposed workers against the risk of eye, skin, and upper respiratory tract irritation potentially associated with exposure to this dust at the uncontrolled levels permitted in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for sodium bisulfite. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

SODIUM HYDROXIDE
CAS: 1310-93-2; Chemical Formula: NaOH
H.S. No. 1367

The current OSHA standard for sodium hydroxide is 2 mg/m³ as an 8-hour TWA. NIOSH recommends a 2 mg/m³ 15-minute ceiling for sodium hydroxide and the ACGIH has established a 2-mg/m³ ceiling limit (maximum duration of 5 minutes in any 3 hours). Sodium hydroxide is a white, deliquescent solid.

Exposure to sodium hydroxide in the form of a caustic dust irritates the upper respiratory tract any may cause ulceration of the nasal passages (ACGIH 1986, p. 535). NIOSH states that rats exposed to unmeasured concentrations of sodium hydroxide for 30 minutes per day developed pulmonary damage after 2 to 3 months (Dluhos et al. 1969, as cited in NIOSH 1976).

Patty (1977) reported that a concentration of 2 mg/m³ sodium hydroxide represents a level that is noticeably irritating to exposed workers. Hervin et al. (1973) noted marked redness and burning sensations in the eyes, throats, and noses of workers exposed to concentrations of sodium hydroxide in the range of 0.005 to 0.7 mg/m³. A study by Lewis (1974, as cited in ACGIH 1986, p. 535) noted throat irritation or eye watering in those employees exposed briefly to concentrations of 0.24, 0.8, or 1.86 mg/m³ of sodium hydroxide; only those workers exposed to 0.24 mg/m³ showed no irritation.

The Agency preliminarily concludes that a 2-ppm ceiling is required to protect exposed workers against the risk of eye and upper respiratory irritation that has been shown to exist at the current 2-mg/m³ TWA. OSHA is proposing a ceiling limit of 2 mg/m³ for sodium hydroxide. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for sodium hydroxide if the Agency determines that this limit will substantially reduce significant risk.

SODIUM METABISULFITE
CAS: 7681-57-4; Chemical Formula: $Na_2S_2O_5$
H.S. No. 1368

OSHA's current Z tables have no exposure limits for sodium metabisulfite. The ACGIH limit is 5 mg/m³ as an 8-hour TWA. Sodium metabisulfite can occur either in the form of a solid or as white crystals; this substance smells like sulfur dioxide.

A 2-year study at the Dow Chemical Company (1977, as cited in ACGIH 1986, p. 535), in which rats ingested 0.215 percent sodium metabisulfite, demonstrated no adverse effects in the rats. If the results of this study are extrapolated to humans, using a safety factor of 10, the equivalent air concentration would be 70 mg/m³ (Dow Chemical Company 1977, as cited in ACGIH 1986, p. 535). Other animal studies show a median lethal dose of 192 mg/kg for rabbits and 115 mg/kg for rats when sodium metabisulfite is injected intravenously (NIOSH 1973). Inhalation of sodium metabisulfite dust is irritating to the lungs, nose, and throat (ACGIH 1986, p. 535).

OSHA is proposing an 8-hour TWA of 5 mg/m³ for sodium metabisulfite. The Agency preliminarily concludes that establishing this limit is necessary and will reduce the risk of irritation of the skin and eyes to which workers are exposed in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for sodium metabisulfite. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

SULFUR MONOCHLORIDE
CAS: 10025-67-9; Chemical Formula: S_2Cl_2
H.S. No. 1376

OSHA's existing PEL for sulfur monochloride is 1 ppm as an 8-hour TWA. The level established by the ACGIH is 1 ppm as a ceiling limit. Sulfur monochloride is an amber, oily, nonflammable, fuming liquid, which has a penetrating odor.

Sulfur monochloride is a primary irritant that affects the upper respiratory tract by releasing hydrochloric acid (HCl) on contact with moisture (Henderson and Haggard 1943). This same study noted that "undecomposed vapor [of sulfur monochloride] might reach the lungs, in which case it would be more toxic than an equivalent quantity of HCl." The ACGIH (1986, p. 545) considers these data indicative of a far greater acute toxicity for sulfur monochloride than for hydrochloric acid. Animal toxicity studies revealed that a dose of 150 ppm sulfur monochloride resulted in death to mice exposed for 1 minute (Flury and Zernik 1931). Cats exposed to 60 ppm sulfur monochloride for 15 minutes all died within a few days, but concentrations of 12 ppm for 15 minutes were tolerated (Henderson and Haggard 1943).

A study by Elkins (1959) of workers in the rubber industry found that concentrations of 2 to 9 ppm sodium monochloride were mildly irritating; however, the concentrations to which these workers were exposed may have included a high proportion of hydrochloric acid.

OSHA is proposing a ceiling of 1 ppm for sulfur monochloride because this substance is a primary irritant. The Agency preliminarily concludes that this limit will protect exposed workers against the risk of primary irritation that could occur at the current 8-hour TWA limit. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for sulfur monochloride if the Agency determines that this limit will substantially reduce significant risk.

SULFUR PENTAFLUORIDE
CAS: 5714-22-7?; Chemical Formula S_2F_{10}
H.S. No. 1377

The current OSHA limit for sulfur pentafluoride is 0.025 ppm as an 8-hour TWA. The ACGIH (1986) has

recommended a ceiling limit of 0.01 ppm for this substance. Sulfur pentafluoride is a colorless gas or liquid with a sulfur-dioxide-like odor.

Sulfur pentafluoride's toxic effects include lung congestion and lesions, and pulmonary edema. In a study in which rats were exposed to sulfur pentafluoride for 16 to 18 hours, levels of 0.1 ppm caused lung irritation, 0.5 ppm resulted in severe pulmonary lesions, and 1 ppm proved fatal (Greenberg and Lester 1950). One-hour exposures to 10 ppm sulfur pentalfluoride resulted in diffuse hemorrhagic lesions in the lungs of rats, while rats exposed to 1 ppm for 1 hour had severe congestion of the lungs. Rats exposed for 1 hour at 0.1 ppm showed no effects. Subsequent examination of rats surviving the 10- and 1-ppm exposures revealed that the lungs had returned to normal after 24 hours (Greenberg and Lester 1950). Saunders, Shoshkes, De Carlo, and Brown (1953) established that the LD_{50} for sulfur pentafluoride in rabbits is 5.8 mg/kg, and that death was due to fulminant pulmonary edema. According to this study, sulfur pentafluoride does not injure the columnar epithelium of the respiratory tract, and exposure is not followed by bronchopneumonia.

OSHA is proposing a ceiling limit for this substance. The 0.01-ppm ceiling has been selected on the basis of evidence showing that even brief exposures to 1 ppm caused pulmonary effects in animals; 0.01 ppm provides a margin of safety against such effects. OSHA preliminarily concludes that this limit for sulfur pentafluoride will reduce the risks of irritation and pulmonary effects to which workers could be exposed in the absence of a ceiling limit. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for sulfur pentafluoride if the Agency determines that this limit will substantially reduce significant risk.

TETRAHYDROFURAN
CAS: 109–99–9; Chemical Formula: $(C_2H_4)_2O$
H.S. No. 1387

OSHA's existing PEL for tetrahydrofuran is 200 ppm as an 8-hour TWA. This is the 8-hour TWA limit recommended by the ACGIH, which also has established a 15-minute STEL of 250 ppm for tetrahydrofuran. Tetrahydrofuran is a colorless liquid with an odor like that of ether.

This proposed limit was selected on the basis of extensive data from experimental animal studies. Lehmann and Flury (1943) reported irritation of the upper respiratory tract as well as kidney and liver injury in a number of animals exposed by inhalation to more than 3000 ppm tetrahydrofuran for 20 days, 8 hours daily. Aqueous solutions exceeding a concentration of 20 percent tetrahydrofuran proved irritating to the skin of rabbits. One study (Stoughton and Robbins 1936) found that amounts in excess of 25,000 ppm were needed to anesthetize dogs. The anesthesia process in these animals showed a delayed induction period and poor recovery. In other studies with dogs (Zapp, cited in ACGIH 1986, p. 564), 200 ppm tetrahydrofuran in daily, 6-hour inhalation exposures produced an observable effect on the pulse pressure of these animals within 3 to 4 weeks; despite an exposure of nine weeks at this dosage level followed by 3 weeks at nearly twice this concentation, no histopathologic changes were observed in the critical organs. Studies (Jochmann 1962) in which tetrahydrofuran was given orally and peritoneally to a variety of laboratory animals resulted in both liver and kidney damage; however, some of the effects observed by this author may have been caused by peroride contamination of the tetrahydrofuran. Oettel (as cited in ACGIH 1986, p. 564) observed no kidney or liver damage in cats, rabbits, rats, or mice exposed repeatedly by inhalation to tetrahydrofuran at concentrations of 3400 to 17,000 ppm for as long as 6 hours.

Technicians involved in the experiment of Stoughton and Robbins (described above) experienced severe headaches when conducting these experiments.

OSHA is proposing an 8-hour TWA of 200 ppm and a STEL of 250 ppm for tetrahydrofuran. The Agency preliminarily concludes that both of these limits are required to protect workers from the risk of headache and irritation potentially associated with exposure to this substance. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for tetrahydrofuran if the Agency determines that this limit will substantially reduce significant risk.

TETRASODIUM PYROPHOSPHATE
CAS: 7722–88–5; Chemical Formula: $Na_4P_2O_7$
H.S. No. 1389

The current OSHA Z tables do not include a limit for tetrasodium pyrophosphate. The ACGIH has established a limit of 5 mg/m³ as an 8-hour TWA. Tetrasodium pyrophosphate may occur either as a white powder or a crystalline substance.

Tetrasodium pyrophosphate is an alkaline dust and therefore causes irritation to the eyes and the respiratory tract (ACGIH 1986, p. 567). For this reason, the ACGIH recommends a time-weighted average TLV of 5 mg/m³, which is half the value recommended for the inert dusts.

OSHA is proposing a 5-mg/m³ 8-hour TWA for this substance. The Agency preliminarily concludes that establishing this limit for this previously unregulated chemical is necessary to reduce the risk of eye and respiratory tract irritation to workers exposed to this substance. The health evidence forms a reasonable basis for proposing a new limit for tetrasodium pyrophosphate. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

THIOGLYCOLIC ACID
CAS: 68–11–1; Chemical Formula: $C_2H_4O_2S$
H.S. No. 1392

OSHA has no current PEL for thioglycolic acid. The ACGIH recommends a 1-ppm 8-hour TWA, with a skin notation, for this colorless liquid, which has an unpleasant odor.

A study by the Dow Chemical Company in which thioglycolic acid was instilled into the eyes of rabbits resulted in severe conjunctival inflammation and pain, dense opacity of the cornea, and severe inflammation of the iris. These effects had not improved 14 days after exposure and washing immediately after exposure did not modify the severity of this ocular response. A single dermal application of the thioglycolic acid to rabbit skin caused necrosis in 5 minutes and was accompanied by hyperemia and edema. The LD_{50} for a 10-percent solution applied percutaneously was 848 mg/kg for rabbits; further studies by Dow (1973) in which female rats were fed a single oral dose of a 10-percent solution of thioglycolic acid showed that this dose resulted in death at the level of 125 mg/kg. Autopsy revealed damage to the liver and gastrointestinal tract. Fassett (1963) reported that the oral LD_{50} for undiluted thioglycolic acid in rats in 50 mg/kg, and that a 10-percent solution applied to the skin of guinea pigs caused fatalities at doses of less than 5 ml/kg. Symptoms prior to death included gasping, convulsions, and weakness.

OSHA is proposing a 1-ppm 8-hour TWA limit for thioglycolic acid. The Agency preliminarily concludes that this limit will protect exposed workers from the risk of eye and skin irritation as well as systemic effects, potentially associated with exposures at the levels permitted by the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for thioglycolic acid. At the time of

the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

1,2,4-TRICHLOROBENZENE
CAS: 120-82-1; Chemical Formula: $C_6H_3Cl_3$
H.S. No. 1405

OSHA currently has no limit for 1,2,4-trichlorobenzene. The ACGIH has established an exposure limit of 5 ppm as a ceiling for this substance. 1,2,4 trichlorobenzene is a colorless liquid.

The inhalation toxicity of trichlorobenzene was studied by Treon (1950), who determined that the target organs of exposure in cats, dogs, rats, rabbits, and guinea pigs included liver, kidneys, ganglion cells at all brain levels, and mucous membranes. Irritation of the lungs and changes in respiration were seen in animals that later died as a result of exposure. Brown et al. (1969) reported that 1,2,4-trichlorobenzene's single-dose oral LD_{50} for rats is 756 mg/kg and for mice is 766 mg/kg. The acute percutaneous LD_{50} for rats was 6139 mg/kg. Sublethal doses administered repeatedly to guinea pigs caused liver damage; acute and short-term (15 6-hour exposures to 70–200 ppm) inhalation studies failed to kill these animals (Gage 1970). In a separate study reported on by Rowe (1975, as cited in ACGIH 1986, p. 593), 20 male rats, 4 rabbits, and 2 dogs were exposed at levels of 30 or 100 ppm 1,2,4-trichlorobenzene 7 hours/day, 5 days/week for 30 exposures in 44 days. No adverse effects were detectable in exposed animals belonging to 30 species as a result of exposure to 30 ppm, with the exception of an elevation of urinary porphyrins in the rats at 15 and 30 exposure days. A second inhalation study was performed with 1,2,4-trichlorobenzene 7 hours/day, 5 days/week for 26 consecutive weeks (Coate, Schoenfisch, Busey, and Lewis n.d.). Thirty rats, 16 rabbits, and 9 monkeys, all males, were exposed at zero, 25, 50, and 100 ppm. Microscopic changes were seen in the parenchymal cells of livers and kidneys of all rats after 4 and 13 weeks of exposure to 1,2,4-trichlorobenzene, but no adverse effects were seen in any of the other species.

In workers, exposure to 1,2,4-trichlorobenzene caused dermal irritation, which may have been attributable to the defatting action of this chemical (Powers, Coate, and Lewis 1975), and in some cases, exposure levels of 3 to 5 ppm caused eye and throat irritation (Rowe 1975, as cited in ACGIH 1986, p. 593).

OSHA is proposing a 5-ppm ceiling limit for 1,2,4-trichlorobenzene. The Agency preliminarily concludes that this limit will protect workers from the risk of eye, throat, and dermal irritation to which they can potentially be exposed in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for 1,2,4-trichlorobenzene. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

TRIETHYLAMINE
CAS: 121-44-8; Chemical Formula: $(C_2H_5)_3N$
H.S. No. 1408

OSHA currently has a limit of 25 ppm TWA for triethylamine. The ACGIH recommends a TLV-TWA of 10 ppm and a TLV-STEL of 15 ppm for this colorless liquid with a strong ammonia-like odor.

Exposure to triethylamine is associated with pulmonary, skin, and eye irritation and central nervous system effects. Guinea pigs exposed for 30 minutes to a concentration of 2,000 ppm triethylamine survived, but four of six animals died when exposed to this level for 2 hours; two of six guinea pigs died during a 4-hour exposure to a concentration of 1,000 ppm, but all survived similar exposures at the 250- and 500-ppm levels (Carpenter, Smyth, and Shaffer 1948). The single-dose oral LD_{50} value in rats is 0.46 g/kg (range: 0.25 to 0.85) (Smyth, Carpenter, and Weil 1951). These investigators also report that triethylamine readily penetrated rabbit skin on contact, with an LD_{50} value of 0.57 ml/kg (range: 0.36 to 0.90); skin irritation and eye injury were also noted from contact with the liquid. One of the six rats died from an acute 4-hour inhalation exposure to 1,000 ppm triethylamine (Smyth, Carpenter, and Weil 1951). Rabbits exposed repeatedly to a level of 50 ppm exhibited marked irritation of the cornea and of pulmonary tissue (Brieger and Hodes 1951; Carpenter and Smyth 1946). The effects of repeated triethylamine exposure correspond to those of ethylamine and diethylamine (Brieger and Hodes 1951). Triethylamine was found to inhibit monoamine oxidase activity, resulting in central nervous system stimulation (DeBruin 1976).

OSHA is proposing an 8-hour TWA limit of 10 ppm and a 15-minute STEL of 15 ppm for triethylamine. The Agency preliminarily concludes that both of these limits are required to protect workers against the risk of CNS effects and acute irritation to the eyes and lungs potentially associated with occupational exposure to triethylamine at elevated levels. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for triethylamine if the Agency determines that this limit will substantially reduce significant risk.

VANADIUM (V_2O_5) DUST
CAS: 1314-62-1; Chemical Formula: V_2O_5
H.S. No. 1421

The current OSHA PEL for vanadium pentoxide dust is a ceiling of 0.5 mg/m³. The ACGIH has established 0.05 mg/m³ as an 8-hour TWA for the respirable dust as vanadium pentoxide. NIOSH recommends a 15-minute short-term limit of 0.05 mg/m³. Vanadium pentoxide is a yellow to rust-brown crystalline compound.

Several studies indicate that OSHA's current exposure limit is insufficient to protect exposed workers against vanadium dust's respiratory effects, which include bronchitis, emphysema, tracheitis, pulmonary edema, and bronchial pneumonia. According to Hudson (1964), vanadium is poisonous to all animals by all routes of administration. The LD_{50} in rabbits injected intravenously is 1.5 mg/kg, and rats fed 25 ppm demonstrated toxic responses within a short time (Hudson 1964).

Seven cases of upper respiratory tract irritation were reported in boiler cleaners exposed to concentrations of from 2 to 85 mg/m³ vanadium pentoxide dust (Sjoberg 1951). Williams (1952) reported eight cases of vanadium poisoning in workers cleaning boilers in an atmosphere ranging from 30 to 104 mg/m³. Gulko (1956) observed eye and bronchial irritation in workers exposed to 0.5 to 2.2 mg/m³. A study by Lewis (1959) indicated that workers exposed to levels of 0.2 to 0.5 mg/m³ experienced a higher incidence of respiratory symptoms than did controls. Tebrock and Machle (1968) reported that workers exposed to average concentrations of 1.5 mg/m³ vanadium pentoxide in a mixed dust developed conjunctivitis, tracheobronchitis, and dermatitis. A single average 8-hour exposure to 0.2 mg/m³ respirable vanadium dust caused severe upper respiratory tract irritation in five human volunteers, and two other subjects exposed to a 0.1-mg/ ³ concentration also developed a delayed cough and an increase in mucous production (Zenz and Berg 1967).

OSHA is proposing a limit of 0.05 mg/m³ as an 8-hour TWA for vanadium dust as vanadium pentoxide. The Agency preliminarily concludes that this limit will prevent or substantially reduce the risks of eye and bronchial irritation, respiratory symptoms, conjunctivitis, and coughing seen in exposed workers at levels ranging from 0.1 mg/m³ to 2.2 mg/m³. The health evidence forms a reasonable basis for proposing a

revision to this level. At the time of the final rule, OSHA will establish a new limit for vanadium dust if the Agency determines that this limit will substantially reduce significant risk.

VANADIUM (V_2O_5) FUME
CAS: 1314-62-1; Chemical Formula: V_2O_5
H.S. No. 1422

OSHA's present PEL for vanadium pentoxide fume is 0.1 mg/m³. The ACGIH has set a 0.05-mg/m³ 8-hour TWA limit for vanadium pentoxide fume, and NIOSH recommends 0.05 mg/m³ as a 15-minute short-term limit. Vanadium pentoxide is a yellow to rust-brown crystalline compound.

Vanadium pentoxide fume's chief toxic effects are manifested in the respiratory passages: Bronchitis, emphysema, tracheitis, pulmonary edema, and bronchial pneumonia result from exposure to vanadium pentoxide fume. According to Hudson (1964), vanadium is poisonous to all animals by all routes of administration. The LD_{50} in rabbits injected intravenously is 1.5 mg/kg, and rats fed 25 ppm demonstrated toxic responses within a short time (Hudson 1964).

Seven cases of upper respiratory tract irritation were reported in boiler cleaners exposed to concentrations of from 2 to 85 mg/m³ vanadium pentoxide fume (Sjoberg 1951). Williams (1952) reported eight cases of vanadium poisoning in workers cleaning boilers in an atomosphere ranging from 30 to 104 mg/m³. Gulko (1956) observed eye and bronchial irritation in workers exposed to 0.5 to 2.2 mg/m³. A study by Lewis (1959) indicated that workers exposed to levels of 0.2 to 0.5 mg/m³ experienced a higher incidence of respiratory symptoms than did controls. Tebrock and Machle (1968) reported that workers exposed to average concentrations of 1.5 mg/m³ vanadium pentoxide in a mixed dust developed conjunctivitis, tracheobronchitis, and dermatitis. A single average 8-hour exposure to 0.2 mg/m³ respirable vanadium dust caused severe upper respiratory tract irritation in five human volunteers, and two other subjects exposed to a 0.1-mg/m³ concentration also developed a delayed cough and an increase in mucous production (Zenz and Berg 1967).

OSHA is proposing an 8-hour TWA limit of 0.05 mg/m³ for vanadium fume as vanadium pentoxide. The Agency preliminarily concludes that this limit will protect exposed workers from the risks of eye, skin, and upper respiratory tract irritation, conjunctivitis, pulmonary damage, and systemic poisoning potentially associated with vanadium pentoxide fume exposures. OSHA believes that this limit will substantially reduce these risks. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for vanadium fume if the Agency determines that this limit will substantially reduce significant risk.

VINYL ACETATE
CAS: 108-05-4; Chemical Formula: $CH_3COOCH=CH_2$
H.S. No. 1424

There is no current OSHA limit for vinyl acetate. The ACGIH has recommended a 10-ppm TLV-TWA and a 20-ppm TLV-STEL. NIOSH (1978m) recommends a 4-ppm ceiling limit, measured over a 15-minute period.

The basis for the ACGIH-recommended limits is an epidemiologic report by Deese and Joyner (1969) describing 15 years of industrial experience with vinyl acetate production. They reported that vinyl acetate is not a significant irritant at exposure levels of 5 to 10 ppm but causes cough and hoarseness at around 22 ppm. They also found no evidence of adverse chronic effects resulting from exposure to 5 to 10 ppm, as determined from medical records and examinations. While conducting air sampling for the study, the primary author (Deese) experienced hoarseness at concentrations of 4.2 and 5.7 ppm, and eye irritation at 5.7 and 6.8 ppm. Three chemical operators and one technician did not report any subjective responses at these levels. The ACGIH also cited a personal communication from the Mellon Institute (1968) that vinyl acetate concentrations of less than 5 ppm are detectable by odor, although some individuals may detect the odor at concentrations of 0.5 ppm.

NIOSH (1978m) reviewed these data and concluded that the recommended exposure limit be designed to protect even the most sensitive individuals from sensory irritant effects. Since the lowest level reported to cause upper respiratory tract irritation was 4.2 ppm (Deese and Joyner 1969), NIOSH recommended that workplace exposures not exceed 4 ppm measured over a 15-minute period.

The NIOSH REL of 4 ppm (ceiling) relies on a report concerning a single individual and provides insufficient information as a basis for standardsetting. The ACGIH recommended TLVs are based on a 15-year epidemiology study that suggests that a 10-ppm TWA and 20-ppm STEL will provide protection against the risk of irritation associated with exposure to vinyl acetate at higher levels. Therefore, the Agency proposes this 8-hour TWA and STEL combination as the revised limits for vinyl acetate. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for vinyl acetate if the Agency determines that this limit will substantially reduce significant risk.

VM & NAPHTHA
CAS No. 8032-32-4; Chemical Formula: none
H.S. No. 1429

OSHA currently has no PEL for VM & P (Varnish Makers' and Printers') naphtha. The ACGIH has established an 8-hour TWA of 300 ppm and a STEL of 400 ppm, which is not to be exceeded in any 15-minute period, for this substance. NIOSH recommends a 10-hour TWA of 75 ppm and a 400-ppm 15-minute STEL. VM & P naphtha, also known as ligroin, is a colorless, flammable liquid.

A study in which rats and beagles received doses by inhalation of 500 ppm for 30 hours per week for 13 weeks resulted in no chronic or latent effects (Carpenter et al. 1975). These authors also noted that the acute toxicity of VM & P naphtha for rats and other species was 4 times greater than that of rubber solvent naphtha, which has a limit of 400 ppm. Carpenter and associates (1975) also reported on an experiment in which rats lost coordination and went into convulsions in 15 minutes during exposures to saturation concentrations at ambient room temperature. The 4-hour inhalation LC_{50} was 3400 ppm, and the acclimated rats survived 5800 ppm for 6 hours.

Seven human volunteers exposed to 880 ppm VM & P naphtha for 15 minutes reported upper respirator tract, eye, nose irritation, and olfactory fatigue (ACGIH 1986, p. 631). Elkins (1959) noted one case of a worker, exposed to levels of VM & P naphtha averaging 800 ppm, who developed unspecified chronic effects. Elkins also reported that the VM & P naphtha level producing significant irritation in human volunteers was about half as great for this form of naphtha as for rubber solvent naphtha.

OSHA is proposing limits of 300 ppm as an 8-hour TWA and 400 ppm as a 15-minute STEL for VM & P naphtha. The Agency preliminarily concludes that both of these limits are required to protect exposed workers against the risk of upper-respiratory effects, eye irritation, and possible chronic effects associated with naphtha exposure. The health evidence forms a reasonable basis for proposing a new limit for VM & P naphtha. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

XYLENE, o, m, and p ISOMERS

CAS: 1330-20-7; 95-47-6; 108-38-3; 106-42-3; Chemical Formula: $C_6H_4(CH_3)_2$
H.S. No. 1431

The current OSHA limit for xylene is 100 ppm as an 8-hour TWA. The ACGIH also recommends a TLV-TWA of 100 ppm, but adds a 15-minute STEL of 150 ppm. NIOSH recommends an exposure limit of 100 ppm TWA, with a 200-ppm 10-minute ceiling. Xylene and its isomers are clear, flammable liquids with an aromatic hydrocarbon odor.

Rats and rabbits exposed to a mixture of xylene isomers at a concentration of 690 ppm for 8 hours daily, six days per week showed no blood abnormalities, but rabbits exposed on the same regimen at 1150 ppm for 55 days showed a decrease in red and white blood cell counts and an increase in platelet count (Fabre and Truhaut 1954).

Studies of workers exposed to xylene revealed headache, fatigue, lassitude, irritability, and gastrointestinal disturbances as the most common symptoms (Gerarde 1960). At unspecified exposure levels, Browning (1965) also noted gastrointestinal disturbances, in addition to kidney, heart, liver, and neurological damage; blood dyscrasias, some of which result in death, were also reported in these workers. A study by Nelson, Ege, Ross et al. (1943), in which human volunteers were exposed to 200 ppm xylene, found eye, nose, and throat irritation in the subjects at this level of exposure.

NIOSH developed a Criteria Document for xylene in 1975, in which the work of Morley and his colleagues (1970) was discussed. These authors observed liver dysfunction and renal impairment in three workers overexposed to xylene (estimated concentration of 10,000 ppm). One of these workers died, but the others recovered slowly. Furniture polishers were reported by Matthaus (1964) to have suffered corneal damage as a result of exposure to xylene at unknown concentrations.

OSHA preliminarily concludes that both a TWA and STEL are necessary to prevent risk of narcosis, blood effects, and irritant effects at the elevated levels possible at the current exposure limit. To reduce this risk, OSHA is proposing a 150-ppm STEL and a 100-ppm TWA. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for xylene if the Agency determines that this limit will substantially reduce significant risk.

ZINC CHLORIDE (FUME)
CAS: 7646-85-7; Chemical Formula: $ZnCl_2$
H.S. No. 1435

OSHA's current PEL for zinc chloride is 1 mg/m³ as an 8-hour TWA. The ACGIH has established a TLV-TWA of 1 mg/m³, with a STEL of 2 mg/m³. Zinc chloride fume is white and has an acrid odor.

Zinc chloride fume is highly caustic and damages the mucous membranes of the nasopharynx and respiratory tract. Exposure to the fumes of zinc chloride may result in a severe pneumonitis that is caused by irritation of the respiratory tract (Gafafer 1964). One instance in which a worker inhaled zinc chloride fumes resulted in advanced pulmonary fibrosis that ended in death (Milliken, Waugh, and Kadish 1963), and 10 deaths and 25 non-fatal cases of pneumonitis occurred in workers caught in a tunnel when 79 smoke generators caught fire and generated zinc chloride fumes (Humter 1955). Other studies have shown that zinc chloride exposures cause skin ulceration (Sax 1957). It has also been suggested that zinc chloride exposure may have chronic effects (Hamilton and Hardy 1974). In an investigation of the adverse effects of zinc chloride fume exposures, Ferry (personal communication 1966, as cited in the ACGIH 1986, p. 643) reported that no sensory effects occurred when 30 minute exposures were limited to 0.07 and 0.4 mg/m³; however, this researcher noted that these levels did corrode metal.

OSHA preliminarily concludes that the risk of damage to the eyes, skin, and respiratory tract associated with exposure to zinc chloride fume should be substantially reduced by establishing a STEL and TWA to protect against elevated short-term and long-term exposures to this substance. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for zinc chloride if the Agency determines that this limit will substantially reduce significant risk.

Preliminary Conclusions

OSHA's preliminary finding is that sensory irritation poses an occupational health risk to workers exposed to these substances at the existing hazardous levels. Among the adverse health consequences of exposure to sensory irritants are acute breathing difficulty, eye tearing, conjunctivitis, sensitization, persistent coughing, and upper respiratory tract irritation. In addition to the pain and suffering associated with these signs and symptoms, workers experiencing irritant effects find it difficult if not impossible to concentrate on the job at hand; thus they work less safely and less productively than non-exposed employees. Reducing exposures from levels that have been associated with these effects to levels where such consequences are substantially less likely to occur will reduce the risk posed to workers at current levels.

The health evidence for these substances forms a reasonable basis for proposing revised or new limits. At the time of the final rule, OSHA will establish new or revised limits for these sensory irritants if the Agency determines that these limits will substantially reduce significant risks.

4. Substances for Which Proposed Limits Are Based on Avoidance of Liver or Kidney Effects

Introduction

The liver or kidneys are the primary target organs affected by toxic exposures to a number of industrial chemicals. In recognition of this target organ toxicity, OSHA is proposing new or revised limits for 17 hepato- or nephrotoxic compounds (12 hepatotoxins and five nephrotoxins). For these substances, the liver or kidney is probably the organ most sensitive to the effects of exposure. Thus, establishing permissible exposure limits that are low enough to prevent toxicity to these target organs generally also protects other organ systems.

Seventeen compounds for which revised limits are being proposed produce kidney or liver effects in overexposed individuals. For seven of these substances, OSHA is proposing to lower the PEL, and for one other substance, OSHA is also proposing the adoption of a short-term exposure limit. For five liver or kidney toxins, OSHA is proposing a PEL where none formerly existed, and in three cases, OSHA proposes to retain the current PEL but to add a STEL where none formerly existed. For four chemicals in this category, NIOSH recommends limits lower than those established by the ACGIH, and for one of these substances, OSHA proposes adoption of the NIOSH RELs; in three cases, the NIOSH and ACGIH limits are essentially the same. The sections below discuss liver and kidney toxins separately. Table C4-1 shows these hepatotoxic substances, their OSHA, ACGIH, and NIOSH limits, and their CAS and HS numbers; Table C4-2 provides the same information for the nephrotoxins in this group.

Liver Toxicity

Description of the Health Effects

Although the precise mechanisms by which these compounds cause liver damage are only partly understood, the development and manifestation of liver

toxicity are similar for all of them. In general, liver toxicity is a graded response, i.e., the severity of the lesion is directly proportional to the intensity/duration of exposure. Although many of the effects caused by exposure to these substances are reversible, some are not.

Liver damage is not a single entity; the manner in which it is manifested depends on the dose, duration, and particular chemical agent involved. For example, acute exposures may cause lipid to accumulate in the hepatocytes, cellular death, and/or hepatobiliary dysfunction. In contrast, chronic exposures may lead to cirrhotic changes and the development of neoplasms. Fatty accumulation and necrosis can be either localized or widespread, and chemical-induced lesions resulting from chronic exposures can cause marked changes of the entire liver (Plaa 1986).

Typically, the earliest and most sensitive indicators of liver toxicity are alterations in biochemical liver functions, such as changes in specific enzyme activities. These may be accompanied by changes in the morphology of specific organelles in hepatocytes. For example, relatively low doses of halogenated aliphatic hydrocarbons, such as allyl chloride, carbon tetrabromide, and 1,1-dichloroethane, cause an increase in the activity of microsomal mixed function oxidase enzymes. This increase is ordinarily accompanied by proliferation of the endoplasmic reticulum.

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Table C4-1. List of Substances For Which Limits Are Based Primarily on Avoidance of Liver Toxicity

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1011 Allyl chloride	107-05-1	1 ppm TWA	1 ppm TWA 2 ppm STEL	1 ppm TWA 3 ppm Ceiling (15 min)
1072 Carbon tetrabromide	558-13-4	-	0.1 ppm TWA 0.3 ppm STEL	-
1089 o-Chlorostyrene	2039-87-4	-	50 ppm TWA 75 ppm STEL	-
1108 Cyclohexanone	108-94-1	50 ppm TWA	25 ppm TWA, Skin	25 ppm TWA
1145 Dioxane	123-91-1	100 ppm TWA, Skin	25 ppm TWA, Skin	1 ppm Ceiling (30 min)
1168 Ethylene dichloride+	107-06-2	50 ppm TWA 100 ppm STEL 200 ppm Ceiling	10 ppm TWA	1 ppm TWA 2 ppm Ceiling (15 min)
1205 Hydrazine	302-01-1	1 ppm TWA, Skin	0.1 ppm TWA, Skin	0.03 ppm Ceiling (120 min)
1269 Methylcyclohexanol	25639-42-3	100 ppm TWA	50 ppm TWA	-
1295 Octachloro-naphthalene	2234-13-1	0.1 mg/m^3 TWA, Skin	0.1 mg/m^3 TWA 0.3 mg/m^3 STEL Skin	-

Table C4-1. List of Substances For Which Limits Are Based Primarily on Avoidance of Liver Toxicity (continued)

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1341 Propylene dichloride	78-87-5	75 ppm TWA	75 ppm TWA 110 ppm STEL	—
1385 1,1,2,2-Tetrachloroethane	79-34-5	5 ppm TWA, Skin	1 ppm TWA, Skin	Lowest feasible level
1407 1,2,3-Trichloropropane	96-18-4	50 ppm TWA	10 ppm TWA, Skin	—

* OSHA's TWA limits are for 8-hour exposures; its STELs are for the durations specified; and its ceilings are peaks not to be exceeded for any period of time.

** The ACGIH TWA-TLV is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

*** NIOSH TWA limits are for 10-hour exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

+ Proposed limit is the NIOSH REL.

Table C4-2. List of Substances For Which Limits are Based Primarily on Avoidance of Kidney Toxicity

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1129 1,3-Dichloropropene	542-75-6	-	1 ppm TWA, Skin	--
1132 Dicyclopentadiene	77-73-6	-	5 ppm TWA	--
1166 Ethyl silicate	78-10-4	100 ppm TWA	10 ppm TWA	--
1195 Hexachlorobutadiene	87-68-3	-	0.02 ppm TWA, Skin	--
1203 Hexone (Methyl isobutyl ketone)	108-10-1	100 ppm TWA	50 ppm TWA 75 ppm STEL	50 ppm TWA

* OSHA's TWA limits are for 8-hour exposures; its STELs are for the durations specified; and its ceilings are peaks not to be exceeded for any period of time.

** The ACGIH TWA-TLV is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

*** NIOSH TWA limits are for 10-hour exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

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Many compounds that damage the liver, such as 1,1,2,2-tetrachloroethane, also cause an abnormal accumulation of fat, especially triglycerides, in liver cells. In experimental animals this effect is manifested as an accumulation of microscopic vacuoles in liver cells. In humans, however, the only grossly detectable manifestation of this effect is increased liver size, which is an indication of severe fat accumulation in the liver.

At sufficiently high doses, most substances that damage the liver cause cell death that leads to tissue necrosis or gangrene. This necrosis may initially be localized, but at higher or more sustained exposure levels the entire liver may be involved. Moderate to severe liver necrosis is usually accompanied by increased concentrations of marker enzymes such as glutamate-pyruvate transaminase or glutamate-oxaloacetate transaminase in the serum; the detection of these substances in the serum of exposed individuals can thus be a useful diagnostic tool.

Dose-Response Characteristics

The development of liver and other organ damage in humans and animals is progressive; it begins with subcellular changes, progresses to the cellular level, and is finally manifested as whole-organ damage. This progression is related to the intensity/duration of dose; i.e., as dose increases, cellular death becomes widespread and eventually causes liver dysfunction. The extent to which liver damage is reversible follows a similar continuum; since the liver can regenerate, minor cellular damage or transient disease states are usually reversible if exposure ceases. However, if exposure continues, the capacity of the liver to regenerate is exceeded and permanent damage results.

As is the case for some chemically induced toxic effects, there appears to be a NOE level below which hepatotoxic effects do not occur.

The following paragraphs describe OSHA's preliminary results for all of the substances in this group of hepatotoxins and discuss the nature of the risk experienced by exposed workers.

ALLYL CHLORIDE
CAS: 107–05–1; Chemical Formula: $CH_2=CHCH_2Cl$
H.S. No. 1011

The current OSHA PEL for allyl chloride is a 1 ppm (3 mg/m³) 8-hour TWA; the ACGIH-recommended TLV–TWA is also 1 ppm, with a 15-minute STEL of 2 ppm. NIOSH has recommended a 1 ppm 10-hour TWA and a 3 ppm 15-minute STEL for this substance. Allyl chloride is a colorless liquid with an unpleasant, pungent odor.

Studies of animal exposures to allyl chloride indicate that the chemical is among the most toxic of the halogenated aliphatic hydrocarbons, producing mucous membrane irritation, mild narcosis, and, at higher concentrations, histologic lesions of the lungs and kidneys (Adams, Spencer, and Irish 1940). Even single exposures lasting only a few minutes at concentrations between 1 and 100 mg/liter (332 to 32,000) caused mucous membrane irritation in various laboratory animals (Adams, Spencer, and Irish 1940). Further animal studies have confirmed liver and kidney pathology in many species (Torkelson, Wolf, Oyen, et al. 1959) and female rats exhibited kidney pathology after exposure to 3 ppm for 6 months.

Human exposures to concentrations of 1 to 113 ppm caused abnormal liver test results (Hausler and Lenich 1968). OSHA therefore proposes that both a STEL of 2 ppm and a 1 ppm TWA are required. The Agency preliminarily concludes that a combined limit is necessary to protect employees from the risk of mucous membrane irritation potentially associated with the elevated short-term exposures to allyl chloride currently permitted by the 8-hour TWA alone. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for allyl chloride if the Agency determines that this limit will substantially reduce significant risk.

CARBON TETRABROMIDE
CAS: 558–13–4; Chemical Formula: CBr_4
H.S. No. 1072

OSHA's current Z tables have no limits for exposure to carbon tetrabromide. The ACGIH limit is a 0.1 ppm TWA and a 0.3 ppm STEL. Carbon tetrabromide's hepatotoxic effects include both fatty infiltration and necrosis. The 0.1 ppm and 0.3 ppm levels were selected based on an observed no-effect level at 0.1 ppm; this finding derives from a study in which rats were exposed to carbon tetrabromide by inhalation for 7 hours per day, 5 days per week for 6 months (Torkelson and Rowe 1981). OSHA believes that controlling workplace exposures to these levels will prevent adverse effects in exposed workers. OSHA preliminarily concludes that establishing a limit for this previously unregulated chemical will protect workers against the risk of experiencing its hepatotoxic effects and will achieve a substantial reduction in this risk. The health evidence forms a reasonable basis for proposing a new limit for carbon tetrabromide. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

o-CHLOROSTYRENE
CAS: 2039–87–4; Chemical Formula: C_8H_7Cl
H.S. No. 1089

OSHA has no current limit for o-chlorostyrene. The ACGIH recommends a TLV–TWA of 50 ppm with a TLV–STEL of 75 ppm. o-Chlorostyrene is a liquid.

In an unpublished report, the Dow Chemical Company (1973) describes the results of an o-chlorostyrene inhalation study in rats, rabbits, guinea pigs, and dogs. Dow exposed the animals to an average concentration of 101 ppm for 7 hours daily, 5 days a week, for a total of 130 exposures in 180 days. No adverse effects were observed in any species in terms of appearance, growth, behavior, mortality, hematology, BUN, alkaline phosphatase, SGPT, BSP, organ weights, or gross pathology (Dow Chemical Company, unpublished report, 1973). Microscopic examination of animal tissue revealed a somewhat higher incidence of pathological liver and kidney changes. There is evidence indicating that the warning properties of o-chlorostyrene do not permit workers to recognize concentrations of o-chlorostyrene of 100 ppm. Based on o-chlorostyrene's structural analogy to styrene, for which short-term exposures of 100 ppm have been demonstrated to produce neuropathic and narcotic effects (Stewart, Dodd, Baretta, and Schaffer 1968), a short-term limit is necessary (ACGIH 1986, p. 136).

OSHA is proposing a PEL of 50 ppm as an 8-hour TWA and a 15-minute STEL of 75 ppm for o-chlorostyrene. The Agency preliminarily concludes that both of these limits will protect against the risk of narcosis and neuropathy to which workers could potentially be exposed in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for o-chlorostyrene if the Agency determines that this limit will substantially reduce significant risk.

CYCLOHEXANONE
CAS: 108–94–1; Chemical Formula: $C_6H_{10}O$
H.S. No. 1108

OSHA has a current limit of 50 ppm TWA for cyclohexanone. Both the ACGIH and NIOSH recommend a time-weighted average of 25 ppm, and the ACGIH also recommends a skin notation. Cyclohexanone is a white to pale yellow oily liquid with an odor

similar to that of acetone and peppermint.

Cyclohexanone has a low order of acute toxicity in animals. A concentration of 2000 ppm inhaled for 4 hours was lethal to 1 of 6 rats; at 4000 ppm, all of the exposed animals died. In rabbits, the dermal LD_{50} was 1000 mg/kg (Smyth et al. 1969). Rabbits showed marked irritation and some corneal injury when undiluted cyclohexanone was instilled in the eye (Carpenter and Smyth 1946). Guinea pigs exposed to 4000 ppm for 6 hours showed narcotic symptoms, lacrimation, salivation, depression of body temperature and heart rate, and corneal opacity (Specht et al. 1940). Rabbits exhibited degenerative changes of the liver and kidneys after 50 daily 6-hour inhalation exposures to 190 ppm (Treon, Crutchfield, and Kitzmiller 1943). Exposures to 309 ppm cyclohexanone on the same regimen caused conjunctival congestion, while exposures to 3000 ppm were lethal to some of the exposed animals (Treon, Crutchfield, and Kitzmiller 1943). In humans, Nelson and co-workers (1943) report that irritation caused by exposure to cyclohexanone was intolerable at 50 ppm, however, 25 ppm was not objectionable to most subjects in 3- to 5-minute exposures.

OSHA is proposing a 25-ppm 8-hour TWA and a skin notation for cyclohexanone. The Agency preliminarily concludes that these two limits will prevent the risk of respiratory and skin irritation associated with cyclohexanone exposures at levels below the existing PEL of 50 ppm. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for cyclohexanone if the Agency determines that this limit will substantially reduce significant risk.

DIOXANE
CAS: 123-91-1; Chemical Formula: $O(CH_2CH_2)_2O$
H.S. No. 1145

OSHA's current PEL for dioxane is 100 ppm as an 8-hour TWA, with a skin notation. NIOSH recommends a 1-ppm 30-minute ceiling for dioxane, and the ACGIH has established a 25-ppm TLV-TWA, with a skin notation. The proposed 25-ppm PEL reflects new toxicological data for this substance. A 2-year drinking water study conducted by the Dow Chemical Company, in which male and female rats were given water containing 1.0, 0.1, or 0.01 percent dioxane, showed that animals given the highest dose developed liver and nasal tumors, as well as pathological changes in the liver and kidney. Rats in the 0.1-percent group showed renal tubular sloughing and hepatocellular degeneration but no significant increase in neoplasms. Because this study demonstrated hepato- and nephrotoxic effects at doses 10 times lower than the dose causing cancer in animals, the ACGIH established the TLV based on dioxane's liver and kidney effects rather than its carcinogenicity. A study by Torkelson et al. (1974) in four species of animals exposed to multiple daily airborne exposures of dioxane at 50 ppm showed no gross or histopathologic organ changes, leading the ACGIH to recommend 25 ppm as an appropriate level to protect against liver and kidney effects in exposed workers (ACGIH 1986). In this case, the ACGIH is using a safety factor to establish the 8-hour limit, i.e., is applying a safety factor of 2 to the results of an animal study in four species that showed no gross or histopathological changes at 50 ppm.

The 25-ppm TLV is based on hepato- and nephrotoxic effects, since the ACGIH evaluation determined that these effects were more severe than the potential for carcinogenicity. OSHA does not generally accept such a rationale; however, OSHA is unable to perform the necessary risk assessment to evaluate the question of carcinogenicity in time for this rulemaking. The 1-ppm (ceiling) REL is based on cancer potential and appears to represent the "lowest concentration reliably measured," a criterion that would not satisfy OSHA feasibility requirements. OSHA therefore proposes that a PEL of 25 ppm TWA, with a skin notation, be adopted at the present time to reduce the risk that currently exists. As future priorities permit, OSHA will consider the need for a more stringent PEL.

ETHYLENE DICHLORIDE
CAS: 107-06-2; Chemical Formula: $ClCH_2CH_2Cl$
H.S. No. 1168

The current OSHA standard for ethylene dichloride is 50 ppm as an 8-hour TWA, a 100-ppm ceiling (maximum duration of 5 minutes in any 3 hours), and a 200-ppm peak; these limits were derived from limits recommended by the American National Standards Institute in 1969. In 1980, the ACGIH reduced its TLV to 10 ppm as an 8-hour TWA. NIOSH (1978) has concluded that ethylene dichloride should be considered a potential human carcinogen and has recommended 1-ppm 10-hour TWA and 2-ppm 15-minute short-term limits. Several studies indicate that the current OSHA PELs are insufficient to protect workers against hepatotoxic and other adverse effects. A paper by Kozik (1957) reported that workers generally exposed below 16 ppm but occasionally exposed to levels between 30 and 50 ppm experienced adverse liver and nervous system effects. In addition, Brzozowski (1954) reported abnormal changes in the blood of 50 percent of workers (8 of 16) exposed to between 10 and 37 ppm of ethylene dichloride. The ACGIH also cited numerous animal studies that consistently show hepatotoxic effects caused by exposure to ethylene dichloride; the ACGIH concluded that ethylene dichloride "clearly belongs in the group of hepatotoxic halogenated hydrocarbons" (ACGIH 1986). Based on these findings, the ACGIH (1986) recommended a reduction in the 8-hour TLV-TWA to 10 ppm.

In August 1978, NIOSH recommended that exposure to ethylene dichloride be minimized in response to an NCI bioassay (1978) showing that ethylene dichloride induced liver cancer in both sexes of mice and rats. NIOSH (1978) subsequently recommended a 1-ppm 10-hour TWA and a 2-ppm 15-minute short-term limit.

OSHA preliminarily concludes that the studies of Kozik (1957) and Brzozowski (1954) clearly indicate that a risk of hepatotoxicity, nervous system effects, and hematopoietic effects exists at the current 50-ppm 8-hour TWA PEL. The 10-ppm TLV recommended by the ACGIH does not afford protection at the levels (10 to 37 ppm) where abnormal blood changes were measured in 50 percent of the workers studied. Therefore, OSHA believes it necessary to reduce its current limits for ethylene dichloride to reduce this risk (as well as to protect against potential carcinogenic effects) and is proposing to revise these limits to 1 ppm as a TWA and 2 ppm as a STEL. The Agency's preliminary feasibility analysis is based on limited data at these levels; OSHA requests additional feasibility information from the public. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for ethylene dichloride if the Agency determines that this limit will substantially reduce significant risk.

HYDRAZINE
CAS: 302-01-2; Chemical Formula: H_2N-NH_2
H.S. No. 1205

The current OSHA limit for hydrazine is 1 ppm as an 8-hour TWA, with a skin notation. The ACGIH (1986) has recommended a TLV-TWA of 0.1 ppm, also with a skin notation. Because of its potential carcinogenic hazard, NIOSH (1978a) has recommended that

workplace exposure to hydrazine not exceed 0.03 ppm as determined by a 2-hour air sample; this level represents the lowest detectable level over this sampling period.

A hepatotoxic response in mice and anemia and weight loss in dogs were reported to occur following a 6-month exposure to 1 ppm for 6 hours per day, 5 days per week or to 0.2 ppm continuously (Haun and Kinkead 1973). It was this finding that led the ACGIH to conclude that the former TLV of 1 ppm was too high. The ACGIH has also assigned an A2 designation (suspect human carcinogen), based on a study by MacEwen et al. (1979) showing significant increases in nasal tumors in rats exposed to 1 or 5 ppm hydrazine, in thyroid adenocarcinoma in rats exposed to 5 ppm, and in lung adenoma among mice exposed to 1 ppm. Other studies are cited by NIOSH (1978a) that demonstrate the carcinogenicity of hydrazine in rodents by a variety of dose routes. Because of this evidence, NIOSH has recommended a workplace exposure limit equal to the limit of detection for a 2-hour sample (0.03 ppm).

The animal studies conducted by Haun and Kinkead (1973) and by MacEwen et al. (1979) clearly demonstrate that exposure to the current 1 ppm PEL presents a risk of respiratory cancer, liver disease, and adverse blood effects; animals exposed to airborne concentrations at the current PEL have exhibited all of these effects. Therefore, OSHA preliminarily concludes that a reduction in the PEL for hydrazine is warranted to reduce this risk and is proposing to revise its PEL to 0.1 ppm as an 8-hour TWA, with a skin notation. The Agency has selected this limit rather than the 0.03-ppm REL recommended by NIOSH because it does not have sufficient feasibility data or risk assessment information regarding use of the 0.03-ppm level. Furthermore, the NIOSH REL appears to be based on the sampling and analytical limits of detection, and OSHA cannot base its limits on such criteria. The Agency believes that the 0.1-ppm limit will control hepatotoxic effects and reduce potential carcinogenic risk. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for hydrazine if the Agency determines that this limit will substantially reduce significant risk.

METHYLCYCLOHEXANOL
CAS: 25639-42-3; Chemical Formula: $CH_3C_6H_{10}OH$
H.S. No. 1269

OSHA currently has an 8-hour TWA limit of 100 ppm for methylcyclohexanol. The ACGIH recommends a limit of 50 ppm TWA. Methylcyclohexanol is a colorless, viscous liquid with an aromatic odor.

Exposure to methylcyclohexanol produces narcotic effects, liver and kidney impairment, and eye and respiratory irritation. Treon, Crutchfield, and Kitzmiller (1943) have reported the oral LD_{50} in rabbits to be between 1.25 and 2 g/kg; liver damage was observed in surviving animals. Repeated inhalation exposures to the vapor caused salivation, eye irritation and lethargy in rabbits exposed at 500 ppm, but exposures to 230 ppm caused no observable effects. Fifty 6-hour exposures at a level of 120 ppm caused "barely discernible" microscopic changes in the liver and kidney tissue of rabbits (Treon, Crutchfield, and Kitzmiller 1943).

In humans, headaches and eye and respiratory irritation have been reported to occur from prolonged exposure to high concentrations of methycyclohexanol (Fillipi 1914). Smyth (1956) considered an exposure limit of 100 ppm to be sufficiently low to prevent narcotic effects and, perhaps, significant liver and kidney damage.

OSHA is proposing an 8-hour TWA of 50 ppm for methylcyclohexanol. The Agency preliminarily concludes that this limit will protect workers against the risk of narcosis and hepatic and renal effects that potentially exists at the levels permitted by the current 100-ppm limit. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for methylcyclohexanol if the Agency determines that this limit will substantially reduce significant risk.

OCTACHLORONAPHTHALENE
CAS: 2234-13-1; Chemical Formula: $C_{10}Cl_8$
H.S. No. 1295

OSHA currently has a limit of 0.1 mg/m³ TWA, with a skin notation, for octachloronaphthalene. The ACGIH recommends a TLV-TWA of 0.1 mg/m³ and a TLV-STEL of 0.3 mg/m³, also with a skin notation. Octachloronaphthalene is a nonflammable, pale yellow, and waxy solid containing 70 percent chlorine.

Inhalation toxicity data for octachloronaphthalene fumes or dust are lacking, but exposure to the chloronaphthalenes causes acne-like lesions that itch severely. Repeated exposure to the fumes of molten chlorinated naphthalenes can cause severe and sometimes fatal systemic poisoning and is especially damaging to the liver (Patty 1963). Ingestion studies of cattle have shown different toxicities for different naphthalenes, with the toxicity increasing with the degree of chlorination (Sikes, Wise, and Bridges 1952). However, these data are controverted by another report in which octachloronaphthalene was found to be less toxic than the hexachloro derivative (Bell 1953). This divergence in the data may be due to differing methods of administration (suspension versus solution) or may reflect the soluble form's greater capacity for absorption (ACGIH 1986, p. 447).

OSHA proposes a PEL of 0.1 mg/m³ TWA and a STEL of 0.3 mg/m³, with a skin notation, for octachloronaphthalene. The Agency preliminarily concludes that this combined limit will protect workers against the risk of dermal effects and serious liver damage potentially associated with exposure to this substance at the levels permitted by the 8-hour limit alone. The skin notation is retained because of octachloronaphthalene's severe dermal effects. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for octachloronaphthalene if the Agency determines that this limit will substantially reduce significant risk.

PROPYLENE DICHLORIDE
CAS: 78-87-5; Chemical Formula: $CH_3CHClCH_2Cl$
H.S. No. 1341

OSHA currently has a limit of 75 ppm TWA for propylene dichloride. The ACGIH recommends a 75-ppm TLV-TWA and a TLV-STEL of 110 ppm. Propylene dichloride is a colorless, flammable, mobile liquid with an odor like that of chloroform.

The primary hazards associated with exposure to propylene dichloride are inhalation toxicity to liver tissue and mild skin and eye irritation. Repeated inhalation exposures to 1000 ppm have been reported to kill dogs (after 24 exposures), guinea pigs (after 22 exposures), and rats (in some cases after 7 exposures); some animals survived more than 100 7-hour exposures. Necropsy showed severe liver damage; the hepatotoxicity of propylene dichloride appears to be greater than that of carbon tetrachloride and less than that of ethylene dichloride (Heppel, Neal, Highman, and Porterfield 1946). Animals of these same species (rats, dogs, and guinea pigs) survived 128 to 140 7-hour exposures to 400 ppm for 5 days/week without histologic effects, while mice died from similar exposures; surviving mice displayed hepatomas (Heppel, Highman, and Peake 1948). The

oral LD_{50} for rats has been reported as 1.19 ml/kg (Smyth et al. 1969); the acute 8-hour inhalation LC_{50} for rats is 3000 ppm (Pozzani et al. 1959).

OSHA proposes an 8-hour TWA PEL for propylene dichloride of 75 ppm and a 15 minute STEL of 110 ppm. The Agency preliminarily concludes that this combined limit will protect workers against the risk of hepatotoxic effects potentially associated with exposures at the levels permitted by the absence of a short-term limit above the 8-hour TWA PEL for even brief periods. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for propylene dichloride if the Agency determines that this limit will substantially reduce significant risk.

1,1,2,2–TETRACHLOROETHANE
CAS: 79–34–5; Chemical Formula: $CHCl_2CHCl_2$
H.S. No. 1385

OSHA's existing PEL for 1,1,2,2–tetrachloroethane is 5 ppm with a skin notation; a 1-ppm 8-hour TWA, also with a skin notation, is the level established by the ACGIH. The NIOSH recommendation is that exposure be reduced to the lowest feasible level. One study by Jevey et al. (1957) revealed identifiably adverse effects on the liver, including hepatitis, in humans exposed to concentrations of tetrachloroethane ranging from 1.5 to 247 ppm; liver damage was still evident after exposures were reduced to 15 ppm. An animal study by Schmidt et al. (1972) found "barely detectable" fatty infiltration of the liver in rats exposed to 2 ppm tetrachloroethane for 11 months.

Based on this evidence, OSHA preliminarily concludes that the current level does not protect against fatty infiltration of the liver and probably does not protect against more serious liver damage; these health consequences clearly pose an occupational risk. OSHA believes that reducing the 8-hour TWA for tetrachloroethane to 1 ppm will substantially reduce the risk posed to workers exposed at the current PEL and thus proposes adoption of a 1-ppm 8-hour TWA, with a skin notation. The proposed limit was selected on the basis of relatively extensive data from health surveys conducted among occupationally exposed workers, supplemented by results from experimental animal studies. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for 1,1,2,2–tetrachloroethane if the Agency determines that this limit will substantially reduce significant risk.

1,2,3–TRICHLOROPROPANE
CAS: 96–18–4; Chemical Formula: $C_3H_5Cl_3$
H.S. No. 1407

OSHA's existing PEL for 1,2,3–trichloropropane is 50 ppm as an 8-hour TWA, and the ACGIH has a TLV–TWA of 10 ppm and a skin notation. 1,2,3–Trichloropropane is a colorless to straw-colored, combustible liquid with an odor similar to chloroform.

1,2,3-Trichloropropane is not irritating to intact skin, but it is absorbed through the skin. It is highly irritating to the eyes (Smyth, Carpenter, Weil et al. 1962). Five of six rats exposed to 1000 ppm died after 4-hour exposures. Rats and guinea pigs exposed at 800, 2100, or 5000 ppm for 30 minutes showed central nervous system depression, which progressed at the higher levels to narcosis and convulsions (Lewis, as cited in ACGIH 1986, p. 602). Several mice exposed for 20 minutes to 5000 ppm died, some as long as several days later from liver damage. Daily 10-minute exposures at 2500 ppm for 10 days killed 7 of 10 mice (McOmie and Barnes 1949). Animals exposed once for 4 hours to 1,2,3-trichloropropane at concentrations of 125, 340, 700, or 2150 ppm showed dose-related signs of irritation, which included, at 700 or 2150 ppm, labored respiration, inactivity, and eye and nose irritation; at autopsy, however, no organ or other damage was apparent (McOmie and Barnes 1949).

Human volunteers found exposure to 1,2,3-trichloropropane objectionable because of eye and upper respiratory tract irritation, and many found 50 ppm an unacceptable level for a full-shift exposure (Silverman, Schulte, and First 1946). Drew and colleagues (1978) noted changes in liver enzymes after a single 4-hour exposure to 500 ppm, and Russian studies indicate that morphologic changes and metabolic lesions of the liver, kidney, and lungs occurred in mice exposed continuously to concentrations of 0.007 to 0.3 ppm (Siderenko, Tsulaya, Bonashevskaya, and Shaipak 1979; Siderenko, Tsulaya, Koreneveskaya, and Banashevskaya 1976; Tsulaya, Bonashevskaya, Zykova et al. 1977).

A National Toxicology Program prechronic study in which rats were gavaged daily with 1,2,3-trichloropropane at 8, 16, 32, 63, 125, and 250 mg/kg body weight for 120 days showed good survival in all but the highest dose group (National Toxicology Program 1983). Statistically significant changes in the liver and kidney, and necrosis and irritation of the nasal passages, occurred in the 63 and 125 mg/kg dose groups. Decreases in the red blood cell count and hematocrit were seen even in the 16-mg/kg dose group. 1,2,3-Trichloropropane did not affect sperm count or morphology or testicular weight. The NTP found this substance to be genetically active in three bioassays. However, Hardin, Bond, Silkov et al. (1981) did not find 1,2,3-trichloropropane to be fetotoxic or teratogenic.

The ACGIH (1986, p. 602) concluded that the most sensitive sex and species, female rats, had a no-adverse-effect level for kidney or liver effects of 8 mg/kg/day, and extrapolated from this level to humans to establish a TLV–TWA of 10 ppm.

OSHA is proposing an 8-hour PEL of 10 ppm and a skin notation for 1,2,3-trichloropropane. The Agency preliminarily concludes that these limits are necessary to protect exposed workers against the risks of liver and kidney damage, eye and throat irritation, and systemic toxicity via inhalation or skin absorption that are potentially associated with exposures to this substance at levels considerably below the Agency's current PEL. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for 1,2,3-trichloropropane if the Agency determines that this limit will substantially reduce significant risk.

Kidney Toxicity

Introduction

Kidney damage is the basis for revising the PELs for five of the compounds in this group. These compounds, their CAS and HS numbers, and their current OSHA, ACGIH, and NIOSH limits are shown in Table C4–2. Three of these substances will be regulated by OSHA for the first time, and in the other two cases, the 8-hour TWA will be reduced. In one of the latter cases, a STEL will also be added.

Description of the Health Effects

The precise mechanism by which these chemicals damage the kidneys is unknown. Typically, these compounds are selectively toxic to cells in the renal tubule, perhaps because impaired transport causes the chemical to collect in these cells. In addition to the excretion of wastes, the kidney plays an important role in the regulation of total body homeostasis. This organ regulates extracellular volume, controls electrolyte and acid-base balance, and forms several hormones that control systemic metabolism. Depending on their particular site of action,

nephrotoxicants thus can interfere with the proper excretion of the body's wastes, hydration, electrolytic balance, metabolism, and the maintenance of the correct acid-base balance.

Like the hepatotoxic effects previously described, the least severe lesions caused by nephrotoxic compounds are graded and reversible. The earliest changes are usually alterations in the activities of specific enzymes in the tubular cells. These changes may be accompanied by minor morphological alterations of the cells that are visible only with an electron microscope. Higher doses or more sustained exposures are required to cause cellular necrosis that might be visible with light microscopy. Because of the reserve capacity of the kidneys, a significant degree of tubular cellular necrosis must occur before it is reflected by measurable alterations in kidney function. Thus, indicators of impaired renal function that can be measured in humans, such as proteinuria, glucosuria, and increased BUN, are relatively insensitive indicators of kidney damage. Other indicators of significant kidney damage include increased kidney weight, swelling of the tubular epithelium, fatty degeneration of tubular epithelium, and the presence of tubular casts in the urine.

OSHA is proposing to establish or revise occupational exposure limits for five nephrotoxic agents. NIOSH has a REL for one of these substances (hexone), and OSHA is currently regulating two chemicals in this group. These limits were selected based primarily on the results of studies in experimental animals. For example, the limit for hexone is based on results from a study in which rats were continuously exposed by inhalation to 100 or 200 ppm of hexone for 90 days. The lowest effects observed in this study were degeneration and necrosis of the renal tubules, which were associated with the 100 ppm exposures (MacEwen 1971). In a study of effects caused by exposure to ethyl silicate at 88, 50 or 23 ppm, no decrease in kidney weight was seen in rats exposed to this substance 7 hours per day, 5 days per week for 90 days (Pozzani and Carpenter 1951). OSHA preliminarily concludes that the 10-ppm level will provide protection against any nephrotoxic effects, and will reduce the significant risk associated with exposure to these hazardous substances.

Dose-Response Characteristics

Kidney damage, like liver damage, is progressive; only at earlier stages are nephrotoxic effects reversible. The toxicity of the kidney-damaging chemicals included in the group for which OSHA is proposing limits also increases as dose increases. For most nephrotoxins there appears to be a NOEL. Workplace exposures to concentrations of these substances at levels at or below the proposed limits are unlikely to cause kidney effects in most workers. OSHA believes that the nephrotoxic risks being protected against are significant at the current PELs for these substances, because the PELs are often at levels well above the concentration demonstrated to produce the toxic effect. In addition, for three of the chemicals, there have previously been no OSHA limits, and workers could therefore be exposed to levels well above the NOEL.

1,3-DICHLOROPROPENE
CAS: 542–75–6; CHEMICAL FORMULA: $CHCl=CH—CH_2Cl$
H.S. No. 1129

OSHA currently has no limit for 1,3-dichloropropene. The ACGIH has established an 8-hour TLV–TWA of 1 ppm, with a skin notation, for this straw-colored clear liquid with a chloroform-like odor. This compound occurs in the form of two isomers, cis and trans.

In male and female rats, the acute oral LD_{50}'s for a 92-percent mixture of the cis and trans isomers of 1,3-dichloropropene were 713 and 470 mg/kg, respectively; post mortem examination showed liver and kidney damage and, perhaps, evidence of lung injury (Torkelson and Oyen 1977). The dermal LD_{50} in rabbits for a 92-percent undiluted mixture was 504 mg/kg, but a 10-percent solution administered by gavage at a dose of 125 or 250 mg/kg was lethal to some of the animals (Torkelson and Oyen 1977). Contact with the liquid was irritating to the eyes and skin of rabbits (Torkelson and Oyen 1977).

Inhalation exposures to 1,3-dichloropropene vapor concentrations above 2700 ppm produced eye and nasal irritation and severe lung, nasal, kidney, and liver damage in rats (Torkels,n and Oyen 1977). Exposure to 1000 ppm caused eye and nasal irritation, lacrimation, and, if prolonged, unconsciousness; rats exposed to 1000 ppm for 2 hours died, but those exposed for 1 hour survived (Torkelson and Oyen 1977). Guinea pigs exposed to 400 ppm for a single 7-hour period died, while rats exposed similarly survived but had obvious lung con9estion (Torkelson and Oyen 1977). Rats, rabbits, guinea pigs, and dogs were exposed 7 hours/day' 5 days/week for 6 months to 1-ppm or 3-ppm concentrations of 1,3-dichloropropene (Torkelson and Oyen 1977). No adverse effects were observed in any of the animals exposed at 1 ppm. Of the animals exposed at 3 ppm, only male rats showed adverse effects; these animals had reversible cloudy swelling of the renal tubular epithelium (Torkelson and Oyen 1977).

In humans, acute exposures to 1,3-dichloropropene cause skin, eye, and respiratory irritation (Torkelson and Oyen 1977). There are no data on the effects in humans of chronic exposure to this substance.

OSHA is proposing an 8-hour TWA limit of 1 ppm, with a skin notation, for 1,3-dichloropropene. The A9ency preliminarily concludes that this limit will protect workers against the risk of eye and mucous membrane irritation and lung, kidney, and liver damage potentially associated with exposure to this substance at the levels permitted by the absence of any OSHA limit. A skin notation is proposed to protect against 1,3-dichloropropene's ability to be absorbed through the skin. The health evidence forms a reasonable basis for proposing a new limit for 1,3-dichloropropene. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

DICYCLOPENTADIENE
CAS: 77–73–6; Chemical Formula: $C_{10}H_{12}$
H.S. No. 1132

OSHA currently has no limit for dicyclopentadiene (DCPD). The ACGIH recommends a TLV–TWA of 5 mg/m^3. DCPD is a solid at room temperature and has a disagreeable odor.

The health effects associated with exposure to DCPD include mild eye, skin, and respiratory irritation, as well as possible pulmonary and renal damage. By the oral and intraperitoneal routes, DCPD is extremely toxic, with an oral LD_{50} value of 0.35 ml/kg and an intraperitoneal LD_{50} value of 0.31 ml/kg in rats: rat fatalities occurred within 60 minutes of exposure to an unspecified concentration of the saturated vapor (Kinkead, Pozzani, Geary, and Carpenter 1971). However, Gage (1970) regards approximately 660 ppm as the 4-hour LC_{50} in rats and reports that ten 6-hour daily exposures to DCPD at a concentration of 250 ppm were survived only by three of four rats; when the animals were subjected to a concentration of 100 ppm for 15 similar exposures, all survived (Gage 1970). Other species were less susceptible than mice to the effects of DCPD exposure, but they exhibited eye irritation, incoordination, and convulsions preceding death (Kinkead, Pozzani, Geary, and Carpenter 1971).

Kinkead and associates (1971) report that rats exposed repeatedly for 10 days

survived concentrations of 72 or 146 ppm but succumbed at the 332 ppm level, with convulsions, lung hemorrhage and blood in the intestines; female rats also suffered hemorrhage of the thymus. Mice similarly exposed succumbed at all three concentration levels (Kinkead, Pozzani, Geary, and Carpenter 1971). Chronic exposures of 7 hours/day for 89 days produced kidney damage and some pulmonary effects in rats exposed at levels of 35 and 74 ppm; a no-effect level for rats was determined to be below 19.7 ppm. Dogs exposed at concentrations of 9, 23, or 32 ppm on the same regimen exhibited only minimal effects (Kinkead, Pozzani, Geary, and Carpenter 1971).

Human sensory response tests resulted in findings of mild eye and throat irritation in 7 minutes on exposure to 1 ppm DCPD vapor and olfactory fatigue in 24 minutes; a 30-minute exposure to 5.5 ppm produced no olfactory fatigue (ACGIH 1986, p. 194). Subjective complaints of headache during the first 2 months of occupational exposure disappeared during the following 3 months of exposure, suggesting a developed tolerance for this substance (ACGIH 19B6, p. 194).

OSHA is proposing a PEL of 5 ppm TWA for dicyclopentadiene. The Agency preliminarily concludes that this limit will protect workers against the potential risk of kidney injury, pulmonary effects, and irritation potentially associated with workplace exposure to DCPD at the levels permitted by the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for dicyclopentadiene. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

ETHYL SILICATE
CAS: 78–10–4; Chemical Formula: $Si(OC_2H_5)_4$
H.S. No. 1166

OSHA's current permissible exposure limit for ethyl silicate is 100 ppm as an 8-hour TWA. The ACGIH recommends a limit of 10 ppm TWA for this colorless, flammable liquid with a faint odor.

Ethyl silicate has been reported to cause both irritation and systemic toxicity. In guinea pigs and rats, a 60-minute exposure of 2000 ppm was reported as the maximal time-concentration that did not cause serious disturbances; 500 ppm was the maximal no-effect exposure level for an exposure of several hours' duration (Smyth and Seaton 1940). Thirty-day exposures to 400 ppm ethyl silicate for 7 hours/day caused significant mortality in rats and damage to the lungs, liver, and kidney in the surviving animals. Exposures of rats, guinea pigs, and mice to 88, 50, or 23 ppm for 90 days (7 hours/day, 5 days/week) resulted only in decreased kidney weights in mice exposed at the 88-ppm level (Pozzani and Carpenter 1951). In another study, Kasper, McCord, and Frederick (1937) showed that animals exposed to 164 ppm ethyl silicate for 17 8-hour days showed less weight gain than did controls. Rowe and associates (1948) reported that three 7-hour exposures at 1000 ppm were fatal to 4 of 10 rats; similar exposures to 500 ppm caused pronounced kidney changes and slight lung irritation. Four to 10 similar exposures at 250 ppm caused slow weight loss and some lung and renal changes; at 125 ppm, slight to moderate kidney damage was observed (Rowe, Spencer, and Bass 1948).

Smyth and Seaton (1940) reported that exposure to a concentration of 1200 ppm causes lacrimation in humans and that 250 ppm causes eye and nose irritation.

OSHA is proposing a PEL of 10 ppm TWA for ethyl silicate. The Agency preliminarily concludes that this limit is required to protect exposed workers from the risk of renal damage potentially associated with exposures to higher concentrations. OSHA believes that this reduced limit will substantially reduce this risk. The health evidence forms a reasonable basis for proposing a new limit for ethyl silicate. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

HEXACHLOROBUTADIENE
CAS: 87–68–3; Chemical Formula: $CCl_2=CCl-CCl=CCl_2$
H.S. No. 1195

OSHA has no current limit for hexachlorobutadiene (HCBD). The ACGIH recommends a TLV–TWA of 0.02 ppm with a skin notation, and classifies this substance as a suspected human carcinogen (A2). Hexachlorobutadiene is a heavy, clear liquid.

Hexachlorobutadiene has a moderate-to-high acute oral toxicity. The LD_{50}s reported for mice, rats, and guinea pigs are 87, 350, and 90 mg/kg, respectively (Murzakev 1964). Gulko and co-workers reported LD_{50} values of 116 mg/kg for mice and 270 mg/kg for rats (Gulko, Zimina, and Shroit 1965). Skin absorption has been demonstrated in rabbits (Kociba et al. 1977). The dose range reported to be lethal via skin absorption is comparable to that which is lethal via the oral administration route. A single exposure of 133 to 150 ppm via inhalation has been fatal in rats when the exposure lasts for 4 to 7 hours. All rats survived exposures at 161 ppm for 0.88 hour or 34 ppm for 3.3 hours; similar exposure of guinea pigs and cats to the same concentrations resulted in the death of most animals (Kociba et al. 1977). Another inhalation study in rats showed eye and nose irritation, respiratory difficulty, and damage to kidney tissue and adrenal cortex after two 4-hour exposures at 250 ppm; twelve 6-hour exposures to 100 ppm caused eye and nose irritation, respiratory difficulty, weight loss, anemia in the female animals, and kidney and adrenal damage; fifteen 6-hour exposures at 25 ppm caused retarded weight gain in females, respiratory difficulty, and kidney damage; fifteen 6-hour exposures at 10 ppm caused retarded weight gain in females but no systemic injury; and fifteen 6-hour exposures at 5 ppm resulted in no adverse effects (Gage 1970).

Reproductive studies in male and female rats demonstrated multiple toxicological effects, including kidney damage in both sexes and increased liver weight in males, at the high dose level of 20 mg/kg/day. Dietary administration of 20, 2, or 0.2 mg/kg daily had no effect on conception percentages, gestational survival, neonatal survival, neonatal sex ratio, neonatal morphology, or neonatal body weights (except for the high-dose neonates) (Schwetz et al. 1977). Results of lifetime dietary studies suggest that the no effect level for hexachlorobutadiene in rats is 0.2 mg/kg/day, that a clear dose-response relationship exists for HCBD-induced toxicity affecting primarily the kidney, and that carcinogenic effects (i.e., renal neoplasms) result from ingestion of 20 mg/kg/day (Kociba et al. 1977). These authors also reported that HCBD-induced neoplasms occur only at HCBD doses higher than those causing discernible renal injury. The ACGIH states that "HCBD would seem to qualify as a carcinogen of intermediate potency" (ACGIH 1986, p. 299).

OSHA is proposing an 8-hour TWA limit of 0.02 ppm, with a skin notation, for this hazardous substance. Assuming a 10-m^3 per day breathing volume per 8-hour workshift and a 70-kg body weight for humans, this limit corresponds to a daily intake of approximately 0.03 mg/kg. This is about 10 times below the observed no-effect level in rats fed hexachlorobutadiene. The Agency preliminarily concludes that the proposed limit of 0.02 ppm will protect workers exposed to HCBD from the risks of eye, skin, and pulmonary irritation, kidney damage, and renal neoplasms potentially associated with exposure to HCBD at the levels

permitted in the absence of any OSHA limit. In addition, the proposed skin notation will prevent the systemic toxicities that can occur as a result of dermal absorption of HCBD. The health evidence forms a reasonable basis for proposing a new limit for hexachlorobutadiene. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

METHYL ISOBUTYL KETONE
CAS: 108-10-1; CHEMICAL FORMULA: $CH_3COCH_2CH(CH_3)_2$
H.S. No. 1203

OSHA's current 8-hour TWA standard for methyl isobutyl ketone (MIBK) is 100 ppm. The ACGIH has established a TLV-TWA of 50 ppm and a 15-minute STEL of 75 ppm for this substance. NIOSH recommends a TWA of 50 ppm for MIBK, which is a clear liquid with a characteristic odor.

A 4-hour exposure to 4000 ppm killed all exposed rats, but a similar exposure to 2000 ppm was not fatal to these animals (Smyth et al. 1951). Guinea pigs exposed to a MIBK concentration of 10,000 ppm immediately showed signs of irritation (Specht et al. 1940, as cited in ACGIH 1986, p. 402).

MacEwen, Vernst, and Haun (1971) exposed rats, mice, dogs, and monkeys to 100 or 200 ppm MIBK for two weeks and noted no signs of intoxication; however, rats exposed to 100 ppm had heavier kidneys and higher kidney-to-body-weight ratios, and, at 200 ppm, livers were heavier as well. Postmortem examination revealed nephrosis of the proximal tubules.

The same authors (MacEwen, Vernst, and Haun 1971), exposed rhesus monkeys, dogs, and rats continuously for 90 days to MIBK concentrations of 100 ppm. These authors observed no significant changes in clinical chemistry or blood test results, although the rats had heavier kidneys and livers, reversible hyaline dioplet degeneration of the proximal tubules of the kidneys, and some necrosis of the tubules.

Silverman and co-workers (1946) determined that the maximum dose of MIBK tolerable to human volunteers for 8 hours was 100 ppm; at 200 ppm, these subjects found the odor of MIBK objectionable and the vapor irritating. Linair and co-workers (1964) reported that more than half of all workers exposed to 500 ppm of MIBK for 20 to 30 minutes daily, and perhaps to 80 ppm for the remainder of the shift, experienced weakness, loss of appetite, headache, burning eyes, nausea, vomiting, and sore throat; several of these workers also reported insomnia, somnolence, heartburn, and intestinal pain. Some workers had enlarged livers and others had colitis. Clinical test results on these workers were normal.

In a follow-up study on this same group of centrifuge operation workers, Armeli and co-workers (1968) determined that reduction of MIBK levels during the 15 to 30 minutes of centrifuge operation to 100 to 105 ppm, and, for the remainder of the shift, to 50 ppm, had also significantly reduced the symptomatology reported earlier by these workers. However, liver enlargement persisted in two workers, and a few workers continued to report gastrointestinal and nervous system effects.

Elkins (1959) noted that exposure to 100 ppm during boot waterproofing operations caused workers to develop headache and nausea; another similarly exposed group experienced only irritation at 100 ppm.

OSHA is proposing an 8-hour TWA of 50 ppm and a 15-minute STEL of 75 ppm for methyl isobutyl ketone. The Agency preliminarily concludes that these limits will work together to protect workers from the risk of headache, nausea, and irritation, as well as potential kidney and liver effects, determined to be associated with exposures to the levels permitted at a 100-ppm TWA limit. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for methyl isobutyl ketone if the Agency determines that this limit will substantially reduce significant risk.

Preliminary Conclusion for Both Liver and Kidney Toxins

The health effects associated with occupational exposure to the hepato- and nephrotoxins shown in Tables C4–1 and C4–2 can be acute or chronic, reversible or irreversible, temporarily disabling or threatening to life. Workers experiencing chemically induced hepatotoxic or nephrotoxic effects may have enlarged livers, high blood pressure, hormonal imbalances, and/or organ necrosis. The health evidence forms a reasonable basis for proposing new or revised limits for the substances in this section. At the time of the final rule, OSHA will establish new or revised limits if the Agency determines that significant risk will be thereby reduced.

5. Substances for Which Proposed Limits Are Based on Avoidance of Ocular Effects

Introduction

Five of the chemicals for which OSHA is proposing limits have the potential to cause serious ocular effects. Certain chemicals in this group are also sensory irritants and are distinguished from other such irritants by their ability to cause permanent damage to the corneas, lenses, or optic nerves of exposed individuals.

Table C5–1 lists these five chemicals, along with OSHA's current PEL, the NIOSH REL, the ACGIH TLV, and the chemical's CAS number and HS number. In two cases, the proposed 8-hour PELs correspond to the current ACGIH TLV-TWAs for these substances. For one substance, methyl alcohol, OSHA is proposing to retain the existing 8-hour TWA and to add a STEL. In the case of methyl silicate, the Agency proposes to add an 8-hour PEL where none formerly existed. For N-ethylmorpholine, the PEL is being reduced from 20 to 5 ppm.

In two instances, the ACGIH and NIOSH limits differ in some respects. For methyl alcohol, the ACGIH recommends a 200-ppm TWA and a 250-ppm STEL, while NIOSH recommends a 200-ppm TWA and an 800-ppm STEL (15-minute ceiling). For hydrogen sulfide, the ACGIH has established both a STEL and a TWA, while NIOSH recommends only a 10-minute STEL. These differences, and the Agency's decision with regard to them, are discussed further below.

Description of the Health Effects

Damage to the eye caused by exposure to the five chemicals in this group can occur in the form of corneal, lens, retinal, ganglion cell layer, or optic nerve effects. Depending on the severity of the exposure, individual susceptibility, and the particular chemical involved, this damage may be transient, temporarily disabling, or permanently blinding.

Corneal effects. The cornea and conjunctiva are the outer surfaces of the eye and are thus directly exposed to external insults. Since the cornea must maintain transparency to remain functional, scar formation after injury to the cornea can destroy visual function completely. Recent evidence suggests that the transparency of the cornea is maintained by thin inner and outer

boundary layers and that the death of these layers leads to loss of transparency (Potts 1986). The corneal epithelium (outer layer) sometimes regenerates, depending on the depth of the burn or insult and the nature of the toxicant.

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Table C5-1. Substances for Which Limits Are Based on Avoidance of Ocular Effects

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1172 N-Ethylmorpholine	100-74-3	20 ppm TWA, Skin	5 ppm TWA, Skin	--
1209 Hydrogen sulfide	7783-06-4	20 ppm STEL 50 ppm Ceiling	10 ppm TWA 15 ppm STEL	10 ppm Ceiling (10 min)
1252 Methyl alcohol	67-56-1	200 ppm TWA	200 ppm TWA 250 ppm STEL, Skin	200 ppm TWA 800 ppm Ceiling (15 min)
1266 Methyl silicate	681-84-5		1 ppm TWA	--
1282 Naphthalene	91-20-3	10 ppm TWA	10 ppm TWA 15 ppm STEL	--

* OSHA's TWA limits are for 8-hour exposures; its STELs are for the durations specified; and its ceilings are peaks not to be exceeded for any period of time.

** The ACGIH TWA-TLV is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

*** NIOSH TWA limits are for 10-hour exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

BILLING CODE 4510-26-C

Some chemicals, including methyl silicate, produce painful corneal epithelial injuries that have a delayed symptom onset. These substances can continue to cause pain and loss of corneal epithelial cells for several hours after exposure. Typically, there is no discomfort during the actual exposure, but several hours later, the eyes begin to burn, vision blurs, and conjunctival hyperemia, tearing, photophobia, and squinting occur (Grant 1986). Possible mechanisms are enzyme inhibition, denaturing of other proteins, alteration of the DNA, and interference with the mitotic process, so that after a period of exposure, the affected cells die. Although the damaged epithelium may regenerate after this type of injury, the damage can also involve the corneal stroma and endothelium, leading to scarring, vascularization, opacity, and loss of vision. These substances' poor warning properties, i.e., absence of immediate effect, make the establishment of protective exposure limits especially critical for these chemicals.

Exposure to some chemical vapors produces painless edema of the corneal epithelium, which is accompanied by the delayed onset of visual haloes. A chemical that produces these effects is N-ethylmorpholine, a catalyst used to manufacture urethane foam. Painless edema generally occurs in workers who have been exposed for several hours to levels that do not produce discomfort during the exposure itself. The visual effect produced by such exposures consists of the appearance of colored haloes around lights, an effect that is caused by the diffraction of light through the swollen epithelial cells of the eye. Visual haloes are severely distracting and restrict activity substantially, and the mechanism underlying this effect is not well understood (Grant 1986).

Lens effects. The lens is a transparent, avascular tissue surrounded by a thin, collagenous capsule. The major portion of the lens is composed of long, thin fibers that form closely packed, onion-like layers. Transparency is dependent on several factors: A highly ordered cellular arrangement; fiber size, shape, and uniformity; molecular structure; and regularity of fiber packing (Potts 1986). Interference with lens metabolism, transport across cell boundaries, or the integrity of the lens capsule itself can cause a loss of lens transparency and lead to decreased visual acuity (Potts 1986). All such changes in lens transparency are referred to as cataracts.

Retinal effects. The retina is a compact neural structure that is responsible for converting the ocular light image to neural impulses. Because the retina is an internal structure, it is not generally affected by exposure to dust, splashes of liquids, or vapors. However, exposure to certain internally absorbed substances, such as methyl alcohol, may cause changes or lesions in the retina, including retinal edema or hemorrhage. Exposure to a few of these substances can cause acute narrowing of the retinal arteries themselves, which can lead, in turn, to damage to the optic nerve and loss of vision.

Effects on ganglion cell layer and optic nerve. Below the retinal surface layer lies the ganglion cell layer, which is composed of the cell bodies of neurons that extend to the midbrain via the optic nerve. Ganglion cells may be damaged directly when the chemical acts on the cell bodies themselves or secondarily when the toxin destroys the optic nerve. Depending on the severity of the exposure, loss of visual acuity or vision may ensue.

Dose-Response Relationships and Ocular Effects. For most of the chemicals on this list, limits have been established on the basis of health surveys and case reports of occupationally exposed populations. These studies indicate that exposures to concentrations of these substances at levels above the NOE level cause damage or pain to the eyes of exposed workers. In some cases only limited human data are available, and evidence from animal studies or knowledge of a chemical's structural analogy to another chemical known to have ocular effects provides the basis for proposing the limit. Animal models are generally good predictors of ocular effects in humans because the eyes of rodents, especially those of guinea pigs and rabbits, closely resemble human eyes. Thus, animal studies of the effects of acid burns on the eye can be relied on to predict accurately how the chemicals that produce these effects in animals will behave in workers exposed in industrial situations. OSHA's preliminary findings and the available toxicologic data for the chemicals in this group are described below.

N-ETHYLMORPHOLINE
CAS: 100-74-3; Chemical Formula: $C_6H_{13}NO$
H.S. No. 1172

The current OSHA 8-hour TWA PEL is 20 ppm, and the ACGIH TLV is an 8-hour TWA of 5 ppm; both limits have skin notations. NIOSH has no REL for ethylmorpholine. N-Ethylmorpholine is a severe eye irritant. Prolonged exposure to fairly low concentrations of ethylmorpholine causes corneal edema, blue-gray vision, and colored haloes. Typically, vision becomes misty and haloes appear a few hours after workers have been exposed to vapors for a period of hours. Distortion of vision can occur even at levels considerably lower than those that cause irritation (Mastromatteo 1965).

Reversible corneal edema has been observed in workers exposed to 40 ppm or more of ethylmorpholine for several hours (Dernehl 1966). Workers routinely exposed to 3 to 4 ppm and never exposed to concentrations above 11 ppm complained of haloes and foggy vision as well as drowsiness (ACGIH 1987). The irritant effects of N-ethylmorpholine were also seen in a controlled- exposure experiment on volunteer subjects. Ten subjects exposed for 2.5 minutes to 100 ppm experienced irritation of the eyes, nose, and throat; those exposed for 2.5 minutes to 50 ppm experienced slight irritation; and no irritation was reported after exposure for 2.5 minutes to 25 ppm (ACGIH 1986).

OSHA's current 20-ppm PEL for N-ethylmorpholine does not protect exposed workers against the occurrence of corneal edema, workers are especially likely not to be aware of the danger of exposure to N-ethylmorpholine because corneal edema is painless as it is developing and has a delayed onset, and thus no warning occurs during the actual exposure itself. In addition, the effects on visual function of repeatedly exposing the eyes to episodes of corneal edema are not known. OSHA therefore preliminarily concludes that reducing the PEL to 5 ppm as an 8-hour TWA (with a skin notation) is necessary to improve the protection of occupationally exposed individuals from ethylmorpholine's injurious effects on the eyes. This reduction in the PEL will reduce the risk of corneal edema, visual distraction, and impaired vision associated with exposure to this substance. The health evidence forms a reasonable basis for proposing a new limit for N-ethylmorpholine. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

HYDROGEN SULFIDE
CAS: 7783-06-4; Chemical Formula: H_2S
H.S. No. 1209

OSHA's current limits (based on an earlier ANSI standard) for hydrogen sulfide are a 20-ppm STEL (10-minute maximum duration) and a 50-ppm peak limit. The ACGIH has established a TLV of 10 ppm TWA and a 5-ppm STEL for hydrogen sulfide. NIOSH has recommended a 10-minute limit of 10 ppm. Hydrogen sulfide is widely used as

an analytical reagent and in the manufacture of heavy water. However, occupational exposure to hydrogen sulfide occurs most frequently when it is encountered in natural oil or gas deposits or as a byproduct in chemical reactions.

The ACGIH cites several reports (Brieger 1964; Kranenburg and Kessner 1941; Elkins 1950; Masure 1963) of the occurrence of adverse ocular effects, including conjunctivitis, associated with exposure to 20 ppm or less of hydrogen sulfide. The ACGIH also cites a publication by Poda (1966), who reported that the voluntary adoption of 10 ppm as a limit in two heavy-water plants proved to be a satisfactory limit. Based on this information, the ACGIH recommended 10 ppm as a TLV–TWA and 15 ppm as a TLV–STEL.

In recommending a 10-minute limit of 10 ppm for hydrogen sulfide, NIOSH also cited the Poda (1966) report. In addition, NIOSH relied on many of the same studies cited by the ACGIH (1986) to demonstrate the occurrence of ocular damage and eye irritation at exposure levels below 20 ppm. In discussing the ocular effects of hydrogen sulfide exposure, NIOSH (1977i) points out that the effects are predominately acute and, although there are no reports of permanent eye damage, recovery may require several days' absence from work. NIOSH also cites a study done by Flury and Zernik (1931) that reported a case of enduring conjunctivitis in a person exposed to 10 to 15 ppm hydrogen sulfide for 6 hours.

OSHA preliminarily concludes that the current 20-ppm (10-minute) short-term limit and 50-ppm peak limit are inadequate to ensure worker protection against the adverse ocular effects associated with exposure to concentrations of less than 20 ppm hydrogen sulfide, as reported in several studies. OSHA believes that the eye irritation and conjunctivitis associated with such exposures represent a risk to workers, who will be forced to seek medical treatment after exposure and who may also be absent from work. OSHA is proposing to reduce its current limits for hydrogen sulfide to 10 ppm as a TWA and 15 ppm as a STEL. The limit being proposed by OSHA for hydrogen sulfide is a level that has been found to be effective in preventing such effects in the workplace (Poda 1966). Promulgating the proposed limit for hydrogen sulfide will substantially reduce the risk of adverse ocular effects that can occur as a consequence of exposure at the Agency's existing Z table limits. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for hydrogen sulfide if the Agency determines that this limit will substantially reduce significant risk.

METHYL ALCOHOL
CAS: 67–56–1; Chemical Formula: CH_3H
H.S. No. 1252

The OSHA 8-hour TWA limit for methyl alcohol is 200 ppm. The ACGIH has established a 200-ppm TWA and a 250-ppm STEL for methyl alcohol, with a skin notation. NIOSH recommends the same 8-hour limit as the ACGIH, but would supplement the TWA with an 800-ppm rather than a 250-ppm STEL (15-minute ceiling). Methyl alcohol is a widely used industrial solvent whose principal toxic effects are headaches, loss of vision, conjunctivitis, and other adverse effects.

Workers exposed to concentrations of methyl alcohol between 200 and 375 ppm experience severe recurrent headaches, and at levels between 1200 and 8300 ppm, studies by Kingsley and Hirsch (1954; 1955) report that their visual capacity is diminished. The ACGIH recommends the addition of a 250-ppm STEL because it believes that an 8-hour PEL of 200 ppm does not ensure that workers may not be exposed to short-term peaks above the 200 to 375 ppm levels that have been shown to cause severe recurrent headaches in exposed workers.

OSHA believes that observance of the current 200-ppm TWA, supplemented by the proposed STEL of 250 ppm and a skin notation, will eliminate or substantially reduce the risk of severe and recurring headaches and other symptoms potentially associated with industrial exposures to methyl alcohol at such peaks. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for methyl alcohol if the Agency determines that this limit will substantially reduce significant risk.

METHYL SILICATE
CAS: 681–84–5; Chemical Formula: $(CH_3O)_4Si$
H.S. No. 1266

Methyl silicate damages the cornea and produces a delayed response. The current Z tables have no limit for methyl silicate. The ACGIH recommends a 1 ppm 8-hour TWA TLV, and NIOSH has no REL for this substance.

In many cases of methyl silicate exposure, the eyes recover completely, but there are reports of damage to the deep layers of the cornea that caused permanent opacification and, in one worker, loss of the vision in one eye (Grant 1986). It is estimated that exposing humans to methyl silicate at concentrations of 200 to 300 ppm for 15 minutes will produce minimal lesions, and that exposure to 1000 ppm for this period will produce injury requiring hospitalization (ACGIH 1986).

Rabbits exposed to 1000 ppm of methyl silicate in dry air experienced delayed eye burns (ACGIH 1986). Exposure of these animals to approximately 15,000 ppm for 5 minutes caused eye burns, but exposure to this level for 4 minutes caused no appreciable effect. Guinea pigs, which are the test animals with the most sensitive eyes, showed maximum no-effect levels of 135 ppm for 15 minutes, 90 ppm for 1 hour, and 20 ppm for eight 1-hour periods. The latency period for ocular changes was 16 hours for serious effects and up to 3 days for mild involvement (ACGIH 1986). The ACGIH applied a safety factor to the NOEL observed in guinea pigs and established a 1-ppm TWA.

OSHA is proposing a 1-ppm 8-hour limit to reduce the risk of potentially severe ocular effects associated with the uncontrolled occupational exposures currently permitted in the workplace. The health evidence forms a reasonable basis for proposing a new limit for methyl silicate. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

NAPHTHALENE
CAS: 91–20–3; Chemical Formula: $C_{10}H_8$
H.S. No. 1282

OSHA's current exposure limit for naphthalene is 10 ppm as an 8-hour TWA. The ACGIH has established a 10-ppm 8-hour TWA and a 15-ppm 15-minute STEL for this substance, which occurs as a colorless-to-brown solid and has the odor of mothballs.

The oral LD_{50} in rats is 1760 mg/kg (Flury and Zernik 1931).

In humans, the inhalation of naphthalene vapor causes headache, loss of appetite, and nausea (Flury and Zernik 1931; Patty 1949). These authors also report that exposure causes optical neuritis, corneal damage, and kidney injury. Eight of 21 workers exposed for 5 years to unspecified levels of naphthalene developed opacities of the lens of the eye (Ghetti and Mariani 1956). Ingestion of large amounts of naphthalene causes severe hemolytic anemia and hemoglobinuria (Stokinger and Mountain 1963).

The lethal dose in humans has been reported as 50 mg/kg (NIOSH 1977). Concentrations somewhat above 15 ppm are reported to cause marked eye irritation (Robbins 1951).

OSHA is proposing an 8-hour TWA of 10 ppm and a 15-minute STEL of 15 ppm for naphthalene. The Agency preliminarily concludes that these limits will protect workers from the risks of eye irritation and serious ocular effects potentially associated with exposure to the levels permitted by an 8-hour limit alone. The health evidence forms a reasonable basis for proposing a new limit for naphthalene. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

Preliminary Conclusions

OSHA believes that adoption of the limits being proposed for this group of chemicals, which have the potential to cause adverse ocular effects ranging from transient discomfort to permanent blindness, will substantially reduce the risk of visual impairment associated with exposure to these substances. The toxicological bases for the proposed limits include evidence derived from occupationally exposed workers, results obtained in animal models that have been shown to be excellent predictors of human responses, and, in a few instances, evidence that a chemical having a similar chemical structure produces serious adverse ocular effects. The risks being protected against have serious consequences, both in terms of the health and functional capacity of the exposed workers themselves and the safety and well-being of these workers and their co-workers.

The available health evidence for the substances described in this section forms a reasonable basis for proposing the revision or addition of these limits. At the time of the final rule, OSHA will promulgate revised or new limits for these substances if the Agency determines that these limits will reduce significant risk.

6. Substances for Which Proposed Limits Are Based on Avoidance of Respiratory Effects

Introduction

Limits are being proposed for a total of 32 substances or materials for which exposure has been shown to cause adverse respiratory effects. The chemicals in this group cause acute pulmonary edema, alveolar damage, or chronic respiratory damage through the general mechanisms of cellular damage or fibrosis. At sufficient doses, these effects can be permanent, disabling, and life-threatening.

Some of the materials in this group are composites of naturally occurring minerals, and for these, the Agency is proposing limits based on the most hazardous component. For several materials (coal dust, crystalline tripoli, silica, and graphite), OSHA proposes that the TWA be measured as the respirable quartz fraction of the dust, because it is exposure to this fraction that presents the greatest risk to exposed workers. In cases where only a portion of the dust is in the form of a respirable dust, OSHA is proposing limits that are to be measured as the respirable fraction rather than as total dust.

Table C6–1 lists the 32 substances in this group, along with the current OSHA PELs, ACGIH TLVs, NIOSH RELs, CAS numbers, and OSHA HS numbers. There is no current OSHA PEL for ten of these substances. For 15 substances, OSHA is proposing to replace its existing TWA–PELs with different TWA–PELs. For one substance, OSHA is proposing to establish a ceiling limit to replace an existing 8-hour TWA, and for three substances, a lower TWA and a new STEL are proposed. In three instances, OSHA is proposing to establish a STEL to augment its current TWA–PELs. NIOSH has recommended limits for 7 substances in this group.

Description of the Health Effects

The respiratory system is a major route of occupational exposure for toxic substances. Because of the vital nature of pulmonary function, respiratory toxicants present a serious health hazard both from acute and chronic exposures. Acute respiratory disease can be life threatening; however, in most cases, such severe effects are associated only with high exposure levels. (One exception to this general rule is exposure to chemicals that can cause allergic sensitization reactions that lead, in turn, to anaphylactic shock; these substances are discussed in a different section of this preamble describing sensitization reactions.)

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C6-1. Substances for Which Limits Are Based on Avoidance of Respiratory Effects

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1017 Aluminum (pyro powders)	7429-90-5	-	5 mg/m^3 TWA	-
1034 Bismuth telluride (Se-doped)	1304-82-1	-	5 mg/m^3 TWA	-
1080 Chlorine dioxide	10049-04-4	0.1 ppm TWA	0.1 ppm TWA 0.3 ppm STEL	-
1093 Chromium metal	7440-47-3	1 mg/m^3 TWA	0.5 mg/m^3 TWA	-
1096 Coal dust, < 5% quartz	None	2.4 mg/m^3 TWA[a]	2 mg/m^3 TWA[a]	-
1097 Coal dust, > 5% quartz	None	$\frac{10\ mg/m^3}{\%\ SiO_2+2}$	0.1 mg/m^3 TWA[b]	-
1161 Ethyl acrylate	140-88-5	25 ppm TWA, Skin	5 ppm TWA 25 ppm STEL, Skin	-
1177 Ferrovanadium dust	12604-58-9	1 mg/m^3 TWA	1 mg/m^3 TWA 3 mg/m^3 STEL	1 mg/m^3 TWA
1190 Grain dust (oat, wheat, barley)	None	-	4 mg/m^3 TWA	-

C6-1. Substances for Which Limits Are Based on Avoidance of Respiratory Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1191 Graphite, natural, respirable < 1% quartz	7782-42-5	15 mppcf TWA	2.5 mg/m^3 TWA	—
1213 Indium & compounds	7440-74-6	—	0.1 mg/m^3 TWA	—
1215 Iron oxide (dust and fume)	1309-37-1	10 mg/m^3 TWA	5 mg/m^3 TWA	—
1272 Methylene bis (4-Cyclohexyliso-cyanate)	5124-30-1	-	0.01 ppm Ceiling	—
1276 Mica	12003-38-2	20 mppcf TWA	3 mg/m^3 TWA	—
1289 Nitrogen dioxide+	10102-44-0	5 ppm Ceiling	3 ppm TWA 5 ppm STEL	1 ppm Ceiling (15 min)
1300 Oxygen difluoride	7783-41-7	0.05 ppm TWA	0.05 ppm Ceiling	—
1301 Ozone	10028-15-6	0.1 ppm TWA	0.1 ppm TWA 0.3 ppm STEL	—
1303 Paraquat, respirable dust	4685-14-7	0.5 mg/m^3 TWA, Skin	0.1 mg/m^3 TWA	—

C6-1. Substances for Which Limits Are Based on Avoidance of Respiratory Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1354 Silica, crystalline cristobalite	14464-46-1	1/2 value calculated for quartz	0.05 mg/m^3 TWA	50 ug/m^3 TWA
1355 Silica, crystalline quartz, respirable	14808-60-7	$\frac{10\ mg/m^3}{\%\ SiO_2+2}$	0.1 mg/m^3 TWA	50 ug/m^3 TWA
1356 Silica, crystalline tridymite	15468-32-3	1/2 value calculated for quartz	0.05 mg/m^3 TWA	50 ug/m^3 TWA
1357 Silica, crystalline tripoli (as quartz dust)	1317-95-9	$\frac{10\ mg/m^3}{\%\ SiO_2+2}$	0.1 mg/m^3 TWA	50 ug/m^3 TWA
1358 Silica, fused	60676-86-0	$\frac{10\ mg/m^3}{\%\ SiO_2+2}$	0.1 mg/m^3 TWA	—
1363 Soapstone, total dust	None	20 mppcf TWA	6 mg/m^3 TWA	—
1363A Soapstone, respirable dust	None	20 mppcf TWA	3 mg/m^3 TWA	—
1375 Sulfur dioxide	7446-09-5	5 ppm TWA	2 ppm TWA 5 ppm STEL	0.5 ppm TWA
1378 Sulfur tetrafluoride	7783-60-0	—	0.1 ppm Ceiling	—
1381 Talc (non-asbestiform)	14807-96-6	20 mppcf TWA	2 mg/m^3 TWA	—

C6-1. Substances for Which Limits Are Based on Avoidance of Respiratory Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1395 Tin oxide	18282-10-5	-	2 mg/m^3 TWA	--
1409 Trimellitic anhydride	552-30-7	-	0.005 ppm TWA	--
1430A Wood dust, hard wood	--	-	1 mg/m^3 TWA	--
1430B Wood dust, soft wood	--	-	5 mg/m^3 TWA 10 mg/m^3 STEL	-- --

[a] For coal dust, respirable fraction less than 5 percent SiO_2.

[b] For coal dust, respirable fraction more than 5 percent SiO_2.

* OSHA's TWA limits are for 8-hour exposures; its STELs are for the durations specified; and its ceilings are peaks not to be exceeded for any period of time.

** The ACGIH TWA-TLV is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

*** NIOSH TWA limits are for 10-hour exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

+ Proposed limit is the NIOSH REL.

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Chronic pulmonary disease can result from long-term exposure to respiratory toxicants and is potentially crippling because it greatly reduces the quality of life and the productivity of its victims. In addition, the onset of respiratory disease can be insidious, because it may be indicated only by the gradual development of a few nonspecific signs (Petersdorf et al. 1983).

The difficulties of detecting irreversible respiratory effects complicate the prevention of pulmonary disease. Pulmonary function can be evaluated with a variety of tests, including measurements of the vital capacity and of the resting and forced expiratory volumes. However, certain conditions, including emphysema and fibrosis, are difficult to diagnose even with such tests. In addition, these same diseases often continue to progress even after the affected individual has recognized the problem and obtained medical assistance. Furthermore, these diseases may continue to progress even after exposure has ceased, which makes prevention even more vital.

In addition to the threat posed to the general occupational population by respiratory toxins, certain subpopulations, such as persons with impaired lung function caused by asthma, bronchitis, emphysema, and pulmonary fibrosis, are at special risk from the adverse effects of respiratory toxins. Tobacco smoking can cause or aggravate all of the respiratory conditions discussed above and can interact additively or synergistically with respiratory toxins to increase their adverse effects on the pulmonary system. For example, tobacco smoking acts additively with coal dust to diminish pulmonary function. Because tobacco smoke contains nitrogen oxides, cadmium, and ammonia, occupationally exposed workers who smoke have an additional source of exposure to these respiratory toxins (U.S. HEW 1979).

Two general categories of lung injuries are relevant to the group of substances under consideration:

- Damage to cells lining the airways, which results in necrosis (localized areas of dead cells), increased permeability, and edema.
- Production of fibrosis, which may become massive and greatly reduce lung capacity.

Cellular damage resulting in edema and emphysema. A number of substances cause damage to cells lining the airways. This can result in increased permeability of cell membranes and subsequent edema, hemorrhage, and localized necrosis (areas of dead cells). Chronic inhalation of certain chemicals causes destruction of the alveolar septa and results in emphysema. Cellular damage may be either localized or diffuse, depending on the distribution of the toxicant in the lung.

Edema is the release of fluid into the lumen (open spaces of the airways) or alveoli. Serious edema can take several hours to develop so that, in some cases, life-threatening or even fatal exposures can take place without the individual's being aware at the time of exposure of the extent of the damage. Ozone, nitrogen dioxide, and paraquat all cause localized cellular damage leading to edema (Klaassen et al. 1986). Fatalities from pulmonary edema have resulted from exposures to concentrations of nitrogen dioxide of about 200 ppm (Sax 1984). Paraquat is unusual in that it can cause delayed pulmonary damage following exposure, even when exposure occurs via routes other than inhalation (Klaassen et al. 1986).

Necrotic changes can reduce the functional surface area of the lung. One type of lesion often noted in persons exposed to respiratory toxins is benign granulomas, which are localized masses formed when the immune system attempts to sequester a foreign object. Depending on the extent of the damage, these masses may reduce the functional capacity of the lung. Exposure to selenium-doped bismuth telluride has been associated with the production of benign granulomas without fibrosis (Wagner et al. 1974).

Emphysema is caused by a gradual destruction of the cells of the alveolar septa, which causes a loss of elasticity in the lung. A slight degree of emphysema is present in much of the adult population and does not cause any functional impairment. As the disease progresses, however, serious and life-threatening reductions in functional capacity can occur. Once the disease has advanced to the point of serious functional impairment, it is, for the most part, irreversible (Petersdorf et al. 1983). There is evidence that a number of the substances in this group cause emphysema, including sulfur tetrafluoride (ACGIH 1986), ozone, and nitrogen dioxide (Klaassen et al. 1986).

Fibrotic changes. Pulmonary fibrosis was one of the earliest recognized forms of occupational disease. Fibrosis should be distinguished from pneumoconiosis, although these terms are often used interchangeably. Pneumoconiosis is a more general term indicating the presence of a foreign substance in the lung, as determined by radiographic (X-ray) analysis. This definition encompasses a variety of conditions and does not by itself necessarily indicate functional damage (Petersdorf et al. 1983). In contrast, fibrosis is a seriously debilitating disease. One type of fibrosis is interstitial fibrosis, which is a kind of pneumoconiosis characterized by deposition of fibrous tissue in the interstitial spaces between the alveolar membrane and the pulmonary capillary membrane. Interstitial fibrosis greatly reduces the diffusing capacity of the lung and thus causes oxygen deprivation in the body (Guyton 1981). Like emphysema, fibrosis is largely irreversible; it sometimes progresses even in the absence of further exposure (Petersdorf et al. 1983).

Silicosis is a form of interstitial fibrosis that is caused by exposure to respirable silica particles (Klaassen et al. 1986). Exposure to coal dust causes a pneumoconiosis with fibrosis that can be severely debilitating (Petersdorf et al. 1983). In addition, exposure to graphite, mica, and grain dust have all been associated with fibrosis in workers (ACGIH 1986).

Dose-Response Relationships and Respiratory Effects

For most of the substances in this group, permissible exposure limits have been based on health surveys and case reports of occupationally exposed populations. In some cases, animal studies provide the evidence of a substance's toxicity. As is the case for most of the substances for which OSHA is proposing new, reduced, or revised limits, the dose-response curve for respiratory irritants tends to be S-shaped.

Table C6–2 presents dose-response data on the adverse pulmonary effects of representative chemicals in this group, the populations exposed, and the endpoints observed. The following discussions of respiratory toxins present OSHA's preliminary findings for all the substances on Table C6–1 and describe the nature of the risks faced by workers exposed to them.

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TABLE C6-2. Summary of Dose-Response Evidence for Adverse Respiratory Effects

H.S. Number/ Chemical Name	CAS No.	CURRENT PEL*	ACGIH TLV**	NIOSH REL***	Dose-Response Data: Dose/Duration Associated With Observed Effects	Species	Comments
1034 Bismuth telluride (Se-Doped)	1304-82-1		5 mg/m^3 TWA	-	15 mg/m^3 1 year	Dogs Rats Rabbits	Granulomatous lesions in lungs seen after 6 months of exposure.
1096 Coal Dust, < 5% quartz	None	2.4 mg/m^3 TWA	2 mg/m^3 TWA	-	4 mg/m^3 35 years	Humans	Calculated estimate of 10 percent probability of developing pneumoconiosis with fibrosis after 35 years of exposure to coal dust. (Quartz content not identified.)
1097 Coal Dust, > 5% quartz	None	$\frac{10 \text{ mg/m}^3}{\% \text{ SiO}_2+2}$	0.1 mg/m^3 TWA	-			
1190 Grain Dust (oat, wheat, barley)	None		4 mg/m^3 TWA	-	20 mg/m^3	Humans	Chronic bronchitis, shortness of breath, reduced pulmonary function.
					13.9 mg/m^3	Humans	Increased incidence of respiratory symptoms.
					4 mg/m^3	Humans	No increased incidence of respiratory symptoms.
						Humans	Fibrosis and mottling, pneumoconiosis.
1191 Graphite, natural, respirable	7782-42-5	15 mppcf TWA	2.5 mg/m^3 TWA	-	N/A	Humans	Anthracosilicosis, similar to that seen in coal miners.

N/A = Not available.

TABLE C6-2. Summary of Dose-Response Evidence for Adverse Respiratory Effects (continued)

H.S. Number/ Chemical Name	CAS No.	CURRENT PEL*	ACGIH TLV**	NIOSH REL***	Dose-Response Data: Dose/Duration Associated With Observed Effects	Species	Comments
1213 Indium & compounds	7440-74-6	-	0.1 mg/m^3 TWA	-	24-97 mg/m^3	Rats	Widespread alveolar edema following exposure to In_2O_2.
1276 Mica[1]	12003-38-2	20 mppcf TWA	3 mg/m^3 TWA	-	N/A	Humans	Signs and symptoms resembling silicosis and pneumoconiosis in 8 of 57 workers.
1289 Nitrogen dioxide*	10102-44-0	5 ppm Ceiling	3 ppm TWA 5 ppm STEL	1 ppm ceiling (15 min)	N/A	Humans	Fatal pulmonary edema.
					0.4-2.7 ppm Chronic	Humans	Change in pulmonary vital capacity.
1300 Oxygen difluoride	7783-41-7	0.05 ppm TWA	0.05 ppm Ceiling	-	0.5 ppm two 7-hr exposures	Lab. Animals	Lethal to a wide variety of laboratory species, causing pulmonary edema and hemorrhage after several hours of exposure.
1301 Ozone	10028-15-6	0.1 ppm TWA	0.1 ppm TWA 0.3 ppm STEL		1.5 ppm 3 hrs/day	Humans	Significant reduction in pulmonary vital capacity.
					1 ppm 1 day	Mice	Damage to alveolar tissue.
1303 Paraquat, respirable dust	4685-14-7	0.5 mg/m^3 TWA, Skin	0.1 mg/m^3 TWA	-	N/A	Humans	69 accidental deaths from pulmonary injury reported through 1972.

[1]Measured as total dust.

TABLE C6-2. Summary of Dose-Response Evidence for Adverse Respiratory Effects (continued)

H.S. Number/ Chemical Name	CAS No.	CURRENT PEL*	ACGIH TLV**	NIOSH REL***	Dose-Response Data: Dose/Duration Associated With Observed Effects	Species	Comments
1354 Silica, crystalline Cristobalite	14464-46-1	1/2 value for quartz	0.05 mg/m^3 TWA	50 ug/m^3 TWA	0.5 mg/m^3 (as total dust) 2.5 years	Dogs	Cellular infiltration of lung and fibrotic nodules in pulmonary lymph nodes.
1355 Silica, crystalline quartz, respirable	14808-60-7	$\frac{10\ mg/m^3}{\%\ SiO_2+2}$	0.1 mg/m^3 TWA	50 ug/m^3 TWA	0.1 mg/m^3 chronic	Humans	Accelerated loss of pulmonary function over effects of aging alone.
1356 Silica, crystalline Tridymite	15468-32-3	1/2 value for quartz	0.05 mg/m^3 TWA	50 ug/m^3 TWA	N/A	Rats	Most active form of free silica when administered by intratracheal injections.
1357 Silica, crystalline Tripoli (as quartz dust)	1317-95-9	$\frac{10\ mg/m^3}{\%\ SiO_2+2}$	0.1 mg/m^3 TWA	50 ug/m^3 TWA	N/A	Lab. Animals	Progressive nodular fibrosis.
1375 Sulfur dioxide	7446-09-5	5 ppm TWA	2 ppm TWA 5 ppm STEL	0.5 ppm TWA	1 ppm	Humans	Accelerated loss of pulmonary function predicted based on data in smelter workers.

TABLE C6-2. Summary of Dose-Response Evidence for Adverse Respiratory Effects (continued)

H.S. Number/ Chemical Name	CAS No.	CURRENT PEL*	ACGIH TLV**	NIOSH REL***	Dose-Response Data: Dose/Duration Associated With Observed Effects	Species	Comments
1378 Sulfur tetrafluoride	7783-60-0	--	0.1 ppm Ceiling	-	4 ppm 4 hrs/day/ 10 days	Rats	Emphysema, marked clinical signs of respiratory impairment.
1409 Trimellitic anhydride	552-30-7	-	0.005 ppm TWA	-		Rats	Intra-alveolar hemorrhage. (No exposure duration indicated.)

* OSHA's TWA limits are for 8-hour exposures; its STELs are for the durations specified; and its ceilings are peaks not to be exceeded for any period of time.

** The ACGIH TWA-TLV is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

*** NIOSH TWA limits are for 10-hour exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

+ Proposed limit is the NIOSH REL.

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ALUMINUM (PYRO POWDERS)
CAS: 7429-90-5; Chemical Formula: Al
H.S. No. 1017

OSHA currently has no permissible exposure limits for aluminum pyro powders. The ACGIH recommends an 8-hour TLV-TWA of 5 mg/m³.

Aluminum pyro powders have a higher reported toxicity than aluminum metal dusts (Stokinger 1981). Several British studies have examined the effects of this finely flaked aluminum on workers in paints and pyrotechnics plants. Their findings revealed that pulmonary fibrosis may result from exposure to pyro powders, although epidemiologic evidence indicated that additives used to prevent oxidation and agglomeration may have contributed to the incidence and nature of the disease (Edling 1961; Jordan 1961; Mitchell 1961). The ACGIH observes that exposures that have previously caused lung changes in workers are presumed to have been extremely high (ACGIH 1986, p. 22).

OSHA preliminarily concludes that the proposed permissible exposure limit of 5 mg/m³ TWA for aluminum pyro powders will prevent the occurrence of lung changes in exposed workers. The health evidence forms a reasonable basis for proposing a new limit for aluminum pyro powders. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

BISMUTH TELLURIDE (DOPED)
CAS: 1304-82-1; Chemical Formula: Bi_2Te_3
H.S. No. 1034

OSHA has no current limit for doped bismuth telluride. The ACGIH recommends a TLV-TWA of 5 mg/m³ for Bi_2Te_3 that has been doped with selenium sulfide. Bismuth telluride appears as gray, hexagonal platelets; it is also available as ingots or single crystals.

Wagner and co-workers conducted a 1-year study in which rabbits, dogs, and rats were exposed 6 hours/day, 5 days/week to doped bismuth telluride dust (containing 80.04 mol % Bi_2Te_3 and 0.20 mol % SnTe, plus a small stoichiometric excess of Te) of 1.04 um particle diameter at a mean concentration of 15 mg/m³. Small, granulomatous lesions without fibrosis appeared in the lungs of dogs at 6 months. In dogs that were sacrificed 4 months after an 8-month exposure, the lesions had regressed, and the affected lymph nodes were without cellular reaction. Rabbits exhibited similar histologic effects, but with decreased numbers of pulmonary macrophages, no fibrous tissue proliferation, and no cellular or fibrous tissues reaction around the dust deposits in the lymph nodes. The rats showed fewer granulomas but some areas of epithelialization of the alveolar walls. As was true for the other species, the rats showed neither fibrosis nor cellular reaction in the lymph nodes, despite accumulation of the intermetallic dust (Wagner, Madden, Zimber et al. 1974).

A PEL of 5 mg/m³ TWA is proposed for Se-doped bismuth telluride to prevent the occurrence of pulmonary lesions seen in experimental animals. OSHA believes this limit will reduce the risk of these pulmonary effects. The health evidence forms a reasonable basis for proposing a new limit for doped bismuth telluride. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

CHLORINE DIOXIDE
CAS: 10049-04-4; Chemical Formula: ClO_2
H.S. No. 1080

OSHA currently has an 8-hour TWA limit of 0.1 ppm for chlorine dioxide. The ACGIH recommends the same time-weighted average and a 15-minute STEL of 0.3 ppm. Chlorine dioxide is a red-yellow gas at ordinary temperatures.

Rats exposed to 0.1-ppm concentrations of chlorine dioxide for 10 weeks at 5 hours daily showed no adverse effects from exposures. Other data in animals are not available (Dalhamn 1957).

Data on human exposures indicate that marked irritation occurs on inhalation of 5 ppm (no time specified) and that one death occurred at 19 ppm (Elkins 1959). Repeated exposures in an exposed individual have been linked to bronchitis and pronounced emphysema (Petry 1954). Clinical studies conducted by Gloemme and Lundgren (1957) revealed that the majority of workers who had been exposed for 5 years to average concentrations of chlorine dioxide below 0.1 ppm, in combination with about 1.0 ppm chlorine, experienced eye and respiratory irritation and slight bronchitis. Some gastrointestinal irritation was also observed in these workers. Gloemme and Lundgren (1957) attributed all of these effects to elevated short-term exposures involving excursions above the 0.1 ppm level. Ferris et al. (1964) have shown that concentrations occasionally ranging as high as 0.25 ppm were associated with respiratory effects in workers concomitantly exposed to chlorine.

OSHA proposes a 0.1-ppm 8-hour TWA and a 15-minute STEL of 0.3 ppm for chlorine dioxide. The Agency preliminarily concludes that both of these limits are necessary to protect exposed workers against the risk of respiratory, skin, and eye irritation known to occur as a result of short-term exposures above the TWA of 0.1 ppm. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for chlorine dioxide if the Agency determines that this limit will substantially reduce significant risk.

CHROMIUM, METAL
CAS: 7440-47-3
H.S. No. 1093

OSHA currently has an 8-hour TWA of 1 mg/m³ for chromium metal. The ACGIH has established an 8-hour TWA of 0.5 mg/m³ for this element. NIOSH has no REL for the elemental form of chromium. Chromium is a steel-grey metal.

The ACGIH (1986, p. 139) reports that exposure to chromium metal does not cause pulmonary fibrosis or pneumoconiosis. The ACGIH has established the 0.5-mg/m³ TLV-TWA for this metal on the basis of its low order of toxicity (ACGIH 1986, p. 139), and states that this limit "should be adequate to prevent pulmonary disease or other toxic effects."

OSHA is proposing an 8-hour TWA limit of 0.5 mg/m³ for chromium metal. The Agency preliminarily concludes that this limit will protect against the risk of pulmonary or other toxic effects potentially associated with exposure to chromium. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for chromium metal if the Agency determines that this limit will substantially reduce significant risk.

COAL DUST, <5% QUARTZ
COAL DUST, >5% QUARTZ
CAS: None; Chemical Formula: None
H.S. Nos. 1096 and 1097

OSHA has a current formula limit of 10 mg/m³/% SiO_2 + 2 for coal dust containing a respirable quartz fraction greater than 5 percent, and a 2.4-mg/m³ limit for coal dust containing a respirable quartz fraction of less than 5 percent. The ACGIH recommends a TLV-TWA of 0.1 mg/m³ for the respirable quartz fraction of coal dust containing more than 5 percent quartz, and 2 mg/m³ for the respirable dust fraction of coal dust containing less than 5 percent quartz. Coal is a natural dark brown to black-colored solid formed from fossilized plants.

The National Coal Board of the United Kingdom has calculated the

probabilities of developing pneumoconiosis from various concentrations of coal dust, based on statistical data from the first 10 years of an epidemiologic study (1969). The study involved 4,122 men from 20 coal pits, for whom radiologists compared pairs of chest X-rays taken at 10-year intervals. The radiologists classified the degree of pneumoconiosis observed in the X-rays in accordance with the International Labour Office's (ILO) pneumoconiosis classification scheme. For each of the 20 collieries, data concerning mean respirable dust concentrations at the coal face were analyzed. Mean coal, carbon, and quartz content of the dust from each of the collieries were also analyzed. Results indicated that the progression of pneumoconiosis correlates significantly with mean respirable dust concentrations and with the respirable quartz fraction of that dust. The carbon content of the dust did not correlate with the development of pneumoconiosis. Estimates of the risk of developing ILO category 2 or greater pneumoconiosis after a 35-year exposure to coal dust containing a respirable quartz fraction of greater than 5 percent quartz were projected to be a 10-percent probability of disease at a concentration of 6.5 mg/m^3, and a zero probability at a concentration of 2.2 mg/m^3. For exposures to coal dust containing a respirable quartz fraction of less than 5 percent quartz, a 10-percent probability of developing ILO category 1 or greater disease was projected at a concentration of 4 mg/m^3, and a zero probability of disease was projected at 1.6 mg/m^3. In 1979, Gormley confirmed these calculations, but reduced the concentration of dust containing more than 5 percent respirable quartz associated with a zero probability of developing pneumoconiosis to 1 mg/m^3 (ACGIH 1986, p. 142).

OSHA proposes an 8-hour TWA PEL of 0.1 mg/m^3, measured as respirable silica, for coal dust with a respirable quartz fraction containing more than 5 percent quartz, and an 8-hour TWA PEL of 2 mg/m^3 TWA for coal dust with a respirable quartz fraction containing less than 5 percent quartz. The Agency's current formula limit is similar to the 0.1 mg/m^3 limit and, thus, does not represent a change in the limit (see discussion for crystalline silica-quartz below). OSHA is proposing to revise its formula limit for coal dust containing more than 5 percent quartz to 0.1 mg/m^3 to simplify employee exposure monitoring. OSHA also concludes that the reduction in the 8-hour TWA PEL for coal dust containing less than 5 percent quartz will protect exposed workers from the risk of pneumoconiosis at the existing limit for coal dust with this quartz content. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for coal dust if the Agency determines that this limit will substantially reduce significant risk.

ETHYL ACRYLATE
CAS: 140-88-5; Chemical Formula: $CH_2=CHCOOC_2H_5$
H.S. No. 1161

OSHA has a current 8-hour TWA of 25 ppm for ethyl acrylate, with a skin notation. The ACGIH recommends a TLV-TWA of 5 ppm and a TLV-STEL of 15 ppm for ethyl acrylate, which is a colorless liquid. The ACGIH also recommends a skin notation for this substance.

Ethyl acrylate produces irritation of the skin, eyes, mucous membranes, gastrointestinal tract, and respiratory system (Dreisbach 1974). The oral LD_{50} in rats fed this substance is 1020 mg/kg and the 4-hour inhalation LC_{50} for these animals ranges between 1000 ppm and 2000 ppm. In rabbits, the dermal LD_{50} is 1790 mg/kg (Pozzani et al. 1949), and the minimum oral LD_{50} is 280 to 420 mg/kg (Treon et al. 1949). Animal studies also indicate that severe chronic effects may result from exposure to this substance. Rats exposed to levels of 70, 300, or 540 ppm of ethyl acrylate for up to 30 days showed accelerated mortality and pathologic changes in the lungs, liver, and kidneys. In those animals that developed pneumonia, renal and hepatic lesions were also seen. In a parallel study, rats, rabbits and guinea pigs who were subjected to ethyl acrylate concentrations in excess of 75 ppm for fifty, 7-hour inhalation periods exhibited pulmonary edema, degenerative changes in the heart, liver, and kidneys, and death (Treon et al. 1949). Miller et al. (1980) reported that rats and mice exposed to 75 or 225 ppm, 6 hours per day for 30 days, developed nasal lesions and other degenerative inflammatory changes in the nasal structure. In other studies, rats and mice administered 100 or 200 mg/kg ethyl acrylate by gavage 5 times per week for 103 weeks developed inflammation and hyperplasia of the forestomach, in addition to squamous cell carcinomas and papillomas in the same area (NTP 1983, as cited in ACGIH 1986, p. 240). Based on a study by Miller et al. (1985) in which rats and mice exposed to 25 or 75 ppm ethyl acrylate for 6 hours per day, 5 days per week for 27 months developed lesions in the nasal cavity even at the lowest dose, the ACGIH (1986, p. 240) concurs with the American Industrial Hygiene Association (1966) that a 25-ppm limit for ethyl acrylate is too high to prevent irritating effects in exposed humans.

In a study by Nemec and Bauer (1978), human volunteers experienced drowsiness, headache, and nausea after prolonged inhalation exposures at 50 to 75 ppm. Opdyke (1975) reported that the application of a 4-percent concentration of ethyl acrylate produced skin sensitization reactions in 10 out of 24 volunteers.

OSHA is proposing an 8-hour TWA of 5 ppm, a 15-minute STEL, and a skin notation, for ethyl acrylate. The Agency preliminarily concludes that these limits will protect workers from the risk of severe nasal irritation and eye and skin irritation associated with exposure to this substance at the level permitted by OSHA's current limit. The skin notation is necessary to protect against skin sensitization and skin absorption. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for ethyl acrylate if the Agency determines that this limit will substantially reduce significant risk.

FERROVANADIUM DUST
CAS: 12604-58-9; Chemical Formula: FeV
H.S. No. 1177

OSHA currently has a limit of 1 mg/m^3 for ferrovanadium dust. The ACGIH recommends a TLV-TWA limit of 1 mg/m^3 with a TLV-STEL of 3 mg/m^3. The NIOSH-recommended exposure limit for metallic vanadium is 1 mg/m^3 as a 10-hour TWA. Ferrovanadium dust exists as dark, odorless, solid particles.

Soviet studies in animals showed ferrovanadium dust to be less toxic than vanadium pentoxide. Roshchin (1952) reported that no acute intoxication occurred in animals exposed to ferrovanadium dust at concentrations as high as 10,000 mg/m^3, serious chronic pulmonary changes were observed after short-term exposures (one hour) on alternate days for two months to concentrations in the 1000- to 2000-mg/m^3 range. These pulmonary changes consisted of chronic bronchitis and chronic lung inflammation.

OSHA proposes a PEL of 1 mg/m^3 TWA and a STEL of 3 mg/m^3 for ferrovanadium dust, in order to reduce the risk of chronic pulmonary damage potentially associated with exposures to this substance at the elevated short-term levels permitted by the TWA limit alone. The Agency preliminarily concludes that the proposed TWA and STEL will substantially reduce this risk. The health evidence forms a reasonable

basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for ferrovanadium dust if the Agency determines that this limit will substantially reduce significant risk.

GRAIN DUST (OAT, WHEAT, AND BARLEY)
CAS: None; Chemical Formula: None
H.S. No. 1190

A judicial decision has held that there is no OSHA exposure limit for grain dust, and NIOSH also has no REL for this substance. The ACGIH has recommended that worker exposure to grain dust (oat, wheat, and barley) not exceed 4 mg/m³ as total dust.

Exposure to grain dust was reported to result in radiographic changes consistent with pneumoconiosis and fibrosis in 11 of 57 workers handling grain. No exposure information was available (Dunner et al. 1946). Chronic bronchitis was reported in an epidemiologic investigation of workers exposed to concentrations of oat dust ranging from 214 mg/m³ to 308 mg/m³ and of wheat dust ranging from 20.2 to 4:06 mg/m³ (Williams et al. 1964). In a third study by Rankin and do Pico (1980), respirable grain dust concentrations of 20 mg/m³ caused pre- to post-workshift changes of more than 20 percent in the forced expiratory volumes of exposed individuals, indicating reduced lung function. In contrast, lung function changes were infrequent among workers exposed to total grain dust concentrations below 15 mg/m³ (Rankin and do Pico 1980). These same authors reported that grain fever occurred among volunteers exposed to 15 mg/m³ of grain dust for 1 to 3 hours. They also reported a higher incidence of respiratory symptoms among workers exposed to 13.9 mg/m³ TWA. However, acute bronchial symptoms did not appear among workers exposed at or below 4 mg/m³. Although dose response data for grain dusts other than wheat are not available' two recent studies (Darke et al. 1976; Cockcroft et al. 1983) have demonstrated the appropriateness of applying the same PEL to all three of these grain dusts. The work of these authors showed that exposure to any of these dusts produced the same type and degree of respiratory distress.

Based on the observed no-effect level reported by Rankin and do Pico (1966), OSHA is proposing an 8-hour TWA of 4 mg/m³ for grain dust. OSHA preliminarily finds that this PEL for grain dust will greatly reduce the risk of chronic bronchitis, reduced pulmonary function, grain fever, and respiratory symptoms currently faced by workers exposed at uncontrolled levels. The health evidence forms a reasonable basis for proposing a new limit for grain dust. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

GRAPHITE, NATURAL
CAS: 7782-42-5; Chemical Formula: None
H.S. No. 1191

The current OSHA limit for natural graphite (total dust) is 15 million particles per cubic foot (mppcf), which is equivalent to 2.5 mg/m³ as respirable dust (assuming that respirable mass is one-half total particle mass). The ACGIH has recommended a graphite TLV of 2.5 mg/m³ for respirable dust containing less than 1 percent quartz. Graphite is a mineral substance that is best known for its use as the "lead" in pencils.

Early reports established that graphite deposited in the lungs of occupationally exposed workers caused pneumoconiosis (Koopman 1924). Subsequent research described the condition produced by exposure to graphite as anthracosilicosis, a pulmonary condition similar to that seen in coal miners, based on radiographic and histologic examinations in exposed individuals (Harding and Oliver 1949). The fibrotic changes seen in graphite workers appear to be related to the silica content of the graphite; experimental animals that were administered graphite that did not contain silica did not develop fibrotic changes (Ray et al. 1951), while another study found that graphite containing only a small amount of silica produced fibrotic changes in exposed animals (Ottowicz and Paradowski 1961). Radiologic changes were also observed among graphite mine and production workers exposed to graphite containing from 3.6 to 10 percent silica (Pendergass et al. 1967).

Although the role of silica in the development of pneumoconiosis among graphite workers remains unclear, OSHA preliminarily concludes that the current limit of 15 mppcf (2.5 mg/m³ for the respirable fraction of graphite containing less than 1 percent quartz) will protect employees from pneumoconiosis that may develop from exposure to graphite, whether silica-containing or not. However, OSHA is proposing to revise its limit to 2.5 mg/m³ as respirable dust to simplify the monitoring of employee exposures, because the use of impingers and microscopic analyses are not required to measure exposures that are expressed in mg/m³ rather than mppcf.

INDIUM AND COMPOUNDS
CAS: 7440-74-6; Chemical Formula: In
H.S. No. 1213

There is no current OSHA limit for indium and compounds, and NIOSH does not have a REL for these substances. The ACGIH has recommended that exposures to indium not exceed 0.1 mg/m³ as an 8-hour TWA.

Although there is no direct human evidence of the effects of indium compounds, severe effects have been produced by indium exposures in experimental animals. Rats that inhaled the sesquioxide form of indium at airborne concentrations ranging from 24 to 97 mg/m³ daily for a total of 224 hours developed widespread alveolar edema; these histologic lesions did not change after a 12-week post-exposure period. Exposure of animals to indium reduces alveolar clearance and may be associated with chronic respiratory insufficiency, recurrent acute pneumonitis, and death.

Because of the severity of indium-induced injury and the persistence of such injuries, OSHA preliminarily concludes that, in the absence of an exposure limit, exposed employees are placed at risk of developing chronic lung function impairment. The health evidence forms a reasonable basis for proposing a new limit for indium and compounds. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

IRON OXIDE (DUST AND FUME)
CAS: 1309-37-1; CHEMICAL FORMULA: Fe_2O_3
H.S. No. 1215

OSHA currently has an 8-hour TWA limit of 10 mg/m³ for iron oxide fume. The ACGIH has established a limit of 5 mg/m³, measured as iron, total particulate. The appearance of iron oxide depends on the shape and size of the particles and the amount of combined water (Merck Index 1983, p. 579). The fume of iron oxide is red-brown in color.

Animals exposed to iron oxide or to iron oxide mixed with less than 5 percent silica by inhalation or by intratracheal injection did not develop pulmonary fibrosis (Naeslund 1940; Harding, Grout, Durkan et al. 1950). Inhalation of iron oxide dust did not produce lung cancer in mice (Muller and Erhardt 1956).

The evidence in humans is conflicting. Drinker, Warren, and Page (1935) concluded that exposures to iron oxide fume should be maintained below 10 mg/m³, and a U.S. Department of Labor study (1941) found that exposures below 30 mg/m³ were without adverse effect.

There are several studies, on the other hand, that report chest X-ray abnormalities in miners, welders, silver polishers, electrolytic iron oxide workers, foundry workers, and boiler scalers (Doig and McLaughlin 1936; Stewart and Faulds 1934; Doig and McLaughlin 1948; McLaughlin, Grout, Barries, and Harding 1945; Davidson, as cited in ACGIH 1986, p. 325; Prendergass and Leopold 1945; Dunner and Herman 1944) exposed to iron oxide dust or fume. However, the exposures of many of these workers were mixed and included exposure to varying amounts of silica; some of these workers developed disabling penumoconiosis.

McLaughlin (1951), whose opinion on the subject is widely accepted, found that the presence of iron oxide dust or fume in the lung caused a pigmentation (termed siderosis) that was responsible for the changes seen in exposed individuals' chest X-rays. Siderosis is believed not to progress to fibrosis (Fawcitt 1943; Fleischer, Nelson, and Drinker 1945; Hamlin and Weber 1950).

It is believed that 6 to 10 years of exposure to about 15 mg/m³ of iron oxide is needed before siderosis develops (Fawcitt 1943; Fleischer, Nelson, and Drinker 1945; Hamlin and Weber 1950), although no studies are available to correlate exposure levels with X-ray changes.

Some studies have shown that workers with exposures to iron oxide and such other substances as silica, radon gas, diesel exhaust, core oils, and the thermal decomposition products of synthetic resins (Faulds 1957; Dreyfus 1936; Bidstrup 1959; Boyd, Doll, Faulds, and Leiper 1970; Braun, Guillorm, Pierson, and Sadoul 1960; Monlibert and Roubille 1960; Jorgensen 1973; Muller and Erhardt 1956; Kosela, Hernberg, Karava et al. 1976; Gibson, Martin, and Lockington 1978) have a greater risk of developing lung cancer.

The ACGIH states that, "at this time, it is not generally accepted that exposure to iron oxide dust or fume causes cancer in man" (1986, p. 325). A review of the world literature by Stokinger (1984) concluded that exposure to iron oxide *per se* was not carcinogenic.

OSHA is proposing an 8-hour TWA of 5 mg/m³ for iron oxide dust and fume, measured as total particulate (Fe). The Agency preliminarily concludes that this limit will protect workers from the risk of siderosis and its accompanying generalized pulmonary densities associated with exposure at the existing PEL. The Agency believes that this limit will substantially reduce this risk. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for iron oxide if the Agency determines that this limit will substantially reduce significant risk.

METHYLENE BIS-(4-CYCLOHEXYLISOCYANATE)
CAS: 5124-30-1; Chemical Formula: $C_{15}H_{22}N_2O_2$
H.S. No. 1272

OSHA has no current limit for methylene bis-(4-cyclohexylisocyanate). The ACGIH recommends a TLV ceiling of 0.01 ppm for this alicyclic diisocyanate compound.

Methylene bis-(4-cyclohexylisocyanate) is a pulmonary, skin, and eye irritant. The oral LD_{50} in rats is 9.9 g/kg. A 5-percent solution applied to the skin of guinea pigs produced strong erythema and edema, and rabbits treated with 0.1 mg showed severe skin reactions (Younger Laboratories 1965, as cited in ACGIH 1986, p. 392).

Rats inhaling a lethal concentration of 20 ppm for 5 hours exhibited marked respiratory irritation, tremors, and convulsions during exposure, and their lungs revealed severe congestion and edema after death (E.I. du Pont de Nemours and Company 1976, as cited in ACGIH 1986, p. 392). Repeated inhalation exposure at 0.4 ppm produced initial weight loss in rats; exposure at 1.2 ppm caused respiratory irritation and decreased growth (E.I. du Pont de Nemours and Company 1978). Guinea pigs exposed to 0.12 ppm and mice exposed to 0.65 ppm did not exhibit dermal sensitivity (Stadler and Karol 1984). Unlike toluene diisocyanate, which is a sensory irritant, methylene bis-(4-cyclohexylisocyanate) depresses respiration by producing pulmonary irritation, e.g., an exposed mouse showed a 50-percent decrease in respiration rate, along with lung irritation, when exposed to 3.7 ppm of this substance (weyel and Schaffer 1985).

Human exposures to this compound have resulted in skin sensitization but only infrequently in pulmonary sensitization (Emmett 1976; Israeli et al. 1981).

OSHA is proposing a ceiling limit of 0.01 ppm for methylene bis-(4-cyclohexylisocyanate). The Agency preliminarily concludes that this limit will protect workers against the risk of eye, skin, and pulmonary irritation potentially associated with occupational exposures to this substance at the levels permitted in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for methylene bis-(4-cyclohexylisocyanate). At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

MICA
CAS: 12001-26-2; Chemical Formula: $K_2Al_4(Al_2Si_6O_{20})(OH)_4$
H.S. No. 1276

OSHA currently has a PEL of 20 mppcf TWA for mica containing less than 1 percent crystalline silica; this limit is equivalent to a 3-mg/m³ limit. The ACGIH recommends a limit of 3 mg/m³ TWA for the respirable dust of mica containing less than 1 percent quartz. Mica is a colorless, odorless, nonflammable, nonfibrous, water-insoluble silicate occurring in plate form and containing less than 1 percent quartz; it includes nine different species.

OSHA proposes an 8-hour TWA limit of 3 mg/m³ for respirable mica dust containing less than 1 percent quartz; this limit corresponds to the existing 20 mppcf PEL and is in keeping with the Agency's decision to delete mppcf values in favor of respirable dust values expressed in mg/m³. The Agency is proposing to express this and other similar limits as mg/m³ to facilitate employee exposure monitoring.

NITROGEN DIOXIDE
CAS: 10102-44-0; Chemical Formula: NO_2
H.S. No. 1289

Both the ACGIH and NIOSH have recommended occupational limits for nitrogen dioxide. The current ACGIH recommendation is for a 3-ppm TWA and a 5-ppm STEL. The NIOSH REL is 1 ppm as a 15-minute short-term limit. OSHA's current PEL is 5 ppm as a ceiling value.

The previous ACGIH TLV of 5 ppm as a ceiling concentration (the basis for the current OSHA limit) was based primarily on the animal studies of Gray et al. (1952 1954) and Wagner et al. (1965). Gray et al. (1952 1954) demonstrated lung injury among rats exposed for 8 or more weeks to an 8 ppm concentration of a mixture of NO_2 and nitric acid, but these authors did not see such lesions in rats exposed for 6 months to 4 ppm concentrations of this mixture. Wagner et al. (1965) reported transient, mild acute effects and no adverse chronic effects in rats exposed to 1 ppm, 5 ppm, or 25 ppm pure NO_2 for 18 months. The ACGIH's recommendation that the 5 ppm TLV be defined as a ceiling rather than an 8-hour TWA was based on reports that NO_2 accelerated lung tumor development among lung-tumor susceptible mice; in the late 1960s, the ACGIH believed that a TLV-ceiling

value would minimize the risk of accelerating lung tumor development.

The current ACGIH TLVs for NO_2 of a 3-ppm 8-hour TWA and a 5-ppm STEL are based on human studies that indicate that normal respiratory function may be compromised at exposures below the current OSHA ceiling limit of 5 ppm NO_2. In particular, Kosmider et al. (1972) reported a slight reduction in vital capacity and maximum respiratory volume in 70 men exposed to 0.4- to 2.7-ppm concentrations of the oxides of nitrogen 6 to 8 hours daily for 4 to 6 years. These authors also reported an unspecified number of cases of chronic bronchitis among men in this group. Another study by Vigdortschik et al. (1937) reported possible cases of chronic bronchitis and emphysema among 127 workers generally exposed below 2.8 ppm NO_2; these workers were also believed to be exposed to sulfuric acid mist at levels sufficient to cause dental erosion.

The NIOSH REL for NO_2 of 1 ppm as a 15-minute STEL is based on the two human studies discussed above, as well as some human studies involving short-term exposure. Abe (1967) found a 40-percent decrease in effective lung compliance among healthy adult males 30 minutes after a 10-minute exposure to 4- to 5-ppm NO_2. Expiratory and inspiratory maximum viscous resistance also increased after exposure. NIOSH (1976c) concluded that Abe's results "document a definite and undesirable effect" at exposures approaching the current OSHA limit. A significant decrease in carbon monoxide diffusing capacity was observed by Von Nieding et al. (1973) in healthy adults exposed to 5 ppm for 15 minutes. NIOSH also cites the work of Von Nieding et al. and Krekeler (1971), who reported significant increases in airway resistance among 88 chronic bronchitis patients after a 15-minute exposure to a concentration of NO_2 as low as 1.5 ppm. NIOSH (1976c) concluded that the specific concentration of NO_2 required to produce pulmonary changes in normal, healthy adults is unknown, but is "likely to be about the same or perhaps a slightly higher concentration than the one inducing pulmonary changes in humans with existing chronic bronchitis" (1.5 ppm). Therefore, NIOSH recommended a 1-ppm 15-minute short-term limit for nitrogen dioxide. To provide additional support for a short term rather than a TWA limit, NIOSH cites several animal studies that indicate that the toxic effects associated with exposure to NO_2 are primarily determined by peak, and not average, concentrations of exposure.

After reviewing the evidence cited by ACGIH (1986) and NIOSH (1976c), OSHA preliminarily concludes that the current 5-ppm ceiling limit for nitrogen dioxide is not sufficient to protect workers against impairment of pulmonary function. The studies by Abe (1967) and by Von Nieding et al. (1973) clearly show that even brief exposures to levels at or just below the current OSHA limit are associated with measurable declines in pulmonary function. The work of Von Nieding et al. and Krekeler (1971) suggests that workers whose respiratory systems are already compromised will be adversely affected during exposures even to lower concentrations of NO_2. Therefore, OSHA believes it necessary to reduce the current occupational exposure limit of NO_2 to reduce this risk, and is proposing to change the limit for nitrogen dioxide to 1 ppm as a 15-minute short-term limit. Human health studies indicate respiratory effects at levels ranging from 0.4 to 2.8 ppm. Health effects have also been observed following short-term exposures ranging from 1.5 to 5.0 ppm. OSHA therefore concludes that the 3-ppm TWA), 5-ppm (STEL) TLV is not sufficiently protective and proposes that a 1-ppm (STEL) be adopted as the PEL. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for nitrogen dioxide if the Agency determines that this limit will substantially reduce significant risk. OSHA's preliminary feasibility analysis is based on limited data at this exposure level; additional feasibility information is requested from the public.

OXYGEN DIFLOURIDE
CAS: 7783-41-7; Chemical Formula: OF_2
H.S. No. 1300

The current PEL for oxygen difluoride is 0.05 ppm as an 8-hour TWA. NIOSH has no REL for this substance. The ACGIH has established a limit of 0.05 ppm as a ceiling value. The revision of the TLV for oxygen difluoride from an 8-hour TWA to a ceiling value reflects the general position of the ACGIH that ceiling TLVs are more appropriate for chemicals that cause acute but not chronic health effects.

Oxygen difluoride is a substance having extremely high acute toxicity; it is an acute irritant and causes fatal pulmonary edema and hemorrhage in animals exposed to 0.5 ppm for a few hours (ACGIH 1986). A single exposure to 0.1 ppm also had an effect on the lung as evidenced by development in animals of a tolerance to the acute effects of this substance after an isolated exposure. Animals acutely exposed to oxygen difluoride have also exhibited gross changes in the kidney and internal genitalia (LaBelle et al. 1945; Lester and Adams 1965).

Because of the extreme acute toxicity of this compound, OSHA believes that the current TWA-PEL of 0.05 ppm is not sufficiently protective of workers, in that this limit would permit brief periods of high exposure (i.e., at 0.5 ppm or more) that have been associated with severe lung damage. Therefore, to reduce the risk of acute lung damage associated with brief excursion exposures to oxygen difluoride, OSHA proposes to revise its 0.05 TWA-PEL to a ceiling value. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for oxygen difluoride if the Agency determines that this limit will substantially reduce significant risk.

OZONE
CAS: 10028-15-6; Chemical Formula: O_3
H.S. No. 1301

The current OSHA PEL for ozone is 0.1 ppm TWA. In the interval since this limit was adopted in 1971, the ACGIH has recommended that 15-minute short-term exposures to ozone not exceed 0.3 ppm. NIOSH has no REL for ozone.

Ozone is highly injurious and lethal in experimental animals at concentrations as low as a few parts per million (Stokinger 1957). A study in which young mice were exposed to 1 ppm ozone for 1 or 2 days reported damage to alveolar tissue (Bils 1970). Human populations chronically exposed to lower concentrations of ozone have been observed to have changes in lung function. In one study, human volunteers exposed to 0.5 ppm ozone for 3 hours a day, 6 days a week, for 12 weeks showed significant changes in lung function (Jaffe 1967). Other authors reported a 20 percent reduction in timed vital capacity in persons exposed to average concentrations of ozone of 1.5 ppm (range not indicated) for 2 hours (Griswold et al. 1957). Welders exposed to maximal ozone concentrations of 9 ppm were observed to have pulmonary congestion (Kleinfeld and Giel 1956).

OSHA is proposing a STEL based on observations that significant declines in pulmonary function can result from repeated intermittent exposures or even from a single short term exposure (Bils 1970; Jaffe 1967; Griswold et al. 1957). OSHA believes that, in the absence of a STEL, employees will continue to be at risk of the impairment in pulmonary functional capacity associated with short-term exposures that could occur if exposures are controlled only by an 8-hour TWA. Thus the Agency

preliminarily concludes that it is necessary to supplement the existing PEL with the proposed STEL of 0.3 ppm. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for ozone if the Agency determines that this limit will substantially reduce significant risk.

PARAQUAT
CAS: 4685–14–7; Chemical Formula:
H.S. No. 1303

OSHA's current limit for paraquat is 0.5 mg/m³ as an 8-hour TWA, with a skin notation. The ACGIH has established a limit of 0.1 mg/m³ as an 8-hour TWA. Paraquat refers to a group of compounds that are odorless, yellow solids. The principal compounds are: 1,1'-dimethyl–4,4'-bipyridinium; 1,1'-dimethyl-4,4'-bipyridinium bis (methyl sulfate); and 1,1'-dimethyl 4,4'-bipyridinium dichloride.

The toxicity of these compounds depends on the compound's cationic moiety. Acute oral toxicity is reported as 30 mg/kg ion as cation for guinea pigs and 127 mg/kg ion for female rats, while the dermal LD_{50} in rabbits is 240 mg/kg ion (Clark 1964; Clark, McElligott, and Hurst 1966; Elligott 1965). Paraquat can penetrate broken skin after it has broken down the skin's usual barriers (Swan 1969; Clark 1966). By inhalation or intratracheal injection, paraquat is very toxic because of its irritant properties (Gage 1968). Rats exposed once for 6 hours to a concentration of 1 mg/m³ died if the aerosol contained particles with diameters of 3 to 5 microns (Gage 1968). Rats exposed 6 hours/day for 3 weeks to the same aerosol at 0.4 mg/m³ exhibited signs of pulmonary irritation; no effects were observed for the same exposure regimen at 0.1 mg/m³ (Gage 1968).

When the diameter of the particles in the aerosol are not of respirable size, toxicity is greatly reduced. The 4-hour LC_{50} for rats is 6400 mg/kg, and dogs, rats, and guinea pigs tolerated 3 weeks of daily exposures to 100 mg/m³ without apparent pulmonary effect (although nosebleeds were observed) (Palazzolo 1965, as cited in ACGIH 1986, p. 456).

Dietary administration, for 90 days, of doses ranging from 300 to 700 ppm showed dose-related effects ranging from pulmonary edema to intraalveolar hemorrhage and death (Kimbrough and Gaines 1970).

Paraquat's teratogenic potency in mice is low (Bus et al. 1975), although 100 ppm administered in the drinking water of pregnant rats increased postnatal mortality significantly (Bus and Gibson 1975).

In humans, 69 accidental deaths and 81 suicides were attributed to the effects of paraquat exposure up to 1972 (Chipman Chemicals 1972, as cited in ACGIH 1986, p. 456). Bouletreau, Ducluzeau, Bui-Xuan et al. (1977) reported 31 cases of renal insufficiency, and a spray applicator was killed when he absorbed a lethal dose of inadequately diluted paraquat through the skin (Jeros 1978). Workers using a 0.05- to 1-percent solution of paraquat developed skin and mucous membrane irritation but experienced no symptoms of systemic poisoning (Howard 1978). Fugita, Suzuki, and Ochiai (1976) reported 5 cases of reversible kerato-conjunctivitis, with corneal injury, after a month of exposure to paraquat.

OSHA is proposing an 8-hour TWA limit of 0.1 mg/m³ for paraquat, with a skin notation. The Agency preliminarily concludes that this limit will protect workers from the risk of skin, eye, and pulmonary irritation observed in animals exposed to aerosols of respirable size at levels below OSHA's existing PEL for paraquat. The Agency believes that this reduction in permissible exposure level will substantially reduce this risk. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for paraquat if the Agency determines that this limit will substantially reduce significant risk. OSHA is retaining the skin notation for this substance because of its capacity to penetrate the skin.

SILICA, CRYSTALLINE-CRISTOBALITE
CAS: 14464–46–1; Chemical Formula: SiO_2
H.S. No. 1354

The current OSHA PEL for respirable cristobalite is expressed by the formula 5 mg/m³/% SiO_2 measured as total respirable dust (i.e., one-half the value calculated for respirable quartz dust). This formula corresponds to a range of 0.04 to 0.05 mg/m³, measured as silica, for dusts containing 10 to 100 percent tridymite. The ACGIH recommends an 8-hour TWA limit of 0.05 mg/m³, measured as silica dust. The ACGIH limit is based on a study by Gardner (1938) that was confirmed by King, Mahanty, Harrison, and Nagelschmidt (1953). Experimental animals injected with cristobalite showed a more severe response than that produced by quartz, and the fibrosis that followed was diffuse rather than nodular.

Although expressed in different units, the current ACGIH and OSHA limits for cristobalite are comparable. The ACGIH's mg/m³ limit, adopted in 1985, does not reflect a re-evaluation of cristobalite's toxicity but was adopted merely to simplify the monitoring of cristobalite dust concentrations.

OSHA is proposing to replace its limit for cristobalite, which is expressed as the formula presented above, with a numerically equivalent limit of 0.05 mg/m³; the Agency is making this change to simplify employee exposure monitoring.

SILICA, CRYSTALLINE—QUARTZ
CAS: 14808–60–7; Chemical Formula: None
H.S. No. 1355

The current OSHA limit for silica-containing dusts is a respirable dust limit expressed as the following formula:

(10 mg/m³)/(% respirable quartz + 2).

The ACGIH formerly also expressed its silica limit in terms of this formula. However, the current ACGIH TLV is 0.1 mg/m³, measured as respirable quartz dust. The ACGIH does not see this change in the value of its limit for occupational exposure to silica as significant; instead, the ACGIH made this change to conform its limit for this dust to its TLVs for other dusts. If the OSHA formula is used to calculate a limit for a dust containing 100 percent quartz, the limit would be 0.098 mg/m³, not appreciably different from the ACGIH's revised limit of 0.1 mg/m³ for respirable quartz dust. For quartz dusts containing less than 100 percent free silica, the current OSHA formula would yield a limit of, for example, 0.83 mg/m³ for respirable dust containing 10 percent quartz. This result is only slightly more stringent than the ACGIH's TLV of 0.1 mg/m³. For cristobalite and tridymite, the OSHA formula and the ACGIH limits yield approximately the same results: both are approximately one-half the limit established by these two entities for quartz dust (see discussions below).

Occupational exposure to free silica has been known for many years to produce silicosis, a chronic, disabling lung disease characterized by the formation of silica-containing nodules of scar tissue in the lungs. Simple silicosis, in which the nodules are less than 1 cm in diameter (as measured on chest x-ray films) is generally asymptomatic but can be slowly progressive, even in the absence of continued exposure. Complicated silicosis (i.e., nodules greater than 1 cm in diameter) is more often associated with disability and can also progress in the absence of continuing exposure.

The health basis underlying the ACGIH's limit for crystalline silica is the work of Russell (1929), which suggested that a limit of 10 mppcf would protect workers from the effects of exposure to granite dust; a study by Ayer (1969) demonstrated that 10 mppcf of granite

dust is approximately equal to 0.1 mg/m³ of respirable quartz dust (ACGIH 1986).

NIOSH has recommended an exposure limit of 0.05 mg/m³ as respirable free silica for all crystalline forms of silica. As applied to cristobalite and tridymite, the NIOSH REL is 0.05 mg/m³, the same as the ACGIH TLV, but NIOSH's 0.05 mg/m³ REL for quartz dust is one-half the value of the ACGIH TLV for quartz dust. To support its more stringent REL for quartz dust, NIOSH cites the work of Hosey et al. (1957), which reported that no new cases of silicosis occurred in workers in Vermont granite sheds who were generally exposed to 0.05 mg/m³ or less of granite dust. The recommendation was also partly based on studies by Theriault, Burgess et al. (1974); Theriault, Peters and Fine (1974); and Theriault, Peters and Johnson (1974) that found that annual declines in pulmonary function and abnormal chest x-rays occurred among 192 granite shed workers exposed to an average quartz concentration of 0.05 mg/m³. NIOSH noted that the exposure estimates reported in the Theriault studies probably failed to account for the higher exposures that probably occurred in the years before exposure sampling was initiated and thus that Theriault's exposure data may have understated average exposures to quartz. Thus, NIOSH believes that the exposures responsible for the declines in pulmonary function were actually above 0.05 mg/m³. The ACGIH (1986) found NIOSH's reasoning unpersuasive, citing a report by Graham et al. (1981), who measured the pulmonary function of the same group of workers studied by Theriault et al., and found, in contrast to Theriault, that these workers experienced "an overall increase in FVC and FEV" (ACGIH 1986).

The 0.1-mg/m³ TLV represents no real change from the existing PEL. However, the use of an mg/m³ unit will simplify sampling procedures by using recently developed technologies which in turn will reduce the cost of air sampling to evaluate potential silica exposures. NIOSH admits to significant error in the exposure estimates used to establish its 0.05-mg/m³ REL, and OSHA believes that use of this limit will introduce feasibility problems. Before this REL can be considered, for adoption as an OSHA limit, a more detailed analysis would be required. OSHA therefore proposes that 0.1 mg/m³ be adopted as the PEL.

SILICA, CRYSTALLINE-TRIDYMITE
CAS: 15468-32-3; Chemical Formula: SiO_2
H.S. No. 1356

The current OSHA PEL for respirable tridymite is expressed by the formula 5 mg/m³/% SiO_2 measured as total respirable dust (i.e., one-half the value calculated for respirable quartz dust). This formula corresponds to a range of 0.04 to 0.05 mg/m³, measured as silica, for dusts containing 10 to 100 percent tridymite. The ACGIH recommends an 8-hour TWA limit of 0.05 mg/m³, measured as silica dust. The ACGIH limit is based on a study conducted by King, Mahanty, Harrison, and Nagelschmidt (1953) that found tridymite to be the most active of the free silica forms when injected intratracheally into rats.

Although expressed in different units, the current ACGIH and OSHA limits for tridymite are comparable. The ACGIH's mg/m³ limit, adopted in 1985, does not reflect a re-evaluation of tridymite's toxicity but was adopted merely to simplify the monitoring of tridymite dust concentrations. OSHA is proposing to replace its limit for tridymite, which is expressed as the formula presented above, with a numerically equivalent limit of 0.05 mg/m³; the Agency is making this change to simplify employee exposure monitoring.

SILICA, CRYSTALLINE-TRIPOLI
CAS: 1317-95-9; Chemical Formula: SiO_2
H.S. No. 1357

Tripoli is a colorless microcrystalline form of quartz. Although OSHA's Table Z-2 does not specifically indicate a limit for tripoli, OSHA currently 10 mg/m³/% SiO_2+2 specifies a limit for crystalline quartz based on the formula measured as total respirable dust. Expressed as mg/m³, this limit corresponds to a limit in the range of 0.08 to 0.1 mg/m³ for respirable dust containing from 10 to 100 percent silica. The 8-hour TWA ACGIH limit for tripoli is 0.1 mg/m³, measured as respirable silica dust. This limit was adopted by the ACGIH in 1985 to simplify the monitoring of quartz dust concentrations. Thus, this revision does not represent a re-evaluation of toxicity data for tripoli.

OSHA is proposing to replace its limit for quartz, which is expressed as the formula presented above, with a numerically equivalent limit of 0.1 mg/m³ as total respirable dust, and to add the same limit for tripoli.

SILICA, FUSED
CAS: 60676-86-0; Chemical Formula: SiO_2
H.S. No. 1358

Fused silica is a colorless, odorless solid that is a form of quartz. As such, it is currently covered by OSHA's limit for quartz (Table Z-3). Exposure to fused silica has long been known to cause the fibrogenic lung disease, silicosis. OSHA's current limit for quartz dust is the formula 10 mg/m³/% SiO_2+2 measured as total respirable dust. This limit corresponds to a respirable quartz concentration ranging from 0.08 to 0.1 mg/m³, measured as free silica. The ACGIH recommends an 8-hour TWA limit of 0.1 mg/m³, measured as free silica; the ACGIH adopted this limit in 1985 to simplify the monitoring of quartz dust concentrations. Thus, this revision does not represent a re-evaluation of the toxicity data for fused silica.

OSHA is proposing to replace its limit for fused silica, which is expressed as the formula presented above, with a numerically equivalent limit of 0.1 mg/m³ as total respirable dust; the Agency is making this change to simplify employee exposure monitoring.

SOAPSTONE, TOTAL DUST
SOAPSTONE, RESPIRABLE DUST
CAS: None; Chemical Formula: 3 Mg-4 SiO_2-H_2O
H.S. No. 1363 (total dust)
H.S. No. 1363A (respirable dust)

OSHA's current exposure limit for soapstone total dust is 20 mppcf (6 mg/m³), and the Agency has no separate limit for the respirable fraction. The ACGIH has established two TLV-TWAs for these two forms of soapstone: 6 mg/m³ for total dust, and 3 mg/m³ for the respirable fraction, both measured as total dust or respirable dust containing less than 1 percent quartz. Because the ratio of total dust mass to the mass of the respirable fraction is 2:1 (ACGIH 1984, p. 480), the 6-mg/m³ total dust limit automatically implies a 3-mg/m³ limit for the respirable fraction.

A study by Dreessen and DallaValle (1935) of mill workers exposed to soapstone showed lung changes in these workers, but it is believed that the dusts involved in these exposures were actually steatite talc, which had a tremolite content of 10 percent. Experiments by Miller and Sayers (1941) showed no measurable toxic effects in guinea pigs injected intraperitoneally with various samples of soapstone.

OSHA is proposing to express the limit for soapstone total dust in mg/m³, rather than mppcf, to simplify employee sampling and analysis. The total dust limit of 6 mg/m³ is equivalent to the previous limit of 20 mppcf, and the new limit of 3 mg/m³ for respirable dust is actually implicit in the total dust limit.

SULFUR DIOXIDE
CAS: 7446-09-5; Chemical Formula: SO_2
H.S. No. 1375

OSHA's current limit for sulfur dioxide is 5-ppm TWA. ACGIH has recommended a 2-ppm TLV-TWA and a 5-ppm STEL. In its written testimony at

OSHA's 1977 hearing on sulfur dioxide, NIOSH recommended a 10-hour TWA of 0.5 ppm (NIOSH 1977).

ACGIH cites one epidemiologic study (Scalpe 1964) showing an increased incidence of respiratory symptoms among pulpmill workers exposed to 10 to 20 ppm sulfur dioxide. Another study by Ferris et al. (1967) found a high incidence (30 percent) of respiratory disease among pulpmill workers exposed to 2 to 13 ppm; however, this was not statistically different from the incidence of disease in a group of workers at a nearby papermill. Kehoe et al. (1932) also found an increased incidence of respiratory irritation among refrigeration workers exposed to 20 to 30 ppm. Weir et al. (1972) exposed 12 healthy subjects to sulfur dioxide continuously for 120 hours. No subjective complaints or decline in respiratory function was found in subjects exposed to 0.3 or 1 ppm. Slight increases in airway resistance resulted from exposure to 3 ppm. Based on these data, the ACGIH recommended a TLV–TWA of 2 ppm and a STEL of 5 ppm.

In recommending a 0.5-ppm TWA limit. NIOSH (1977j) cited four epidemiological studies that became available after publication of their criteria document (NIOSH 1974b). Archer and Gillam (1977) found increases in the incidence of respiratory disease and reductions in pulmonary function among workers exposed to 0.4 to 4 ppm (mean = 2 ppm) for many years. Smith et al. (1977) reported pulmonary function decrements among workers exposed to between 1 and 4 ppm sulfur dioxide; the decrements were statistically significant compared to workers whose mean exposures were less than 1 ppm. A third study (Ministry of Health 1976) also reported increases in respiratory disease incidence among smelter workers exposed to an average of 2.5 ppm for 10 years or more. A fourth study reported finding no definite effects among 10,000 workers exposed to mean sulfur dioxide levels of 0.35 ppm. Based on these studies, NIOSH (1977j) revised its original recommendation from 2 ppm TWA to a 0.5-ppm TWA.

The 2-ppm TLV–TWA is based on limited data showing no effects from exposures at levels less than 1 ppm. The 0.5-ppm REL is based on additional information indicating respiratory disease associated with exposures ranging from 0.4 to 4 ppm, with no observed effects at 0.35 ppm. Although these findings suggest that the ACGIH TLV may not be sufficiently protective, OSHA is concerned that the NIOSH limit may present feasibility problems. The Agency's preliminary feasibility analysis has considered only a PEL of 2 ppm. At this time, OSHA tentatively proposes that a 2-ppm 8-hour TWA, supplemented by a 5-ppm STEL, be adopted as the PEL. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for sulfur dioxide if the Agency determines that this limit will substantially reduce significant risk. The Agency specifically solicits comments on feasibility and on any evidence related to adverse health effects at exposures at or below these levels. The proposed 2-ppm limit will be reconsidered in light of the feasibility and health effects data obtained prior to the final rulemaking.

SULFUR TETRAFLUORIDE
CAS: 7783–60–0; Chemical Formula: SF_4
H.S. No. 1378

OSHA's current Z tables have no exposure limits for sulfur tetrafluoride. The ACGIH recommends 0.1 ppm as a ceiling limit. Sulfur tetrafluoride is a colorless, non-combustible gas.

On contact with moisture, sulfur tetrafluoride produces sulfur dioxide and hydrogen fluoride (HF) (Lester 1971, as cited in ACGIH 1986, p. 546), and it is the release of HF that is primarily responsible for sulfur tetrafluoride's toxic effects (Zapp 1971, as cited in ACGIH 1986, p. 546). A du Pont (1961) study of rats exposed for 4 hours to 4 ppm sulfur tetrafluoride over a period of 10 days reported that the animals demonstrated nasal discharge, difficulties in breathing, and weakness. Autopsies of these animals revealed evidence of emphysema, but those rats surviving exposure and given a 2-week rest period after exposure showed no significant pathological changes. In the same study by du Pont, a 4-hour exposure to 20 ppm sulfur tetrafluoride proved lethal to one of two rats. In a study by Clayton (1962), irregular breathing and signs of irritation were observed at exposures of 20 ppm and lower; animals receiving lethal amounts of sulfur tetrafluoride showed pulmonary edema on autopsy, and those with sub-lethal exposures demonstrated no pathologic changes 14 days later.

OSHA is proposing a 0.1-ppm ceiling limit for this highly toxic gas. The Agency preliminarily concludes that establishing this limit for this previously unregulated chemical will reduce the risk of chronic respiratory effects potentially associated with exposure to the chemical at the levels permitted by the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for sulfur tetrafluoride. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

TALC (non-asbestiform)
CAS: 14807–96–6: Chemical Formula: H_2O_3Si 3/4Mg
H.S. No. 1381

The current OSHA PEL for non-asbestiform talc is 20 million particles per cubic foot of air (mppcf) as an 8-hour TWA; this is roughly equivalent to 3 mg/m^3. The ACGIH recommends a TLV–TWA of 2 mg/m^3 (15 mppcf) for talc, measured as respirable dust. Talc is a white to gray-white, fine powder.

The health effects evidence for talc is complicated by the fact that talcs contain amphiboles and other minerals, in addition to platiform talc crystals; adverse health effects appear to be related to the non-platiform content (that is, to the fiber content) of the talc in question (ACGIH 1986, p. 550). There are conflicting views over the extent to which the non-platiform constituents are asbestos; however, no health effects information is available that is specifically related to non-asbestiform talc (ACGIH 1986, p. 550).

Numerous epidemiological studies have documented the effects on workers of long-term exposures to talc. In 1942, Porro et al. (cited in Patty 1981) published a report in which 15 cases of talc pneumoconiosis, including 5 postmortem examinations, showed that asbestotic bodies were almost always present in fibrotic areas of the lungs of those workers with talcosis. Siegal and his colleagues (1943, as cited in Patty 1981) noted that an advanced fibrosis incidence rate of 14.5 percent existed among a group of 221 talc miners and millers. These workers were primarily exposed to fibrous talc, which was held to be responsible for the pathology of the asbestos-like lung lesions. A study by McLaughlin et al. (1949, as cited in Patty 1981) revealed that talc-induced pneumoconiosis was caused by the fibrous varieties of talc; in animal studies by Schepers and Durkin (1955, as cited in Patty 1981), the degree of fibrosis in the lung tissue was found to be a function of the length of the talc fibers, rather than of the composition of the talc itself. A paper by Kleinfeld et al. in 1963 (as cited in Patty 1981) reported that postmortem examinations on 6 talc industry workers showed that bodies found in the lung bronchioles or embedded in fibrous tissue were indistinguishable from the asbestos bodies seen in cases of asbestosis.

Kleinfeld et al. (1967) later conducted a cohort study of 220 workers employed in a mine that produced talc with a

tremolite and anthophyllite content. Of the 91 deaths in this group, 10 resulted from respiratory cancer and 28 were attributed to pneumoconiosis. The proportional mortality from respiratory cancer was four times the expected rate. In 1974, when Kleinfeld performed a follow-up study of this group, which at that time consisted of 260 workers (108 deaths), he found significant differences between expected and observed mortality in the period 1950 to 1954, but not during 1960 to 1969. This investigator attributed this finding to the reduction in talc dust counts (from averages of 25 to 73 mppcf in the years 1948 to 1965 to averages of 9 to 43 mppcf in the period 1966 to 1969). This study also showed a decrease of greater than 50 percent in deaths due to pneumoconiosis in the 1965-to-1969 time period.

Studies by NIOSH (Dement and Zumwald 1978) of 398 white male workers employed between 1947 and 1959 in the talc industries found that 74 of these men had died. Bronchogenic cancer was the cause of death in 9 men, whereas only 3.3 deaths from this cause would have been expected. Non-malignant respiratory disease (NMRD) exclusive of influenza, pneumonia, and tuberculosis accounted for 3 deaths; 1.5 would have been expected. From these data, NIOSH concluded that a significant increase in mortality due to bronchogenic cancer and NMRD had occurred from occupational exposure to talc dust. NIOSH's report also included a morbidity study of 12 talc industry workers, currently employed, in which chest X-rays, lung function tests, and questionnaires were used. This study concluded that a higher prevalence of cough, phlegm, dyspnea, and irregular opacities in chest X-rays existed in these workers than in potash miners; instances of pleural thickening and calcification were greater than in coal and potash miners; and the pulmonary function of talc workers overall was reduced in comparison with that of coal and potash miners employed for the same length of time. The reductions in pulmonary function were dose- and duration-related.

The ACGIH (1986, p. 552) concludes that serious health effects have been associated in the past (i.e., prior to 1945) with exposures to amphibole-containing talc. However, the ACGIH believes that the introduction of mining improvements has all but eliminated "the excess of death rates from pneumoconiosis and lung cancer" (ACGIH 1986, p. 552).

Two recent studies of talc exposures (Rubino, Scansetti et al. 1976; Selevan, Dement et al. 1979) are available. The Rubino et al. study found that miners and millers exposed to an average of 849 to 8470 mppcf-years (miners) and 76 to 651 mppcf-years (millers) showed no increase in the number of observed (compared to expected) deaths from causes other than silicosis. These authors concluded that the disease-causing factor in these workers was silica rather than talc (Rubino, Scansetti et al. 1976).

Selevan and Dement's studies (1979) of 392 workers exposed to talc in five mines found non-malignant respiratory deaths for millers to be almost eight times the expected rate, while miners experienced more than three times the expected mortality rate for these same diseases. The ACGIH (1986, p. 552) concludes that the Selevan and Dement study is incomplete because confounders were not adequately identified and controlled for.

OSHA is proposing an 8-hour TWA limit of 2 mg/m^3 for the respirable dust of talc containing no asbestos fibers and less than 1 percent silica. The Agency preliminarily concludes that this limit will protect workers from the risk of non-malignant respiratory effects associated with exposure to the dust of non-fibrous talc.

With regard to the finding by NIOSH (Dement and Zumwald 1978) of excess cancer deaths among talc workers, OSHA is currently reviewing data describing the effects of exposure to non-asbestiform varieties of mineral fibers that are found in talc deposits. OSHA is considering a separate rulemaking to address this issue.

In addition, OSHA is proposing to express the limit for talc in mg/m^3 rather than mppcf, to facilitate employee exposure monitoring. The Agency is proposing this change for all of its Z table limits that are currently expressed as mppcf.

TIN OXIDE
CAS: 7440-31-5; Chemical Formula: SnO
H.S. No. 1395

OSHA currently has no exposure limit for tin oxide. The ACGIH recommends an exposure limit of 2 mg/m^3 as an 8-hour TWA. Tin oxide may be a white or yellow-brown powder.

Injection of tin dust intraperitoneally into guinea pigs resulted in a non-specific, well-vascularized chronic granulomatous reaction (Oyanguren, Haddad, and Maass 1958). Chronic exposure to tin oxide fume and dust results in stannosis, a form of pneumoconiosis. The fume is considered a more important source of stannosis than the dust (Dundon and Hughes 1950), but other authorities consider the quality of the dust and the duration of exposure equally important (Robertson and Whittaker 1954). The onset of the symptoms of stannosis may be delayed for years; the appearance of the condition is signalled by the onset of difficulty in breathing. One worker who had been exposed to unspecified tin oxide levels for 22 years was tested for stannosis and registered a vital breathing capacity 70 percent of normal and a maximal breathing capacity 61 percent of the predicted value (Spencer and Wycoff 1954).

More than 150 cases of stannosis have been reported in the world literature (Robertson and Whittaker 1954), and five cases were reported in the United States before 1954. No cases of massive fibrosis caused by exposure to tin oxide dust or fume have been reported (ACGIH 1986, p. 574).

OSHA is proposing an 8-hour TWA of 2 mg/m^3 for tin oxide dust and fume. The Agency preliminarily concludes that this limit will protect workers from the risk of reduced pulmonary capacity and stannosis associated with exposure to this substance in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for tin oxide. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

TRIMELLITIC ANHYDRIDE (TMAN)
CAS: 552-30-7; Chemical Formula: $C_9H_4O_5$
H.S. No. 1409

OSHA has no exposure limit for trimellitic anhydride. In 1981, the ACGIH set 0.005 ppm (0.04 mg/m^3 as the 8-hour TWA limit for this substance. Trimellitic anhydride is a colorless solid.

Exposure to trimellitic anhydride (TMAN) causes irritation of the eyes, nose, skin, and pulmonary tract. NIOSH (1978) reported in a Current Intelligence Bulletin that trimellitic anhydride should be considered an extremely toxic workplace hazard, because exposure to it can cause noncardiac pulmonary edema and immunological sensitization, as well as upper respiratory tract irritation.

Pulmonary edema has occurred in workers exposed to TMAN at unreported air concentrations; the development of pulmonary edema in these workers without upper respiratory tract irritation suggests that TMAN is a sensitizer (Rice et al. 1977). Zeiss and colleagues (1977) described TMAN-related illnesses among a group of workers synthesizing TMAN. These authors believe there are three separate syndromes associated with TMAN exposure: Rhinitis/asthma; a flu-like condition; and irritation of the upper

respiratory tract. Another case of TMAN-related occupational sensitization occurred in a worker exposed during the application of an epoxy resin coating (Fawcett et al. 1977).

At levels averaging 1.5 and 2.8 mg/m³ in two processes, NIOSH reported that employees reported eye and nose irritation, shortness of breath, coughing, nausea, headache, skin irritation, and throat irritation (NIOSH 1974). Pulmonary hemorrhage and hemolytic anemia have been reported in workers exposed to TMAN at unspecified levels (Ahmed et al. 1979).

Rats have shown intralveolar hemorrhage after TMAN exposures to concentrations of 0.01 ppm.

OSHA is proposing to regulate trimellitic anhydride to an 8-hour TWA level of 0.005 ppm. The Agency preliminarily concludes that this limit will protect workers from the severe pulmonary effects, sensitization and skin and upper respiratory tract irritation observed in workers exposed to this extremely toxic substance. OSHA believes that this limit will substantially reduce this risk, which is presently not controlled due to the absence of any OSHA PEL. The health evidence forms a reasonable basis for proposing a new limit for trimellitic anhydride. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

WOOD DUST
CAS: None; Chemical Formula: None
H.S. No. 1430A (Hard Wood)
H.S. No. 1430B (Soft Wood)

Before 1980, OSHA regulated wood dust under its nuisance dust standard of 15 mg/m³ (29 CFR 1910.1000, Table Z–3). However, in an enforcement proceeding, wood dust was held not to be an inert mineral dust, and the Agency has subsequently not regulated this substance. Consequently, OSHA has no current PEL for hard wood or soft wood dust. The ACGIH recommends a TLV–TWA of 1 mg/m³ for hard wood dust, and a TLV–TWA of 5 mg/m³ and STEL of 10 mg/m³ for soft wood dust. Wood dust is defined as any wood particles arising from the processing or handing of woods. Hard woods derive from the deciduous broad-leaved flowering species of trees, and soft woods include the coniferous species that do not shed their leaves in the winter.

Exposure to wood dust has long been associated with a variety of adverse health effects, including dermatitis, allergic respiratory effects, mucosal and non-allergic respiratory effects, and cancer. The toxicity data in animals are limited, particularly with regard to exposure to wood dust alone.

Animal Studies

Groups of male guinea pigs were injected intratracheally with suspensions containing 75 mg of sheesham or mango wood dust or of hemp or bagasse fibers, or 20 mg of jute fiber (Bhattacharjee et al. 1979; Bhattacharjee and Zaidi 1982). Animals were sacrificed serially at intervals up to 90 days after injection. Lung examination revealed that, at 90 days, Grade I fibrosis of the lungs had occurred in the guinea pigs injected with mango or jute, while those treated with sheesham or hemp had developed Grade II pulmonary fibrosis.

In another experiment involving guinea pigs, animals were exposed by inhalation to average respirable dust concentrations of 1143 mg/m³ for 30 minutes/day, 5 days/week for 24 weeks (McMichael et al. 1983). Histopathological examination showed lung changes, described by the authors as moderate to severe, in all exposed guinea pigs. The changes seen included an increase in septal connective tissue components and aggregation of lymphocytes; however, no pulmonary fibrosis or extensive destruction of the parenchymal tissue occurred. The authors of this study concluded that exposure to fir bark dust may cause inflammatory changes in the lung.

Two studies examined the effect of exposing Syrian golden hamsters to beech wood dust by inhalation, with or without concurrent administration of the known carcinogen diethylnitrosamine (DEN) (Wilhelmsson et al. 1985a, b; Drettner et al. 1985). In each study, the animals were divided into four separate groups. In Study 1, there were 12 animals per group. Two groups were exposed to fresh beech wood dust (a hard wood dust) at a mean total dust concentration of 15 mg/m³ for 6 hours/day, 5 days/week for 36 weeks, and one of these groups was also given 1.5 mg of DEN once a week for the first 12 weeks. The third group in Study 1 was given the DEN doses only (positive control) and the fourth group was given no exposure at all (negative control).

In Study II, there were 24 animals in each of four groups. Two groups of animals were exposed to fresh beech wood dust at a mean total dust concentration of 30 mg/m³ for 6 hours/day, 5 days/week for 40 weeks. The positive and negative control groups were treated as in Study I.

In Study I, none of the hamsters had lung or nasal tumors or metaplasia. Four hamsters exposed to wood dust and DEN exhibited squamous cell papillomas of the trachea, as did three animals in the positive control group and one in the negative control group. No differences in organs other than the respiratory organs were seen between the treated and control groups in Study I.

In Study II, all DEN-exposed hamsters had nasal lesions ranging from hyperplasia and dysplasia to papillomas. In addition, half of all DEN-exposed hamsters developed nasal adenocarcinomas, whether or not they had also been exposed to wood dust. Half of the DEN-exposed animals also had papillomas of the larynx and trachea. In the wood-dust-exposure-only group, two of the animals had nasal lesions, one of which was an unclassifiable malignant nasal tumor and the other of which consisted of fo[illegible] metaplasia with mild dysplasia. The authors concluded that exposure to wood dust did not increase the tumor incidence in DEN-exposed animals but did affect the respiratory tract of all exposed animals.

Human Studies

Dermatitis. There are a large number of case reports, epidemiological studies, and other data on the health effects of wood dust exposure in humans. Dermatitis caused by exposure to wood dusts is common, and can be caused either by chemical irritation, sensitization (allergic reaction), or either of these together. As many as 300 species of trees have been implicated in wood-caused dermatitis.

The chemicals associated with allergic reactions are generally found in the inner parts of a tree, e.g., the heartwood, and the workers most prone to these reactions are those involved in secondary wood processing (carpenters, joiners, finishers).

The symptoms of sensitization are redness, scaling, and itching, which may progress to vesicular dermatitis and, after repeated exposures, to chronic dermatitis. The parts of the body most often affected are the hands, forearms, eyelids, face, neck, and genitals. This form of dermatitis generally appears after a few days or weeks of contact.

Allergic Respiratory Effects. Allergic respiratory responses are mediated by the immune system, as is also the case with allergic dermatitis. Many authors have reported cases of allergic reactions in workers exposed to wood dust. (Sosman et al. 1969; Greenberg 1972; Pickering et al. 1972; Eaton 1973; Booth et al. 1976; Chan-Yeung et al. 1978; Edwards et al. 1978; Innocenti and Angotzi 1980; Bush and Clayton 1983; Cartier et al. 1986). Asthma is the most

common response to wood dust exposure, and the allergic nature of such reactions has been demonstrated by the presence of IgE antibodies and positive skin reactions on patch testing. The best-studied of the allergic reactions to wood dust is Western red cedar (WRC) asthma; it is estimated that 5 percent of the exposed population is allergic to this wood. However, only one study is available that relates exposure level to ventilatory function. In that study, exposure to concentrations of 2 mg/m³ of WRC dust caused significant decreases in forced vital capacity and forced expiratory volume (Vedel et al. 1986). These authors also found that exposures to concentrations above 3 mg/m³ produced eye irritation.

Mucosal and Nonallergic Respiratory Effects. This section discusses changes in the structure and function of the nasal mucosa and respiratory tract that are caused by exposure to wood dust. These changes include nasal dryness, irritation, bleeding, and obstruction; coughing, wheezing, and sneezing; sinusitis; and prolonged colds. These symptoms have been observed even at wood dust concentrations below 4 mg/m³.

Bellion et al. (1964) found that 97 of 225 workers (carpenters, sawmill workers, woodworkers) exposed from 3 to 24 years to the dust of several different hard woods showed radiologic evidence of pulmonary abnormalities. Black et al. (1974) studied nine woodworkers from a woodworking factory in England. In all of these workers, mucociliary movement was markedly depressed, leading these authors to conclude that exposure to wood dust in the furniture industry for 10 years or more can impair mucociliary clearance. These findings were confirmed in a Danish study involving furniture makers (Solgaard and Andersen 1975; Andersen et al. 1976, 1977); compared with controls, the mucociliary transport rate was also significantly impaired in these woodworkers and dose-response effects were noted.

A respiratory survey conducted by Chan Yeung et al. (1980) in pulp and paper mill workers in British Columbia showed that workers exposed to wood dust at a mean total dust concentration of 0.5 mg/m³ had a slight but statistically significant decrease in pulmonary function values compared with controls. The authors concluded that the chemical preservatives used to treat the wood could also have been responsible for these adverse effects.

In a cross-sectional survey of 1,157 American woodworkers (both hard and soft wood), Whitehead et al. (1986) found that exposure to higher (10+ mg/years/m³) compared with lower (0–2 mg-years/m³) dust concentrations was associated with a statistically significant and higher incidence of decreased pulmonary function. However, dose-response effects were observed only for soft wood (i.e., pine) dusts. A later study by Beckman et al. (1981) examined subgroups of the workers studied by Whitehead and found no correlation between years of exposure to pine wood dust and pulmonary function.

In a pilot study of 55 workers in a North Carolina hardwood furniture plant, Goldsmith (1983) found that, at mean area wood dust concentrations of 2 mg/m³ or below, peak ventilatory flow correlated significantly with cumulative person-years of exposure. Goldsmith interpreted this finding to mean that inhalation of wood dust may impair large airway function.

A study of Italian woodworkers showed that the number of wood-dust-exposed workers who had developed anosmia (loss of smell) was significantly higher than in a control group of non-exposed workers (Innocenti et al. 1985). Amoore (1986) confirmed this finding in other workers exposed to hardwood dusts.

Summary of mucosal and nonallergic respiratory effects. A large number of studies have demonstrated that occupational exposure to wood dust causes both statistically significant and non-significant increases in respiratory symptoms. These symptoms range from irritation to bleeding, wheezing, sinusitis, and prolonged colds. In addition, chronic wood dust exposure reduces the rate of mucociliary transport in the nose and, in some workers, also causes changes in the nasal mucosa. Several studies have demonstrated decreased pulmonary function among wood-dust-exposed workers, although other studies have not confirmed these findings.

Carcinogenicity

The association between occupational exposure to wood dust and various forms of cancer has been explored in many studies and in many countries. In 1987, the International Agency for Research on Cancer (IARC) classified furniture manufacturing in Category I (confirmed human carcinogen) and carpentry in category 2B (suspected human carcinogen).

The discussion below focuses on selected U.S. studies.

Nasal and Sinus Cavity Cancer. The earliest U.S. study of wood dust exposure and nasal cancer was conducted by Brinton et al. in 1976. These authors analyzed cancer death rates between 1950 and 1969 in 132 U.S. counties having at least 1 percent of their population employed in furniture and wood fixture manufacturing. This study revealed that the age-adjusted mortality rate for cancer of the nasal cavity and sinuses among white males in the "furniture" counties was significantly higher than in non-furniture counties.

In a later case-control study, these authors (Brinton et al. 1984) analyzed cases of nasal and sinus cancers occurring in North Carolina and Virginia between 1970 and 1980. This study identified a significantly elevated risk of adenocarcinomas in males working in the furniture manufacturing industry, but no increased risk among lumber, carpentry, or construction workers. There was no significant increase in the risk of squamous cell carcinoma in workers from any other wood-related industry.

In a study sponsored by the Inter-Industry Wood Dust Task Force, Viren and colleagues (1982) described a death certificate case-control study of nasal cancer deaths for 1963 to 1977 in North Carolina, Mississippi, Washington, and Oregon. Findings of this study included a relative nasal cancer risk of 1.95 for industries involving lumber and wood products; however, no significant relative risk of nasal cancer was seen for workers in the furniture manufacturing industry.

Imbus and Dyson conducted a study of nasal cancer and North Carolina furniture workers (1985). This study found: (1) That there was a statistically significant increase of nasal cancer among furniture workers; (2) that the nasal cancer rates among North Carolina furniture workers were much lower than those reported for English furniture workers; (3) that the number of nasal cancer deaths among North Carolina furniture workers decreased between 1956 and 1977; and (4) that a slight excess in nasal cancer may have existed among North Carolina furniture workers but is currently either declining or non-existent.

At present, the National Cancer Institute is conducting a cohort mortality study of 36,622 workers employed in the wood, metal, and plastic furniture manufacturing industries (Miller 1987). Results are too preliminary to be described at this time.

Summary of evidence for nasal and sinus cavity cancers. NIOSH (1987) concluded that the literature clearly demonstrates an association between occupational wood dust exposure and nasal cancer. English studies first

identified this link by showing a 10- to 20-times greater incidence of nasal adenocarcinoma among woodworkers in the furniture industry than among other woodworkers and 100 times greater than in the general population. In the United States, three studies have reported a fourfold risk of nasal cancer or adenocarcinoma in furniture workers, and another study noted a similar relationship between nasal cancer and wood dust exposure. One other study failed to find such an association for furniture workers, but did find an increase among logging and timber industry workers. Although hard wood dust has most often been implicated, soft-wood-dust exposure has also been a risk factor in some studies. In addition, there is some evidence that the development of adenocarcinomas is associated with quantitatively higher levels of wood dust exposure and that the risk of this form of cancer increases with latency from first exposure.

Pulmonary Cancer. A number of studies investigating the association between wood dust exposure and the development of lung cancer have been conducted. Milham (1974) found a significant excess of malignant tumors of the bronchus and lung in workers who had belonged to the AFL–CIO United Brotherhood of Carpenters and Joiners of America. Only construction workers showed a statistically significant increase in lung cancer rate.

In a study of lung cancer in Florida residents, Blot et al. (1982) found that an elevated risk of lung cancer that was statistically significant existed among workers in the lumber and wood industry and in construction; however, smoking may have been a confounding factor in these results.

Summary of evidence for pulmonary cancer. The association between lung cancer and occupational wood dust exposure is inconclusive, although several epidemiological studies have reported increases in lung cancer among wood-dust-exposed workers.

Hodgkin's Disease. The data on the relationship between exposure to wood dust and the development of Hodgkin's disease are conflicting. Milham (1967) and Milham and Hesser (1967) concluded, on the basis of a case-cohort study of 1,549 white males dying of this disease between 1940–1953 and 1957–1964, that there was an association between Hodgkin's disease and exposure to wood dust.

Another study (Spiers 1969) concluded that men working in the wood industries in the eastern United States were at special risk for Hodgkin's disease, and suggested that pine pollen exposure might be responsible for the increase.

A Washington State epidemiological study (Petersen and Milham 1974) also found that woodworkers had an increase risk of Hodgkin's disease, and the work of these authors was supported by the results of another study (Gufferman et al. 1976), which showed a non-significant increase in the relative risk for Hodgkin's disease among woodworkers.

Summary of evidence for Hodgkin's disease. Although the data are conflicting, several epidemiological studies of U.S. workers do report increases in the incidence of Hodgkin's disease among woodworkers. This excess is particularly apparent among carpenters.

Other Cancers. NIOSH (1987) concluded that the data on the relationship between occupational exposure to wood dust and the development of cancers other than nasal, Hodgkin's disease, or lung cancers are insufficient and inconclusive.

Basis for the Proposed Limits

OSHA is proposing an 8-hour TwA limit of 1 mg/m^3 for hard wood dust and a 5 mg/m^3 8-hour TWA and 10 mg/m^3 15-minute STEL for soft wood dust. These limits reflect evidence in the literature that exposures to these substances cause allergic respiratory effects and eye irritation (Vedal et al. 1986), mucosal and nonallergic respiratory effects (Bellin et al. 1964; Black et al. 1974; Goldsmith 1983), nasal and sinus cavity cancers (Brinton et al. 1976, 1984; Viren et al. 1982; Imbus and Dyson 1985), and, perhaps, Hodgkin's disease (Milham 1967; Milham and Hesser 1967; Spiers 1969; Petersen and Milham 1974; Gufferman et al. 1976). OSHA preliminarily concludes that these limits will protect exposed workers from the risks of the many conditions and diseases associated with wood dust exposure. However, the Agency notes that many of the studies described above implicate both hard wood and soft wood dusts or do not distinguish between these two substances for cancer, asthma, and dermatitis. OSHA requests information on the health effects, the exposure levels, and the protectiveness of the proposed limits. The health evidence forms a reasonable basis for proposing a new limit for wood dust. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

Preliminary Conclusions for All Respiratory Toxicants

As Table C6–2 and the discussions above show, limits for substances in this group have been established to control employee exposures to or below concentrations of substances that have been associated with acute or chronic respiratory effects. For most of these substances, the evidence is sufficient to identify the NOE or low-effect levels that are related to these effects in humans or animals. Accordingly, OSHA preliminarily concludes that maintaining employee exposures at or below these limits will greatly decrease the likelihood that employees will be at significant risk of respiratory effects when they are exposed to these substances in the workplace. Because chronic pulmonary disease caused by exposure to toxic dusts can be incapacitating, such exposures can effectively end the working life of severely affected individuals. Less serious pulmonary disease can result in lost work days, both as a result of the associated symptoms themselves and as a consequence of increased susceptibility to respiratory infections. The effects of exposure to acute pulmonary toxins, such as ozone or trimellitic anhydride, range from reduced lung function to life-threatening pulmonary edema. Lowering the Agency's current limits or establishing limits where none previously existed will substantially reduce these risks. The health evidence for the substances in this group provides a reasonable basis for revising or adding limits for these substances. At the time of the final rule, OSHA will establish new or revised limits for these respiratory toxins if the Agency determines that these limits will substantially reduce significant risks.

7. Substances for Which Proposed Limits Are Based on Avoidance of Cardiovascular Effects

Introduction

For seven chemicals, OSHA is proposing limits based on their adverse effects on the cardiovascular system. Table C7–1 lists the current Z table limits for these substances, along with the ACGIH TLVs, NIOSH RELs, CAS numbers and HS numbers for these substances. For two of these substances, chloropentafluoroethane and sodium azide, neither OSHA nor NIOSH has current limits. OSHA is proposing to replace its current ceiling limits for two substances (ethylene glycol dinitrate and nitroglycerin) with lower short-term limits. OSHA is proposing to reduce its

current TWA-PEL for carbon disulfide to 1 ppm. For one other substance (fluorotrichloromethane), OSHA is proposing to replace its current TWA-PEL with a ceiling value. For the remaining substance (1,1,2-trichloro-1,2,2-trifluoroethane), OSHA proposes to add a STEL to its existing 8-hour TWA. For three substances in this group, NIOSH recommends limits substantially lower than those established by the ACGIH.

Description of the Health Effects

Although the cardiovascular system can be adversely affected in many different ways by exposure to toxic substances, the adverse effects caused by exposure to the seven chemicals in Table C7-1 are limited to three categories: (1) Cardiac sensitization, (2) vasodilation, and (3) atherosclerosis.

Cardiac sensitization is not related to the type of sensitization that is mediated by the immune system and that causes an allergic reaction. Instead, it results when a chemical "sensitizes" the heart to the effects of a class of biological compounds called sympathomimetic amines. The physiological action of sympathomimetic amines is to stimulate the heart to beat faster. The hormone adrenaline, also called epinephrine, is an example of a sympathomimetic amine. It is normally secreted into the bloodstream when the body anticipates an increase in physical exertion, such as occurs when someone is frightened. A concentration of epinephrine equal to or higher than the noe-ffect level for this substance is necessary to increase the heartbeat rate in exposed individuals. The effect of a cardiac sensitizer is to lower the no-effect level so that the heartbeat rate is stimulated by a lower concentration of adrenaline.

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TABLE C7-1. List of Substances for Which Limits Are Based on Avoidance of Cardiovascular Effects

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1070 Carbon disulfide+	75-15-0	20 ppm TWA 30 ppm STEL 100 ppm Ceiling	10 ppm TWA, Skin	1 ppm TWA 10 ppm Ceiling (15 min)
1087 Chloropenta-fluoroethane	76-15-3	--	1000 ppm TWA	--
1170 Ethylene glycol+ dinitrate	628-96-6	0.2 ppm Ceiling, Skin	0.3 mg/m^3 TWA, Skin	0.1 mg/m^3 Ceiling (20 min)
1180 Fluorotrichloro-methane	75-69-4	1000 ppm TWA	1000 ppm Ceiling	--
1290 Nitroglycerin+	55-63-0	0.2 ppm Ceiling, Skin	0.5 mg/m^3 TWA, Skin	0.1 mg/m^3 Ceiling (20 min)
1364 Sodium azide	26628-22-8	--	0.1 ppm Ceiling	--

TABLE C7-1. List of Substances for Which Limits Are Based on Avoidance of Cardiovascular Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1403 1,1,2-Trichloro-1,2,2-trifluoro-ethane	76-13-1	1000 ppm TWA	1000 ppm TWA 1250 ppm STEL	--

* OSHA's TWA limits are for 8-hour exposures; its STELs are for the durations specified; and its ceilings are peaks not to be exceeded for any period of time.

** The ACGIH TWA-TLV is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

*** NIOSH TWA limits are for 10-hour exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

+ Proposed limit is the NIOSH REL.

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The region of the heart that becomes sensitized is the pacemaking and conduction system, which determines the rhythm and rate of the heartbeat. Unregulated or unnecessary interference with this region of the heart can result in arrhythmia, an abnormality in the rhythm or rate of the heartbeat (Wyngaarden and Smith 1985). The clinical consequences of an arrhythmia vary among individuals. A young person with a healthy heart may not be adversely or seriously affected. However, fatal arrhythmias have occurred in healthy young people, and in older people or individuals whose cardiovascular systems have already been compromised, arrhythmias can cause symptoms of cerebral or myocardial ischemia, shock, or congestive heart failure.

Vasodilators are compounds that cause blood vessels to expand, resulting in a decrease in blood pressure (hypotension) and a decrease in the amount of blood reaching the organs. Acute hypotension is a common cause of shock (Petersdorf et al. 1983). Chronic hypotension may result in a number of symptoms, including lethargy, weakness, easy fatigability, and dizziness or faintness.

Atherosclerosis is a serious disease produced by a degenerative process in the arteries. Plaques containing lipids, complex carbohydrates, blood products, and calcium form on the inside walls of arteries, usually on major blood vessels. These plaques are also called atheromas; their presence makes arteries narrower. Depending on which arteries in the body contain atheromas, different clinical consequences may result, including renal hypertension, stroke, and myocardial ischemia (inadequate circulation of blood to the myocardium) (Balazs et al. 1986). Some chemicals can enhance or accelerate the formation of atheromas and thereby encourage the development of atherosclerosis, a major cause of coronary heart disease.

Dose-Response Relationships and Cardiac Effects

For four of the chemicals in Table C7–1 (carbon disulfide, ethylene glycol dinitrate, nitroglycerin, and sodium azide), the ACGIH-recommended limits are based primarily on health surveys and case reports indicating that occupationally exposed workers subjected to concentrations above a no-effect level experience these cardiovascular effects. Human data for the other three chemicals (chloropentafluoroethane, fluorotrichloromethane, and 1,1,2-trichloro-1,1,2-trifluoroethane), however, are scarce. For these chemicals, limits are based on the results of studies in laboratory animals.

Chemically induced cardiovascular disease occurs in a pattern that appears to correspond to a typical effect level dose-response relationship. That is, an exposure level and exposure duration exist below which the substance appears unlikely to exert an adverse effect. Thus, the limits for substances in this group are designed to maintain exposures below this apparent no-effect level.

The following discussions describe OSHA's preliminary findings for some substances in this group and point to the seriousness of the cardiovascular effects potentially associated with exposure to these substances.

CARBON DISULFIDE
CAS: 75–15–0: Chemical Formula: CS_2
H.S. No. 1070

The current OSHA PELs for carbon disulfide include a TWA of 20 ppm, a STEL of 30 ppm, and a ceiling of 100 ppm. The ACGIH has recommended a TLV–TWA for this substance of 10 ppm without a ceiling or STEL, but with a skin notation, and NIOSH recommends a 1-ppm TWA and a 10-ppm 15-minute ceiling.

The serious adverse effects of exposure to carbon disulfide on the cardiovascular and neurological systems have been documented for decades (ACGIH 1986). Evidence has accumulated indicating that even exposure to low levels of carbon disulfide exerts harmful effects on the cardiovascular and neurological systems of occupationally exposed populations. The current PEL of 20 ppm (i.e., the 1968 ACGIH TLV) was established primarily to prevent neurological disturbances. Since that time, carbon disulfide has been implicated as a risk factor in the development of coronary heart disease at the 20-ppm PEL.

The cardiovascular effects of exposure to carbon disulfide have been well documented in three studies. Tiller et al. (1968), Tolonen et al. (1975), and Tolonen et al. (1979) all identified exposure to carbon disulfide as a contributing factor in coronary heart disease. The latter two studies were epidemiological investigations in Finland in which viscose rayon workers were exposed to low levels of carbon disulfide for at least 5 years; at times, they were exposed to high carbon disulfide concentrations.

Mihail et al. (1968) observed "significant vascular, nervous and biochemical changes" in workers exposed to an average carbon disulfide concentration of 9 ppm for 2 years. According to the ACGIH (1986) and NIOSH (1977b), other studies have reported cardiovascular disorders among the effects of carbon disulfide exposure at levels between 10 and 40 ppm.

Several other studies demonstrate that exposure to carbon disulfide is associated with the development of neurological symptoms, including nervousness, irritability, indigestion, bizarre dreams, insomnia, excessive fatigue, loss of appetite, and headache. More severe symptoms such as psychosis, polyneuritis, and tremors have also been reported (Vigliani 1954; Gordi and Trumper 1938).

Seppalainen and Tolonen (1974) conducted a neurological study on a subgroup of the same workers whose exposure to carbon disulfide at levels between 10 and 30 ppm was considered a contributing factor to coronary heart disease (Tolonen et al. 1975; 1979). They reported an increased incidence of pathologically reduced nerve conduction velocities and abnormal electroencephalograms in exposed workers relative to unexposed workers. The observed decreases in nerve conduction velocity were believed by these researchers to be irreversible.

Several scientists have urged that the exposure limits for carbon disulfide be reduced; Gordi and Trumper (1938) and Kleinfeld and Tabershaw (1955) all advocated a TWA of 10 ppm for carbon disulfide on the basis of its adverse neurological effects. In light of these recommendations and the reports of adverse cardiovascular effects in workers exposed to "relatively low concentrations," the ACGIH (1986) recommended a TLV of 10 ppm TWA. However, NIOSH (1977) considered 10 ppm to be the "lowest concentration causing demonstrated adverse health effects" (p. 137), and believed it necessary to apply a safety factor because "coronary heart disease frequently results in sudden death" (p. 137). Therefore, NIOSH recommended a 10-hour TWA of 1 ppm with a 15-minute short-term limit of 10 ppm with a 15-minute short-term limit of 10 ppm to protect against acute neurological effects associated with exposure to high concentrations of carbon disulfide.

OSHA preliminarily concludes that the epidemiological studies showing neurological and cardiovascular disease among workers exposed to 9 ppm and to between 10 and 40 ppm demonstrate that a risk exists at the current OSHA TWA PEL of 20 ppm for carbon disulfide. OSHA believes that the reduction of the current PEL from 20 ppm to a 1-ppm TWA and a 10-ppm 15-

minute STEL is essential to reduce the risk of adverse effects on the cardiovascular and neurological systems caused by exposure to carbon disulfide at the existing PEL. Because there are reports of cardiovascular and biochemical changes at levels below the ACGIH's limit, the 10-ppm TLV does not provide adequate protection. OSHA is proposing the NIOSH recommendations of a 1-ppm TWA and a 10-ppm STEL as the permissible exposure limits. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for carbon disulfide if the Agency determines that this limit will substantially reduce significant risk.

CHLOROPENTAFLUOROETHANE
CAS: 76–15–3; Chemical Formula: $ClCF_2CF_3$
H.S. NO. 1087

OSHA has no current limit for chloropentafluoroethane (FC–115); the ACGIH recommends a TLV–TWA of 1000 ppm for this colorless, odorless gas.

Chloropentafluoroethane vapors have rather low acute toxicity. In dogs and rats, gastrointestinal absorption following intragastric intubation has been shown to be minimal (Terrill 1974; Clayton, Hood, Nick, and Waritz 1966). The inhalation toxicity for this compound is very low; rats exposed to 800,000 ppm for 4 hours showed no clinical or histopathologic effects (Clayton, Hood, Nick, and Waritz 1966). Rats and guinea pigs showed no adverse clinical effects at inhalation levels of 600,000 ppm for 2 hours (Weigand 1971), and guinea pigs exposed to 200,000 ppm for varying intervals also exhibited no adverse signs (Breen and Wallis 1963). Rats, mice, rabbits and dogs have also demonstrated that FC–115 has a very low subchronic inhalation toxicity. These species have tolerated 6-hour daily exposures of 100,000 ppm for 90 days without adverse effects (Clayton, Hood, Nick, and Waritz 1971), and laboratory animals have tolerated doses of 200,000 ppm for 3.5 hours daily, 5 days per week, for 4 weeks (Weigand 1971). The potential for cardiac sensitization appears to be very low for FC–115; only one of 13 unanesthetized dogs showed cardiac sensitization after exposure to 150,000 ppm intravenously (Trochimoicz, Azar, Terrill, and Mullin 1974). However, several other studies indicate that unanesthetized dogs, rats, and monkeys receiving dosages between 100,000 ppm and 200,000 ppm may show increased blood pressure, accelerated heart rate, myocardial depression, or altered pulmonary effects under certain conditions (Belej and Aviado 1975; Friedman, Cammarato, and Aviado 1973; Aviado and Belej 1975). There are no reports of mutagenic, teratogenic, or carcinogenic toxicities in these studies.

OSHA is proposing an 8-hour TWA permissible exposure limit of 1000 ppm for chloropentafluoroethane. The Agency preliminarily concludes that this limit will protect workers from the risk of cardiac effects at the extremely high levels permitted by the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for chloropentafluoroethane. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

ETHYLENE GLYCOL DINITRATE
CAS: 628–96–6; Chemical Formula: $CH_2H_4(NO_3)_2$
H.S. No. 1170

NITROGLYCERIN
CAS: 55–63–0; Chemical Formula: $CH_2NO_3CHNO_3NO_3$
H.S. No. 1290

The current OSHA PELs for ethylene glycol dinitrate (EGDN) and nitroglycerin (NG) are ceilings of 0.2 ppm (1 mg/m³ and 2 mg/m³, respectively, with skin notations). The ACGIH (1986) has established a TWA of 0.05 ppm (0.3 mg/m³) for EGDN and a TWA of 0.05 ppm (0.05 mg/m³) for NG, both with skin notations, to protect against the hypotension and headaches caused in occupationally exposed populations by the vasodilatory effects of these substances. NIOSH recommends a 20-minute ceiling of 0.1 mg/m³ for both of these substances.

Most occupational exposures to EGDN actually involve mixtures of EGDN and nitroglycerin (NG). Because EGDN is 160 times more volatile than nitroglycerin, and most of the mixtures of these two substances used in industry consist of 60 to 80 percent EGDN, the adverse effects associated with inhalation of such mixtures can be attributed primarily to EGDN.

Trainor and Jones (1966) reported that exposure to EGDN:NG at a level of 0.7 mg/m³ for 25 minutes was sufficient to produce decreased blood pressure and a slight headache in humans. The authors also reported that workers at a munitions plant developed headaches when exposed to EGDN:NG concentrations between 0.1 and 0.53 mg/m³ (0.36 mg/m³ average). Morikawa et al. (1967) found that workers in an explosives plant exposed to low concentrations of EGDN:NG (0.066 ppm was the highest average level) had a much higher incidence of abnormal pulse waves than controls (143 out of 1,271 versus 0 out of 175). (Abnormal pulse waves often indicate a clinically significant defect in the functioning of the heart and/or circulatory system (Braunwald 1978).)

In its Criteria Document for NG and EGDN, NIOSH (1978b) refers to a report of a dynamite worker who died when exposed to EGDN:NG concentrations between 0.3 and 1.4 mg/m³, and to another report of two workers who died suddenly following exposure to EGDN:NG at concentrations ranging from 1.7 to 2.7 mg/m³. NIOSH (1978) observed that skin absorption may have contributed significantly to these deaths.

Based on the evidence described above, the ACGIH recommended a 0.05-ppm TLV–TWA and a 0.1-ppm TLV–STEL in 1981. Because the studies relied on were primarily reports of adverse effects resulting from chronic exposure, ACGIH adopted the TLV–TWA without the STEL in 1983. After reviewing the same evidence, NIOSH (1978) concluded that exposure to EGDN:NG should be controlled to a level below that associated with vasodilation, which is the most sensitive indicator of toxicity. Relying primarily on the Trainor and Jones (1966) study, NIOSH concluded that workers exposed to or below 0.1 mg/m³ will not develop vasodilation, as indicated by the development of headache. Because vasodilation and associated headache were found to occur even on short-term exposure to EGDN:NG, NIOSH recommended a 20-minute short-term limit of 0.1 mg/m³ approximately 0.01 ppm).

Hypotension and headache have been observed in populations exposed below 0.5 mg/m³, and fatalities have occurred at EGDN:NG exposures of between 0.3 and 1.4 mg/m³ in one instance, and between 1.7 and 2.7 mg/m³ in another. OSHA's existing standard is 0.3 mg/m³, as is the ACGIH TLV. Since worker deaths have occurred at or near the current PEL, OSHA proposes adoption of the more stringent 0.1-mg/m³ (0.01-ppm) NIOSH short-term limit as the PEL to prevent fatalities and protect against adverse health effects. It should be noted that toxicity can be produced readily through the skin, and therefore the recommended environmental limit is protective only if skin contact is prevented. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for ethylene glycol dinitrate and nitroglycerin if the Agency determines that this limit will substantially reduce significant risk.

FLUOROTRICHLOROMETHANE
CAS: 75–69–4; Chemical Formula: CCl_3F
H.S. No. 1180

Fluorotrichloromethane, also known as FC–11, is a member of a large family of chemicals, the chlorofluorocarbons. The current OSHA PEL is an 8-hour TWA of 1000 ppm. NIOSH has no REL for fluorotrichloromethane. OSHA proposes that the standard be revised to a ceiling limit of 1000 ppm, which corresponds to the current ACGIH TLV for this substance.

Inhalation of large doses of FC–11 has caused cardiac sensitization and death in humans. Experimental mice that inhaled aerosol containing 10 percent FC–11 exhibited cardiac arrhythmias. In the same study, dogs that inhaled aerosol containing 2.5 percent FC–11 had decreased myocardial function; monkeys that inhaled an aerosol containing 5 percent FC–11 developed tachycardia and hypotension (NRC 1977).

Exposure to 5000 ppm FC–11 has induced cardiac sensitization and arrhythmia in dogs that were intravenously injected with epinephrine (Reinhardt, Azar, Maxfield et al. 1971). Jenkins, Jones, Coon, and Siegel (1970) found that four species of animals (monkeys, dogs, rats, and guinea pigs) suffered no ill effects after 90 days of continuous exposure to 1000 ppm FC–11.

Because 1000 ppm appears to represent a no-effect level for FC–11, OSHA recommends a ceiling of 1000 ppm, which will provide workers with a greater margin of safety against cardiac sensitization than the Agency's current 1000-ppm 8-hour TWA. The cardiac sensitization exhibited by FC–11-exposed animals is an acute effect. OSHA's current 1000-ppm TWA PEL would permit workers to be exposed to short-term concentrations of FC–11 that are sufficiently high to sensitize the heart to sympathomimetic amines. Accordingly, OSHA finds that, at the current limit, workers are at risk of experiencing arrhythmia. Reducing this to a 1000-ppm ceiling limit (the observed no-effect level in animals) should ensure that no worker is placed at risk of cardiac sensitization as a result of exposure to FC–11. Therefore OSHA is proposing to revise its 1000-ppm TWA–PEL for this substance to a 1000-ppm ceiling value. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for fluorotrichloromethane if the Agency determines that this limit will substantially reduce significant risk.

SODIUM AZIDE
CAS: 26628–22–8; Chemical Formula: NaN_3
H.S. No. 1364

There is no current OSHA PEL for sodium azide and NIOSH also has no REL for this substance. The ACGIH (1986) recommends ceiling limits of 0.1 ppm for sodium azide (as hydrazoic acid vapor), or 0.3 mg/m³ as NaN_3).

Sodium azide is known to produce hypotension in laboratory animals and humans. An intravenous dose of 1 mg/kg has been reported to lower blood pressure in cats (Graham 1949). In the 1950s, the medicinal usefulness of sodium azide as a hypotensive agent was tested in 30 hypertensive patients. Their hypertension was reduced, but observed side effects included headaches; in addition, 20 of 30 patients developed increased sensitivity to sodium azide, necessitating a reduction in the dose (Black et al. 1954).

Acute inhalation by humans of hydrazoic acid vapor (which forms when sodium azide contacts water) results in lowered blood pressure, eye irritation, bronchitis, headache, weakness, and collapse (Fairhall et al. 1943; Graham 1949). The exposure levels that produce these effects were not reported by these authors. Haas and Marsh (1970) report that exposure to concentrations of hydrazoic acid vapor as low as 0.5 ppm "cause some discomfort to laboratory personnel."

Because of its hypotensive effect in humans, OSHA believes that a limit should be established for sodium azide that will reduce the risk posed to workers at the previously uncontrolled levels of this substance permitted in the workplace. To reduce this risk substantially, OSHA is proposing to establish a ceiling limit of 0.1 ppm HN_3 (0.3 mg/m³ NAN_3) for sodium azide. The health evidence forms a reasonable basis for proposing a new limit for sodium azide. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

1,1,2-TRICHLORO-1,2,2-TRIFLUOROETHANE
CAS: 76–13–1; Chemical Formula: CCl_2FCClF_2
H.S. No. 1403

1,1,2-Trichloro-1,2,2-trifluoroethane (FC–113) is a member of the chlorofluorocarbon family. The current OSHA PEL is an 8-hour TWA of 1000 ppm. The ACGIH has an 8-hour TLV–TWA of 1000 ppm and a 15-minute STEL of 1250 ppm. NIOSH has no REL for 1,1,2-trichloro-1,2,2-trifluoroethane.

Cardiac sensitization following the administration of epinephrine is the most significant effect observed after exposure to FC–113. Reinhardt, Mullin, and Maxfield (1973) observed that 10 out of 29 dogs exposed to 5000 ppm FC–113 for 5 minutes and simultaneously injected with epinephrine developed serious arrhythmias. Similar experiments in which the dogs were exposed to 2000 to 2500 ppm of this substance for longer periods of time (from one-half to 6 hours) and administered epinephrine resulted occasionally in arrhythmia (Aviado 1975). However, when the experiment was repeated using four 6-hour exposures to 1000 ppm in conjunction with an injection of epinephrine, no arrhythmias were observed.

A study by Stopps and McLaughlin (1967) of human volunteers revealed that exposure to 2500 ppm FC–113 resulted in impairment of psychomotor performance (described as loss of ability to concentrate and lethargy). This effect was not observed at concentrations below 2500 ppm.

The evidence described above demonstrates that FC–113 can exert toxic effects at levels of exposure comparable to the levels that are permitted by excursions above the current OSHA TWA of 1000 ppm; thus, current levels pose a risk of cardiac sensitization to exposed workers. OSHA finds that a STEL of 1250 ppm will provide a wider margin of safety against cardiac sensitization and impaired psychomotor performance by limiting the potentially high, short-term exposures currently permitted by the 8-hour TWA. OSHA believes that establishing a STEL of 1250 ppm will reduce this risk of FC–113-induced cardiovascular toxicity. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for 1,1,2-trichloro-1,2,2-trifluoroethane if the Agency determines that this limit will substantially reduce significant risk.

Preliminary Conclusions

Of all the physiological systems, the cardiovascular system is especially vulnerable to occupational hazards because cardiovascular diseases are already so prevalent in our society. According to Levy (1985), "an estimated 40 million Americans have some form of cardiovascular disease." The major risk factors, as revealed by epidemiology, are age, male sex, hypertension, cigarette smoking, the existence of low-density and high-density plasma lipoproteins, cholesterol, and diabetes (Levy 1985). Many American workers exposed to the chemicals grouped here on the basis of their cardiovascular

effects have one or more of these risk factors and are therefore particularly susceptible to exposure to cardiovascular toxicants. Although the precise interactions among these risk factors and exposures to cardiovascular toxins are difficult to demonstrate with accuracy, few would argue that they do not occur.

OSHA preliminarily concludes that the potential for cardiovascular system damage associated with exposure to these cardiac sensitizers, vasodilators, and atherosclerosis-causing substances poses a risk to employees in a broad range of workplaces. The effects experienced by exposed workers range from arrhythmia, low blood pressure, stroke, and blockage of the flow of blood to the myocardium. Reducing the exposure limits for these cardiovascular toxins from levels where such effects could occur to concentrations where their occurrence is unlikely will substantially reduce these risks. OSHA believes that the health evidence for these cardiovascular toxins forms a reasonable basis for proposing new or revised limits. At the time of the final rule, OSHA will establish new or revised limits for these substances if the Agency determines that such limits will substantially reduce significant risk.

8. Substances for Which Proposed Limits Are Based on Avoidance of Systemic Toxicity

Introduction

For a number of substances, OSHA's proposed limits are based primarily on evidence that exposure to these substances is associated with general systemic toxicity. This group of substances is unique among the groupings discussed so far in that no single low-dose target organ system can be identified for these chemicals. Instead, these substances have been shown either to affect several organ systems simultaneously or to cause a variety of nonspecific adverse signs and symptoms that are indicative of general toxicity.

The 33 substances belonging to this group and their CAS numbers, HS numbers, current PELs, ACGIH TLVs, and RELs are shown in Table C8–1. OSHA is proposing to establish exposure limits for 17 substances in this group for which there are currently no Z table limits. OSHA is also proposing to retain the Agency's PELs for eight substances and to add STELs to them. For five substances, OSHA is proposing to lower its existing TWA PELs. For two additional substances that have existing TWA PELs, OSHA is proposing to delete the TWA PELs and to adopt ceiling values instead. For one remaining substance, OSHA is proposing to delete its existing ceiling limit and to adopt a new TWA PEL. NIOSH recommends limits for 11 of the substances that differ in some respects from the ACGIH TLVs.

Description of the Health Effects

For each substance included in this grouping, limits have been established to protect against a variety of adverse exposure-related effects that are manifested at multiple target organ sites. In some instances, the nature of the toxic effects associated with exposure is well-defined and clearly understood (for example, CNS depression, histological organ changes, embryotoxicity, methemoglobinemia, conjunctivitis). The effects of exposure to other substances in this group, however, have been demonstrated only by such nonspecific indicators as weight loss or decreased weight gain, lethargy, loss of appetite, nervousness, or gastrointestinal disturbances. Although the specificity of the systemic effect caused by exposure to the substances in this group may vary, all of these substances have been shown to be biologically active in mammalian species, to interfere significantly with biological processes, and to impair normal organ function.

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TABLE C8-1. Substances for Which Limits Are Based on Avoidance of Systemic Toxicity

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1005 Acetonitrile[+]	75-05-8	40 ppm TWA	40 ppm TWA 60 ppm STEL, Skin	20 ppm TWA
1006 Acetylsalicylic acid (Aspirin)	50-78-2	--	5 mg/m^3 TWA	--
1019 Aluminum (Welding fumes)	7429-90-5	--	5 mg/m^3 TWA	--
1046 2-Butoxy ethanol	111-76-2	50 ppm TWA, Skin	25 ppm TWA, Skin	--
1052 n-Butyl glycidyl ether	2426-08-6	50 ppm TWA	25 ppm TWA	5.6 ppm Ceiling (15 min)
1067 Captan	133-06-2	--	5 mg/m^3 TWA	--
1088 Chloroprene	126-99-8	25 ppm TWA, Skin	10 ppm TWA, Skin	1 ppm Ceiling (15 min)
1109 Cyclohexylamine	108-91-8	-	10 ppm TWA	-
1112 Cyhexatin	13121-70-5	-	5 mg/m^3 TWA	-
1120 2-N-Dibutylamino-ethanol	102-81-8	-	2 ppm TWA, Skin	-

TABLE C8-1. Substances for Which Limits Are Based on Avoidance of Systemic Toxicity (continued)

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1139 Diglycidyl ether	2238-07-5	0.5 ppm Ceiling	0.1 ppm TWA	0.2 ppm Ceiling (15 min)
1159 Ethanolamine	141-43-5	3 ppm TWA	3 ppm TWA 6 ppm STEL	--
1167 Ethylene chlorohydrin	107-07-3	5 ppm TWA, Skin	1 ppm Ceiling, Skin	--
1189 Glycidol	556-52-5	50 ppm TWA	25 ppm TWA	--
1198 Hexafluoroacetone	684-16-2	--	0.1 ppm TWA, Skin	--
1207 Hydrogen cyanide+	74-90-8	10 ppm TWA, Skin	10 ppm Ceiling, Skin	4.7 ppm Ceiling (10 min)
1210 Hydrogenated terphenyls	61788-32-7	--	0.5 ppm TWA	--
1223 2-Isopropoxyethanol	109-59-1	--	25 ppm TWA	--
1227 Isopropyl glycidyl ether	4016-14-2	50 ppm TWA	50 ppm TWA 75 ppm STEL	50 ppm Ceiling (15 min)
1273 4,4'-Methylene bis (2-chloroaniline)	101-14-4	--	0.02 ppm TWA ($0.22\ mg/m^3$), Skin	3 ug/m^3 TWA (Lowest detectable limit)

TABLE C8-1. Substances for Which Limits Are Based on Avoidance of Systemic Toxicity (continued)

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1317 Phenylhydrazine	100-63-0	5 ppm TWA, Skin	5 ppm TWA 10 ppm STEL, Skin	0.14 ppm Ceiling (120 min)
1318 Phenylphosphine	638-21-1	-	0.05 ppm Ceiling	-
1321 Phosphine	7803-51-2	0.3 ppm TWA	0.3 ppm TWA 1 ppm STEL	--
1330 Piperazine dihydrochloride	142-64-3	--	5 mg/m^3 TWA	--
1340 n-Propyl nitrate	627-13-4	25 ppm TWA	25 ppm TWA 40 ppm STEL	--
1366 Sodium fluoroacetate	62-74-8	0.05 mg/m^3 TWA, Skin	0.05 mg/m^3 TWA 0.15 mg/m^3 STEL, Skin	--
1412 Trimethylbenzene	25551-13-7	-	25 ppm TWA	--
1416 Tungsten Compounds (insoluble)	7440-33-7	--	5 mg/m^3 TWA 10 mg/m^3 STEL	5 mg/m^3 TWA
1417 Tungsten Compounds (soluble)	7440-33-7	--	1 mg/m^3 TWA 3 mg/m^3 STEL	1 mg/m^3 TWA

TABLE C8-1. Substances for Which Limits Are Based on Avoidance of Systemic Toxicity (continued)

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1428 Vinylidene chloride	75-35-4	--	5 ppm TWA 20 ppm STEL	Lowest feasible level
1430 Welding fumes (Total particulate)	--	-	5 mg/m^3 TWA	--
1437 Zinc oxide (Fume)	1314-13-2	5 mg/m^3 TWA	5 mg/m^3 TWA 10 mg/m^3 STEL	5 mg/m^3 TWA 15 mg/m^3 Ceiling (15 min)
1439 Zirconium compounds	7440-67-7	5 mg/m^3 TWA	5 mg/m^3 TWA 10 mg/m^3 STEL	--

* OSHA's TWA limits are for 8-hour exposures; its STELs are for the durations specified; and its ceiling are peaks not to be exceeded for any period of time.

** The ACGIH TWA-TLV is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

*** NIOSH TWA limits are for 10-hour exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

+ Proposed limit is the NIOSH REL.

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Table C8–2 summarizes the toxic effects reported in humans and experimental animals to support the establishment of limits for these substances. This table shows the variety of adverse health effects that adoption of the proposed limits will minimize or prevent. The table also shows that, for the vast majority of substances in this group, the risks of exposure have been defined in studies of humans or animals and are known to include respiratory effects, neurological effects, adverse effects on the reproductive system, organ damage, hematopoietic effects, sensitization, and mucosal irritation. All of these effects are indicative of generalized systemic effects rather than localized effects occurring at the site of chemical contact.

Dose-Response Relationships and Systemic Effects

As Table C8–2 shows, adverse toxic reactions have been reported to occur in humans for 18 of the 33 substances in this group; thus, for more than half of these substances, it has been established conclusively that exposure is associated with adverse health effects in humans. Experimental animal data comprise the principal evidence for the toxicologic action of the remaining substances. As is the case for many substances for which limits are being proposed, apparent no-observed effect levels, supplemented by the use of an additional safety factor to take more sensitive individuals into account, provide the basis for setting limits. The systemic toxic effects caused by exposure to substances in this group appear to follow an NOE dose-response pattern. That is, as intensity and/or duration of exposure decreases, the severity of the effect on organ systems also decreases until a point is reached where there is no detectable effect, at least at observable levels, on organ systems at or below the NOE level. No-effect exposure levels have been identified in humans and animals for several of the substances in this group; where no-effect levels have been identified (i.e., for diglycidyl ether and phenylphosphine), they have provided the primary basis for the proposed limits.

TABLE C8–2.—SUMMARY OF ADVERSE HEALTH EFFECTS REPORTED FOR SUBSTANCES PRODUCING GENERAL SYSTEMIC TOXICITY

H.S. No./Chemical name	Effects reported in humans	Effects reported in animals
1005 Acetonitrile	Tightness in chest	Embryotoxicity.
	Flushing of face	Teratogenicity at maternally toxic doses.
		Liver, blood count changes.
1006 Acetylsalicylic acid	Mucosal irritation	Teratogenicity at "very large" doses.
	Respiratory allergic response	
	Internal bleeding	
1019 Aluminum welding fumes	No data	Respiratory effects.
1046 2-Butoxy ethanol	Mild sensory irritation	Severe hemoglobinuria; lung, kidney, liver changes.
		Hemolytic anemia, increased osmotic fragility in erythrocytes
1052 n-Butyl glycidyl ether	Dermatitis, skin sensitization	Delirium, depression.
1067 Captan	Recurrent urticaria	Decreased fertility index in males.
		Polyploid carcinoma of duodenum.
1088 Chloroprene	CNS depression	"Mineral systemic effect."
	Lung, liver, kidney injury	
	Conjunctivitis, necrosis of cornea	
	Lowering of blood pressure	
1109 Cyclohexylamine	Acute toxicity	Mutagenic and reproductive effects.
	Sensitization	
1112 Cyhexatin	No data	Microscopic changes in liver, kidney, adrenal glands.
1120 2-N-Dibutylaminoethanol	No data	Weight loss.
		Elevated liver- and kidney-to-body-weight ratios.
1139 Diglycidyl ether	Mucosal irritation	CNS depression.
		Clouding of cornea.
		Respiratory irritation.
		Hematopoietic effects.
1159 Ethanolamine	No data	Pulmonary, hepatic, and renal lesions.
		Decreased alertness.
		Temporary weight loss.
1167 Ethylene chlorohydrin	Damage to liver and brain at lethal concentrations.	Respiratory depression.
		Liver and kidney damage.
	Mucosal irritation	
	Gastrointestinal disturbances	
1189 Glycidol	No data	Pneumonitis, emphysema.
1198 Hexafluoroacetone	No data	Renal dysfunction.
		Increased lung weight.
		Testicular damage. Hematopoietic effects.
		Fetotoxicity.
1210 Hydrogenated terphenyls	No data	Decreased weight gain.
		Liver, kidney damage.
		Lung changes, bronchopneumonia.
1207 Hydrogen cyanide	Cyanide poisoning	None reported.
	Weakness	
	Mucosal irritation	
	Colic	
	Nervousness	
	Enlargement of thyroid	
1223 2-Isopropoxyethanol	No data	Anemia.
		Hemoglobinuria.
		Lung congestion.
1227 Isopropyl glycidyl ether	Mucosal irritation	Reduced weight gain.
		Hemoglobin increase.
		Emphysematous changes in lungs.
		CNS depression.

TABLE C8-2.—SUMMARY OF ADVERSE HEALTH EFFECTS REPORTED FOR SUBSTANCES PRODUCING GENERAL SYSTEMIC TOXICITY—Continued

H.S. No./Chemical name	Effects reported in humans	Effects reported in animals
1273 4,4'-methylene-bis (2-chloroaniline)	Hematuria	Cyanosis. Methemoglobinemia. Liver, lung tumors.
1317 Phenylhydrazine	Skin sensitization	Anemia. Irregular growth. General weakness. Blood vessel tumors.
1318 Phenylphosphine	No data	Mild hemolytic anemia. Testicular degeneration. Hind leg tremor. Nausea, loss of appetite. Hypersensitivity to sound and touch.
1321 Phosphine	Pulmonary edema Gastrointestinal disturbances Dizziness	Respiratory irritation.
1330 Piperazine dihydrochloride	Skin burns, sensitization, asthma	No data.
1340 n-Propyl nitrate	No data	Cyanosis. Methemoglobinemia. Hypotension. Respiratory depression.
1366 Sodium fluoroacetate	No data	Fluctuation in growth rate. Tissue changes.
1412 Trimethylbenzene	Nervousness, tension, anxiety Asthmatic bronchitis Hypochromic anemia	Lymphopenia, neutrophilia.
1416 Tungsten compounds (insoluble)	No data	Gross changes in liver and spleen. Lung tissue changes.
1417 Tungsten compounds (soluble)	No data	Generalized cellular asphyxiation. Colic. Incoordination. Dyspnea.
1428 Vinylidene chloride	No data	Nasal irritation. Liver cell degeneration. Retarded weight gain. Embryotoxicity Kidney adenocarcinoma.
1430 Welding fumes (total particulate)	Pulmonary irritation	Pulmonary irritation.
1437 Zinc oxide (fume)	Metal fume fever, gastritis	No data.
1439 Zirconium compounds	No data	Toxic effects from zirconium tetrachloride due to liberation of hydrochloric acid.

In instances where no-effect levels have not been reported (e.g., for n-butyl glycidyl ether, trimethylbenzene, and acetylsalicylic acid), OSHA has used safety factors and expert judgment to derive an NOE value.

The following discussions describe OSHA preliminary findings for these systemic toxicants.

ACETONITRILE
CAS: 75-05-8; Chemical Formula: CH_3CN
H.S. No. 1005

Acetonitrile is most widely used in industry as a specialty solvent and chemical intermediate. The current occupational exposure limit for acetonitrile is a 40-ppm TWA. The ACGIH has recommended a 40-ppm TLV-TWA with a 60-ppm TLV-STEL, in addition to a skin notation. NIOSH (1978) has also evaluated the toxicity of acetonitrile and has recommended a TWA limit of 20 ppm.

The only human evidence describing the toxic effects associated with exposure to acetonitrile is a report by Pozzani et al. (1959), who exposed human subjects to acetonitrile vapor. None of three subjects exposed to 40 ppm for 4 hours reported any adverse responses during the exposure period, but one subject experienced slight tightness of the chest a few hours after termination of exposure as well as a cooling sensation in the lungs the following day. None of the subjects had elevated blood cyanide levels; one subject showed a slightly elevated urinary thiocyanate level. Pozzani et al. (1959) also exposed two subjects to 80 ppm and 160 ppm of acetonitrile for 4 hours. Subjects exposed to 80 ppm reported no adverse response. One subject exposed to 160 ppm experienced slight flushing of the face and chest tightness a few hours after exposure (Pozzani et al. 1959).

In animal studies, acetonitrile has been found to be embryotoxic and teratogenic in rodents only at exposure levels sufficiently high to cause maternal toxicity (Berteau et al. 1982; Willhite 1983). A 13-week inhalation study conducted by the National Toxicology Program (Hazelton Laboratories 1983) found pathological changes in the liver and some blood changes in mice and rats exposed to concentrations of 400 ppm acetonitrile.

The human study by Pozzani et al. (1959) provides the primary basis for both the ACGIH's 40-ppm TWA and 60-ppm STEL and NIOSH's REL of 20 ppm (TWA). In addition to this study, NIOSH (1978) cites a report by Amdur (1959), who investigated an incident in which 16 painters became ill (with one death) after using an acetonitrile-containing material in a confined space. Amdur (1959) reported no further incidents after adequate ventilation was installed and acetonitrile levels were maintained at about 17 ppm. NIOSH concluded that exposure to 40 ppm "produced minimal effects, whereas no observable effects were produced in humans at 17 ppm" (NIOSH 1978, p. 97). Therefore, NIOSH recommended that exposure not exceed 20 ppm as a 10-hour TWA.

OSHA proposes a 20-ppm PEL for acetonitrile. Minor health effects have been reported at the 40-ppm TLV-TWA (with a 60-ppm STEL) level; the identification of 17 ppm as a no-effect

level is a major consideration in selection of the 20-ppm REL value that the Agency proposes to adopt. This health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for acetonitrile if the Agency determines that this limit will substantially reduce significant risk. OSHA's preliminary feasibility analysis is based on limited data at this level, and additional feasibility information is requested from the public.

ACETYLSALICYLIC ACID (ASPIRIN)
CAS: 50-78-2; Chemical Formula: $CH_3COOC_6H_4COOH$
H.S. No. 1006

There is no current OSHA limit or NIOSH REL for acetylsalicylic acid. The ACGIH has established a TLV of 5 mg/m³ as an 8-hour TWA.

The work of O'Brien (1968) reports that a normal therapeutic dose of 600 mg aspirin will interfere with platelet aggregation in exposed subjects for a period of five days or more. Hart (1947) also reported that 150 mg is the smallest oral dose of acetylsalicylic acid that will produce this effect. Unpublished data from the Dow Chemical Company (cited in ACGHIH 1986, p. 10) indicate that aspirin concentrations exceeding 100 mg/m³ are tolerated except for occasional skin irritation. However, no data are available on the long-term effects on organ systems of inhalation exposure to aspirin. Secondary sources report that aspirin is an acute irritant to the gastric mucosa and respiratory tract.

OSHA is proposing an 8-hour TWA of 5 mg/m³ for acetylsalicylic acid. The Agency preliminarily concludes that this limit will prevent blood effects and gastric and respiratory irritation, and proposes to protect workers from these adverse effects by reducing this uncontrolled risk. This health evidence forms a reasonable basis for proposing a new limit for acetylsalicylic acid. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

ALUMINUM (WELDING FUMES)
CAS: 7429-90-5; Chemical Formula: Al
H.S. No. 1019

OSHA currently has no permissible exposure limits for aluminum welding fume. The ACGIH recommends an 8-hour TLV-TWA of 5 mg/m³.

Aluminum welding fumes are produced by arc-welding in a protective, inert atmosphere such as argon. Some aluminum fume is created by these arcs, as is an intense radiation that can produce ozone. Because workers exposed to arc welding fumes have previously not been protected by a permissible exposure limit, OSHA proposes a PEL of 5 mg/m³ TWA for these welding fumes. The Agency preliminarily concludes that this limit will protect welders and other workers in the vicinity of the welding from exposure to the significant irritation potentially associated with inhalation of these fumes. This health evidence forms a reasonable basis for proposing a new limit for aluminum (welding fumes). At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

2-BUTOXYETHANOL
CAS: 111-76-2; Chemical Formula: $C_4H_9OCH_2CH_2OH$
H.S. No. 1046

OSHA's current permissible exposure limit for 2-butoxyethanol, one of the family of substances known as the glycol ethers, is 50 ppm as an 8-hour TWA, with a skin notation. The ACGIH recommends a limit of 25 ppm TWA, also with a skin notation, for this colorless liquid with a mild ether odor.

2-Butoxyethanol has long been known to be toxic, with early studies indicating that a single 7-hour exposure to 700 ppm was lethal to laboratory animals (Werner, Mitchel, Miller et al. 1943). Exposures near the lethal level caused systemic toxicity in the form of hemoglobinuria and lung, kidney, and liver changes. Carpenter and associates (1956) reported hemolytic anemia and increased fragility of the red blood cells in rats repeatedly exposed to 2-butoxyethanol at 320 ppm for 5 weeks. However, repeated exposure for 12 weeks at 400 ppm was only slightly injurious to dogs (Werner, Mitchell, Miller et al. 1943).

Humans appear to be less susceptible to butoxyethanol poisoning than experimental animals. In humans, several single 8-hour exposures at levels of 200 ppm and 100 ppm caused urinary excretion of butoxyacetic acid. However, these subjects experienced irritation and discomfort after these exposures (Carpenter, Pozzani, Weil et al. 1956). However, a recent study has confirmed that the increased erythrocyte osmotic fragility observed in rats exposed to many of the glycol ethers is a very sensitive indicator of toxicity and correlates with the development of hemoglobinuria at higher exposure levels (Moffett, Linnett, and Blair 1976). These findings indicate that the no-effect level in animals is approximately 25 ppm. The ACGIH suggests that 2-butoxyethanol's toxicity may be more likely to occur as a result of skin absorption than as a consequence of inhalations (ACGIH 1986, p. 71).

OSHA preliminarily concludes that the current PEL of 50 ppm is insufficiently protective against the risk of 2-butoxyethanol's hematological effects. The proposed limit of 25 ppm will reduce this risk to a level below that at which these toxic effects have been observed. This health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for 2-butoxyethanol if the Agency determines that this limit will substantially reduce significant risk.

n-BUTYL GLYCIDYL ETHER
CAS: 2426-08-6; Chemical Formula: $C_4H_9OH_2CHOCH_2$
H.S. No. 1052

The current OSHA limit for n-butyl glycidyl ether is 50 ppm TWA. The ACGIH-recommended TLV is 25 ppm; NIOSH has recommended that occupational exposure to n-butyl glycidyl ether not exceed 5.6 ppm as a 15-minute short-term level.

OSHA's current PEL of 50 ppm, which was adopted from the ACGIH's 1968 TLV list, was based on a Dow Chemical Company report (cited in ACGIH 1986, p. 81) that showed that repeated applications of n-butyl glycidyl ether to the skin of humans caused irritation and sensitization; at the time, the ACGIH concluded that a limit of 50 ppm would prevent these irritation responses. The ACGIH proposed to reduce the TLV to 25 ppm in 1978, noting that the 50-ppm limit was only 13 times lower than the 8-hour LC_{50} (670 ppm) reported for this chemical in rats and that a wider margin of safety was desirable.

The NIOSH limit of 5.6 ppm was recommended in the Institute's June 1978 Criteria Document on Glycidyl Ethers. This limit was based, in large part, on mutagenic studies conducted in microbial and mammalian test systems, as well as some evidence for other glycidyl ethers that exposure is associated with testicular atrophy and hematopoietic abnormalities in laboratory animals. After publication of its Criteria Document, NIOSH received a confidential report prepared for the Shell Development Company by Anderson et al. (1957), who had conducted a rat inhalation study. In the research, rats were exposed to 38 ppm, 75 ppm, 150 ppm, or 300 ppm for seven hours daily, 5 days per week for 10 weeks. Atrophic testes were found in 5 of 10 rats exposed to 300 ppm, very small testes were found in 1 of 10 rats exposed to 150 ppm, and patchy atrophy was found in 1 of 10 rats exposed to 75 ppm. No effects were observed in rats exposed at 38 ppm. Based on this

additional evidence, NIOSH reaffirmed its REL for n-butyl glycidyl ether in a Current Intelligence Bulletin published in October, 1978.

The NIOSH REL of 5.6 ppm (15-minute STEL) is based on in-vitro testing of microbial and mammalian systems, and extensive extrapolation of these data is required to predict effects in humans. The 25-ppm TLV is based on an increased factor of safety applied to the LC_{50} rate determined in animal studies and is below the observed no-effect level of 38 ppm. OSHA therefore proposes that a PEL of 25 ppm TWA be adopted to protect exposed workers from the serious adverse effects of butyl glycidyl ether exposure. This health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for n-butyl glycidiy ether if the Agency determines that this limit will substantially reduce significant risk.

CAPTAN
CAS: 133-06-2; Chemical Formula: $C_9H_8Cl_3NO_2S$
H.S. No. 1067

OSHA does not currently regulate captan. The ACGIH recommends a TLV-TWA of 5 mg/m³ for this substance, which is a white, crystalline, odorless solid.

Skin applications of 900 mg/kg captan produce skin irritation in experimental animals. Long-term feeding studies did not reveal adverse effects in dogs fed captan in the diet at levels of 100 mg/kg/day for 66 weeks or in rats fed 1000 mg/kg/day for 2 years (Martin 1971; Spencer 1968). Male mice showed decreased fertility at levels of 50 or 100 mg/kg/day for 5 days (Collins 1972).

Studies on the mutagenicity of captan indicate that the substance acts as an alkylating agent and induces chromosome rearrangements in rats and point mutations in *Neurospora crassa* (Epstein and Legator, as cited in ACGIH 1986, p. 98). Legator and colleagues (1969) reported that concentrations of 10 ug/ml inhibited DNA in human embryo cells, and concentrations of 1.5 ug/ml produced chromosomal aberrations in somatic and germ cells of kangaroo rats. Animal evidence concerning carcinogenicity is contradictory, although high doses caused significant incidences of polypoid carcinoma of the duodenum in mice, as well as adenomatous polyps (NCI 1977).

Some exposed individuals experience skin irritation (Spencer 1968). A case of recurrent urticaria caused by captan exposure has been reported and confirmed (Croy 1973), and captan exhibited high reactivity when administered in a battery of patch tests (Rudner 1977).

OSHA is proposing a PEL of 5 mg/m³ TWA to protect workers exposed to captan from the risk of skin irritation and reproductive effects associated with exposure to this previously unregulated substance. This health evidence forms a reasonable basis for proposing a new limit for captan. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

CHLOROPRENE
CAS: 126-99-8; Chemical Formula: $CH_2{:}CClCH{:}CH_2$
H.S. No. 1088

The current OSHA limit for chloroprene is 25 ppm TWA with a skin notation. The ACGIH has recommended a 10-ppm TLV-TWA, with a skin notation, and NIOSH (1977c) recommended a limit of 1 ppm, measured over a 15-minute period.

The ACGIH recommended a reduction in the TLV from 25 ppm to 10 ppm in 1981 based on a review of the world literature by Trochimowicz, who prepared the 1980 ACGIH proposed documentation, and by Reinhardt (personal communication 1980, as cited in ACGIH 1986, p. 135). Reinhardt concluded that there was no evidence indicating a lack of safety associated with the 25-ppm TLV, but "minimal systemic effects" (i.e., growth retardation) seen in rats and hamsters exposed to 39 ppm for 4 weeks or 50 ppm for a lifetime suggest that an additional margin of safety was in order. Therefore, the ACGIH reduced the TLV-TWA to 10 ppm.

In recommending a 1-ppm 15-minute exposure limit, NIOSH (1977c) cited three reports on plants in the Soviet Union. Katosova (1973) reported finding a significant excess of chromosomal abnormalities in the blood of workers exposed to approximately 5 ppm chloroprene. Volkova et al. (1976) reported similar findings in a plant where chloroprene levels ranged from 0.8 to 1.95 ppm. In the third study, Sanotskii (1976) reported abnormal sperm morphology among workers exposed from 0.28 to 1.94 ppm; a threefold increase in the rate of spontaneous abortion among wives of the workers was also found. In addition, NIOSH (1977c) cited a study by Davtian et al. (1973), who reported a significant excess of embryonic mortality in female rats that were mated to male rats exposed to 1 ppm chloroprene. The investigators also found chromosomal aberrations in bone marrow cells of exposed male rats. NIOSH (1977c) also cited a number of reports showing chloroprene to be mutagenic in a variety of test systems. NIOSH concluded that it was prudent to limit exposure to 1 ppm over a 15-minute period, to limit the risk of genetic abnormalities being transmitted to subsequent generations. This limit represents the lowest concentration that can be measured reliably over a 15-minute period.

The 1-ppm (15-minute STEL) value recommended by NIOSH is based on studies reported in the USSR literature, and it represents an analytical and sampling limit of detection. The 10-ppm TLV-TWA is based on a 1981 critical review of the world literature and the observation that minimal systemic effects are observed at 38 ppm. OSHA proposes to adopt the 10-ppm TWA as a PEL to substantially reduce the systemic effects associated with chloroprene exposure. However, the Agency solicits comments on the significance of the Russian studies as their findings relate to risks to employees. This health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for chloroprene if the Agency determines that this limit will substantially reduce significant risk

CYCLOHEXYLAMINE
CAS: 108-91-8; Chemical Formula: $C_6H_{13}N$
H.S. No. 1109

OSHA has no current limit for cyclohexylamine. The ACGIH recommends a TLV-TWA of 10 ppm. Cyclohexylamine is a liquid with a strong, fishy, amine odor.

Data concerning the acute toxicity of cyclohexylamine were reported by Eastman Kodak in 1958. In rats, the oral LD_{50} of a 5-percent solution in water was between 400 and 800 mg/kg; mice fed a diet of the 1-percent aqueous solution or the undiluted amine had LD_{50}s of between 200 and 400 mg/kg. Injection of the 5-percent aqueous solution in rats produced LD_{50}s of between 5 and 25 mg/kg, while mice injected intraperitoneally with the 1-percent solution had LD_{50}s of between 5 and 10 mg/kg. In guinea pigs, the dermal LD_{50} of undiluted cyclohexylamine is reported to be between 1 and 5 mL/kg. Edema, necrosis, and eschars were reported as a consequence of these dermal exposures. In rabbits, one drop of a 50-percent solution caused complete destruction of the eye. Six-hour inhalation exposures at a vapor concentration of 12,000 ppm caused deaths in rats, but exposure to 1000 ppm caused neither toxic effects nor deaths.

Legator, Palmer, Green, and Petersen (1969) considered cyclohexylamine to be a potential carcinogen, mutagen, or

teratogen on the basis of dose-dependent chromosomal abnormalities observed in rats injected intraperitoneally with cyclohexylamine. Khera and Stolz (1971) noted adverse effects on rat fertility, and Becker and Gibson (1970) reported embryotoxic effects in mice intraperitoneally injected with cyclohexylamine. In contrast, Kennedy, Sanders, Weinberg, et al. (1969) reported no effects of exposure to cyclohexylamine on rabbit and rat fertility, reproduction, embryogenesis, or perinatal and postnatal development.

In general, there is agreement concerning the moderate-to-severe toxicity of cyclohexylamine and its potential for intense skin irritation and moderate skin sensitization (Sax 1969). The chemical is well known to be pharmacologically active, having sympathomimetic activity (Barger and Dale 1910). However, Lichfield and Swan (1971) report that human dietary levels of 5 g/day for 7 to 8 days produced no pharmacologically active levels in the tissues; furthermore, no changes were detected in blood pressure or heart rate, or in the electrocardiograms of exposed subjects. Chronic experimental toxicity data are lacking, but Watrous and Shulz (1950) have reported that exposure to 4 to 10 ppm of cyclohexylamine caused no symptoms of any kind in acutely exposed workmen.

OSHA is proposing an 8-hour TWA PEL of 10 ppm for cyclohexylamine. The Agency preliminarily concludes that limiting workplace exposures to this previously unregulated substance to the 10-ppm level will protect workers against the risk of severe skin and eye irritation, and sensitization that are potentially associated with exposure to cyclohexylamine. This health evidence forms a reasonable basis for proposing a new limit for cyclohexylamine. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

CYHEXATIN
CAS: 13121-70-5; Chemical Formula: $(C_6H_{11})_3SnOH$
H.S. No. 1112

OSHA currently has no limit for cyhexatin. The ACGIH recommends a TLV-TWA of 5 mg/m³. At room temperature, cyhexatin exists in the form of white crystals.

Cyhexatin has oral LD_{50}s of 500, 700, and 654 mg/kg for rabbits, guinea pigs, and chickens, respectively. The intraperitoneal LD_{50} for the rat is 13 mg/kg (NIOSH 1977). and the oral LD_{50} for the rat has been reported to be 190 mg/kg (ACGIH 1974). Skin exposure to a 1- to 2-percent solution in goats and cattle caused mild effects; sheep showed mild effects after application of a 0.5-percent solution. One of 5 sheep died from multiple skin applications of a 1-percent suspension (Johnson et al. 1975).

The toxicity of cyhexatin is considered to be moderate, although it is greater than the toxicity of most other organic tin compounds. Long-term feeding studies in the rat produced no behavioral changes, mortality, tissue changes, or hematologic or biochemical changes in response to a 2-year oral dosage at 12 mg/kg per day for 2 years; however, dosed animals were smaller than controls. After daily doses by gavage of 24 mg/kg per day for 2 weeks, rats showed microscopic changes in the liver, kidneys, and adrenal glands at autopsy. Six mg/kg is considered to be the no-effect level in rats, and in dogs, the no-effect feeding level is reported to be 3 mg/kg. Rats fed 4 to 6 mg/kg, and rabbits fed 3 mg/kg, showed no ill effects on indices for fertility, gestation, viability, or lactation (Dow Chemical Company 1973). No inhalation data on animals are available and there are no human data.

OSHA proposes an 8-hour TWA limit of 5 mg/m³ for cyhexatin. OSHA preliminarily concludes that a PEL of 5 mg/m³ will protect workers against the risk of skin and other irritation associated with exposure to this tin compound in the absence of a current limit. This health evidence forms a reasonable basis for proposing a new limit for cyhexatin. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

2-N-DIBUTYLAMINOETHANOL
CAS: 102-81-8; Chemical Formula: $(C_4H_9)_2NCH_2CH_2OH$
H.S. No. 1120

OSHA currently has no limit for 2-N-dibutylaminoethanol (DBAE). The ACGIH recommends a TLV-TWA of 2 ppm, with a skin notation, for this colorless, combustible liquid, which has a faint, amine-like odor.

In rats, 2-N-dibutylaminoethanol has a single-dose oral LD_{50} of 1.7 g/kg and a corresponding intraperitoneal LD_{50} of 0.14 g/kg; these values are approximately analogous to the oral and intraperitoneal LD_{50}s for diethanolamine (Hartung and Cornish 1968). The LD_{50} for skin absorption in rabbits is 1.68 g/kg (Smyth et al. 1954). In male rats, the lowest 5-week drinking water dose tolerated without weight loss was 0.13 g/kg/day. Rats that ingested a dose of 0.43 g/kg/day showed elevated kidney-to-body-weight ratios but no histologic changes at autopsy (Cornish, Dambrauskas, and Beatty 1969). In inhalation studies of rats, 6-hour exposures at 70 ppm for 5 days killed one rat, and the surviving rats showed a 57 percent average body weight loss, as well as a doubling of kidney-to-body-weight ratio, a 10-fold increase in serumbilirubin, a slight increase in clotting time, and an elevated hematocrit. Inhalation of 33 ppm for one week caused a 3-percent body weight loss and a slight increase in clotting time, but no significant changes in the other variables observed. Twenty-seven weeks of exposure to 22 ppm caused no differences in the variables measured between exposed rats and controls (Cornish, Dambrauskas, and Beatty 1969). 2-N-dibutylaminoethanol is a more potent inhibitor of acetylcholinesterase in vitro than is diethylamine (DEA) (Hartung and Cornish 1968).

OSHA is proposing an 8-hour TWA PEL of 2 ppm, with a skin notation, for 2-N-dibutylaminoethanol. The Agency preliminarily concludes that this limit will protect exposed workers from the risk of metabolic effects associated with inhalation exposure at the levels permitted in the absence of any OSHA limit. In addition, OSHA concludes that this substance presents a risk of systemic toxicity via percutaneous absorption and that a skin notation is required to substantially reduce this risk. This health evidence forms a reasonable basis for proposing a new limit for 2-n-dibutylaminoethanol. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

DIGLYCIDYL ETHER
CAS: 2238-07-5; Chemical Formula: $C_6H_{10}O_3$
H.S. No. 1139

The current OSHA limit for diglycidyl ether (DGE) is 0.5 ppm as a ceiling concentration, and the ACGIH recommended TLV is 0.1 ppm as an 8-hour TWA. NIOSH recommends a limit of 0.2 ppm as a 15-minute ceiling.

Both the previous ACGIH 0.5 ppm TLV and that organization's current TLV are based on the results of an animal study reported by Hine and Rowe (1963) in which rats were administered repeated 4-hour exposures of 20 ppm, 3 ppm, or 0.3 ppm DGE. Rats exposed to 20 ppm of DGE showed respiratory irritation, loss of body weight, decreased leukocyte count, involution of the spleen and thymus, and hemorrhagic bone marrow. Residual hematopoietic effects were observed among rats exposed to 3 ppm, and no observed effects were

noted among rats exposed to 0.3 ppm after as many as 60 exposures. The previous TLV of 0.5 ppm as a ceiling value was based on the no-observed effect level of 0.3 ppm reported in this study and on industrial experience. In 1979, the ACGIH reconsidered its limit for DGE, noting that "in view of the seriousness of some of the effects produced [in the rat study], a TLV below the no-ill-effect level [of 0.3 ppm] would normally be adopted" (ACGIH 1986). The ACGIH consequently revised the TLV to 0.1 ppm as a TWA.

OSHA preliminarily finds that the revised TWA limit of 0.1 ppm will protect exposed workers against the hematopoietic and irritant effects to which they are potentially exposed at OSHA's current PEL. The risks of DGE exposure range from respiratory irritation to bone marrow effects. The proposed limit for DGE is intended to reduce this risk substantially. This health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, DSHA will establish a new limit for diglycidyl ether if the Agency determines that this limit will substantially reduce significant risk.

ETHANOLAMINE
CAS: 141–43–5; Chemical Formula:
$NH_2CH_2CH_2OH$
H.S. No. 1159

OSHA currently has an 8-hour TWA limit of 3 ppm for ethanolamine. The ACGIH recommends the same TWA limit and a 15-minute STEL of 6 ppm. Ethanolamine is a colorless liquid with a mild smell like that of ammonia.

The health hazards associated with exposures to ethanolamine include skin irritation and necrosis and central nervous system depression. The oral LD_{50} in rats is reported as 3.32 g/kg and the intraperitoneal LD_{50} in rats as 981 mg/kg (Hartung and Cornish 1968). The dermal toxicity is considerably higher, with the LD_{50} being reported as 1 mg/kg in the rabbit. Dermal application of the undiluted liquid also caused redness, swelling, and burns comparable to mild first-degree burns (Union Carbide Corporation, as cited in ACGIH 1986, p. 235). The eye injury potential of ethanolamine is just slightly less than that of undiluted ammonia (Carpenter and Smyth 1946). Rats fed 0.5 percent in their food for 90 days (Smyth, Carpenter, and Weil 1951) showed no adverse effects, but at 1.28 g/kg/day, fatalities occurred. Treon and associates (1975) reported lung, liver, and kidney damage in various species exposed to high concentrations of the vapor and mist. In tests of various species, Weeks and coworkers (1960) reported marked dermal effects from continuous exposures (24 hours/day, 7 days/week for from 24 to 90 days) at various concentrations of the vapor; at 12 to 26 ppm, dermal effects were less severe, but at 5 ppm, skin irritation was still evident. Dogs also experienced a slight and temporary weight loss after 90-day exposure to 5 ppm, as well as decreased activity and alertness (Weeks, Dowing, Musselman et al. 1960). Luck and Wilcox (1953) demonstrated that a portion of low-dose ethanolamine is not excreted and is presumably retained in the body of cats, rats, and rabbits.

In studies of anesthetized dogs, Priddle (1954) reported that low doses cause central nervous system stimulation, while lethal doses cause CNS depression. Ethanolamine's irritant and necrotic effect on the skin is not related to its alkalinity (Hinglais 1948).

OSHA is proposing a PEL of 3 ppm TWA and a 15-minute STEL of 6 ppm for ethanolamine. The Agency preliminarily concludes that both of these limits are required to protect workers against the risk of irritation and neuropathic effects potentially associated with exposure to the levels permitted in the absence of a short-term limit. This health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for ethanolamine if the Agency determines that this limit will substantially reduce significant risk.

ETHYLENE CHLOROHYDRIN
CAS: 107–07–3; Chemical Formula:
$ClCH_2CH_2OH$
H.S. No. 1167

OSHA currently has an 8-hour TWA limit of 5 ppm, with a skin notation, for ethylene chlorohydrin. The ACGIH recommends a ceiling limit of 1 ppm, with a skin notation. Ethylene chlorohydrin is a colorless liquid with a faint ethereal odor.

The primary health hazards associated with exposure to this substance are central nervous system effects, cardiovascular effects, liver damage, kidney damage, gastrointestinal effects, skin irritation, eye irritation, and mutagenic effects. The oral LD_{50} for rats is 72 mg/kg, and the intra peritoneal LD_{50} is 56 mg/kg (Goldblatt and Chiesman 1944). In guinea pigs, the intraperitoneal LD_{50} is 98 mg/kg, and the percutaneous LD_{50} is 205 mg/kg (Wahlberg and Bowman 1978).

The skin absorption rate for ethylene chlorohydrin is high; Semenova and associates (1978) determined that the LD_{50} must be reduced to one-fifth if ethylene chlorohydrin is administered daily for 20 days (Semenova, Kazanina, Fedyanina et al. 1978).

Inhalation toxicity is also high. Ambrose (1950) has reported that a single 1-hour exposure at 7.5 ppm and repeated 1-hour exposures at 2 ppm can be fatal to rats. Exposures of 15 minutes daily at concentrations of from 900 to 1000 ppm were fatal to rats within a few days (Goldblatt and Chiesman 1944).

In subacute and chronic studies, rats have shown fatalities from a daily dietary dose of 67.5 mg/kg (Oser, Morgareidge, Cox, and Carson 1975). Semenova and associates (1978) reported a 4-month no-effect inhalation level of 0.0033 ppm; at 0.017 ppm, slight CNS inhibition changes in the cavity of acid phosphatases and urinary secretion of nitrogen were observed after 4 months. These investigators also observed increased chromosomal aberrations in rat bone marrow at the 0.22-ppm level for 4 months (Semenova, Kazanina, Fedyanina et al. 1978).

Voost and Vet (1969) tested ethylene chlorohydrin in Klebsiella pneumoniae and found it strongly mutagenic. This finding was confirmed by the Ames test in Salmonella typhimurium; ethylene chlorohydrin reacts with DNA, since it inhibits the growth of DNA-deficient bacteria (Rosenkranz and Whodkowski 1974). A dose related increase of liver protein and glutathion depletion was observed in rats after a single dose of ethylene chlorohydrin of from 10 to 50 mg/kg (Friedman, Scalera, Balazs et al. 1977).

One fatal and several non-fatal cases of poisoning in industrial workers have been reported from exposure (for unspecified periods) to levels between 300 and 500 ppm. Autopsy of the worker who died revealed severe damage to the liver and brain, as well as effects in other organs. The survivors experienced nausea, vomiting, and irritation of the eyes, nose, and lungs (Bush, Abrams, and Brown 1949). Dierker and Brown (1944) reported that a 2-hour inhalation exposure to 300 ppm was fatal in one accidental exposure.

OSHA is proposing a ceiling limit of 1 ppm for ethylene chlorohydrin, with a skin notation. The Agency preliminarily concludes that this limit will protect workers against the risk of central nervous system and other systemic effects associated with workplace exposures at the levels permitted with a TWA limit alone. The skin notation is retained because ethylene chlorohydrin is readily absorbed through the skin. This health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for ethylene

chlorohydrin if the Agency determines that this limit will substantially reduce significant risk.

GLYCIDOL (2,3-EPOXY-1-PROPANOL)
CAS: 556–52–5; Chemical Formula: $C_3H_6O_2$
H.S. No. 1189

OSHA currently has an 8-hour TWA limit of 50 ppm TWA for glycidol. The ACGIH recommends a limit of 25 ppm TWA for this colorless liquid.

Glycidol causes eye, respiratory, and pulmonary irritation. Hine and associates (1956) conducted a study of animal toxicity caused by glycidol exposure and reported that glycidol is irritating to the lungs, with mice and rats exhibiting pneumonitis and emphysema resulting from vapor inhalation. The LC_{50} reported for mice is 450 ppm for a 4-hour exposure; the 8-hour LC_{50} for rats is 580 ppm (Hine, Kodama, Wellington et al. 1956). A single dermal application was only mildly irritating (Draize score, 4.5); repeated daily skin applications were severely irritating after 4 days. One drop of pure glycidol in the rabbit eye caused severe but reversible corneal injury (Hine, Kodama, Wellington et al. 1956). In rats, chronic exposures to 400 ppm (7 hours/day for 50 days) did not cause systemic toxicity, but eye irritation and respiratory distress were observed after the first few exposures (Hine, Kodama, Wellington et al: 1956). A study to determine glycidol's tumorigenic potential on the skin of mice showed negative results (Van Duuren et al. 1967).

OSHA proposes an 8-hour TWA limit of 25 ppm TWA for glycidol. The Agency preliminarily concludes that this limit will protect workers against the risk of eye, respiratory, and pulmonary irritation potentially associated with exposures to this substance. This health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for glycidol (2,3-epoxy-1-propanol) if the Agency determines that this limit will substantially reduce significant risk.

HEXAFLUOROACETONE
CAS: 684–16–2; Chemical Formula: C_3F_6O
H.S. No. 1198

OSHA currently has no limit for hexafluoroacetone. The ACGIH recommends a TLV–TWA of 0.1 ppm, with a skin notation, for this colorless, non-flammable, and highly reactive gas.

Inhalation studies of hexafluoroacetone in animals have shown varied systemic toxicities, including injury to the liver, kidney, testes, thymus, and bone marrow. In rats and dogs exposed 6 hours/day, 5 days/week for 13 weeks at concentrations of about 0.1, 1.0, or 12 ppm, no effects (other than increased lung weights in dogs) were observed in either species at 0.1 ppm. However, the 12-ppm exposures produced severe effects in both species, including marked but reversible testicular damage and slight hypoplasia of the spleen, thymus, and lymph nodes (E.I. du Pont de Nemours & Company, Inc. 1971 as cited in ACGIH 1986, p. 303). Reversible kidney damage in rats and increased lung weights in dogs occurred during the 1.0-ppm exposures. An earlier 4-hour acute exposure of rats demonstrated that 300 ppm was a lethal concentration (E.I. du Pont de Nemours and Company, Inc., as cited in ACGIH 1986, p. 303).

In rats, 2-week dermal exposures of 65, 130, or 250 mg/kg resulted in numerous adverse effects, including testicular damage and corresponding changes in lipid metabolism (Kennedy et al. 1982). A dermal dose of 13 mg/kg produced no adverse effects (Lee and Gillies 1984). An injected dose of radiolabeled hexafluoroacetone was, for the most part, rapidly excreted in unmetabolized form in the urine; this material also did not accumulate in rat testes (Gillies and Rickard 1984). Britelli and co-workers reported that hexafluoroacetone was fetotoxic in rats (1979). Dermal application of 90 mg/kg/day to pregnant rats resulted in maternal toxicity. Fetal toxicity occurred at maternal doses of 25 mg/kg, and fetal size was reduced at maternal doses of 5 and 25 mg/kg; however, 1 mg/kg produced no fetal effect. Although soft-tissue damage and external abnormalities were observed, teratogenicity could not be demonstrated definitively (Britelli et al. 1979).

OSHA proposes an 8-hour TWA PEL of 0.1 ppm TWA and a skin notation for hexafluoroacetone. The Agency preliminarily concludes that these limits, taken together, will protect hexafluoroacetone-exposed workers from the systemic injuries that can be manifested at multiple organ sites, reproductive effects, kidney damage, and fetotoxic effects associated with exposure to this substance at previously uncontrolled levels. This health evidence forms a reasonable basis for proposing a new limit for hexafluoroacetone. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

HYDROGEN CYANIDE
CAS: 74–90–8; Chemical Formula: HCN
H.S. No. 1207

The current OSHA limit for hydrogen cyanide is a 10-ppm TWA, with a skin notation. The ACGIH recommends a 10-ppm ceiling limit, also with a skin notation. NIOSH (1976) has recommended that workplace exposures not exceed 4.7 ppm as a 10-minute ceiling.

The ACGIH summarized the extensive body of human evidence on the adverse effects resulting from exposure to hydrogen cyanide. They note that exposure to levels of 45 to 54 ppm can be tolerated for 1 hour with no immediate or delayed effects, and that 18 to 36 ppm produces "slight" symptoms after several hours of exposure. They also cite Grabois (1954), who reported that workers in apricot kernel processing plants experienced no ill effects when exposed to hydrogen cyanide on the order of 10 ppm.

The NIOSH recommendation of 4.7 ppm as a 10-minute ceiling limit is based largely on an epidemiologic study by El Ghawabi (1976), showing an increase in symptoms of headache, weakness, throat irritation, vomiting, dyspnea, lacrimation, colic, and nervousness among workers exposed for an average of 7.5 years to cyanide concentrations ranging from 4.2 to 12.4 ppm. NIOSH also cited other papers reporting similar symptoms among cyanide-exposed workers. NIOSH acknowledged that such symptoms can be caused by a wide variety of other chemical or physical factors, but believed that these symptoms were sufficiently well characterized as being associated with cyanide exposure to warrant their consideration as indicators of health impairment. Therefore, NIOSH recommended a 4.7 ppm limit, determined from a 10-minute sample. Because of this recommendation, ACGIH considered recommending a 3-ppm TLV-ceiling for hydrogen cyanide; however, a majority of committee members favored retention of the 10-ppm ceiling limit at ACGIH's 1979 meeting.

The 10-ppm (ceiling) TLV is based on the observation of some health effects at 18 to 36 ppm and no observed effects at 10 ppm. More recent epidemiological data indicate that a variety of symptoms may be associated with exposure to hydrogen cyanide at levels less than 10 ppm. This indicates that neither the existing PEL nor the ACGIH TLV may be sufficiently protective. OSHA therefore proposes a 4.7 ppm (ceiling) as the PEL. This health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for hydrogen cynanide if the Agency determines that this limit will substantially reduce significant risk.

OSHA's preliminary feasibility analysis is based on limited data at this exposure level, and the Agency therefore requests additional feasibility information from the public.

HYDROGENATED TERPHENYLS
CAS No.: 61788-32-7; Chemical Formula: None
H.S. No. 1210

OSHA does not currently regulate the hydrogenated terphenyls. The ACGIH recommends a TLV-TWA of 0.5 ppm (approximately 5 mg/m³) TWA for these complex mixtures of ortho-, meta-, and para-terphenyls in various stages of hydrogenation.

Acute exposure to the hydrogenated terphenyls poses a risk of potential lung, eye, and skin damage. Chronic exposure presents a risk of systemic toxicity involving injury to the liver, kidneys, and blood-forming organs, as well as possible metabolic disturbances and cancer (ACGIH 1986, p. 311).

Early studies of unhydrogenated terphenyl isomers determined that the LD_{50} in rats is low, i.e., 1900 mg/kg for the ortho isomer, 2400 mg/kg for the meta isomer, and 10,000 mg/kg for the para isomer (Cornish 1962). Thirty-day oral administration of 500 mg/kg/day in the diet of rats indicated possible liver and kidney damage, which was suggested by increases in the liver and kidney to body-weight ratios and decreases in the rate of weight gain (Cornish 1962). Other studies have demonstrated nephrotoxicity and liver damage in rats fed 33 mg/kg or more of unirradiated terphenyl isomers (Petkau and Hoogstraater 1965; Young et al. 1969). Inhalation studies showed that bronchopneumonia is associated with exposure to the ortho and meta isomers, but not to the para isomer (Haley et al. 1959). Cornish's work (1962) showed that none of the isomers caused skin irritation in rabbits following a 24-hour dermal application. For terphenyls that are approximately 40 percent hydrogenated, the acute oral LD_{50} in rats is reported as 17,500 mg/kg; in mice, it is 12,500 mg/kg (Adamson and Weeks 1973). This study also demonstrated that an irradiated hydrogenated terphenyl mixture is three times more acutely toxic by ingestion than is a non-irradiated mixture. This finding was confirmed in 16-week chronic ingestion studies (Adamson and Weeks 1969); these authors found that 1200 mg/kg of an irradiated mixture was lethal to mice, while the same dose in non-irradiated form produced only an irreversible interstitial nephritis. In the same study, no effects were observed for either mixture at a dose level of 250 mg/kg.

Eight-day inhalation studies in mice showed some pathologic changes in lung tissue after 500 mg/m³ exposures to non-irradiated hydrogenated terphenyls; 8-week exposures at 2000 mg/m³ resulted in the same lung damage, as well as some proliferation of the smooth endoplastic reticulum in the liver (Adamson and Weeks 1969, 1973). Carcinogenesis in mice has been reported from 8-week skin exposures to the irradiated mixture (Henderson and Weeks 1973). The significance of the changes observed by Adamson and Furlong (1974) in the mouse lung after 8 weeks of inhalation exposure to the irradiated mixture is difficult to interpret in terms of the potential of the hydrogenated terphenyls to cause pulmonary cancer; particles were found to clear the lungs rapidly but to accumulate and clear more slowly in the intestine, kidney, and liver.

OSHA is proposing a 0.5-ppm 8-hour TWA for the complex mixtures of ortho-, meta-, and para-terphenyls (either irradiated or non-irradiated) in various stages of hydrogenation. The Agency preliminarily concludes that this limit will protect exposed workers from the risk of eye, skin, and lung damage, and of systemic toxicity to the liver, kidney, and blood-forming organs, potentially associated with exposure at the levels possible in the absence of any OSHA limit for this substance. This health evidence forms a reasonable basis for proposing a new limit for hydrogenated terphenyls. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

2-ISOPROPOXYETHANOL
CAS: 109-59-1; Chemical Formula: $(CH_3)_2CHOCH_2CH_2OH_2$
H.S. No. 1223

OSHA has no current limit for 2-isopropoxyethanol. The ACGIH recommends a TLV-TWA of 25 ppm for this mobile liquid.

2-Isopropoxyethanol has been demonstrated to produce systemic toxicity in laboratory animals. In studies of rats, fifteen 6-hour exposures at 1000 ppm caused hemoglobinuria, anemia, and lung congestion, but no fatalities (Gage 1970). At 300 ppm, Gage reported transient hemoglobin and MCHC decreases and lung congestion after 15 exposures. Exposure at the 100-ppm level produced no effect (Gage 1970). Another study reported a significant increase in the osmotic fragility of erythrocytes in female rats after a 4-hour inhalation exposure to 62 ppm, but no effect was observed at 32 ppm (Carpenter et al. 1956). Studies of four species exposed at concentrations of 200, 50, or 25 ppm 6 hours/day for 26 weeks resulted in hematologic changes only in rats; increased osmotic fragility of erythrocytes was marked at 200 ppm, slight at 50 ppm, and minimal at 25 ppm (Moffet, Linnett, and Blair 1976).

OSHA is proposing an 8-hour TWA PEL of 25 ppm for 2-isopropoxyethanol. The Agency preliminarily concludes that this limit will protect exposed workers from the risk of hemolytic effects associated with exposure to this substance, which is not presently regulated. This health evidence forms a reasonable basis for proposing a new limit for 2-isopropoxyethanol. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

ISOPROPYL GLYCIDYL ETHER
CAS: 4016-14-2; Chemical Formula: $C_6H_{12}O_2$
H.S. No. 1227

OSHA's current limit for isopropyl glycidyl ether (IGE) is 50 ppm as an 8-hour TWA. The ACGIH has established an 8-hour TWA of 50 ppm and a 15-minute STEL of 75 ppm for IGE. NIOSH recommends a limit of 50 ppm for IGE as a 15-minute ceiling. IGE is a colorless and volatile liquid.

The 4-hour LC_{50} for mice was 1500 ppm and the 8-hour LC_{50} in rats was 1100 ppm (Hine, Kodama, Wellington et al. 1956). The intragastric LD_{50}s in mice and rats were 1.30 and 4.2 g/kg, respectively; in rabbits, the dermal LD_{50} was 9.65 g/kg (Hine, Kodama, Wellington et al. 1956). Fifty daily 7-hour exposures of rats to 400 ppm caused a reduced rate of weight gain, an increase in hemoglobin, a decrease in peritoneal fat, and, in some animals, emphysematous lungs and mottling of the liver (Hine, Kodama, Wellington et al. 1956). Animals in this study also exhibited signs of ocular irritation and respiratory distress.

In humans, eye, nose, and upper respiratory irritation occurred in the technicians handling the animals in the Hine and co-workers' study; exposure levels were not specified. Dermatitis has also been reported in workers exposed to other glycidyl ethers during manufacture, and one such case involved IGE exposure (ACGIH 1986, p. 340).

OSHA is proposing an 8-hour TWA of 50 ppm and a 15-minute STEL of 75 ppm for IGE. The Agency preliminarily concludes that these limits will work together to protect workers from the risk of eye, skin, and upper respiratory tract irritation associated with exposures at the levels permitted in the absence of a short-term limit. This health evidence

forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for isopropyl glycidyl ether if the Agency determines that this limit will substantially reduce significant risk.

4,4'-METHYLENE BIS(2-CHLOROANILINE)
CAS: 101-14-4; Chemical Formula: $CH_2(C_6H_4ClNH_2)_2$
H.S. No. 1273

OSHA currently has no limit for 4,4-methylene bis (2-chloroaniline), or MBOCA, a tan-colored solid. The ACGIH recommends a limit of 0.02 ppm TWA, with a skin notation; MBOCA is classified as a suspected human carcinogen (A2) by the ACGIH. NIOSH recommends a TWA limit of 3 mg/m^3, which is the lowest detectable limit.

MBOCA is highly toxic, causing cyanosis, kidney irritation, and methemoglobinemia. It is similar in effect to the other aromatic amines (Hosein and van Roosmalen 1978; Mastromatteo 1965.)

Steinhoff and Grundman (1969) demonstrated that feeding MBOCA at unspecified levels to rats on a protein-deficient diet caused a high incidence of liver cancer. Russfield and associates (1975) reported liver and lung tumors in rats fed MBOCA while on a standard diet. Dogs fed MBOCA at a dose of 100 mg/day, 5 days/week showed no hepatic cancer, but malignant nodules in the bladder occurred in a dog fed MBOCA for 9 years (Stula et al. 1977).

In industry, reversible hematuria has been reported among MBOCA-exposed workers, but precise concentration data are lacking (Mastrometteo 1965). However, a study of workers exposed for as long as 18 years to MBOCA showed no adverse effects, although the substance and its metabolites were detected in the urine of these subjects (Linch et al. 1971). Hosein and van Roosmalen (1978) reported an industrial incident where molten MBOCA was splashed in a worker's face; urinary levels of 3.6 mg/L MBOCA, as well as protein, were detected in the urine, and the subject experienced nausea. However, this worker recovered quickly.

A recent NIOSH retrospective study involving 370 workers employed in a MBOCA-manufacturing plant evaluated the carcinogenicity of this substance, which is structurally similar to benzidene. The study has found 2 bladder cancers in very young workers (less than 30 years of age), both of whom are non-smokers.

OSHA is proposing an 8-hour TWA limit of 0.02 ppm for MBOCA, with a skin notation. The Agency preliminarily concludes that this limit will protect workers against the risk of cyanosis, methemoglobinemia, kidney irritation, and cancer potentially associated with exposures to this substance at the levels permitted in the absence of any OSHA limit. A skin notation is proposed to protect against the percutaneous absorption and systemic toxicity demonstrated for this substance in industrial accidents. This health evidence forms a reasonable basis for proposing a new limit for 4,4'-methylene bis (2-chloroaniline). At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

PHENYLHYDRAZINE
CAS: 100-63-0; Chemical Formula: $C_6H_5NHNH_2$
H.S. No. 1317

OSHA's current limit for phenylhydrazine is 5 ppm TWA, with a skin notation. The ACGIH recommends a TLV-TWA of 5 ppm with a STEL of 10 ppm, and a skin notation. NIOSH (1978a) recommends that workplace exposures not exceed 0.14 ppm as measured over a 2-hour period.

No data are available on the effects of phenylhydrazine resulting from inhalation. The ACGIH limits are based on the high acute toxicity of the compound when administered orally or subcutaneously to animals; single doses on the order of 20 mg/kg have resulted in the death of dogs within 22 days (Hease et al. 1935) and produced a marked decrease in erythrocyte count in rodents (von Oettingen and Deichmann 1936). Anemia and hemolysis are the characteristic responses seen in animals fed or injected with phenylhydrazine.

In its criteria document on hydrazines, NIOSH (1978a) reviewed four studies on the carcinogenicity of phenylhydrazine in mice. One study (Toth and Shimizu 1976) found significant increases in blood vessel tumors. Another study (Clayson et al. 1966) reported increased incidences of lung adenomas and adenocarcinomas. Two other studies (Roe et al. 1967; Kelly et al. 1969) were negative. NIOSH concluded that phenylhdyrazine should be considered a potential human carcinogen and recommended that exposures not exceed 0.14 ppm over a 2-hour sampling period, which represents the lowest level that can be detected reliably. The ACGIH (1986) has placed phenylhydrazine on its A2 list.

As discussed previously, the NIOSH REL is based on the limitations of the available sampling and analytical methods for this substance; this approach does not necessarily satisfy OSHA's requirements regarding significant risk and feasibility. In addition, OSHA is not able to conduct the risk assessment necessary to consider the potential carcinogenic risks associated with occupational exposure to phenylhydrazine in time for this rulemaking. The Agency therefore proposes that a 5-ppm (8-hour) TWA and 10-ppm STEL be adopted as an interim PEL to reduce the risk of acute blood-related toxicity that has been associated with phenylhydrazine. This health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for phenylhydrazine if the Agency determines that this limit will substantially reduce significant risk. As future priorities dictate, OSHA may consider the need for a lower limit for this substance.

PHENYLPHOSPHINE
CAS: 638-21-1; Chemical Formula: $C_6H_5PH_2$
H.S. No. 1318

OSHA has no current requirement for limiting worker exposure to phenylphosphine: NIOSH also has no REL for this substance. The ACGIH has recommended a ceiling limit of 0.05 ppm.

A 90-day inhalation study conducted by the duPont Company in which rats and beagle dogs were exposed to average concentrations of 0.6 ppm or 2.2 ppm for 6 hours per day, 5 days per week, showed that rats exposed to 2.2 ppm had significant hematologic changes and testicular degeneration (as cited in ACGIH 1986, p. 479). These effects were not noted among rats exposed to 0.6 ppm, but rats exposed at the lower level did show hypersensitivity to sound and touch and mild hyperemia. The dogs tolerated the higher exposure level better than the rats in that some regeneration of testicular damage occurred during a 1-month recovery period. Dogs exposed to 0.6 ppm exhibited intermittent nausea, diarrhea, lacrimation, and hind leg tremor (ACGIH 1986). The ACGIH considered 0.6 ppm to be an NOE level for severe effects in animals and recommended a 0.05-ppm ceiling TLV to provide a 10-fold safety margin to protect workers against the changes exhibited by the test animals at the 0.6 ppm level.

OSHA preliminarily concludes that workers currently exposed to uncontrolled levels of phenylphosphine are at risk of experiencing the nausea, irritation, and CNS effects found to be associated with such exposures in animals. The Agency believes the proposed ceiling of 0.05 ppm will reduce this risk substantially. This health evidence forms a reasonable basis for

proposing a new limit for phenylphosphine. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

PHOSPHINE
CAS: 7803-51-2; Chemical Formula: PH_3
H.S. No. 1321

OSHA currently has a PEL of 0.3 ppm TWA for phosphine. The ACGIH recommends a TLV-TWA of 0.3 ppm and a TLV-STEL of 1.0 ppm. Phosphine is a colorless gas with a disagreeable, garlic-like odor.

Early studies reported that laboratory animals could tolerate phosphine in 4-hour daily exposures of 5 ppm for 2 months, but fatalities were observed from seven similar exposures at 10 ppm (Muller 1940). In 1975, Waritz and Brown reported a 4-hour LC_{50} of 11 ppm in rats; these lethal exposures caused effects typical of respiratory irritation.

Prior to 1958, numerous cases of phosphine-related occupational poisonings and deaths were reported including a fatality caused by pulmonary edema that was attributed to an exposure to 8 ppm for 2 hours daily (Harger and Spolyar 1958). Sublethal symptoms (without chronic effects) occurred at phosphine exposures averaging 10 ppm or less, with excursions of up to 35 ppm: recorded symptoms included diarrhea, nausea, vomiting, respiratory distress, and dizziness (Jones, Jones, and Longely 1964). The literature contains no documented reports of chronic poisoning caused by prolonged exposure to phosphine, although several authorities have asserted that this is a possibility (Henderson and Haggard, 1963; Fairhall 1957; Johnstone and Miller, 1940; Patty 1963; American Industrial Hygiene Association, 1964).

OSHA proposes an 8-hour TWA PEL for phosphine of 0.3 ppm and a 15-minute STEL of 1 ppm. The Agency preliminarily concludes that both of these limits are required to protect exposed workers from the risk of lung damage, diarrhea, nausea, and other effects potentially associated with elevated short-term and long-term exposure to this gas. This health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for phosphine if the Agency determines that this limit will substantially reduce significant risk.

PIPERAZINE DIHYDROCHLORIDE
CAS: 142-64-3; Chemical Formula: $C_4H_{10}N_2 \cdot 2HCl$
H.S. No. 1330

OSHA currently has no limit for piperazine dihydrochloride. The ACGIH recommends a TLV-TWA limit of 5 mgm³. Piperazine dihydrochloride is a solid. Piperazine dihydrochloride is a water-soluble solid with low systemic toxicity and mild irritant properties; the compound is biologically active. The oral LD_{50} for rats has been reported as 4.9 g/kg (NIOSH 1984).

Eye and skin irritation have been reported as a result of human exposures to high (not further specified) levels of piperazine dihydrochloride; subjects experienced mild to moderate skin burns and sensitization. Inhalation of the dust has been associated with asthmatic reactions (Dow Chemical Company, as cited in ACGIH 1986, p. 491).

OSHA proposes a limit of 5 mg/m³ as an 8-hour TWA for piperazine dihydrochloride. The Agency preliminarily concludes that this limit will reduce the risk of sensitization and eye and skin irritation potentially associated with exposures to this substance at the previously uncontrolled level. This health evidence forms a reasonable basis for proposing a new limit for piperazine dihydrochloride. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

n-PROPYL NITRATE
CAS: 627-13-4: Chemical Formula: $CH_3CH_2CH_2ONO_2$
H.S. No. 1340

OSHA currently has an 8-hour TWA limit of 25 ppm for n-propyl nitrate. The ACGIH recommends a 25-ppm TWA and adds a 15-minute STEL of 40 ppm. n-Propyl nitrate is a pale yellow liquid with a sickly sweet odor.

Rats inhaling propyl nitrate vapor for 4 hours at a concentration of 10,000 ppm demonstrated cyanosis and methemoglobinemia before they died (Hood 1953, as cited in ACGIH 1986, p. 505). Subsequent cardiac muscle effects and respiratory depression. The intravenous LD_{50} in unanesthetized rabbits has been reported to be between 200 and 250 mg/kg; in anesthetized dogs and cats, intravenous doses of between 100 and 200 mg/kg were usually fatal (Murtha, Stabile, and Wills 1956). Murtha and associates, who conducted these studies, concluded that n-propyl nitrate exerts a direct action on the vascular smooth muscle and that the ensuing cardiac effects and respiratory depression contribute to the compound's hypotensive action (Murtha, Stabile, and Wills 1956). Inhalation trials in mice, rats, hamsters, guinea pigs, and dogs have established 4-hour LC_{50} values ranging from 9000 to 10,000 ppm for rats, 6000 to 7000 for mice, and 2000 to 2500 for dogs. Dogs survived repeated exposures (6 hours/day, 5 days/week) at 260 ppm for 6 months, although slight clinical signs were observed during the first 2 weeks of exposure (Rinehart, Garbers, Greene, and Stoufer 1958). The percutaneous toxicity of n-propyl nitrate is low but may cause inflammation and thickening of the skin after repeated exposures; these effects are sometimes transient, however (ACGIH 1986, p. 505). The odor of n-propyl nitrate is detectable at 50 ppm. To protect against cardiovascular and respiratory depressant effects requires both TWA and STEL limits.

OSHA proposes a limit of 25 ppm TWA with a STEL of 40 ppm for n-propyl nitrate. The Agency preliminarily concludes that this combined PEL-STEL limit will protect workers against the risk of cyanosis, methemoglobinemia, and hypotension that have been observed in laboratory animals. This health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for n-propyl nitrate if the Agency determines that this limit will substantially reduce significant risk.

SODIUM FLUOROACETATE
CAS: 62-74-8; Chemical Formula: $CH_2FCOONa$
H.S. No. 1366

The current OSHA standard for sodium fluoroacetate is 0.05 mg/m³ as an 8-hour TWA with a skin notation. The ACGIH has established exposure limits of 0.05 mg/m³ TLV-TWA and 0.15-mg/m³ TLV-STEL, with a skin notation. Sodium fluoroacetate is a fine white powder, which is sometimes dyed black for commercial use.

Sodium fluoroacetate causes vomiting, convulsions, and ventricular fibrillation. It is highly toxic by inhalation, ingestion, or via absorption through the skin (NIOSH/OSHA, 1981). The ACGIH calculated and set the threshold limit of 0.05 mg/m³ based on studies of rats indicating an oral LD_{50} of 1.7 mg/kg (Lehman 1951). Tissue changes in rats were noted in a later study by the same author in which the animals were fed 0.25 mg sodium fluoroacetate/kg/day (Lehman 1952); the equivalent level in humans would be 17 mg/person/day. A further study by Miller and Phillips (1955) examined growth rates in rats fed various dosages of sodium fluoroacetate. Rats who received 10 ppm in their diet experienced a transient fluctuation in growth rate. At 20 ppm (approximately 2 mg/kg in young rats),

the growth rate declined markedly the first week; the rats survived and resumed growth at the normal rate in 3 to 4 weeks. Tolerance for the chemical lasted less than 2 weeks, and those rats who had adjusted to sodium fluoroacetate showed a second retardation of growth when returned to a dietary level of 20 ppm, after a 2-week interval of eating a normal diet. Miller and Phillips (1955) noted that rats conditioned to a dietary level of 20 ppm were then able to adjust to a level of 40 ppm (a dose that is greater than the single LD_{50} dose per day).

OSHA is proposing an 8-hour TWA of 0.05 mg/m³ a STEL of 0.15 mg/m³, and a skin notation. The Agency preliminarily concludes that the 8-hour and short-term exposure limits will reduce the risk of systemic effects possible as a result of exposures above the 8-hour TWA of 0.05 mg/m³. A skin notation is proposed because this substance causes systemic toxicity when absorbed dermally. This health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for sodium fluoroacetate if the Agency determines that this limit will substantially reduce significant risk.

TRIMETHYLBENZENE
CAS: 25551–13–7; Chemical Formula: $(CH_3)_3C_6H_3$
H.S. No. 1412

There is no current OSHA exposure limit for trimethylbenzene. The current ACGIH TLV for all isomers of trimethylbenzene is 25 ppm as an 8-hour TWA. NIOSH has no REL for this substance or its isomers.

A study by Battig et al. (1957) provides the basis for the proposed limit; this work reports symptoms among 27 workers exposed to a solvent containing 30 percent 1,3,5-trimethylbenzene and 50 percent 1,2,3-trimethylbenzene. A "significant number" of these workers were reported to have experienced symptoms of nervousness, tension and anxiety, and asthmatic bronchitis. The peripheral blood of these workers "showed a tendency to hypochronic anemia" and somewhat abnormal clotting ability. This group of workers had been occupationally exposed to total hydrocarbon concentrations ranging from 10 to 60 ppm for several years. The authors of the study recommended maintaining employee exposures below 35 ppm (Battig et al. 1957).

OSHA agrees that workers exposed to trimethylbenzene should be protected by an OSHA limit. OSHA believes that a 25-ppm 8-hour PEL will provide protection for trimethylbenzene-exposed workers, and a PEL set at this level is therefore proposed. This level will reduce the risk of bronchitis and anxiety previously reported in exposed workers. This health evidence forms a reasonable basis for proposing a new limit for trimethylbenzene. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

TUNGSTEN AND COMPOUNDS (insoluble)
CAS: 7440–33–7; Chemical Formula: W
H.S. No. 1416

OSHA presently has no exposure limits for insoluble tungsten and its compounds. The ACGIH has established 5 mg/m³ as an 8-hour TWA and 10 mg/m³ as a short-term exposure limit for these substances. NIOSH recommends a limit of 5 mg/m³ as a 10-hour TWA. Tungsten is a gray, hard metal.

Rats fed a diet containing 0.5 percent insoluble tungsten compounds died, and another group of rats fed 0.1 percent of these compounds suffered noticeable weight loss (Kinard and van de Erve 1941). Studies in rats fed tungsten at 2, 5, or 10 percent of their diet showed that females in all dose groups has a 15-percent reduction in weight gain (Kinard and van der Erve 1943). The intraperitoneal LD_{50} tungsten metal powder of 5 g/kg body weight; survivors showed minor liver and spleen changes at necropsy (Frederick and Bradley 1946). Studies of the tissues of guinea pigs intratracheally injected with tungsten metal and tungsten carbide revealed moderate interstitial cellular proliferation and no changes, respectively. However, Soviet studies involving similar intratracheal injections showed proliferation of the intra-alveolar septa (Kaplun and Mezentseva 1960). The NIOSH criteria document for tungsten (1977) reports that Russian investigators found a 9- to 11-percent incidence of pulmonary fibrosis in workers exposed to tungsten (Kaplun and Mezentseva 1959; Mezentseva 1967). NIOSH recommended that the standard for the tungsten and its insoluble compounds be set at 5 mg/m³ to protect against pulmonary effects. To date, studies of industrial conditions have yielded evidence that pneumoconiosis does not develop from exposure to tungsten metal or its insoluble compounds (Dernehl 1966, as cited in ACGIH 1986, p. 614).

OSHA is proposing an 8-hour TWA of 5 mg/m³ and a STEL of 10 mg/m³ for tungsten and its insoluble compounds. The Agency preliminarily concludes that these limits will protect exposed workers against the risk of pulmonary fibrosis and other lung effects associated with exposure to this metal and its compounds at the levels permitted by the absence of any OSHA limit. This health evidence forms a reasonable basis for proposing a new limit for tungsten and compounds (insoluble). At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

TUNGSTEN AND COMPOUNDS (soluble)
CAS: 7440–33–7; Chemical Formula: W
H.S. No. 1417

OSHA has no current limit for exposure to tungsten and its soluble compounds. The ACGIH limit is 1 mg/m³ TWA with a 3 mg/m³ STEL. NIOSH recommends a 1-mg/m³ 10-hour TWA for tungsten and its soluble compounds. Tungsten is a grey, hard metal.

Animal studies have shown that the LD_{50} for soluble sodium tungstate when injected subcutaneously in rats ranges from 140 to 160 mg/kg (Kinard and van de Erve 1940). Soluble tungsten's lethal effects are the result of systemic poisoning that occurs as the compound is absorbed by multiple organs; this is followed by cellular asphyxiation (International Labour Office [ILO] 1934). Karantassis (1924) also observed a systemic response in guinea pigs given soluble sodium tungstate or pure soluble tungsten either orally or intravenously, developed anorexia, colic, trembling, and difficulty in breathing prior to death. Rats fed a diet containing 0.5 percent tungsten as soluble sodium tungstate or tungsten oxide died from this dose. Dietary doses of 0.1 percent tungsten oxide and the sodium salt caused weight loss in rats, but no deaths (Kinard and van der Erve 1941). Tungsten is believed to act by antagonizing the action of molybdenum (Higgins, Richert, and Westerfield 1956). NIOSH states in its criteria document for tungsten (1977) that information on the effects of exposure to soluble tungsten compounds in the working population is unknown. The ACGIH (1986, p. 614) recommends a lower TLV for the soluble compared with the insoluble compounds of tungsten because of the former's greater systemic toxicity.

OSHA proposes an 8-hour TWA of 1 mg/m³ and a STEL of 3 mg/m³ for tungsten and its soluble compounds. The Agency preliminarily concludes that these limits will protect exposed workers against the risk of systemic toxicity, anorexia, colic, incoordination, trembling, and dyspnea associated with exposure to these compounds, which are presently not regulated by OSHA. This health evidence forms a reasonable

basis for proposing a new limit for tungsten and compounds (soluble). At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

VINYLIDENE CHLORIDE (1,1-DICHLOROETHYLENE)
CAS: 75-35-4; Chemical Formula: $CH_2=CCl_2$
H.S. No. 1428

Currently, OSHA's Z tables do not have a limit for vinylidene chloride (VCD). The ACGIH has established 5 ppm as an 8-hour TWA and 20 ppm as a 15-minute STEL. NIOSH recommends that employee exposure to VCD be reduced to the lowest feasible level, and considers VCD a carcinogen. Vinylidene chloride is a colorless liquid that polymerizes readily.

The acute oral LD_{50} for male rats is 2500 mg/kg (Jenkins, Trabulus, and Murphy 1972). The LC_{50} for rats exposed to a single 4-hour exposure of VCD vapor was reported as 6350 ppm in one study (Siegel, Jones, Coon, and Lyon 1971) and 32,000 ppm in an earlier study (Carpenter, Smyth, and Pozzani 1949). Liquid VCD causes transient irritation to the eyes of rats but has little effect on exposed skin if the VCD is allowed to evaporate (Torkelson and Rowe 1981).

Prendergast and co-workers exposed rats, rabbits, guinea pigs, and monkeys 8 hours/day, 5 days/week for 6 weeks to 395 mg/m³ (100 ppm); these authors saw no visible signs of toxicity while the exposure was in process, but rabbits and monkeys lost weight. These same species were exposed continuously to VCD concentrations of 5, 15, 25, or 47 ppm for 90 days; only the animals exposed to 5 ppm showed no increases in mortality (Prendergast, Jones, Jenkins, and Siegel 1967).

Nasal irritation, liver cell degeneration, and retarded weight gain were reported in rats following twenty 6-hour exposures to 500 ppm VCD (Gage 1970); at 200 ppm, only nasal irritation occurred. Studies by Torkelson and Rowe (1981) in which rats, rabbits, guinea pigs, and dogs were exposed to 25, 50, or 100 ppm VCD for 8 hours per day, 5 days per week for 6 months revealed injury of the kidneys and liver in all animals at all levels of exposure. Maltoni (1977) and Maltoni, Cotti, Morisi, and Chieco (1977) conducted an evaluation of VCD's carcinogenicity in which mice, rats and hamsters were exposed to levels from 10 ppm to 150 ppm for 4 hours per day, 5 days per week for 52 weeks, with results reported through week 98 of the study. In those mice exposed to 25 ppm VCD, 21 percent of the males and 1.5 percent of the females developed kidney adenocarcinomas; these tumors were not seen in rats exposed to amounts of VCD up to 150 ppm. Exposures of 100 or 150 ppm in rats did produce a significant increase in mammary adenocarcinomas, and this response was dose-related (Maltoni 1977; Maltoni, Cotti, Morisi, and Chieco 1977). Overt toxicity and mortality occurred early in the studies after 4-hour exposures at levels of 50 ppm in mice and 200 ppm in rats; hamsters exposed to 20 ppm VCD showed no increase in tumor incidence (Maltoni 1977; Maltoni, Cotti, Morisi, and Chieco 1977).

A study by Murray, Nitschke, Rampy, and Schwetz (1979) investigated the embryotoxic, fetotoxic, and teratogenic effects of inhaled and ingested VCD (in rats) and inhaled VCD (in rabbits). In the inhalation studies, rats were exposed to 20, 80, or 160 ppm VCD for 7 hours per day. VCD was toxic to both the adults and their embryos at levels of 80 and 160 ppm among the rats, and 160 ppm in rabbits. At exposure levels of 20 ppm in rats and 80 ppm in rabbits, neither maternal toxicity nor effects on embryonic or fetal development were noted. In the ingestion study with rats, drinking water containing 200 ppm VCD caused no toxic effects in either the rats or their offspring.

Two strains of rats exposed to 75 or 100 ppm VCD for 5 days/week, 6 hours/day for 12 months did not show a significant increase in tumors (Viola and Caputo 1977). Other investigators exposed rats to 25 or 75 ppm by inhalation for 6 hours/day, 5 days/week for 18 months or to 60, 100, or 200 ppm VCD in their drinking water for 2 years and found no increase in tumor incidence in these animals (Rampy, Quast, Humiston et al. 1977). In mice, VCD was not active either as a whole mouse skin carcinogen or by subcutaneous injection.

In other studies, VCD proved mutagenic in both E. coli and S. typhimurium strains (Greim, Bonse, Radwan et al. 1975; Bartsch, Malaveille, Montesano, and Tomatis 1975). VCD has been implicated as a tumor initiator in a carcinogenesis bioassay by Van Duuren (1979). Studies by Reitz, Watanabe, McKenna et al. (1980) suggest that VCD's tumorigenicity is a result of its ability to initiate cell injury and not of its ability to alter the genetic material of an injured cell. The actual cell injury is caused by VCD metabolites which are highly reactive and cytotoxic (Maltoni 1977; Hathaway 1977; Henschler and Bonse 1977).

A cohort study of 138 VCD-exposed workers did not identify any VDC-related health effects in these workers (Ott, Fishbeck, Townsend, and Schneider 1976).

OSHA is proposing an 8-hour TWA of 5 ppm and a 15-minute STEL of 20 ppm for vinylidene chloride. The Agency preliminarily concludes that these limits will protect workers from the risk of kidney and liver damage and carcinogenicity potentially associated with exposure to VCD at the levels permitted by the absence of any OSHA limit. This limit may be an interim limit; as future priorities permit, the Agency may perform a quantitative risk assessment for VCD and consider further rulemaking. This health evidence forms a reasonable basis for proposing a new limit for vinylidene chloride (1,1-dichloroethylene). At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

WELDING FUMES
CAS: None; Chemical Formula: Not available
H.S. No. 1430

OSHA currently has no limits for exposure to welding fumes, which it defines as fumes that are generated by the manual metal arc or oxy-acetylene welding of iron, mild steel, or aluminum. The ACGIH has set an 8-hour TWA of 5 mg/m³ for welding fumes, measured as total particulate inside the welding helmet.

Although welding of these types generally produces fumes made up of aluminum, iron, or zinc oxides, other toxic gases may be produced in large amounts (Ferry and Ginther 1952; Ferry 1954; Silverman 1956; Homer et al. 1957). Iron metals may give off fumes of manganese, silicate, and various organic binders. Aluminum welding may result in fumes consisting of fluorine, arsenic, copper, silicon, and beryllium (NIOSH n.d.; American Welding Society 1973). Eighteen different substances, including fluoride, manganese, silicon, titanium, and sodium and potassium silicates, have been measured in the fumes resulting from the welding of mild steel (ACGIH 1986, p. 634). The process of shielded arc welding is known to produce ozone, and when carbon dioxide is used as a shield gas, carbon monoxide is given off (NIOSH n.d.; American Welding Society 1973).

The adverse health effects associated with over exposure to welding fumes are those of metal fume fever—chills and fever, profuse sweating, and weakness—and respiratory irritation.

OSHA preliminarily concludes that a PEL for welding fumes is needed to protect workers involved in the welding of aluminum, iron, or mild steel from the

risk of metal fume fever and respiratory irritation associated with welding fumes, and is proposing to adopt a TWA of 5 mg/m³ for welding fumes as total particulate. This health evidence forms a reasonable basis for proposing a new limit for welding fumes. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

ZINC OXIDE (FUME)
CAS: 1314–13–2; Chemical Formula: ZnO
H.S. No. 1437

OSHA's existing exposure limit for zinc oxide fume is 5 mg/m³ as an 8-hour TWA. The ACGIH also recommends a 5-mg/m³ TWA and also has a STEL of 10 mg/m³. NIOSH recommends a 5-mg/m3 10-hour TWA limit with a 15-minute ceiling of 15 mg/m³. When heated. zinc oxide produces a white fume.

The most prevalent toxic effect of zinc oxide fume is a condition known as "metal fume fever," whose symptoms include chills, fever, muscular pain, nausea, and vomiting (Turner and Thompson 1926). Studies in the workplace have shown that welders exposed to zinc oxide fumes at concentrations of 320 to 580 mg/m³ reported nausea, with the development of chills, shortness of breath, and severe chest pains 2 to 12 hours later. Most workers took approximately 4 days to recover, and some eventually developed pneumonia (Hammond 1984). Other studies have reported the frequent occurrence of chills in workers exposed to zinc oxide at levels as low as 5 mg/m³ (Hickish 1963; Wall 1970). Hammond (1984) reported that workers exposed to 8 to 12 mg/m³ of zinc oxide fume did not suffer from metal fume fever.

Exposure of guinea pigs lasting only an hour caused a drop in body temperature, followed 6 to 18 hours later by an increase above normal levels (Turner and Thompson 1926). The animals in the high exposure group (2500 mg/m³ for 3 to 4 hours) died after exposure.

Early studies (Drinker and Fairhall 1957) suggested that metal fume fever was unlikely to occur at concentrations below 15 mg/m³, but subsequent experience shows that exposures even at 5 mg/m³ can cause this syndrome (Hickish, private communication, 1963; Wall, private communication, 1970, as cited in ACGIH 1986, p. 646).

NIOSH's Criteria Document (1975) reported that the development of metal fume fever was unlikely at levels as low as 5 mg/m³, but the institute stated that exposures to the fume could cause chronic respiratory effects.

OSHA is proposing a 5-mg/m³ TWA and a STEL of 10 mg/m³. The Agency preliminarily concludes that both of those limits will protect exposed workers from the risk of metal fume fever associated with exposure to zinc oxide fumes at the elevated short term levels permitted in the absence of a STEL. This health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for zinc oxide (fume) if the Agency determines that this limit will substantially reduce significant risk.

ZIRCONIUM COMPOUNDS
CAS: 7440–67–7; Chemical Formula: Zr
H.S. No. 1439

The current OSHA limit for zirconium compounds is an 8-hour TWA of 5 mg/m³. The ACGIH has established a TLV–TWA of 5 mg/m³, supplemented by a 10 mg/m³ STEL. Zirconium compounds may be either bluish-black powders or grayish-white lustrous metals.

The toxic effects of inhalation exposures to zirconium compounds include the formation of granulomas, both in the lungs and on the skin. Sax (1984) reports cases of pulmonary granulomas in workers exposed to zirconium aerosols. In laboratory animals, oral toxicity is low (NIOSH 1972), and inhalation studies conducted for one year at levels of 3.5 mg zirconium/m³ dust and mist resulted in limited toxicity (Stokinger 1981).

OSHA preliminarily concludes that the 5-mg/m³ TWA and 10-mg/m³ STEL limits for zirconium compounds will protect exposed workers from the risk of pulmonary effects potentially associated with the short-term exposures permitted by the 8-hour TWA alone. This health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for zirconium compounds if the Agency determines that this limit will substantially reduce significant risk.

Preliminary Conclusions

For the group of systemic toxicants shown on Table C8–1, OSHA preliminarily concludes that the risks associated with occupational exposures to these substances are substantial. As Table C8–2 shows, the systemic effects caused by such exposures include liver and kidney damage, testicular damage, fetal poisoning, central nervous system depression, and asthma. Affected employees may experience dizziness, nausea, generalized weakness, respiratory irritation, blood in the urine, chest tightness, hives, and necrosis of the cornea. These effects represent risks to health and functional capacity, and reducing the limits for these systemic toxins will substantially reduce these risks. The health evidence for these substances is a reasonable basis for proposing revised or new limits for substances in this group. At the time of the final rule, OSHA will establish revised or new limits for these systemic toxins if the Agency determines that these limits will substantially reduce significant risks.

9. Substances for Which Proposed Limits Are Based on Observed No-Effects Levels

Introduction

For a group of 23 toxic substances, OSHA is proposing limits based on evidence that the levels chosen have been shown not to produce adverse effects in exposed populations. These substances are shown in Table C9–1, along with their CAS numbers and H.S. numbers and current OSHA, ACGIH, and NIOSH limits. OSHA is proposing limits for 17 chemicals in this group that have not formerly been regulated by the Agency. The Agency is proposing to retain or decrease the 8-hour limit and to add a STEL in a total of five cases and to reduce the TWA in one instance. NIOSH has RELs only for one substance in this class.

Description of the Health Effects

The substances included in this group cause a wide range of adverse health effects in both animals and humans. Unlike most of the other groupings described in this preamble, these toxicants do not affect the same target organ or system: Some are central nervous system depressants, several are upper respiratory tract irritants, and still others have their primary effect on the liver and/or kidney.

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Table C9-1. Substances for Which Limits Are Based On A No-Effect Level

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1029 Atrazine	1912-24-9	--	5 mg/m^3 TWA	--
1041 Bromacil	314-40-9	--	1 ppm TWA	--
1056 p-tert-Butyltoluene	98-51-1	10 ppm TWA	10 ppm TWA 20 ppm STEL	--
1085 Chlorodifluoromethane	75-45-6	--	1000 ppm TWA 1250 ppm STEL	--
1090 o-Chlorotoluene	95-49-8	--	50 ppm TWA 75 ppm STEL	--
1110 Cyclonite	121-82-4	--	1.5 mg/m^3 TWA, 3 mg/m^3 STEL, Skin	--
1117 2,6-di-tert-Butyl-p-cresol	128-37-0	--	10 mg/m^3 TWA	--
1134 Diethanolamine	111-42-2	--	3 ppm TWA	--
1136 Diethyl phthalate	84-66-2	--	5 mg/m^3 TWA	--

Table C9-1. Substances for Which Limits Are Based On A No-Effect Level (continued)

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL**
1144 Dinitolmide	148-01-6	--	5 mg/m^3 TWA	--
1147 Diphenylamine	122-39-4	--	10 mg/m^3 TWA	--
1153 Diuron	330-54-1	--	10 mg/m^3 TWA	--
1249 Methyl acetate	79-20-9	200 ppm TWA	200 ppm TWA 250 ppm STEL	--
1275 Metribuzin	21087-64-9	--	5 mg/m^3 TWA	--
1297 Oil mist (mineral)	8012-95-1	5 mg/m^3 TWA	5 mg/m^3 TWA 10 mg/m^3 STEL	--
1312 Petroleum distillates (naphtha)	8002-05-9	500 ppm TWA	400 ppm TWA	87 ppm TWA 450 ppm Ceiling (15 min)
1327 m-Phthalodinitrile	626-17-5	--	5 mg/m^3 TWA	--
1332 Platinum, metal	7440-06-4	--	1 mg/m^3 TWA	--
1346 Resorcinol	108-43-3	--	10 ppm TWA 20 ppm STEL	--

Table C9-1. Substances for Which Limits Are Based On A No-Effect Level (continued)

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL**
1382 Tantalum	7440-25-7	5 mg/m^3	5 mg/m^3 TWA 10 mg/m^3 STEL	—
1410 Trimethyl phosphite	121-45-9	—	2 ppm TWA	—
1415 Triphenyl amine	603-34-9	—	5 mg/m^3 TWA	—
1418 Uranium (insoluble compounds)	7440-61-1	0.25 mg/m^3 TWA	0.2 mg/m^3 TWA 0.6 mg/m^3 STEL	—

* OSHA's TWA limits are for 8-hour exposures; its STELs are for the durations specified; and its ceilings are peaks not to be exceeded for any period of time.

** The ACGIH TWA-TLV is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

*** NIOSH TWA limits are for 10-hour exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

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For some compounds in this category, no adverse health effects have been observed in animals or humans. This is the case because the research conducted did not reveal effects at the doses and durations tested. Compounds falling into this latter sub-group include 2,6-di-tert-butyl-p-cresol, diethanolamine, dinitolmide, diuron, oil mist, m-phthalodinitrile, resorcinol, triphenyl amine, and the insoluble uranium compounds.

The commonality among these otherwise diverse substances is that apparent no-effect levels have been defined for all of them. Permissible exposure limits have been developed for these chemicals by applying safety factors to these no-effect levels. Table C9–2 shows the health effects observed in humans and animals after exposure to these substances.

Dose-Response Relationships and No-Effect Levels

The concept of setting limits based on a NOE level assumes that there is a concentration at which repeated and prolonged exposure to a toxic substance causes no adverse effect in the majority of workers. A similar concept is widely used by a variety of federal agencies, for example the Food and Drug Administration, to set contaminant tolerances, acceptable daily intake values, and other limits.

All of the limits for these substances have been set, with varying degrees of confidence, at a no-effect or minimal effect level, regardless of the specific effect being protected against. At least in part, the limits proposed for the 23 items listed in Table C9–1 are based on published or unpublished data or information indicating that these limits are already being maintained in work environments and that industrial experience shows these levels to be both feasible and free of associated health effects or employee complaints. OSHA believes that these limits will also protect against any effects these substances have been shown to cause at higher concentrations (e.g., minimal effects are noted in animals at 50 times the TLV–TWAs for trimethylphosphite and chlorodifluoromethane). The substances in this group have effects that range in toxicity from low to high at relatively low exposure concentrations.

TABLE C9–2.—HEALTH EFFECTS ASSOCIATED WITH SUBSTANCES FOR WHICH LIMITS ARE BASED ON NO-EFFECT LEVELS

H.S. number chemical name	CAS No.	Health effects observed in animals	Health effects observed in humans
1029 Atrazine	1912-24-9	Ataxia, dyspnea, convulsions.	
1041 Bromacil	314-40-9	Mild irritation.	
1056 p-tert-Butyl-toluene	98-51-1	CNS depression, respiratory tract irritation, liver and kidney changes	Nasal irritation, nausea, headache, weakness.
1085 Chlorodifluoromethane	75-45-6	Cardiac sensitization.	
1090 o-Chlorotoluene	95-49-8	Weakness, vasodilation, incoordination, convulsions.	
1110 Cyclonite	121-82-4		Nausea, vomiting, convulsions.
1117 2,6-di-tert-Butyl-p-cresol	128-37-0		
1134 Diethanolamine	111-42-2	Impaired vision skin irritation.	
1136 Diethyl phthalate	84-66-2		Pain, numbness, transient irritation, polyneuritis.
1144 Dinitolmide	148-01-6	Liver changes.	
1147 Diphenylamine	122-39-4	Liver, kidney, spleen changes	Tachycardia, bladder symptoms, hypertension, eczema.
1153 Diuron	330-54-1		
1249 Methyl acetate	79-20-9		Eye, mucous membrane irritation, chest tightness.
1275 Metribuzin	21087-64-9	CNS depression, thyroid and liver changes.	
1297 Oil mist (mineral)	8012-95-1	Lung irritation.	
1312 Petroleum distillates (naphtha).	8002-05-9	Motor incoordination, convulsions	Eye,throat irritation.
1327 m-Phthalodinitrile	626-17-5	Skin irritation.	
1332 Platinum, metal	7440-06-4		
1346 Resorcinol	108-43-3		
1382 Tantalum	7440-25-7	Bronchitis, pneumonitis, hyperamia.	
1410 Trimethyl phosphite	121-45-9	Teratogenicity, ocular irritation.	
1415 Triphenyl amine	603-34-9	Skin irritation.	
1418 Uranium (insoluble compounds).	7440-61-6	Kidney damage, blood disorders.	

The following discussions describe OSHA's preliminary findings for some of the substances in this group and illustrate the nature of the risk faced by workers exposed to these toxicants.

ATRAZINE

CAS: 1912-24-9; Chemical Formula: $C_8H_{14}ClN_5$

H.S. No. 1029

OSHA has no current limit for atrazine. The ACGIH recommends a TLV–TWA of 5 mg/m.[3] Atrazine is a stable, white, crystalline compound.

Animal studies indicate that the toxicity of the s-triazine herbicides, of which atrazine is the best known, is low. No observable effects have been shown in rats, dogs, horses, and cattle fed dietary levels of over 25 ppm for extended periods. In addition, the s-triazine herbicides are excreted in urine and feces in relatively short periods of time (Bakke et al. 1972). Atrazine has not shown teratogenic effects in studies of rats, mice, and sheep (the Merck Index 1983; Peters and Cook 1973; Binns and Johnson 1970). *In vitro* studies have shown no mutagenic effects, and a 2-year feeding study in rats and mice showed no carcinogenic effects (Innes et al. 1969). The only reports of toxicity indicate that high-dose ingestion of atrazine can cause ataxia, dyspnea, and convulsions in animals (as cited in ACGIH 1986, p. 44). The s-triazines appear to interfere with carbohydrate metabolism by blocking the production of sugars (Gysin 1962; Gast 1958).

In humans there are no reports of atrazine poisoning (ACGIH 1986, p. 44). Because there are no reports of human reactions to atrazine that can be related to air concentrations, the ACGIH's limit was set on the basis of animal studies. Long-term feeding studies in dogs have established 3.75 mg/kg as the highest no-observed-effect level (U.S. EPA 1979). Applying appropriate safety factors to this value, and assuming that lung absorption is less than 50 percent, yields an 8-hour TWA limit of 5 mg/m^3 (Zielhuis and Van der Kreek 1979).

OSHA proposes a PEL of 5 mg/m³ TWA for atrazine and preliminarily concludes that this limit will protect employees from the risk of metabolic effects to which they could potentially be exposed in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for atrazine. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

BROMACIL
CAS: 314-40-9; Chemical Formula: $C_9H_{13}BrN_2O_2$
H.S. No. 1041

OSHA has no current limit for bromacil. The ACGIH recommends a TLV–TWA of 1 ppm. Bromacil is a white, crystalline solid.

Bromacil has a low order of acute and chronic toxicity (Sherman 1975). In 2-year feeding studies of rats, no-effect dietary concentrations were determined to be greater than 250 ppm but less than 1250 ppm for rats and 1250 ppm for dogs. Rats and rabbits exhibited no teratogenic or carcinogenic effects as a result of dietary intake (Sherman 1975). Inhalation studies in rats have shown that all rats tolerate a 4-hour exposure equivalent to 4800 mg/m³. Studies of guinea pigs showed no skin sensitization and only mild irritation after exposures at unspecified levels. Rabbits have shown no clinical signs of toxicity as a result of the skin application of 5000 mg/kg (ACGIH 1986, p. 64).

OSHA proposes a permissible exposure limit of 1 ppm for bromacil, and preliminarily concludes that this limit will protect exposed employees against the risk of irritation potentially associated with exposure to bromacil at uncontrolled levels. The health evidence forms a reasonable basis for proposing a new limit for bromacil. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

p-tert-BUTYLTOLUENE
CAS: 98-51-1; Chemical Formula: $(CH_3)_3C—C_6H_4CH_3$
H.S. No. 1056

OSHA currently has a limit of 10 ppm TWA for p-tert-Butyltoluene. The ACGIH recommends a TLV–TWA of 10 ppm and a TLV–STEL of 20 ppm. p-tert-Butyltoluene is a colorless liquid with an aromatic, gasoline-like odor.

p-tert Butyltoluene has been shown to be slightly toxic on ingestion, moderately toxic when inhaled, and negligibly toxic through skin exposure (Hine, Unger, Anderson et al. 1954). Repeated exposures in animals have shown liver and kidney changes and microscopic degenerative hemorrhages in the spinal cord and brain, even at relatively low concentrations. The chief acute effects in animals are central nervous system depression and respiratory irritation; in rats exposed for 1 to 7 hours daily over a 26-week period, 25 ppm daily appeared to be the no-effect level (Gerarde 1960).

In humans, Hine, Unger, Anderson, et al. (1954) observed nasal irritation, nausea, malaise, headache, and weakness associated with exposure to p-tert-butyltoluene at unspecified levels. These authors also observed cardiovascular effects, as well as effects on the central nervous system, the skin, and the respiratory tract. Half of the subjects exposed to p-tert-butyltoluene developed tremor and anxiety, and 25 percent of exposed individuals showed evidence of chemical contact irritation of the respiratory tract.

OSHA is proposing a TWA of 10 ppm and a STEL of 20 ppm for p-tert-butyltoluene. The Agency preliminarily concludes that a STEL as well as a TWA will protect exposed workers against the risk of central nervous and cardiovascular system effects and of irritation and nausea potentially associated with short-term exposures to p-tert-butyltoluene. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for p-tert-butyltoluene if the Agency determines that this limit will substantially reduce significant risk.

CHLORODIFLUOROMETHANE
CAS: 75-45-6; Chemical Formula: $CHClF_2$
H.S. No. 1085

OSHA has no current limit for chlorodifluoromethane (Freon 22). The ACGIH recommends a TLV–TWA of 1000 ppm, with a short-term limit of 1250 ppm for 15 minutes. Chlorodifluoromethane is a colorless, nearly odorless, non-flammable gas.

Exposure to very high atmospheric levels of Freon 22 cause stimulation and then depression of the central nervous system, followed by asphyxiation. Rats and guinea pigs exposed to concentrations of 75,000 to 100,000 ppm over a 2-hour period exhibited excitation and disequilibrium; narcosis was observed at 200,000 ppm and mortality at 300,000 and 400,000 ppm (Weigand 1971). In mice, similar exposures at 320,000 ppm were the maximum tolerated, and the minimum lethal dose was 370,000 ppm (Karpov 1965). In rabbits, the minimum concentration altering reflex responses was 11,000 to 20,000 ppm (Karpov 1965). Studies of guinea pigs reported no fatalities as a result of exposure for 2 hours at 200,000 ppm, but mild clinical changes were observed at 50,000 ppm and minimal effects at 25,000 ppm (Underwriters' Laboratories, Inc. 1940). Thirty-minute exposures at 500,000 ppm were lethal to guinea pigs (Booth and Bixby 1932). Karpov has also reported the results of a 10-month study of inhalation effects in rats, guinea pigs, dogs, and cats. Six-hour inhalation exposures to 14,000 ppm and 2000 ppm for 5 days/week were studied, and alterations in weight, endurance, blood chemistry, and pathology of the lungs, central nervous system, heart, liver, kidney, and spleen were seen at the 14,000-ppm level in rats, mice, and rabbits. At the 2000-ppm daily inhalation level, rats and mice showed no effects. In dogs, cardiac sensitization was not observed at the 25,000-ppm level, but it did occur at the 50,000-ppm level (Reinhardt, Azar, and Maxfield 1971). No data have been published concerning the carcinogenicity, mutagenicity, or teratogenicity of this substance.

The Agency is proposing an 8-hour TWA limit of 1000 ppm for chlorodifluoromethane and a 1250-ppm 15-minute STEL. OSHA preliminarily concludes that these limits will provide protection against the life-threatening asphyxiant effects that could occur in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for chlorodifluoromethane. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

o-CHLOROTOLUENE
CAS: 95-49-8; Chemical Formula: C_7H_7Cl
H.S. No. 1090

OSHA has no current limit for o-chlorotoluene. The ACGIH recommends a TLV–TWA of 50 ppm and a 15-minute STEL of 75 ppm for this colorless liquid.

The oral LD_{50} in rats for o-chlorotoluene is greater than 1600 mg/kg. When the undiluted material was administered orally in doses ranging from 50 to 100 mg/kg, the animals experienced weakness and vasodilation at the higher dose levels, but all survived and were gaining weight 2 weeks later (ACGIH 1986 p. 137). When the undiluted liquid was applied to the skin of guinea pigs in doses of 1 cc or 10 cc/kg for 24 hours, moderately severe skin irritation occurred at both dose levels. The guinea pigs lost weight over the 2-week period following application, indicating percutaneous absorption of this substance. One drop of undiluted material in the eyes of rabbits produced

a delayed erythema of the conjunctiva, although this effect cleared after 14 days (Ely, as cited in ACGIH 1986, p. 137). Rats exposed to an atmosphere of 21 mg/L, or about 4000 ppm, for 6 hours exhibited loss of coordination within 1.5 hours, prostration at 1.75 hours, and tremors at 2 hours. At 14,000 ppm, rats showed loss of coordination, vasodilation, labored respiration, narcosis, and tearing. Rats exposed at 4000 and 14,000 ppm survived. At 175,000 ppm, one of three rats died (Ely, as cited in ACGIH 1986 p. 137). In another study, mice, rats, and guinea pigs were exposed to o-chlorotoluene at a concentration of about 4400 ppm. Mice showed gasping and convulsions within 30 minutes, and guinea pigs and rats exhibited gasping, hyperpnea, ataxia, and convulsions in 45 minutes. All animals were comatose within 60 minutes, and, except for two guinea pigs that still survived at 14 days, all of the animals died (Hazleton Laboratories, Inc. 1966).

In rabbits, the 24-hour patch test resulted in moderate skin irritation; albino rabbits displayed conjunctival irritation from a single instillation of 0.1 ml of undiluted o-chlorotoluene, but no corneal damage was observed 7 days later (Hazleton Laboratories, Inc. 1966).

Data concerning human exposures are lacking, but no cases of dermatitis or poisoning have been reported as a result of occupational exposure. Personal communications from several occupational health experts recommend limits ranging from 25 ppm to 200 ppm TWA for human exposures (Hopton; Mastromatteo; Elkins; Torkelson, as cited in ACGIH 1986, p. 137). These limits were recommended on the basis of analogy with similar compounds, such as the chlorinated benzenes.

OSHA is proposing 50 ppm as an 8-hour TWA PEL. The Agency preliminarily concludes that this limit will protect against the risk of eye and skin irritation and systemic poisoning possible in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for o-chlorotoluene. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

CYCLONITE
CAS: 121-82-4; Chemical Formula: $C_3H_6N_6O_6$
H.S. No. 1110

OSHA currently has no permissible exposure limit for cyclonite. The ACGIH recommends a TLV-TWA of 1.5 mg/m³ and a STEL of 3 mg/m³, with a skin notation. Cyclonite exists in the form of orthorhombic crystals.

Cyclonite, a high explosive and a rat poison, has not been shown in animal studies to be acutely toxic. In industry, reports of poisonings as a result of occupational exposures to cyclonite were widespread as late as 1962 (Kaplan, Berghout, and Peczenik 1962). Exposure causes central nervous system effects, including nausea, vomiting, convulsions, and unconsciousness. These clinical signs result from repeated gastrointestinal and respiratory exposures and from skin absorption (Sunderman et al. 1944; von Oettingen, Donahue, Yagoda et al. 1949). In an epidemiological study, Hathaway (1977) reported that 8-hour TWA exposures ranging up to 1.57 and averaging 0.28 mg/m³ caused no identifiable abnormalities attributable to cyclonite exposure.

OSHA is proposing a limit of 1.5 mg/m³ TWA, a 3-mg/m³ 15-minute STEL, and a skin notation for cyclonite. The Agency preliminarily concludes that establishing these limits for this previously unregulated chemical will protect workers from the risk of neuropathic effects associated with cyclonite exposure, either as a result of inhalation or percutaneous exposures, in the absence of an OSHA PEL. The health evidence forms a reasonable basis for proposing a new limit for cyclonite. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

2,6-DI-tert-BUTYL-p-CRESOL
CAS: 128-37-0; Chemical Formula: $C_{15}H_{24}O$
H.S. No. 1117

OSHA currently has no limit for 2,6-di-tert-butyl-p-cresol (DBPD). The ACGIH recommends a TLV-TWA of 10 mg/m³ for this white crystalline compound, which is prepared from p-cresol and isobutylene. DBPD is widely used as a food preservative.

DBPD is considered to have a low order of toxicity; in extensive animal studies, ingestion has not been associated with toxic effects (ACGIH 1986, p. 227). Deichmann and associates (1955) reported oral LD_{50} values of 10.7 g/kg for guinea pigs, 1.7 and 1.97 g/kg for male and female rats, respectively, and ranges of between 0.94 and 2.1 g/kg for cats and between 2.1 and 3.2 g/kg for rabbits. One year of daily oral administration of 0.17 to 0.9 g/kg in dogs produced no effects, nor did a 24-month oral administration of 0.2, 0.5, or 0.8 percent DBPD in rats (Deichmann et al. 1955). Other studies have confirmed these overall results, although some growth rate decreases and liver weight increases were demonstrated in rats fed 0.01 to 0.5 percent DBPD, total daily diet (Brown, Johnson, and O'Halloran 1959; Creaven, Davies, and Williams 1966).

The estimated human intake of DBPD in the United States does not exceed a few milligrams daily (Gilbert and Goldberg 1965), perhaps 0.2 mg/kg body weight. These authors further observe that the no-effect dietary level in rats is 25 mg/kg.

OSHA proposes a TWA limit of 10 mg/m³ for 2,6-di-tert-butyl-p-cresol. The Agency preliminarily concludes that this limit will protect workers against the risk of any acute or chronic effects potentially associated with occupational exposure to this substance in the absence of any OSHA PEL. The health evidence forms a reasonable basis for proposing a new limit for 2,6-di-tert-butyl-p-cresol. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

DIETHANOLAMINE
CAS: 111-42-2; Chemical Formula: $HO(CH_2)_2NH(CH_2)_2OH$
H.S. No. 1134

OSHA currently has no limit for diethanolamine. The ACGIH has established an 8-hour TWA limit of 3 ppm. Diethanolamine exists as either a solid or a liquid at room temperature.

Diethanolamine has a low order of toxicity. The oral LD_{50} for both rats and guinea pigs has been reported to be about 2 g/kg (Dow Chemical Company, as cited in ACGIH 1986, p. 197). Acute toxicity studies have shown that direct contact may impair vision and denature the skin if exposure is repeated. Dietary studies in rats showed no ill effects after 90 days of feeding at 20 mg/kg/day (Smyth et al. 1951).

OSHA is proposing a PEL of 3 ppm TWA for diethanolamine. The Agency preliminarily concludes that this limit will protect exposed workers from the risk of eye damage and skin irritation potentially associated with exposure to diethanolamine at the levels permitted by the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for diethanolamine. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

DIETHYL PHTHALATE
CAS: 84-66-2; Chemical Formula: C_6H_4-$(COOC_2H_5)_2$
H.S. No. 1136

OSHA currently has no limit for diethyl phthalate. The ACGIH recommends a TLV-TWA of 5 mg/m³ for this stable, colorless, odorless, and oily liquid with a bitter taste.

Diethyl phthalate exposure may cause polyneuritis and disturbance of the vestibular function. By most routes of administration, this substance has low acute toxicity in laboratory animals and borders on the "relatively harmless" classification according to the systematic grouping developed by Hine and Jacobson (1954). Oral LD_{50} values in the rat range between 9.5 and 31 g/kg (Shibko and Blumenthal 1973); the intraperitoneal LD_{50} for the rat is 5.08 ml/kg (Singh, Lawrence, and Autrian 1972) and, for the mouse, 2.8 g/kg (Calley, Autrian, and Guess 1966). Chronic feeding studies lasting 6 or more weeks resulted in no-effect levels of 2.5 g/kg/day for the rat and 1.25 g/kg/day for the dog, with no specific lesion attributable to diethyl phthalate and no unusual incidence of tumors (Shibko and Blumenthal 1973).

A study of workers exposed to a mixture of diethyl phthalate, dibutyl phthalate, and di-2-ethyl hexyl phthalate vapors in air at concentrations of 8 to 53 mg/m³ resulted in findings of no phthalates in the blood (before or after the exposure) and no peripheral polyneuritis (Raleigh, as cited in ACGIH 1986, p. 200). Fassett (1963) reported transient nasal and throat irritation produced by exposure to the heated vapors of diethyl phthalate, but no cumulative effects have been noted. A Russian study of workers (employed for between 0.5 and 19 years) who were exposed to several phthalate plasticizers (e.g., butyl phthalate, the higher aryl phthalates, dioctyl phthalate, and benzyl butyl phthalate), as well as to sebacates, adipates, and tri-o-cresyl phosphate, at concentrations ranging from 1.7 to 66 mg/m³ resulted in subjective complaints of pain, numbness, and spasms in the upper and lower extremities. These complaints were related to the duration of exposure and usually began after the 6th or 7th year of employment (Milkov et al. 1969). These investigators reported polyneuritis in 32 percent of the 47 persons examined for this health effect; of 81 persons evaluated for disturbance of the vestibular function, 78 percent showed depression of vestibular receptors (Milkov et al. 1969).

OSHA proposes a PEL of 5 mg/m³ for diethyl phthalate. The Agency preliminarily concludes that this limit will protect workers against the risk of polyneuritis and vestibular function disturbance potentially associated with occupational exposure to this substance in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for diethyl phthalate. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

DINITOLMIDE (3,5-DINITRO-O-TOLUAMIDE)
CAS: 148-01-6; Chemical Formula: $C_8H_7N_3O_5$
H.S. No. 1144

OSHA currently has no limit for dinitolmide. The ACGIH recommends a limit of 5 mg/m³ TWA for this yellowish solid.

In rats, the oral LD_{50} for males is 560 mg/kg, and for females, 650 mg/kg; the ACGIH concludes that it has a moderate oral toxicity in rats (1986, p. 213). Two-year dietary studies of rats fed 62.5 ppm (or 3 mg/kg/day) dinitolmide showed no ill effects. Rats of both sexes fed 6 mg/kg/day showed slight fatty changes in the liver; female rats also exhibited slight liver weight increases. Dogs fed 10 mg/kg/day showed no effects after 1 year. A 3-generational study of rats fed 6 or 3 mg/kg/day revealed no effects on fertility, gestation, viability, or lactation (Dow Chemical Company, as cited in ACGIH 1986, p. 213). There are no inhalation data.

OSHA is proposing an 8-hour TWA PEL for dinitolmide of 5 mg/m³. The Agency preliminarily concludes that this limit will protect exposed workers from the risk of hepatic changes possible as a result of exposure at the levels permitted in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for dinitolmide. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

DIPHENYLAMINE
CAS: 122-39-4; Chemical Formula: $(C_6H_5)_2NH$
H.S. No. 1147

OSHA currently has no limit for diphenylamine. The ACGIH recommends a TLV of 10 mg/m³ TWA. Diphenylamine exists as monoclinic crystalline leaflets that discolor when exposed to light.

Acute oral toxicity data for diphenylamine are limited. A single report describes a study in which a dietary dose of 1,500 mg/kg killed two of twenty rats within 30 days of ingestion (Griswold et al. 1966). This suggests that diphenylamine is significantly less toxic than aniline (Hamblin 1963). Dietary studies of rats fed 0.025, 0.1, 0.5, 1.0, or 1.5 percent diphenylamine for 226 days demonstrated non-malignant renal cysts at the three highest doses (Thomas et al. 1957). However, rats given diphenylamine crystals encapsulated in collodion developed bladder papillomas within 125 days (Yoshida et al. 1941). Exposure to diphenylamine dust has been linked to liver, spleen, and kidney changes in experimental animals (Robert et al. 1937).

A report of industrial diphenylamine poisoning in France described bladder symptoms, tachycardia, hypertension, and eczema (Fairhall 1957).

OSHA is proposing a PEL of 10 mg/m³ TWA for diphenylamine. The Agency preliminarily concludes that this limit will protect workers against the risk of liver, kidney, cardiovascular, and other systemic effects potentially associated with exposures to this substance in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for diphenylamine. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

DIURON
CAS: 330-54-1; Chemical Formula: $C_9H_{10}Cl_2N_2O$
H.S. No. 1153

OSHA currently has no limit for diuron. The ACGIH recommends a TLV of 10 mg/m³ TWA for this white crystalline solid.

Hodge and associates (1967, 1968) have reported a low order of acute and chronic toxicity for diuron. For male rats, the oral LD_{50} is 3,400 mg/kg. In 2-year feeding studies of rats and dogs, the no-effect levels were reported to be 250 and 125 ppm, respectively. A concentration of 125 ppm in the diet did not cause reproductive or carcinogenic effects in a 3-generational study of rats (Hodge, Downs, and Panner 1967; Hodge, Downs, Smith et al. 1968); 1,400 ppm did not have carcinogenic effects in mice (Innes et al. 1969). Skin irritation and sensitization test findings in guinea pigs have been negative (ACGIH 1986, p. 228).

OSHA is proposing an 8-hour limit of 10 mg/m³ TWA for diuron. This is the limit being proposed by the Agency for all inert dust and particulates; OSHA preliminarily concludes that this limit will protect exposed workers from the risks potentially associated with workplace exposure in the absence of any OSHA PEL. These risks include accidents, skin and upper respiratory tract irritation, and interference with vision. The health evidence forms a reasonable basis for proposing a new limit for diuron. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

METHYL ACETATE
CAS: 79-20-9; Chemical Formula: CH_3COOCH_3
H.S. No. 1249

OSHA currently has a limit of 200 ppm TWA for methyl acetate. The ACGIH also recommends an 8-hour TWA limit of 200 ppm, with the addition of a TLV–STEL of 250 ppm. Methyl acetate is a highly volatile, colorless liquid with a pleasant odor.

Methyl acetate is mildly narcotic and is a known irritant to the mucous membranes of the eyes and respiratory passages. Occupational exposure to this substance by vapor inhalation at unreported levels resulted in inflammation of the eyes, nervous irritation, and tightness in the chest (Duquenois and Revel 1934; Fairhall 1957). Duquenois and Revel (1934) suggested that, like methyl alcohol, methyl acetate may produce atrophy of the optic nerve.

Other researchers have suggested that the methanol formed by hydrolysis in the body may be responsible for the toxicity of methyl acetate and, on this basis, have recommended a limit of 250 ppm in the occupational setting (Henderson and Haggard 1943). However, Lehmann and Flury (1943) have attributed toxic effects (e.g., blood changes, weight loss, lung irritation), as well as some deaths, to chronic exposures at 6600 ppm. These health effects require a PEL which will protect workers against both chronic and acute exposures.

No cases of irritation or systemic injury have been reported from industrial exposures to methyl acetate below 200 ppm.

OSHA is proposing an 8-hour PEL of 200 ppm TWA and a 15-minute STEL of 250 ppm for methyl acetate. The Agency preliminarily concludes that both of these limits will protect exposed workers from the risk of narcosis, eye and skin irritation, and pulmonary irritation possible at elevated exposure levels. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for methyl acetate if the Agency determines that this limit will substantially reduce significant risk.

METRIBUZIN
CAS: 21087–64–9; Chemical Formula: $C_8H_{14}N_4OS$
H.S. No. 1275

OSHA currently has no limit for metribuzin. The ACGIH recommends a TLV–TWA of 5 mg/m.[3] Metribuzin is a crystalline solid.

The herbicide metribuzin has a low order of acute toxicity; single exposures to high concentrations produce central nervous system depression, and repeated high doses affect the thyroid and liver function (Deutsche Forschungsgemeinshaft 1981, as cited in ACGIH 1986, p. 411). The oral LD_{50} in rats has been reported to be 2000 mg/kg; in cats and rabbits, the LD_{50} is up to 500 mg/kg. A 4-hour aerosol exposure at concentrations of between 860 and 892 mg/m[3] was tolerated by rats and mice; no skin or eye irritation was observed in rabbits. No sensitizing effects were seen in guinea pigs, and a skin application of the 70-percent wettable powder of 1000 mg/kg per day for 3 weeks produced no effects in rats (Deutsche Forschungsgemeinschaft 1981 as cited in ACGIH 1986, p. 411).

Inhalation studies have shown no adverse effects in rats exposed to 31 mg/m[3] of the aerosol for 6 hours/day, 5 days/week during a 3-week period (Bayer 1981, as cited in ACGIH 1986, p. 411). No carcinogenic effects were observed in rats and mice fed 20, 800, or 3200 ppm for 2 years (Kimmerle 1982, as cited in ACGIH 1986, p. 411). A no-effect level of 100 ppm was observed in a 2-year dietary study of rats and dogs (Deutsche Forschungsgemeinschaft 1981, as cited in ACGIH 1986, p. 411); these same investigators observed no teratogenic, embryotoxic, or reproductive effects in rats or rabbits. In Chinese hamsters and mice, no mutagenic activity was observed (Siebert and Lemperle 1974).

No human poisonings with metribuzin have been reported. In oral long-term studies, the highest no-observed effect levels (NOELs) were 2.5 to 5 mg/kg per day (ACGIH 1986, p. 411). Single and repeated patch tests in humans resulted in neither irritation nor sensitization (Deutsche Forschungsgemeinschaft 1981, as cited in ACGIH 1986, p. 411).

OSHA is proposing a PEL of 5 mg/m[3] TWA for metribuzin. The Agency preliminarily concludes that this limit will protect workers against the risks of metabolic and CNS effects that potentially exist from workplace exposure to metribuzin at the levels permitted by the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for metribuzin. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

OIL MIST (MINERAL)
CAS: 8012–95–1; Chemical Formula: None
H.S. No. 1297

OSHA currently has a limit of 5 mg/m[3] TWA for oil mist. The ACGIH has the same limit and recommends the addition of a 10 mg/m[3] TLV–STEL. Oil mist (mineral) refers to the airborne mist of petroleum-based cutting oils or of white petroleum oil; its odor is described as that of burned lubrication oil.

Studies in animals have shown no ill effects as a result of repeated 6-hour daily exposures to 5 mg/m[3] level (Wagner, Wright, and Stokinger 1964). At 100 mg/m[3], slight changes (not further specified) were observed in exposed animals (Lushbauch, Green, and Redemann 1950). Oil fumes and the role of additives have not yet been evaluated, but it has been suggested that heat-decomposed oil fumes are irritating to the lungs (Wagner, Dobrogorski, and Stokinger 1961). Some lung effects have been reported as a result of exposures in animals at 100 mg/m[3] Lushbauch, Green, and Redemann 1950).

OSHA proposes a PEL of 5 mg/m[3] TWA and a STEL of 10 mg/m[3] for mineral oil mist. The Agency preliminarily concludes that both a TWA limit and a STEL are necessary to protect against the risk of lung irritation potentially associated with elevated short-term exposures to oil mist. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for mineral oil mist if the Agency determines that this limit will substantially reduce significant risk.

PETROLEUM DISTILLATES (NAPHTHA)
CAS: None; Chemical Formula: None
H.S. No. 1312

For petroleum distillates (naphtha), also identified as rubber solvents, OSHA is proposing to reduce its current 8-hour limit of 500 ppm to 400 ppm. The ACGIH has a TLV–TWA of 400 ppm, and NIOSH recommends a TWA of 87 ppm and a ceiling (15 min) of 450 ppm for these substances.

A 1975 study performed by Carpenter et al. exposed rats to between 2800 and 24,200 ppm of naphtha. Motor incoordination occurred at 5300 ppm, and convulsions and death occurred in all animals at 24,200 ppm. Animals exposed to 480 ppm for 63 days showed no signs of toxicity (Carpenter et al. 1975).

NIOSH (1977i) noted that rubber solvent (naphtha) is composed primarily of C_6–C_8 alkanes and, as such, the limit of 350 mg/m[3] (85 ppm) recommended for C_6–C_8 alkanes should apply to naphtha. This recommendation presumes that all C_6–C_8 alkanes possess neuropathic capability; as discussed above in the section on narcotic agents, OSHA has preliminarily concluded that not all C_6–C_8 alkanes are neuropathic agents.

In establishing its recommended 400-ppm TLV–TWA for petroleum distillates, the ACGIH relied on the

observations that slight irritation occurs in humans at exposure to 430 ppm and that no signs of toxicity occur in animals exposed to 480 ppm. The NIOSH-recommended 85-ppm limit is based on the assumption that all C_6–C_8 alkanes possess equivalent neuropathic properties. OSHA has tentatively rejected this hypothesis and proposes therefore to reduce the PEL to 400 ppm TWA in order to avoid the risk of extensive irritation. The Agency requests comments on the issue of equivalent toxicity for all C_6–C_8 alkanes. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for petroleum distillates if the Agency determines that this limit will substantially reduce significant risk.

m-PHTHALODINITRILE
CAS: 626–17–5; Chemical Formula: $C_8H_4N_2$
H.S. No. 1327

OSHA currently has no limit for m-phthalodinitrile. The ACGIH has recommended a TLV–TWA of 5 mg/m³. meta-Phthalodinitrile exists in the form of needles obtained from solutions containing either water or ligroin as the solvent.

In rabbits, slight skin reactions have been reported from dermal applications of m phthalodinitrile to intact or abraded skin for 6 hours/day, 5 days/week during a 3-week period. The doses applied were 0.5, 1.0, and 2.0 g/kg; at the two higher dose levels, some changes in organ size, without histopathologic changes, were observed. Female rabbits exposed at the highest dose lost body weight (Owen 1972, as cited in ACGIH 1986, p. 488).

A 15-year review of industrial experience revealed no reports of adverse effects from exposure to m-phthalodinitrile (Zeller, Hofmann, Thiess, and Hey 1963). Williams (1959) attributes this absence of exposure effects to the fact that the aromatic nitriles, of which m-pthalodinitrile is one, do not liberate cyanide in the body, as is the case with the aliphatic nitriles.

OSHA is proposing an 8-hour TWA limit for m-phthalodinitrile of 5 mg/m³. The Agency preliminarily concludes that this limit will protect exposed workers from the risk of skin irritation that exists in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for m-phthalodinitrile. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

PLATINUM (METAL)
CAS: 7440–06–4; Chemical Formula: Pt
H.S. No. 1332

OSHA currently has no limit for platinum metal. The ACGIH recommends a limit of 1.0 mg/m³ TWA for platinum metal dust. Platinum is a silver gray, lustrous, malleable, and ductile precious metal.

Extrapolating from its TLV for platinum soluble salts and recognizing no major health effects associated with exposure to the metal dust, the ACGIH recommended a TLV of 1.0 mg/m³ for platinum metal dust. This level apparently was based on good industrial hygiene practices and the expectation that a heavy metal dust will be more toxic than nuisance dusts (which are controlled to 10 mg/m³.

OSHA therefore proposes a limit of 1.0 mg/m³ TWA for platinum metal dust. The Agency preliminarily concludes that this limit will protect workers against the risk of adverse health effects potentially associated with workplace exposure in the absence of any OSHA PEL. The health evidence forms a reasonable basis for proposing a new limit for platinum metal dust. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

RESORCINOL
CAS: 108–46–3; Chemical Formula: $C_6H_4(OH)_2$
H.S. No. 1346

OSHA currently has no limit for resorcinol. The ACGIH recommends an 8-hour TWA limit of 10 ppm with a TLV–STEL of 20 ppm. Resorcinol occurs in the form of sweet-tasting white crystals that may turn pink on exposure to air and light or on contact with iron.

Resorcinol has been reported to be less toxic than either catechol or phenol by ingestion or skin penetration (von Oettingen 1949; Koppers Company 1974, as cited in ACGIH 1986, p. 511). The oral LD_{50} in rats is 301 mg/kg (NIOSH 1977). Daily 6-hour exposures at 8 ppm for 2 weeks produced no ill effects in rats, guinea pigs, and rabbits. Acute inhalation exposures to a resorcinol-water aerosol at concentrations as high as 7800 mg/m³ for 1 hour and 2800 mg/m³ for 8 hours caused no toxic effects in laboratory animals (Koppers Company 1974, as cited in ACGIH 1986, p. 511).

In humans, regular exposure to 10 ppm also caused no irritation and no complaints of discomfort (Koppers Company 1974, as cited in ACGIH 1986, p. 511).

OSHA proposes a PEL of 10 ppm TWA and a STEL of 20 ppm for resorcinol. The Agency preliminarily concludes that this combined limit will protect workers against the risk of irritation that exists in the absence of any OSHA limit for occupational exposure to this substance. The health evidence forms a reasonable basis for proposing a new limit for resorcinol. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

TANTALUM (METAL DUST AND OXIDE)
CAS: 7440–25–7; Chemical Formulas: (Tantalum metal)Ta; (Tantalum oxide)Ta_2O_5
H.S. No. 1382

OSHA's existing PEL for tantalum is 5 mg/m³; the ACGIH recommends a 5-mg/m³ and TWA and a 15-minute STEL of 10 mg/m³. Tantalum dust is a black powder and tantalum oxide is a white, microcrystalline powder.

Animal studies by Miller, Davis, Goldman, and Wyatt (1953) have not implicated tantalum as a cause of pnuemoconiosis, although an exposure to 100 mg tantalum oxide produced "soft white circumscribed pigmented dust lesions" (ACGIH 1986, p. 554) in the lungs of these animals. Additionally, this particular study demonstrated transient bronchitis, interstitial pneumonitis, and hyperemia at the 100-mg exposure level. Tantalum oxide has been used as a dressing for burns (Olsen 1944), and the use of tantalum gauze in surgical repair showed no long-term adverse effects (Dales and Kyle 1958). No adverse health effects have been associated with industrial exposures to tantalum or its compounds (Cochran, Doull, Mazur, and DuBois 1950). A single oral dose of 6500 mg/kg oxide was virtually nontoxic to rats (ACGIH 1986, p. 554).

The ACGIH (1986, p. 554) believes that tantalum dust and oxide should be classified as an inert or nuisance dust and, accordingly, placed tantalum on its 1987–88 Notice of Intended Changes list with a recommendation for increasing the TLV–TWA to 10 mg/m³, the ACGIH's limit for all nuisance dusts, and deleting the current STEL of 10 mg/m³. However, OSHA notes that inhalation exposures to tantalum have produced pulmonary lesions, bronchitis, interstitial pneumonitis, and hyperemia. The Agency preliminarily concludes that the existing 5-mg/m³ TWA for these compounds should be supplemented with a short-term limit of 10 mg/m³ to protect exposed workers from these respiratory effects of exposure. OSHA therefore proposes a PEL of 5 mg/m³ TWA and a STEL of 10 mg/m³ for tantalum (metal dust and oxide). The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for tantalum dust and oxide if the Agency determines

that this limit will substantially reduce significant risk.

TRIMETHYL PHOSPHITE
CAS: 121–45–9; Chemical Formula: $(CH_3O)_3P$
H.S. No. 1410

OSHA currently has no limit for trimethyl phosphite. The ACGIH limit is a 2-ppm 8-hour TWA. Trimethyl phosphite is a colorless liquid with a pungent odor.

Trimethyl phosphite's toxic effects include lung, skin, and eye irritation. In a chronic inhalation study of rats, Levin and Gabriel (1973) found that exposure to trimethyl phosphite at concentrations of 500±75 ppm for 7.5 hours daily, 5 days/week for 8 weeks caused an adverse effect on body weight and, at necropsy, revealed evidence of severe pulmonary and cutaneous pathology. At exposures of 600 ppm 6 hours/day, 5 days/week for 4 weeks, 70 percent of the rats died, and 10 percent of those exposed even at 300 ppm on the same regimen died (Mobil Oil Corporation 1979).

Rats exposed at 100 ppm showed signs of eye irritation, and at 300 to 600 ppm, mild to severe cataracts developed. At doses of 164 mg/kg, trimethyl phosphite caused gross abnormalities in the offspring of treated rats (Mobil Oil Corporation 1979).

Skin contact with trimethyl phosphite produced severe skin irritation in rabbits, and instillation in the eyes of rabbits caused temporary swelling and irritation but no permanent effects (Fassett 1963).

In a group of 179 workers exposed to average concentrations of trimethyl phosphite of between 0.3 and 4 ppm, no ocular changes were observed (Mobil Chemical Corporation 1980, as cited in ACGIH 1986, p. 609).

OSHA is proposing an 8-hour TWA for trimethyl phosphite of 2 ppm. The Agency preliminarily concludes that this limit will protect exposed workers against the risk of eye damage, skin irritation, and upper respiratory tract irritation potentially associated with exposures to this substance at the levels permitted in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for trimethyl phosphite. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

TRIPHENYL AMINE
CAS: 603–34–9; Chemical Formula: $(C_6H_5)_3N$
H.S. No. 1415

OSHA currently has no exposure limit for triphenyl amine. The ACGIH recommends a 5-mg/m³ 8-hour TWA limit for this substance. Triphenyl amine exists as colorless monoclinic prisms.

Animal studies conducted by the Eastman Kodak Company (as cited in ACGIH 1986, p. 612) showed an oral LD_{50} in rats of 3200 to 6400 mg/kg and an oral LD_{50} in mice of 1600 to 3200 mg/kg. The LD_{50} by intraperitoneal administration for both rodent species exceeded 6400 mg/kg. Skin and eye sensitivity tests in both rabbits and guinea pigs were essentially negative except that application of 5 to 20 ml/kg occlusively for 4 hours produced slight erythema (Eastman Kodak Company, as cited in ACGIH 1986, p. 612).

OSHA is proposing a 5-mg/m³ TWA limit for triphenyl amine. The Agency preliminarily concludes that this limit will protect exposed workers against the risk of skin irritation potentially associated with exposure to this substance in the absence of any OSHA PEL. The health evidence forms a reasonable basis for proposing a new limit for triphenyl amine. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

URANIUM (INSOLUBLE COMPOUNDS)
CAS: 7440–61–1; Chemical Formula: U
H.S. No. 1418

OSHA's existing PEL for insoluble uranium compounds is 0.25 mg/m3; the ACGIH has a TLV–TWA of 0.2 mg/m³ and a 0.6-mg/m³ STEL. Uranium is a silver-white radioactive metal.

OSHA's existing limit for the insoluble compounds of uranium was based on several early studies of uranium's toxic effects in animals; these effects included kidney damage and blood changes (Voegtlin and Hodge 1953). In the intervening years, a considerable body of evidence has accumulated based on the actual occupational exposures of uranium plant workers over a period as long as 25 years. This evidence shows that, before 1950, workers were often exposed to uranium levels between 0.2 and 1.5 mg/m³, but that after 1950, only about 6 percent were exposed at 0.05 mg/m³ or above; despite these relatively high early exposures, the incidence of all diseases, whether or not linked to radiation exposure, has been no higher than is the case for workers in the general population (ACGIH 1986, p. 617). However, there is also evidence that several workers were exposed to brief excursions during which exposure levels reached a concentration as much as five times the TLV (Wing, Heatherton, and Quigley 1963, as cited in ACGIH 1986, p. 617).

OSHA is proposing an 8-hour TWA of 0.2 mg/m³ and a STEL of 0.6 mg/m³ for the insoluble forms of uranium. The Agency preliminarily concludes that these limits are required to protect uranium plant workers from the risk of kidney or blood disorders potentially associated with both full-shift and excursion exposures to these compounds. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for uranium if the Agency determines that this limit will substantially reduce significant risk.

Preliminary Conclusions

For the group of substances shown in Table C9–1, OSHA preliminarily concludes that workplace exposures to these substances cause a broad range of adverse health consequences in exposed individuals; effects include CNS depression, respiratory irritation, liver and kidney damage, cardiac sensitization, and hepatocellular cancer. OSHA believes that the health evidence forms a reasonable basis for proposing new or revised limits for these substances. At the time of the final rule, the Agency will establish new or revised limits if it determines that significant risk will be substantially reduced thereby.

10. Substances for Which Proposed Limits Are Based on Avoidance of Adverse Nuisance Effects

Introduction

OSHA is proposing limits for a group of substances that cause a variety of adverse effects. These substances are recognized universally as posing a substantial threat to the health, safety, and well-being of workers exposed to them. The term "nuisance" for these particulates can be misleading because the hazards they pose in the workplace are real and often serious. The ACGIH classifies many of these substances as nuisance particulates and applies a single workplace limit to them. OSHA has no substance-specific limits for these dusts; the Agency's limit for the category of nuisance dusts as a whole is 15 mg/m³ as total dust and 5 mg/m³ as respirable dust (see Table Z–3 of 29 CFR 1910.1000). Table C10–1 shows the 47 substances included in this group. NIOSH does not recommend a limit for the category of nuisance dusts but does have a REL for malathion and fibrous glass dust, two substances included in this group by the ACGIH. For all of these substances except fibrous glass dust, OSHA is proposing to establish an

8-hour TWA limit of 10 mg/m³ as total dust. For fibrous glass dust, the Agency is proposing a limit of 5 mg/m³ as a TWA.

Description of the Health Effects

The adverse effects caused by exposure to these substances include: Interference with vision; deposition of these substances in the eyes, ears, nasal passages, and upper respiratory tract; and skin irritation. Thus, workers exposed to excessive concentrations of these substances may have difficulty seeing or be subjected to attacks of uncontrolled coughing and sneezing. Moreover, workers may injure their skin and mucous membranes when they attempt to remove these substances, which settle everywhere. These undesirable exposure effects also have serious safety implications, because they lead to workplace accidents and injuries.

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TABLE C10-1. - Substances Causing Adverse Nuisance Effects

H.S. Number/ Chemical Name	CAS No.	OSHA Nuisance Dust Limit	ACGIH TLV	NIOSH REL
1014 Alpha-alumina	1344-28-1	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1016 Aluminum metal dust	7429-90-5	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1024 Ammonium sulfamate	7773-06-0	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1031 Barium sulfate	7727-43-7	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1032 Benomyl	17804-35-2	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1035 Bismuth telluride (undoped)	1304-82-1	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1039 Boron oxide	1303-86-2	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1057 Calcium carbonate	1317-65-3	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1061 Calcium silicate, total dust	--	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1062 Calcium sulfate	7778-18-9	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1076 Cellulose	9004-34-6	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1082 2-Chloro-6-trichloro-methyl pyridine	1929-82-4	15 mg/m^3 TWA	10 mg/m^3 TWA	--

TABLE C10-1. - Substances Causing Adverse Nuisance Effects (continued)

H.S. Number/ Chemical Name	CAS No.	OSHA Nuisance Dust Limit	ACGIH TLV	NIOSH REL
1095 Clopidol	2971-90-6	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1102 Crag herbicide (sesone)	136-78-7	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1133 Dicyclopentadienyl iron	102-54-5	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1155 Emery	112-62-9	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1176 Ferbam	14484-64-1	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1178 Fibrous glass dust+	--	--	10 mg/m^3 TWA	5 mg/m^3 TWA*
1188 Glycerin (mist)	56-81-5	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1191A Graphite, synthetic, total dust	--	15 mg/m^3 TWA	10 mg/m^3 TWA	-
1192 Gypsum, total dust	-	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1230 Kaolin, total dust	-	15 mg/m^3 TWA	10 mg/m^3 TWA	--

* NIOSH has an additional limit for fibrous glass of 3 million fibers/m^3; fibers less than or equal to 3.5 microns in diameter and equal to or greater than 10 microns in length are counted.

TABLE C10 1. Substances Causing Adverse Nuisance Effects (continued)

H.S. Number/ Chemical Name	CAS No.	OSHA Nuisance Dust Limit	ACGIH TLV	NIOSH REL
1232 Limestone, total dust	1317-65-3	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1233 Magnesite, total dust	--	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1234 Magnesium oxide fume	1309-48-4	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1235 Malathion	121-75-5	15 mg/m^3 TWA, Skin	10 mg/m^3 TWA, Skin	15 mg/m^3 TWA
1239 Marble, total dust	1713-65-3	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1246 Methoxychlor	72-43-5	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1277 Mineral wool fiber	-	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1278 Molybdenum (insoluble compounds)	7439-98-7	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1294 Nuisance particulates, total dust	-	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1305 Pentaerythritol, total dust	115-77-5	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1310 Perlite	-	15 mg/m^3 TWA	10 mg/m^3 TWA	-

TABLE C10-1. - Substances Causing Adverse Nuisance Effects (continued)

H.S. Number/ Chemical Name	CAS No.	OSHA Nuisance Dust Limit	ACGIH TLV	NIOSH REL
1328 Picloram	1918-02-1	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1331 Plaster of Paris, total dust	--	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1333 Portland cement	65997-15-1	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1351 Rouge, total dust	--	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1359 Silicon	7440-21-3	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1360 Silicon carbide	409-21-2	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1369 Starch, total dust	--	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1374 Sucrose, total dust	--	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1383 Temephos	3383-96-8	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1391 4,4'-Thiobis (6-tert-butyl-m-cresol)	96-69-5	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1396 Titanium dioxide	13463-67-7	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1423 Vegetable oil mist	--	15 mg/m^3 TWA	10 mg/m^3 TWA	--
1434 Zinc stearate	557-05-1	15 mg/m^3 TWA	10 mg/m^3 TWA	--

TABLE C10-1. - Substances Causing Adverse Nuisance Effects (continued)

H.S. Number/ Chemical Name	CAS No.	OSHA Nuisance Dust Limit	ACGIH TLV	NIOSH REL
1438 Zinc oxide, total dust	1315-13-2	15 mg/m^3 TWA	10 mg/m^3 TWA	--

* OSHA's TWA limits are for 8-hour exposures; its STELs are for the durations specified; and its ceilings are peaks not to be exceeded for any period of time.

** The ACGIH TWA-TLV is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

*** NIOSH TWA limits are for 10-hour exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

+ Proposed PEL is the NIOSH REL.

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When exposures to the substances shown in Table C10–1 are kept under proper control in the workplace, exposures are not likely to result in significant organic disease or irreversible toxic effects. However, it is inappropriate to consider these nuisance particulates biologically inert, because, if inhaled in sufficient amount, these dusts do cause pulmonary responses.

The lung-tissue reactions associated with exposure to these nuisance particulates leave the structure of the air spaces intact and do not cause scar tissue formation to any significant extent (ACGIH 1986). In addition, the reactions caused by inhalation are reversible if exposure is stopped. Controlling occupational exposures to these nuisance particulates and the other substances included in this category to levels below 10 mg/m³ as an 8-hour TWA will prevent these adverse effects. Workers protected by the proposed 10 mg/m³ limit will therefore not be at risk of the seriously distracting and often painful effects associated with exposures above this NOE level.

The following discussions describe OSHA's preliminary findings for the particulates included in this group. In addition, the health effects potentially associated with exposures to these substances are reviewed.

ALPHA-ALUMINA
CAS: 1344–28–1; Chemical Formula: Al_2O_3
H.S. No. 1014

OSHA presently has no specific limit for alpha-alumina, although OSHA's general nuisance dust limit of 15 mg/m³ applies. The ACGIH recommends an 8-hour TWA of 10 mg/m³, measured as total dust. Alpha-alumina, also called aluminum oxide, is a white powder that is widely used as an abrasive grinding material.

A study by Miller and Sayers (1941) determined that alumina particles with diameters less than 40 microns produced no reaction in laboratory animals. The results of a study by Stacy, King, Harrison et al. (1959) confirmed the findings of Miller and Sayers; these authors found a-alumina to be nearly inert when injected in the lungs of rats (Stacy, King, Harrison et al. 1959). Inhalation of fine aluminum powders at unspecified levels did not cause fibrosis in rats, guinea pigs, or hamsters (Gross et al. 1973).

In 1923, shortly after a-alumina replaced sandstone as the industrial abrasive of choice. Macklin and Middleton (1923) reported that workers exposed to aluminum oxide dust using the new, synthetic abrasive had much less pulmonary disease than workers using sandstone abrasives. Other studies (Sutherland, Meiklejohn, and Price 1937; Meiklejohn and Posner 1947; Meiklejohn and Posner 1948) reported that workers exposed to aluminum oxide dust in the chinaware industry and in aluminum production showed no evidence of pneumoconiosis. However, some early studies (Clark and Simmons 1925; Clark 1929) reported that workers engaged in aluminum oxide production and exposed to dust levels generally between 50 and 100 mppcf showed X-ray evidence of pulmonary fibrosis; these workers are likely also to have been exposed to silica. Workers exposed during World War II to bauxite fumes containing both alumina and silica developed pulmonary fibrosis and emphysema; the authors believe that silica fume was involved in the development of these diseases (Shaver and Riddell 1947). The ACGIH (1986, p. 21) states that alpha-alumina acts as an inert material.

OSHA is proposing an 8-hour limit of 10 mg/m³ for alpha-alumina, the limit being proposed for all inert particulates. The Agency preliminarily concludes that this limit will protect workers from the safety and health risks potentially associated with exposures to inert particulates in the workplace. These risks include safety accidents, skin and eye irritation, interference with vision, and distraction from the task at hand. This health evidence forms a reasonable basis for proposing a new limit for alpha-alumina. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

ALUMINUM METAL DUST
CAS: 7429–90–5; Chemical Formula: Al
H.S. No. 1016

OSHA currently has no specific permissible exposure limit for aluminum metal dust; however, OSHA's current nuisance dust limit of 15 mg/m³ TWA applies. The ACGIH recommends an 8-hour TWA limit of 10 mg/m³ as total dust.

Aluminum metal dust has been shown to present a minimal health hazard, according to results from the McIntyre Foundation's 27-year study of aluminum oxide dust. No deleterious lung or systemic effects were observed as a result of exposure to aluminum metal dust having a particle size of 1.2 um at calculated concentrations equivalent to 2 mg/m³ over an 8-hour workshift. Even much higher concentrations (not further specified) over 10- or 20-minute periods produced no adverse effects (ACGIH 1986, p. 22).

Therefore, OSHA has preliminarily concluded that aluminum metal dusts are essentially inert and is proposing a PEL of 10 mg/m³ TWA, the standard being proposed for all nuisance dusts. This level will provide protection against the physical irritation possible at the previously uncontrolled level and prevent the safety hazards potentially associated with overexposure to dusts. This health evidence forms a reasonable basis for proposing a new limit for aluminum metal dust. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

AMMONIUM SULFAMATE
CAS: 7773–06–0; Chemical Formula: $NH_4SO_3NH_2$
H.S. No. 1024

OSHA currently regulates ammonium sulfamate under its general nuisance dust limit of 15 mg/m³. The ACGIH recommends a limit of 10 mg/m³ as an 8-hour TWA. Ammonium sulfamate is a colorless, non-combustible, white crystalline substance.

Ammonium sulfamate has a low order of toxicity and should be considered a nuisance dust. Lehman (1951) found oral LD_{50}s of 3900 mg/kg, 5700 mg/kg, and 3000 mg/kg in rats, mice, and quail, respectively. He also reported that no effects were noted in rats administered 10,000 ppm in the diet for 105 days. The hazards associated with exposure to ammonium sulfamate include eye and nose irritation, interference with vision, and the danger of accidents caused by the distraction and avoidance reactions typical of workers overexposed to dusts in the workplace.

OSHA proposes a PEL of 10 mg/m³ TWA for ammonium sulfamate, which is the limit being proposed for all workplace dusts with a low order of toxicity. The Agency preliminarily concludes that this revised limit will protect workers against physical and other irritation and against workplace accidents associated with exposure to this substance. The health evidence forms a reasonable basis for proposing a new limit for ammonium sulfamate. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

BARIUM SULFATE
CAS: 7727–43–7; Chemical Formula: $BaSO_4$
H.S. No. 1031

OSHA has no specific limit for barium sulfate, although OSHA's nuisance dust limit applies; the ACGIH recommends a TLV–TWA of 10 mg/m³, total dust, for this substance. Barium sulfate is a white or yellowish, odorless, tasteless powder.

Because barium sulfate is insoluble, it is considered an inert dust. Einbrodt et

al. (1972) exposed rats to a concentration of 40 mg/m³ for 2 months and concluded that barium sulfate is an inert dust. As an inert dust of the non-collagenous type; however, barium sulfate has the potential to cause pneumoconiosis through tissue reaction to accumulated dust in the lung (Anon., *Brit. Med. J.* 1972). Barium sulfate has been observed to cause no adverse effects in industrial workers exposed over periods of several years (Doig 1976).

OSHA proposes that a PEL for barium sulfate be established at the 10 mg/m³ TWA level; this limit is the limit proposed by OSHA for all inert dusts. The Agency preliminarily concludes that this limit will protect workers against the hazards associated with exposures to these dusts, which include safety hazards, eye irritation, and upper respiratory tract irritation. The health evidence forms a reasonable basis for proposing a new limit for barium sulfate. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

BENOMYL
CAS: 17804–35–2; Chemical Formula: $C_{14}H_{18}N_4O_3$
H.S. No. 1032

OSHA currently regulates benomyl under its general nuisance dust limit of 15 mg/m³. The ACGIH has established a TLV TWA of 10 mg/m³ for this substance. Benomyl is a white crystalline solid, and exposures to it occur in its particulate form.

Studies of rats and rabbits indicate that the oral and skin absorption LD_{50}s are greater than 10,000 mg/kg, and studies of guinea pigs show a very low risk of skin irritation. Application to the shaved intact skin of ten male guinea pigs (as aqueous suspensions containing 5, 12.5, and 25 percent benomyl) resulted in negligible irritation; one of ten guinea pigs had mild erythema 2 days after application at the high rate (E. I. du Pont de Nemours and Co., Inc. 1974). In another study, instillation of 10 mg of dry 50-percent powder or 0.1 ml of 10-percent suspension in mineral oil caused only temporary mild conjunctival irritation (E. I. du Pont de Nemours and Co., Inc. unpublished). No teratogenic or mutagenic effects have been observed in rats, and dogs have been shown to eliminate more than 99 percent of ingested benomyl within 72 hours (Gardiner, Kirkland, and Klopping 1974).

OSHA proposes a PEL of 10 mg/m³ for this substance as an 8-hour TWA. The Agency believes that this limit will protect workers from the risks of benomyl's effects, which include irritation and erythema. The health evidence forms a reasonable basis for proposing a new limit for benomyl. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

BISMUTH TELLURIDE (UNDOPED)
CAS: 1304–82–1; Chemical Formula: Bi_2Te_3
H.S. No. 1035

OSHA has no current limit for undoped bismuth telluride; however, OSHA's general nuisance dust limit of 15 mg/m³ applies. The ACGIH recommends a TLV–TWA of 10 mg/m³ for the undoped substance. Bismuth telluride appears as gray, hexagonal platelets; it is also available as ingots or single crystals.

An 11-month inhalation study of dogs, rabbits, and rats exposed to pure bismuth telluride dust at 15 mg/m³ showed the pulmonary responses typical of exposure to inert dust (Wagner, Madden, Zimber et al. 1974).

Thus, OSHA proposes a permissible exposure limit of 10 mg/m³ TWA for pure undoped bismuth telluride. This limit is the same as the limit proposed for all nuisance dusts. The Agency believes that a 10-mg/m³ PEL will protect workers from the hazards associated with workplace dust exposures, which include skin and eye irritation and safety accidents. The health evidence forms a reasonable basis for proposing a new limit for undoped bismuth telluride. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

BORON OXIDE
CAS: 1303–86–2; Chemical Formula: B_2O_3
H.S. No. 1039

OSHA currently regulates boron oxide under its general nuisance dust limit of 15 mg/m³, and the ACGIH recommends a TLV–TWA of 10 mg/m³. Boron oxide occurs as a white powder or granular solid, and it has a bitter taste.

Animal studies indicate that skin and eye irritation were caused by the topical application of boron oxide to the skin of rabbits and by ocular instillation. Studies of aerosol administration at various exposure levels for varying time periods caused mild nasal irritation and an increase in urine acidity and creatinine coefficient in dogs and rats (Wilding, Smith, Yevich et al. 1959). Young rats that were force-fed a 10-percent slurry of boron oxide in water for 3 weeks showed no growth retardation or other effects (Wilding, Smith, Yevich et al. 1959).

Gabrant and co-workers (1984) determined the prevalence of eye and respiratory irritation among boron-oxide-exposed workers; those exposed to boron oxide concentrations ranging from 1.2 to 8.5 mg/m³ were then compared with controls. Workers exposed at an average concentration of 4.1 mg/m³ reported significant increases in coughing; eye, nose, and throat irritation; dryness of the mouth; and sore throats.

The ACGIH believes that a TLV–TWA of 10 mg/m³ will provide protection against boron oxide's irritant effects (ACGIH 1986). However, OSHA notes that irritation of the upper respiratory tract and eyes occurs among occupationally exposed workers at an average airborne concentration of 4.1 mg/m³. OSHA therefore proposes a PEL of 10 mg/m³ TWA and solicits additional information on the boron oxide exposure levels associated with adverse health effects in workers. The health evidence forms a reasonable basis for proposing a new limit for boron oxide. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

CALCIUM CARBONATE
CAS: 1317–65–3; Chemical Formula: $CaCO_3$
H.S. No. 1057

OSHA currently regulates calcium carbonate under the 15-mg/m³ nuisance dust limit. The ACGIH recommends a TLV–TWA of 10 mg/m³, total dust. Calcium carbonate is an odorless, tasteless powder or crystal.

The ACGIH considers calcium carbonate a nuisance dust and has accordingly established a 10-mg/m³ TWA limit for it. Beal, Griffith, and Nagelschmidt (as cited in the ACGIH 1986, p. 90) believe that exposure to this substance in its pure form does not cause pneumoconiosis, and Hunter (1975) confirms this finding.

OSHA is proposing to reduce its current PEL of 15 mg/m³ for calcium carbonate to 10 mg/m³ to protect workers against the risks associated with dust exposures in the workplace. These risks include eye and skin irritation, as well as the danger of accidents caused by interference with vision and the distractive effects of these substances. The health evidence forms a reasonable basis for proposing a new limit for calcium carbonate. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

CALCIUM SILICATE
CAS: 1344–95–2; Chemical Formula: None
H.S. No. 1061

OSHA has no limit specifically for calcium silicate; the Agency classifies this substance as a nuisance dust and assigns it an 8-hour TWA limit of 15 mg/m³, as total dust. The ACGIH also classifies calcium silicate as a nuisance dust and has established an 8-hour limit of 10 mg/m³ for this white powder.

There are no reported health effects in humans or animals as a result of exposure to calcium silicate. Calcium silicate is thus a nuisance dust, without long-term adverse health effects if exposures are kept under reasonable control.

OSHA is proposing an 8-hour limit of 10 mg/m³ TWA for calcium silicate; this limit is being proposed for all nuisance dusts and inert particulates. The Agency preliminarily concludes that this limit will protect workers from the risk of reduced visibility and distraction that can cause accidents in the workplace and will also prevent them from experiencing the eye and skin irritation associated with exposures to higher levels of this or other nuisance dusts. The health evidence forms a reasonable basis for proposing a new limit for calcium silicate. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

CALCIUM SULFATE
CAS: 7778-18-9; Chemical Formula: $CaSO_4$
H.S. No. 1062

OSHA currently regulates calcium sulfate under its general nuisance dust limit of 15 mg/m³. The ACGIH recommends a TLV-TWA of 10 mg/m³ total dust for this crystalline or powdery substance.

Calcium sulfate dust is known to have a low order of toxicity, and it is not reported to have specific irritant properties (ACGIH 1986, p. 93). One report has indicated that no lung diseases are associated with exposure to calcium sulfate in miners (Hunter 1975). Because calcium sulfate appears to be biologically inert, it is appropriately classified as a nuisance dust.

OSHA is proposing to reduce the permissible exposure limit to an 8-hour TWA of 10 mg/m³ (total dust) for calcium sulfate, the limit being proposed for all dusts in this category. The Agency preliminarily concludes that this limit will prevent eye and skin irritation, interference with vision, and lack of concentration associated with high dust exposures in the workplace. As is the case for all of the substances classified as nuisance dusts, calcium sulfate presents a safety as well as a health hazard to exposed workers. The health evidence forms a reasonable basis for proposing a new limit for calcium sulfate. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

CELLULOSE
CAS: 9004-34-6; Chemical Formula: $(C_6H_{10}O_5)n$
H.S. No. 1076

OSHA currently regulates cellulose under the 8-hour TWA limit of 15 mg/m³ for nuisance dusts. The ACGIH recommends a TLV-TWA of 10 mg/m³ (total dust). Technical cellulose refers to that portion of the plant cell wall derived exclusively from glucose and resembles cotton cellulose in its physical and chemical properties (ACGIH 1986, p. 113).

Technical cellulose is inert, and inhalation of cellulose dust is not irritating or toxic in exposed humans (Schreiber 1974). In industry, cellulose dust occurs in combination with other substances, such as quartz dust, wood, cotton, flax, jute, and hemp fibers, and these substances have demonstrated toxicities that are unrelated to their cellulose content (ACGIH 1986, p. 113).

OSHA is proposing to reduce the PEL for this dust to 10 mg/m³ TWA total dust for cellulose dust containing less than 1 percent quartz. This limit is the limit OSHA is proposing for all of the "nuisance" dusts. The Agency preliminarily concludes that this reduced limit will protect exposed workers from safety and health risks associated with exposure to cellulose dust in the workplace. These adverse effects include eye and skin irritation, coughing, interference with vision, loss of the ability to concentrate, and accidents. The health evidence forms a reasonable basis for proposing a new limit for cellulose. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

2-CHLORO-6-(TRICHLOROMETHYL) PYRIDINE (NITRAPYRIN)
CAS: 1929-82-4; Chemical Formula: $C_6H_3Cl_4N$
H.S. No. 1082

OSHA currently has no specific limit for nitrapyrin; however, OSHA's general nuisance dust limit of 15 mg/m³ TWA applies. The ACGIH recommends a TLV-TWA of 10 mg/m³ and a TLV-STEL of 20 mg/m³. Nitrapyrin is a crystalline substance.

Nitrapyrin's very low vapor pressure limit makes hazardous inhalation exposures unlikely. Torkelson (as cited in ACGIH 1986, p. 428) has reported feeding dogs and rats a dosage of 15 mg/kg daily for 93 days. He observed no adverse effects in appearance, behavior, growth, food consumption, body and organ weight, mortality, or blood chemistry, and no tissue or organ changes.

OSHA proposes a PEL of 10 mg/m³ TWA for this dust, which is the limit that OSHA is proposing for all nuisance dusts to protect workers from the health and safety risks associated with exposure to these dusts. With regard to the 20-mg/m³ STEL, the ACGIH (1986) provided no basis for this limit. OSHA therefore requests additional information that will assist the Agency in determining the need for the 20-mg/m³ STEL. The health evidence forms a reasonable basis for proposing a new limit for nitrapyrin. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

CLOPIDOL (COYDEN)
CAS: 2971-90-6; Chemical Formula: $C_7H_7Cl_2NO$
H.S. No. 1095

OSHA has no current limit specifically for clopidol; however, OSHA's general nuisance dust limit of 15 mg/m³ TWA applies. The ACGIH recommends a TLV-TWA of 10 mg/m³ and a TLV-STEL of 20 mg/m³. Clopidol is a solid.

Clopidol has a low reported acute oral toxicity. The oral LD_{50} in rats, rabbits, and guinea pigs is greater than 8 g/kg (Dow Chemical Company 1973). Long-term (two-year) studies of rats and dogs fed at levels of 15 mg/kg and 5 mg/kg per day, respectively, showed no adverse effects. Similarly, there were no adverse effects on fertility, gestation, viability, or lactation in rats and rabbits, and no increase in teratogenicity (Dow Chemical Company 1973). The chronic toxicity of clopidol is also reported to be low (ACGIH 1986, p. 141).

OSHA is proposing a PEL of 10 mg/m³ TWA for clopidol, the same limit as for all nuisance dusts. OSHA preliminarily concludes that this limit will protect workers from the health and safety risks associated with exposure to these dusts. OSHA notes that the ACGIH (1986) provided no basis for the 20-mg/m³ STEL recommendation. OSHA requests additional information that will assist the Agency in determining the need for a STEL for clopidol. The health evidence forms a reasonable basis for proposing a new limit for clopidol. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

CRAG HERBICIDE (SESONE)

CAS: 136–78–7; Chemical Formula: $C_8H_7Cl_2NaO_2S$
H.S. No. 1102

OSHA currently applies a TWA limit of 15 mg/m³ for crag herbicide; this is the Agency's limit for all inert dusts. The ACGIH recommends a TLV–TWA of 10 mg/m³ for this colorless, odorless, non-combustible solid.

An early study reported an oral LD_{50} in rats of 1500 mg/kg for this herbicide (Smyth 1956). At high concentrations, crag herbicide is a gastrointestinal irritant (NIOSH 1984). Rats fed a diet containing 60 mg sesone/100 gm experienced minor liver damage; when fed 20 mg sesone/100 gm of diet for 2 years, rats showed no adverse effects (ACGIH 1986, p. 519). In 1984, NIOSH reported the oral LD_{50} in rats to be 730 mg/kg. There are no reported incidents of human poisonings associated with the use of sesone.

OSHA is proposing a reduction in the PEL to 10 mg/m³ TWA, the limit being proposed for all nuisance dusts. OSHA preliminarily concludes that this level will protect exposed workers from the safety and health hazards potentially associated with exposures to these dusts. These risks include accidents, interference with vision, and eye and skin irritation. This health evidence forms a reasonable basis for proposing a new limit for crag herbicide. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

DICYCLOPENTADIENYL IRON
CAS: 102–54–5; Chemical Formula: $C_{10}H_{10}Fe$
H.S. No. 1133

OSHA currently regulates dicyclopentadienyl iron under its nuisance dust limit of 15 mg/m³. The ACGIH recommends a TLV–TWA of 10 mg/m³ for this bright-orange, crystalline solid that smells like camphor.

Available evidence in animals suggests that dicyclopentadienyl iron has a relatively low order of oral toxicity. In mice, the oral LD_{50} has been reported as 600 mg/kg (Madinaveitia 1956). In rats, 1000 mg/kg has been reported as the lethal dose, and subacute oral toxicity tests have shown no fatalities when 10 feedings of 200 mg/kg were given over a 2-week period (E.I. du Pont de Nemours and Company 1955, as cited in ACGIH 1986, p. 195). Madinaveitia (1956) has determined that this substance is a hematinic agent in animals.

OSHA proposes an 8-hour TWA limit of 10 mg/m³ for dicyclopentadienyl iron. The Agency preliminarily concludes that this limit will protect workers against the risk of hematinic effects potentially associated with occupational exposure to this substance at the levels permitted in the absence of any OSHA PEL. The health evidence forms a reasonable basis for proposing a new limit for dicyclopentadienyl iron. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

EMERY
CAS: 112–62–9; Chemical Formula: Al_2O_3
H.S. No. 1155

OSHA currently regulates emery at an 8-hour TWA of 15 mg/m³ TWA, the Agency's limit for all nuisance dusts. The ACGIH recommends a limit of 10 mg/m³ TWA, total dust, for emery containing less than 1 percent quartz. Emery is impure corundum (aluminum oxide) and is found in certain mineralogical deposits.

The only report of ill effects from emery dust inhalation is a report of a case of pneumoconiosis occurring in France, and it is questionable whether this incident was caused by emery dust or silica impurities in the dust (*Archives des Maladies Professionelles de Medcin du Travail et de Securite* 1970). Exposure to emery dust containing less than 1 percent silica produces little if any effect on the health of exposed workers; it does not affect the lungs or produce organic disease at commonly encountered levels (ACGIH 1986, pp. 21, 229).

OSHA is therefore proposing a PEL of 10 mg/m³ TWA, total dust, for emery, in keeping with the Agency's decision to lower the TWA for all nuisance dusts. OSHA preliminarily concludes that this limit will prevent the safety and health risks associated with exposures to dusts in the workplace; these risks include the danger of accidents, interference with vision, distraction, and skin and eye irritation. The health evidence forms a reasonable basis for proposing a new limit for emery. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

FERBAM
CAS: 14484–64–1; Chemical Formula: $[(CH_3)_2NCS_2]_3Fe$
H.S. No. 1176

OSHA currently applies its general nuisance dust limit of 15 mg/m³ TWA to ferbam. The ACGIH recommends a TLV–TWA of 10 m/gm³ for this odorless black solid.

Ferbam, which is a fungicide, has been reported to have an oral LD_{50} of more than 17 mg/kg in rats, but rabbits and guinea pigs demonstrated less sensitivity to this substance (Hodge, Maynard, Downs, and Blanchet 1952). Thirty-day dietary studies of rats showed no effect at ferbam doses of 0.01 percent, with fatalities occurring at 0.5 percent. Dogs showed no adverse effects when fed 25 mg/kg of ferbam daily for 6 months.

Inhalation of ferbam affects the upper respiratory tract in humans, in a manner typical of airborne exposures to inert dusts.

OSHA is proposing to reduce the PEL to a 10-mg/m³ 8-hour TWA because the Agency has decided that a decrease in the limit from 15 to 10 mg/m³ is warranted for all the nuisance dusts. The Agency preliminarily concludes that this reduction is necessary to prevent the health and safety risks associated with workplace exposures to these dusts. These risks include skin and eye irritation, on-the-job accidents, and interference with vision. The health evidence forms a reasonable basis for proposing a new limit for ferbam. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

FIBROUS GLASS DUST
CAS: None; Chemical Formula: None
H.S. No. 1178

Fibrous glass dust is used primarily for thermal and acoustical insulation of residential and commercial buildings. There is currently no OSHA limit governing occupational exposure to fibrous glass dust. The ACGIH (1986) has established a 10-mg/m³ TLV–TWA, reflecting its belief that a nuisance-dust limit is appropriate. NIOSH (1977) recommended a 5-mg/m³ 10-hour TWA limit for total dust and a 3-fibers/cc limit for airborne fibers less than 3.5 um in diameter and longer than 10 um in length.

Epidemiologic studies of workers exposed to fibrous glass dust have generally failed to detect any significant increase in respiratory effects. One study of 2,028 workers (Nasr et al. 1976) did not identify any increased prevalence of abnormal radiographic findings among production workers compared to office workers; results of spirometric tests and administration of a chronic bronchitis questionnaire did not discern any difference in health between workers with the heaviest exposure and those with minimal exposure. Another study of 416 retired workers reported no difference in overall mortality or morbidity rates compared to general population rates (Enterline and Henderson 1975). Bayliss et al. (1976) failed to find any excess in overall mortality or lung cancer among 1,448 workers, but did report an excess of

mortality from nonmalignant respiratory disease; the authors could not attribute this excess to fibrous glass because smoking histories were not generally available. A review of 691 physicians' reports of adverse effects caused by exposure to vitreous fibers identified 66 reports of nondisabling upper respiratory tract symptoms (Milby and Wolf 1969).

In animal studies, intratracheal injection of thin glass fibers longer than 10 um into guinea pigs produced peribronchiolar fibrosis (Kuschner and Wright 1976). When such fibers were injected into the abdomen of rats, a dose-related increase in sarcomas and mesotheliomas resulted (Stanton and Wrench 1972; Wagner et al. 1973; Pott and Friedrichs 1972). The response mimics that found with asbestos, but the magnitude of the response is generally less than is found with asbestos. NIOSH (1977) did not consider fibrous glass to present a carcinogenic hazard after reviewing these data.

Based on the lack of reported adverse health effects in epidemiologic studies, the ACGIH (1986) considered fibrous glass dust to be essentially a nuisance dust and applied a 10-mg/m³ TLV–TWA. In contrast, NIOSH (1977) concluded that "available data are sufficient to demonstrate that fibrous glass does not act like an inert or nuisance dust because it can produce fibrosis in animals and respiratory tract irritation in humans" (NIOSH 1977, p. 94). Relying on several investigations that showed a lack of adverse effects among workers exposed to mean respirable fiber concentrations generally less than 5 to 6 f/cc, NIOSH recommended that exposures be limited to 3 f/cc for fibers 3.5 um or less in diameter and greater than 10 um in length. In addition, NIOSH recommended that exposures to total fibrous glass dust be limited to 5 mg/m³ as a 10-hour TWA.

The 10-mg/m³ TLV–TWA considers only the health effects associated with fibrous glass particles. However, the PEL will also be applied by OSHA to situations where the fibrous form of this material is present. Recent concerns have emerged regarding the potential carcinogenicity of the long fibers (TIMA 1988). While a 3-fibers/cc limit for long, thin fibers has also been proposed by NIOSH, it is impossible in this rulemaking to adequately evaluate that limit for the fibrous form of this substance. Therefore, as an interim measure, OSHA proposes that a 5-mg/m³ TWA be adopted as the PEL for fibrous glass dust. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for fibrous glass dust if the Agency determines that this limit will substantially reduce significant risk. Because of the concern with the fibrous forms of this substance, feasibility is not expected to be a problem at this level. If future information and priorities indicate the need for a more restrictive standard for the fibrous form of this material, OSHA will initiate individual-substance rulemaking.

GLYCERIN (MIST)
CAS: 56–81–5; Chemical Formula: $CH_2OHCHOHCH_2OH$
H.S. No. 1188

OHSA currently has no specific limit for glycerin mist, although this substance is currently regulated at 15 mg/m³ as a general nuisance dust. The ACGIH recommends a TLV–TWA of 10 mg/m³. Glycerin is an oily hygroscopic liquid with a warm, sweet taste.

Glycerin was long considered to be nontoxic; however, more recent information has indicated that the mist may be injurious to the kidneys at very high exposure levels (Campanacci 1965). Ackerman, Bassler, and Wagner (1975) have reported that glycerin mist is easily metabolized and excreted. In the adult human of average weight, 2 grams of glycerol can be metabolized and excreted in an 8-hour workday. At this metabolic and elimination rate, the ACGIH believes that no ill effects are likely to occur as a result of exposure at or below 10 mg/m³ as an 8-hour TWA, the level set for nuisance dusts (ACGIH 1986, p. 286).

OSHA proposes a limit of 10 mg/m³ TWA for glycerin mist, which is the level the Agency is proposing for all inert dusts. The health evidence forms a reasonable basis for proposing a new limit for glycerin mist. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk. However, OSHA notes that glycerin may not be truly inert, as evidenced by the study of Campanacci (1943). The Agency specifically requests comments on the toxicity (if any) of exposures to various levels of glycerin.

GRAPHITE, SYNTHETIC
CAS: None; Chemical Formula: None
H.S. No. 1191A

OSHA currently has no specific standard for synthetic graphite but regulates it as a nuisance dust. OSHA's 8-hour TWA for all nuisance dusts is 15 mg/m³. The ACGIH recommends a TLV–TWA limit of 10 mg/m³ for graphite as total dust.

Synthetic graphite is a crystalline form of carbon made from high-temperature treatment of coal or petroleum products; it has the same properties as natural graphite. Meiklejohn reported that synthetic graphite injected intraperitoneally in mice produced effects characteristic of inert dusts (1958).

In humans, exposure to natural graphite has long been associated with the development of pneumoconiosis (Koopman 1924; Ruttner et al. 1952; Pendergass et al. 1967). Lister (1961, 1972) reported fibrotic changes in the lungs of a worker who had been engaged for 17 years in the production and milling of synthetic graphite. Other reports of lung injury caused by exposure to graphite have not distinguished between the form of the graphite (i.e., natural or synthetic) causing the injury; in addition, exposures to impurities, such as quartz silica, were involved in many of the reported cases (ACGIH 1986, p. 291).

OSHA is proposing to reduce the 8-hour TWA limit for all the nuisance dusts from 15 mg/m³ to 10 mg/m³, to protect against the health and safety risks potentially associated with dust exposures in the workplace. The health evidence forms a reasonable basis for proposing a new limit for synthetic graphite. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk. However, the Agency notes that occupational exposure to synthetic graphite has been associated with pneumoconiosis, and preliminarily concludes that a lower limit may be appropriate. Specific comment is solicited on the exposure levels and health effects associated with synthetic graphite related occupational disease.

GYPSUM, TOTAL DUST
CAS: 7778–18–9; Chemical Formula: $CaSO_4 \cdot H_2O$
H.S. No. 1192

The current OSHA limit for gypsum is an 8-hour TWA of 15 mg/m³, the Agency's current limit for all inert dusts. The ACGIH recommends a TWA of 10 mg/m³, measured as total dust, for gypsum and other inert particulates. Gypsum exists as either colorless or white crystals.

The ACGIH (1986) states that gypsum does not "produce significant organic disease or toxic effect when exposures are kept under reasonable control." Exposures in excess of the recommended limit may result in reduced visibility, deposits of gypsum dust in the eyes, ears, and nasal passages, and skin irritation.

OSHA is proposing to reduce the current limit for gypsum total dust to 10 mg/m³ as an 8-hour TWA, which is the limit being proposed for all of the inert particulates. The Agency preliminarily concludes that this limit will protect exposed workers from the safety and health risks associated with exposures to gypsum or other dusts at higher levels. These risks include eye and skin irritation, interference with vision, and safety accidents. The health evidence forms a reasonable basis for proposing a new limit for gypsum total dust. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

KAOLIN, TOTAL DUST
CAS: None; Chemical Formula: $H_2Al_2Si_2O_8O \cdot H_2O$
H.S. No. 1230

OSHA's current limit for kaolin is 15 mg/m³, measured as total dust; this is the Agency's current limit for all inert particulates. The ACGIH recommends a TLV–TWA of 10 mg/m³, also measured as total dust. Kaolin may be a white powder or a white or yellow-white earthy mass.

Exposure to excess amounts of kaolin dust or other inert particulates may cause reduced visibility in the workplace, injury to the skin or mucous membranes, and a buildup of dust deposits in the eyes, ears, and nasal passages (ACGIH 1986).

OSHA is proposing a PEL of 10 mg/m³ TWA for kaolin, measured as total dust, which is the limit being proposed for all of the inert particulates. The Agency preliminarily concludes that this limit will protect exposed workers from the safety and health risks potentially associated with exposure to the dusts at higher levels. These risks include skin and mucous membrane injury, accumulations of kaolin deposits in the eyes, ears and nose, and reduced visibility in the workplace, which may endanger worker safety. The health evidence forms a reasonable basis for proposing a new limit for kaolin, measured as total dust. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

LIMESTONE, Total Dust
CAS: 1317–65–3; Chemical Formula: $CaCO_3$
H.S No. 1232

The current OSHA PEL for limestone is an 8-hour TWA of 15 mg/m³, as total dust. The ACGIH recommends 10 mg/m³ TWA for limestone, measured as total dust. Limestone is a hard white solid.

Exposure to limestone dust has not been associated with the development of pneumoconiosis, and ACGIH considers it to be a nuisance dust (see discussion on calcium carbonate above).

Exposure to excess levels of limestone dust may result in deposits in the eyes, ears, and nasal passages, or may injure the skin or mucous membranes (ACGIH 1986). OSHA proposes an 8-hour TWA of 10 mg/m³ for limestone dust, the limit being proposed for all of the nuisance dusts and particulates. The Agency preliminarily concludes that this limit will reduce the safety and health hazards posed to exposed employees by nuisance dusts in the workplace. The health evidence forms a reasonable basis for proposing a new limit for limestone dust. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

MAGNESITE, TOTAL DUST
CAS: None; Chemical Formula: $(MgCO_3)_4 \cdot Mg(OH)_2 \cdot 5H_2O$(approx)
H.S. No. 1233

OSHA's existing PEL for magnesite is 15 mg/m³, measured as total dust; this is the Agency's limit for all inert particulates. The ACGIH recommends a TLV–TWA of 10 mg/m³, also measured as total dust. Magnesite occurs as a white powder.

Magnesite is considered by both OSHA and the ACGIH to be one of the "nuisance dusts," which "do not produce significant organic disease or toxic effect when exposures are kept under reasonable control" (ACGIH 1986). Exposure to excess levels of magnesite or the other nuisance particulates in the workplace causes skin or mucous membrane irritation resulting from contact with the magnesite itself or from rigorous cleansing procedures necessary for removing the dust; buildup of dust deposits in the eyes, ears, and nasal passages; and reduced visibility in the workplace.

OSHA is proposing a PEL of 10 mg/m³ TWA for magnesite, measured as total dust. This is the limit being proposed for all of the inert particulates. The Agency preliminarily concludes that this limit is necessary to protect exposed workers from the health and safety risks potentially associated with exposure to magnesite or other inert dusts. These risks include skin and mucous membrane injury, the deposition of magnesite deposits in the eyes, ears, and nose, and reduced workplace visibility, which may endanger employee safety. The health evidence forms a reasonable basis for proposing a new limit for magnesite. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

MAGNESIUM OXIDE (FUME)
CAS: 1309–48–4 Chemical Formula: MgO
H.S. No. 1234

OSHA's current limit for magnesium oxide (as fume) is 15 mg/m³ as an 8-hour TWA, the Agency's limit for inert dusts. The ACGIH recommends a TLV–TWA limit of 10 mg/m³ for the fume of this white, odorless, very fine powder.

Slight reactions (not further specified) have been reported in human subjects after exposures of less than 10 minutes to freshly generated MgO fume at concentrations of from 400 to 600 mg/m³ Drinker, Thomson, and Finn 1927). Animal and human studies of magnesium oxide fume exposure have shown toxicities less marked than but similar to those attributable to zinc oxide fume (Drinker and Drinker 1928). The symptoms of exposure include those of metal fume fever (fever, chills, muscular pain, nausea, and vomiting) and leukocytosis, symptoms analogous to those caused by exposure to zinc oxide fume.

OSHA is proposing a PEL of 10 mg/m³ TWA for magnesium oxide fume, the limit OSHA is proposing for all of the toxicologically inert nuisance particulates included in this rulemaking. OSHA preliminarily concludes that this limit will protect exposed workers from the safety and health risks potentially associated with exposures to these dusts in the workplace. These risks include accidents, skin and eye irritation, and interference with vision. The health evidence forms a reasonable basis for proposing a new limit for magnesium oxide fume. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

MALATHION
CAS: 121–75–5; Chemical Formula: $C_{10}H_{19}O_6PS_2$
H.S. No. 1235

Both OSHA and NIOSH have 15-mg/m³ limits for malathion (and OSHA has a skin notation); the ACGIH TLV for this substance is 10 mg/m³ as a TWA, also with a skin notation.

Malathion is a widely used organophosphorus insecticide having a relatively low level of toxicity; some authors have determined that malathion is approximately 1/100th as toxic as parathion (Johnson et al. 1952). Rats fed malathion at a concentration of 100 ppm for 2 years exhibited no toxic effects (Hazleton and Holland 1953). Several occupational and research exposures

involving scientists or human volunteers produced no change in blood cholinesterase or other effects (Rider et al. 1959; Hayes et al. 1960; Culver et al. 1956).

Fatalities have been reported in the Japanese and Indian literature, but these deaths have always involved extremely high doses of malathion (Chabra 1970; Horiguchi 1973). Thus, malathion's principal workplace effect is that it acts like a nuisance dust. NIOSH concurs in this view (NIOSH 1978i) and recommends a higher TWA (15 mg/m³) for this substance than would be permitted by the ACGIH.

The 10-mg/m³ TLV–TWA is part of a generally accepted, broad guideline to limit exposure to nuisance dusts; the 15-mg/m³ REL represents an earlier approach to the control of these substances. As discussed in this section, nuisance dusts must be controlled to prevent skin, respiratory tract, and eye irritation, as well as a variety of other effects. This level is readily attainable in the workplace, using generally available industrial hygiene practices. OSHA is therefore proposing a PEL of 10 mg/m³ TWA for malathion with a skin notation. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for malathion if the Agency determines that this limit will substantially reduce significant risk.

MARBLE, TOTAL DUST
CAS: 1317–65–3; Chemical Formula: None
H.S. No. 1239

OSHA currently has no specific limit for marble dust, but regulates this substance as a nuisance dust, for which the 8-hour TWA limit as total dust is 15 mg/m³. The ACGIH has established an 8-hour TLV–TWA of 10 mg/m³ for marble dust as total dust containing less than 1 percent quartz. Marble dust, a metamorphic form of calcium carbonate dust, is an odorless and tasteless powder or crystal.

OSHA is proposing an 8-hour TWA limit of 10 mg/m³ for marble dust as total dust containing less than 1 percent quartz. This is the limit being proposed by the Agency for all nuisance dusts and inert particulates. OSHA preliminarily concludes that this limit will protect exposed workers from the safety and health risks associated with exposure to the inert dusts at higher levels. These risks include the danger of accidents caused by interference with vision and distraction, and eye and skin irritation. The health evidence forms a reasonable basis for proposing a new limit for marble dust. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

METHOXYCHLOR
CAS: 72–43–5; Chemical Formula: $C_{16}H_{15}Cl_3O_2$
H.S. No. 1246

OSHA currently applies its 15-mg/m³ TWA limit for nuisance dusts to methoxychlor. The ACGIH recommends a limit of 10 mg/m³ TWA for this white crystalline solid, which is assigned the limit for all inert dusts.

Methoxychlor has a low level of toxicity. The reported oral LD_{50} for rats is 6000 mg/kg (Lehman 1954). Lehman also determined that 100 ppm for 2 years is the lowest dietary level producing no effect in rats: this corresponds to a level of 350 mg/man/day. Results of another dietary study indicated that rats fed 200 ppm methoxychlor for 2 years were not affected in terms of growth or survival (Hodge, Maynard, and Blanchet 1952). Tegeris and co-workers reported that dogs fed 1 g/kg daily for 6 months showed weight loss; most animals died within 9 weeks when the dietary level was increased to 2 g/kg daily (Tegeris, Earl, Smalley, and Curtis 1966). Morgan and Hickenbottom (1978) reported that male Holtzman rats fed 10, 40, 160, or 640 mg/kg for 24 hours showed no liver abnormalities. Extrapolating from animal data, Lehman estimated the dose levels that would produce toxic effects in humans as follows: The fatal oral dose would be 450 grams; adverse health effects would occur at 6430 mg/kg orally; and 2414 mg/kg is the level at which dermal effects would be predicted to occur (Lehman 1954).

OSHA is proposing to reduce the existing 8-hour TWA limit from 15 mg/m³ to 10 mg/m³ to reduce the health and safety risks of exposure to the inert dusts in the workplace. These risks include interference with vision, skin and eye irritation, and accidents. The health evidence forms a reasonable basis for proposing a new limit for methoxychlor. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

MINERAL WOOL FIBER
CAS: None; Chemical Formula: None
H.S. No. 1277

OSHA currently has no limit specifically for mineral wool fiber, but this substance is covered by the Agency's 15-mg/m³ 8-hour TWA limit for all inert dusts and particulates. The ACGIH recommends an 8-hour TWA limit of 10 mg/m³ for mineral wool fiber as total dust containing less than 1 percent quartz. Rock or mineral wool is composed of mineral fibers produced by blowing steam or air through molten furnace slag; the fibers contain less than 1 percent quartz, and the substance is therefore vitreous.

In a study of cats, the only ill effects of inhalation that were observed were pulmonary changes attributed to silicate deposition (Fairhall, Webster, and Bennett 1935). A Russian study showed that mineral wool dust containing little free silica and moderate amounts of combined silica produced moderate diffuse and nodular sclerosis in the lungs of rats after 2 to 3 months (Grimailovskaya et al. 1957). The ACGIH (1986, p. 414) states that these conclusions are suspect, however, because rat lungs show only cellular foci even after 2 to 3 months of exposure to heavy quartz dust; the ACGIH believes that the Soviet investigators were not familiar with the chronic bronchitis that is endemic in rats.

Chronic exposure of 84 workers to mineral wool (with a free silica content of no more than 0.5 percent) for from 7 to 29 years resulted in no X-ray evidence of silicosis (Carpenter and Spolyor 1945).

OSHA is proposing a PEL of 10 mg/m³ TWA for mineral wool fiber as total dust containing less than 1 percent quartz. This is the limit being proposed for all inert or "nuisance" particulates. The Agency preliminarily concludes that this limit will protect workers against the safety risks associated with on-the-job exposures to inert particulates; these risks include interference with vision, accidents caused by the distracting effects of these substances, eye irritation, and irritations of the skin and mucous membranes. The health evidence forms a reasonable basis for proposing a new limit for mineral wool fiber. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

MOLYBDENUM (INSOLUBLE COMPOUNDS)
CAS: 7439–98–7; Chemical Formula: None
H.S. No. 1278

OSHA has a current limit of 15 mg/m³ TWA for the insoluble compounds of molybdenum, which include molybdenum metal and the dioxide. This is the Agency's limit for all inert dust and particulates. The ACGIH recommends a TLV–TWA of 10 mg/m³ as molybdenum. Molybdenum is a silver-white metal or a dark-gray or black powder.

In general, the compounds of molybdenum have a low order of toxicity. The insoluble compounds of molybdenum have not been reported to have any definite toxicities, although no specific exposure data are available.

Mogilvskaya (1950) concluded that the dust of molybdenum metal and molybdenum dioxide caused only transitory irritation of mucosal surfaces in white mice after an intensive dusting for 1 hour; in a similar 30-day exposure, the metal and the dioxide proved only minimally poisonous.

OSHA is proposing a PEL for the insoluble compounds of molybdenum of 10 mg/m³ TWA, measured as molybdenum. This is the limit being proposed for all inert dusts and particulates. The Agency preliminarily concludes that this limit will protect workers from the safety and health risks of overexposure to inert particulates. These risks include distraction and interference with vision, which can lead to safety accidents, and eye, nose, and skin irritation. The health evidence forms a reasonable basis for proposing a new limit for molybdenum. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

NUISANCE PARTICULATES
CAS: None; Chemical Formula: None
H.S. No. 1294

OSHA, currently regulates nuisance dusts at an 8-hour TWA limit of 15 g/m³ as total dust. The ACGIH recommends a TLV-TWA of 10 mg/m³ (as total dust) for nuisance particulates having a quartz content of less than 1 percent. Nuisance particulates are air-suspended particles having diameters of greater than respirable size.

According to the ACGIH:

> Nuisance particulates differ from fibrogenic dusts in that the former produce little adverse effect on the lungs and do not cause organic disease or toxic effects when exposures are reasonably controlled; fibrogenic dusts, on the other hand, cause the formation of pulmonary scar tissue when they are inhaled in excessive amounts. Nuisance dusts are sometimes referred to as "biologically inert," and, in the sense that they do not affect the architecture of pulmonary air spaces, do not form collagen or scar tissue, and do not cause irreversible tissue damage, this term is appropriate. Excessive concentrations of nuisance dusts may reduce visibility, cause deposits in the eyes, ears, and nasal passages, or injure the skin or mucous membranes by mechanical action or by the rigorous skin-cleansing procedures necessary for their removal (ACGIH 1986, p. 445).

OSHA proposes an 8-hour TWA limit of 10 mg/m³ as total dust, for nuisance particulates containing less than 1 percent quartz. The Agency preliminarily concludes that this limit will protect workers against the safety and health risks associated with exposure to excessive concentrations of these dusts, including reduced visibility, deposits in the eyes, ears, and nasal passages, and skin injury. The health evidence forms a reasonable basis for proposing a new limit for nuisance particulates. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

PENTAERYTHRITOL, TOTAL DUST
CAS: 115-77-5; Chemical Formula: $C(CH_2OH)_4$
H.S. No. 1305

OSHA currently has no separate limit for pentaerythritol, but this substance is classified as a nuisance dust and is regulated at 15 mg/m³ TWA, the Agency's limit for all such dusts. The ACGIH recommends a TLV-TWA of 10 mg/m³ for total dust containing less than 1 percent quartz. Pentaerythritol is an odorless, white, crystalline solid.

Pentaerythritol has a low acute toxicity and only mild irritative effects. Rats exposed at 11,000 mg/m³ for 6 hours were reported to show no ill effects from a single exposure, and rats, dogs, and guinea pigs exposed 6 hours daily for 90 days also showed no effects (Keplinger and Kay 1964). The oral LD_{50}s in guinea pigs and mice are 11.3 g/kg and 22.5 g/kg, respectively; rats survived oral doses as high as 16 g/kg. At higher doses, animals displayed diarrhea, tremors, ataxia, and loss of righting reflex (Keplinger and Kay 1964). Daily applications of a saturated aqueous solution of technical pentaerythritol to rabbit skin produced no significant irritation; a single application of 10 g/kg aqueous paste on intact or abraded rabbit skin produced no evidence of percutaneous absorption (Keplinger and Kay 1964; Hercules, Inc., as cited in ACGIH 1986, p. 462). Instillation of a 50-percent aqueous suspension into the conjunctival sac of rabbits' eyes resulted in slight transient irritation (Hercules, Inc., as cited in ACGIH 1986, p. 462).

Human volunteers are reported to have eliminated 85 percent of dietary pentaerythritol unchanged in the urine within 30 hours. A slight and transient increase in apparent blood sugar that was proportional to the ingested dose appeared in these subjects soon after administration (Berlow, Barth, and Snow 1958).

OSHA is proposing an 8-hour PEL of 10 mg/m³ TWA for pentaerythritol, which is the level the Agency is proposing for all nuisance dusts. The Agency preliminarily concludes that this limit will protect exposed employees from the safety and health risks potentially associated with exposure to this substance at higher levels. These risks include the risk of accidents and of eye and skin irritation. The health evidence forms a reasonable basis for proposing a new limit for pentaerythritol. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

PERLITE
CAS: None; Chemical Formula: None
H.S. No. 1310

OSHA currently regulates perlite as a nuisance dust at an 8-hour PEL of 15 mg/m³. The ACGIH recommends a TLV-TWA of 10 mg/m³ for perlite as total dust containing less than 1 percent quartz. Perlite is a natural volcanic glass. It is essentially an amorphous mineral consisting of fused sodium potassium aluminum silicate.

Perlite is reported to have a free silica content varying from zero to 3 percent (Anderson, Selvig, Baur et al. 1956; Perlite Institute, as cited in ACGIH 1986, p. 467). In its processed crude and expanded forms, perlite is reported to have a measurable quartz content of 0.4 percent quartz and 0.2 percent cristobalite (Sheckler, as cited in ACGIH 1986, p. 467). There are no published reports of adverse physiologic effects from exposure to perlite dust.

OSHA has preliminarily concluded that perlite is non-toxic when airborne concentrations are maintained at levels of 10 mg/m³ or below and when its quartz content is limited to a level below 1 percent crystalline silica. For these reasons, the Agency proposes a PEL of 10 mg/m³ TWA for total perlite dust containing less than 1 percent quartz, which is the limit being proposed for all nuisance particulates. OSHA believes that this limit will protect exposed workers from the risk of safety and health hazards associated with workplace exposures to inert particulates; these risks include the danger of accidents, interference with vision, and eye and skin irritation. The health evidence forms a reasonable basis for proposing a new limit for perlite. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

PICLORAM
CAS: 1918-02-1; Chemical Formula: $C_6H_3Cl_3N_2O_2$
H.S. No. 1328

OSHA currently has no limit for picloram. The ACGIH recommends a TLV-TWA of 10 mg/m³ and a TLV-STEL of 20 mg/m³ for this white powder, which has an odor like that of chlorine.

Picloram has low acute oral toxicity, with LD_{50} values of 3.75 g/kg for rats, 1.5

g/kg for mice, and 2.0 g/kg for rabbits (NIOSH 1979). Two-year feeding studies showed no ill effects in albino rats and beagle dogs from ingestion of doses up to and including 150 mg/kg/day (McCallister and Leng 1969). At 225 mg/kg/day, rats displayed moderate liver and kidney changes and, in females, slight body weight loss after 90 days (McCallister and Leng 1969). McCallister and Leng (1969) reported no fertility, reproduction, or lactation effects in albino rats fed at levels of up to 3000 ppm (0.3 percent) in a 3-generational study. However, maternal toxicity in rats was reported at dietary levels of 750 and 1000 mg/kg administered during days 6 through 15 of gestation, but neither teratogenic nor neonatal effects were observed when sub-toxic or maternally toxic doses of picloram were administered during organogenesis (Thomson et al. 1972). The National Cancer Institute (NCI) has concluded that picloram is not carcinogenic in mice or rats (NCI 1977).

OSHA proposes a TWA limit of 10 mg/m³ and a 15-minute STEL of 20 mg/m³ for picloram. The Agency preliminarily concludes that this combined limit will minimize the risk of systemic effects, such as liver and kidney damage, potentially associated with exposure to this substance in the absence of any OSHA PEL. The health evidence forms a reasonable basis for proposing a new limit for picloram. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

PLASTER OF PARIS, Total Dust
CAS: None; Chemical Formula: CaO_4S
H.S. No. 1331

Plaster of Paris is a fine white powder. OSHA's Z–3 table lists an 8-hour TWA exposure limit of 15 mg/m³ for Plaster of Paris. This is the Agency's limit for all of the inert particulates and dusts. The ACGIH has established a 10-mg/m³ TWA for Plaster of Paris, measured as total dust.

Where occupational exposures to Plaster of Paris have been limited, no toxic effects or organic diseases of the lungs have occurred (see discussion on calcium sulfate above). Exposure to excessive levels of dust in the work area may result in reduced visibility or injury to the skin or mucous membranes from the dust itself, or damage to the skin from the rigorous skin-cleansing procedures required to remove the dust (ACGIH 1986).

OSHA is proposing an 8-hour TWA of 10 mg/m³ for Plaster of Paris dust. The Agency preliminarily concludes that this limit will reduce the risk of the safety and health hazards described above, which include accidents, interference with vision and concentration, and skin and eye irritation. The health evidence forms a reasonable basis for proposing a new limit for plaster of paris dust. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

PORTLAND CEMENT
CAS: 65997–15–1; Chemical Formula: None
H.S. No. 1333

OSHA currently has a limit of 50 mppcf (approximately 15 mg/m³) for Portland cement containing less than 1 percent crystalline silica. The ACGIH recommends a TLV–TWA of 10 mg/m³ for Portland cement as total dust containing less than 1 percent quartz. Portland cement refers to a class of hydraulic cements that are odorless gray powders containing less than 1 percent crystalline silica. Portland cement is insoluble in water and contains tri- and di-calcium silicate, with varying amounts of alumina, tricalcium aluminate, and iron oxide.

Intraperitoneal injection of Portland cement in guinea pigs produced an absorptive reaction, which is typical of inert particulates. Portland cement is eventually eliminated from tissue and is generally considered harmless when ingested (Miller and Sayers 1941).

In a study of industrial exposures, Gardner and associates (1939) found no evidence of Portland-cement-related pneumoconiosis in 2,278 workers who had been heavily exposed to this substance for prolonged periods of time (Gardner, Durkan, Brumfiel, and Sampson 1939). Conflicting reports of pneumoconiosis (Parmeggiani 1951; Prosperi and Barsi 1957) are attributed to the presence of silica in the inhaled dust rather than to exposure to Portland cement itself (ACGIH 1986, p. 494). Cement dermatitis does occur among exposed workers, however, as a consequence of the alkaline, abrasive, and hygroscopic properties of the wet cement, which cause general irritation of the skin (Schwartz, Tulipan, and Birmingham 1957).

OSHA is proposing an 8-hour TWA PEL of 10 mg/m³ for Portland cement as total dust containing less than 1 percent quartz. The Agency preliminarily concludes that this limit will protect workers against the safety risks associated with on-the-job exposures to inert particulates. These risks include interference with vision, accidents caused by the distracting effects of these substances, eye irritation, and irritation of the skin and mucous membranes. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for Portland cement if the Agency determines that this limit will substantially reduce significant risk. In addition, revising the limit to 10 mg/m³ TWA will simplify employee exposure monitoring for Portland cement, since gravimetric rather than impinger methods can be used.

ROUGE, TOTAL DUST
CAS: None; Chemical Formula: None
H.S. No. 1351

OSHA currently has no specific limit for rouge, but regulates this substance as a nuisance dust under the Agency's nuisance dust standard of 15 mg/m³ as an 8-hour TWA. The ACGIH has established an 8-hour limit of 10 mg/m³ TWA for rouge as total dust containing less than 1 percent quartz. Rouge is a high-grade red pigment, composed mainly of ferric oxide, that is used as a polishing agent for glass, jewelry, etc.

Rouge is a biologically inert substance. There are no studies demonstrating any effects of exposure to rouge in either animals or humans.

OSHA is proposing an 8-hour TWA of 10 mg/m³ for total dust, the limit being proposed by the Agency for all inert particulates and nuisance dusts. OSHA preliminarily concludes that this limit will protect workers from the health and safety risks associated with workplace exposure to higher levels of rouge and other nuisance dusts. These effects include reduced visibility and distraction, which may lead to accidents and injuries, and deposits in the ears, eyes, and nasal passages. In addition, rouge and other nuisance particulates can cause skin and mucous membrane irritation. The health evidence forms a reasonable basis for proposing a new limit for rouge. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

SILICON
CAS: 7440–21–3; Chemical Formula: Si
H.S. No. 1359

OSHA's current Z Tables have no specific limit for silicon; however, silicon is currently regulated under OSHA's nuisance dust limit of 15 mg/m³ TWA. The ACGIH recommends a 10-mg/m³ 8-hour TWA for silicon, measured as total dust. Silicon is a black to gray, lustrous, needle-like crystal, which is used in the manufacture of semiconductors.

An early study by McCord, Fredrick, and Stolz (1937) reported no response in guinea pigs and rats injected intraperitoneally with silicon. A more

recent study (Schepers 1971) demonstrated pulmonary lesions in rabbits administered an intratracheal dose of 25 mg silicon dust.

OSHA is proposing an 8-hour TWA limit of 10 mg/m³ for silicon, the limit being proposed for all inert particulates. The Agency preliminarily concludes that this limit will reduce the safety and health risks potentially associated with exposure to this substance at the currently unregulated level. These risks include interference with vision, accidents, and eye and skin irritation. The health evidence forms a reasonable basis for proposing a new limit for silicon. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

SILICON CARBIDE
CAS: 409–21–2; Chemical Formula: SiC
H.S. No. 1360

OSHA currently regulates silicon carbide under its 15-mg/m³ nuisance dust limit. The ACGIH recommends 10 mg/m³ as an 8-hour TWA, measured as total dust. Silicon carbide is a green to blue-black irridescent crystal.

An animal study (Gardner 1923) showed that exposure to silicon carbide alone produced no changes in the lungs, while exposure of guinea pigs infected with tuberculosis to silicon carbide (6 hours/day, 5 days/week for one year) aggravated pulmonary tuberculosis to the extent that extensive fibrosis occurred. Guinea pigs exposed to silicon carbide dust and infected with the tubercle bacteria developed tuberculopneumoconiotic lesions (Gross, Westrick, and McNerney 1959). Miller and Sayers (1941) observed that intraperitoneal injection of guinea pigs produced no reaction.

Bruusgaard (1945) found that X-rays of 10 out of 32 workers exposed to average levels of 34 mppcf of silicon carbide for 15 years or more demonstrated pulmonary changes: these 10 workers also were tuberculin-positive. Miller, Davis, Goldman, and Wyatt (1953) described three cases of pulmonary reactions and hyperglobinemia in tungsten carbide industry workers; these authors concluded that exposure to silicon carbide was not a hazard unless the exposed workers already had pulmonary tuberculosis.

OSHA is proposing a 10-mg/m³ TWA limit for silicon carbide, as total dust. The Agency preliminarily concludes that this limit will protect exposed workers from the risk of safety and health effects potentially associated with exposure to this inert particulate. The health evidence forms a reasonable basis for proposing a new limit for silicon carbide. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

STARCH, TOTAL DUST
CAS: 9005–25–8; Chemical Formula: $(C_6H_{10}O_5)_n$
H.S. No. 1369

The current OSHA limit for starch is 15 mg/m³ as an 8-hour TWA, the Agency's limit for all inert particulates. The ACGIH recommends a TLV–TWA of 10 mg/m³ for starch as total dust that contains no asbestos and less than 1 percent crystalline silica. Starch is a white odorless powder.

Exposure to high concentrations of dust may result in impaired vision, or may cause injury to mucous membranes or skin. Injury may also result from vigorous skin-cleansing procedures necessary for the complete removal of starch (ACGIH 1986).

OSHA is proposing an 8-hour TWA of 10 mg/m³ for starch. The Agency preliminarily concludes that this limit will reduce the risk of safety accidents, eye and skin irritation, and interference with concentration and vision that may result from exposure to high levels of inert dusts in the workplace. The health evidence forms a reasonable basis for proposing a new limit for starch. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

SUCROSE, TOTAL DUST
CAS: 57–50–1; Chemical Formula: $C_{12}H_{22}O_{11}$
H.S. No. 1374

The current OSHA 8-hour TWA limit for sucrose is 15 mg/m³ as total dust, the Agency's limit for all nuisance dusts. The ACGIH includes sucrose in its grouping of nuisance particulates that "do not produce significant organic disease or toxic effect when exposures are kept under reasonable control" (1986), and has therefore established a TLV–TWA limit of 10 mg/m³ for sucrose as total dust containing no asbestos and less than 1 percent quartz. Sucrose is found in the form of white crystals.

Exposure to excess levels of sucrose dust can cause skin and eye irritation, interference with vision, and distraction from the task at hand.

OSHA is proposing an 8-hour TWA of 10 mg/m³ for sucrose dust, the limit being proposed for all of the inert particulates. The Agency preliminarily concludes that this limit will protect exposed workers against the risk of the health and safety hazards described above. The health evidence forms a reasonable basis for proposing a new limit for sucrose dust. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

TEMEPHOS
CAS: 3383–96–8; Chemical Formula: $C_{16}H_{20}O_6P_2S_3$
H.S. No. 1383

The current OSHA Z tables have no specific limit for exposure to temephos, a cholinesterase-inhibiting insecticide. Temephos is currently regulated under OSHA's nuisance dust limit of 15 mg/m³. The ACGIH limit is 10 mg/m³ as an 8-hour TWA. Temephos may be a white crystalline solid or a viscous brown liquid.

In rats and mice, temephos has an acute oral LD_{50} of 400 mg/kg or greater. Various animal species tolerated doses of 10 mg/kg without clinical effect and 1 mg/kg without effect on cholinesterase activity (Gaines, Kimbrough, and Laws 1967). Laws et al. (1967) revealed that human volunteers consuming oral doses of temephos at levels of 256 mg/man/day for 5 days, or 64 mg/man/day for 4 weeks, evidenced no detectable effects on erythrocyte or plasma cholinesterase levels. Murphy and Cheever (1972) reported that 1 mg of temephos per liter of drinking water produces no effect. These authors found that rat liver carboxylesterases were at least 30 times more sensitive to inhibition from temephos than cholinesterases. Assuming that human liver carboxylesterases are proportionately more sensitive than cholinesterases, it is estimated that significant inhibition of these carboxylesterases could occur as a result of consuming 2 liters of drinking water containing 1 mg/L of temephos. Although nonspecific liver carboxylesterase is not critical for normal physiologic function, adverse effects on this enzyme could increase the susceptibility of exposed individuals to chemicals and drugs that contain carboxylesterase linkages (ACGIH 1986, p. 557).

The ACGIH derived the limit of 10 mg/m³ TWA for temephos from studies of malathion, which has an acute LD_{50} of 2100 mg/kg in rats, or roughly one-half that of temephos. Because humans tolerate 16 mg/day oral doses of malathion without effects on blood cholinesterase levels, the ACGIH believes the 10 mg/m³ limit is appropriate for temephos (ACGIH 1986, p. 557).

OSHA is proposing a limit of 10 mg/m³ for temephos. The Agency preliminarily concludes that this limit will protect exposed workers from the risk of cholinesterase inhibition and reduction in carboxylesterase activity

potentially associated with exposure to this substance. This health evidence forms a reasonable basis for proposing a new limit for temephos. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

4,4'-THIOBIS (6-TERT-BUTYL-n-CRESOL)
CAS: 96-69-5; Chemical Formula: $C_{22}H_{30}O_2S$
H.S. No. 1391

OSHA currently regulates 4,4'-thiobis under its general nuisance dust limit of 15 mg/m³ TWA. The ACGIH limit is 10 mg/m³ as an 8-hour TWA, the limit established by the ACGIH for all of the inert dusts. 4,4'-Thiobis is a light gray to tan powder with a slightly aromatic odor.

In a 30-day study, rats fed diets of 500 ppm 4,4'-thiobis exhibited normal weight gain; those rats fed five times this amount exhibited enlarged livers and a reduced rate of weight gain (Lefaux 1968, as cited in ACGIH 1986, p. 570). In a 90-day study reported by the same author, rats fed 50 ppm showed no toxic effects, but male rats fed 500 ppm ate and grew at a slightly lower rate. No pathologic changes were observed in the 500 ppm-dosed rats. A dose of 5 g/kg of 4,4'-thiobis proved lethal to rats, with the predominant symptom being gastroenteritis.

OSHA is proposing an exposure limit of 10 mg/m³ as an 8-hour TWA for 4,4'-thiobis. The Agency preliminarily concludes that this limit will protect exposed workers from the risks to safety and health potentially posed by workplace exposures to this and other nuisance dusts. These risks include distraction and interference with vision, which may cause safety accidents, and eye and skin irritation. The health evidence forms a reasonable basis for proposing a new limit for 4,4'-thiobis. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

TITANIUM DIOXIDE
CAS: 13463-67-7; Chemical Formula: TiO_2
H.S. No. 1396

OSHA's existing PEL for titanium dioxide is 15 mg/m³ as an 8-hour TWA; this is the Agency's current limit for inert particulates. A 10-mg/m³ 8-hour TWA, measured as total dust, has been established by the ACGIH. Titanium dioxide is a white crystalline solid.

Miller and Sayers (1941) reported that intraperitoneal injections of titanium dioxide in guinea pigs showed a tendency to remain in the injected tissues but not to produce a proliferative response; these authors consider it an inert dust. A study by Grandjean (1956) in which rats were administered 50 mg of titanium dioxide intratracheally showed pigmented dust deposits in the lungs. In addition, evidence of infection appeared in the alveoli of one rat and diffuse fibrosis was found in the lungs of a separate test animal. No nodule formation was observed (Grandjean et al. 1956). Another study by Dale (1973) revealed thickening of the walls of the alveoli in the lungs of rabbits injected with titanium dioxide dust; however, lungs had returned to normal 3 months post-treatment. Feeding studies of rats and mice at doses of 2.5 percent or 5 percent titanium dioxide for 103 weeks revealed no signs of carcinogenicity in either species (National Cancer Institute 1978). From these data, the ACGIH determined that there exists "no evidence of danger to health from the inhalation of titanium dioxide dust in concentrations of air that do not exceed 10 mg/m³ total dust containing less than 1 percent quartz" (1986).

OSHA proposes an 8-hour TWA of 10 mg/m³, the limit being proposed for all inert particulates. The Agency preliminarily concludes that this limit will protect workers from the health and safety risks potentially associated with exposure to airborne particulates at higher levels. These risks include accidents, interference with vision, and eye and skin irritation. The health evidence forms a reasonable basis for proposing a new limit for titanium dioxide. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

VEGETABLE OIL MIST (EXCEPT CASTOR OIL, CASHEW NUT, OR SIMILAR IRRITANT OILS)
CAS: 8008-89-7; Formula: None
H.S. No. 1423

Vegetable oil is a pale-yellow oily liquid. The current OSHA standard for vegetable oil mist is 15 mg/m³, the limit applying to all inert dusts at present. The ACGIH has established a 10-mg/m³ 8-hour TWA for all nuisance dusts.

Occupational exposure to this group of dusts is associated with a variety of health and safety hazards. For example, these substances interfere with vision; cause coughing, eye tearing, and irritation; and, by distracting affected employees from the task at hand, can lead to on-the-job accidents and injuries. In addition, the vigorous cleansing necessary to remove the oil mist from the skin may cause skin irritation.

OSHA is proposing to reduce the existing limit for dusts in this category to 10 mg/m³ TWA. The Agency preliminarily concludes that this limit will avoid the risks described above, which include both safety and health risks to exposed employees. The health evidence forms a reasonable basis for proposing a new limit for vegetable oil. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

ZINC STEARATE
CAS: 557-01-1; Chemical Formula: $Zn(C_{18}H_{35}O_2)_2$
H.S. No. 1434

OSHA currently regulates zinc stearate as a nuisance dust with a limit of 15 mg/m³ 3 TWA, total dust. The ACGIH has established an 8-hour TWA of 10 mg/m³ for zinc stearate, measured as total dust. Zinc stearate is a white powder.

A report in *Folia Medica* (1957) documented the case of a worker exposed to zinc stearate dust for 30 years who died from extensive fibrosis of the lungs. More recent studies have revealed incidences of pulmonary fibrosis associated with encephalopathy that stemmed directly from exposure to aluminum dust, which is frequently coated with stearic acid (*British Journal of Industrial Medicine* 1962); the ACGIH (1986, p. 646) is uncertain of the relevance of this report to zinc stearate exposures.

Observations of long-term workers exposed to this dust in the rubber industry revealed no adverse effects of exposure (B.F. Goodrich Rubber Company, private communication, as cited in the ACGIH 1986, p. 646). The ACGIH considers zinc stearate dust to be biologically inert and has assigned a nuisance dust limit to this substance.

OSHA is proposing a 10-mg/m³ limit for this dust (measured as total dust) because the Agency believes that all of its nuisance dust limits should be decreased to 10 mg/m³ as 8-hour TWAs. The Agency believes that this limit will prevent the safety and health risks associated with high workplace exposures to these dusts. These risks include accidents, interference with vision, and eye and skin irritation. The health evidence forms a reasonable basis for proposing a new limit for zinc stearate. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

ZINC OXIDE, TOTAL DUST
CAS: 1314-13-2; Chemical Formula: ZnO
H.S. No. 1438

Zinc oxide dust is a white or pale-yellow powder. OSHA currently has no exposure limit specifically for zinc oxide as total dust. The ACGIH established a limit of 10 mg/m³ as an 8-hour TWA for zinc oxide, measured as total dust.

According to Turken and Thompson (1926), exposure to finely divided zinc oxide dust can produce effects similar to those for metal fume fever. Beeckmans and Brown (1963) reported that catalytically active zinc oxide dust is more toxic when treated with ultraviolet light. Aside from these considerations, the ACGIH considered zinc oxide dust to be a nuisance dust.

OSHA is proposing a limit of 10 mg/m³ for zinc oxide as total dust, the same as for all nuisance dusts. The Agency preliminarily concludes that this limit will protect exposed individuals from the risk of exposure to this dust in the workplace, which can result in safety and health hazards such as accidents, skin and respiratory irritation, and interference with vision. The health evidence forms a reasonable basis for proposing a new limit for zinc oxide. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

Preliminary Conclusions

OSHA's Current 8-hour inert or nuisance dust standard (29 CFR 1910.1000, Table Z–3) was adopted from the 1968 ACGIH TLV–TWA of 15 mg/m³ for total dust and 5 mg/m³ for respirable dust. At the time, the ACGIH considered the 15 mg/m³ value to be "an acceptable limit of good hygienic practice" based on the then prevailing "lack of knowledge" of any adverse effects at exposure levels below this value (ACGIH Documentation 1966). Shortly after OSHA adopted the ACGIH's 1968 limit, the ACGIH revised its limit downward to 10 mg/m³ for total dust and 5 mg/m³ for respirable dust. In justifying this reduction, the ACGIH noted that the lower levels would "result in appreciable improvement of working conditions in plants where the old limit of 15 mg/m³ formerly prevailed" (ACGIH 1971, p. 190).

OSHA preliminarily concludes that the proposed TWA limit of 10 mg/m³ for this group of substances that cause a variety of adverse effects will substantially reduce the risk of upper respiratory tract, eye, and skin irritation and of danger to the safety of workers distracted by the presence of these substances in the workplace. The health evidence forms a reasonable basis for proposing revised or new limits for nuisance dusts. OSHA will establish new limits for the nuisance dusts if the Agency determines that these limits will substantially reduce significant risks.

11. Substances for Which Limits Are Based on Avoidance of Odor and Taste Effects

Introduction

This category includes four substances that have obnoxious odors. The Agency recognizes that working in atmospheres containing detectable concentrations of these substances will endanger workers by distracting them from the task at hand and creating safety hazards. For three of these substances, OSHA is retaining its current 8-hour TWAs. For one substance, propylene glycol monomethyl ether, a new limit is being proposed. OSHA is proposing the retention or adoption of these limits based on the data described below, which show a NOE level for these intolerable taste and odor effects. Table C11–1 shows the substances included in this group and their OSHA and ACGIH limits, as well as their CAS and HS numbers.

Description of the Health Effects

The substances in this group have obnoxious odors and cannot willingly be tolerated by most workers for any extended period of time. Because odor detection occurs at very low concentrations for many of these chemicals, the proposed limit has been set at a level below the concentration at which the odor is intolerable to employees.

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TABLE C11-1. Substances for Which Limits Are Based on Avoidance of Odor and Taste Effects

Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1226 Isopropyl ether+	108-20-3	500 ppm TWA	250 ppm TWA 310 ppm STEL	—
1314 Phenyl ether (vapor)+	101-84-8	1 ppm TWA	1 ppm TWA 2 ppm STEL	—
1343 Propylene glycol monomethyl ether	107-98-2	—	100 ppm TWA 150 ppm STEL	—
1427 Vinyl toluene+	25013-15-4	100 ppm TWA	50 ppm TWA 100 ppm STEL	—

* OSHA's TWA limits are for 8-hour exposures; its STELs are for the durations specified; and its ceilings are peaks not to be exceeded for any period of time.

** The ACGIH TWA-TLV is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

*** NIOSH TWA limits are for 10-hour exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

+ The existing OSHA limit for this substance is being retained.

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The evidence for each of the substances in this group describes their adverse effects both in animals and humans. These effects, which range from nausea to narcosis, generally occur at levels higher than the limits for these substances, which are based not only on toxicological effects but also on the basis of the intolerable odors associated with their presence in workroom air. Because odor effects range in severity from distracting to intolerable, these limits have been set at the concentration at which the odor becomes so seriously objectionable as to create a risk of safety hazards.

Dose-Response Relationships and Odor Effects

Odor effects are threshold effects, although there is wide variation in individual odor response, i.e., in the ability to detect odor. Because of this wide variation and the phenomenon of olfactory fatigue, odor is not a reliable indicator of airborne concentrations and should not be relied on to provide a warning of overexposure. The following paragraphs describe OSHA's preliminary findings with respect to the substances in this group.

ISOPROPYL ETHER
CAS: 108-20-3; Chemical Formula: $(CH_3)_2CHOCH(CH_3)_2$
H.S. No. 1226

OSHA currently has a limit of 500 ppm TWA for isopropyl ether. The ACGIH recommends a TLV-TWA of 250 ppm and a TLV-STEL of 310 ppm for this liquid, which has a sharp, sweet odor similar to that of ether.

Animal studies have shown that exposures to high concentrations cause narcosis and death (Machle, Scott, and Treon 1939). Twenty exposures at a 1-percent vapor concentration produced intoxication and depression but no significant blood or weight changes. In rabbits, the minimum lethal dose has been reported to be 5 to 6.5 g/kg. The liquid is an irritant to the skin and mucous membranes and causes dermatitis in rabbits on repeated exposure (Machle, Scott, and Treon 1939).

Humans exposed for 15 minutes at 300 ppm experienced no irritation but complained about the objectionable odor of isopropyl ether; eye and nose irritation was experienced as a result of 5-minute exposures to 800 ppm. A 15-minute exposure to 500 ppm was not reported by subjects to be irritating (Silverman, Schulte, and First 1946).

The available health evidence for this substance may not be sufficient to support a revision of the current limit, and OSHA is accordingly retaining its current limit. However, there may be other health effects information on isopropyl ether, and OSHA is specifically requesting any available information from the public to support lower limits for this substance. The public may also wish to address the topic of material impairment of health, as discussed in section 6(b) of the Act.

PHENYL ETHER
CAS: 101-84-8; Chemical Formula: $(C_6H_5)_2O$
H.S. No. 1314

OSHA currently has an 8-hour TWA limit of 1 ppm for phenyl ether. The ACGIH recommends the same TWA and the addition of a 2-ppm 15-minute STEL for phenyl ether vapor. Phenyl ether is a colorless liquid or solid with a low volatility; its vapor has a disagreeable odor.

The acute oral lethal dose is approximately 4 g/kg for rats and guinea pigs, and single doses of between 1 and 2 g/kg administered to various species have shown no liver, spleen, kidney, thyroidal or gastrointestinal toxicities in surviving animals (Vogel, Snyder, and Schulman 1964). Repeated inhalation studies in rats, rabbits, and dogs have shown that 20 exposures to 4.9 ppm for 5 days per week, 7 hours per day produced no adverse effects. Eye and nasal irritation were observed in rats and rabbits exposed at 10 ppm (Hefner, Leong, Kociba, and Gehring 1975). Skin and eye irritation have been reported only as a result of prolonged undiluted exposures. There is no evidence that skin absorption presents a health hazard (ACGIH 1986, p. 475).

The primary complaints associated with human exposures to phenyl ether vapor are of disagreeable odor and occasional nausea (Hake and Rowe, as cited in Patty 1963, p. 1698).

The available health evidence for this substance may not be sufficient to support a revision of this current limit, and OSHA is therefore retaining its limit for phenyl ether. However, there may be other health effects information on phenyl ether, and OSHA is specifically requesting any available information from the public to support lower limits for this substance. The public may also wish to comment on the topic of material impairment of health, as discussed in section 6(b) of the Act.

PROPYLENE GLYCOL MONOMETHYL ETHER
CAS: 107-98-2; Chemical Formula: $CH_3OCH_2CHOHCH_3$
H.S. No. 1343

OSHA has no current standard for propylene glycol monomethyl ether (PGME). The ACGIH recommends a TWA of 100-ppm and a STEL of 150 ppm; NIOSH has no REL.

Exposure to propylene glycol monomethyl ether causes anesthesia at a level of approximately 1000 ppm, eye tearing at levels above 100 ppm, and an objectionable odor at 100 ppm (Stewart, Baretta, Dodd, and Torkelson 1970). Ingestion of 3 g/kg in a 35-day period caused minor changes in the livers and kidneys of rats, and repeated dermal applications of 7 to 10 ml/kg/day caused death in rats treated over a 90-day period (Rowe, McCollister, Spencer et al. 1954).

The proposed PELs for PGME of 100 ppm TWA and 150 ppm STEL are designed to protect workers from experiencing this objectionable effect, which is seriously distracting. In addition, the proposed levels will ensure that workers will not experience the eye irritation reported to be associated with exposures to propylene glycol monomethyl ether at levels above 100 ppm. OSHA preliminarily finds that the proposed limits will reduce the risks potentially associated with previously uncontrolled exposures to this substance. The health evidence forms a reasonable basis for proposing a new limit for propylene glycol monomethyl ether.

VINYL TOLUENE
CAS: 25013-15-4; Chemical Formula: $CH_3C_6H_4CH=CH_2$
H.S. No. 1427

The current OSHA standard for vinyl toluene is 100 ppm as an 8-hour TWA. The ACGIH recommends a TWA of 50 ppm with a 100-ppm short-term exposure limit. Vinyl toluene is a colorless liquid with a strong, disagreeable odor.

Wolf, Rowe, McCollister et al. (1956) noted fatty degeneration of the liver and an increase in kidney and liver weights in rats, guinea pigs, rabbits, and monkeys exposed to approximately 100 7- to 8-hour exposures of vinyl toluene at 1250 ppm. Some deaths occurred among the rats in this group. Animals exposed to vinyl toluene at 600 ppm appeared normal and showed no blood or urine abnormalities, no gross or microscopic tissue changes, and no changes in growth rate or organ weight (Wolf, Rowe, McCollister et al. 1956).

Human volunteers reported eye and nose irritation at 400 ppm, and objectionable odor at 300 ppm. At 50 ppm, the odor of vinyl toluene was detectable, but no irritation was experienced (ACGIH 1986, p. 630).

The available health evidence for this substance may not be sufficient to support a revision of the current limit, and OSHA is accordingly retaining its limit. However, there may be other

health effects information on vinyl toluene, and OSHA is specifically requesting any available information on vinyl toluene from the public to support lowering the limit for this substance. The public may also wish to comment on the topic of material impairment of health, as discussed in section 6(b) of the Act.

Preliminary Conclusions

For the one chemical in this group for which OSHA is proposing a new limit, the Agency preliminarily finds that exposure at the uncontrolled levels currently permitted places workers at risk of experiencing the adverse effects associated with this noxious substance. These effects include irritation, distraction, discomfort, and, if exposure is sufficiently severe, danger to affected employees and their co-workers. This risk is created by the safety risks generated by propylene glycol monomethyl ether's intolerable odor.

For the three substances—isopropyl ether, phenyl ether, and vinyl toluene—for which OSHA is at present retaining its current limits, the available health evidence may not be sufficient to support a revision of these limits. However, OSHA is soliciting additional information from the public on the health effects associated with occupational exposure to these substances to support revision or retention of these limits. At the time of the final rule, OSHA will make a final determination, based on the best available evidence, of whether to retain or revise the limits for these substances.

12. Substances for Which Proposed Limits are Based on Avoidance of Adverse Health Effects Caused by Exposure to Analogous Substances

Introduction

OSHA is proposing limits for 73 substances on the basis of their toxicologic and structural similarities to other chemical substances that create significant risks of systemic toxicity, ocular effects, kidney or liver damage, and other similarly adverse health effects. For 46 of these substances, OSHA has not previously had Z table limits. For an additional 12 substances, OSHA is proposing to reduce the 8-hour TWA, and in 13 cases, the Agency is proposing to retain its 8-hour limit and to add a STEL to supplement the TWA. OSHA proposes to delete the 8-hour limit and add a ceiling in the case of acetic anhydride and to delete a ceiling limit and add an 8-hour TWA for another substance. Table C12–1 shows these substances, their CAS and HS numbers, and their current OSHA and ACGIH limits. NIOSH has RELs for four substances in this category.

Description of the Health Effects

The health effects associated with occupational exposures to the diverse group of substances shown in Table C12–1 vary widely, ranging from sensory irritation, systemic toxicity, ocular effects, and neuropathy to renal and liver damage. This variation in target organs reflects the fact that the substances in this group have not been grouped on the basis of their toxic effects or mechanism of action; instead, they are considered together because the specific limits proposed for them have been determined on the basis of toxic effects caused by exposure to analogous chemicals. Table C12–2 shows these substances, along with their adverse health effects and analogous compounds.

The use of analogy is a reasonable basis for making estimates because of the similarities in structure and activity of these substances. Industrial hygienists frequently use this approach. The limits for these chemicals have thus been set based on dose-response information for other compounds that are of similar chemical structure or that have a similar mechanism of action. For example, limits are being proposed for a number of compounds that are known cholinesterase inhibitors (including diazinon, disulfoton, and monocrotophos); since direct dose-response data are not available, OSHA has proposed limits that are similar to the proposed limit for parathion, another cholinesterase inhibitor for which adequate dose-response data are available.

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TABLE C12-1. Substances for Which Limits Are Based on Analogy to Related Compounds

H.S. Number/ Chemical Name	CAS No.	CURRENT PEL*	ACGIH TLV**	NIOSH REL
1003 Acetic anhydride	108-24-7	5 ppm TWA	5 ppm Ceiling	--
1009 Acrylic acid	79-10-7	--	10 ppm TWA	--
1015 Aluminum (alkyls)	7429-90-5	--	2 mg/m^3 TWA	--
1018 Aluminum (soluble salts)	7429-90-5	--	2 mg/m^3 TWA	--
1040 Boron tribromide	10294-33-4	--	1 ppm Ceiling	--
1043 Bromine pentafluoride	7789-30-2	-	0.1 ppm TWA	--
1048 n-Butyl acrylate	141-32-2	--	10 ppm TWA	--
1055 o-sec-Butylphenol	89-72-5	-	5 ppm TWA, Skin	--
1059 Calcium hydroxide	1305-62-0	-	5 mg/m^3 TWA	--
1060 Calcium oxide	1305-78-8	5 mg/m^3 TWA	2 mg/m^3 TWA	--
1074 Carbonyl fluoride	353-50-4	-	2 ppm TWA 5 ppm STEL	--
1075 Catechol	120-80-9	--	5 ppm TWA	--
1081 1-Chloro-1-nitro-propane	600-25-9	20 ppm TWA	2 ppm TWA	--

H.S. Number/ Chemical Name	CAS No.	CURRENT PEL*	ACGIH TLV**	NIOSH REL
1098 Cobalt carbonyl	10210-68-1	-	0.1 mg/m^3 TWA	--
1099 Cobalt hydrocarbonyl	16842-03-8	-	0.1 mg/m^3 TWA	--
1118 Diazinon	333-41-5	-	0.1 mg/m^3 TWA, Skin	-
1121 1,1-Dichloro-1-nitro-ethane	594-72-9	10 ppm Ceiling	2 ppm TWA	--
1125 p-Dichlorobenzene	106-46-7	75 ppm TWA	75 ppm TWA 110 ppm STEL	--
1128 Dichloromono-fluoromethane	75-43-4	1000 ppm TWA	10 ppm TWA	--
1135 Diethyl ketone	96-22-0	-	200 ppm TWA	--
1138 Diethylenetriamine	111-40-0	-	1 ppm TWA, Skin	--
1148 Dipropyl ketone	123-19-3	-	50 ppm TWA	-
1150 Diquat	85-00-7	-	0.5 mg/m^3 TWA	--
1152 Disulfoton	298-04-4	-	0.1 mg/m^3 TWA	--

TABLE C12-1. Substances for Which Limits Are Based on Analogy to Related Compounds (continued)

H.S. Number/ Chemical Name	CAS No.	CURRENT PEL*	ACGIH TLV**	NIOSH REL
1154 Divinyl benzene	108-57-6	-	10 ppm TWA	--
1156 Endosulfan	115-29-7	--	0.1 mg/m^3 TWA, Skin	--
1181 Fonofos	944-22-9	-	0.1 mg/m^3 TWA, Skin	--
1182 Formamide	75-12-7	-	20 ppm TWA 30 ppm STEL	--
1186 Germanium tetra-hydride	7782-65-2	--	0.2 ppm TWA	--
1212 Indene	95-13-6	-	10 ppm TWA	--
1214 Iodoform	75-47-8	--	0.6 ppm TWA	--
1219 Isobutyl alcohol	78-83-1	100 ppm TWA	50 ppm TWA	--
1220 Isooctyl alcohol	26952-21-6	-	50 ppm TWA, Skin	--
1229 n-Isopropylaniline	643-28-7	-	2 ppm TWA, Skin	--
1231 Ketene	463-51-4	0.5 ppm TWA	0.5 ppm TWA 1.5 ppm STEL	--
1244 Methacrylic acid	79-41-4	-	20 ppm TWA	--

H.S. Number/ Chemical Name	CAS No.	CURRENT PEL*	ACGIH TLV**	NIOSH REL
1247 4-Methoxyphenol	150-76-5	--	5 mg/m^3 TWA	--
1250 Methyl acetylene-propadiene mixture	74-99-7	1000 ppm TWA	1000 ppm TWA 1250 ppm STEL	--
1256 Methyl demeton	8022-00-2	--	0.5 mg/m^3 TWA, Skin	--
1257 Methyl ethyl ketone peroxide	1338-23-4	--	0.2 ppm Ceiling	--
1258 Methyl formate	107-31-3	100 ppm TWA	100 ppm TWA 150 ppm STEL	--
1259 Methyl iodide	74-88-4	5 ppm TWA, Skin	2 ppm TWA, Skin	Lowest feasible limit
1260 Methyl isoamyl ketone	110-12-3	--	50 ppm TWA	50 ppm TWA
1262 Methyl isopropyl ketone	563-80-4	--	200 ppm TWA	--
1265 Methyl parathion	298-00-0	--	0.2 mg/m^3 TWA, Skin	0.2 mg/m^3 TWA

TABLE C12-1. Substances for Which Limits Are Based on Analogy to Related Compounds (continued)

H.S. Number/ Chemical Name	CAS No.	CURRENT PEL*	ACGIH TLV**	NIOSH REL
1268 Methylcyclohexane	108-87-2	500 ppm TWA	400 ppm TWA	--
1271 Methylcyclopentadienyl Mn tricarbonyl	12108-13-3	--	0.2 mg/m^3 TWA, Skin	--
1279 Monocrotophos	6923-22-4	--	0.25 mg/m^3 TWA	--
1281 Morpholine	110-91-8	20 ppm TWA, Skin	20 ppm TWA 30 ppm STEL, Skin	--
1286 Nitric acid	7697-37-2	2 ppm TWA	2 ppm TWA 4 ppm STEL	2 ppm TWA
1287 p-Nitroaniline	100-01-6	6 mg/m^3 TWA, Skin	3 mg/m^3 TWA, Skin	--
1292 Nitrotoluene	1321-12-6	5 ppm TWA, Skin	2 ppm TWA, Skin	--
1293 Nonane	111-84-2	--	200 ppm TWA	--
1299 Oxalic acid	144-62-7	1 mg/m^3 TWA	1 mg/m^3 TWA 2 mg/m^3 STEL	--
1309 Perchloryl fluoride	7616-94-6	3 ppm TWA	3 ppm TWA 6 ppm STEL	--

H.S. Number/ Chemical Name	CAS No.	CURRENT PEL*	ACGIH TLV**	NIOSH REL
1320 Mevinphos (Phosdrin)	7786-34-7	0.1 mg/m^3 TWA, Skin	0.1 mg/m^3 TWA 0.3 mg/m^3 STEL, Skin	--
1323 Phosphorus oxychloride	10025-87-3	--	0.1 ppm TWA 0.5 ppm STEL	--
1324 Phosphorus pentasulfide	1314-80-3	1 mg/m^3 TWA	1 mg/m^3 TWA 3 mg/m^3 STEL	--
1326 Phthalic anhydride	85-44-9	2 ppm TWA	1 ppm TWA	--
1335 Propargyl alcohol	107-19-7	--	1 ppm TWA, Skin	--
1336 Propionic acid	79-09-4	--	10 ppm TWA 15 ppm STEL	--
1338 n-Propyl acetate	109-60-4	200 ppm TWA	200 ppm TWA 250 ppm STEL	--
1339 n-Propyl alcohol	71-23-8	200 ppm TWA	200 ppm TWA 250 ppm STEL, Skin	--
1344 Propylene oxide	75-56-9	100 ppm TWA	20 ppm TWA	--

H.S. Number/ Chemical Name	CAS No.	CURRENT PEL*	ACGIH TLV**	NIOSH REL
1361 Silicon tetrahydride	7803-62-5	-	5 ppm TWA	--
1379 Sulfuryl fluoride	2699-79-8	5 ppm TWA	5 ppm TWA 10 ppm STEL	--
1393 Thionyl chloride	7719-09-7	--	1 ppm Ceiling	--
1402 Tributyl phosphate	126-73-8	5 mg/m^3 TWA	2.5 mg/m^3 TWA	--
1404 Trichloroacetic acid	76-03-9	-	1 ppm TWA	-
1411 Trimethylamine	75-50-3	-	10 ppm TWA 15 ppm STEL	-
1420 n-Valeraldehyde	110-62-3	-	50 ppm TWA	-
1432 m-Xylene-alpha, alpha', diamine	1477-55-0	-	0.1 mg/m^3 Ceiling, Skin	--
1433 Xylidine	1300-73-8	5 ppm TWA, Skin	2 ppm TWA, Skin	--

* OSHA's TWA limits are for 8-hour exposures; its STELs are for the durations specified; and its ceilings are peaks not to be exceeded for any period of time.

** The ACGIH TWA-TLV is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

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It is important to note that the establishment of a limit on the basis of analogy to other substances does not reflect a lack of information showing that the substance is toxic; acute animal data are available demonstrating the toxicity of all of the substances for which limits are being proposed in this category, and cases of human poisoning caused by exposure to many of them are reported. Thus the proposed limits reflect more than theoretical considerations of chemical structure and physiologic reaction: The hazardous nature of exposure has been demonstrated beyond doubt, although the precise level at which it will occur cannot be foretold with certainty.

The following sections describe OSHA's preliminary findings for the substances in this grouping.

TABLE C12-2.—SUMMARY OF RATIONALE FOR LIMITS BASED ON ANALOGY TO RELATED COMPOUNDS

H.S. No./Chemical name	Compound on which limit is based	Associated health effects
1003 Acetic anhydride	Acetic acid	Sensory irritation.
1009 Acrylic acid	Acetic acid	Sensory irritation.
1015 Aluminum (alkyls)	Welding fumes	Respiratory irritation.
1018 Aluminum (soluble salts)	Hydrolysis to hydrogen chloride.	Sensory irritation.
1040 Boron tribromide	Hydrogen bromide	Sensory irritation.
1043 Bromine pentafluoride	Chlorine terifluoride	Systemic injury.
1048 n-Butyl acrylate	Methyl acrylate	Acute toxicity.
1055 o-sec-Butylphenol	Phenol and cresol	Respiratory, liver, and kidney effects.
1059 Calcium hydroxide	Sodium hydroxide	Sensory irritation.
1060 Calcium oxide	Sodium hydroxide	Sensory irritation.
1074 Carbonyl fluoride	Hydrolysis to hydrogen fluoride.	Sensory irritation.
1075 Catechol	Phenol	Peripheral vasoconstriction, renal tubule degeneration.
1081 1-Chloro-1-nitropropane	Nitropropane	Acute toxicity, damage to heart muscle, liver, and kidneys.
1098 Cobalt carbonyl	Nickel carbonyl	Systemic toxicity.
1099 Cobalt hydrocarbonyl	Nickel carbonyl	Systemic toxicity.
1118 Diazinon	Parathion	Cholinesterase inhibition.
1121 1,1-Dichloro-1-nitroethane	Related compounds	Systemic toxicity.
1125 p-Dichlorobenzene	o-Dichlorobenzene	Neurological effects, cataract formation.
1128 Dichlorofluoromethane	Chloroform	Hepatotoxicity, cardiac sensitization.
1135 Diethyl ketone	Methyl propyl ketone	Acute toxicity.
1138 Diethylenetriamine	Ethylamine	Irritation, sensitization.
1148 Dipropyl ketone	Methyl isobutyl ketone	Acute toxicity.
1150 Diquat	Paraquat	Ocular effects.
1152 Disulfoton	Parathion	Cholinesterase inhibition.
1154 Divinyl benzene	Styrene	Sensory irritation.
1156 Endosulfan	Aldrin, Dieldrin	Neurological effects.
1181 Fonofos	Ethyl parathion	Cholinesterase inhibition.
1182 Formamide	Dimethyl formamide	Acute toxicity.
1186 Germanium tetrahydride	Stibine	Acute toxicity.
1212 Indene	Naphthalene	Sensory irritation.
1214 Iodoform	Methyl iodide	Acute toxicity.
1219 Isobutyl alcohol	n-Butanol	Acute toxicity.
1220 Isooctyl alcohol	Isoamyl alcohol	Sensory irritation.
1229 n-Isopropylaniline Aniline	N,N-dimethylaniline	Acute toxicity.
1231 Ketene	Phosgene	Sensory irritation.
1244 Methacrylic acid	Acrylic acid	Sensory irritation.
1247 4-Methoxyphenol	Hydroquinone	Ocular effects.
1250 Methyl acetylene-propadiene mixture	Methyl acetylene	Hygienic standard (absence of demonstrated health effects).
1256 Methyl demeton	Demeton	Ocular effects, respiratory effects, inner ear irritation.
1257 Methyl ethyl ketone peroxide	Benzoyl peroxide, hydrogen peroxide.	Sensory irritation.
1258 Methyl formate	Methyl acetate	Sensory irritation.
1259 Methyl iodide	Methyl bromide	CNS effects.
1260 Methyl isoamyl ketone	Methyl isobutyl ketone	Neuropathy.
1262 Methyl isopropyl ketone	Diethyl ketone, methyl propyl ketone.	Narcosis sensory irritation.
1265 Methyl parathion	Parathion	Cholinesterase inhibition.
1268 Methylcyclohexane	Heptane	Acute toxicity.
1271 Methylcyclopentadienyl manganese tricarbonyl	Tetraethyl lead	Central nervous system effects, chronic lung effects.
1279 Monocrotophos	Cholinesterase inhibitors	Cholinesterase inhibition.
1281 Morpholine	Ammonia	Kidney and liver degeneration, sensory irritation.
1286 Nitric acid	Hydrogen chloride, sulfuric acid.	Sensory irritation.
1287 p-Nitroaniline	Aniline	Methemoglobin formation.
1292 Nitrotoluene	Aniline	Methemoglobin formation.
1292 Nitrotoluene	Aniline	Methemoglobin formation.
1293 Nonane	Octane	Acute toxicity.
1299 Oxalic acid	Sulfuric acid, phosphoric acid.	Irritation, burns.
1309 Perchloryl fluoride	Fluoride	Fluorosis.
1320 Phosdrin (Mevinphos)	Parathion	Cholinesterase inhibition.
1323 Phosphorus oxychloride	Phosphorous trichloride	Sensory irritation, respiratory effects.
1334 Phosphorus pentasulfide	Hydrolysis to phosphoric acid.	Sensory irritation.
1326 Phthalic anhydride	Tetrachlorophthalic anhydride, maleic anhydride.	Sensory irritation.

TABLE C12-2.—SUMMARY OF RATIONALE FOR LIMITS BASED ON ANALOGY TO RELATED COMPOUNDS—Continued

H.S. No./Chemical name	Compound on which limit is based	Associated health effects
1335 Propargyl alcohol	Allyl alcohol	Acute toxicity.
1336 Propionic acid	Acetic acid	Sensory irritation.
1338 n-Propyl acetate	Isopropyl acetate, n-butyl acetate.	Sensory irritation.
1339 n-Propyl alcohol	Isopropyl alcohol	Sensory irritation.
1344 Propylene oxide	Ethylene oxide	Central nervous system depression, sensory irritation
1361 Silicon tetrahydride	Germane, stannane	Acute toxicity.
1379 Sulfuryl fluoride	Hydrogen fluoride	Fluorosis.
1393 Thionyl chloride	Hydrolysis to hydrogen chloride.	Sensory irritation.
1402 Tributyl phosphate	Triphenyl phosphate	Narcosis, cholinesterase inhibition.
1404 Trichloroacetic acid	2,2-Dichloropropionic acid.	Sensory irritation.
1411 Trimethylamine	Dimethylamine	Sensory irritation.
1420 n-Valeraldehyde	Saturated aliphatic	Sensory irritationaldehydes.
1432 m-Xylene-alpha, alpha', diamine	Phenylenediamine	Allergic respiratory sensitization.
1433 Xylidine	Aniline	Methemoglobin formation.

ACETIC ANHYDRIDE
CAS: 108-24-27; Chemical Formula: $(CH_3CO)_2O$
H.S. No. 1003

The current OSHA PEL for acetic anhydride is 5 ppm as an 8-hour TWA. The ACGIH has recommended a TLV of 5 ppm as a ceiling, based on analogy with acetic acid and the substance's irritant potential. Acetic anhydride is a colorless, mobile, strongly refractive liquid with a strong odor.

In one study, rats inhaling 1000 ppm of acetic anhydride for 4 hours survived, but 2000 ppm was fatal (Smyth 1956). In human studies, eye, nose, and throat irritation has been observed, and it has been suggested that bronchial and lung injury may occur as a consequence of exposure. (Henderson and Haggard 1943). Skin burns and serious corneal injury have been reported in industrial settings when workers came into contact with the liquid (McLaughlin 1946), and acetic anhydride is a marked lacrimator (Fairhall 1949).

In light of acetic anhydride's potential for acute toxicity, OSHA is proposing to replace the current 5-ppm 8-hour TWA with a 5-ppm ceiling. The Agency preliminarily concludes that this limit will protect workers from the risk of ocular and respiratory effects associated with high, short-term exposures to acetic anhydride at the current level. The proposed limit will substantially reduce this risk among industrially exposed workers. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for acetic anhydride if the Agency determines that this limit will substantially reduce significant risk.

ACRYLIC ACID
CAS: 79-10-7; Chemical Formula: $CH_2=CHCO_2H$
H.S. No. 1009

OSHA has no current permissible exposure limit for acrylic acid. The ACGIH has a recommended 8-hour TWA of 10 ppm. Acrylic acid is a colorless, corrosive liquid with a distinctive acrid odor.

Acrylic acid is known to polymerize explosively with amines, ammonia, oleum, and chlorosulfonic acid, and it is incompatible with strong alkalis and pure nitrogen. Occupational exposure to acrylic acid usually occurs when the chemical is used in the form of methyl, ethyl, or butyl esters in the manufacture of acrylic resins.

Data indicate that the oral LD_{50} in rats is between 0.25 and 0.5 mg/kg (Dow Chemical Company, 1977), and the skin absorption LD_{50} in rabbits is 0.95 ml/kg (Smyth et al. 1962). Another study indicates that rabbits given acrylic acid orally had no ill effects at a level of 0.025 mg/kg (Klimkina et al. 1969), and Gage reports that rats exposed to 80 ppm for 6 hours daily for 20 days showed no adverse effects (1970).

Case reports indicate that acute exposures to acrylic acid in workers have caused skin burns, eye burns, and upper respiratory effects (ACGIH, 1986, p. 14).

OSHA preliminarily concludes that an 8-hour TWA PEL of 10 ppm is necessary to protect exposed workers from the risk of nasal and eye irritation potentially associated with exposure to acrylic acid at the previously uncontrolled level. This limit will substantially reduce this risk and prevent recurrences of the burns and irritation previously associated with industrial exposures. The health evidence forms a reasonable basis for proposing a new limit for acrylic acid. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

ALUMINUM (ALKYLS)
CAS: 7429-90-5; Chemical Formula: Al
H.S. No. 1015

ALUMINUM (SOLUBLE SALTS)
CAS: 7429-90-5; Chemical Formula: Al
H.S. No. 1018

OSHA currently has no permissible exposure limits for the soluble salts of aluminum or for the aluminum alkyls. The ACGIH recommends an 8-hour TLV-TWA limit of 2 mg/m³ for aluminum (soluble salts) and 2 mg/m³ for the aluminum alkyls.

The ACGIH's limits for aluminum soluble salts have been set on the basis of the amount of hydrolized acid, such as hydrochloric acid or sulphuric acid, in their acid compounds. For example, three mols of hydrochloric acid (HCl) hydrolize from one mol of aluminum chloride; since HCl has a PEL of 5 ppm, a PEL of 2 mg/m³ for aluminum chloride, a soluble salt of aluminum, would provide the same degree of protection from irritation as that provided by this current limit for HCl. The acute toxicity of aluminum chloride is generally representative of the toxicity of all of the soluble salts of aluminum. For the aluminum alkyls, toxicity data are sparse. However, all of the nonhalogenated alkyls decompose into aluminum oxide fume, and the halogenated alkyls are even more irritating because of acid hydrolysis.

OSHA proposes an 8-hour TWA limit of 2 mg/m³ for the soluble salts and the alkyls of aluminum. The Agency preliminarily concludes that these limits will protect against the risk of irritation and skin burns presented by the acidic nature of these substances, to which workers can presently be exposed at uncontrolled levels. The health evidence

forms a reasonable basis for proposing new limits for the alkyls and aluminum salts. At the time of the final rule, OSHA will promulgate new limits if the Agency determines that these limits will substantially reduce significant risk.

BORON TRIBROMIDE
CAS: 10294-33-4; Chemical Formula: BBr_3
H.S. No. 1040

OSHA currently has no limit for exposure to boron tribromide. The ACGIH recommends a 1-ppm ceiling limit for boron tribromide, which is a colorless, fuming liquid that is decomposed by water and alcohol.

Boron tribromide has a high potential for acute local irritation, and its potential for systemic toxicity is analogous to that of hydrogen bromide (HBr). On decomposition, one molecule of boron tribromide would be expected to produce three molecules of HBr (ACGIH 1986, p. 62).

Animals repeatedly exposed to boron tribromide develop pneumonia, and exposure at 100 ppm caused a uniformly high mortality rate in six laboratory species (Stokinger, Spiegel et al. 1953). Rats, rabbits, and mice exposed at 1.5, 3.4, or 12.8 ppm boron trifluoride developed pneumonitis and dental fluorosis, although at the lowest level tested, the evidence of pneumonitis was described as "marginal" (Torkelson, Sadek, and Rowe 1961).

Based on this evidence of boron tribromide's severe pulmonary toxicity at exposure levels below 4 ppm, OSHA preliminarily proposes a ceiling limit of 1 ppm. The Agency believes that this limit will protect workers from the risk of serious pulmonary damage associated with exposure to this substance at the levels permitted in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for boron tribromide. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

BROMINE PENTAFLUORIDE
CAS: 7789-30-2; Chemical Formula: BrF_5
H.S. No. 1043

OSHA has no current limit for bromine pentafluoride exposure. The ACGIH recommends a TLV-TWA of 0.1 ppm. This substance is a pale-yellow liquid at temperatures below 40.3°C; above this temperature, it is a colorless, pungent, and corrosive gas.

Bromine pentafluoride has been shown to be acutely toxic in animals. Animals exposed to bromine pentafluoride vapor at 500 ppm exhibited immediate symptoms of gasping, swollen eyelids, clouded corneas, tearing, salivation, and acute distress; these symptoms appeared after exposure for a period as short as 3 minutes. Exposures to 50 ppm were fatal after 30 minutes, and chronic exposures above 3 ppm resulted in severe nephrosis in some animals, as well as marked hepatosis and severe respiratory involvement (The Matheson Co., Inc., as cited in ACGIH, p. 66). Bromine pentafluoride is toxicologically more active than free, elemental fluorine, and its toxicity appears to be closely related to that of chlorine trifluoride (Horn and Wier 1955, 1956). Chlorine trifluoride has caused severe toxicity and some fatalities in dogs and rats exposed to an average concentration of 1.17 ppm 6 hours daily for 6 months (Horn and Wier 1955).

OSHA is proposing the adoption of a PEL of 0.1 ppm as an 8-hour TWA to prevent the risk of serious systemic injury potentially associated with exposure to this substance at the levels permitted in the absence of any OSHA limit. The Agency preliminarily concludes that this limit will substantially reduce this risk. The health evidence forms a reasonable basis for proposing a new limit for bromine pentafluoride. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

n-BUTYL ACRYLATE
CAS: 141-32-2; Chemical Formula: $C_7H_{12}O_2$
H.S. No. 1048

OSHA has no current limit for n-butyl acrylate. The ACGIH's Threshold Limit Value is a 10 ppm TWA. n-Butyl acrylate is a colorless, flammable liquid.

n-Butyl acrylate is a skin and eye irritant and is toxic to animals (rats) at inhalation doses of 1000 ppm for 4 hours (Treon, Sigmon, Wright, and Kitzmiller 1949).

In rabbits, the dermal LD_{50} for n-butyl acrylate was approximately 1800 mg/kg, compared to 1235 mg/kg for methyl acrylate (Smyth, Carpenter, and Weil 1951). n-Butyl acrylate has also been found to be mildly irritating to the skin and to produce corneal necrosis in the unwashed eyes of rabbits (Holland 1974, unpublished memo, as cited in ACGIH 1986, p. 75).

OSHA is proposing an 8-hour TWA PEL of 10 ppm for n-butyl acrylate, based on the similarity of the toxicological response of n-butyl acrylate to methyl acrylate, for which OSHA has a 10 ppm TWA limit. The Agency preliminarily concludes that this limit is necessary to reduce the risk of skin irritation and corneal necrosis to which workers could be exposed in the absence of an OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for n-butyl acrylate. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

o-sec-BUTYLPHENOL
CAS No. 89-72-5; Chemical Formula: $C_2H_5(CH_3)CHC_6H_4OH$
H.S. No. 1055

OSHA has no current limit for o-sec-butylphenol. The ACGIH recommends a 5-ppm 8-hour TLV-TWA with a skin notation. o-sec-Butylphenol is a colorless liquid.

Animal studies indicate that contact with o-sec-butylphenol causes irritation of the skin, eyes, and respiratory tract, and may result in skin burns. A Dow Chemical Company study (1977, as cited in ACGIH 1986, p. 84) showed that the oral and skin absorption LD_{50}'s for guinea pigs ranged between 0.6 and 2.4 g/kg. Prolonged contact of o-sec-butylphenol with the skin of these animals resulted in burns, whereas direct application to the eyes did not cause corneal injury. The oral LD_{50} for rats is 2700 mg/kg (Sax 1984), and rats exposed to saturated air levels of this chemical survived for 7 hours (Dow Chemical Company 1977, as cited in ACGIH 1986, p. 84). The intravenous LD_{50} for mice is 6 mg/kg (Sax 1984).

Acute workplace exposures to o-sec-butylphenol have resulted in mild respiratory irritation and skin burns (ACGIH 1986, p. 84).

OSHA is proposing an 8-hour TWA limit of 5 ppm for o-sec-butylphenol, with a skin notation. The Agency preliminarily concludes that this limit is necessary to protect workers from the risk of eye and respiratory tract irritation and skin burns associated with exposure to this substance at the levels permitted in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for o-sec-butylphenol. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

CALCIUM HYDROXIDE
CAS: 1305-62-0; Chemical Formula: $Ca(OH)_2$
H.S. No. 1059

OSHA currently has no limit for calcium hydroxide; the ACGIH recommends a TLV-TWA of 5 mg/m^3. Calcium hydroxide is a soft, white, odorless crystalline powder with an alkaline, bitter taste.

Calcium hydroxide is a moderately caustic irritant when it comes in contact with the skin, eyes, or mucous membranes of the upper respiratory tract. The oral LD_{50} in rats is reported to

be 7.34 g/kg (Smyth et al. 1969). Industrial experience with this substance has not shown a high incidence of adverse health effects. Calcium hydroxide has less alkalinity than the hydroxides of the alkali series, and the ACGIH has suggested that limits for exposures to calcium hydroxide should be based on its total alkalinity. According to the ACGIH, since calcium hydroxide has 2.5 times the alkalinity of sodium hydroxide, it should have a limit 2.5 times that of sodium hydroxide; this would set the limit for calcium hydroxide at 5 mg/m³.

OSHA is proposing an 8-hour TWA limit for calcium hydroxide of 5 mg/m³ to protect against the risk of skin, eye, and mucous membrane irritation caused by exposure to this substance at the uncontrolled levels permitted by the absence of any OSHA limit. The Agency preliminarily concludes that this limit will reduce the risk substantially. The health evidence forms a reasonable basis for proposing a new limit for calcium hydroxide. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

CALCIUM OXIDE
CAS: 1305–78–8; Chemical Formula: CaO
H.S. No. 1060

OSHA currently has a limit of 5 mg/m³ for calcium oxide, and the ACGIH recommends a TLV–TWA of 2 mg/m³. Calcium oxide is a white or off-white powder.

Calcium oxide is known to be a caustic and irritating material (Sax 1984); it produces severe irritation on contact with the mucous membranes and moist skin. Significant irritation occurs from the local liberation of heat and the dehydration of tissues resulting from the alkalinity of the slaked particles (ACGIH 1986, p. 92). Sax (1984) considers calcium oxide "a powerful caustic to living tissue."

Exposure to calcium oxide can cause inflammation of the respiratory passages and ulceration of the nasal septum (National Safety Council 1936; Schwartz, Tulipan, and Birmingham 1957). The inhalation of calcium oxide dust has also been linked to reports of pneumonia (International Labour Office 1934). However, the most frequent adverse response associated with exposure to calcium oxide is irritation to the skin and eyes. The effects of exposure to calcium oxide are analogous to those of sodium hydroxide.

OSHA proposes a PEL of 2 mg/m³ TWA for calcium hydroxide to afford protection against the risk of skin and eye irritation. The Agency preliminarily concludes that a 2-mg/m³ 8-hour TWA will substantially reduce this risk. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for calcium oxide if the Agency determines that this limit will substantially reduce significant risk.

CARBONYL FLUORIDE
CAS: 353–50–4; Chemical Formula: COF_2
H.S. No. 1074

OSHA has no current limit for carbonyl fluoride. The ACGIH has established an 8-hour TWA limit of 2 ppm and a 15-minute STEL of 5 ppm for this colorless and essentially odorless gas.

The 1-hour LC_{50} for rats is 360 ppm, and the 4-hour LC_{50} for the same species is 90 ppm (ACGIH 1986, p. 111). Carbonyl fluoride hydrolyzes instantly on contact with moisture.

Repeated exposure of animals to carbonyl fluoride is known to have metabolic effects; it inhibits the fluoride-sensitive enzyme succinic dehydrogenase via hydrolysis of carbonyl fluoride to hydrogen fluoride (Scheel, McMillan, and Phipps 1968). Carbonyl fluoride is also a strong irritant to the eyes, skin, mucous membranes, and respiratory tract (Sax 1984).

OSHA is proposing an 8-hour TWA limit of 2 ppm and a 15-minute 5 ppm STEL for carbonyl fluoride; these limits are based on analogy to the 3-ppm TWA limit being proposed for hydrogen fluoride. The Agency preliminarily concludes that both a TWA and a STEL are necessary to provide protection against the risk of marked irritation and metabolic effects associated with exposure to carbonyl fluoride at the levels permitted in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for carbonyl fluoride. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

CATECHOL (PYROCATECHOL)
CAS: 120–80–9; Chemical Formula: $C_6H_4(OH)_2$
H.S. No. 1075

OSHA currently has no established limit for catechol. The ACGIH recommends a TWA–TLV of 5 ppm. Catechol is a colorless crystalline solid that sublimes readily and thus occurs in the vapor state at room temperature.

Catechol is approximately 1.1 to 2.2 times more toxic than phenol, depending on the route of exposure (Acute Toxicity Studies With Catechol 1974, as cited in ACGIH 1986, p. 112). The oral LD_{50} in rats is 300 mg/kg, or approximately half that of phenol. Percutaneous toxicity for catechol in rabbits is 800 mg/kg, only slightly greater than the value for phenol. Eye and nose irritation, as well as muscular spasms and tremor, have been observed in rats at a concentration of 2800 mg/m³ catechol indicating that the acute respiratory toxicity of catechol is approximately one-third that of phenol (Acute Toxicity Studies with Catechol 1974, as cited in ACGIH 1985, p. 112). Metabolic data indicate that the urinary elimination rate of catechol in rabbits is only 10 percent of that of phenol (Williams 1959). In mice, catechol is easily absorbed through the skin and gastrointestinal tract (Forsyth and Quinel 1957). Additional data document a variety of dermal, respiratory, and systemic toxicities that are closely analogous to those of phenol in their metabolic actions (Harald, Nierenstein, and Road 1910; Dietering 1938; Cushny et al. 1940).

Exposure to catechol causes an increase in blood pressure, and, at high doses, kidney damage, eczematous dermatitis, and systemic illness (Harald, Nierenstein, and Road 1910; Dietering 1938; Cushny et al. 1940).

OSHA is proposing a permissible exposure limit of 5 ppm TWA for this substance. The Agency preliminarily concludes that this level will protect workers against the risk of dermal, respiratory, and systemic effects potentially associated with exposure to catechol in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for catechol. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

1-CHLORO-1-NITROPROPANE
CAS: 600–25–9; Chemical Formula: $CH_3CH_2CHClNO_2$
H.S. No. 1081

OSHA's current time-weighted average limit for 1-chloro-1-nitropropane is 20 ppm. The ACGIH recommends a TLV–TWA of 2 ppm for this flammable liquid (ACGIH 1986). The ACGIH (1986, p. 132) notes that a 20-ppm 8-hour TWA "provides little margin of safety, even assuming no chronic effects."

1-Chloro-1-nitropropane is the most acutely toxic of the fungicides known as the chloronitropropanes. In an inhalation experiment, two rabbits were exposed for 6 hours to a concentration of 393 ppm, after which one rabbit died; at an average concentration of 2574 ppm, both rabbits died. Guinea pigs tested under the same conditions survived these exposures. The oral LD_{50} for rabbits determined in the same study

was between 50 and 100 mg/kg (Machle, Scott, Treon et al. 1945). Other members of this family of fungicides show lesser skin and lung irritation but do have higher ingestion toxicities (Patty 1963). Exposure to high concentrations of 1-chloro-1-nitropropane can cause heart muscle, liver, and kidney damage (Patty 1963).

OSHA is proposing an 8-hour TWA PEL of 2 ppm. The Agency preliminarily concludes that this limit will protect exposed employees from the risk of skin and upper respiratory irritation and of systemic toxicity potentially associated with 1-chloro-1-nitropropane exposure at the existing PEL. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for 1-chloro-1-nitropropane if the Agency determines that this limit will substantially reduce significant risk.

COBALT CARBONYL
CAS: 10210-68-1; Chemical Formula: $Co_2(CO)_4$
H.S. No. 1098

OSHA has no current limit for cobalt carbonyl. The ACGIH recommends a TLV-TWA of 0.1 mg/m³ (as cobalt) for this substance, which is a solid that decomposes at 50°C.

Sax (1984) reports that cobalt carbonyl has a moderate-to-high order of toxicity by the oral route. The oral LD_{50} in mice is 377.7 mg/kg; in rats, it is 753.8 mg/kg (Spiridonova and Shabalina 1973). The hazards of exposure to the metal carbonyls range from relatively low (for iron pentacarbonyl) to extremely serious (for nickel carbonyl) (Clayton and Clayton 1982, Vol. 2A, pp. 1797-1806); the greater the toxicity of the metal and the more stable and volatile the carbonyl, the more hazardous the compound. Exposure to any of the metal carbonyls causes the same symptoms of nausea, dizziness, headache, substernal pain, coughing and dyspnea (Clayton and Clayton 1982). Evidence concerning any chronic effects of long-term exposure is lacking (ACGIH 1986, p. 145).

OSHA proposes a PEL of 0.1 mg/m³ TWA for cobalt carbonyl to protect against the risk of headache, nausea, and pulmonary effects associated with occupational exposure to this substance at the levels permitted in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for cobalt carbonyl. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

COBALT HYDROCARBONYL
CAS: 16842-03-8; Chemical Formula: $HCo(CO)_4$
H.S. No. 1099

OSHA has no current limit for cobalt hydrocarbonyl. The ACGIH recommends a TLV-TWA of 0.1 mg/m³ (as cobalt) for this flammable and toxic gas.

Cobalt hydrocarbonyl is approximately half as toxic as nickel carbonyl in terms of acute effects; in animals, it produces clinical signs and symptoms very similar to those produced by nickel carbonyl (ACGIH TLV-TWA of 0.007 mg/m³) and iron pentacarbonyl (ACGIH TLV-TWA of 0.8 mg/m³ (ACGIH 1986, p. 145). These include headache, dizziness, and, after a delay in onset, liver, brain, and lung damage. The 30-minute LC_{50} in rats is 165 mg/kg (Palmes, Nelson, Laskin, and Kuschner 1959). There is no evidence of chronic toxicity or of carcinogenicity.

OSHA proposes a TWA limit of 0.1 mg/m³ to protect exposed employees from the risk of pulmonary, brain, and liver damage, as well as that of acute effects such as headaches and dizziness, which are possible in the absence of an OSHA limit for this substance. The Agency preliminarily concludes that this limit will substantially reduce this risk. The health evidence forms a reasonable basis for proposing a new limit for cobalt hydrocarbonyl. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

DIAZINON
CAS: 333-41-5; Chemical Formula: $C_{12}H_{21}N_2O_3PS$
H.S. No. 1118

OSHA currently has no limit for diazinon. The ACGIH recommends a TLV-TWA of 0.1 mg/m³, with a skin notation. Pure diazinon is a colorless liquid, but the technical grade is pale yellow to dark brown in color and has a faint odor.

Gaines reports the acute oral LD_{50} for male and female rats to be 108 and 76 mg/kg, respectively (1960). Other reports set the acute oral LD_{50}'s in rats, guinea pigs, and rabbits at 76 to 150, 240 to 320, and 130 mg/kg, respectively (Pesticide Chemicals Official Compendium 1969). Hazleton Laboratories (1965) and Radeleff (1958) have shown much greater susceptibility to diazinon in birds and calves, with the oral LD_{50} being less than 10 mg/kg in some instances. However, susceptibility to repeated doses is relatively consistent among species, with dogs showing signs of poisoning at 9 3 mg/kg per day and rats showing complete inhibition of red blood cell cholinesterase and marked inhibition of brain cholinesterase at 50 mg/kg/day (Bruce, Howard, and Elsea 1955). Monkeys were poisoned at 5 mg/kg/day (Woodard and Cronin 1963). Chronic feeding studies in rats have shown no chronic toxicity at 10, 100, and 1000 ppm. For many mammals, diazinon is less toxic than parathion (ACGIH TLV-TWA of 0.1 mg/m³), although this is not true under some circumstances (ACGIH 1986, p. 172).

In humans, Hayes reports that two patients were poisoned by a dermal diazinon dosage of about 1.1 mg/kg (1963); however, Gassman (1957) reports no ill effects from an accidental ingestion of 30 mg/kg. One man received a dose of 250 mg/kg and recovered after treatment, which included gastric lavage (Bockel 1967). In tests, Geigy (1966) found that a series of doses of 0.05 mg/kg/day for 28 days produced plasma cholinesterase inhibition, and it has been suggested that the no-effect level for cholinesterase inhibition in humans is 0.02 mg/kg/day. Skin absorption of diazinon occurs readily, and overexposures are associated with weakness, headache, blurred vision, salivation, sweating, nausea, vomiting, diarrhea, abdominal cramps, slurred speech, and moist rales in the lungs (ACGIH 1986, p. 172).

The Agency is proposing an 8-hour TWA PEL of 0.1 mg/m³, with a skin notation, for diazinon. The Agency preliminarily concludes that these limits will protect exposed workers from the risk of cholinesterase inhibition, weakness, headache, nausea, vomiting, and the other symptoms and signs of diazinon poisoning resulting either from ingestion or dermal absorption at the levels permitted by the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for diazinon. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

1,1-DICHLORO-1-NITROETHANE
CAS: 594-72-9; Chemical Formula: $CH_3CCl_2NO_2$
H.S. No. 1121

OSHA currently has a ceiling limit of 10 ppm for 1,1-dichloro-1-nitroethane. The ACGIH recommends a TLV-TWA of 2 ppm for this colorless liquid.

Toxicity data on 1,1-dichloro-1-nitroethane are largely derived from the 1945 studies conducted by Machle and co-workers. These scientists reported that both rabbits and guinea pigs died from inhaling vapors at 100 ppm for 6 hours; at a concentration of 60 ppm, the animals survived a 2-hour exposure.

Four-hour inhalation exposures at 34 ppm and 6-hour daily exposures at 25 ppm for a total of 204 hours also did not kill rabbits or guinea pigs. Skin and mucous membrane irritation was not produced at the 25 ppm exposure level. At survival concentrations, the primary targets of toxicity were the lungs, which showed edema, congestion, hemorrhage, and acute bronchitis. At lethal exposures, these investigators observed acute myocardial degeneration with interstitial edema, cloudy swelling of the liver with cellular degeneration, and tubular degeneration and interstitial edema of the kidney, as well as edema of the tufts of the glomeruli and kidney necrosis. The compound was also found to be a severe skin irritant when two applications were applied on 2 successive days (Machle, Scott, Treon et al. 1945).

OSHA is proposing a PEL of 2 ppm TWA for 1,1-dichloro-1-nitroethane. The Agency preliminarily concludes that this limit will protect workers against the risk of irritation, lung injury, and liver and kidney damage that exists at the current permissible exposure limit. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for 1,1-dichloro-1-nitroethane if the Agency determines that this limit will substantially reduce significant risk.

p-DICHLOROBENZENE
CAS: 106-46-7; Chemical Formula: $C_6H_4Cl_2$
H.S. No. 1125

OSHA currently has an 8-hour 75 ppm TWA limit for p-dichlorobenzene. The ACGIH recommends a limit of 75 ppm TWA and a STEL of 110 ppm for this white crystalline material, which has a camphor-like odor. The ACGIH's limit recognizes that the para isomer is somewhat less toxic than the ortho isomer, for which the ACGIH has established a ceiling limit of 50 ppm.

In animal studies, an injection of 0.005 gram in rats caused slight liver necrosis (Cameron and Thomas 1937). The intraperitoneal injection LD_{50} for rats has been reported as 2562 mg/kg (Zupko and Edwards 1949). The oral LD_{50} in mice is 2950 mg/kg (Domenjoz 1946); for rats, the oral LD_{50} is 2512 mg/kg (Varshavskaya 1970). Rabbits fed a daily dietary exposure of 5 grams developed opacity of the lens in 3 weeks (Berliner 1939); this finding was not confirmed, however, in repeated studies (Pike 1944).

Reports of a human inhalation exposure to unspecified levels of p-dichlorobenzene describe swelling of the feet, ankles, and hands after day-long use of a moth proofing agent consisting of this substance (Claytor 1935). Other reports describe cataracts caused by exposure to unspecified concentrations of the vapor of p-dichlorobenzene (Berliner 1939). Petit and Champaix (1948) report the case of a woman who experienced tingling of the hands, vertigo, and loss of weight from working for 18 months with a mixture of 90 parts p-dichlorobenzene and 10 parts hexachloroethane (airborne concentration not specified).

OSHA is proposing a PEL of 75 ppm TWA and a STEL of 110 ppm for p-dichlorobenzene. The Agency preliminarily concludes that both a TWA and a STEL are necessary to protect exposed workers from the risk of eye damage, vertigo, and neuropathic effects potentially associated with occupational exposure to p-dichlorobenzene. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for p-dichlorobenzene if the Agency determines that this limit will substantially reduce significant risk.

DICHLOROFLUOROMETHANE
CAS: 75-43-4; Chemical Formula: $CHCl_2F$
H.S. No. 1128

OSHA currently has a limit of 1000 ppm TWA for dichlorofluoromethane (FC-21). The ACGIH recommends a TLV-TWA of 10 ppm for this colorless gas. FC-21 is considered more toxic than the related difluorinated methanes. The major health hazards associated with exposure to this substance are liver damage, cardiac sensitization, and narcosis.

Freon-21 has a 4-hour LC_{50} of 49,900 ppm in rats (Tappan and Waritz 1964, as cited in ACGIH 1986, p. 187). Within an hour, exposure to 100,000 ppm killed rats and guinea pigs (Weigand 1971); other tests with guinea pigs and mice demonstrated that concentrations of 50,000 ppm and higher cause unconsciousness or death (Nuckolls 1935, as cited in ACGIH 1986, p. 187; Booth and Bixby 1932). The clinical signs of overexposure include loss of coordination, tremors, narcosis, and prostration, as well as possible lung and liver changes (Tappan and Waritz 1964).

Two-week exposures of rats to 10,000 ppm for 6 hours daily caused hepatic failure or marked liver damage (Trochimowicz, Moore, and Chiu 1977). A series of 90-day exposures of rats and dogs to concentrations of 1000 and 5000 ppm dichlorofluoromethane resulted in bilateral hair loss, cirrhosis, and excessive mortality in rats at both exposure levels; dogs exhibited weight loss at both levels, but mild liver changes were observed only at the 5000-ppm level (Trochimowicz, Lyon, Kelly, and Chiu 1977). Another uncompleted study reported liver pathology in rats repeatedly exposed for 90 days at 500 ppm, and probable liver pathology from similar exposures to 200 ppm; no hepatic effects were observed after exposure to 50 ppm (Allied Chemical Company 1978, as cited in ACGIH 1986, p. 187).

Two of 12 dogs exposed to 10,000 ppm FC-21 plus intravenous epinephrine developed serious arrhythmia (Mullin, as cited in ACGIH 1986, p. 187). Dogs and monkeys (anesthetized) demonstrated tachycardia and hypotension after exposure to FC-21 levels between 50,000 and 100,000 ppm; bronchoconstriction was observed at 25,000 ppm (Aviado and Smith 1975; Belej and Aviado 1975). Anesthetized mice exposed to a concentration of 100,000 ppm FC-21 showed arrhythmia and cardiac sensitization to epinephrine (Aviado and Belej 1974). Pre-implantation loss has been reported in pregnant rats exposed to FC-21 at 10,000 ppm on days 6 to 15 of gestation (Belej and Aviado 1975).

OSHA proposes a TWA limit of 10 ppm for dichlorofluoromethane. The Agency preliminarily concludes that this limit will protect workers against the risks of hepatotoxic effects, cardiac sensitization, and narcosis that have been shown to exist as a consequence of exposure to this substance at levels substantially below the current OSHA limit. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for dichlorofluoromethane if the Agency determines that this limit will substantially reduce significant risk.

DIETHYL KETONE
CAS: 96-22-0; Chemical Formula: $C_2H_5COC_2H_5$
H.S. No. 1135

OSHA currently has no limit for diethyl ketone. The ACGIH recommends a limit of 200 ppm TWA for this colorless liquid, which has an acetone-like odor.

The oral LD_{50} for diethyl ketone in rats is reported to be 2.14 g/kg. Four of six rats died when exposed to diethyl ketone for 4 hours at 8000 ppm (Smyth et al. 1954). In general, the toxicities of the methyl ketones increase with increasing molecular weight; diethyl ketone is somewhat less toxic than is methyl propyl ketone (NIOSH 1978). All of the ketones cause mucous membrane and eye and skin irritation.

OSHA is proposing an 8-hour TWA PEL of 200 ppm for diethyl ketone, the same limit being proposed for methyl

propyl ketone. The Agency preliminarily concludes that this limit will reduce the risk of eye and skin irritation associated with exposure to diethyl ketone in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for diethyl ketone. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

DIETHYLENE TRIAMINE
CAS: 111–40–0; Chemical Formula: $(NH_2CH_2CH_2)_2NH$
H.S. No. 1138

OSHA currently has no limit for diethylene triamine (DETA). The ACGIH recommends a TLV–TWA of 1 ppm, with a skin notation, for this strongly alkaline, hygroscopic, and somewhat viscous yellow liquid that smells like ammonia.

The acute intraperitoneal LD_{50} values for DETA are reported to be 71 and 74 mg/kg for the mouse and rat, respectively (Hine 1958). In the rat, the reported oral and percutaneous LD_{50} values are the same (1080 mg/kg); the dermal LD_{50} for the rabbit is 1090 mg/kg (Smyth et al. 1949). Exposure to 300 ppm of diethylene triamine vapor for 8 hours failed to kill any of a group of exposed rats (Savitt 1955).

Sutton (1963) has reported that DETA causes severe corneal injury; solutions of 15 to 100 percent caused lasting corneal damage. If improperly controlled, the vapor and liquid cause sensitization of the respiratory tract and skin (American Industrial Hygiene Association 1960). Dernehl demonstrated such sensitization in a study reported in 1951.

OSHA proposes an 8-hour TWA limit of 1 ppm, with a skin notation, for diethylene triamine. The Agency preliminarily concludes that these limits will protect workers against the risk of skin and respiratory tract irritation and sensitization potentially associated with exposure to diethylene triamine in the absence of any OSHA PEL. The skin notation is necessary to reduce the risk of skin sensitization resulting from contact with this substance. The health evidence forms a reasonable basis for proposing a new limit for diethylene triamine. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

DIPROPYL KETONE
CAS: 123–19–3; Chemical Formula: $(CH_3CH_2CH_2)CO$
H.S. No. 1148

OSHA currently has no limit for dipropyl ketone. The ACGIH recommends a TLV of 50 ppm TWA for this colorless liquid with a penetrating odor.

Dipropyl ketone has a moderate oral and inhalation toxicity (Sax 1984). In rats, the oral LD_{50} is 3.73 g/kg. Tests have indicated that rats inhaling 2000 ppm for 4 hours survived, but at 4000 ppm all animals died (Carpenter, Weil, and Smyth 1974). Methyl isobutyl ketone (MIBK) has a similar acute toxicity (OSHA is proposing a 50-ppm 8-hour TWA and a 75-ppm STEL for MIBK) (ACGIH 1986, p. 221).

OSHA is proposing a PEL of 50 ppm TWA for dipropyl ketone. The Agency preliminarily concludes that this limit is necessary to protect workers from the adverse health effects that could occur from the uncontrolled exposures permitted in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for dipropyl ketone. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

DIQUAT
CAS: 85–00–7; Chemical Formula: $C_{12}H_{12}Br_2N_2$
H.S. 1150

OSHA currently has no limit for diquat. The ACGIH recommends a limit of 0.5 mg/m³ TWA for these yellow crystals.

In most species, the acute oral toxicity of diquat is similar to that of paraquat and ranges from 100 to 400 mg/kg in rats, mice, rabbits, and dogs. Cows experience more severe toxic effects, with an acute oral LD_{50} of 30 mg/kg. The 24-hour percutaneous LD_{50} in rabbits is greater than 400 mg cation/kg; no skin irritation or other ill effects were demonstrated at this level (Clark and Hurst 1970; Rowe and Wright 1965). Rats fed 1000 ppm daily (about 50 mg/kg/day) for 2 years survived; reduced food intake and growth were the only consequences observed. At 500 ppm (about 25 mg/kg/day), the only ill effect observed was a pathologic change in the eye. A dietary level of 10 ppm (about 0.5 mg/kg/day) for 2 years did not induce cataract formation, but cataracts do occur at higher levels, with pathology observed at the 500-ppm level; one in four animals demonstrated complete corneal opacity in one or both lenses after 6 months at the 1000-ppm level. Cataract formation requires prolonged exposure and is not induced by single high-level exposures (ACGIH 1986, p. 222).

Unlike paraquat, diquat does not produce lung damage in human or animal exposures. Acute poisoning may produce nonspecific respiratory distress as well as other nonspecific signs of poisoning. In humans, accidental ingestion has shown less toxic reactions than those associated with paraquat (Orepoulos and McEvoy 1969).

OSHA is proposing a PEL of 0.5 mg/m³ TWA for diquat. The Agency preliminarily concludes that this limit will protect against the risk of ocular problems associated with chronic exposure at the exposure levels permitted in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for diquat. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

DISULFOTON
CAS: 298–04–4: Chemical Formula: $C_8H_{19}O_2PS_3$
H.S. No. 1152

OSHA currently has no exposure limit for disulfoton. The ACGIH recommends a limit of 0.1 mg/m³ TWA for this substance. Pure disulfoton is an oily, colorless liquid; the technical grade is a brown liquid.

The acute toxicity of disulfoton is very high by all laboratory-tested routes of administration. For weanling rats, the intraperitoneal LD_{50} is reported to be 5.4 mg/kg; for adult rats, it is 9.4 mg/kg (Brodeur and Dubois 1963). The acute dermal LD_{50} is 6 mg/kg for adult female rats and 25 mg/kg for adult male rats (Gaines 1969). The acute oral LD_{50}s for male and female rats are reported as 6.8 mg/kg and 2.3 mg/kg, respectively (Brodeur and Dubois 1964). Rats have demonstrated an acquired tolerance for disulfoton (Brodeur and Dubois 1964).

Metabolically, disulfoton is highly fat-soluble, and the compound apparently interferes with mixed-function oxidase activity in the same manner shown to be the case for parathion; with respect to median lethal doses, parathion and disulfoton are similar (Stevens et al. 1973).

OSHA proposes an 8-hour TWA PEL for disulfoton of 0.1 mg/m³. The Agency preliminarily concludes that this limit will prevent the risk of acute toxicity and metabolic injury possible in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for disulfoton. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

DIVINYL BENZENE
CAS: 108–57–6; Chemical Formula: $C_6H_4(CHCH_2)_2$
H.S. No. 1154

OSHA currently has no limit for divinyl benzene. The ACGIH

recommends a TLV–TWA of 10 ppm. The commercial grade of divinyl benzene is a straw-colored liquid; it contains all three isomers, but the meta isomer predominates.

Divinyl benzene has low acute toxicity. The oral LD_{50} for rats is reported to be 4.1 g/kg, and an acute inhalation study showed no ill effects from a single 7-hour exposure at 351 ppm. However, repeated or prolonged contact with the liquid may cause skin burns (Dow Chemical Company, as cited in ACGIH 1986, p. 228).

Industrial experience indicates that mild irritation of the respiratory system, skin, and eyes can result from inhalation exposures, but there are no data concerning chronic exposures in humans.

OSHA is proposing a PEL of 10 ppm TWA for divinyl benzene. The Agency preliminarily concludes that this limit will prevent the risk of irritation to the respiratory tract, eyes, and skin potentially associated with exposure to divinyl benzene in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for divinyl benzene. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

ENDOSULFAN
CAS: 115–29–7; Chemical Formula: $C_9H_6Cl_6O_3S$
H.S. No. 1156

OSHA currently has no permissible exposure limit for endosulfan. The ACGIH recommends a TLV–TWA of 0.1 mg/m^3, with a skin notation. Technical endosulfan is a tan, semi-waxy solid mixture; it may have a slight odor similar to that of sulfur dioxide.

The insecticide, endosulfan, is similar in its acute oral toxicity to the related insecticides aldrin and dieldrin, with the exception that it is slightly more toxic than these substances in female laboratory animals. In rats, the oral LD_{50} of endosulfan is 43 mg/kg for males and 18 mg/kg for females (Farm Chemicals Handbook 1974). The dermal LD_{50}s in male and female rats are 130 mg/kg and 74 mg/kg, respectively (Farm Chemicals Handbook 1974). The respiratory LC_{50} for male rats is 50 mg/kg for 4 hours of exposure (Pesticide Chemicals Official Copendium, as cited in ACGIH 1986, p. 230).

In laboratory tests of chronic exposure, rats tolerated oral doses of up to 3.2 mg/kg/day for 3 months without injury (Gaines, as cited in ACGIH 1986, p. 230), and dogs tolerated doses up to 0.75 mg/kg for 1 year (Ely, MacFarlane, Galen, and Hines 1967). A 2-year dietary level of 10 ppm (approximately 0.5 mg/kg/day) in rats was associated with a statistically insignificant decline in female survival rates and caused a reduction in testis weights in males. At 5.0 mg/kg/day, histopathologic findings showed renal tubular damage and some hydropic changes in rat livers (Czech 1958).

Inhalation of endosulfan dust has been associated with slight nausea, confusion, excitement, flushing, and dry mouth (State of California: Department of Industrial Relations, as cited in ACGIH 1986, p. 230). Nine employees who had been working with 50-percent water-wettable endosulfan powder for only a few days had convulsions (Pesticide Chemicals Official Compendium, as cited in ACGIH 1986, p. 230).

OSHA preliminarily concludes that exposure to endosulfan poses a risk of systemic poisoning and renal and testicular damage, and the Agency therefore proposes a PEL of 0.1 mg/m^3 TWA for endosulfan. OSHA believes that this limit will substantially reduce the risk currently associated with exposure to this substance at the levels permitted in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for endosulfan. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

FONOFOS
CAS: 944–22–9; Chemical Formula: $C_{10}H_{15}OPS_2$
H.S. No. 1181

OSHA currently has no limit for fonofos. The ACGIH recommends a limit of 0.1 mg/m^3 TWA, with a skin notation, for this light-yellow liquid.

In male rats, the average acute oral LD_{50} of technical fonofos has been reported to be 13.2 mg/kg (Stauffer Chemical Co., as cited in ACGIH 1986, p. 275). For female rats, an average oral LD_{50} of 3 mg/kg has been reported (NIOSH 1974). The acute dermal LD_{50}s reported for rats and guinea pigs are 147 and 278 mg/kg, respectively (Weir and Hazleton 1981). Weir and Hazleton reported that no localized eye irritation occurred when 0.1 ml of technical fonofos was instilled into rabbit eyes; however, death resulted in these animals within 24 hours after the instillation (1981). Dietary studies of rats lasting 105 weeks have shown 10 ppm (about 0.2 mg/kg) to be a no-effect level. Dogs fed fonofos for 14 weeks showed no-effect dietary levels of 8 ppm; no carcinogenic effects were observed. Rats showed reproductive effects at dietary levels of 10 ppm and 31.6 ppm (about 0.7 mg/kg) (Stauffer Chemical Co., as cited in ACGIH 1986, p. 275).

There are no reports of human poisonings caused by fonofos, although it is known to be a cholinesterase inhibitor (ACGIH 1986, p. 275).

OSHA is proposing a PEL of 0.1 mg/m^3 TWA for fonofos to protect exposed workers from the risk of cholinesterase inhibition that is characteristic of this substance and of other organic phosphate pesticides. A skin notation is also proposed, based on evidence in animals that fonofos can penetrate the skin and cause death. The Agency preliminarily concludes that these limits will reduce the risk to which workers could be exposed in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for fonofos. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

FORMAMIDE
CAS: 75–12–7; Chemical Formula: CH_3NO
H.S. No. 1182

OSHA currently has no limit for formamide. The ACGIH recommends a TLV–TWA of 20 ppm and a TLV–STEL of 30 ppm for this clear, viscous, odorless liquid.

Formamide has a low oral toxicity, with an LD_{50} of approximately 6 g/kg for rats (Thiersh 1962; Zaeva et al. 1969). Dietary administration at 1.5 g/kg for 2 weeks resulted in fatalities in rats; pathologic examination revealed cumulative changes characteristic of gastritis and malnutrition (E.I. du Pont de Nemours and Company, as cited in ACGIH 1986, p. 278). Czajkowska (1981) reports the dermal LD_{50} for skin absorption in rabbits as 6 g/kg; Sturla and Krauss (1977) report the approximate lethal dose in rabbits to be greater than 17 g/kg. Mild and transient irritation, but no allergic skin sensitization, occurred when formamide was applied to the skin of guinea pigs (Sax 1984; E.I. du Pont de Nemours and Company, as cited in ACGIH 1986, p. 278). However, the potential for systemic toxicity through skin absorption has been demonstrated in tests with rats, although effective doses were relatively high (BASF Corporation 1985, as cited in ACGIH 1986, p. 278). Eye irritation tests in rabbits showed only slight, temporary irritation (Carpenter and Smyth 1956). No signs of toxicity in rats were detected in single 6-hour exposures at 3900 ppm formamide dispensed as a mist, or in 6-hour daily exposures for 10 days at approximately 1500 ppm formamide vapor (equivalent

to air saturated with formamide at room temperature); no indications of organ damage were seen in these animals on pathologic examination (E.I. du Pont de Nemours and Company, as cited in ACGIH 1986, p. 278).

Gross fetal malformations were not noted following dermal applications of formamide to skin of pregnant rats; the effects that were observed were weak and were produced at overwhelming concentrations (Stula and Krauss 1977). The no-observed-effect level in a rabbit developmental toxicity study was 22 mg/kg orally (Merkle and Zeller 1980).

There are no reports of industrial poisoning by formamide (E.I. du Pont de Nemours and Company, as cited in ACGIH 1986, p. 278).

OSHA is proposing a PEL of 20 ppm TWA and a 15-minute STEL of 30 ppm for formamide. The Agency preliminarily concludes that this combined limit will protect workers against the risk of eye and skin irritation that exists potentially from workplace exposure at the levels permitted by the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for formamide. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

GERMANIUM TETRAHYDRIDE
CAS: 7782-65-2; Chemical Formula: GeH_4
H.S. No. 1186

OSHA currently has no limit for germanium tetrahydride. The ACGIH recommends a TLV of 0.2 ppm TWA for this colorless gas.

An early study indicated that germanium tetrahydride has a toxicity between that of tin hydride and arsine (Flury and Zernik 1931). In this study, a rabbit survived exposure to 100 ppm for 1 hour. One-hour exposures at 150 and 185 ppm caused fatalities in mice, and a similar exposure involving guinea pigs resulted in sickness at the 150 ppm level and death at 185 ppm (Flury and Zernik 1931). On the other hand, Webster (1956) reported that germanium tetrahydride is less toxic than both tin hydride and arsine. The effect of exposure to germanium tetrahydride is hemolysis. Data concerning chronic or sub-acute toxicities are not available. Based on germanium's acute toxicity, which is approximately half that of stibine, the ACGIH recommends an 8-hour TLV of 0.2 ppm TWA.

OSHA proposes a PEL of 0.2 ppm as an 8-hour TWA for germanium tetrahydride to reduce the risk of hemolytic effects associated with exposure to this substance at the previous uncontrolled level. The Agency preliminarily concludes that implementation of this limit will substantially reduce this risk. The health evidence forms a reasonable basis for proposing a new limit for germanium tetrahydride. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

INDENE
CAS: 95-13-6; Chemical Formula: C_9H_8
H.S. No. 1212

OSHA has no current limit for indene. The ACGIH recommends a TLV-TWA of 10 ppm for this colorless liquid. This limit is recommended by analogy to naphthalene, for which a TLV-TWA of 10 ppm is recommended.

Early inhalation studies of indene reported injury to the spleen, liver, and kidneys of rats exposed to indene vapor concentrations of 800 to 900 ppm for six 7-hour periods (Cameron and Doniger 1939). Some animals were found at necropsy to have severe necrosis of the liver with hemorrhage; kidney necrosis was also observed. No other organ damage was found and no deaths occurred as a result of these exposures (Cameron and Doniger 1939). By analogy with the effects of exposure to other monoaromatic hydrocarbons, exposure to indene is likely to irritate the mucous membranes. In laboratory animals, chemical pneumonitis, pulmonary edema, and hemorrhage have resulted from the aspiration of indene liquid into the lung, and repeated skin contact has caused dermatitis as a result of the defatting properties of indene (Gerarde 1960). In dermal studies of rats, one to eight applications of 0.1 ml to the shaved skin were reported to have no effect; three applications of 0.5 ml to guinea pig skin also produced no effect (Cameron and Doniger 1939). The oral toxicity of indene appears to be moderate, with adult rabbits tolerating a single dose of 1 gram without signs of systemic toxicity (Gerarde 1960). Subcutaneous injection of 1 gram, however, caused liver pathology and fatalities; high oral doses (2.5 ml of a 1:1 v/v mixture in olive oil) were uniformly fatal, with characteristic liver, lung, and gastrointestinal changes. Chronic administration of 3 mg/m^3 indene for 105 days caused catalase inhibition and stimulation of blood cholinesterase in rats, but no effects were observed in rats exposed at 0.6 mg/m^3 (Dyshinevich, as cited in ACGIH 1986, p. 321).

OSHA is proposing a PEL of 10 ppm TWA for indene. The Agency preliminarily concludes that this level will reduce the risk of irritation, pulmonary effects, and systemic toxicity that may occur when workers are exposed to indene at the levels permitted by the absence of any OSHA permissible exposure limit. The health evidence forms a reasonable basis for proposing a new limit for indene. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

IODOFORM
CAS: 75-47-8; Chemical Formula: CHI_3
H.S. No. 1214

OSHA has no current limit for iodoform. The ACIGH has established an 8-hour TWA limit of 0.6 ppm for this yellow-green powder or crystalline solid with a pungent odor.

The subcutaneous LD_{50} for rabbits is 50 mg/kg, and the oral LD_{LO} for iodoform in dogs is 1000 mg/kg (Kutob and Plaa 1962). These authors also report that, on a molar basis, iodoform has an acute toxicity in mice similar to that of methyl iodide; this conclusion is based on parameters of lethality, barbiturate sleeping time, and bromsulphalein (BSP) retention time. An NCI bioassay (1978a) of iodoform indicates that the substance is not carcinogenic nor of high systemic toxicity, although histopathological examination of laboratory animals was inadequate.

No human data are available for this compound.

OSHA is proposing an 8-hour TWA limit of 0.6 ppm for iodoform, based on the proposed limit for methyl iodide (2 ppm TWA); these limits are comparable on a molar iodine basis. The health evidence forms a reasonable basis for proposing a new limit for iodoform. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

ISOBUTYL ALCOHOL
CAS: 78-83-1; Chemical Formula: $(CH_3)_2CHCH_2OH$
H.S. No. 1219

OSHA currently has a limit of 100 ppm as an 8-hour TWA for isobutyl alcohol. The ACGIH recommends a limit of 50 ppm TWA for this flammable, refractive, colorless liquid.

Limited inhalation studies have reported a somewhat higher acute toxicity for isobutyl alcohol than for n-butyl alcohol (which has an ACGIH ceiling of 50 ppm) (Smyth, Carpenter, and Weil 1951; Smyth, Carpenter, Weil, and Pozzani 1954). A 4-hour LC_{50} of 8000 ppm has been reported in rats for isobutyl alcohol. Ingestion studies in rabbits have reported an acute oral toxicity of 3.75 g/kg for isobutyl alcohol (Smyth, Carpenter, and Weil 1951; Smyth, Carpenter, Weil, and Pozzani

1954). The dermal LO_{50} is 4.2 g/kg (Stokinger 1976). Weese (1928) reported that the narcotic inhalation dose over a total of 136 hours is 6400 ppm in mice. Slight changes in the liver and kidneys were reported, but no fatalities occurred after repeated narcotizing doses (Weese 1928).

The effects of liquid isobutyl alcohol on the human eye appear to be comparable to those of n-butanol; no data are available on ocular exposure to the isobutyl alcohol vapor. Dermal application of isobutyl alcohol has caused slight erythema and hyperemia in humans (Schwartz and Tulipan 1939; Oettel 1936).

OSHA is proposing to reduce the current 8-hour TWA PEL of 100 ppm to 50 ppm for isobutyl alcohol. The Agency preliminarily concludes that a 50-ppm limit will reduce the risk of skin irritation associated with exposures to concentrations below those permitted by the current OSHA PEL. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for isobutyl alcohol if the Agency determines that this limit will substantially reduce significant risk.

ISOOCTYL ALCOHOL
CAS: 26952-21-6; Chemical Formula:
$CH_3(CH_2)_3CH(C_2H_5)CH_2OH$
H.S. No. 1220

OSHA currently has no limit for isooctyl alcohol. The ACGIH recommends a TLV-TWA of 50 ppm, with a skin notation, for this colorless liquid mixture.

The single-dose oral LD_{50}s reported for rats and mice are between 3.2 and 6.4 g/kg; intraperitoneal injection LD_{50}s for these species range from less than 0.4 g/kg to 1.6 g/kg (Hodge 1943; Fassett, as cited in ACGIH 1986, p. 332). The dermal LD_{50} for the guinea pig is greater than 10 ml/kg (Fassett, as cited in ACGIH 1986, p. 332). Moderate skin irritation from exposure to isooctyl alcohol has also been reported. Rats and rabbits have shown skin irritation at exposure levels ranging from 1.7 to 3.34 ml/kg (Smyth et al. 1969). Fassett (as cited in ACGIH 1986, p. 332) also reported no fatalities in rats after an 8-hour inhalation test at 235 ppm.

OSHA is proposing an 8-hour TWA PEL of 50 ppm, with a skin notation, for isooctyl alcohol. The Agency preliminarily concludes that these limits will reduce the risk of skin irritation and dermal absorption potentially associated with exposure to this substance at the levels permitted in the absence of any OSHA limit. The health evidence, forms a reasonable basis for proposing a new limit for isooctyl alcohol. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

n-ISOPROPYLANILINE
CAS: 643-28-7; Chemical Formula:
$C_6H_5NHCH(CH_3)_2$
H.S. No. 1229

OSHA currently has no limit for n-isopropylaniline. The ACGIH recommends a TLV-TWA of 2 ppm, with a skin notation, for this liquid.

The oral LD_{50} for rats exposed to n-isopropylaniline is between 0.25 and 0.5 g/kg. Slight irritation of the skin and eyes has been reported in animals as a result of direct contact with this chemical (Dow Chemical Company, as cited in ACGIH 1986, p. 338). No other data concerning chronic toxicity or human exposure are available (ACGIH 1986, p. 338).

Chemical analysis shows n-isopropylaniline to have toxicologic properties similar to those of its parent compound, aniline. The oral LD_{50}s for the two chemicals are approximately equal. The ACGIH has established the 2-ppm TLV-TWA for n-isopropylaniline on the basis of structural analogy with aniline (which has a 2-ppm TLV-TWA) and n,n-dimethylaniline (which has a 5-ppm TLV-TWA and a 10-ppm STEL); exposure to these substances has been shown to cause hemolytic and central nervous system effects in animals and humans. These substances are also toxic when absorbed through the skin.

OSHA is proposing an 8-hour PEL of 2 ppm for n-isopropylaniline, with a skin notation. The Agency preliminarily concludes that this limit will protect exposed workers from the risk of irritation and, by analogy with aniline, of systemic and hemolytic effects caused by inhalation, ingestion, or dermal absorption. The health evidence forms a reasonable basis for proposing a new limit for n-isopropylaniline. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

KETENE
CAS: 463-51-4; Chemical Formula:
$CH_2=C=O$
H.S. No. 1231

OSHA has a current 8-hour TWA limit of 0.5 ppm for ketene. The ACGIH recommends a TLV-TWA of 0.5 ppm and a TLV-STEL of 1.5 ppm for this colorless gas with a sharp, penetrating odor.

Ketene is highly irritating to the respiratory tract (Mendenhall and Stokinger 1959), and the effects of its action are delayed (Treon, Sigmon, Kitzmiller et al. 1949). Mendenhall and Stokinger (1959) have reported a 10-minute LC_{50} for mice of 17 ppm. Chronic exposure at 1 ppm for 6 months on a schedule of 6 hours daily, 5 days per week, was tolerated by animals of several species (Mendenhall and Stokinger 1960, as cited in ACGIH 1986, p. 341). Similar results have been reported in monkeys exposed repeatedly (55 exposures) for 7 hours (Treon, Sigmon, Kitzmiller et al. 1949). Evidence strongly suggests that the development of emphysema and fibrosis may occur in individuals who have developed a tolerance to the acute effects of ketene exposure (Stokinger, Wagner, and Dobrogarski 1957).

OSHA proposes an 8-hour TWA PEL of 0.5 ppm and a 15-minute STEL of 1.5 ppm for ketene. The Agency preliminarily concludes that workers exposed to this highly irritating and toxic gas are at risk of developing respiratory irritation, pulmonary edema, and other severe pulmonary effects. OSHA believes that these limits will substantially reduce this risk. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for ketene if the Agency determines that this limit will substantially reduce significant risk.

METHACRYLIC ACID
CAS: 79-41-4; Chemical Formula:
$CH_2=C(CH_3)COOH$
H.S. No. 1244

OSHA currently has no limit for methacrylic acid. The ACGIH recommends a TLV-TWA of 20 ppm for this substance. Methacrylic acid is a liquid with an acrid, disagreeable odor.

The primary toxic hazard associated with exposure to methacrylic acid is irritation, although the degree of irritation from exposure to this substance is significantly less than that from acrylic acid (ACGIH 1986, p. 362).

Direct contact of methacrylic acid with the skin or eye can cause corrosion of the skin or blindness. In rabbits, the skin absorption LD_{50} for methacrylic acid is 0.5 to 1 g/kg (Dow Chemical Company, as cited in ACGIH 1986, p. 362). Rats exposed by inhalation to approximately 1000 ppm methacrylic acid exhibited eye irritation (Dow Chemical Company, as cited in ACGIH 1986, p. 362). Rats exposed to 300 ppm for 6 hours daily for 20 days showed slight congestion of the kidneys (Gage 1970).

Medical reports of acute exposures (at concentrations of up to 113 ppm) in an industrial setting revealed no respiratory symptoms; however, skin responses and a severe corneal burn

were reported (Dow Chemical Company, as cited in ACGIH 1986, p. 362).

OSHA is proposing a PEL of 20 ppm as an 8-hour TWA for this substance. The Agency preliminarily concludes that this limit will protect workers from the risk of severe eye and skin irritation potentially associated with exposure to methacrylic acid at the levels permitted by the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for methacrylic acid. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

4-METHOXYPHENOL
CAS: 150-76-5; Chemical Formula: $CH_3OC_6H_4OH$
H.S. No. 1247

OSHA currently has no limit for 4-methoxyphenol. The ACGIH recommends a TLV-TWA of 5 mg/m³ for this solid substance.

In rats, the oral LD_{50} for 4-methoxyphenol is between 1 and 2 g/kg; the skin absorption LD_{50} is reported as greater than 1 g/kg in rabbits. Results of a 2-month dietary study demonstrated no ill effects at 0.1 ppm (approximately 50 mg/kg/day). Direct contact of 4 methoxyphenol with the skin or eyes causes burns or moderate corneal damage (Hodge et al. 1949; Dow Chemical Co., as cited in ACGIH 1986, p. 367).

To reduce the risk of dermal and ocular effects resulting from exposure to 4-methoxyphenol, a compound similar in chemical structure and toxicity to hydroquinone, OSHA is proposing a permissible exposure limit of 5 mg/m³ TWA. The Agency preliminarily concludes that this limit will protect workers against the risk of dermal and skin effects potentially associated with exposures to this substance at the levels permitted in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for 4-methoxyphenol. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

METHYL ACETYLENE-PROPADIENE MIXTURE (MAPP)
CAS: 74-99-7; 463-49-0; Chemical Formula: C_3H_4 isomers
H.S. No. 1250

OSHA currently has a standard of 1000 ppm TWA for MAPP. The ACGIH also recommends an 8-hour TWA limit of 1000 ppm, with a TLV-STEL of 1250 ppm. MAPP contains 58 percent of a mixture of propadiene (a colorless, unstable gas with a strong, unpleasant odor) and methyl acetylene (a colorless gas with a sweet odor); the balance of the mixture consists of paraffinic and olefinic C_3 and C_4 hydrocarbons.

Tests of rabbits, dogs, and guinea pigs exposed to an average concentration of 5000 ppm for 7 hours/day, 5 days/week for 4 months resulted in no adverse health effects except decreased lung weights. No changes at all were observed in animals exposed to 1000 ppm for 4 months (Dow Chemical Co., as cited in ACGIH 1986, p. 368).

On the basis of these data, which show MAPP to be a chemical mixture of low toxicity in experimental animals, the Agency proposes a PEL of 1000 ppm and a STEL of 2250 ppm. The Agency preliminarily concludes that both of these limits are necessary to ensure that workers are protected and that good industrial hygiene practice is maintained. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for MAPP if the Agency determines that this limit will substantially reduce significant risk.

METHYL DEMETON
CAS: 8022-00-2; Chemical Formula: $(CH_3O)_2PSO(CH_2)_2SC_2H_5$
H.S. No. 1256

OSHA currently has no limit for methyl demeton. The ACGIH recommends a TLV-TWA limit of 0.5 mg/m³, with a skin notation. Methyl demeton is an oily, colorless to pale-yellow liquid with an unpleasant odor.

Methyl demeton is reported to have an oral LD_{50} value of 40 to 65 mg/kg for the thiolo isomer and 150 to 250 mg/kg for the thiono isomer. Both isomers form sulfoxide or sulfone, with an oral LD_{50} similar to that of the parent compounds (Dubois and Plazak 1962; Heath and Vandekar 1965; Klimmer and Plaff 1955). In solution or storage, methyl demeton may form alkyl sulfonium compounds of very high intravenous toxicity and an oral LD_{50} of 10 to 20 mg/kg. Dermal toxicity is reported to be moderate, with an LD_{50} of approximately 400 mg/kg (Heath and Vandekar 1965).

In humans, methyl demeton causes changes in intraocular pressure, and acute poisonings produce nausea, headache, dizziness, vomiting, and hyperemia of the nasal mucosa. Chronic exposure causes hyperemia of the respiratory organs and inner ear irritation (Dugel'nyy 1971; Rasuleva 1970).

OSHA is proposing an 8-hour TWA for methyl demeton of 0.5 mg/m³, with a skin notation. The Agency preliminarily concludes that this limit will protect workers from the risk of ocular and nasal irritation and pulmonary effects potentially associated with exposure to this substance at the levels permitted by the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for methyl demeton. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

METHYL ETHYL KETONE PEROXIDE
CAS: 1338-23-4; Chemical Formula: $C_8H_{16}O_4$ or $C_8H_{18}O_6$
H.S. No. 1257

OSHA currently has no limit for methyl ethyl ketone peroxide (MEKP). The ACGIH recommends a ceiling limit of 0.2 ppm. MEKP is sold commercially as a colorless liquid mixture of approximately 60 percent MEKP and 40 percent diluent to reduce MEKP's sensitivity to shock.

In mice and rats, the 4-hour LC_{50}s for MEKP have been reported to be 170 ppm and 200 ppm, respectively; by intraperitoneal injection, the LD_{50} in rats is 65 mg/kg, and the oral LD_{50} in this species is 484 mg/kg (Floyd and Stokinger 1958). The same authors report that MEKP is irritating to the eyes and skin. In addition, the effects of MEKP exposure are cumulative; rats died or showed marked evidence of cumulative effects both orally and intraperitoneally after 7 weeks of 3-day/week doses of MEKP that were 20 percent of the LD_{50} level (Floyd and Stokinger 1958). Inhalation of vapors produced petechial and gross hemorrhages of the lungs in rats after 4-hour exposures; liver and kidney damage was also observed by these authors. Acute high-level exposures have caused nasal porphyrin exudate in rats. Although MEKP caused methemoglobin formation in rats, low-level chronic exposures did not cause this effect (Floyd and Stokinger 1958).

OSHA is proposing a ceiling limit of 0.2 ppm for methyl ethyl ketone peroxide. The Agency preliminarily concludes that this limit will protect workers against the risk of eye and skin irritation, kidney and liver damage, and the cumulative effects potentially associated with exposure to this substance at the levels permitted in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for methyl ethyl ketone peroxide. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

METHYL FORMATE
CAS: 107-31-3; Chemical Formula: $HCOOCH_3$
H.S. No. 1258

OSHA has a current limit of 100 ppm TWA for methyl formate. The ACGIH also recommends an 8-hour time-weighted average of 100 ppm, with a TLV–STEL of 150 ppm. Methyl formate is a flammable, colorless liquid with an agreeable odor.

Methyl formate causes nose and eye irritation, vomiting, incoordination, narcosis, and death in guinea pigs exposed at high concentrations (Schrenk, Yant, Chornyak, and Patty 1936). A 5-percent concentration was fatal in 20 to 30 minutes, a 1.5- to 2.5-percent concentration was dangerous in 30 to 60 minutes, and a 0.5-percent concentration (5000 ppm) was considered the maximum concentration tolerable for a 60-minute period without serious consequences. Lehmann and Flury (1943) observed that inhalation of 1.02 percent methyl formate for 2 to 3 hours caused pulmonary edema and death in cats; a concentration of 1600 ppm resulted in lung inflammation after 1 hour (1943). Guinea pigs died when exposed by inhalation to 2.5 percent methyl formate (Lehmann and Flury 1943).

In studies of methyl formate exposure in humans, van Oettingen reported that exposed workers showed temporary blindness, narcosis, mucous membrane irritation, and dyspnea (1959). Fairhall (1957) has reported that methyl formate was more irritating than either methyl or ethyl acetate (1957).

OSHA is proposing both a 15-minute STEL of 150 ppm and an 8-hour PEL of 100 ppm TWA to prevent the risks of irritation, narcotic effects, and pulmonary damage potentially associated with exposure to concentrations of methyl formate even for short periods (one hour or more). The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for methyl formate if the Agency determines that this limit will substantially reduce significant risk.

METHYL IODIDE
CAS: 74–88–4; Chemical Formula: CH_3I
H.S. No. 1259

OSHA currently has a limit of 5 ppm TWA, with a skin notation, for methyl iodide. The ACGIH recommends a TLV–TWA limit of 2 ppm, with a skin notation, for methyl iodide, and classifies it as a suspected human carcinogen (A2). NIOSH recommends reducing exposure to the lowest feasible limit, and also considers this chemical a carcinogen. Methyl iodide is a colorless, sweet-smelling liquid that turns yellow, red, or brown when exposed to light and moisture.

Methyl iodide has been reported to have an LD_{50} in rats of 150 to 200 mg/kg; liver damage was evident after these lethal exposures (Kutob and Plaa 1960). Fifteen-minute exposures to 3800 ppm were fatal in rats (Chambers et al. 1950, as cited in ACGIH 1986, p. 399), and Bachem (1927, as cited in ACGIH 1986, p. 399) has reported that methyl iodide is 6 times as toxic in mice as methyl bromide. Inhalation studies have shown eye irritation and depressed body-weight in rats as a result of 14-week exposures to 30 and 60 ppm (Black et al. 1984). The same authors observed fatalities in rats within 4 weeks of exposure to 143 ppm; 10 ppm was reported to be a no-effect level.

In industry, fatalities have occurred from methyl iodide poisoning in chemical workers (Garland and Camps 1945; Appel, Galen, O'Brien, and Schoenfeldt 1975). The exposure levels associated with these fatal overexposures are not known, however (ACGIH 1986, p. 399).

In tests of carcinogenicity, methyl iodide produced local sarcomas in rats injected subcutaneously and lung tumors in mice given intraperitoneal injections (Druckrey, Kruse, Preussman et al. 1970; Poirer, Stoner, and Shimkin 1975). These carcinogenic effects occurred at a dosage approximately equivalent to a daily 8-hour exposure to 20 or 25 ppm for an adult human (ACGIH 1986, p. 399).

OSHA proposes an 8-hour TWA limit of 2 ppm, with a skin notation, for methyl iodide. The Agency preliminarily concludes that these limits will protect workers from the risk of irritation, liver and kidney damage, and potential carcinogenicity associated with exposure to methyl iodide in the workplace. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for methyl iodide if the Agency determines that this limit will substantially reduce significant risk.

METHYL ISOAMYL KETONE
CAS: 110–12–3; Chemical Formula: $CH_3COCH(C_2H_5)_2$
H.S. 1260

OSHA currently has no limit for methyl isoamyl ketone (MIAK). The ACGIH has established an 8-hour TLV–TWA of 50 ppm. NIOSH recommends a 50-ppm TWA limit for MIAK. Methyl isoamyl ketone is a colorless, clear liquid with a pleasant odor.

The oral LD_{50} value of methyl isoamyl ketone in rats is 1.67 g/kg (Smyth et al. 1962). No data relating exposure levels to specific effects in humans have been reported. However, the ACGIH (1986, p. 400) believes that MIAK is likely to be more irritating and a more potent narcotic than is the case for methyl isobutyl ketone.

The NIOSH criteria document on the ketones states that "because methyl isoamyl ketone contains one more carbon atom than does methyl isobutyl ketone, methyl (isoamyl) ketone might produce irritation and narcosis at concentrations at least as low as those at which methyl isobutyl ketone produces these effects," and NIOSH thus recommends a 50-ppm TWA for MIAK, corresponding to NIOSH's recommendation for methyl isobutyl ketone (NIOSH 1978).

OSHA is proposing an 8-hour TWA limit of 50 ppm for methyl isoamyl ketone. The Agency preliminarily concludes that this limit will protect workers against the risk of narcotic and irritant effects potentially associated with exposure to MIAK at the levels permitted in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for methyl isoamyl ketone. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

METHYL ISOPROPYL KETONE
CAS: 563–80–4; Chemical Formula: $(CH_3)_2CHCOCH_3$
H.S. No. 1262

OSHA currently has no limit for methyl isopropyl ketone (MIPK). The ACGIH recommends a TLV–TWA of 200 ppm. Methyl isopropyl ketone is a colorless, flammable liquid.

Animal studies have shown MIPK to have an acute toxicity somewhat greater than that of diethyl ketone and somewhat lesser than that of di-n-propyl ketone or methyl-n-propyl ketone (ACGIH 1986, p. 405). Rats exposed for 4 hours at a concentration of 5700 ppm died (NIOSH 1977). Other data concerning the inhalation toxicity of MIPK are lacking.

OSHA proposes a limit of 200 ppm TWA for methyl isopropyl ketone. The Agency preliminarily concludes that this limit will protect workers against the risk of irritation associated with exposure to this (and other) ketone(s) in the workplace at the levels permitted by the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for methyl isopropyl ketone. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

METHYL PARATHION
CAS: 298-00-0; Chemical Formula: $C_8H_{10}NO_5PS$
H.S. No. 1265

OSHA currently has no limit for methyl parathion. The ACGIH recommends a TLV-TWA of 0.2 mg/m³, with a skin notation. NIOSH recommends a TWA of 0.2 mg/m³ and a skin notation for methyl parathion. Methyl parathion is a tan to brown liquid with a pungent odor like that of garlic.

Methyl parathion is an acetylcholinesterase inhibitor, and excessive exposure can cause sweating, salivation, diarrhea, bradycardia, bronchoconstriction, muscle fasciculations, and coma. Methyl parathion's acute oral LD_{50} for male rats is almost identical to that of parathion, i.e., 10 to 25 mg/kg; for female rats, the LD_{50} is 24 mg/kg, or approximately one-sixth that of parathion. By the dermal route, methyl parathion is much less toxic than parathion, with an LD_{50} of 67 mg/kg in rats of both sexes (Hayes, as cited in ACGIH 1986, p. 407). Erythrocyte cholinesterase activity was inhibited in dogs fed methyl parathion for 12 weeks at a rate corresponding to approximately 24 mg/day; inhibition of both plasma and erythrocyte cholinesterase activity occurred at doses of 70 mg/day, without accompanying illness (Williams et al. 1959). Dogs fed 6 mg/day methyl parathion for 12 weeks showed no effects from such exposures (Williams et al.). Lifetime feeding studies of rats and mice fed diets containing methyl parathion concentrations of up to 40 ppm and up to 125 ppm, respectively, produced no evidence of cancer (NCI 1979).

Plasma and erythrocyte cholinesterase levels did not differ by more than 20 percent in subjects exposed at 7, 7.5, 8, or 9 mg/man/day, compared with controls (Moeller and Rider 1963). Tiess and associates (1982) have reported a case of protracted methyl parathion poisoning resulting from both percutaneous and inhalation exposure; Dille and Smith (1964) attribute the long-term neuro-psychiatric illness of two pilots to exposure to methyl parathion and other cholinesterase-inhibiting agents. Chronic exposure to small doses of methyl parathion have not caused chromosomal effects (DeCassia Stocco et al. 1982).

OSHA proposes a limit of 0.2 mg/m³ TWA for methyl parathion, with a skin notation. The Agency preliminarily concludes that this limit will protect workers against the risk of acetylcholinesterase inhibition potentially associated with workplace exposures at the levels permitted in the absence of any OSHA limit. The skin notation will protect workers from the risk of percutaneous absorption of this substance. The health evidence forms a reasonable basis for proposing a new limit for methyl parathion. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

METHYLCYCLOHEXANE
CAS: 108-87-2; Chemical Formula: C_7H_{14}
H.S. 1268

OSHA has a current 8-hour TWA limit of 500 ppm for methylcyclohexane. The ACGIH recommends a limit of 400 ppm TWA for this colorless liquid.

Lehmann and Flury (1943) indicate that the acute toxicity of methylcyclohexane is greater than that of heptane but less than that of octane. Lazarew (1929) found that a 2-hour exposure to a concentration of 7500 to 10,000 ppm caused prostration in mice, and exposure to 10,000 to 12,500 ppm caused death. Treon, Crutchfield, and Kitzmiller (1943) reported that exposure to 1200 ppm had no effect in rabbits, and prolonged exposures to 370 ppm had no effect in monkeys. Methylcyclohexane's histologic effects in animals resemble those of cyclohexane; the liver and kidney are the sites affected (ACGIH 1986, p. 384).

OSHA proposes an 8-hour TWA limit of 400 ppm for methylcyclohexane. The Agency preliminarily concludes that this limit will protect workers against the risk of irritation associated with exposure to methylcyclohexane and other alicyclic hydrocarbons at the levels permitted by the current OSHA limit. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for methylcyclohexane if the Agency determines that this limit will substantially reduce significant risk.

2-METHYLCYCLOPENTADIENYL MANGANESE TRICARBONYL
CAS: 12108-13-3; Chemical Formula: $(CH_3)C_5H_6-Mn(CO)_3$
H.S. No. 1271

OSHA currently has no limit for 2-methylcyclopentadienyl manganese tricarbonyl (C1-2). The ACGIH recommends a TLV-TWA of 0.2 mg/m³, as manganese, with a skin notation. C1-2 is a dark orange liquid with a faintly pleasant odor; it is a complex organic compound containing about 25 percent manganese by weight.

2-Methylcyclopentadienyl Mn tricarbonyl is highly toxic in its concentrated form, causing adverse effects primarily on the central nervous system. It is somewhat irritating to the eyes but skin contact does not produce irritation or sensitization: C1-2 is readily absorbed through the skin (ACGIH 1986, p. 387). Animal studies indicate that C1-2 has a toxicity similar to that of tetraethyl lead and is highly toxic by all routes of exposure (U.S. Navy Smoke Abatement Additive, as cited in ACGIH 1986, p. 387).

The single-dose oral LD_{50} for rats is 23 or 39 mg/kg, depending on sex. The skin LD_{50} for rabbits is 1692 ± 145 mg/kg, and the 1-hour inhalation LC_{50} for rats is about 350 mg/m³ (The Ethyl Corporation, as cited in ACGIH 1986, p. 387). Toxic exposures by all routes produce rapidly appearing symptoms of mild excitement, hyperactivity, tremors, severe clonic spasms, weakness, respiratory distress, and occasional clonic convulsions, followed by terminal coma (U.S. Navy Smoke Abatement Additive, as cited in ACGIH 1986, p. 387).

Acute exposure causes damage to the liver, kidneys, and cerebral cortex, as well as changes in lung tissue (ACGIH 1986, p. 387). Browning (1966) observed chronic bronchitis, peribronchitis, interstitial pneumonia, and lung abscesses in animals that subsequently died from long-term inhalation exposure to C1-2: exposure to C1-2 concentrations of approximately 12 mg/m³ for 100 days produced no deviation in weight gain patterns and no gross or microscopic changes in two dogs (Browning 1966). The liver and kidneys are the principal target organs associated with acute overexposures; the lungs of overexposed animals were hemorrhagic (Browning 1966).

In humans, skin contact should be entirely avoided. A 5- to 15-ml spill on one worker's hand and wrist was reported to have caused "thick tongue," nausea, giddiness, and headache within 3 to 5 minutes (U.S. Navy Smoke Abatement Additive, as cited in ACGIH 1986, p. 387).

OSHA proposes a PEL of 0.2 mg/m³ TWA as manganese, with a skin notation, for 2-methylcyclopentadienyl manganese tricarbonyl. The Agency preliminarily concludes that this limit will protect workers against the risk of CNS effects and systemic damage that exists in the absence of any OSHA limit for this substance. A skin notation is proposed because of C1-2's ability to penetrate the skin rapidly. The health evidence forms a reasonable basis for proposing a new limit for manganese. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

MONOCROTOPHOS (AZODRIN)
CAS: 6923-22-4; Chemical Formula: $C_7H_{14}NO_5P$
H.S. No. 1279

OSHA currently has no limit for the systemic insecticide monocrotophos. The ACGIH recommends a TLV-TWA of 0.25 mg/m³ for this reddish-brown solid with a mild ester odor.

Monocrotophos is a highly toxic, direct acting cholinesterase inhibitor that penetrates the intact skin (ACGIH 1986, p. 416). The acute oral LD_{50} values in rats and mice range from 5.7 to 17 mg/kg in a water formulation (Brown et al. 1970; Shellenberger and Newell, as cited in ACGIH 1986, p. 416) and from 10 to 23 mg/kg in an oil formulation (Shellenberger and Newell, as cited in ACGIH 1986, p. 416). These authors also report a percutaneous LD_{50} in the rabbit that ranges from 112 to 709 mg/kg, depending on the vehicle used. A 2-year dietary study of rats ingesting 0, 1, 10, or 100 ppm monocrotophos revealed that both sexes in the 100 ppm group failed to gain as much weight as the controls, but autopsy showed no significant findings; plasma, erythrocyte, and brain cholinesterase decreased at the two highest dose levels but were unaffected at 1 ppm (Johnston 1966, as cited in ACGIH 1986, p. 416; Johnston et al. 1967, as cited in ACGIH 1986, p. 416). Another 2-year feeding study in dogs administered doses of up to 16 ppm monocrotophos revealed no adverse effects at levels of 0.16 and 1.6 ppm, but serious cholinesterase reduction was observed at the 16 ppm level (Johnston 1966, as cited in ACGIH 1986, p. 416; Johnston et al. 1967, as cited in ACGIH 1986, p. 416). Metabolism studies in rats and goats indicate that monocrotophos is excreted rapidly in the rat and does not accumulate in the body (Bull and Lindquist, as cited in ACGIH 1986, p. 416); goats given labeled monocrotophos by mouth showed only traces of the material in their milk (Menzer and Casida; Potter, both as cited in ACGIH 1986, p. 416). Inhalation exposure of rats to an unknown concentration of 75 percent monocrotophos in air for 1 hour was not lethal; a 4-hour exposure to an unknown concentration of the aerosol (0.4 and 0.75 percent) was fatal to 2 out of 6 (0.4 percent aerosol) and 5 out of 8 rats (0.75 percent aerosol). Head-only exposure to the 0.4 percent aerosol resulted in the death of one of eight animals (Wilson, as cited in ACGIH 1986, p. 416).

Intravenous injection of radiolabeled monocrotophos in human volunteers showed maximum excretion at 4 to 8 hours, with 67 ± 5 percent of the material in the urine; absorption of 14 ± 7 percent occurred when the radiolabeled material was applied to the forearm; 33 ± 9 percent of the applied dose was absorbed when it was covered with a vapor-proof film for 72 hours (Maibach 1970). Although gauze patches attached to the clothing and skin of field workers attested to the presence of monocrotophos, no cholinesterase inhibition resulted in post-exposure examinations at 3 hours and at 3 and 7 days (Maibach, as cited in ACGIH 1986, p. 416).

OSHA is proposing a PEL of 0.25 mg/m³ TWA for monocrotophos. The Agency preliminarily concludes that this limit will protect workers against the risk of cholinesterase inhibition potentially associated with exposure in the workplace at the levels permitted by the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for monocrotophos. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

MORPHOLINE
CAS: 110-91-8; Chemical Formula: C_4H_9NO
H.S. No. 1281

OSHA has a current limit of 20 ppm, with a skin notation, for morpholine. The ACGIH recommends a 20-ppm TWA limit and a TLV-STEL of 30 ppm, as well as a skin notation. Morpholine is a colorless liquid with an amine-like odor.

Exposure to morpholine produces nasal and bronchial irritation and liver and kidney impairment in animals (Shea 1939); the substance readily penetrates the skin and is highly irritating to the eyes (Jefferson Chemical Company, Inc. 1961, as cited in ACGIH 1986, p. 417). The single oral LD_{50} in rats is 1.05 g/kg (range: 0.95 to 1.16 g/kg), and the single skin LD_{50} for 24-hour contact is 0.5 mg/kg (Smyth, Carpenter, Weil, and Pozzani 1954). A 1-hour exposure to concentrated vapor was not fatal in rats, nor was an 8-hour exposure to 8000 ppm (Smyth, Carpenter, Weil, and Pozzani 1954). Rats were exposed for 8 hours daily to a concentration of 16,000 ppm for a total of 5 days; after the first day, all animals showed severely reddened thoracic walls, and one fatality (from kidney and liver congestion) occurred. A similar fatality occurred on the third day; on day 4, a third rat died, and postmortem examination revealed degeneration of the epithelial lining of the kidney tubules. Three additional deaths occurred after the exposures had ended; autopsy revealed thickened alveoli, emphysema, and liver and kidney effects (Shea 1939).

Reporting on his own reactions to morpholine exposure at a concentration of 12,000 ppm, Shea (1939) complained of nose irritation (after 1 minute) and coughing (after 90 seconds); in addition, when he transferred morpholine by pipette, he experienced sore throat and mucosal irritation. All symptoms disappeared after the experiment stopped (Shea 1939). Skin contact poses a moderately high degree of hazard, which diminishes as the product is diluted with water to less than 25 percent (Jefferson Chemical Company, Inc. 1961, as cited in ACGIH 1986, p. 417). Respiratory irritation but no chronic effects have been reported as a result of industrial exposure (Patty 1963). In comparison with ammonia, morpholine has a greater potential for systemic toxicity (ACGIH 1986, p. 417).

OSHA proposes an 8-hour TWA limit for morpholine of 20 ppm TWA and a 15-minute STEL of 30 ppm, with a skin notation. The Agency preliminarily concludes that this limit will protect workers against the risk of eye and respiratory tract irritation potentially associated with exposures in the absence of a short-term limit. OSHA is retaining the skin notation for morpholine because of its ability to be absorbed through the skin. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for morpholine if the Agency determines that this limit will substantially reduce significant risk.

NITRIC ACID
CAS: 7697-37-2; Chemical Formula: HNO_3
H.S. No. 1286

OSHA currently has an 8-hour limit of 2 ppm TWA for nitric acid. The ACGIH recommends the same TWA limit and a 15-minute STEL of 4 ppm, and NIOSH recommends a TWA limit of 2 ppm. Nitric acid is a fuming colorless or yellowish liquid.

Rats receiving a single exposure to nitric acid mist at a concentration of 63 mg/m³ exhibited no apparent adverse effects (Diggle and Gage 1954).

Chronic exposure to airborne nitric acid vapor or mist at unspecified levels was reported to cause chronic bronchitis, pneumonitis (Fairhall 1957), and tooth erosion (Lynch and Bell 1947).

Nitric acid's irritant potential is considered similar to that of other strong acids; it typically exists in conjuction with nitrogen dioxide, which is regarded as being more hazardous (ACGIH 1986, p. 428).

OSHA is proposing a PEL of 2 ppm TWA and a STEL of 4 ppm for nitric acid. The Agency preliminarily

concludes that this combined limit will protect workers against the risk of irritation, chronic pulmonary disease, and dental corrosion that potentially exists at exposures at the levels permitted by the TWA alone. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for nitric acid if the Agency determines that this limit will substantially reduce significant risk.

p-NITROANILINE
CAS: 100-01-6; Chemical Formula: $NO_2C_6H_4NH_2$
H.S. No. 1287

OSHA currently has a skin notation and a limit of 1 ppm TWA (6 mg/m³) for p-nitroaniline (PNA). The ACGIH recommends a limit of TLV-TWA 3 mg/m³, with a skin notation. para-Nitroaniline usually exists in the form of yellow needles.

p-Nitroaniline is readily absorbed through the skin and is a strong methemoglobin-forming agent, and prolonged exposure can cause liver damage (ACGIH 1986, p. 430). Anderson (1946) reported several cases of PNA-poisoning among shipboard workers assigned to clean up a p-nitroaniline spill; one man with a history of liver disease became jaundiced and died, and the other exposed workers became cyanotic and complained of headache, sleepiness, weakness, and respiratory distress (Anderson 1946). It has also been reported that children who ingested p-nitroaniline that was contained in wax crayons subsequently became ill (Rieders and Brieger 1953).

Several investigators (Anderson 1946; Gupta 1953; Fairhall 1957; Linch 1974) have concluded that the nitroanilines are more hazardous than aniline, and, on this basis, the ACGIH has recommended a TWA limit for PNA that is lower than the limit for aniline (ACGIH 1986, p. 430).

OSHA is proposing a PEL of 3 mg/m³ TWA for p-nitroaniline, with a skin notation. The Agency preliminarily concludes that this limit will protect workers against the risk of methemoglobinemia and liver damage potentially associated with exposure to PNA at levels above 3 mg/m³. The Agency is retaining the skin notation because this substance is readily absorbed through the skin. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for p-nitroaniline if the Agency determines that this limit will substantially reduce significant risk.

NITROTOLUENE
CAS: 88-72-2; 99-08-1; 99-99-0; Chemical Formula: $CH_3C_6H_4NO_2$
H.S. No. 1292

OSHA currently has an 8-hour limit of 5 ppm, with a skin notation, for nitrotoluene. The ACGIH recommends a TLV-TWA of 2 ppm, also with a skin notation. The ortho- and meta-isomers of nitrotoluene are yellow liquids; the para-isomer is also yellow, but exists in crystalline form.

Nitrotoluene is one of the aromatic nitrogen compounds that may cause methemoglobin formation. Linch (1974) has studied the nitrotoluene isomers and reported that they have relatively low emiagenic potential; he considered nitrotoluene comparable to aniline in its toxic effects (Linch 1974). Cases of poisoning as a result of exposure to nitrotoluene are rare (von Oettingen 1941).

OSHA proposes an 8-hour TWA limit of 2 ppm, with a skin notation, for nitrotoluene. The Agency preliminarily concludes that this limit will protect workers against the risk of methemoglobinemia potentially associated with exposure to this substance; the skin notation is retained because of nitrotoluene's capacity to penetrate the skin. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for nitrotoluene if the Agency determines that this limit will substantially reduce significant risk.

NONANE
CAS: 111-84-2; Chemical Formula: $CH_3(CH_2)_7CH_3$
H.S. No. 1293

OSHA currently has no limit for nonane. The ACGIH recommends a TLV-TWA of 200 ppm for this colorless liquid.

The toxicity of nonane is approximatelv equal to that of VM&P naphtha. Naphtha has a 4-hour inhalation LC_{50} for rats of 3400 ppm, while nonane has an LC_{50} of 3200 ppm (Carpenter et al. 1975, 1978). These investigators found a no-effect level of 590 ppm nonane for rats exposed 6 hours/day, 5 days/week for a 65-day period; under the same exposure conditions, a no-effect level of 560 ppm was reported for rats exposed to VM&P naphtha (Carpenter et al. 1975, 1978). Earlier studies of octane and heptane have resulted in much higher LC_{50} values for mice, i.e., 13,500 ppm and 16,000 ppm, respectively, for 30- to 60-minute exposures (Flury and Zernik 1931). Swann and associates (1974) have reported similarly high values for octane and hexane in mice; mice died from respiratory arrest after 3 to 5 minutes of exposure to 16,000 ppm of octane or to 48,000 ppm of hexane (Swann et al. 1974).

OSHA proposes an 8-hour TWA limit of 200 ppm for nonane. The Agency preliminarily concludes that this limit will protect workers against the risk of narcotic effects potentially associated with exposure to nonane at the levels permitted by the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for nonane. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

OXALIC ACID
CAS: 144-62-7; Chemical Formula: $H_2C_2O_4$
H.S. No. 1299

OSHA currently has a limit of 1 mg/m³ for oxalic acid. The ACGIH has a TLV-TWA of 1 mg/m³ and recommends a TLV-STEL of 2 mg/m³. Anhydrous oxalic acid is usually in the form of a white powder; the dihydrate form is a colorless, odorless, crystalline substance.

Oxalic acid is known to produce severe burns of the eyes, mucous membranes, and skin (The Merck Index 1983, p. 991). There have been human fatalities from ingesting as little as 5 grams of oxalic acid. It appears that these deaths were caused by oxalic acid's ability to disturb the calcium-potassium balance in critical tissues (Klauder, Shelanski, and Gabriel 1955). Solutions of 5- to 10 percent oxalic acid have also been reported to irritate the skin on prolonged exposure.

Because of oxalic acid's severe acute toxicity, OSHA is proposing an 8-hour TWA limit of 1 mg/m³ PEL and a STEL of 2 mg/m³. The Agency preliminarily concludes that both of these limits are required to protect exposed workers from the risk of severe eye and skin burns and respiratory tract irritation that could result from elevated short-term exposures at the present TWA limit. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for oxalic acid if the Agency determines that this limit will substantially reduce significant risk.

PERCHLORYL FLUORIDE
CAS: 7616-94-6; Chemical Formula: ClO_3F
H.S. No. 1309

OSHA's current 8-hour TWA limit for perchloryl fluoride is 3 ppm. The ACGIH has a TLV-TWA of 3 ppm and a STEL of 6 ppm for this colorless gas with a sweet odor.

The 4-hour LD_{50}s in rats and mice were 385 and 630 ppm, respectively.

Dogs exposed for 4 hours to 220- to 450-ppm concentrations of the vapor, followed by exposure to 620 ppm for 2.5 hours, became hyperpneic and cyanotic and showed increased methemoglobin. Dogs succumbing from these exposures had pigment deposition in the liver, spleen, and bone marrow; alveolar hemorrhage and collapse; and emphysema.

Exposure to 185 ppm for 6 hours/day, 5 days/week for 7 weeks killed 18 of 20 rats, 20 of 30 mice, and all exposed guinea pigs (Greene, Colburn, Donati, and Weeks 1960). These animals had difficulty breathing, became cyanotic, and developed alveolar edema and methemoglobinemia; at autopsy, they showed fluorosis; patchy lungs; enlarged spleens; and hemosiderosis of the kidneys, spleen, and liver. When animals were exposed on a similar regimen but to a concentration of 104 ppm for 6 weeks, all guinea pigs but only 1 of 20 rats died (Greene, Colbourn, Donati, and Weeks 1960). After a 6-month exposure to 24 ppm, bone fluoride levels increased 4-fold in guinea pigs, 3-fold in rats, and about 50 percent in dogs. Animals exposed at 24 ppm showed no signs of irritation (Greene, Colbourn, Donati, and Weeks 1960).

OSHA is proposing an 8-hour TWA of 3 ppm and a STEL of 6 ppm for perchloryl fluoride. The Agency preliminarily concludes that this combined limit will protect workers from the risk of fluorosis and hematologic effects potentially associated with exposures to perchloryl fluoride at the levels permitted in the absence of a short term limit. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for perchloryl fluoride if the Agency determines that this limit will substantially reduce significant risk.

MEVINPHOS (PHOSDRIN)
CAS: 7786-34-7; Chemical Formula:
H.S. No. 1320

OSHA currently has an 8-hour TWA limit of 0.1 mg/m³, with a skin notation, for mevinphos (phosdrin). The ACGIH recommends a TLV-TWA of 0.01 ppm (0.1 mg/m³) and a TLV-STEL of 0.03 ppm (0.3 mg/m³), also with a skin notation. Phosdrin is a colorless liquid. The commercial product is a mixture of cis- and trans-isomers that have a yellow color.

The acute oral LD_{50} of phosdrin is 4 to 8 mg/kg for male mice and 6 to 8 mg/kg for female rats (Shell Chemical Corporation 1956, as cited in ACGIH 1986, p. 412). Phosdrin is a cholinesterase inhibitor and has been reported to cause slight plasma cholinesterase depression but no decrease in brain cholinesterase activity in rats fed 2 to 5 ppm. The compound may be absorbed dermally and by inhalation or ingestion; the action of the compound is direct and immediate (Cleveland and Treon 1961). The dermal LD_{50} in rats has been reported to be 4.5 mg/kg (Gaines 1969). Chronic feeding of rats demonstrated a minimal lethal dose of between 100 and 200 ppm. Cholinesterase activity decreased continually when sublethal doses were administered until a maximum reduction in RBC cholinesterase activity of 25 percent was achieved on the 27th day of the administration of 1.5 to 20 mg doses (Huelse and Federspil 1975).

In industry, the primary hazards associated with exposure to phosdrin are absorption of phosdrin through the skin, lung, and mucous membranes, which causes liver damage (Natoff 1970). Phosdrin intoxication is reported to occur in humans, with accompanying symptoms of headache, visual distortion, weakness, cramps, diarrhea, pain, and respiratory distress. Severe exposure may cause convulsions; in one reported case, some symptoms (anxiety, depression, vertigo, and nystagmus) persisted for as long as 4 months (Zavon, as cited in ACGIH 1986, p. 412).

OSHA is proposing a PEL of 0.01 ppm TWA and a STEL of 0.03 ppm for phosdrin, with a skin notation. The Agency preliminarily concludes that these limits will protect workers against the risk of cholinesterase inhibition and hepatic injury that results from absorption of phosdrin through the skin and mucous membranes and from exposure by the inhalation and oral routes. OSHA believes that these limits will substantially reduce this risk. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for phosdrin if the Agency determines that this limit will substantially reduce significant risk.

PHOSPHORUS OXYCHLORIDE
CAS: 10025-87-3; Chemical Formula: $POCl_3$
H.S. No. 1323

OSHA has no current limit for phosphorus oxychloride. The ACGIH recommends a TLV-TWA of 0.1 ppm and a TLV-STEL of 0.5 ppm. This clear, colorless, fuming liquid has a pungent odor.

The primary hazards associated with inhalation of phosphorus oxychloride vapor are irritation of the eyes and respiratory tract, as well as narcotic effects, gastric irritation, pulmonary edema, and nephritis (The International Technical Information Institute 1978).

Weeks and associates (1964) reported 4-hour LC_{50} values for phosphorus oxychloride of 48 ppm and 52 ppm for guinea pigs and rats, respectively. They also observed that ammonia vapor mediates the irritant effects of exposure to phosphorus oxychloride without significantly altering this LC_{50} value (Weeks et al. 1964).

Both chronic and acute occupational intoxication have been reported to occur among workers exposed to phosphorus oxychloride (Sassi 1954).

OSHA is proposing a PEL of 0.1 ppm TWA and a 15-minute STEL of 0.5 ppm for phosphorus oxychloride. The Agency preliminarily concludes that these limits will reduce the risk of narcotic effects and systemic poisoning potentially associated with acute and chronic exposure at the uncontrolled levels permitted by the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for phosphorus oxychloride. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

PHOSPHORUS PENTASULFIDE
CAS: 1314-80-3; Chemical Formula: P_2S_5
H.S. No. 1324

OSHA currently has a limit of 1 mg/m³ as an 8-hour TWA for phosphorus pentasulfide. The ACGIH also recommends a limit of 1 mg/m³ TWA and a 15-minute STEL of 3 mg/m³. These limits are the same as those proposed for phosphoric acid, which the ACGIH believes to be approximately as toxic as phosphorus pentasulfide. Phosphorus pentasulfide is a greenish-yellow crystalline mass with an odor like that of rotten eggs.

The primary hazard associated with exposure to phosphorus pentasulfide is respiratory irritation (Smyth 1956). In the presence of moisture, phosphorus pentasulfide is rapidly hydrolyzed to phosphoric acid and hydrogen sulfide. No toxicity data are available concerning phosphorus pentasulfide per se.

OSHA is proposing a PEL of 1 mg/m³ as an 8-hour TWA, with a 15-minute STEL of 3 mg/m³, for phosphorus pentasulfide. The Agency preliminarily concludes that both of these limits are necessary to reduce the risk of respiratory irritation associated with exposure to this substance at the higher concentrations permitted at the current PEL. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for phosphorus pentasulfide if the

Agency determines that this limit will substantially reduce significant risk.

PHTHALIC ANHYDRIDE
CAS: 85-44-9; Chemical Formula:
$C_6H_4(CO)_2O$
H.S. No. 1326

OSHA currently has an 8-hour TWA limit of 2 ppm for phthalic anhydride. The ACGIH recommends a limit of 1 ppm TWA. Phthalic anhydride exists in the form of white crystalline needles with a mild odor.

The primary exposure hazards associated with phthalic anhydride are severe skin, eye, and respiratory irritation. The substance can also produce skin and, perhaps, pulmonary sensitization (Patty 1963). Baader (1955) has reported irritant effects in animals exposed to 30 mg/m³ (approximately 5 ppm) phthalic anhydride in air.

In studies of workers exposed to phthalic anhydride, symptoms of respiratory tract injury as well as bronchitis, eye irritation, and nasal bleeding have been reported. Precise exposure concentrations were not detectable by the analytic method being used, which had a limit of detection of 25 mg/m³ (i.e., of 4 ppm or lower) (Baader 1955; Menschick 1955). Other industrial acid anhydrides (e.g., tetrachlorphthalic anhydride and maleic anhydride) are considered more irritating than phthalic anhydride (ACGIH 1986, p. 489).

OSHA is proposing an 8-hour TWA limit of 1 ppm for phthalic anhydride, compared with the limit of 0.25 ppm being proposed for maleic anhydride. The Agency preliminarily concludes that a 1 ppm limit will reduce the risk of respiratory irritation and the potential risk of skin and pulmonary sensitization that exists for workers exposed at higher levels permitted by the current PEL. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for phthalic anhydride if the Agency determines that this limit will substantially reduce significant risk.

PROPARGYL ALCOHOL
CAS: 107-19-7; Chemical Formula:
$HC{\equiv}CCH_2OH$
H.S. No. 1335

OSHA has no current limit for propargyl alcohol. The ACGIH has established an 8-hour TWA of 1 ppm, with a skin notation, for this light to straw-colored liquid, which smells like geraniums.

In rats, guinea pigs, and mice, the oral LD_{50}s are 70, 60, and 50 mg/kg, respectively; the inhalation LC_{50} in both the rat and mouse is reported to be about 850 ppm (NIOSH 1977).

Propargyl alcohol is a primary skin irritant, but it is not a skin sensitizer (Antara Chemicals 1952, as cited in ACGIH 1986, p. 496). The toxicity of propargyl alcohol is estimated to be equal to that of allyl alcohol (oral LD_{50} in rats of 64 mg/kg) (NIOSH 1977). The ACGIH limit is based on the structural and toxicological similarity of propargyl alcohol to allyl alcohol (ACGIH 1986, p. 496).

OSHA is proposing an 8-hour TWA for propargyl alcohol of 1 ppm, with a skin notation. The Agency preliminarily concludes that these limits will protect workers against the risk of skin and mucous membrane irritation potentially associated with exposure to this substance at the levels permitted in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for propargyl alcohol. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

PROPIONIC ACID
CAS: 79-09-4; Chemical Formula:
CH_3CH_2COOH
H.S. No. 1336

OSHA currently has no limit for propionic acid. The ACGIH recommends a TLV-TWA of 10 ppm and a TLV-STEL of 15 ppm. Propionic acid is a colorless, oily liquid with a pungent odor.

The primary health effects associated with exposure to propionic acid are skin burns and irritation of the eyes and respiratory system. Smyth and co-workers (1962) reported that the oral LD_{50} for rats is 4.3 g/kg; NIOSH (1977) stated that the intravenous LD_{50} for mice is 625 mg/kg and the skin absorption LD_{50} for rabbits is 500 mg/kg. Inhalation of the saturated vapor for 8 hours caused no rat fatalities (ACGIH 1986, p. 498).

Acute industrial exposures have been reported to cause mild to moderate skin burns, eye irritation, and, in a single incident, asthmatic cough. No irritation was observed as a consequence of exposures in humans averaging below 0.25 ppm with excursions to 2.1 ppm in an 8 hour-period (Dow Chemical Company, as cited in ACGIH 1986, p. 498).

OSHA proposes limits of 10 ppm TWA and 15 ppm as a 15-minute STEL for propionic acid. The Agency preliminarily concludes that both of these limits are required to protect workers against the risk of eye and respiratory tract irritation that exists at the levels permitted by the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for propionic acid. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

n-PROPYL ACETATE
CAS: 109-60-4; Chemical Formula:
$CH_3COOCH_2CH_2CH_3$
H.S. No. 1338

OSHA currently has an 8-hour TWA limit of 200 ppm for n-propyl acetate. The ACGIH also recommends a 200 ppm TWA limit and a TLV-STEL of 250 ppm. n-Propyl acetate is a pleasant-smelling liquid.

The primary health effects associated with exposure to n-propyl acetate are narcosis and eye and respiratory irritation. The limiting 5-hour narcotic concentrations for cats and mice have been reported as 9000 ppm and 6000 ppm, respectively (Flury and Wirth 1933). n-Propyl acetate's narcotic action is 1.3 times that of ethyl acetate; salivation and irritation of cats' eyes occurred at 2600 ppm (Flury and Wirth 1933). A 4-hour exposure at 8000 ppm killed four of six rats (Smyth 1964, as cited in ACGIH 1986, p. 500).

OSHA is proposing a PEL of 200 ppm TWA and a STEL of 250 ppm for n-propyl acetate. The Agency preliminarily concludes that both of these limits are required to prevent the risk of narcosis and eye and respiratory tract irritation that exists for workers exposed to levels above the level permitted by the 8-hour TWA limit alone. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for n-propyl acetate if the Agency determines that this limit will substantially reduce significant risk.

PROPYL ALCOHOL
CAS: 71-23-8; Chemical Formula:
$CH_3CH_2CH_2OH$
H.S. No. 1339

OSHA currently has a limit of 200 ppm TWA for n-propyl alcohol. The ACGIH recommends the same TWA limit, a 250-ppm 15-minute STEL, and a skin notation. Propyl alcohol is a colorless liquid with an alcohol-like odor.

The primary health effect associated with exposure to propyl alcohol is mild narcosis. Propyl alcohol's toxicity is somewhat greater than that of isopropyl alcohol (Gleason, Gosselin, and Hodge 1963).

The inhalation LD_{50} for propyl alcohol in rats is reported as 1.9 g/kg (Smyth, Carpenter, Weil, and Pozzani 1954). Starrek reported deep narcosis in mice inhaling the vapor at a concentration of 4100 ppm for 240 minutes and of 24,500

ppm for 60 minutes; ataxia appeared in 90 to 120 minutes at 3250 ppm (Starrek in Patty 1963 pp. 1434–1438). These effects are almost twice as intense as those reported for exposure to the vapor of isopropyl alcohol. The dermal LD_{50} in rabbits is 5040 mg/kg (Sax 1984).

Nelson, Ege, Ross, and associates (1943) reported mild eye, nose, and throat irritation in humans exposed at 400 ppm to the vapor of isopropyl alcohol, but no data exist on human sensory response to propyl alcohol vapor. The ACGIH (1986, p. 500) reports that many industrial hygienists consider the vapor of propyl alcohol to be more irritating to the throat than the vapor of the isomer.

OSHA is proposing an 8-hour TWA PEL of 200 ppm and a STEL of 250 ppm for propyl alcohol, with a skin notation. The Agency preliminarily concludes that these limits will protect workers against the risk of narcosis and irritation that exists at levels above the PEL. OSHA also proposes to add a skin notation because propyl alcohol can be absorbed through the skin. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for propyl alcohol if the Agency determines that this limit will substantially reduce significant risk.

PROPYLENE OXIDE
CAS: 75–56–9; Chemical Formula: CH_3CHOCH_2
H.S. No. 1344

OSHA currently has an 8-hour TWA limit of 100 ppm for propylene oxide. The ACGIH recommends a limit of 20 ppm TLV–TWA. Propylene oxide is a colorless, highly flammable, volatile, and ethereal liquid.

The health hazards associated with exposure to this substance are primary skin, eye, and respiratory irritation, as well as central nervous system depression. The oral LD_{50} values reported for rats and guinea pigs are 930 mg/kg and 690 mg/kg, respectively. In mice, the inhalation LC_{50} has been reported to be at 1740 ppm for 4 hours. Dogs and guinea pigs exposed for 4 hours at 2000 and 4000 ppm, respectively, died (NIOSH 1977). Although some species tolerate daily exposures to 200 ppm, all species tested tolerated 100 ppm without ill effects (Rowe, Hollingsworth, Oyen et al. 1956). Jacobson and associates (1956) considered the toxic effects of propylene oxide to be one-half to one third as intense as those of ethylene oxide (Jacobson, Hackley, and Feinsilver 1956).

Corneal burns and skin necrosis, as well as respiratory and pulmonary irritation, have been reported in humans as a result of direct contact with the liquid or vapor (Patty 1963); central nervous system effects include ataxia, incoordination, and general depression.

OSHA proposes an 8-hour TWA limit of 20 ppm for propylene oxide to protect workers against the risk of primary irritation and CNS depression potentially associated with exposure to higher levels. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for propylene oxide if the Agency determines that this limit will substantially reduce significant risk.

SILICON TETRAHYDRIDE
CAS: 7803–62–5; Chemical Formula: SiH_4
H.S. No. 1361

OSHA currently has no limit for silicon tetrahydride. The ACGIH limit of 5 ppm as an 8-hour TWA was established in 1983. Silicon tetrahydride, which is a colorless gas, is used in the manufacture of semiconductors.

Studies of rats exposed to silicon tetrahydride at levels of 126 ppm for 1 hour (Matheson Gas Products 1971, as cited in ACGIH 1986, p. 528) and at 1400 ppm for 6 hours (Union Carbide Corporation 1980, as cited in ACGIH 1986, p. 528) have failed to identify any systemic effects associated with exposure to this chemical. Sax (1984) lists the effects of acute exposure to silicon tetrahydride as moderate irritation to the eyes, skin, and mucous membranes.

OSHA is proposing a limit of 5 ppm TWA for silicon tetrahydride. The Agency preliminarily concludes that this limit will protect exposed workers from the risk of eye, skin, and upper respiratory tract irritation potentially associated with exposure to this substance at the unregulated levels permitted by the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for silicon tetrahydride. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

SULFURYL FLUORIDE
CAS: 2699–79–8; Chemical Formula: SO_2F_2
H.S. No. 1379

The current OSHA limit for sulfuryl fluoride is 5 ppm as an 8-hour TWA. The ACGIH recommends 5 ppm as a TWA, with the addition of a STEL of 10 ppm. Sulfuryl fluoride is a colorless gas with a sulfide odor. When selecting this limit, the ACGIH took into consideration the fact that, compared with hydrogen fluoride (TLV–TWA ceiling of 3 ppm), only a small portion of the inhaled gas is retained and converted to inorganic fluorides.

In extensive animal studies conducted by the Dow Chemical Company (1962 and 1970, as cited in ACGIH 1986, p. 546), sulfuryl fluoride was determined to exhibit one-half to one-third the acute inhalation toxicity of methyl bromide. Acute exposures of animals resulted in tremors that later developed into severe convulsions. Pulmonary edema was seen in laboratory animals following a single severe exposure. Repeated exposures of rats, guinea pigs, and mice to 20 ppm sulfuryl fluoride for 7 hours per day produced both kidney and lung injuries after 6 months. After 12 months of exposure, slight effects were seen that reversed after exposure was terminated. Some evidence of fluorosis was observed in the incisors of mice, but not in the teeth of the rats or guinea pigs (Dow Chemical Company 1962, 1970, as cited in ACGIH 1986, p. 546).

A report by Taxay (1966) that examined an incident of workplace exposure to sulfuryl fluoride noted that abdominal pain, nausea, vomiting, and itching were the major symptoms. On the day following exposure, the serum of the affected worker tested positive for fluoride.

OSHA is proposing an 8-hour TWA limit of 5 ppm and a STEL of 10 ppm for sulfuryl fluoride. The Agency preliminarily concludes that these limits will protect workers against the risks of kidney and lung injury and of fluorosis potentially associated with chronic exposure to this substance. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for sulfuryl fluoride if the Agency determines that this limit will substantially reduce significant risk.

THIONYL CHLORIDE
CAS: 7719–09–7; Chemical Formula: Cl_2OS
H.S. No. 1393

OSHA's current Z tables have no limits for thionyl chloride. The ACGIH has established a ceiling limit of 1 ppm for this substance. Thionyl chloride is a colorless to pale yellow liquid with a suffocating odor.

Thionyl chloride vapors are skin, eye, and mucous membrane irritants, probably as a result of the formation of sulfur dioxide and hydrogen chloride (ACGIH 1986, p. 572). An inhalation exposure of 17.5 ppm proved lethal to cats within 20 minutes (Sax 1979).

The ACGIH's exposure limit for thionyl chloride is based on the exposure limits for the decomposition products (hydrogen chloride and sulfur

dioxide) of thionyl chloride when mixed with water. The reaction of one mole of thionyl chloride with water produces two moles of hydrogen chloride and one of sulfur dioxide, so that 1 ppm of thionyl chloride can be shown to produce a total irritant gas concentration of 3 ppm. The exposure limit for hydrogen chloride is 5 ppm as a ceiling limit; for sulfur dioxide, the limit is a TWA of 2 ppm. Thus, "the * * * ceiling limit of 1 ppm for thionyl chloride should prevent the irritant effects of its reaction products" (ACGIH 1986, p. 572).

The Agency is proposing a ceiling limit of 1 ppm for thionyl chloride. OSHA preliminarily concludes that this limit will protect workers from the risk of irritation of the eyes, skin, and mucous membranes potentially associated with exposure to this substance at the levels permitted in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for thionyl chloride. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

TRIBUTYL PHOSPHATE
CAS: 126–73–8; Chemical Formula: $(C_4H_9)_3PO_4$
H.S. No. 1402

The current OSHA standard for tributyl phosphate is 5 mg/m³ as an 8-hour TWA. The ACGIH has established a 2.5-mg/m³ TWA. Tributyl phosphate is a clear, colorless, odorless liquid.

Tributyl phosphate's toxicity affects the skin and mucous membranes, the lungs and the central nervous system, and this substance is also a cholinesterase inhibitor.

A paper by Smyth and Carpenter (1944) reported that contact with liquid tributyl phosphate caused severe eye injury and skin irritation when tested in rabbits. Chambers and Casida (1967) found that mice injected with 1 g/kg tributyl phosphate intraperitoneally became paralyzed. A study by Vandekar (1957) in which mice were given tributyl phosphate by gavage revealed that a dose of 80 mg/kg resulted in a 1-hour period of anesthesia, and a dose of 100 mg/kg resulted in 8 to 10 minutes of anesthesia, followed by respiratory failure and death. Administered intraperitoneally to rats, tributyl phosphate inhibited cholinesterase activity and stimulated plasma beta-glucuronidase activity (Suzuki, Kikuchi, Kato et al. 1977). This substance did not exhibit mutagenic activity in bacterial or fruit fly assays (Hanna and Dyer 1975). Nausea and headache were reported by workers exposed to levels of 15 mg/m³ of tributyl phosphate (Mastromatteo, as cited in ACGIH 1986, p. 591).

OSHA is proposing to reduce the 8-hour PEL from 5 mg/m³ to 2.5 mg/m³. OSHA preliminarily concludes that this limit will protect workers against the risk of paralysis, anesthetic effects, and skin or eye irritation potentially associated with exposure to tributyl phosphate. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for tributyl phosphate if the Agency determines that this limit will substantially reduce significant risk.

TRICHLOROACETIC ACID
CAS: 76–03–9; Chemical Formula: CCl_3COOH
H.S. No. 1404

OSHA currently has no exposure limits for trichloroacetic acid. The ACGIH recommends an 8-hour TWA of 1 ppm to protect against the corrosive effects of trichloroacetic acid. Trichloroacetic acid is a relatively strong acid that forms deliquescent crystals.

The Dow Chemical Company (1977, as cited by the ACGIH 1986, p. 592) reported that the oral LD_{50} of trichloroacetic acid for rats is 3.33 g/kg. Studies on mice conducted by NIOSH (1984) established that the oral LD_{50} for this species is 4.97 g/kg, and that a 500-mg/kg dose was fatal when administered intraperitoneally.

Medical reports show mild to moderate skin and eye burns in workers exposed to unspecified levels of trichloroacetic acid; although corrosive, trichloroacetic acid is not readily absorbed by the skin (ACGIH 1986, p. 592).

OSHA is proposing an 8-hour TWA limit for trichloroacetic acid of 1 ppm. The Agency preliminarily concludes that this limit will protect exposed workers from the risk of skin and eye irritation associated with exposure to this substance at the levels permitted in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for trichloroacetic acid. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

TRIMETHYLAMINE
CAS: 75–50–3; Chemical Formula: $(CH_3)_3N$
H.S. No. 1411

OSHA presently has no exposure limit for trimethylamine. The ACGIH recommends a 10-ppm limit as an 8-hour TWA and 15 ppm as a 15-minute STEL. Trimethylamine has a pungent, fishy odor and is a gas at room temperature.

Little toxicological data are available for trimethylamine. One study reports that the intravenous LD_{50} for this substance is 90 mg/kg in mice (Dechezlepretre and Cheymol 1967). The ACGIH established the TLV for trimethylamine on the basis of its chemical similarity to dimethylamine, for which the current TLV–TWA is 10 ppm. Dimethylamine is a central nervous system depressant and cause methemoglobinemia.

OSHA is proposing an 8-hour TWA limit of 10 ppm and a STEL of 15 ppm (15 minutes) for trimethylamine. Based on analogy to dimethylamine, the Agency preliminarily concludes that these limits will protect workers exposed at previously unregulated levels from the risk of eye, mucous membrane, and upper respiratory tract irritation associated with this substance. The health evidence forms a reasonable basis for proposing a new limit for dimethylamine. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

n-VALERALDEHYDE
CAS: 110–62–3; Chemical Formula: $CH_3(CH_2)_3CHO$
H.S. No. 1420

OSHA currently has no limit for n-valeraldehyde. The ACGIH limit is 50 ppm as an 8-hour TWA. n-Valeraldehyde is a colorless liquid.

n-Valeraldehyde's toxic effects include both skin and eye irritation. Animal studies showed n-valeraldehyde to be severely irritating when applied to guinea pig skin and to rabbits' eyes (Fassett, as cited in ACGIH 1986, p. 619). The dermal LD_{50} for guinea pigs exceeds 20 ml/kg (Fassett, as cited in ACGIH 1986, p. 619).

A series of studies of the relative acute inhalation toxicity of 13 aliphatic saturated and unsaturated aldehydes in mice, guinea pigs, and rabbits showed that valeraldehyde was relatively nontoxic systemically (Salem and Cullumbine 1960).

OSHA is proposing a 50-ppm 8-hour TWA limit for this previously unregulated chemical. The Agency preliminarily concludes that this limit will protect workers from the risk of valeraldehyde's potential to cause severe eye and skin irritation. The health evidence forms a reasonable basis for proposing a new limit for n-valeraldehyde. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

m-XYLENE ALPHA, ALPHA'-DIAMINE

CAS: 1477-55-0; Chemical Formula: $C_6H_4(CH_2NH_2)_2$
H.S. No. 1432

OSHA presently has no exposure limit for this substance. The ACGIH has established a limit of 0.1 mg/m³ as a ceiling limit that should not be exceeded during any part of the working day, and has added a skin notation to indicate that substantial percutaneous absorption can occur through the eyes, mucous membranes, and skin. m-Xylene alpha, alpha'-diamine (MXDA) is a colorless liquid.

Animal studies have demonstrated that MXDA is strongly irritating to the skin (Haskell Laboratory 1973, private communication; Sherwin-Williams Company 1978). Research at Du Pont (1973) showed that pure MXDA was corrosive when applied to the skin of guinea pigs, and a 50-percent MXDA solution caused severe irritation in these animals. In a separate study (Sherwin-Williams Company 1978), a 10-percent mixture of MXDA caused severe skin irritation and erythema in guinea pigs. Sherwin-Williams (1978) also reported that rats exposed to levels of MXDA ranging from 1.74 to 6.04 mg/liter even for 1 hour sustained liver, kidney, and lung damage, as determined at necropsy. One study showed mild sensitization when MXDA was applied to guinea pig skin, but this effect was not observed in a second study (Sherwin-Williams Company 1978).

OSHA preliminarily concludes that a ceiling limit of 0.1 mg/m³ is necessary to protect against the risk of skin irritation, percutaneous absorption of MXDA, and potential systemic effects. The health evidence forms a reasonable basis for proposing a new limit for MXDA. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

XYLIDINE
CAS: 1300-73-8; Chemical Formula: $(CH_3)_2C_6H_3NH_2$
H.S. No. 1433

OSHA's current Z tables list an exposure limit of 5 ppm as an 8-hour TWA for xylidine, with a skin notation. In 1982, the ACGIH reduced its TLV to 2 ppm as an 8-hour TWA and retained the skin notation. Xylidine is a pale-yellow to brown liquid. Commercial xylene is a mixture of isomers.

Several studies indicate that the current OSHA PEL for xylidine is insufficient to protect workers against hepatotoxic and other adverse effects. A paper by Von Oettingen et al. (1974) reported liver damage in dogs, rats, cats, and mice repeatedly exposed to 45 ppm xylidine for 7 hours per day for a period of 20 to 40 weeks; these exposures also caused death in dogs, cats, and mice. Treon, Sigmon, Wright, et al. (1950) noted cardiac, liver, and kidney damage in animals fatally exposed at the following doses: Cats, 17 ppm; guinea pigs, 50 ppm; and rabbits, 60 ppm; cyanosis was also observed in these animals.

OSHA is proposing to reduce the existing 8-hour TWA to 2 ppm and to retain the skin notation. The Agency preliminarily concludes that these limits will protect workers from the risk of cardiac, kidney, and liver damage potentially associated with exposure to this substance. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for xylidine if the Agency determines that this limit will substantially reduce significant risk.

Preliminary Conclusions

Exposure to the 72 substances included in this category place workers at risk of material health impairment or functional incapacity. The adverse health consequences of exposure to these chemicals include neuropathies, skin and respiratory tract irritation, kidney and liver damage, and gastrointestinal disorders. The available health evidence for this large group of substances forms a reasonable basis for proposing to reduce existing limits or to add new limits where none formerly existed. At the time of the final rule, OSHA will establish new or revise existing limits, if the Agency determines that these limits will substantially reduce significant risk.

13. Substances for Which Proposed Limits Are Based on Avoidance of Biochemical/Metabolic Effects

Introduction

One basis for establishing exposure limits is the ability of many toxic substances to interfere with the normal metabolism or biochemistry of the body. A total of 26 substances for which OSHA is proposing limits fall into this group. Table C13-1 shows these substances, their current OSHA PELs, ACGIH TLVs, and NIOSH RELs, and their CAS and HS numbers. For four of these substances, OSHA is proposing only to lower the 8-hour TWA; for two other substances, the Agency is proposing to retain the 8-hour limit and to add a STEL. In one instance, OSHA is proposing a reduced TWA and the addition of a ceiling. In one case (terphenyls), OSHA proposes a reduction in the ceiling level, and for 17 substances, new limits are being proposed. In the case of p-nitrochlorobenzene, OSHA proposes to retain OSHA's current limit of 1 mg/m³ as an 8-hour TWA. NIOSH has a REL for only two of these substances, carbon monoxide and carbon dioxide.

Description of the Health Effects

The compounds shown in Table C13-1 are further divided into the following sub-classes, based on their mechanism of action:

- Substances that are cholinesterase inhibitors;
- Substances that interfere with the oxygen carrying capacity of blood;
- Substances with Antabuse-like effects.

The disruption of metabolic processes by toxic substances, if severe enough, results in potentially dangerous effects on the neurological, cardiovascular, and respiratory systems. The adverse health consequences caused by exposure to chemicals having cholinesterase inhibition effects range from wheezing, nausea, vomiting, and confusion to respiratory failure, coma, and death. If exposure has localized rather than systemic effects, the signs and symptoms of cholinesterase inhibition can include sweating, blurred vision, and constriction of the bronchial tubes. Substances that interfere with the ability of the blood to carry oxygen cause a broad range of symptoms, including fainting, loss of consciousness, rapid heartbeat, headache, nausea, coma, and death. Carbon monoxide (CO) is the best known substance in this category of chemicals, and exposure to CO is common throughout industry.

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Table C13-1. Substances for Which Limits Are Based on Avoidance of Metabolic Effects

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***	Biochemical/ Metabolic Effect
1025 Aniline and homologs	62-53-3	5 ppm TWA, Skin	2 ppm TWA, Skin	-	Methemoglobinamia
1058 Calcium cyanamide	156-62-7		0.5 mg/m^3 TWA		Antabuse-like effect
1068 Carbofuran	1563-66-2		0.1 mg/m^3 TWA		Cholinesterase inhibition
1069 Carbon dioxide	124-38-9	5000 ppm TWA	5,000 ppm TWA 30,000 ppm STEL	10,000 ppm TWA 30,000 ppm Ceiling (10 min)	Hyperventilation
1071 Carbon monoxide†	630-08-0	50 ppm TWA	50 ppm TWA 400 ppm STEL	35 ppm TWA 200 ppm Ceiling	Carboxyhemoglobinemia
1091 Chlorpyrifos	2921-88-2		0.2 mg/m^3 TWA 0.6 mg/m^3 STEL, Skin		Cholinesterase inhibition
1103 Crufomate	299-86-5		5 mg/m^3 TWA 20 mg/m^3 STEL		Cholinesterase inhibition

Table C13-1. Substances for Which Limits Are Based on Avoidance of Metabolic Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***	Biochemical/ Metabolic Effect
1104 Cyanamide	420-04-2		2 mg/m^3 TWA	-	Antabuse-like effect
1131 Dicrotophos	141-66-2	-	0.25 mg/m^3 TWA, Skin	-	Cholinesterase inhibition
1143 Dimethylaniline	121-69-7	5 ppm TWA, Skin	5 ppm, TWA, Skin 10 ppm STEL		Methemoglobinemia
1146 Dioxathion	78-34-2		0.2 mg/m^3 TWA, Skin	-	Cholinesterase inhibition
1151 Disulfiram (Antabuse)	97-77-8		2 mg/m^3 TWA		Antabuse effects
1160 Ethion	563-12-2		0.4 mg/m^3 TWA, Skin		Cholinesterase inhibition
1173 Fenamiphos	22224-92-6		0.1 mg/m^3 TWA, Skin	-	Cholinesterase inhibition
1174 Fensulfothion	115-90-2		0.1 mg/m^3 TWA		Cholinesterase inhibition

Table C13-1. Substances for Which Limits Are Based on Avoidance of Metabolic Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***	Biochemical/ Metabolic Effect
1175 Fenthion	55-38-9		0.2 mg/m^3 TWA, Skin	-	Cholinesterase inhibition
1245 Methomyl	16752-77-5		2.5 mg/m^3 TWA		Cholinesterase inhibition
1280 Monomethylaniline	100-61-8	2 ppm TWA, Skin	0.5 ppm TWA, Skin	-	Methemoglobinemia
1288 p-Nitrochloro-benzene[++]	100-00-5	1 mg/m^3 TWA, Skin	3 mg/m^3 TWA, Skin		Methemoglobin-emia
1319 Phorate	298-02-2		0.05 mg/m^3 TWA, 0.2 mg/m^3 STEL, Skin	-	Cholinesterase inhibition
1337 Propoxur	114-26-1		0.5 mg/m^3 TWA		Cholinesterase inhibition
1349 Ronnel	299-84-3	15 mg/m^3 TWA	10 mg/m^3 TWA		Cholinesterase inhibition
1380 Sulprofos	35400-43-2		1 mg/m^3 TWA		Cholinesterase inhibition

Table C13-1. Substances for Which Limits Are Based on Avoidance of Metabolic Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***	Biochemical/ Metabolic Effect
1384 Terphenyls	26140-60-3	1 ppm Ceiling	0.5 ppm Ceiling		Mitochondrial changes
1401 m-Toluidine	108-44-1	-	2 ppm TWA, Skin		Methemoglobinemia
1413 2,4,6-Trinitrotoluene	118-96-7	1.5 mg/m^3 TWA, Skin	0.5 mg/m^3 TWA, Skin	-	Methemoglobinemia

* OSHA's TWA limits are for 8-hour exposures; its STELs are for the durations specified; and its ceilings are peaks not to be exceeded for any period of time.

** The ACGIH TWA-TLV is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

*** NIOSH TWA limits are for 10-hour exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

+ Proposed PEL is the NIOSH REL.

++ OSHA limit is retained.

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The Antabuse-like effects associated with exposure to three chemicals—disulfiram, cyanamide, and calcium cyanamide—include facial flushing, nausea, and a racing heartbeat. However, these effects are manifested only if the exposed individual has ingested alcohol. The three chemicals in this sub-group cause this effect by inhibiting aldehyde dehydrogenase activity, which is involved in the biotransformation of alcohol.

For chemicals that cause systemic toxicity in animals and/or humans, the grossly observable signs and symptoms of intoxication are usually secondary to the interaction of the chemical with a molecular target. In other words, the chemical interacts with (binds with or modifies) an endogenous molecular constituent (protein, nucleic acid, lipid, etc.) in the target tissue(s). The result of the interaction is ordinarily a modification or elimination of the normal function of the specific molecular constituent which, if sufficiently severe, may lead to secondary effects within the affected cells and/or tissues. It is possible for a number of molecules to be affected by the toxic chemical without there being any overt manifestation of toxicity. In other words, there is an apparent no-effect level governing the overt manifestation of toxicity, although there are usually metabolic effects at levels below those that cause overt effects.

For chemicals for which the molecular target is known and for which methods are available to detect the altered molecular target, it is possible to use the measure of altered biochemical function as a sensitive indicator of exposure to the chemicals at levels below those that cause grossly observable signs and symptoms of poisoning. For some other classes of chemicals, studies in animals and/or humans have shed light on the biochemical basis of their toxicity. For some of these classes of chemicals, it is possible to base limits of human exposure on biochemical, metabolic, or pharmacologic indicators of their interaction with molecular targets rather than on grossly visible signs and symptoms of adverse systemic effects.

Cholinesterase inhibition. A number of organophosphate and carbamate insecticides produce acute toxicity in humans through inhibition of acetylcholinesterase at cholinergic synapses in the central and peripheral nervous systems. This inhibition causes an accumulation of acetylcholine at the effector sites and elicits signs and symptoms consistent with excessive cholinergic activity. These include bronchoconstriction; increased bronchial secretions, salivation, and lacrimation; nausea; vomiting; cramps; constriction of the pupils; muscular weakness; and cardiac irregularities. If sufficiently severe, acetyl-cholinesterase inhibition **may** cause coma, irreversible CNS damage, and death.

The mechanisms by which carbamates and organophosphates inhibit acetylcholinesterase differ. In general, carbamates form a non-covalently bound complex with the enzyme, while most organophosphates bind covalently with the enzyme. The net result, inactivation of the enzyme, is similar for both groups. In either case, the inhibition is usually reversible. The carbamate-cholinesterase complex dissociates to regenerate the active enzyme, while cholinesterase inactivated by organophosphates is replaced by the *de novo* synthesis of active enzyme. Therefore, unless the inhibition is sufficiently severe to cause brain damage or death, the manifestations of acute toxicity are reversible, and poisoned individuals recover without sequelae. A significant proportion of endogenous cholinesterase activity may be inhibited before the overt manifestations of intoxication appear. The fraction of total cholinesterase activity that can be inhibited without there being signs and symptoms of toxicity varies from individual to individual and also appears to depend on the intensity and duration of exposure. The lack of warning signs at low levels of exposure increases the need to set exposure limits at levels that will protect those individuals who do not readily manifest the symptoms and signs of toxicity from experiencing the subclinical effects of exposure.

Compounds that interfere with the oxygen-carrying capacity of the blood. A number of compounds produce their immediate toxicity in humans by altering the ability of hemoglobin in the red blood cells to bind, transport, and release oxygen. Perhaps the best studied of these is carbon monoxide. Carbon monoxide binds to hemoglobin with a greater affinity than does oxygen. It also alters the dissociation characteristics for the oxygen-hemoglobin complex. The overall effect is to reduce the oxygen-carrying capacity of the blood. Also included in this overall category of compounds is a group of aromatic amines and nitro compounds that react with hemoglobin in the blood to reduce it to methemoglobin. Methemoglobin will not bind with oxygen and therefore is not an effective carrier of oxygen.

Because these compounds reduce the ability of the blood to transport oxygen, the overt signs and symptoms of acute toxicity are those of tissue anoxia, i.e., neurobehavioral disturbances, dizziness, cardiac irregularities, cyanosis, unconsciousness, and death. The severity of the symptoms are a function of the degree to which the oxygen-carrying capacity of the blood has been depleted and of the state of the exposed individual's health. In the case of carbon monoxide, individuals may experience only very subtle neurobehavioral effects when 10 percent of the hemoglobin is bound to carbon monoxide, and healthy individuals may tolerate carboxyhemoglobin concentrations of 50 percent for short periods of time without experiencing lasting adverse effects.

In the cases of both carbon monoxide and the methemoglobin-forming compounds, the primary effect (i.e., formation of carboxyhemoglobin or methemoglobin) is reversible. In the absence of additional carbon monoxide exposure, carboxyhemoglobin dissociates to carbon monoxide and fully functional hemoglobin. Methemoglobin can be reoxidized to hemoglobin by endogenous mechanisms, but the major recovery mechanism is via the synthesis of new hemoglobin.

Substances that cause Antabuse-like effects. The ingestion of alcoholic beverages following exposure to disulfiram, cyanamide, or calcium cyanamide results in a characteristic syndrome consisting of flushing of the face, nausea, vomiting, hypotension, and increased heart rate. If exposure is particularly severe, the reaction may trigger convulsions, cardiac arrhythmias, or heart attacks and has in some cases caused death. In the vast majority of less severe cases, the reaction is fully reversible, although the symptoms are temporarily completely disabling. Disulfiram (Antabuse) is used therapeutically in the treatment of chronic alcoholism; as such, employees who are currently being treated with disulfiram for alcoholism are at particularly high risk if they are also occupationally exposed to these substances that cause Antabuse-like effects. These compounds do not cause any signs or symptoms of toxicity in the absence of alcohol ingestion unless exposure levels are far above those that trigger the alcohol response.

Dose-Response Relationships and Biochemical/Metabolic Effects

Cholinesterase inhibition. Typically, the cholinesterase inhibition potential of a compound is assessed by measuring plasma cholinesterase activity in the treated organism. Data from experiments in animals and limited data

from human clinical trials indicate that the percentage of basal plasma cholinesterase activity decreases with increasing dose and that the dose-response curve is S-shaped. Because there is inter-individual variation in this relationship, the dose-response curve for a population exposed to a cholinesterase inhibitor would be expected to be much shallower in slope and to have longer tails than the dose-response curve for any single individual.

The relationship between the dose-response curve for plasma cholinesterase inhibition and the dose-response curves for more direct indicators of clinical intoxication, such as acetylcholinesterase activity in the CNS or the actual appearance of signs of intoxication, is not known. Evidence suggests that there is considerable inter-individual variability in these relationships. Some individuals may be free of the symptoms and signs of intoxication when their plasma cholinesterase levels have been inhibited by as much as 90 percent, while others may experience symptoms after only a small decrease in plasma cholinesterase activity. Because of this variability, any exposure limit should be set with this individual variability in mind.

Substances that interfere with oxygen transport. Both carboxyhemoglobin and methemoglobin formation exhibit a classical sigmoidal dose-response relationship in relation to exposure to carbon monoxide or methemoglob-informing compounds. The loss in the oxygen-carrying capacity of the blood is a function of the intensity and duration of exposure. As stated above, the majority of healthy individuals can tolerate some reduction in the oxygen-carrying capacity of their blood without experiencing symptoms of overt toxicity. However, there is great inter-individual variability in the degree of decreased oxygen-carrying capacity that can be tolerated without apparent ill effect. Individuals with pre-existing anemia or with high carboxyhemoglobin levels as a result of other environmental exposures may already be at or above the level at which they will display the signs or experience the symptoms of tissue anoxia. For these individuals, even a small incremental decrease in the oxygen-carrying capacity of the blood can have serious consequences.

Substances causing Antabuse-like effects. The dose-response characteristics of disulfiram, cyanamide, and calcium cyanamide follow the usual S-shaped curve. The effect of exposure to cyanamide is approximately one-half that of exposure to disulfiram (ACGIH 1986). The proposed limits for the substances in this group have been set at levels below those associated with the Antabuse effect in workers ingesting alcohol either during or after work.

The following paragraphs describe the Agency's preliminary findings with respect to the substances that cause metabolic disturbances. The discussions below illustrate the serious nature of the risk associated with exposure to these substances.

PHORATE
CAS: 298-02-2; Chemical Formula: $C_7H_{17}O_2PS_3$
H.S. No. 1319

Phorate is an organophosphorus cholinesterase inhibitor that is used as an insecticide. The ACGIH has recommended exposure limits of 0.05 mg/m³ 8-hour TWA and 0.2 mg/m³ STEL for phorate, with a skin notation.

Phorate has been shown to be a highly toxic compound in animals. Rats exposed to daily doses of phorate showed effects at levels above 0.15 mg/kg/day but no effects at levels below this level. The no-effect level in dogs is between 0.01 and 0.05 mg/kg/day Gaines 1969).

The proposed limit of 0.05 mg/m³ as an 8-hour TWA, supplemented by a STEL of 0.2 mg/m³ and a skin notation, is based on calculations that the no-effect level in humans would lie in the range between 0.21 and 0.7 mg/day, and that use of an appropriate safety factor would suggest an 8-hour limit of 0.05 mg/m³ with a STEL of 0.2 mg/m³ to ensure against excursions greatly in excess of the TWA limit.

OSHA preliminarily finds that these limits will protect workers exposed to phorate against cholinesterase inhibition and its associated effects, which include respiratory symptoms, nausea, confusion, and vomiting. The Agency believes that, in the absence of any OSHA limit, phorate-exposed employees are at risk of experiencing such effects and that establishing a PEL will substantially reduce these risks. The health evidence forms a reasonable basis for proposing a new limit for phorate. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

CARBON MONOXIDE
CAS: 630-08-0; Chemical Formula: CO
H.S. No. 1071

OSHA's current limit for carbon monoxide is 50 ppm TWA. The ACGIH recommends a TLV-TWA of 50 ppm with a TLV-STEL of 400 ppm. NIOSH (1972) recommends an 8-hour TWA limit of 35 ppm with a 200-ppm ceiling.

Carbon monoxide readily combines with hemoglobin to form carboxyhemoglobin (COHb). Excessive accumulations of COHb cause hypoxic stress in healthy individuals as a result of reduced oxygen-carrying capacity of the blood. In patients with cardiovascular disease, such stress can further impair cardiovascular function. The ACGIH (1986) cites a number of studies showing that exposure to 50 ppm TWA carbon monoxide generally results in COHb levels of 8 to 10 percent, and that such levels are not associated with signs or symptoms of health impairment in healthy individuals. However, ACGIH comments that a TLV of 25 ppm, which results in COHb levels of 4 percent or less, may be necessary for workers with disease that places them at higher risk of serious cardiovascular injury. The NIOSH recommendation of 35 ppm TWA is also based on protecting workers with chronic heart disease; NIOSH believed that such workers should not be allowed to approach a COHb level of 5 percent.

The basis for the recommendation of a 400-ppm TLV-STEL by the ACGIH is not entirely clear, but may be based on a study by Schulte (1964), who stated that exposure to 100 ppm carbon monoxide for 4 hours is excessive. NIOSH recommended a 200-ppm short-term limit to supplement the TWA limit since it appears that transient exposures up to 200 ppm do not significantly alter a worker's equilibrium COHb level.

Exposure to the TLV-TWA of 50 ppm generally results in COHb levels of 8 to 15 percent. These levels are not associated with toxic effects in healthy individuals. NIOSH recommends an 8-hour TWA limit of 35 ppm and a ceiling limit of 200 ppm, based on the need to protect workers with chronic heart disease. OSHA proposes that these NIOSH limits be adopted as the PEL to ensure that COHb levels are less than 5 percent and thus protect workers who may be at greater risk because of cardiovascular or pulmonary impairment. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for carbon monoxide if the Agency determines that this limit will substantially reduce significant risk. OSHA's preliminary feasibility analysis is based on limited data at this level, and the Agency therefore requests additional feasibility information from the public.

CRUFOMATE
CAS: 299-86-5; Chemical Formula: $C_{12}H_{19}ClNO_3P$
H.S. No. 1103

This pesticide actively inhibits both plasma and erythrocyte cholinesterase. Neither OSHA nor NIOSH presently has a limit for crufomate; the ACGIH has set a TWA-TLV of 5 mg/m³ and a STEL of 20 mg/m³ for this substance.

A study in humans showed that ingestion of 200 mg of crufomate daily for 7 days caused no apparent cholinesterase inhibition in the subjects of this controlled study. Rats and dogs receiving higher doses (5 mg/kg/day) for two years did show this effect (McCollister et al. 1968).

Because cholinesterase inhibition is a very sensitive indicator of exposure, OSHA preliminarily concludes that the proposed level of 5 mg/m³ will provide a margin of safety below the ingestion NOEL for humans, which corresponds approximately to an 8-hour inhalation of 20 mg/m³. The substantial risk of experiencing such effects will be significantly reduced from the uncontrolled levels possible in the absence of an OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for crufomate. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

CARBON DIOXIDE
CAS: 124-38-9; Chemical Formula: CO_2
H.S. No. 1069

OSHA's current limit for carbon dioxide is 5000 ppm TWA. ACGIH also recommends a 5000-ppm TLV-TWA with a 30,000-ppm TLV-STEL. NIOSH recommends a 10-hour TWA limit of 10,000 ppm with a 10-minute 30,000 ppm ceiling limit.

Both ACGIH (1986) and NIOSH (1976f) cite studies indicating that continuous exposure to between 1.5 and 3 percent carbon dioxide (15,000 to 30,000 ppm) results in few, if any, adverse effects. However, electrolyte imbalances and other mild metabolic changes have been associated with prolonged exposure to 10,000 to 20,000 ppm (Schulte 1964; Gray et al. 1950). Increases in the rate of respiration have been observed among resting subjects exposed to 39,500 ppm for periods shorter than a day and among exercising subjects exposed to airborne concentrations below 30,000 for the same period (Sinclair et al. 1969).

OSHA is proposing to add a 30,000-ppm STEL to the existing PEL of 5000 ppm TWA to protect employees from experiencing elevated short term exposures; the Agency preliminarily concludes that this limit will substantially reduce the risk associated with such short-term exposures to CO_2. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for carbon dioxide if the Agency determines that this limit will substantially reduce significant risk.

ANILINE (AND HOMOLOGUES)
CAS: 62-53-3; Chemical Formula: $C_6H_5NH_2$
H.S. No. 1025

The current OSHA 8-hour TWA permissible exposure limit for aniline is 5 ppm, with a skin notation. The ACGIH-recommended 8-hour TLV is a 2-ppm TWA, with a skin notation. Aniline, when first distilled, is an oily, colorless liquid that darkens on exposure to air.

Occupational aniline poisoning was a relatively common occurrence in earlier years (ACGIH 1986, p. 30). The early limits for aniline were set to guard against acute toxicity manifested as cyanosis (Henderson and Haggard 1943). Cirrhosis and chronic CNS effects were also reported (Holstein 1955; von Oettingen 1941). Skin absorption occurs when aniline vapor contacts the skin (Dutkiewicz 1962), and skin contact should therefore be avoided.

Early studies suggested that less than full-shift exposures of 7 to 53 ppm of aniline vapor caused mild symptoms, while 1-hour inhalation exposures to concentrations in the range of 100 to 160 ppm caused severe effects (Henderson and Haggard 1943). Later studies in several species of animals found no effects, other than a slight increase in methemoglobin in the blood of rats, after the animals had been exposed to aniline concentrations of 5 ppm for 6 months (Oberst, Hackley, and Comstock 1956). An early NCI aniline hydrochloride cancer bioassay in Fischer-344 rats and B6C3F1 mice demonstrated carcinogenic effects, primarily in the spleen of rats, but multiple organ sites were also involved in rats fed 0.6 percent or 0.3 percent aniline hydrochloride for 103 weeks (NCI 1978).

OSHA has preliminarily concluded that the current limit of 5 ppm is not protective, since systemic effects have been observed in humans exposed to levels as low as 7 ppm and in animals at levels as low as 5 ppm. Accordingly, OSHA proposes an 8-hour TWA of 2 ppm for aniline and retains the skin notation to protect against percutaneous absorption. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for aniline if the Agency determines that this limit will substantially reduce significant risk. The Agency intends to analyze the evidence of aniline carcinogenicity further.

CALCIUM CYANAMIDE
CAS: 156-62-7; Chemical Formula: $CCaN_2$
H.S. No. 1058

OSHA currently has no limit for calcium cyanamide. The ACGIH recommends a TLV-TWA of 0.5 mg/m³ for this crystalline gray material.

The acute toxicity of calcium cyanamide is low, although evidence of its toxic effects is sparse. The oral LD_{50} reported for rabbits is 1400 mg/kg, and that for rats is 1000 mg/kg (Guide to Chemicals Used in Crop Production, London, Ontario, 1973).

Skin and eye irritation have been reported in rats and rabbits, with a significant irritative effect occurring when 100 mg is placed directly into the eyes of rabbits (Martin 1975). Severe skin irritation developed in rabbits when a paste of this substance was applied to the shaved abdominal skin for 24 hours (Martin 1975). Two of five animals died when the dose was 10 g/kg, but all survived a dose of 5 g/kg.

Most industrial toxicities involve primary skin irritation or sensitizing dermatitis. This skin irritation develops in the form of an erythematous rash over the body surfaces exposed to the substance or those areas irritated by clothing or perspiration. Some individuals develop a macular rash on exposure, and this may progress to the weeping stage. In addition, exposed workers may develop temporary vasomotor disturbances of the upper body, with susceptibility increasing with alcohol intake (Fassett 1963). Calcium cyanamide is used medically for its Antabuse-like effect, and the maintenance dose in adults is between 50 and 100 mg/day (Hald, Jacobson, and Varson 1952).

OSHA proposes a TWA of 0.5 mg/m³ for calcium cyanamide. The Agency preliminarily concludes that this level will substantially reduce the risks of eye and skin irritation, sensitizing dermatitis, and the occurrence of Antabuse-like effects possible at previously unregulated levels of exposure. The health evidence forms a reasonable basis for proposing a new limit for calcium cyanamide. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

CARBOFURAN
CAS: 1563-66-2; Chemical Formula: $C_{12}H_{15}NO_3$
H.S. No. 1068

OSHA does not currently regulate carbofuran. The ACGIH recommends a TLV-TWA of 0.1 mg/m³ for this white crystalline solid.

The inhalation toxicity of carbofuran is low, with Tobin reporting the LC_{50} of

50-percent wettable powder to be 108 mg/m³ for male and 133 mg/m³ for female rats; a respiratory LC_{50} of 53 mg/m³ for guinea pigs exposed to 75-percent wettable powder is also reported (Tobin 1970). Rhesus monkeys did not display cholinesterase depression at levels equivalent to 0.56 mg/m³ of 75-percent wettable powder (Tobin 1970). Chronic feeding studies in the rat have shown no effect at 25 ppm; in the dog, the no-effect level was 20 ppm (Gaines, unpublished data). Inhibition of plasma, erythrocyte, and brain cholinesterase levels were evident at levels of 50 ppm in the diet (Tobin 1970). Six-hour exposures at levels of 0.86 mg/m³ caused significant cholinesterase inhibition in animals (Tobin 1970).

Workers exposed at concentrations approaching 0.1 mg/m³ have not shown any adverse effects (Tobin, personal communication to ACGIH TLV Committee, as cited in ACGIH 1986, p. 100).

OSHA is proposing a permissible exposure limit of 0.1 mg/m³ TWA for this substance to protect exposed employees from the risk of metabolic effects potentially associated with exposure to this previously unregulated substance. The health evidence forms a reasonable basis for proposing a new limit for carbofuran. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

CHLORPYRIFOS
CAS: 2921-88-2; Chemical Formula: $C_9H_{11}Cl_3NO_3PS$
H.S. 1091

OSHA has no current limit for chlorpyrifos. The ACGIH recommends a TLV-TWA of 0.2 mg/m³ and a 0.6-mg/m³ STEL, with a skin notation, for this white crystalline solid.

Chlorpyrifos has an acute oral LD_{50} of 135 mg/kg for female rats and 163 for male rats (The Merck Index 1983, pp. 309-310). Other sources have reported the acute oral LD_{50} as 82 mg/kg in rats and the acute dermal LD_{50} as about 2000 mg/kg for rabbits (Gray 1965; Gaines 1969).

Chlorpyrifos is absorbed through the skin. It is an active inhibitor of plasma cholinesterase but has only moderate capacity to reduce red blood cell cholinesterase or to cause cholinergic symptoms and systemic injury (ACGIH 1986, p. 138). Particle inhalation has been shown to cause mild depression in plasma cholinesterase in dogs exposed for 4 hours at the upper end of a 140- to 280-mg/m³ range (Spencer 1968).

Dogs and rats fed 3.0 mg/kg of chlorpyrifos daily for 2 years showed no adverse effects (FAO/WHO 1972). Male and female rats showed no teratogenic or reproductive effects when fed 1.0 mg/kg per day (Dow Chemical Company 1972).

Five out of seven human exposures to 0.5 percent chlorpyrifos resulted in a measurable decrease (50 percent) in plasma and red cell cholinesterase (Eliason, Cranmer, von Windeguth et al. 1969). However, another study showed no ill effects on cholinesterase metabolism when human volunteers were exposed to an ultra-low-volume spray (0.8 um/m³ for 3 to 8 minutes) (Ludwig, Kilian, Dishburger, and Edwards 1970). Human cholinesterase levels appear to be less affected by dermal exposure than do those of rabbits (ACGIH 1986, p. 138). In human volunteers, four repeated dermal doses of 10 mg/kg, applied for 12 hours each, caused no depression in cholinesterase levels, but a similar dose of 25 mg/kg did depress plasma cholinesterase. Human subjects ingesting 0.03 mg/kg for 3 weeks showed no cholinesterase effects, but subjects ingesting 0.1 mg/kg demonstrated plasma cholinesterase depression (Dow Chemical Company 1973).

Workers applying chlorpyrifos as a spray were exposed to 0.5 percent chlorpyrifos emulsion and exhibited a marked decrease in plasma and red cell cholinesterase levels (Eliason, Cranmer, von Windeguth et al. 1969). In five of seven exposed sprayers, this reduction was greater than 50 percent.

OSHA proposes a PEL of 0.2 mg/m³ TWA and a 0.6 mg/m³ STEL for chlorpyrifos to protect exposed workers against the risk of organic injury and cholinesterase inhibition caused by this currently unregulated substance. A skin notation is also proposed to protect workers from the significant risk of systemic effects caused by percutaneous absorption. The health evidence forms a reasonable basis for proposing a new limit for chlorpyrifos. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

CYANAMIDE
CAS: 420-04-2; Chemical Formula: $H_2NC{=}N$
H.S. No. 1104

OSHA currently has no limit for cyanamide. The ACGIH recommends a TLV-TWA of 2 mg/m³. Undiluted cyanamide is a deliquescent crystalline solid.

The average oral LD_{50} for cyanamide in rats is 125 (85 to 180) mg/kg, and cyanamide has been observed to be very irritating and caustic to the skin (American Cyanamide Company n.d.). As a 25-percent solution, which is commonly used, 10 mL/kg applied to the skin of rabbits caused no fatalities or signs of systemic toxicity. Irritation occurred in the form of primary skin irritation and, following instillation into the eye, slight irritation of the conjunctival sac (American Cyanamide Company n.d.).

When cyanamide is ingested or inhaled by a person who has also consumed an alcoholic beverage, the person experiences vasodilation of the face and neck, tachycardia, tachypnea, nausea, vomiting, and hypotension. This syndrome is referred to as the Antabuse effect. Studies of cyanamide's Antabuse-like effects indicate that the effect is about one-half that of tetraethylthiuram disulfide (Antabuse) and one-sixth that of tetramethyl thiuram disulfide (Hald, Jacobsen, and Larsen et al. n.d.).

OSHA proposes a limit of 2 mg/m³ TWA for cyanamide. The Agency preliminarily concludes that this limit will protect exposed workers from the risk of irritation and of an adverse reaction in individuals who have ingested alcohol. The health evidence forms a reasonable basis for proposing a new limit for cyanamide. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

DICROTOPHOS (BIDRIN)
CAS: 141-66-2; Chemical Formula: $C_8H_{16}NO_5P$
H.S. No. 1131

OSHA currently has no limit for dicrotophos; the ACGIH recommends a TLV of 0.25 mg/m³ TWA, with a skin notation, for this brown liquid with a mild ester odor.

Dicrotophos is a cholinesterase inhibitor (ACGIH 1986, p. 193). The acute oral LD_{50} in rats is reported as 22 mg/kg, and the percutaneous LD_{50} in rabbits is 224 mg/kg (Stanford Research Institute 1962, as cited in ACGIH 1986, p. 193). Another study reports the oral LD_{50} in rats as 16 to 21 mg/kg and the dermal LD_{50} in the same species as 42 mg/kg (Gaines 1969). Two-year feeding studies in rats given 0, 1, 10, or 100 ppm dicrotophos showed no detectable effects at the 1-ppm concentration. At the higher concentrations, decreased body weights (as compared to controls) and cholesterinase inhibition were observed (Woodward Research Corporation 1967, as cited in ACGIH 1986, p. 193). Dietary studies in dogs showed both plasma and erythrocyte cholinesterase inhibition at a 16-ppm concentration, but no significant ill effects at concentrations of 0, 0.16, or 1.6

ppm (Woodward Research Corporation 1967, as cited in ACGIH 1986, p. 193). Studies of vapor inhalation in male rats have shown that transient illness occurred after a 1-hour exposure to 910 mg/m³ technical dicrotophos, and 2620 mg/m³ or 2120 mg/m³ of 38-percent dicrotophos (Kettering Laboratories 1965, as cited in ACGIH 1986). Dicrotophos does not cause demyelinization in chickens (Tunstall Laboratory 1965; Kettering Laboratory 1963, as cited in ACGIH 1986, p. 193), and it is metabolized in a fashion similar to mono-microtophos (Menzer and Casida 1965). The ACGIH (1986, p. 193) reports that dicrotophos penetrates the skin.

The ACGIH recommendation is based on the data described above and, in part, by analogy to other cholinesterase-inhibiting substances. OSHA is proposing an 8-hour TWA permissible exposure limit of 0.25 mg/m³, with a skin notation, for dicrotophos. The Agency preliminarily concludes that this limit will protect exposed workers from the metabolic effects, such as cholinesterase inhibition, potentially associated with inhalation, ingestion, and dermal exposure to this substance. The health evidence forms a reasonable basis for proposing a new limit for dicrotophos. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

DIMETHYLANILINE
CAS: 121–69–7; Chemical Formula: $C_6H_5N(CH_3)_2$
H.S. No. 1143

OSHA's current permissible exposure limit for dimethylaniline is 5 ppm as an 8-hour TWA, with a skin notation. The ACGIH recommends an 8-hour TWA limit of 5 ppm, with a 15-minute STEL of 10 ppm and a skin notation. Dimethylaniline is a yellow-to-brown oily liquid.

One of the major toxic effects of dimethylaniline exposure is methemoglobinemia, although authorities disagree concerning the level at which humans can tolerate exposure to this substance (ACGIH 1986, p. 207).

Hamblin (1962) reported that dimethylaniline is quantitatively less toxic than aniline. Dogs administered a single oral dose of 50 mg/kg exhibited methemoglobinemia, and absorption through the skin can increase the overall exposure (Hamblin 1962). Mayer (1930) reported that dimethylaniline's necrotic potential was markedly lower than that of aniline, which has a TLV–TWA of 2 ppm. However, von Oettingen (1941) stated that dimethylaniline has a greater depressant effect on the nervous system than does aniline.

The literature on industrial experience with dimethylaniline is limited. Hamilton (1919) reported collapse, prolonged unconsciousness, visual disturbance, and intense abdominal pain following severe exposure of two workers.

The Agency is proposing an 8-hour TWA PEL of 5 ppm and a short-term limit of 10 ppm, with a skin notation, for dimethylaniline. OSHA believes that the STEL is necessary to afford protection from CNS depression following acute exposures. OSHA preliminarily concludes that these limits, taken together, will provide exposed workers with protection from the risks of skin absorption, methemoglobinemia, and neuropathic effects associated with exposure to this substance. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for dimethylaniline if the Agency determines that this limit will substantially reduce significant risk.

DIOXATHION (DELNAV)
CAS: 78–34–2; Chemical Formula: $C_{12}H_{26}O_6P_2S_4$
H.S. No. 1146

OSHA currently has no permissible exposure limit for dioxathion. The ACGIH recommends a limit of 0.2 mg/m³ as an 8-hour TWA, with a skin notation. Dioxathion is a nonvolatile, very stable, dark amber liquid.

The pesticide, dioxathion, contains both the cis- and trans-isomers of 2,3-p-dioxanedithiol; the cis-isomer is approximately four times as acutely toxic as the trans-isomer ACGIH 1986, p. 219). The oral LD_{50} values reported for rats range from 23 to 118 mg/kg (with most values in the 23- to 64-mg/kg portion of the range); in dogs, oral LD_{50}s range from 10 to 40 mg/kg. The LC_{50} in rats is 1398 mg/m;³ in mice, it is 340 mg/m³ (Hercules, Inc. 1973, as cited in ACGIH 1986, p. 219). The percutaneous LD_{50}s in rats and rabbits are reported to be 63 and 85 mg/kg, respectively (NIOSH 1983). Instillation of 0.1 ml dioxathion into the rabbit eye produces mild, transient conjunctivitis but no corneal damage (ACGIH 1986, p. 219).

In subacute oral toxicity studies, the no-effect dose level in rats was reported to be 0.22 mg/kg/day; in dogs, a no-effect level of between 0.075 and 0.25 mg/kg/day was indicated (Frawley, Weir, Tusing et al. 1963). Dioxathion was reported not to produce myelin degeneration in chickens (Frawley, Weir, Tusing et al. 1963). The no-effect level in multigenerational studies of reproductive effects in rats was reported to be 10 ppm (Kennedy, Frawley, and Colandra 1973).

Human volunteers who ingested 0.075 mg/kg/day had no symptoms related to plasma or blood cholinesterase activity, while those ingesting 0.15 mg/kg/day exhibited a slight decrease in plasma cholinesterase activity (Frawley, Weir, and Tusing et al. 1963). The World Health Organization has estimated an acceptable daily intake for man of 0.0015 mg dioxathion/kg (WHO, as cited in ACGIH 1986, p. 219). Other organophosphorous compounds have been demonstrated to produce levels of cholinesterase inhibition analogous to those produced by dioxathion (ACGIH 1986, p. 219).

OSHA is proposing an 8-hour TWA PEL of 0.2 mg/m³ for dioxathion; the Agency also proposes a skin notation for dioxathion. OSHA preliminarily concludes that these limits will protect exposed workers against the risk of metabolic effects associated with inhalation and oral exposure and with dermal penetration of this substance, which is currently not regulated by OSHA. The health evidence forms a reasonable basis for proposing a new limit for dioxathion. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

DISULFIRAM
CAS: 97–77–8; Chemical Formula: $C_{10}H_{20}N_2S_4$
H.S. No. 1151

OSHA currently has no limit for disulfiram. The ACGIH recommends a limit of 2 mg/m³ TWA for this crystalline solid.

Disulfiram has a very low order of acute oral toxicity in laboratory animals (ACGIH 1986, p. 225). The LD_{50} for rats is reported as 8.6 g/kg (The Merck Index 1983, pp. 491–492), and the oral LD_{50} for rabbits is reported to be 2.05 g/kg (Brieger 1947). The compound is highly toxic when injected intraperitoneally, with an LD_{50} of 75 mg/kg for mice (National Technical Information Service, as cited in ACGIH 1986, p. 225). The effects of high-dose ingestion include degenerative changes in the liver and kidneys. Very high doses can cause leukopenia and marked hypoplasia or aplasia of the bone marrow; in the most seriously afflicted animals, the blood urea nitrogen sometimes increased and the thymol turbidity test was positive (Brieger 1947).

Adverse health effects occur in humans consuming alcohol and simultaneously exposed to disulfiram. This represents a significant concern since disulfiram, under the trade name

Antabuse, is used as a medication in the treatment of chronic alcoholism. For individuals who drink alcohol and are exposed to disulfiram, the symptoms of exposure are facial vasodilation, tachycardia, tachypnea, nausea, vomiting, pallor, and hypotension. High doses of disulfiram can induce convulsions, cardiac arrhythmias, and myocardial infarction, and the compound has also been associated with polyneuropathy, peripheral neuritis, and skin eruption (Compendium of Pharmaceuticals and Specialties 1968). In industry, there have been reports of minimal skin irritation (Mastromatteo, as cited in ACIGH 1986, p. 225) and of optic neuritis (Norton and Walsh 1972).

OSHA is proposing a PEL of 2 mg/m³ TWA for disulfiram. The Agency preliminarily concludes that this limit will protect workers against the risk of Antabuse-like effects associated with exposure to airborne concentrations of disulfuram in combination with alcohol consumption. The health evidence forms a reasonable basis for proposing a new limit for disulfiram. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

ETHION (NIALATE)
CAS: 563–12–2; Chemical Formula:
$C_9H_{22}O_4P_2S_2$
H.S. No. 1160

OSHA currently has no permissible exposure limit for ethion. The ACGIH recommends a limit of 0.4 mg/m³ TWA, with a skin notation. Pure ethion is an odorless and colorless liquid. The technical material has a very disagreeable odor.

Ethion, is an insecticide that is used in a variety of forms, including 25-percent wettable powder, 2-, 3-, and 4-percent dust, 5-percent granules, and in several oil solutions and combinations with other chemicals. As a result, the acute toxicity values reported vary considerably.

NIOSH (1974) reports an oral LD_{50} in rats of 13 mg/kg. Other reported values for oral LD_{50}s in rats include 65 mg/kg, 96 mg/kg, and 208 mg/kg (Clinical Handbook on Economic Poisons; Farm Chemicals Handbook A74; Pesticide Chemicals Official Compendium; all as cited in ACGIH 1986, p. 236). Studies with 95 percent technical ethion report oral LD_{50}s of 87.4±0.16 mg/kg for albino rats and 24.4 mg/kg for female rats (Niagara Chemical Division, FMC Corp., as cited in ACGIH 1986, p. 236). Inhalation studies report LC_{50} values of 710 mg/m³ for female rats exposed to 25-percent wettable powder dust for 1 hour, and 7200 mg/m³ for male rats similarly exposed. Dermal exposure studies employing technical Nialate report a median acute dermal lethal dose of 915 mg/kg, demonstrating ethion's ability to penetrate skin; instillation of 0.05 ml ethion in the rabbit eye is immediately irritating but does not cause corneal scarring (Niagara Chemical Division, FMC Corp., as cited in ACGIH 1986, p. 236). Dietary studies of rats fed 600, 1000, or 1500 ppm showed complete cholinesterase inhibition; 300 ppm in the diet produced marked cholinesterase inhibition (Pesticide Chemicals Official Compendium, as cited in ACGIH 1986, p. 236).

Ethion poisonings have been reported in workers harvesting grapes and peaches (State of California: Department of Industrial Relations, as cited in ACGIH 1986. p. 236).

OSHA is proposing a PEL of 0.4 mg/m³ TWA for ethion. The Agency preliminarily concludes that this limit will protect exposed workers from the risk of organophosphate poisoning and cholinesterase inhibition posed to workers in the absence of any OSHA limit. The Agency notes this substance's potential for dermal absorption in laboratory animals and is proposing a skin notation to protect against the risk of systemic toxicity possible in the absence of a skin notation. The health evidence forms a reasonable basis for proposing a new limit for ethion. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

FENAMIPHOS
CAS: 22224–92–6; Chemical Formula:
$C_{13}H_{22}NO_3PS$
H.S. No. 1173

OSHA currently has no limit for fenamiphos. The ACGIH recommends a TLV–TWA of 0.1 mg/m³ for this substance, with a skin notation. Fenamiphos is a tan-colored and waxy solid.

Fenamiphos is a cholinesterase inhibitor that produces both central and peripheral cholinergic reactions (WHO 1975). The acute oral LD_{50} values reported for fenamiphos are 2 to 19 mg/kg in rats, 22 mg/kg in mice, 56 to 100 mg/kg in guinea pigs, 10 to 17 mg/kg in rabbits, and approximately 10 mg/kg in cats and dogs. Acute dermal LD_{50} values are 72 to 154 mg/kg in rats and 178 to 225 mg/kg in rabbits. One- and 4-hour exposures of rats to fenamiphos aerosols resulted in LC_{50} values of 110 to 175 and 91 mg/m³ to 100 mg/m³ of air, respectively. Rabbits exhibited no dermal or eye irritation (WHO 1975; Loeser and Kimmerle 1971).

Rats exposed to fenamiphos aerosol at concentrations of 0.03, 0.25, or 3.5 mg/m³ of air for 3 weeks exhibited no symptoms. At 3.5 mg/m³, rats showed significant depression of plasma cholinesterase; 0.25 mg/m³ was the highest no-effect concentration observed (Kimmerle, as cited in ACGIH 1986, p. 265). Two-year feeding studies of dogs (0.5, 1.0, and 10 ppm) and rats (3, 10, and 30 ppm) revealed no treatment-related toxic or oncogenic effects or tissue changes at a dietary level of 10 ppm; no-observable-effect levels were 3 ppm for the rat and 1 ppm for the dog (WHO 1975). Studies of rabbits and rats showed no embryotoxic or teratogenic effects, and results of a 3-generation study in rats showed that fenamiphos had no effect on reproduction (WHO 1975). Studies of mice have also shown no mutagenic effects, and a study of chickens demonstrated no delayed neurotoxic effects (WHO 1975; Kimmerle, as cited in ACGIH 1986, p. 265). Fenamiphos is metabolized rapidly and excreted primarily in the urine, as demonstrated in absorption tests of the skin and digestive and respiratory tracts of rats and cows (Waggoner and Khasawinah 1974).

There are no reports of human poisonings caused by exposure to fenamiphos, and no quantitative data are available relating adverse health effects to measurable airborne concentrations of fenamiphos.

OSHA proposes a PEL for this substance of 0.1 mg/m³ TWA to protect against the risk of anti-cholinesterase effects. A skin notation is also proposed based on the evidence of percutaneous absorption of fenamiphos in experimental animals. The Agency preliminarily concludes that these limits will substantially reduce this risk. The health evidence forms a reasonable basis for proposing a new limit for fenamiphos. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

FENSULFOTHION (DASANIT)
CAS: 115–90–2; Chemical Formula:
$C_{11}H_{17}O_4PS_2$
H.S. No. 1174

OSHA currently has no limit for fensulfothion. The ACGIH recommends a TLV–TWA of 0.1 mg/m³ Fensulfothion is a brown liquid at room temperature.

Fensulfothion has an acute oral LD_{50} of 4 mg/kg in male rats, and 1.8 mg/kg in female rats. Aerosol inhalation studies in rats have shown LC_{50}s of 113 mg/m³ for a 1-hour exposure and 29.5 mg/m³

for a 4-hour exposure (Luser and Kimmerle 1971). This insecticide has been shown to have effects similar to those of the other thiophosphates, which cause cholinesterase inhibition. Dermal toxicity is relatively high, with LD_{50} values ranging between 14 and 30 mg/kg for male rats and between 3.5 and 3.0 mg/kg for females (NIOSH 1974). Tests of mice and rabbits have shown no embryotoxic, reproductive or mutagenic effects. The no-effect dietary level in subchronic feeding studies is reported to be 1 ppm in rats and 2 ppm in dogs. The no-effect level for cholinesterase inhibition is reported as 1 ppm in the diet for both dogs and cats (ACGIH 1986, p. 266).

In humans, dermal studies have shown irritation without cholinesterase effects from 2-hour twice-daily applications of a 5-percent granular formulation to the forearms of three subjects. Systemic absorption through the lungs has been demonstrated after inhalation of fensulfothion aerosols (ACGIH 1986, p. 266).

OSHA is proposing a PEL of 0.1 mg/m^3 TWA for this previously unregulated substance to reduce the risks of metabolic effects and skin irritation. The Agency preliminarily concludes that this limit will substantially reduce this risk. The health evidence forms a reasonable basis for proposing a new limit for fensulfothion. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

FENTHION
CAS: 55–38–9; Chemical Formula: $C_{10}H_{15}O_3PS$
H.S. No. 1175

OSHA currently has no limit for fenthion. The ACGIH recommends a TLV–TWA of 0.2 mg/m^3, with a skin notation. Fenthion is a yellow-to-tan, oily liquid that smells slightly like garlic.

The primary health effect associated with exposure to fenthion is plasma cholinesterase inhibition. The oral LD_{50} values for the rat and rabbit are 215 mg/kg and 150 mg/kg, respectively, and the dermal LD_{50} in rats is 330 mg/kg (*Farm Chemicals Handbook* 1976; NIOSH 1977). Rats given single intramuscular injections of 5, 25, or 50 mg/kg of fenthion exhibited enduring changes in the electroretinogram (ERG) and cholinesterase activity; pseudocholinesterase activity in the plasma dropped to 50 percent of normal on the fourth day after injection. The retinal effects of fenthion persisted for as long as 50 days (Imai 1975). Groups of Donryn rats fed 300 ppm fenthion daily showed symptoms of organophosphate intoxication, including nervousness, general spasms, diarrhea, salivation, and ophthalmologic effects (Kawai, Tojo, Miyazawa et al. 1976). The no-effect inhalation level for rats has been reported to be 1 ppm for exposures of 6 hours/day, 5 days/week for 3 weeks (Thyssen 1979, as cited in ACGIH 1986, p. 267); the 4-hour inhalation LC_{50} in the rat is between 800 and 1200 mg/m^3 Thyssen 1978, as cited in ACGIH 1986, p. 267).

No mutagenic, carcinogenic, or reproductive effects have been reported (WHO 1976; Food and Agriculture Organization 1979; Shirasu, Moriya, Kato et al. 1976; Hanna and Dyer 1975; Oesch 1977; Simmon, Mitchell, and Jergenson 1977; Herbold 1980). Single and repeated applications of the compound have shown no delayed neurotoxic effects in chickens (WHO 1972). Two-year feeding studies of rhesus monkeys have shown plasma cholinesterase inhibition only at the highest oral dose given, i.e., 0.2 mg/kg daily (Rosenblum 1980).

Griffin, Rosenblum, and Coulston (1979) reported cholinesterase depression in humans at oral doses of 0.07 mg/kg daily for 4 weeks, but no effect was observed at 0.02 mg/kg. The lowest lethal dose for humans is 50 mg/kg (*Farm Chemicals Handbook* 1976; NIOSH 1977).

OSHA is proposing an 8-hour TWA limit of 0.2 mg/m^3, with a skin notation, for fenthion. The Agency preliminarily concludes that these limits will protect workers against the risk of cholinergic effects associated with exposures to this substance at the levels permitted by the absence of any OSHA limit. A skin notation is proposed because of evidence that fenthion is readily absorbed through the skin. The health evidence forms a reasonable basis for proposing a new limit for fenthion. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

METHOMYL
CAS: 16752–77–5; Chemical Formula: $C_5H_{10}N_2O_2S$
H.S. No. 1245

OSHA currently has no limit for methomyl. The ACGIH recommends a TLV–TWA of 2.5 mg/m^3 for this white crystalline solid with a slightly sulfurous odor.

Methomyl is a cholinesterase-inhibiting insecticide. The oral LD_{50} in rats is reported to be between 25 and 40 mg/kg (Dashiell and Kennedy 1984). Studies of dermal effects have reported no appreciable irritation or sensitization effects in guinea pigs. Instillation of a 10-percent solution of methomyl in propylene glycol or of the dry material into rabbit eyes caused mild conjunctivitis without corneal injury. However, marked pupillary constriction, a health effect produced commonly by cholinesterase inhibitors, occurred (E.I. du Pont de Nemours and Co., Inc., as cited in ACGIH 1986, p. 363). The LC_{50} of unformulated methomyl as mist is 0.3 mg/L at 4-hour exposures; the lethal concentration in rats exposed to a 90-percent water-soluble formulation with a particle size of less than 10 microns was approximately 0.45 mg/L.

Inhalation studies have reported no fatalities resulting from 4-hour exposures to the saturated vapor. There is no clinical evidence of cumulative toxicity resulting from 10 doses of 5.1 mg/kg/day over a 14-day period (Harvey, Jelnek, and Sherman 1973). Methomyl is rapidly metabolized and excreted in the urine, and cholinesterase inhibition is thus quickly reversed. In dogs, a dose of 20 mg/kg (one-half the lethal dose) produced symptoms of intoxication and cholinesterase inhibition that disappeared within 2 to 4 hours after cessation of exposure (E.I. du Pont de Nemours and Co., Inc. as cited in ACGIH 1986, p. 363). No depression of cholinesterase activity could be detected in rats fed at levels of 0, 200, 400, or 800 ppm methomyl for 79 days. In dogs, 90-day and 2-year feeding studies have shown no effect at 0, 50, 100, or 400 ppm; however, animals fed at 1000 ppm did demonstrate toxicity. Similar studies of rats have shown kidney, liver, and spleen damage at higher feeding levels, but the no-effect level for both rats and dogs has been reported to be 100 ppm (Kaplan and Sherman 1977).

OSHA is proposing a PEL of 2.5 mg/m^3 TWA for methomyl. The Agency preliminarily concludes that this limit will protect exposed workers against the risk of cholinesterase inhibition to which they could otherwise be exposed in the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for methomyl. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

MONOMETHYLANILINE
CAS: 100–61–8; Chemical Formula: $C_6H_5NH(CH_3)$
H.S. No. 1280

OSHA's existing PEL for monomethylaniline (N-methyl aniline) is 2 ppm, measured as an 8-hour TWA, and this limit is accompanied by a skin notation, indicating that this chemical may penetrate the skin to a degree such that the total amount absorbed by the body via all routes of exposure may be

significantly increased. The ACGIH has a limit of 0.5 ppm TWA for monomethylaniline, with a skin notation. Monomethylaniline is a colorless liquid which turns reddish-brown after standing.

Treon, Deichmann, Sigmon, and associates (1949) found that monomethylaniline applied to the skin of laboratory animals resulted in systemic poisoning, and that the oral LD_{50} in rabbits was 280 mg/kg. A later study by Treon and his associates (1950) showed that guinea pigs, rabbits, and rats died from 130 or fewer 7-hour exposures to 7.6 ppm monomethyl aniline. In the same study, a monkey survived the same number and length of exposures at 2.4 ppm, and a dog survived 50 exposures at 86 ppm. Exposed animals later developed blood changes, including methemoglobinemia and Heinz bodies.

OSHA is proposing a 0.5-ppm TWA limit, with a skin notation, for this substance. The Agency preliminarily concludes that these two limits, taken together will protect exposed workers from the risk of metabolic and blood effects, such as methemoglobinemia, potentially associated with exposure to monomethylaniline. The skin notation will protect workers from the risk of systemic poisoning posed by skin absorption of this substance. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for monomethylaniline if the Agency determines that this limit will substantially reduce significant risk.

p-NITROCHLOROBENZENE
CAS: 100–00–5; Chemical Formula: $NO_2C_6H_6H_4Cl$
H.S. No. 1288

OSHA currently has an 8-hour TWA limit of 1 mg/m³, with a skin notation, for p-nitrochlorobenzene (PNCB). The ACGIH recommends a TLV–TWA of 3 mg/m³ with a skin notation. para-Nitrochlorobenzene exists as yellow crystals and has a sweet odor.

The primary hazards associated with exposure to PNCB include systemic toxicity to the liver, spleen, bone marrow, and kidneys, as well as methemoglobinemia and DNA damage. The Monsanto Company (1977) reported an oral LD_{50} in rats of 530 mg/kg and a dermal LD_{50} in rabbits of greater than 3040 mg/kg; PNCB was absorbed through rabbit skin to produce methemoglobinemia (Kubota 1960), although application to the skin or eyes did not produce irritation (Monsanto Company 1977). Rusakov and associates (1973) described sensitization in guinea pigs after dermal application of PNCB.

A 4-hour inhalation exposure of rats (heads only) showed that the lethal concentration was approximately 16.1 mg/L (Du Pont Company 198l, as cited in ACGIH 1986, p. 432). Head-only exposures at 0.05, 0.29, or 0.64 mg/L PNCB for 6 hours/day, 5 days/week for 2 weeks resulted in spleen weight increases and blood effects in all groups. In addition, there were dose-related effects in blood methemoglobin levels, i.e., decreased hemoglobin, hematocrit, and red blood cell count values. Microscopic changes in the spleen, bone marrow, and kidneys were seen in the two higher-dose groups, and pathological degeneration of the seminiferous tubules and abnormal epididymal sperm contents were also observed in these groups (Du Pont Company 1984, as cited in ACGIH 1986, p. 432).

The Monsanto Company (1981, as cited in ACGIH 1986, p. 432) reported that a 90-day gavage administration of PNCB at daily doses of 0.3, 10, or 30 mg/kg to male and female rats produced hemolytic effects and spleen changes at all levels, kidney and liver effects at mid- to high-level doses, and hyperplasia of bone marrow and testicular atrophy at the highest dose (30 mg/kg/day). In 1985, Monsanto reported the results of another gavage study in rats. After 2 years of PNCB feeding at 0.1, 0.7, or 5.0 mg/kg/day, animals in the mid- and high-dose groups exhibited hemolytic effects; in addition, mid- and high-dose groups showed microscopic spleen, kidney, and liver changes and, at the highest dose, bone marrow hyperplasia and testicular atrophy were seen (Monsanto Company 1985, as cited in ACGIH 1986, p. 432).

Rats fed PNCB at doses of 0, 0.1, 0.7, or 5 mg/kg/day for up to 2 years showed methemoglobinemia at the two highest levels, and animals in the 5 mg/kg/day group had indications of anemia and pigment accumulation in spleen cells. No treatment-related increase in tumors was observed (Monsanto Company 1985, as cited in ACIGH 1986, p. 432). In a dietary cancer bioassay, rats and mice were given PNCB at unspecified levels for 2 years (Weisberger, et al. 1978). Only mice were affected, with mice of both sexes showing an increase in vascular tumors at the highest dose and male mice showing an increase in liver tumors at the lowest dose (Weisberger et al. 1978).

Maternal toxicity was seen in rats given PNCB by gavage at doses of 15 and 45 mg/kg/day on days 9 through 16 of gestation; at the 45-mg/kg level, fetotoxicity and teratogenicity were also observed (Nair et al. 1985). At 15 mg/kg, maternal toxicity but no fetotoxicity or teratogenic effects occurred; at the lowest dose, the only effect was a small increase in maternal spleen weight. A two-generation reproductive study resulted in a reduced mating index in rats given 0.7 or 5.0 mg/kg/day (Monsanto Company) 1984, as cited in ACGIH 1986, p. 432). Positive responses were observed in a mutation assay of L5178Y TK mouse lymphoma cells (both in the presence and absence of metabolic activation) and in a microbial assay of *Salmonella* strain TA 1535 (in the absence of metabolic activation); however, no evidence of mutagenicity was noted in assays of three other *Salmonella* strains or in assays of Chinese hamster ovary cells, rat hepatocyte primary culture/DNA repair, or rat bone marrow cell clastogenesis (Monsanto Company 1980–1984, as cited in ACGIH 1986, p. 432). PNCB produced DNA damage in the liver, kidney, and brain cells of rats after a single intraperitoneal dose of 30 to 1000 mg/kg (Cesarone et al. 1983) and in cultured hepatocytes at 1.5 hours after a 3-hour treatment (Cesarone et al. 1984).

p-Nitrochlorobenzene may be absorbed through the lungs and skin in humans to produce methemoglobin. Reports of industrial exposures indicate that overexposure causes cyanosis, weakness, and headache (Saita and Moreo 1958; Renshaw 1926). In a study of workmen exposed to average concentrations of PNCB at 55, 125, and 143 ppm and to a PNCB-nitrophenol mixture at 23 ppm, the authors concluded that the mixed exposure did not produce chronic intoxication, but did cause increased methemoglobin, the appearance of Heinz bodies, headache, vertigo, and occasional eczema; these effects could not be attributed definitely either to skin absorption or to the level of PNCB in the mixture (Pacseri et al. 1958). No data are reported for the p-nitrochlorobenzene exposures only (Pacseri et al. 1958).

OSHA is proposing to retain its current 8-hour TWA limit of 1 mg/m³ for p-nitrochlorobenzene, with a skin notation. The Agency preliminarily concludes that these limits are necessary to protect workers from the risk of methemoglobinemia and changes in the spleen, liver, and kidney possible at higher exposure levels. OSHA is retaining the skin notation because dermal absorption of PNCB has been shown to cause systemic effects in animals.

PROPOXUR
CAS: 114–26–1; Chemical Formula: $C_{11}H_{15}NO_3$
H.S. No. 1337

OSHA has no current limit for propoxur. The ACGIH has established an 8-hour TLV–TWA of 0.5 mg/m³ for this white, odorless, crystalline compound.

The oral LD_{50}s in male and female rats are 83 and 86 mg/kg, respectively; for both sexes, the dermal LD_{50} is greater than 2400 mg/kg (Gaines 1969).

Dietary studies in rats at levels of 7.5 mg/kg/day for 28 days or at 800 ppm for 3 months produced no adverse effects (Association of American Pesticide Control Officials, Inc., 1966, as cited in ACGIH 1986, p. 499).

Rats were exposed to propoxur concentrations of 5, 7, 18.7, or 31.7 mg/m³ 6 hours/day, 5 days/week for 12 weeks; animals in the high-dose group showed depressed red blood cell and brain cholinesterase levels, and plasma cholinesterase was depressed by as much as 20 to 30 percent (Association of America Pesticide Officials, as cited in ACGIH 1986, p. 499).

In humans, a few cases of mild propoxur poisoning have been reported among sprayers of this insecticide and among residents of propoxur-treated homes (Vandekar, Hedayat, Plestina, and Ahmady 1968). In a study of human volunteers, a single oral dose of 1.5 mg/kg propoxur caused gastrointestinal symptoms that disappeared two hours after ingestion and a depression in red blood cell cholinesterase; oral doses of 0.75 to 1.0 mg/kg produced no symptoms but did depress erythrocyte cholinesterase (Vandekar, Plestina, and Wihelm 1971).

OSHA is proposing an 8-hour TWA of 0.5 mg/m³ for propoxur. The Agency preliminarily concludes that this limit will protect workers against the risk of cholinesterase inhibition potentially associated with exposure to this substance at the levels permitted by the absence of any OSHA limit. The health evidence forms a reasonable basis for proposing a new limit for propoxur. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

RONNEL
CAS: 299–84–3; Chemical Formula: $(CH_3O)_2PSOC_6H_2Cl_3$
H.S. No. 1349

OSHA currently has a limit of 15 mg/m³ TWA for ronnel. The ACGIH recommends a TLV–TWA of 10 mg/m³ for this white, noncombustible powder.

Ronnel is an indirect cholinesterase inhibitor that affects the blood plasma rather than the red cell acetyl cholinesterase (Plapp and Casida 1958). The acute oral LD_{50} for rats is reported as 1250 and 2630 mg/kg for males and females, respectively. The oral LD_{50} in dogs is greater than 500 mg/kg (McCollister, Oyen, and Rowe 1959). Two-year dietary studies of rats fed up to 50 mg/kg/day showed no effect on growth rate, food consumption, survival or hematopoesis (McCollister, Oyen, and Rowe 1959). In a study by Gladenko and Stuk (1972), albino rats developed clinical symptoms of motor irritation, tremor, increased auditory and tactile sensitivity, lacrimation, and salivation within 2 weeks at exposure levels between 164 and 328 mg/kg; some animals died during the latter part of the study. At exposures below 16.4 mg/kg, no ill effects were observed (Gladenko and Stuk 1972). A 2-year feeding study in dogs exposed at 10 mg/kg showed no ill effects except cholinesterase depletion (Worden, Noel, and Mawdsley-Thomas 1972).

Patch tests of 50 human subjects showed that ronnel has no skin-sensitizing potential (McCollister, Oyen, and Rowe 1959).

OSHA proposes an 8-hour TWA limit of 10 mg/m³ for ronnel. The Agency preliminarily concludes that this limit will protect workers against the risk of cholinergic effects potentially associated with exposure to this substance. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for ronnel if the Agency determines that this limit will substantially reduce significant risk.

SULPROFOS
CAS: 35400–43–2; Chemical Formula: $C_{12}H_{19}O_2PS_3$
H.S. No. 1380

OSHA's Z tables currently have no limit for sulprofos. The ACGIH recommends an exposure limit of 1 Mg/m³ as an 8-hour TWA. Sulprofos, also known as the insecticide Bolstar^R, is a tan liquid.

Kimmerle (1982, as cited in ACGIH 1986, p. 547) conducted an extensive animal study on the effects of sulprofos. He reported that the acute toxicity of sulprofos is species-dependent; rats have an oral LD_{50} of 100 to 300 mg/kg and mice have an oral LD_{50} of 1600 to 1800 mg/kg. The acute dermal toxicity of this substance is low, with an LD_{50} greater than 1000 ml/kg in rats and 800 to 1000 mg/kg in rabbits. In rabbits, sulprofos did not irritate the skin or eyes, and it had no dermal sensitization effects In guinea pigs. Inhalation studies showed no fatalities in rats exposed to aerosol concentrations of up to 4130 mg/m³ of sulprofos over a period of 4 hours. In a 3-week inhalation study in which rats were exposed to aerosol concentrations of 6, 14, or 74 mg/m³, the two highest concentrations produced cholinergic symptoms; no observable effects were seen at the lowest concentration. Two-year feeding studies by Kimmerle (as cited in ACGIH 1986, p. 547) in dogs, rats, and mice showed that sulprofos concentrations of 150 ppm, 250 ppm, or 400 ppm were tolerated by all species, with no sulprofos-related tissue changes, signs of toxicity, or oncogenic effects. The overall NOELs were 10 ppm in dogs, 6 ppm in rats, and 2.5 ppm in mice. Kimmerle's ingestion studies in rats and rabbits at levels of 3, 10, or 30 mg/kg/day of sulprofos showed no embryotoxic or teratogenic effects in these animals, and a three-generation diet study in rats also produced no adverse reproductive effects. Mutagenic studies reported by the same author in mice were negative. Separate subacute inhalation studies also showed no effects on blood cholinesterase levels in rats exposed to 6 mg/m³ Zielhuis and Van der Kreek 1979).

There are no reported cases of poisoning in humans (ACGIH 1986, p. 547).

OSHA is proposing an 8-hour TWA limit of 1 mg/m³ for sulprofos. The Agency preliminarily concludes that this limit will protect workers from the risk of cholinesterase inhibition, the most sensitive indicator of exposure to this currently unregulated substance. The health evidence forms a reasonable basis for proposing a new limit for sulprofos. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

TERPHENYLS
CAS: 26140–60–3; Chemical Formula: $C_{18}H_{14}$
H.S. No. 1384

The current OSHA limit for the terphenyls is 1.0 ppm as a ceiling limit. The ACGIH recommends a 0.5-ppm ceiling limit for these substances. Terphenyls are colorless or light-yellow solids and are used as coolants in nuclear reactors. Commercial preparations contain mixtures of ortho, meta, and para terphenyls.

The terphenyls are primary irritants that cause eye, skin, and respiratory irritation.

Haley, Detrick, Kosmeau et al. (1959) reported that mixtures of terphenyls caused conjunctival irritation when instilled into the eyes of rabbits and damaged guinea pig skin following intracutaneous injection. Cornish, Bahor, and Ryan (1962) determined LD_{50} values of 1900, 2400, and greater than 10,000 mg/kg for the ortho, meta-, and para-terphenyls, respectively. These authors

also conducted 30-day feeding studies of rats involving doses of 250 or 500 mg/kg/day of the individual terphenyl isomers. Rats fed ortho-terphenyl showed elevated liver and kidney weight ratios; rats fed meta-terphenyl displayed elevated kidney weight ratios only; and rats fed para-terphenyl showed no elevation in liver or kidney weight ratios. Two studies by Petkau and Hoogstraaten (1965) and Young, Petkau, and Hoogstraaten (1969) have shown that the terphenyls have nephrotoxic effects and cause hepatic damage in rats fed 33 mg/kg/day. Adamson, Bowden, and Wyatt (1969) published a study in which rats exposed to terphenyl aerosols for 7 hours per day at a concentration of 50 mg/m^3 for a period of 8 days developed morphological changes in their pulmonary cell mitochondria: the number of vacuolated mitochondria was directly related to duration of exposure.

Weeks and Lentle (1970, 1971) conducted a clinical survey of 47 workers with ongoing exposure to terphenyl coolant in a nuclear facility. The study represented 122 man-years of occupational exposure, with duration of exposure ranging from 6 months to 7 years. The airborne concentrations of terphenyl varied, measuring 0.094 mg/m^3 in general working areas and up to 0.89 mg/m^3 in areas with organic piping equipment. The terphenyl coolant was determined to be a primary irritant, even in those workers wearing protective clothing, because skin moistness increased sensitivity to the terphenyls (Weeks and Lentle 1970, 1971). Testa and Masi (1964) reported that at concentrations above 10 mg/m^3 (approximately 1 ppm), workers reported both eye and respiratory irritation.

OSHA is proposing a ceiling of 0.5 ppm for the terphenyls. The Agency preliminarily concludes that this limit will protect exposed workers against the risk of primary irritation of the eyes, skin, and upper respiratory tract and of mitochondrial changes potentially associated with exposure to very low levels of the terphenyls. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for terphenyls if the Agency determines that this limit will substantially reduce significant risk.

m-TOLUIDINE
CAS: 108–44–1; Chemical Formula: C_7H_9N
H.S. No. 1401

m-Toluidine currently has no OSHA permissible exposure limit. The ACGIH recommends a 2-ppm 8-hour TWA, with a skin notation. m-Toluidine is a light yellow liquid.

When m-toluidine was tested on the eyes and skin of rabbits, moderate to strong irritation effects resulted (NIOSH 1979). A mean maximal methemoglobinemia of 60.2 percent was reported to occur following the intravenous administration of 27 mg m-toluidine per kilogram body weight in cats (McLean, Starner, and Thomas 1969). Rodent carcinogenicity studies cited by the ACGIH (1986, p. 589) were either inconclusive or negative.

The effects in humans of exposure to m-toluidine, either absorbed through the skin or via inhalation, are hematuria and methemoglobinemia. Exposure to 40 ppm for 60 minutes causes severe poisoning (Goldblatt 1955). There are no epidemiological studies of workers exposed only to m-toluidine (ACGIH 1986, p. 589).

OSHA is proposing a 2-ppm 8-hour TWA and a skin notation for this previously unregulated chemical. The Agency preliminarily concludes that this limit will protect workers from the risk of metabolic effects, such as hematuria and methemoglobinemia, associated with exposure to m-toluidine at levels currently permitted in the absence of any OSHA PEL. The health evidence forms a reasonable basis for proposing a new limit for m-toluidine. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

2,4,6-TRINITROTOLUENE
CAS: 118–96–7; Chemical Formula: $C_7H_5N_3O_6$
H.S. No. 1413

OSHA's current PEL for 2,4,6-trinitrotoluene (TNT) is 1.5 mg/m^3 as an 8-hour TWA with a skin notation. The ACGIH has set a standard of 0.5 TLV–TWA with a skin notation for this chemical. TNT occurs as yellow needle-like crystals and is used as an explosive.

The ACGIH's limit was selected on the basis of health surveys conducted among occupationally exposed workers. Fairhall (1957) describes dermatitis, cyanosis, gastritis, acute yellow atrophy of the liver, and aplastic anemia as possible effects of exposure to TNT. According to Sollman (1957), blood destruction, leucocytosis or leucopenia, and varying degrees of central nervous system change (probably resulting from anoxia, peripheral neuritis and muscular pains, cardiac muscular and menstrual irregularities, and urinary and renal irritation) can also occur as a consequence of TNT exposure. TNT has irritant properties, and may cause sneezing, sore throat, or skin irritation (von Oettingen 1941).

A study by Goodwin (1972) revealed 36 cases of liver damage in a munitions plant where workers were exposed to a mean air level of 2.38 mg/m^3 TNT over a period of 20 years. Another study (Morton et al. 1976) found elevated levels of liver enzymes in 43 TNT shell-packers and loaders who worked where TNT exposures ranged from 0.3 to 0.8 mg/m^3 over a period of 5 months. In 1975, Djerassi and Vitany published a paper describing hemolytic episodes in three TNT workers with glucose-6-phosphate dehydrogenase deficiency; although these workers were from Iraq, where G–6–PDase deficiency has a high (25 percent) frequency of occurrence, the study is also of concern for other workers having a high frequency of G–6–PDase deficiency.

OSHA is proposing an 8-hour TWA of 0.5 mg/m^3 with a skin notation for 2,4,6-trinitrotoluene. The Agency preliminarily concludes that this limit is necessary to protect workers against the risk of liver damage and hemolytic effects potentially associated with exposure to TNT. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for 2,4,6-trinitrotoluene if the Agency determines that this limit will substantially reduce significant risk.

Preliminary Conclusions

For the class of toxic substances having biochemical/metabolic effects, OSHA preliminarily concludes that occupational exposure presents significant risks. The effects associated with exposure to these substances (which inhibit cholinesterase activity, interfere with the blood's ability to carry oxygen, and produce Antabuse-like symptoms and signs) range from nausea, bronchoconstriction, cardiac irregularities, neurobehavioral effects, and unconsciousness, to coma and death, depending on the severity of the exposure. Because many of these substances are relatively new on the industrial scene, OSHA has no limits for them. This situation means that occupational exposures can potentially be uncontrolled, which increases the urgency that limits be adopted. The Agency thus preliminarily finds that establishing or revising limits for this group of toxicants will reduce occupational risks. The health evidence discussed in this section provides a reasonable basis for proposing revised or new limits for these substances. At the time of the final rule, OSHA will revise or add new limits for this group of metabolic toxins if the Agency

determines that these limits will substantially reduce significant risk.

14. Substances for Which Proposed Limits Are Based on Avoidance of Sensitization Effects

Introduction

OSHA is proposing limits for eight substances based on their sensitization potential. Table C14–1 lists the current OSHA PELs, ACGIH Limits, and NIOSH RELs for each chemical in this group, along with their CAS and HS numbers. In four cases, current OSHA regulations set no limit on exposure. For two substances, OSHA is proposing to reduce its current TWA–PEL. In one other instance, OSHA is proposing to replace its current ceiling limit with an 8-hour TWA–PEL and a 15-minute STEL. For the remaining substance, the addition of a STEL with no change in the current TWA–PEL is proposed. NIOSH has recommended exposure limits for three substances that cause sensitization; OSHA is proposing the REL value for one of these chemicals, isophorone diisocyanate.

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Table C14-1. Substances for Which Limits Are Based on Avoidance of Sensitization

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1066 Captafol (Difolatan)	2425-06-1		0.1 mg/m^3 TWA, Skin	
1100 Cobalt, metal, fume, dust	7440-48-4	0.1 mg/m^3 TWA	0.05 mg/m^3 TWA	-
1222 Isophorone diisocyanate	4098-71-9		0.01 ppm TWA, Skin	0.005 ppm TWA 0.02 ppm Ceiling (10 minutes)
1313 Phenothiazine	92-84-2		5 mg/m^3 TWA, Skin	-
1315 Phenyl glycidyl ether	122-60-1	10 ppm TWA	1 ppm TWA	1 ppm Ceiling (15 min)
1329 Picric acid	88-89-1	0.1 mg/m^3 TWA, Skin	0.1 mg/m^3 TWA 0.3 mg/m^3 STEL, Skin	
1373 Subtilisins (Proteolytic enzymes)	9014-01-1		0.06 ug/m^3 Ceiling**	

Table C14-1. Substances for Which Limits Are Based on Avoidance of Sensitization (continued)

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1398 Toluene-2,4-diisocyanate	584-84-9	0.02 ppm Ceiling	0.005 ppm TWA 0.02 ppm STEL	0.005 ppm TWA 0.02 ppm Ceiling (20 min)

* OSHA's TWA limits are for 8-hour exposures; its STELs are for the durations specified; and its ceilings are peaks not to be exceeded for any period of time.

** The ACGIH TWA-TLV is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

*** NIOSH TWA limits are for 10-hour exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

+ Proposed PEL is the NIOSH REL.

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Description of the Health Effects

A sensitization reaction, also known as an allergic reaction, is defined as an adverse response to a chemical following a previous exposure to that chemical or to a structurally similar one (Klaasen 1986). The subject who suffers an allergic reaction is said to have become sensitized to that chemical. Sensitization is the result of an immune reaction to the chemical; although the initial exposure does not generate an immediate response, the immune system "remembers" the chemical and reacts strongly at the next encounter. A related phenomenon is cross-sensitization. Cross-sensitization occurs when exposure to one chemical elicits a sensitization reaction not only on subsequent exposure to the same chemical but also on exposure to a different chemical, usually one with a similar structure.

The toxic manifestations of a sensitization reaction vary both in their location and severity. In humans, common target organs are the skin and the eyes; typical allergic conditions in these organ systems are allergic contact dermatitis and conjunctivitis, respectively. The respiratory system can also be sensitized; the resulting pathologies include bronchitis and asthma (Dean et al. 1986). These allergic reactions are mediated by the two immunoglobulins IqD or IqE. Involvement of IqD results in delayed contact dermatitis. In contrast, IqE-mediated reactions cause very severe effects, such as acute asthmatic attack, urticaria, and anaphylactic shock, which can be fatal. The unpredictability and potential seriousness of sensitization reactions demand that exposures be carefully controlled.

Sensitivity to a chemical frequently persists throughout the lifetime of an individual; occasionally it may gradually disappear over time. Usually symptoms are not observed after exposure to the sensitizing agent is discontinued. Although treatment for some allergies is possible, avoidance is considered the best way, and sometimes the only way, to regain good health.

An additional cause for concern about exposure to sensitizing chemicals is recent evidence that residual respiratory symptoms may continue even after exposure is discontinued. For example, in the case of toluene-2,4-diisocyanate (TDI), Weill et al. (1981) and Innocenti et al. (1981) found that sensitized workers may exhibit decreased pulmonary function or chronic bronchitis for as long as 3½ years after cessation of exposure.

Dose-Response Relationships and Sensitization Effects

Like other toxic effects, allergic reactions are dose-related; that is, in response to increasing doses of the chemical, increasing numbers of subjects may be sensitized, and the subsequent reactions may be more severe. The time course of sensitization for any one individual is unpredictable. Some individuals are sensitized after only one exposure; others remain resistant to sensitization after a lifetime of exposure. Different people are generally sensitive to different chemicals, although some chemicals are more universally reactive than others, such as the active agent in poison ivy. Various parameters influence the likelihood of sensitization by a particular chemical, such as "the nature of the chemical, concentration, type of exposure, genetic susceptibility and nongenetic idiosyncrasies" (Emmett 1986). Sensitization reactions observed in occupational settings are often the result of dermal or inhalation exposure.

For most of the substances in this group, the proposed limits are based on health surveys and reports of occupationally exposed populations. These studies indicate that exposures below a certain no-effect level generally do not result in individuals becoming sensitized. Where human data were absent or sparse, OSHA has relied on animal evidence to set the proposed limit. However, since chemically induced immunological sensitization occurs among laboratory animals by the same mechanism as in humans (that is, immune reactions in animals can be mediated by either IqB or IqE immunoglobulins), sensitization reactions in animals are generally good predictors of immune reactions in humans.

The discussions below describe OSHA's preliminary findings for the substances in this group. These discussions illustrate the nature of the risk confronting exposed employees, and the extent to which the risk of developing immune sensitization will be reduced among workers by the promulgation of the proposed limits.

CAPTAFOL (DIFOLATAN)
CAS: 2425-06-1; Chemical Formula: $C_{10}H_9Cl_4NO_2S$
H.S. No. 1066

OSHA has no current permissible exposure limit for captafol. The ACGIH recommends a TLV-TWA of 0.1 mg/m³, with a skin notation. Captafol is a white, crystalline substance with a slight but characteristic odor.

A 2-year study conducted by the World Health Organization (Reinhardt and Brittelli 1981) reported growth depression in rats at captafol dietary levels of 1500 and 5000 ppm, and histopathologic examination revealed changes in the livers and kidneys of the animals exposed at these levels. No tumors were observed. In male rats, an increase in liver-to-body-weight ratio was observed at levels of 250 ppm and higher after 12 months of captafol feeding (Reinhardt and Brittelli 1981).

In humans, skin irritation has been reported in both American and Japanese studies of farmers applying captafol as a fungicide. Arimatsu (1970) reports that farmers using captafol have experienced acute contact dermatitis manifesting as erythematous dermatitis and phototoxic eruptions. Khan reports that workers cleaning up in an area where captafol was handled experienced skin and respiratory sensitization (written communication, 1975, as cited in ACGIH 1986, p. 97).

OSHA is proposing a permissible exposure limit for captafol of 0.1 mg/m³ TWA, with a skin notation, to protect exposed workers against the risk of contact dermatitis and respiratory and skin sensitization associated with exposure to this substance at the levels permitted in the absence of any OSHA limit. The Agency preliminarily concludes that the 8-hour TWA, combined with a skin notation, will substantially reduce this risk. The health evidence forms a reasonable basis for proposing a new limit for captafol. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

COBALT (METAL, DUST AND FUME)
CAS: 7440-48-4; Chemical Formula: Co
H.S. No. 1100

OSHA has a current 8-hour TWA limit of 0.1 mg/m³ for cobalt metal dust and fume. The ACGIH recommends a TLV-TWA of 0.05 mg/m³ for cobalt, which is a gray, hard, magnetic, and somewhat malleable metal.

Animal studies indicate that high intratracheal doses (10, 25, or 50 mg) of cobalt metal dust can cause obliterative bronchiolitis adenomatosis in guinea pigs (Schepers 1955). Additional studies in animals have shown that exposure to cobalt dust or fumes causes hypersensitivity reactions. Increases in serum A-globulin and neuraminic acid occurred in dogs and rabbits exposed by inhalation to cobalt metal, metal fume, or carbide blend; injections of $CoCl_2$ produced similar reactions (Stokinger and Wagner 1958). Recent studies conducted on miniswine have shown that inhalation of 0.1 mg/m³ cobalt

metal dust (50 percent alpha and 50 percent beta variety, with a size range of from 0.4μ to 3.6μ) has caused early (onset in 3 months) pulmonary disease. Wheezing, which indicates hypersensitivity, occurred during the fourth week of exposure to 0.1 or 1.0 mg/m³ for 6 hours/day, 5 days/week, for 3 months following a 1-week sensitizing dose (Kerfoot, Frederick, and Domeier 1975).

Pulmonary disease has been reported frequently in workers exposed to cobalt in the manufacture of cemented tungsten carbide (Miller, Davis, Goldman, and Wyatt 1953; Lundgren and Ohman 1954; Lundgren and Swensson 1953). The adverse effect of exposure is generally chronic interstitial pneumonitis. Fatalities have been reported occasionally from exposures to cobalt of 1 to 2 mg/m³ or less (Fairhall, Castberg, Carrozzo, and Brinton 1947; Fairhall, Keenan, and Brinton 1959). An increase in serum A–2 globulin fraction was reported in the case of a welder exposed to fumes containing cobalt; the welder had a history of exertional dyspnea and an abnormal chest X-ray (Seigesmund et al. 1974). Schwartz, Tulipan, and Birmingham (1957) reported that allergic dermatitis has been caused by contact with cobalt and its compounds.

In studies undertaken by the Michigan Department of Health, it was demonstrated that, in the period between 1946 and 1964, improved control measures had successfully reduced cobalt metal dust and fume levels from 14.42 mg/m³ to a level below 0.1 mg/m³. No new cases of systemic toxicity or dermatitis have since been associated with cobalt exposure. The Pennsylvania Department of Health demonstrated that concentrations could be controlled easily to 0.07 mg/m³; without control, concentrations were about 0.5 mg/m³ (ACGIH 1986, p. 144).

OSHA is proposing an 8-hour TWA limit of 0.05 mg/m³ for cobalt metal fume and dust. The Agency preliminarily concludes that this limit will provide workers exposed to cobalt metal dust and fume with protection against the risk of serious pulmonary injury demonstrated to occur at levels above 0.1 mg/m³. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for cobalt if the Agency determines that this limit will substantially reduce significant risk.

ISOPHORONE DIISOCYANATE
CAS: 4098–71–9; Chemical Formula: $C_{12}H_{18}N_2O_2$
H.S. No. 1222

There is no current OSHA limit for isophorone diisocyanate (IPDI). The ACGIH has recommended a TLV–TWA of 0.01 ppm, with a skin notation. NIOSH (1978) recommended a 10-hour TWA of 0.005 ppm for all isocyanates, with a 10-minute ceiling limit of 0.02 ppm.

To date, there is little direct information on health effects associated with exposure to this chemical. However, diisocyanates, in general, cause irritation of the respiratory tract, decreases in pulmonary function, and sensitization. The ACGIH (1986) cited two reports in which workers exposed to isophorone diisocyanate suffered asthma or dyspnea (Clark and Aldons 1981; Tyrer 1979). Neither of these reports contained quantitative exposure data. The ACGIH recommended that the TLV–TWA for 2,4-toluene diisocyanate (TDI) apply to isophorone diisocyanate until more information becomes available; however, the ACGIH did not include its 0.02-ppm TLV–STEL for TDI in this recommendation. NIOSH (1978) came to the same conclusion, reasoning that other diisocyanates would react similarly to TDI on a molar basis; therefore, NIOSH recommended that the limits established for TDI (0.005 ppm TWA and 0.02 ppm as a 10-minute ceiling) apply to all diisocyanates. In supporting the need for a ceiling limit for diisocyanates, NIOSH (1978) cites a report in which 12 workers in an automobile plant developed severe respiratory symptoms after exposure to 0.03 to 0.07 ppm TDI for one week. The ceiling limit recommended by NIOSH is designed to prevent the irritation effects of exposure to the diisocyanates in nonsensitized workers.

OSHA is proposing a 0.005-ppm TWA, a 0.02-ppm ceiling (10-minute), and a skin notation for IPDI. The Agency believes that these limits will protect exposed nonsensitized workers against IPDI's sensitizing effects and minimize asthmatic reactions among sensitized workers. OSHA's preliminary feasibility analysis is based on limited data at this level; the Agency requests additional feasibility information from the public. The health evidence forms a reasonable basis for proposing a new limit for isophorone diisocyanate. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

PHENOTHIAZINE
CAS: 92–84–2; Chemical Formula: $S(C_6H_4)_2NH$
H.S. No. 1313

Currently, neither OSHA nor NIOSH has an occupational exposure limit for phenothiazine. The ACGIH has established an 8-hour TWA–PEL for this substance of 5 mg/m³, with a skin notation.

The Agency's recommendation is based primarily on a study by Mawhinney and Rakow (1968) that showed that exposure to 15 to 48 mg/m³ of phenothiazine was associated with skin sensitization in workers but not with more acute systemic effects. Symptoms of sensitization included burning and an itching sensation of the skin. The skin effects were identifiable as an allergic reaction because the employees developed a tolerance to phenothiazine exposure after a few weeks (Mawhinney and Rakow 1968). Accompanying the sensitization reaction were pinkish-red-colored hair and brown fingernails.

OSHA is proposing an 8-hour TWA limit of 5 mg/m³, which represents a threefold margin of safety over the low end of the exposure range that causes sensitization reactions in workers. Because uncontrolled occupational exposures to phenothiazine pose a risk of developing sensitization reactions, OSHA preliminarily concludes that adopting an exposure limit of 5 mg/m³ will markedly reduce the risk of sensitization for workers. The health evidence forms a reasonable basis for proposing a new limit for phenothiazine. At the time of the final rule, OSHA will promulgate a new limit if the Agency determines that this limit will substantially reduce significant risk.

PHENYL GLYCIDYL ETHER
CAS: 122–60–1; Chemical Formula: $C_6H_5OCH_2CHOCH_2$
H.S. No. 1315

OSHA currently has an 8-hour TWA limit of 10 ppm for phenyl glycidyl ether. The ACGIH recommends a TLV–TWA of 1 ppm for this substance, and NIOSH recommends a 15-minute ceiling limit of 1 ppm. Phenyl glycidyl ether is a colorless liquid.

Exposure to phenyl glycidyl ether causes systemic effects and irritation. Studies in 1956 by Hine, Kodama, Wellington, and colleagues showed pulmonary inflammation and liver changes in some of the rats exposed for 7 hours daily for 50 days to 100 ppm; respiratory distress and minimal eye irritation were also observed in the exposed animals. Intragastric LD_{50} values of 1.40 g/kg for mice and 3.85 g/kg for rats were reported. Animals displayed central nervous system (CNS) depression, and death was caused by respiratory paralysis; in the survivors, these CNS effects were transient. The percutaneous LD_{50} reported for rabbits was 2.99 g/kg. Other studies have

reported a single-dose oral LD_{50} of 4.26 g/kg, although exposure for 8 hours to the near-saturated vapor was not lethal (Smyth, Carpenter, Weil, and Pozzani 1954). Terrill and Lee (1977) reported kidney, liver, spleen, thymus, and testicular changes in rats exposed to phenyl glycidyl ether at 29 ppm for 4 hours daily, 5 days/week for 2 weeks. At concentrations of 12 or 5 ppm, these authors observed no effects other than hair loss after exposures of 6 hours/day, 5 days/week for 9 weeks; however, after 18 weeks, 10 percent of male and 25 percent of female rats exhibited alopecia. These health effects were attributed by the authors to direct irritation of the skin rather than to systemic absorption (Terrill and Lee 1977).

Reports of workers using or handling this substance have described moderate skin irritation on prolonged or repeated contact, as well as several cases of skin sensitization (ACGIH 1986, p. 476).

NIOSH (1978) notes that the glycidyl ethers are biologically reactive compounds because of the presence of the epoxide group; these compounds have also been shown to have cytotoxic effects and to be mutagenic in short-term bioassays. Terrill and Lee (1977) exposed rats repeatedly to 1 ppm PGE and observed no effects, although skin damage was observed at 5 ppm. Inconclusive evidence of testicular degeneration was reported in some of the rats exposed to levels as low as 1.75 ppm (Haskell Laboratory Reports, as cited in NIOSH 1978, p. 114). At 10 ppm, 5 day/week exposures for 10 weeks caused respiratory tract irritation and early signs of liver necrosis in rats (Hine, Kodama, Wellington, Dunlap, and Anderson 1956).

OSHA is proposing to reduce the 8-hour TWA for phenyl glycidyl ether to 1 ppm. The Agency preliminarily concludes that this limit will protect exposed workers from the risk of skin and respiratory tract irritation, skin sensitization, testicular damage, and liver necrosis potentially associated with exposure to the current limit of 10 ppm. OSHA believes that the revised limit will substantially reduce these risks. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for phenyl glycidyl ether if the Agency determines that this limit will substantially reduce significant risk.

PICRIC ACID
CAS: 88–89–1; Chemical Formula: $HOC_6H_2(NO_2)_3$
H.S. No. 1329

OSHA currently has a limit of 0.1 mg/m³ TWA, with a skin notation, for picric acid. The ACGIH also recommends an 8-hour TWA of 0.1 mg/m³, as well as a 15 minute short-term limit of 0.3 mg/m³ and a skin notation. Picric acid occurs as colorless to pale yellow, odorless, and intensely bitter crystals.

Picric acid and its salts are toxic by ingestion, skin contact, or inhalation, and the substances also have skin-sensitization potential (Schwartz 1944). Available reports concerning human exposures describe edema, papules, vesicles, and desquamations of the face, mouth, and nose (Sunderman, Weidman, and Batson 1945). Systemic poisoning following absorption has been reported to produce headache, vertigo, vomiting, nausea, diarrhea, and skin and conjunctival discoloration, as well as discoloration of urine and albuminuria; high-dose exposures caused destruction of erythrocytes and produced gastroenteritis, hemorrhagic nephritis, and acute hepatitis (Sunderman, Weidman, and Batson 1945). Occupational exposure to ammonium picrate dust at concentrations of 0.0088 to 0.1947 mg/m³ caused dermatitis only in those workers who were least exposed; the ACGIH believes that this suggests that desensitization or adaptation occurs with repeated exposure (ACGIH 1986, p. 490).

OSHA proposes an 8-hour TWA of 0.1 mg/m³, a 15-minute STEL of 0.3 mg/m³, and a skin notation for picric acid. The Agency has preliminarily concluded that these limits will protect exposed workers from the risk of systemic poisoning resulting from skin absorption and of sensitization caused by contact with this substance. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for picric acid if the Agency determines that this limit will substantially reduce significant risk.

SUBTILISINS
CAS: 1395–21–7; 9014–01–1; Chemical Formula: None
H.S. No. 1373

Currently, neither OSHA nor NIOSH has an occupational exposure limit for subtilisins. The ACGIH has established a ceiling limit of 0.06 µg/m³. Subtilisins are proteolytic bacterial enzymes used primarily in laundry detergents. They are considered a threat to occupational health because they cause bronchoconstriction and respiratory allergies in addition to irritation of the skin and respiratory tract (ACGIH 1986; Pepys et al. 1969).

A report by the California Department of Public Health (1969) showed that several workers were hospitalized after exposure to subtilisins in a detergent formulation plant where the "safe limit" was set at 0.12 µg/m³. Whether this limit was exceeded or the workers failed to wear the protective gear they were given is unclear.

OSHA is proposing a ceiling limit of 0.06 µg/m³ for the subtilisins; the incident described above suggests that OSHA's proposal to set a ceiling limit of 0.06 µg/m³ for exposure to subtilisins is appropriate and will reduce the risk of skin irritation, respiratory allergies, and sensitization in the exposed worker population. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for subtilisins if the Agency determines that this limit will substantially reduce significant risk.

TOLUENE-2,4-DIISOCYANATE
CAS: 584–84–9; Chemical Formula: $CH_3C_6H_3(NCO)_2$
H.S. No. 1398

The current OSHA limit for toluene-2,4-diisocyanate (TDI) is 0.02 ppm as a ceiling limit. The ACGIH and NIOSH both recommend a TWA of 0.005 ppm and a STEL of 0.02 ppm.

TDI is one of the most frequently encountered sensitizers in the workplace, and is also a known cross-sensitizer. The proposed limit is based on human data showing that workers can develop sensitization reactions at exposure levels below the 0.02-ppm ceiling. Elkins et al. (1965) reviewed the incidence of TDI intoxication in 14 plants in Massachusetts between 1957 and 1962. In eleven instances of TDI intoxication, the average concentration of TDI was 0.015 ppm, and in nine cases the average concentration was below 0.01 ppm. In all plants where the average levels were above 0.01 ppm, TDI had caused respiratory problems. TDI-related respiratory problems were not observed when the average concentration of TDI was maintained below 0.007 ppm.

Williamson conducted two TDI studies (1964, 1965) that revealed a 5-percent sensitization rate in 99 workers exposed for 18 months to levels of TDI averaging below 0.02 ppm. The author believed that accidental spills accounted for the high sensitization rate. Williamson also found that six sensitized workers out of 18 exposed to concentrations of TDI below 0.02 ppm for 14 months showed marked decreases in lung function.

A NOEL (no-observed-effect level) for TDI has been documented. In 1975, Roper and Cromer failed to observe any

symptoms of respiratory illness or changes in pulmonary function in nine employees working in a plant where breathing zone samples showed TDI concentrations of 0.001 to 0.002 ppm.

Wegman et al. (1974, 1977, 1982) observed a dose-response relationship among TDI-exposed employees in the long-term decline of lung function, as documented in test results. Only for those workers exposed to less than 0.002 ppm TDI were the results of lung function tests normal. In keeping with these findings, OSHA is proposing a TWA of 0.005-ppm and a STEL of 0.02-ppm.

OSHA preliminarily concludes that the evidence clearly demonstrates that workers are at risk of pulmonary sensitization reactions at the current PEL, as evidenced by declines in pulmonary function observed among workers exposed below this level. OSHA also believes that establishing a 0.005-ppm TWA with a 0.02-ppm STEL will reduce this risk; TDI-related respiratory effects have not generally been observed among workers exposed below 0.01 ppm. The health evidence forms a reasonable basis for proposing a revision to this level. At the time of the final rule, OSHA will establish a new limit for toluene-2,4-diisocyanate if the Agency determines that this limit will substantially reduce significant risk.

Preliminary Conclusions

For the eight sensitizing agents included in this category of substances, OSHA preliminarily concludes that there are occupational risks associated with exposure. The effects caused by such exposures include skin sensitization, substantial decrements in lung function, bronchoconstriction, and severe skin irritation. Reducing or establishing exposure limits for these toxic substances will substantially reduce these workplace risks.

The health evidence for these sensitizers forms a reasonable basis for proposing revisions to or additions of exposure limits for these substances. At the time of the final rule, OSHA will establish new limits or revise existing limits if the Agency determines that these limits will substantially reduce significant risk.

15. Substances for Which Proposed Limits Are Based on Avoidance of Cancer

The ACGIH has established new TLVs or lowered previous TLVs for 17 substances based on evidence that occupational exposure may be associated with an increased cancer risk. Table C15–1 lists the current OSHA permissible exposure levels (PELs), the ACGIH TLVs, the NIOSH RELs, and the CAS and HS numbers for these substances. OSHA is proposing to revise existing TWA and/or STEL limits for six substances; retain a PEL for three substances currently listed on Table Z–2; adopt NIOSH ceiling limits for two substances; and add limits for six substances not currently listed on OSHA's Z-tables.

The following discussion addresses some general aspects of carcinogenicity, together with the methodology used by OSHA in previous rulemakings to assess carcinogenic hazards. Two representative substances are reviewed in terms of their effects, dose-response considerations, and quantitative risk assessments to evaluate the decrease in risk of developing cancer that is expected after revising or establishing PELs for these substances. In this section, quantitative risk models that are widely accepted by the scientific community are used as a means of estimating cancer risks. The multistage model, which is the model primarily used by OSHA, is preferred over other models because it is based on a more plausible biological mechanism of cancer than the other models.

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TABLE C15 1. Substances for Which Limits Are Based on Avoidance of Cancer

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1008 Acrylamide	79-06-1	0.3 mg/m^3 TWA, Skin	0.03 mg/m^3 TWA, Skin	0.3 mg/m^3 TWA
1020 Amitrole (3-Amino-1,2,4-triazole)	61-82-5	-	0.2 mg/m^3 TWA	-
1028 Asphalt fumes	8052-42-4	--	5 mg/m^3 TWA	5 mg/m^3 Ceiling (15 min)
1033 Beryllium & compounds++	7440-41-7	0.002 mg/m^3 TWA 0.005 mg/m^3 STEL 0.025 mg/m^3 Ceiling (30 min)	0.002 mg/m^3 TWA	0.5 ug/m^3 (No time specified)
1073 Carbon tetrachloride+	56-23-5	10 ppm TWA 25 ppm STEL 200 ppm Ceiling (5 min/4 hr)	5 ppm TWA, Skin	2 ppm Ceiling (60 min)
1086 Chloroform+	67-66-3	50 ppm Ceiling	10 ppm TWA	2 ppm Ceiling (60 min)

TABLE C15-1. Substances for Which Limits Are Based on Avoidance of Cancer (continued)

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1092 Chromic acid & chromates++	7440-47-3	0.1 mg/m^3 Ceiling	0.05 mg/m^3 TWA	25 ug/m^3 TWA 50 ug/m^3 Ceiling (15 min)
1094 Chromyl chloride	14977-61-8	-	0.025 ppm TWA	--
1142 Dimethyl sulfate	77-78-1	1 ppm TWA, Skin	0.1 ppm TWA, Skin	--
1283 Nickel (soluble compounds)	7440-02-0	1 mg/m^3 TWA	0.1 mg/m^3 TWA	15 ug/m^3 TWA
1291 2-Nitropropane	79-46-9	25 ppm TWA	10 ppm TWA	Lowest feasible level
1372 Styrene	100-42-5	100 ppm TWA 200 ppm STEL 600 ppm Ceiling (5 min/3 hr)	50 ppm TWA 100 ppm STEL	50 ppm TWA 100 ppm STEL (15 min)
1399 o-Toluidine	95-53-4	5 ppm TWA Skin	2 ppm TWA Skin	--
1400 p-Toluidine	106-49-0	-	2 ppm TWA Skin	-

TABLE C15-1. Substances for Which Limits Are Based on Avoidance of Cancer (continued)

H.S. Number/ Chemical Name	CAS No.	Current PEL*	ACGIH TLV**	NIOSH REL***
1425 Vinyl bromide	593-60-2	--	5 ppm TWA	--
1426 Vinyl cyclohexene dioxide	106-87-6	--	10 ppm TWA, Skin	--
1436 Zinc chromates (CrVI)++	13530-65-9	0.1 mg/m^3 Ceiling	0.05 mg/m^3 TWA	0.001 mg/m^3 TWA

* OSHA's TWA limits are for 8-hour exposures; its STELs are for the durations specified; and its ceilings are peaks not to be exceeded for any period of time.

** The ACGIH TWA-TLV is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

*** NIOSH TWA limits are for 10-hour exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

+ Proposed PEL is the NIOSH REL.

++ Existing limit is retained.

BILLING CODE 4510-26-C

Description of the Health Effects

Cancer is a life-threatening and particularly insidious disease that is brought about by the invasion of organ systems by abnormal tissue growth. The abnormal tissue is comprised of cells that have been altered in such a way as to cause unrestricted cell growth. As this unrestricted growth progresses, the abnormal tissue begins to interfere with the vital functions of normal organ systems. In the absence of medical intervention, most forms of cancer are ultimately lethal. In some instances (e.g., colon cancer, breast cancer), life can be prolonged through chemotherapy, radiation treatment, surgery, or some combination of these; however, the quality of life of the victims of cancer is usually severely affected. In other instances, such as lung cancer, there is little hope of survival, even when aggressive treatment strategies are employed.

An increased risk of developing cancer has been associated with occupational or environmental exposure to a number of chemical substances. The development of chemically induced cancer in humans and animals is a complex and multi-step process that is not completely understood. It is currently believed that the mechanism by which cancer develops requires at least two stages: initiation and promotion. Initiation occurs when chemicals interact either directly or indirectly with DNA to cause a heritable mutation. Alterations in DNA structure may cause an incorrect reading of the DNA sequence during replication and result in more altered cells, which may eventually be expressed as a tumor. There is a correlation between substances that are mutagenic in *in-vitro* test systems and their ability to cause cancer. Although genotoxic assays are not capable of predicting carcinogenic potential with certainty, such assays are useful for the preliminary identification of substances that may have the potential to cause cancer.

The second stage in the carcinogenic process is promotion. Promotion is considered to be the likely mechanism of action when there is no evidence that a substance interacts with genetic material, e.g., when *in-vitro* mutagenicity assays are negative. Peroxisome proliferation, immunosuppression, and hormonal alterations are examples of promotional events; these events facilitate the unrestricted multiplication of initiated cells, leading to the development of cancer. When a substance or its metabolite possesses both initiation and promotion capabilities, it is considered to be a complete carcinogen, i.e., exposure to the substance alone is sufficient to cause cancer. Examples of such substances that OSHA has recently regulated include asbestos, benzene, ethylene oxide, and formaldehyde.

In all of OSHA's past rulemakings for carcinogens, the Agency has used a weight-of-evidence approach to assess the carcinogenic potential of chemical substances. This approach involves examining all available human epidemiologic studies, clinical and case studies, animal studies, mutagenicity studies, and metabolic studies, combined with a quantitative assessment of cancer risk, to make determinations regarding the potential that occupational exposure to a substance increases the risk of cancer. OSHA relies most heavily on epidemiologic studies of worker populations and well-conducted animal bioassays to make these determinations. OSHA's overall approach to promulgating regulations for carcinogens has been upheld in a number of Court decisions.

The following discussion summarizes how epidemiologic and animal studies are used to assess cancer risk.

Epidemiology studies. Epidemiological studies that include detailed exposure data provide the best evidence for describing a causal relationship between exposure to a substance and the onset of cancer in humans. Epidemiologic evidence has been relied on heavily in OSHA's decisions to promulgate standards for the carcinogens benzene, asbestos, and arsenic. At a minimum, positive epidemiologic studies provide qualitative proof of a causal relationship between exposure to a substance and the development of cancer. A general lack of quantitative exposure data and the long latencies between onset of exposure and appearance of disease may make it difficult to derive quantitative dose-response relationships from epidemiological studies. However, the ability of such studies to link exposures to carcinogens to cancer in humans outweighs these limitations.

Because of the long latency periods associated with chemically induced cancer in humans, these studies cannot be used to detect disease until after irreparable harm has been done. To protect workers or other human populations, therefore, it is necessary to assess the risks of such effects before they occur. The data used for this purpose derive from animal bioassays; these data are used to predict potential human responses and to infer a causal relationship between exposure to a substance and the onset of disease.

Animal data. Animal studies frequently provide the best dose-response data for chemically induced cancer. When relying on such studies, assumptions must be made in order to extrapolate from animal bioassay data to humans; the most important of these are that physiologic, pharmacokinetic, and biochemical parameters are similar between mammalian species. To the extent that adequate metabolic data are available, such data may be used to refine the extrapolation from animals to humans. Despite the need to make such assumptions, it is widely accepted that animals are acceptable surrogates for estimating potential cancer risks in humans. This confidence derives from the observation, after many years of conducting bioassay studies, that there appears to be a reasonable concordance between carcinogenic effects in animals and these effects in humans.

Dose-Response and Quantitative Assessment of Risk

Unlike other chemically induced toxic effects discussed in this preamble, a large body of scientific knowledge has accumulated regarding the mechanisms by which carcinogens act and the quantitative relationship between dose and biological response. As a result of these investigations, several mathematical approaches have been developed that permit estimates to be made of the cancer risk that is associated with exposure to low doses of carcinogenic substances.

Since the dominant view of the carcinogenic process holds that most cancer initiators cause irreversible damage to DNA, there is reason to assume that the dose-response of most carcinogens will follow a linear, non-threshold relationship. The Office of Science and Technology Policy (OSTP 1985) recommends the use of models that incorporate low-dose linearity when the data are limited and uncertainty exists regarding the mechanisms of carcinogenic action. In conducting risk assessments for prior rulemakings, OSHA has generally relied on the linearized multistage model.

The multistage model used to assess cancer risks associated with exposure to substances in this group is GLOBAL83, a model developed by K.S. Crump and colleagues. If P(d) represents the lifetime risk of cancer at dose d, and A(d) is the extra risk over the background rate at dose d, then the multistage model has the following form:

$$A(d) = 1 - \exp[-(q_1 d + q_2 d^2 + \ldots + q_k d^k)]$$

where

$q_i > 0$
$i = 1,2,3,....,k$
and $A(d) = [P(d) - P(0)]/[1 - P(0)]$

For a unique set of q_i, this function will adequately describe (or fit) the experimentally derived data. How well the model describes the data may be mathematically determined by what are termed goodness-of-fit tests. Once the model is fit to the data, the maximum likelihood estimate (MLE) and the 95 percent upper confidence limit (UCL) of A(d) are calculated using the 95 percent upper confidence limit on parameter q_i (q_i*). The MLE is the point estimate of A(d), and is therefore considered the best estimate of extra risk at dose d.

The following discusses the carcinogenicity evidence for the chemicals listed in Table C15–1. A brief discussion of the data and a quantitative risk assessment (where appropriate) are included to demonstrate the reduction in cancer risk that could result from lowering the current OSHA PELs for these potential carcinogens.

ACRYLAMIDE
CAS: 79–06–1; Chemical formula: $CH_2=CHCONH_2$
H.S. No. 1008

The current OSHA PEL for acrylamide is an 8-hour TWA PEL of 0.3 mg/m³. The ACGIH has established a TLV–TWA of 0.03 mg/m³ for acrylamide based on the increased incidence of cancer seen in laboratory animals chronically exposed to acrylamide (Johnson et al. 1986; Bull et al. 1984). Acrylamide has been classified by the ACGIH as a suspected human carcinogen (A2).

Acrylamide is commonly used as a reactive monomer or intermediate in organic syntheses. Polyacrylamide is a polymer used in the manufacture of adhesives, fibers, paper sizing, molded parts, water coagulant aids, and textiles (ACGIH 1986, p. 12).

Past industrial experience and several animal studies have demonstrated that chronic exposure to acrylamide has clinically significant neurotoxic effects (ACGIH 1986, p. 12). In fact, the original ACGIH recommendation of 0.3 mg/m³ as a TWA was intended to protect workers against central nervous system toxicity.

Assays of acrylamide mutagenicity in *S. typhimurium* were negative, with or without microsomal activation (Bull et al. 1984; Miller et al. 1984). However, subchronic exposure to 75 mg acrylamide/kg/day in the diet caused chromosome breaks and aberrations in spermatogonia in mice (Shiraishi 1978). Furthermore, Smith et al. (1985) found that females mated with male rats that had been given 30 or 60 mg acrylamide/liter in drinking water suffered a significantly increased incidence of post-implantation loss. Litters sired by males exposed to 60 mg/L had a significant increase in pre-implantation loss. The authors concluded that acrylamide produces dominant lethality in the male rat (Smith et al. 1985).

No data are available at the present time on the carcinogenic effects of acrylamide exposure in humans. The evidence that acrylamide causes cancer in experimental animals is described in two studies, Bull et al. (1984) and Johnson et al. (1986). Bull et al. (1984) tested acrylamide as a skin tumor initiator in female Sencar mice, using 12-o-tetradecanoyl-phorbol-13-acetate (TPA) as a promoter. Acrylamide was administered 6 times during a 2-week period in doses ranging from 0 to 50 mg/kg body weight. Tumor incidence increased in a dose-related manner, regardless of whether the route of exposure was gastric intubation, i.p. injection, or topical application. The systemic routes of exposure were the most effective. A dose-response relationship was also observed in the induction of lung adenomas in both sexes of A/J mice exposed to acrylamide by either gastric intubation or i.p. injection. These mice were exposed to doses of acrylamide ranging from 0 to 25 mg/kg body weight 3 times per week for 8 weeks.

Johnson et al. (1986) exposed male and female Fischer 344 rats to acrylamide in drinking water for 2 years. Doses ranged from 0 to 2.0 mg acrylamide/kg body weight/day. A statistically significant increase in mortality caused by cancer was observed at the highest dose level during the last 4 months of the study. The incidences of several types of tumors in both sexes were also significantly increased at this dosage. In females, the more frequently observed tumor types were tumors of the mammary gland, central nervous system, thyroid gland (follicular epithelium), oral tissues, uterus, and clitoral gland. In males, increased incidences of tumors in the central nervous system, thyroid gland, and scrotum were observed.

Risk estimate for acrylamide. For the EPA, Crump et al. (1987) performed a risk assessment for acrylamide, derived in part from the results of the Johnson et al. study (1986). For the purposes of risk assessment, the incidences of certain tumors in female rats were pooled: tumors of the mammary gland, brain and spinal cord, thyroid gland, uterus, and oral cavity. The Maximum Likelihood Estimates (MLEs) and Upper Confidence Limits (UCLs) for the current and proposed exposure limits are presented in Table C15–2. GLOBAL 83, a multistage model developed by K.S. Crump, was used to predict these risks. The risk estimate shows that, at OSHA's current PEL of 0.3 mg/m³, the excess risk of death from cancer for workers exposed over their working lifetimes is 10 per 1,000 workers. At the proposed PEL of 0.03 mg/m³, this number would be reduced to 1 per 1,000 exposed workers.

OSHA preliminarily concludes, based on the results of the quantitative risk estimate shown in Table C15–2, that the risks of cancer posed to workers at the current PEL are clearly significant. The Agency finds that reducing the exposure limit to the proposed 0.03 mg/m³ level will reduce this significant risk by a factor of 10, to 1 per 1,000 workers, which is consistent with the Supreme Court's guidance in the *Benzene* decision. OSHA therefore proposes to lower the 8-hour TWA PEL to 0.03 mg/m³.

TABLE C15-2.—MULTISTAGE MODEL ESTIMATES OF CANCER RISK ASSOCIATED WITH WORKING LIFETIME EXPOSURE TO ACRYLAMIDE

Exposure level	Excess cancer deaths per 1,000 workers	
	MLE	UCL
0.3 mg/m³[a]	10	45
0.03 mg/m³[b]	1	5

[a] Current OSHA PEL.
[b] Proposed PEL.
MLE = Maximum likelihood estimate of risk.
UCL = 95 percent upper confidence limit on maximum likelihood estimate of risk.

AMITROLE
CAS: 61–82–5: Chemical Formula: $C_2H_4N_2$
H.S. No. 1020

Amitrole is used as an herbicide and plant growth regulator. There is no current OSHA PEL for amitrole. OSHA proposes a TWA limit of 0.2 mg/m³, the same limit recommended by the ACGIH, based on positive carcinogenicity studies conducted in rats and mice. NIOSH recommends a 10-hour TWA of 0.3 mg/m³ for amitrole.

Amitrole is a potent anti-thyroid agent and has been shown to cause tumors, particularly of the thyroid and pituitary glands, in experimental animals (ACGIH 1986, p. 25). Its tumor-producing activity is thought to be related to its goitrogenic effects, which cause an increase in thyroid stimulating hormone (TSH). Other antithyroid agents that cause TSH stimulation, such as propylthiouracil, have also been shown to produce thyroid tumors (Guyton 1981).

Amitrole has not been shown to be mutagenic in the Ames bacterial mutation assay, a dominant lethal test in male mice, or in assays that measure recessive sex-linked lethal mutations in Drosophila melanogaster (ACGIH 1986, p. 25).

An excess incidence of tumors has been reported to occur among pesticide workers exposed to amitrole alone and in combination with phenoxy herbicides. Although these studies indicate the possible association of increased tumor incidence with exposure to amitrole, confounding factors, such as smoking and concurrent exposure to other pesticides, complicate interpretation of these data.

The Swedish National Board of Occupational Safety and Health ordered an epidemiological evaluation to assess the incidence of cancer among railroad workers exposed to herbicides (Axelson and Sundell 1974). Amitrole was among the pesticides utilized by these workers. Cohorts were separated into groups according to whether they were exposed to amitrole and combinations of other herbicides, phenoxy acids and combinations of other herbicides, or other herbicides alone. A statistically significant increase in the incidence of total tumors and lung tumors was found among workers exposed to amitrole and combinations of other herbicides. Smoking frequency among members of this group was reported to be similar to the frequency of smoking in the general Swedish population.

In a 1980 follow-up to Axelson and Sundell (1974), Axelson and co-workers combined data from the earlier study with data on workers exposed from 1972 to 1978. Cohorts were divided into the following exposure groups: Amitrole alone, phenoxy acids alone, and amitrole and phenoxy acids combined. The reanalyzed data did not show a statistically significant increase in cancer incidence among the workers exposed to amitrole alone; however, the incidence of tumors among workers exposed to amitrole and phenoxy acids together was significantly increased.

Amitrole has been found to be carcinogenic in laboratory animals following dietary exposure to relatively high doses. Attempts to induce tumors by dermal application and subcutaneous injection have been unsuccessful. Studies investigating the carcinogenic potential of amitrole in laboratory animals are reviewed below. The inhibitory effects of amitrole on the production of liver tumors induced by 4-dimethylaminoazobenzene are also discussed.

The effects of lifetime exposure to amitrole were investigated in rats, mice, and hamsters fed diets containing 1, 10, or 100 ppm amitrole (Steinhoff et al. 1983). There was a significant increase in the incidence of thyroid tumors in male and female rats and in the incidence of pituitary tumors in female rats exposed to 100 ppm. An excess incidence of tumors was not found in male or female rats exposed to 1 or 10 ppm. The results of this experiment are presented in Table C15-3. Tumor induction was not observed in male or female mice or hamsters. Another study reported negative results for rats fed diets containing 10, 50, or 100 ppm amitrole (Jukes and Schaffer 1960).

Dermal applications of 0.1 or 10 mg of amitrole produced no increased incidence of tumors in mice (IARC 1974).

In contrast to the negative results obtained in mice following lifetime dietary exposure to 1, 10, or 100 ppm amitrole (Steinhoff et al. 1983), positive results were observed in male and female mice following dietary exposure to higher levels (2192 ppm) of amitrole for 1 year (Innes et al. 1969). Carcinomas of the thyroid were observed in 89 percent (64/72) of the exposed animals (tumor incidence in controls is not reported).

TABLE C15-3. Incidence of Rat Thyroid and Pituitary Tumors Associated With Ingestion of Amitrole

Tumor Site	Concentration in diet (ppm)			
	0	1	10	100
Thyroid (Male)				
- Benign	5/75	9/75	4/75	45/75*
- Malignant	3/75	0/75	3/75	18/75*
Thyroid (Female)				
- Benign	7/75	12/75	8/75	44/75*
- Malignant	0/75	1/75	4/75	28/75*
Pituitary (Female)				
- Benign	14/75	20/75	15/75	36/75*
- Malignant	1/75	2/75	4/75	5/75

* $p < 0.001$, Fisher Exact Test

Positive results were also observed in mice exposed to 1 percent (10,000 ppm) amitrole in the diet (duration of exposure not indicated; Feinstein et al. 1978). Liver tumors developed in 7 percent of the exposed mice; however, the incidence of tumors in unexposed controls was not reported. A small number of thyroid tumors was also reported. The authors hypothesized that the reason more thyroid tumors were not seen was because the animals died of the high toxic doses before such tumors were expressed.

Chronic dietary administration of amitrole in dogs (10, 50, 100, or 500 ppm) and in rainbow trout (1200 or 4800 ppm) did not result in the development of tumors (IARC 1974).

Risk estimate for amitrole. The study by Steinhoff et al. (1983) provides sufficient information to estimate quantitatively the excess cancer risk associated with exposure to amitrole in the workplace. The linearized multistage model was chosen to estimate risk. The incidence of malignant thyroid tumors in female rats was used because these tumors demonstrate a clear monotonic response. Female rats were assumed to weigh 250 g and to consume 25 g of food per day. Human risks were estimated at exposure levels corresponding to the proposed PEL of 0.2 mg/m³, as well as for exposure levels of 0.4 mg/m³ and 1.0 mg/m³. The excess estimated cancer risk, in terms of excess deaths per 1000 employees, is shown in Table C15–4.

TABLE C15–4. Multistage Model Estimates of Cancer Risk Associated With Working Lifetime Exposure to Amitrole

Exposure Level	Excess Cancer Deaths per 1,000 Workers	
	MLE	UCL
0.2 mg/m³[a]	13	17
0.4 mg/m³	26	35
1.0 mg/m³	65	84

[a] Proposed OSHA PEL.
MLE = Maximum likelihood estimate of risk.
UCL = 95 percent upper confidence limit on maximum likelihood estimate of risk.

Exposure to 0.2 mg/m³ of amitrole for an occupational lifetime (45 years) is associated with an estimated 13 excess cancer deaths per thousand employees (1.3 percent). This rate is based on the maximum likelihood estimate (MLE). The 95-percent upper-bound estimate of risk corresponding to this dose is 17 excess cancer deaths per 1,000. By comparison, the maximum likelihood estimates of risk for lifetime exposure to 0.4 mg/m³ or 1.0 mg/m³ are 26 or 65 excess deaths per 1,000 employees, respectively.

Exposure to amitrole has been shown to be associated with an increased incidence of thyroid and pituitary tumors in experimental animals. Although human studies have not demonstrated conclusively that amitrole is carcinogenic, the studies by Axelson and Sundell (1974) and Axelson et al. (1980) provide evidence that amitrole may increase cancer risk among exposed workers. OSHA's risk assessment, based on the animal data, shows that this significant excess cancer risk can be substantially reduced for employees who are currently exposed above the proposed 0.2 mg/m³ limit. Therefore, OSHA is proposing to establish a 0.2 mg/m³ TWA exposure limit for amitrole.

ASPHALT FUMES
CAS: 8052–42–4; Chemical formula: None
H.S. No. 1028

There is no current OSHA PEL for asphalt fumes. The ACGIH recommends a TLV–TWA of 5 mg/m³, based on asphalt's ability to induce skin tumors in mice. NIOSH recommends a ceiling (15 minutes) of 5 mg/m³ for asphalt fumes, measured as total particulates.

Asphalt, also called bitumen, is a mixture of hydrocarbons that is produced by the evaporation of the lighter hydrocarbons from petroleum distillation and subsequent partial oxidation of the residue. Occupational exposure to asphalt fumes can occur during its manufacture or as a result of the secondary heating of asphalt in processes such as road building, roofing, and the coating of construction metals (Thayer et al. 1981). Asphalt is considered a potential carcinogen because of its physical and chemical similarity to coal tar pitch, a recognized human carcinogen. (Benz(a)pyrene (BaP), which is found in coal tar pitch, is a known carcinogen that has been regulated by OSHA in a 6(b) rulemaking.)

The process of manufacturing asphalts or bitumens removes many of the lower molecular weight compounds that have low boiling points, including PAHs with 3 to 7 rings. Therefore, the amount of BaP in asphalt is lower than the amount in coal tar pitch.

The mutagenicity data on asphalt are limited. Claxton and Hufsingh (1980) and Lewtas (1981, 1983) reported positive results for extracts of roofing tar fumes in the Ames *Salmonella*

typhimurium assay. Penalva et al. (1983) also reported that an extract of asphalt was mutagenic in *Salmonella typhimurium* in the presence of biological activation by cytochrome P_{450}. The vapors, particles, and aerosols of this substance were weakly mutagenic in *Salmonella typhimurium*, both in the presence and the absence of biological activation.

Several studies of the carcinogenicity of asphalt have been conducted. Four studies reported negative results (Hueper and Payne 1960; Wallcave et al. 1972; Bingham et al. 1980; and Emmett et al. 1981), and three studies reported positive results (Simmers 1965, 1966; Thayer et al. 1981). These studies are summarized below.

Four studies have reported findings on the carcinogenicity of asphalt by skin painting. Thayer et al. (1981) reported positive tumorigenic activity when two different types of condensates from roofing asphalt volatiles and two types of condensates from coal tar pitch volatiles were tested at two temperatures, 232 and 316 degrees F.

Groups of CD–1 and CH3/HeJ mice were treated with one of the four types of asphalt condensates or one of the four types of coal tar pitch condensates. The mice treated with asphalt received a 25-mg dose of the condensate twice weekly for 18 months. The mice treated with coal tar solids received a dose of 1.5 to 4.2 mg of the condensate (0.15 μg and 0.45 μg BaP, respectively) twice weekly for 18 months. In addition to these treatments, separate groups of mice were treated with the asphalt or coal tar preparations plus ultraviolet light (UVL).

CH3/HeJ mice were more sensitive than the CD–1 mice, with nearly 100 percent of the CH3/HeJ mice and about half of the CD–1 mice developing skin papillomas. UVL decreased the carcinogenic response in all test groups. Also, the asphalt volatiles prepared at the higher temperature produced a greater response. Although Thayer et al. (1981) report a statistically significant increase in the incidence of skin tumors, it is difficult to estimate quantitatively the cancer risk from these data because of the lower reliability of the estimate of q_1 (the slope of the dose-response curve) determined from data providing one dose level and a 100-percent tumorigenic response.

An additional skin painting study (Simmers 1965) supports the results of Thayer et al. (1981). However, in the Simmers study (1965), the asphalt was applied in a solution containing benzene, a human carcinogen.

There is conflicting evidence concerning the carcinogenicity of asphalt following dermal application and inhalation exposure. Hueper and Payne (1960), Wallcave et al. (1971), Bingham et al. (1980), and Emmett et al. (1981) reported negative results by skin painting. Hueper and Payne (1960) also reported negative results when guinea pigs and rats were exposed to asphalt fumes by inhalation.

A positive carcinogenic response was reported by Simmers (1966) and by Hueper and Payne (1960) when rats and mice were exposed to asphalt by injection.

Studies by Hammond et al. (1976), Menck and Henderson (1976), and Milham (1982) reported increased standardized mortality ratios or proportionate mortality ratios for lung cancer among roofers and slaters. It is difficult to show a definite association between asphalt exposure and lung cancer because most people who work with asphalt have concomitant exposure to coal tars (Hammond et al. 1976).

A survey by Baylor and Weaver (1968) reported no significant differences in the health of 462 asphalt workers, compared with the health of controls. These workers had asphalt exposures for a period of at least 5 years and were then given a physical examination, which included a medical and occupational history. Additional information was obtained by questionnaire from paving companies, roofing manufacturers, and truck operators. No adverse health effects were reported in this survey (Baylor and Weaver 1968). However, without mortality data and a longer period of follow-up, no firm conclusions can be drawn from these studies.

Asphalt's ability to induce skin tumors directly in mice and rats demonstrates this substance's potential carcinogenicity. Exposures to asphalt fumes should also be reduced because these fumes contain benzo(a)pyrene. BaP is present in coal tar and is the constituent primarily responsible for the high carcinogenic risk associated with exposure to coal tar pitch. OSHA considered the possibility of performing a quantitative risk assessment for asphalt and concluded that the studies described above did not have sufficient dose-response data to provide an adequate basis for such a risk assessment. OSHA preliminarily concludes that workers exposed at the current unregulated level are at significant risk of cancer. The Agency believes that establishing a limit of 5 mg/m³ for daily exposure to asphalt fumes will substantially reduce this significant risk.

BERYLLIUM AND COMPOUNDS
CAS No.: 7440–41–7
H.S. No. 1033

OSHA's current limits for beryllium are 0.002 mg/m³ as an 8-hour TWA, 0.005 mg/m³ as a ceiling, and 0.025 mg/m³ as a 30-minute peak. The ACGIH has established a TLV–TWA of 0.002 mg/m³. NIOSH (1977) has recommended a ceiling limit of 0.5 μg/m³ (0.0005 mg/m³).

The ACGIH recommendation is based on human evidence describing non-malignant respiratory disease and berylliosis associated with exposure to beryllium. Because of the uncertainty regarding the minimal concentrations of beryllium necessary to produce chronic respiratory disease, and because of the serious nature of the disease, ACGIH recommended a TLV–TWA of 0.002 mg/m³.

At the time of publication of NIOSH's 1972 criteria document on beryllium, NIOSH judged the evidence on beryllium-related cancer to be equivocal. In testimony at OSHA's 1977 hearing on a standard for beryllium, NIOSH presented additional epidemiologic and animal evidence indicating that beryllium is carcinogenic. In particular, NIOSH cited the studies of Bayliss and Wagoner (1977) and Mancuso (personal communication, 1977) showing significant increases in bronchogenic cancer among beryllium-exposed workers. NIOSH therefore recommended that exposure to beryllium not exceed the reliable limit of detection of 0.5 μg/m³.

The ACGIH TLV of 0.002 mg/m³ (TWA) is less stringent than OSHA's existing PELs. The NIOSH REL is based on analytical and sampling limits of detection, which do not necessarily satisfy OSHA's requirements regarding significant risk and feasibility. To consider the carcinogenic effects of beryllium, OSHA would have to perform a quantitative risk assessment, which could not be completed in time for this rulemaking. In light of these facts, OSHA proposes to maintain the Agency's existing PELs of 0.002 mg/m³ TWA, 0.005 mg/m³ as a 30-minute ceiling, and 0.025 mg/m³ as a peak.

CARBON TETRACHLORIDE
CAS: 56–23–5; Chemical Formula: CCl_4
H.S. No. 1073

The current OSHA PELs for carbon tetrachloride are 10 ppm as an 8-hour TWA, 25 ppm as a ceiling limit not to be exceeded for more than 5 minutes every 4 hours, and 200 ppm as a peak limit. The ACGIH has established a 5 ppm 8-hour TWA limit, with a skin notation. A 60-minute limit of 2 ppm has been recommended by NIOSH (1977); this limit reflects the lowest reliably

detectable airborne concentration at the time that the recommendation was made. Carbon tetrachloride is classified as a probable human carcinogen by EPA (group B2) and IARC (group 2B) and as a suspected human carcinogen by ACGIH (category A2), based on positive carcinogenicity studies in rats, mice, and hamsters.

In humans, there have been three case reports of liver tumors developing after carbon tetrachloride exposure (Tracey and Sherlock 1968; Johnstone 1948; Simler et al. 1964). In each case, the patient had been acutely overexposed to carbon tetrachloride, leading to nausea, stomach pains, and signs of severe liver damage.

Blair et al. (1979) studied causes of death in 330 laundry and dry cleaning workers potentially exposed to carbon tetrachloride, as well as to trichloroethylene and tetrachloroethylene. Causes of death based on death certificates were compared to the age, sex, race, and cause-specific distribution of U.S. deaths from the same time period. The proportionate mortality ratio (PMR) for all malignant neoplasms was 128, which was statistically significant, indicating that the study group had a 28 percent higher proportion of total deaths due to cancer compared with the U.S. general population. The excess cancer deaths were due to liver, lung, and cervical cancer and leukemia. Although the excess lung and cervical cancer may reflect socioeconomic differences among these workers, the excess liver cancer seen in this study is consistent with findings in animal studies on carbon tetrachloride.

In animals, carbon tetrachloride has produced hepatocellular carcinomas in all species evaluated (rats, mice, and hamsters). Male rats were given 47 or 94 mg/kg carbon tetrachloride and females were given 80 or 159 mg/kg by gavage for 78 weeks (NCI 1976a,b; 1977). The incidence of hepatocellular carcinomas was increased in animals exposed to carbon tetrachloride as compared with pooled colony controls but was statistically significant only for low-dose females. The lower incidence of carcinomas in female rats at the high dose (1/49) compared to the low-dose (4/49) was attributed by the authors to the increased lethality that occurred among these rats before tumors could be expressed.

In this same study, mice of both sexes received 1250 or 2500 mg/kg carbon tetrachloride by gavage. Hepatocellular carcinomas were found in 49/49 low-dose and 47/48 high-dose males (compared with 5/77 in the control males) and in 40/40 low-dose and 43/45 high-dose females (compared with 1/80 in the control females) (NCI 1976a,b; 1977).

Edwards et al. (1942) administered carbon tetrachloride by gavage to a mouse strain known to have a low incidence of spontaneous hepatomas. The incidence of hepatomas was 52 percent (28/54) for males and 32 percent (6/19) for females. Previous hepatoma incidence data for untreated mice of this strain were 2/71 for males and 0/81 for females. Carbon tetrachloride administered by gavage has also been shown to produce neoplastic changes in the livers of four additional strains of mice (Andervont 1958; Edwards 1941; Eschenbrenner and Miller 1943).

Della Porta et al. (1961) gave weekly gavage treatments to hamsters for 30 weeks, and the animals were observed for an additional 25 weeks. All 10 hamsters dying or killed between weeks 43 and 55 had liver cell carcinomas, in comparison with 0/254 in historical controls.

Risk estimate for carbon tetrachloride. Four data sets have sufficient dose response information to allow quantitative risk estimation: The rat and mouse bioassay data (NCI 1976a, 1976b, 1977); the Edwards et al. (1942) mouse data; and the Della Porta et al. (1961) hamster data. In order to increase sample sizes, the data for male and female animals in each of the studies were pooled. The estimated risk presented in Table C15–11 is the geometric mean of the risk calculated from each data set.

Inhalation risk was calculated assuming an air intake of 20 m^3 per 24-hour day and a 40 percent absorption rate for humans (U.S. EPA 1984). All four studies suggest that a common biological mechanism, cell death and regeneration, occurs and leads to the development of the same tumor type.

Table C15–11 presents the estimates of lifetime human risk from carbon tetrachloride exposure, calculated by the linearized multistage model (GLOBAL83), at the proposed 2 ppm limit, the ACGIH limit of 5 ppm, and the current 10 ppm OSHA PEL. Both the maximum likelihood estimates (MLE) and the 95 percent upper confidence limits of human risk are given, as well as the corresponding expected number of excess cancer deaths per 1,000 exposed over a working lifetime.

TABLE C15-11. Multistage Model Estimates of Cancer Risk Associated with Working Lifetime Exposure to Carbon Tetrachloride

Exposure Level	Excess Cancer Deaths per 1,000 Workers	
	MLE	UCL
2 ppm[a]	3.7	5.2
5 ppm[b]	9.2	13.0
10 ppm[c]	17.9	26.0

[a] Proposed OSHA PEL.
[b] ACGIH TLV.
[c] Current OSHA PEL.
MLE = Maximum likelihood estimate of risk.
UCL = 95 percent upper confidence limit on the maximum likelihood estimate of risk.

Based on this risk estimate, the MLE at the current OSHA limit of 10 ppm is 17.9 excess deaths per 1000 exposed workers, clearly indicating that a significant cancer risk exists at the current PEL.

Risk at the current ACGIH limit of 5 ppm is estimated to be 9.2 excess deaths per 1000 workers exposed over their working lifetimes. At the proposed limit of 2 ppm, residual risk continues to be significant, according to the Supreme Court's guidance in the *Benzene* decision; the risk predicted at 2 ppm is 3.7 excess deaths for 1000 workers exposed over their working lifetimes. However, risk at the 2 ppm limit is substantially reduced compared with risk at the current OSHA PEL of 10 ppm. The estimate shows that approximately 14 cancer deaths would potentially be avoided by reducing the limit to 2 ppm. However, because the 2 ppm limit was set by NIOSH on the basis of the limits for the sampling and analytical method available at the time this 60-minute PEL was recommended. OSHA solicits comments on the technological feasibility of the proposed limit.

CHLOROFORM
CAS: 67-66-3; Chemical Formula: $CHCl_3$
H.S. No. 1086

The current OSHA PEL for chloroform is 50 ppm as a ceiling limit. The ACGIH has established a TLV-TWA of 10 ppm and assigned chloroform an A2 designation. NIOSH (1977) recommends that workplace exposures not exceed 2 ppm as detected by a 60 minute sample; this limit represented the limit of detection at the time NIOSH made the recommendation.

Chloroform is considered to be a probable carcinogen in humans by the ACGIH and the United States Environmental Protection Agency (EPA) and International Agency for Research on Cancer (IARC). Chloroform is given an overall weight-of-evidence classification of B2 by the EPA, and an IARC classification of 2B. These classifications are based on sufficient animal evidence for carcinogenicity and insufficient epidemiological evidence to reach a conclusion based on the human data. The following discussion is based on information from the EPA Health Assessment Document for chloroform (U.S. EPA 1985).

It is currently believed that the carcinogenicity of chloroform results from the formation of reactive metabolites, such as phosgene, that bind to cellular macromolecules. Although there is some evidence to suggest that chloroform is weakly mutagenic, the results of most mutagenicity tests are negative.

In humans, there are no epidemiological studies that evaluate populations exposed only to chloroform, although there are several studies that examine populations exposed to chloroform in chlorinated drinking water. However, because chloroform is not the only potential carcinogen present in chlorinated water, the epidemiological data are considered inadequate to use as the basis for a quantitative risk assessment. Thus, a causal relationship between cancer and chloroform exposure cannot be determined based on epidemiological studies alone, although these studies can be used to provide general support for findings in animal studies.

A case-controlled study indicates a significant association between colon cancer and exposure to chlorinated drinking water contaminated with organic material (Young et al. 1981). Significant positive associations were also found for chloroform levels in drinking water and the incidence of mortality due to cancer of the bladder, rectum, and large intestine (Hogan et al. 1979). Similar results also have been found by others (Cantor et al. 1978 and Gottlieb et al. 1981). However, although these studies suggest an association between exposure to chloroform and an increased risk of cancer, a definite causal relationship between the development of colon and bladder cancer and exposure to chloroform

cannot be determined solely from these studies.

In animals, several long-term studies provide strong evidence for the carcinogenic activity of chloroform. Chloroform has been shown to produce statistically significant increases in renal epithelial tumors in male rats and hepatocellular carcinomas in several strains of mice. The carcinogenic activity of chloroform in these studies is specific to the kidney and liver.

The carcinogenic activity of chloroform was investigated in rats exposed to chloroform by gavage for 78 weeks (NCI 1976). Male rats were administered doses of 90 or 180 mg/kg/day, and female rats were administered doses of 100 or 200 mg/kg/day. A statistically significant dose-related increase in renal epithelial tumors was observed in treated male rats compared with untreated, matched controls; these tumors were described as carcinomas and adenomas. No increase in the incidence of tumors was observed in chloroform-treated female rats.

In this same study, the carcinogenicity of chloroform was evaluated in mice exposed chronically to chloroform by gavage (NCI 1976). Male mice were exposed to doses of 138 or 277 mg/kg/day and females to 238 or 477 mg/kg/day for 78 weeks. There were significant dose-related increases in the incidence of hepatocellular carcinomas in chloroform-treated male and female mice. The increase of tumors in male mice for low and high doses was 36 percent and 98 percent, respectively. For female mice, the increases were 80 percent for the low dose and 95 percent for the high dose of chloroform.

The carcinogenic potential of chloroform in mice was further investigated in two additional studies (Roe et al. 1979; Jorgenson et al. 1985). Doses of 17, 60, or 100 mg/kg/day were administered to four different strains of male and female mice (C57BL, CBA, CF/1, and ICI) by gavage for 80 weeks (Roe et al. 1979). The incidence of kidney tumors, described as hypernephromas, was significantly elevated in the ICI strain. Moderate to severe renal changes were observed in the male mice of the other strains, but no significant increase in renal tumors was reported. Tumors were not observed in female mice.

The carcinogenicity of chloroform administered in drinking water was investigated in male rats and female mice (Jorgenson et al. 1985). Animals were treated with drinking water containing chloroform concentrations of 200, 400, 900, or 1800 mg/L for 104 weeks. There was a marked increase in the number of kidney tumors (described as tubular cell adenomas and adenocarcinomas) in rats. However, the incidence of tumors in female mice was not significantly increased.

Risk estimate for chloroform. The NCI (1976) rat study, which demonstrated a statistically significant increase in the incidence of renal tumors in male rats, was the data set used for the quantitative risk estimation. Although there are no data concerning the carcinogenicity of chloroform following inhalation exposure, the risk from inhaled chloroform is considered to be equivalent to the risk from ingested chloroform. The linearized multistage, one hit, and Weibull models were used. The maximum likelihood estimates of excess cancers over an occupational lifetime for a population of 1000 and the 95 percent upper bound estimates are summarized in Table C15–12. The Weibull model is similar to the logit and probit models. However, by using only one data set, the logit, probit, and multihit models failed to converge.

The results of the data analysis presented here are similar to the results of other models described by the EPA (1985) for chloroform. These three models clearly demonstrate, based on the MLE estimates, that a significant cancer risk exists at the current PEL of 50 ppm. The risks estimated to exist at the current PEL are of the same order of magnitude as the risks determined by OSHA to be associated with other carcinogens that OSHA has regulated (e.g., benzene, ethylene oxide). Therefore, OSHA preliminarily concludes that a significant risk of cancer exists at the current PEL of 50 ppm, with estimated risks ranging from 9 to 32 excess deaths per 1,000 workers. The Supreme Court indicates that a reasonable person "might well consider a risk of 1.0 per 1000 significant, and take steps to decrease or eliminate that risk" (*I.U.D.* v. *A.P.I.*, 448 U.S. 655). OSHA also preliminarily finds that revising the PEL to 2 ppm will substantially reduce this risk by from 92 to 99 percent. Therefore, OSHA is proposing a 2 ppm short-term limit (15 minutes) as the PEL. OSHA's preliminary feasibility analysis is based on limited data at this level, and the Agency accordingly requests additional feasibility information from the public.

Table C15-12. Multistage Model Estimates of Cancer Risk Associated with Working Lifetime Exposure to Chloroform

Exposure Level	Excess Cancer Deaths Per 1,000 Workers	
	MLE	UCL
Multistage		
2 ppm[a]	0.22	3.6
10 ppm[b]	1.17	8.90
50 ppm[c]	12.20	44.20
One Hit		
2 ppm[a]	2.58	3.81
10 ppm[b]	6.43	9.44
50 ppm[c]	32.00	46.80
Weibull		
2 ppm[a]	0.07	0.65
10 ppm[b]	0.56	3.22
50 ppm[c]	9.08	31.80

[a] Proposed OSHA PEL.
[b] ACGIH TLV.
[c] Current OSHA PEL.
MLE = Maximum likelihood estimate of risk.
UCL = 95 percent upper confidence limit on the maximum likelihood estimate of risk.

CHROMIC ACID, CHROMATES: ZINC CHROMATES
CAS: 7440-47-3; 13530-65-9
H.S. No. 1092; 1436

The current OSHA limit for chromic acid and chromates is a ceiling limit of 0.1 mg/m³ measured as CrO_3. The ACGIH has established a TLV-TWA of 0.05 mg/m³ as Cr(VI) for both the soluble and insoluble forms of chromate (except zinc chromate), and has designated insoluble chromates as confirmed human carcinogens (A1). NIOSH (1975) has recommended that exposure to the noncarcinogenic forms of chromium (VI) be limited to 0.025 mg Cr(VI)/m³ as a 10-hour TWA and 0.05 mg Cr(VI)/m³ as a 15-minute ceiling. For the carcinogenic (i.e., insoluble) forms of chromium (VI), NIOSH recommends a 10-hour TWA limit of 0.001 mg Cr(VI)/m³.

The ACGIH recommendation for both soluble (noncarcinogenic) and insoluble (carcinogenic) forms of Cr(VI) is based largely on reports by Bloomfield and Blum (1928) and by the U.S. Public Health Service (1953) that demonstrate nasal irritation and some evidence of liver enlargement and kidney dysfunction among chromate workers exposed to 0.06 to 0.07 mg Cr(VI)/m³. The ACGIH also cites a report by Mancuso and Hueper (1951) of excess lung cancer among chromate workers exposed to 0.01 to 0.15 mg/m³ soluble chromate and 0.1 to 0.58 mg/m³ insoluble chromate. Animal data cited by the ACGIH indicate that insoluble chromate salts were likely to have been responsible for the increased incidence of cancer seen in the Mancuso and Hueper study. The ACGIH (1986) concluded that the 0.05 mg/m³ TLV-TWA would protect workers from chromium-induced nasal irritation and possible liver or kidney damage, and, in the case of the insoluble chromates, would provide an adequate margin of safety from respiratory cancer. (It should be noted that the 0.05 mg/m³ limit, expressed as Cr(VI), approximates 0.01 mg/m³ measured as CrO_3.)

NIOSH (1975) cited several studies showing inflammation and ulceration of the nasal cavity at short-term exposure levels greater than 0.1 mg CrO_3/m³. In its 1973 Criteria Document on chromic acid, NIOSH recommended that the current OSHA ceiling limit (0.1 mg CrO_3/m³) be supplemented with an 0.05 mg CrO_3/m³ 10-hour TWA limit. In its 1975 Criteria Document on chromium (VI), NIOSH reaffirmed these limits but extended their application to all forms of noncarcinogenic chromate. Thus, the 0.1 mg CrO_3/m³ ceiling limit corresponds to a 0.05 mg Cr(VI)/m³ ceiling limit, and the 0.05 mg CrO_3 TWA limit corresponds to a 0.025 mg Cr(VI)/m³ TWA. For the carcinogenic (insoluble) forms of Cr(VI), NIOSH recommends the lowest detectable level, which is 0.001 mg Cr(VI)/m³ as a 10-hour TWA.

Zinc chromate is an insoluble, carcinogenic form of chromate. As such, the current OSHA limit for chromic acid and chromates applies, as does the NIOSH limit of 0.001 mg/m³ limit for

carcinogenic chromates. The ACGIH (1986) reviewed several small epidemiologic studies of zinc chromate workers, all of which reported excesses of lung cancer. Because of the consistent evidence, the ACGIH (1986) classified zinc chromate as a confirmed human carcinogen (A1) and reduced the TLV to 0.05 mg Cr(VI)m^3.

Evaluation of the alternate recommendations is complicated by the different valence states of chromium compounds, the different methods of measurement (CrO_3 or Cr(VI)), and differences in defining those substances that present a cancer hazard (soluble vs. insoluble or valence state). The 0.05 ppm TWA-TLV is less restrictive than the current 0.05 ppm ceiling limit (as Cr(VI)), and would not be considered a revised PEL. OSHA therefore tentatively proposes that the existing PEL of 0.1 mg/m^3 (measured as CrO_3) be maintained. Because of the problems noted above, OSHA will consider whether the NIOSH REL should be adopted in place of the 0.1 mg/m^3 limit (measured as CrO_3) during the public hearing phase of this rulemaking. OSHA will also consider whether to place these substances on its regulatory agenda for future consideration for section 6(b) rulemaking, rather than making any changes as part of this rulemaking.

CHROMYL CHLORIDE
CAS: 14977-61-8; Chemical Formula: CrO_2Cl_2
H.S. No. 1094

There is no existing OSHA PEL for chromyl chloride. The ACGIH recommends that a TWA of 0.025 ppm be established, based on this substance's carcinogenic potential (ACGIH 1986, p. 141). The evidence in humans is considered sufficient for the carcinogenicity of chromium and chromium compounds, and these have been given a Group 1 classification by the International Agency for Research on Cancer. As discussed below in connection with chromic acid, chromates, and zinc chromates, the chromium compounds present several important issues that require detailed analysis and can most appropriately be handled in an individual section 6(b) rulemaking. OSHA intends to commence work on this rulemaking as priorities and research permit.

DIMETHYL SULFATE
CAS: 77-78-1; Chemical Formula: $(CH_3)_2SO_4$
H.S. No. 1142

OSHA's current limit for dimethyl sulfate is 1 ppm. The ACGIH considers this substance a suspected human carcinogen and has given it a classification of A2 (ACGIH 1986, p. 212). The ACGIH's TLV-TWA for this substance is 0.1 ppm.

Dimethyl sulfate is commonly used in the manufacture of many organic chemicals. It has been shown to be carcinogenic in rats by inhalation exposure, subcutaneous injection, and prenatal exposure. The rat is the only animal species in which the carcinogenesis of dimethyl sulfate has been tested (IARC 1974).

The carcinogenic activity of dimethyl sulfate was investigated in male rats chronically exposed to subcutaneous injections of 8 or 16 mg/kg body weight per week (Druckrey et al. 1966). Local sarcomas with metastases to the lung and regional lymph nodes were observed at both dose levels. A single subcutaneous injection of dimethyl sulfate (50 mg/kg) also produced local sarcomas with metastases to the lung (Druckrey et al. 1970). However, tumors did not develop following chronic weekly intravenous injections of dimethyl sulfate (2 or 4 mg/kg) (Druckrey et al. 1970). Control data were not reported for either of these studies.

The carcinogenic potential of dimethyl sulfate exposure by inhalation was also evaluated in male rats (Druckrey et al. 1970). Animals were exposed to approximately 3 or 10 ppm dimethyl sulfate for 1 hour per day five times weekly for 130 days. Malignant tumors developed in 15 percent (3/20) of the rats exposed at 3 ppm and in 18 percent (5/27) of the rats exposed at 10 ppm.

Pregnant rats were exposed to a single intravenous injection of dimethyl sulfate (20 mg/kg body weight) on day 15 of gestation and the incidence of malignant tumors in the offspring was investigated for 1 year. Tumors were reported in 7/59 of the offspring. However, the incidence of tumors in the control group was not indicated. The results of this study are complicated because several rats died (number of deaths not specified) from the acute toxic effects of dimethyl sulfate, and the incidence of tumors in the control group was not reported.

There is little information available regarding the carcinogenicity of dimethyl sulfate in humans. A case study of workers exposed to dimethyl sulfate reported that three workers developed bronchial cancer (Druckrey et al. 1966). However, an epidemiological study by the E.I. du Pont de Nemours Company (cited in ACGIH 1986, p. 213) demonstrated no increase in the incidence of respiratory cancer among workers exposed to dimethyl sulfate.

OSHA considered the possibility of performing a quantitative risk assessment for dimethyl sulfate and concluded that the studies described above did not have sufficient dose-response data to provide an adequate basis for such a risk assessent. Dimethyl sulfate induces malignant tumors in animals both by inhalation and ingestion, and there is thus sufficient evidence in animals to predict that workers exposed to dimethyl sulfate are at significant risk of developing cancer; exposures at levels only three times the existing PEL resulted in a significant number of tumors. OSHA preliminarily concludes that reducing the current limit to 0.1 ppm as an 8-hour TWA will reduce this risk and will substantially reduce this significant risk of cancer mortality.

NICKEL (SOLUBLE COMPOUNDS)
CAS: 7440-02-0; Chemical Formula: Varies
H.S. No. 1283

The current OSHA PEL for all forms of inorganic nickel (as Ni) is 1 mg/m^3 TWA. The ACGIH has recommended that the TLV-TWA for soluble forms of nickel be reduced to 0.1 mg/m^3. NIOSH recommends that exposure to any form of inorganic nickel be maintained at or below 0.015 mg/m^3.

A variety of toxic effects results from exposure to nickel compounds. Soluble nickel salts cause contact dermatitis in sensitized individuals and eye irritation (ACGIH 1986, p. 422). High rates of asthmatic lung disease have been reported among nickel-plating workers (EPA 1986).

Three soluble nickel compounds have been tested for their carcinogenic potential: Nickel chloride, nickel sulfate, and nickel acetate. In addition, the sparingly soluble compounds, nickel carbonate and nickel hydroxide, have been studied. As a whole, the results of animal studies suggest that some soluble nickel compounds are potentially carcinogenic. Results from occupational studies are inconclusive for soluble nickel compounds because of the presence of several types of nickel compounds in the facilities studied. One cohort of nickel refinery electrolysis workers exposed to nickel sulfate experienced an increased risk of lung cancer compared with the facility's roasting and smelting workers (Doll 1958). OSHA proposes to lower the PEL for soluble nickel compounds to 0.1 mg/m^3, due to recent evidence that, in animals, exposure to low levels causes lung damage that is indicative of pre-neoplastic changes.

Nickel chloride has been reported to be mutagenic in *Salmonella typhimurium and Cornebacterium*, but negative in *E. coli* (EPA 1986). The positive studies are not considered conclusive, however, because the *S. typhimurium* report is an abstract lacking detailed data and *Cornebacterium* is not the usual species

used in these tests. Amacher and Paillet (1980) reported that nickel chloride was mutagenic in mouse lymphoma cells and demonstrated a dose-response relationship.

Some *in-vitro* studies using soluble nickel compounds report finding chromosomal aberrations (EPA 1986). These studies do not demonstrate a dose-response relationship or statistical significance, which weakens their findings. Several *in-vivo* studies have failed to detect chromosomal aberrations (EPA 1986). However, several *in-vitro* studies on nickel sulfate and nickel chloride have reported findings of sister chromatid exchanges (EPA 1986).

Some animal studies on soluble nickel compounds suggest that these compounds are carcinogenic in animals. Strain A mice receiving intraperitoneal injections of nickel acetate had an increased rate of lung adenomas and adenocarcinomas that was statistically significant in the high dose group (Stoner et al. 1976). The animals were injected 3 times per week for 8 weeks at 72, 180, or 360 mg/kg.

EPA (1986) reported a study in which rats were given monthly intramuscular injections of 35 mg/kg nickel acetate for 4 to 6 months (Haro et al. 1968, as reviewed by Rigaut 1983). Twenty-two percent of the treated rats developed sarcomas. Payne (1964) observed tumor responses in rats after intramuscular implantation of 7 mg nickel acetate, nickel sulfate, nickel chloride, or nickel carbonate. Implant site sarcomas developed in 1 of 35 rats exposed to nickel acetate, 1 of 35 rats exposed to nickel sulfate, none of 35 rats exposed to nickel chloride, and 4 of 35 rats exposed to nickel carbonate.

Results of other studies on nickel sulfate have been negative. Three studies used intramuscular injection in rats and reported that no tumors developed in the treated group (Gilman 1962; Gilman 1966; and Kasprazak et al. 1983). An ingestion study also reported no tumors among treated rats or dogs (Ambrose et al. 1976).

Gilman (1966) administered 5 mg nickel hydroxide to rats by intramuscular injection in each thigh. Nineteen out of 40 injection sites developed sarcomas. Kasprazak et al. (1983) gave rats intramuscular injections of nickel hydroxide in gel, crystalline, or colloidal form. Five out of 19 animals receiving the gel developed sarcomas (2 with metastasis to the lung), 3 out of 20 receiving the crystalline form developed sarcomas (1 with metastasis to the lung), and none of 13 rats receiving the colloid developed tumors.

Bingham et al. (1972) exposed rats by inhalation to 0.1 mg/m^3 nickel chloride for 12 hours a day for 2 weeks. Animals showed evidence of pulmonary damage and hyperplasia. Rats and guinea pigs exposed daily to 1.0 mg/m^3 (as Ni) nickel chloride for 6 months showed increased lung weight, which is an indication of pulmonary damage and hyperplasia (Clary 1977). Rabbits inhaling 0.3 mg/m^3 (as Ni) nickel chloride aerosol for 30 days showed a doubling in alveolar cell number and volume of alveolar epithelial cells, as well as nodular accumulation of macrophages and laminated structures (Johansson et al. 1983). These studies indicate that exposure above the current OSHA PEL of 1.0 mg/m^3 for soluble nickel is associated with increased cell turnover in the lung; the hyperplasia observed in the lungs of treated animals is indicative of pre-neoplastic change.

Electrolysis workers at a refinery in Kristiansand, Norway experienced the highest lung cancer risk in the plant (Mangus et al. 1982). Electrolysis workers were exposed to an aerosol composed predominantly of nickel sulfate, which was estimated (to contain nickel) at a concentration of 0.2 mg/m^3 (EPA 1986). However, exposure to nickel subsulfide and oxides may have occurred in the electrolysis building, and the electrolysis workers may have worked in other process departments (Grandjean et al. 1988). Roasting and smelting workers were exposed to an estimated average of 0.5 mg/m^3 (as Ni) of roasting dust.

The standardized mortality ratios (SMRs) for lung cancer were 550 for electrolysis workers, 390 for other process workers, and 360 for roasting and smelting workers. The pattern of SMRs for nasal cancer was different: 2600 for electrolysis workers, 2000 for other process workers, and 4000 for roasting and smelting workers. The results seem consistent with studies showing that roasting and smelting workers have the highest concentrations of nickel in the nasal mucosa, presumably because of the relatively larger particles resulting from roasting. Electrolysis workers have higher plasma and urine levels of nickel, suggesting that nickel aerosolized by this process penetrates to the deep lung (EPA 1986).

In contrast to the study of Norwegian nickel refinery workers, an increased risk of lung cancer was not found among electrolysis workers at Port Colborne, Ontario (EPA 1986). The characteristics of exposure, however, may not have been similar to those experienced by the workers in Norway.

Risk estimate for soluble nickel compounds. OSHA considered the possibility of performing a quantitative risk assessment for the nickel compounds. However, the animal studies on the effects of exposure to the soluble compounds of nickel do not contain sufficient dose-response data for such an assessment. Risk estimates can be derived from the study by Mangus et al. (1982), which reported excesses in lung cancer and nasal tumors among electrolysis workers exposed predominantly to soluble nickel aerosols. To perform the risk assessment, OSHA used the multiplicative and average relative risk models (EPA 1986) applied to the excess lung cancer response reported by Mangus et al. (1982). The results of the risk assessment are reported in Table C15–5.

TABLE C15-5. Multistage Model Estimates of Lung Cancer Risk Associated With Working Lifetime Exposure to Soluble Nickel

Exposure Level	Excess Cancer Deaths Per 1,000 Workers: Estimate Using Multiplicative Relative Risk Model	Estimate Using Average Relative Risk Model	Midpoint of Range
0.1 mg/m^3 [a]	0.25	2.5	1.4
1.0 mg/m^3 [b]	2.5	25.0	14.0

[a] Proposed OSHA PEL.
[b] Current OSHA PEL.

Animal studies have clearly demonstrated that lung changes indicative of pre-neoplastic responses occur as a result of exposure to soluble nickel at levels ranging from 0.1 mg/m^3 to 1.0 mg/m^3 for periods of time that were far less than the lifetime of the animals. From these studies, it is clear that the current OSHA PEL of 1.0 mg/m^3 for soluble nickel is inadequate to offer protection against these pathological changes.

For example, using the midpoint of the range of excess deaths predicted by the two models (Table C15–5), 14 excess deaths would be predicted to occur among 1,000 workers exposed over their working lifetimes at the current limit. This risk is reduced by 90 percent at the proposed level, i.e., is reduced to 1.4 excess deaths per 1,000 exposed workers. The residual risk at 0.1 mg/m^3 is still significant, if the guidelines provided by the Supreme Court in the *Benzene* decision are used. Soluble nickel has exhibited carcinogenic activity in animals treated by injection. The finding by Mangus et al. (1982) of excess lung and nasal tumor cancer among workers exposed to soluble nickel, although complicated because of concurrent exposure to insoluble nickel, is consistent with these animal findings. A quantitative assessment of cancer risk based on the Mangus et al. (1982) study shows that the potential cancer risk at the current PEL represents a significant cancer risk, and that reducing the exposure limit for soluble nickel to 0.1 mg/m^3 results in a substantial reduction of that risk. Because the 0.015 mg/m^3 NIOSH limit is based on analytical and sampling limits of detection, the significant risk and feasibility requirements to which the Agency may not be satisfied. OSHA preliminarily concludes that reducing the current PEL for soluble nickel is necessary and proposes to reduce its limit to a 0.1-mg/m^3 8-hour TWA, measured as elemental nickel, as an interim PEL. As future priorities permit. OSHA may consider the need for a more restrictive standard for these compounds.

2-NITROPROPANE
CAS: 79–46–9; Chemical Formula: $CH_3CH(NO_2)CH_3$
H.S. No. 1291

OSHA's current limit for 2-nitropropane (2-NP) is 25 ppm; the ACGIH has an established limit for this substance of 10 ppm as an 8-hour TWA and classifies 2-nitropropane as a suspected human carcinogen (A2). 2-Nitropropane is used as a chemical intermediate, solvent, and a component in paint, ink, and varnishes (Fiala et al. 1987). Approximately 185,000 workers are exposed to 2-NP during its production and use in printing, highway maintenance (traffic markings), shipbuilding and maintenance (marine coatings), furniture and plastic product finishes, and food packaging (NIOSH 1980).

In rats and chimpanzees, 2-NP is metabolized by microsomal enzymes in the liver to acetone, low levels of isopropanol, and nitrite (Mueller et al. 1983). Methemoglobin formation is associated with the metabolism of nitropropane and has been reported in cats exposed to 280 ppm of 2-NP for 7 hours. Sensitivity to the toxic effects of 2-NP in animals varies by species (Dequidt et al. 1972; ACGIH 1986, p. 441).

The mechanisms of carcinogenicity of 2-NP are thought to involve the release of nitrite and the formation of a reactive azoxy intermediate that can react with cellular macromolecules (Williams and Weisburger 1986).

In mutagenicity tests, 2-NP increased the frequency of mutations in all strains of *Salmonella typhimurium* with and without metabolic activation. Positive mutagenicity results were reported in *Salmonella typhimurium* strains TA100, TA1535, and TA98 by Lofroth et al. (1981) and Speck et al. (1982). 2-NP was not shown to be mutagenic in the mouse micronucleus test (Hite and Skeggs 1979).

Acute exposures to 2-NP from occupational accidents have been reported to cause severe liver toxicity and subsequent death in humans (ACGIH 1986, p. 441). However the

available epidemiology data on the chronic health effects of occupational exposure to 2-NP do not contain sufficient dose-response data to use as a basis for quantitative risk estimation. An unpublished retrospective mortality study of 1,481 potentially exposed workers from a nitropropane production plant found no increase in liver cancer or liver disease mortality. However, lack of exposure data, the small number of workers with long exposures (greater than 15 years), and a short latency period make interpretation of the results of this study difficult (Miller and Temple 1979; Bolender 1983).

There are two studies that report high incidences of liver tumors in male rats exposed to 2-NP by gavage and inhalation. Fiala et al. (1987) administered, by gavage, 1 mmol/kg body weight (approximately 27 mg/treatment per 300-gram rat) of 2-NP in a 10 percent aqueous Emulphor EL-620 vehicle to male Sprague-Dawley rats three times weekly for 16 weeks. Dosing was discontinued after 16 weeks because of excessive mortality in the treated rats. Seventy-seven weeks from the first treatment, the surviving rats were sacrificed and subjected to necropsy. All (100 percent) of the treated rats examined had developed hepatocarcinomas.

The results of the Fiala et al. study (1987) support the earlier positive results reported by Lewis et al. (1979). In the Lewis et al. study (1979), male Sprague-Dawley rats and male New Zealand White rabbits were exposed via inhalation to 27 ppm or 207 ppm of 2-NP for 7 hours/day, 5 days/week for 6 months. At the end of 6 months, all 10 rats in the high-dose group exhibited hepatocellular carcinomas and neoplastic nodules. No exposure related lesions were seen in the rats exposed to 27 ppm, and no exposure related lesions were observed in any of the rabbits.

One high-dose and two low-dose studies reported negative results for rats exposed to 2-NP vapors. Griffin et al. (1978) reported no hepatic carcinomas on exposing male and female rats to 200 ppm of 2-NP by inhalation using a protocol similar to that described by Lewis et al. (1979). Although no hepatic carcinomas were observed, the following effects (generally occurring more extensively in males) were seen: Increased liver weights (both sexes); hepatic nodules; hepatocellular necrosis; and peripheral compression.

Two low-dose studies by Griffin et al. (1980, 1981) also produced negative results. Male and female Sprague-Dawley rats were exposed by inhalation to 25 ppm of 2-NP for 7 hours/day, 5 days/week for 22 months. No pathological changes associated with exposure to 2-NP were seen.

Although the results of both the Lewis et al. (1979) and the Fiala et al. (1987) studies show statistically significant increases in liver carcinomas, neither study provides sufficient dose-response information to use as a basis to quantify the excess cancer risk to humans exposed to 2-NP. Both studies were terminated before the natural lifetime expectancy of the controls, so it is not possible to determine a background incidence of cancer risk. No historical information is provided on tumor incidence for these animals.

2-Nitropropane produced a high incidence of liver tumors in male rats by two routes of administration: Inhalation and ingestion. Its ability to cause mutations in *Salmonella typhimurium* further supports the premise that 2-NP is a potential human carcinogen. OSHA considered whether to perform a quantitative risk assessment on 2-NP. OSHA preliminarily concludes that the studies described above do not contain sufficient dose-response data to use as the basis for quantitative risk estimation using standardized risk assessment models. However, two studies (Fiala et al. 1987, Lewis et al. 1979) demonstrate that exposure to 2-NP, either by gavage or inhalation, produced hepatocarcinomas in rats. In addition, this substance produced positive results in two mutagenic assays (Lofroth et al. 1981, Speck et al. 1982).

OSHA is proposing an 8-hour PEL for 2-NP of 10 ppm. The Agency preliminarily concludes that a reduction in the PEL is necessary to protect exposed workers from the significant risk of cancer potentially associated with exposure to 2-NP at the current PEL. The proposed limit will substantially reduce this significant occupational risk.

STYRENE
CAS: 100–42–5; Chemical Formula: $C_6H_5CH=CH_2$
H.S. No. 1372

OSHA's current occupational limits for styrene are 100 ppm as an 8-hour TWA, 200 ppm as a ceiling limit not to be exceeded for more than 5 minutes in any 3-hour period, and 600 ppm as a peak limit. The ACGIH has recommended limits of 50 ppm as an 8-hour TWA and 100 ppm as a 15-minute STEL for styrene. NIOSH has a recommended standard for styrene of 50 ppm as a 10-hour TWA, with a 15-minute ceiling of 100 ppm.

An increased incidence of cancer has been reported among workers exposed to styrene. In addition, styrene has been demonstrated to be carcinogenic in animals. The discussion below summarizes the human and animal data and presents OSHA's quantitative assessment of the cancer risk potentially associated with occupational exposure to styrene.

McMichael et al. (1976), in a nested case-control study, identified a sample of male workers who had worked for 5 or more years at a tire manufacturing plant where exposure to styrene (and butadiene) occurred. The authors found the age-standardized relative risk for this cohort (128 workers), compared with that of the total plant population (6,678 workers), to be 6.2 for lymphatic and hematopoietic cancer, 3.9 for lymphatic leukemia, and 2.2 for stomach cancer. In a follow-up unpublished analysis (discussed in EPA 1987), the relative risk for lymphatic and hematopoietic cancer was revised to 2.4.

In a retrospective cohort mortality study, Meinhardt et al. (1982) identified five deaths from leukemia and aleukemia (1.8 expected, SMR = 278) among 600 workers in a synthetic rubber manufacturing facility. Workers at this plant had been exposed to styrene (and butadiene) for at least 6 months; the mean concentration of styrene at the plant was 0.94 ppm. Because of the concurrent exposure to butadiene reported among the study cohort, these studies can be said to support but not conclusively to prove that exposure to styrene is associated with an elevated risk in workers of hematopoietic cancer and leukemia.

In a study sponsored by the Chemical Manufacturers Association (Dow 1978, as cited in EPA 1987), male and female Sprague-Dawley rats were exposed to styrene vapor at concentrations of 600 to 1200 ppm, 6 hours per day, 5 days per week, for 18 or 20 months. The higher exposure level was reduced to 1000 ppm after the first 2 months of exposure because of excessively reduced weight in the male rats. A statistically significant increased incidence of mammary tumors was reported in low-dose female rats (7 of 87) compared with controls (1 of 85); no increase in mammary tumors was reported among high-dose female rats. The authors questioned the significance of this response, since historical control animals from the same laboratory showed a higher background incidence of mammary tumors than the controls used in this study.

An increased incidence of leukemia and lymphosarcoma was also observed in both low-dose and high-dose females; both experimental groups exhibited the same incidence (6 of 85), compared with 1 of 85 in the controls. Although the

increased incidence was not statistically significant compared with the incidence in concurrent controls, the increase was highly significant compared with historical control rates. The tumor response in male rats was confounded by excessive non-treatment-related mortality.

In a 1979 NCI study, male and female B6C3F1 mice and Fischer 344 rats were treated by gavage 5 days per week for 78 weeks (low-dose rat groups were treated for 103 weeks). The study was terminated at 91 weeks for mice and 104 to 105 weeks for rats. Dose-related increases in alveolar/bronchiolar adenomas and carcinomas were observed only in the low-dose (150 mg/kg) and high-dose (300 mg/kg) male mice; the incidence of tumors for vehicle controls, low dose, and high-dose male mice was 0/20, 6/44, and 9/43, respectively. Although the historical incidence of tumors among untreated controls was 12 percent (32/271), the historical incidence of vehicle controls was 0/40.

Quantitative Risk Assessment for Styrene. OSHA assessed the excess cancer risk associated with 45 years of occupational exposure to styrene using both the NCI (1979) male mouse lung tumor response and the Dow (1978) female rat leukemia/lymphosarcoma response. Risk estimates derived using the multistage model and the NCI data set yielded estimated excess cancer deaths of 280 or 150 per 1,000 employees exposed to 100 ppm or 50 ppm over their working lifetimes, respectively (95 percent upper-bound estimates were 460 or 220 per 1,000, respectively). However, the daily doses associated with 45 years of occupational exposure to 100 ppm or 50 ppm styrene are well above the daily dose administered to the high-dose group of mice in the NCI gavage study, which places the estimated human doses outside the linear range of the model. As a consequence, the NCI gavage study is not suited to estimating risk in the range of exposure represented by the current and proposed limits for this substance.

Because the daily human doses corresponding to 45 years of exposure to 100 ppm or 50 ppm styrene in the Dow (1978) study are well below the daily doses administered to the female rats, these data can be used as the basis for a quantitative risk assessment. The risk estimates based on this study are presented in Table C15-6.

TABLE C15-6. Multistage Model Estimates of Cancer Risk Associated With Working Lifetime Exposure to Styrene*

Exposure Level	Excess Cancer Deaths per 1,000 Workers	
	MLE	UCL
50 ppm[a]	9.5	17
100 ppm[b]	19.0	33

* Based on 1978 Dow study sponsored by the Chemical Manufacturers Association.
[a] Proposed OSHA PEL.
[b] Current OSHA PEL.
MLE = Maximum likelihood estimate of risk.
UCL = 95 percent upper confidence limit on maximum likelihood estimate of risk.

Styrene has been shown to be carcinogenic in two species of animals and by two routes of administration. Human studies also implicate styrene as a potential human carcinogen, but these studies are confounded by concurrent exposure to butadiene in the cohorts studied. However, it is noteworthy that the excess cancers that have been found in workers exposed to styrenebutadiene (hematopietic cancer and leukemia) involve the same tumor site affected in rats exposed to styrene by inhalation.

OSHA's quantitative risk assessment based on the rat inhalation study indicates that an excess of 19 cancer deaths will occur among 1,000 employees exposed to the current PEL of 100 ppm styrene for 45 years; clearly, this represents a significant risk. Reduction of the PEL to 50 ppm will reduce this significant risk substantially; the reduction from 100 to 50 ppm reduces the risk existing at the current PEL by 50 percent. Therefore, OSHA proposes to revise its existing PEL for styrene to 50 ppm as an 8-hour TWA; OSHA also proposes to supplement the TWA-PEL with a 100-ppm 15-minute STEL to ensure that workplace exposures are maintained under good industrial hygiene control. OSHA is proposing the 50-ppm limit as an interim measure. As future priorities permit, the Agency will consider additional rulemaking for this potent occupational carcinogen, at which time OSHA will investigate whether it is feasible to reduce exposures further.

o-TOLUIDINE

CAS: 95-53-4; Chemical Formula: $CH_3C_6H_4NH_2$

H.S. No. 1399

OSHA's current 8-hour TWA for o-toluidine is 5 ppm. The ACGIH identifies o-toluidine as a suspected human carcinogen and has accordingly placed it in the A2 category (ACGIH 1986, p. 586). The ACGIH has a TLV-TWA of 2 ppm, with a skin notation, for this substance. The International Agency for Research on Cancer (IARC 1982) classifies o-toluidine as a probable carcinogen (category 2A) based on sufficient evidence of its carcinogenicity

in rats and mice following oral administration (IARC 1982). IARC judged the human evidence inadequate to establish o-toluidine's carcinogenicity in human tests.

o-Toluidine is mutagenic in short-term tests, inducing sister chromatid exchanges and unscheduled DNA synthesis in mammalian cells in vitro and chromosomal anomalies in yeast. o-Toluidine was negative in the micronucleus test in mice in vivo, but induced cell transformations in the BHK (baby hamster kidney) assay. IARC considers these data to be sufficient evidence of o-toluidine's activity in short-term tests (IARC 1982).

There are a number of studies that report an excess of bladder tumors in dyestuff workers exposed to o-toluidine and other chemicals; however, there are no studies that examine a population of workers exposed to o-toluidine alone. Workers exposed to toluene, o-nitrotoluene, o-toluidine, and 4,4 methylene bis (2-methylaniline) in manufacturing were observed to have an excess of bladder tumors. However, the concurrent exposures of these workers to these other potential carcinogens make these data inappropriate for use in the quantitative assessment of o-toluidine's carcinogenic risk in human populations. A few reports of bladder tumors in persons exposed primarily to o-toluidine have been reported, but insufficient follow-up time and incomplete data have prevented the establishment of a clear quantitative association between o-toluidine exposure and cancer in humans. For this reason, IARC considers the data from human studies inadequate to establish an association between exposure to o-toluidine and cancer (IARC 1982).

o-Toluidine has been determined to be carcinogenic in rats and mice following oral administration. In rats, statistically significant increases in subcutaneous fibromas, fibrosarcomas, and cancers of the urinary bladder have been reported. Studies in mice have resulted in statistically significant increases in hemangiosarcomas and hepatocellular carcinomas.

The National Cancer Institute (NCI 1979) conducted long-term carcinogenicity studies with o-toluidine in rats and mice. Both of these studies were positive for carcinogenicity. The mouse study used groups of 50 female and 50 male B6C3F1 mice fed o-toluidine hydrochloride in the diet at levels of 1000 ppm or 3000 ppm for 102 to 103 weeks. There was no excess mortality in the tested animals. At the 3000-ppm dose level, there was a statistically significant increase in hemangiosarcomas at all sites in males and a statistically significant increase in hepatocellular carcinomas and adenomas in females.

The National Cancer Institute also conducted a 2-year feeding study with 50 male and 50 female Fischer 344 rats. There was a dose-related trend in mortality (which was not caused by cancer); all the males in the high-dose group died by 100 weeks. However, the females at both dose levels were observed to have significant increases in transitional-cell carcinomas or papillomas of the urinary bladder, and the high-dose females developed fibroadenomas of the mammary gland. The males at both dose levels showed significant increases in fibromas of the subcutaneous tissue and mesotheliomas in multiple organs (NCI 1979). The high mortality in the males complicates the interpretation of these latter findings.

Weisburger et al. (1978) reported positive findings for o-toluidine in long-term feeding studies in rats and mice. The study in rats was conducted with two groups of 25 male CD rats fed o-toluidine in the diet via one of two regimens: 8000 ppm for 3 months and then 4000 ppm for an additional 15 months; or 16,000 ppm for 3 months and then 8000 ppm for an additional 15 months. Statistically significant increases in the incidence of subcutaneous fibromas and fibrosarcomas were observed in both dose groups. In addition, there was a non-statistically significant increase in the incidence of transitional-cell carcinomas of the urinary bladder in these animals.

Weisburger et al. (1978) also reported the results of a long-term study in mice. Groups of 25 male and 25 female CD-1 mice were fed diets containing o-toluidine at two dose levels: 16,000 ppm for 3 months and then 8000 ppm for an additional 15 months; or 32,000 ppm for 3 months and then 8000 ppm for an additional 15 months. There was a statistically significant, dose-related increase in the incidences of vascular tumors (hemangiosarcomas and hemangiomas of the abdominal viscera) in both sexes of treated mice, compared with results in control mice.

Risk estimate for o-toluidine. Four of these carcinogenicity studies of o-toluidine have yielded sufficient and adequate data for quantitative risk estimation: The two NCI studies (NCI 1978) and the two Weisburger et al. (1978) studies. OSHA has used the NCI study in rats as the basis for its quantitative risk assessment because it provides the most appropriate data. Table C15-7 presents the Maximum Likelihood Estimates (MLE) of excess deaths per 1,000 employees predicted to result from exposure to o-toluidine at the current OSHA PEL of 5 ppm and at the proposed PEL of 2 ppm. These data were calculated using a multistage model, GLOBAL83.

Table C15-7 shows an excess MLE estimate of risk of 1.4 per 10,000 workers exposed over their working lifetimes at the current PEL. This risk would be reduced to 0.5 per 10,000 exposed workers after promulgation of the proposed limit of 2 ppm. This level of risk is lower than the levels OSHA has regulated for some carcinogens, such as ethylene oxide, arsenic, and benzene. However, this risk is approximately the same as that associated with exposure to formaldehyde; in the case of formaldehyde, OSHA considered a reduction in exposure of approximately this amount to be appropriate (see the discussion at 52 FR 46211-37, December 4, 1987). Based on that analysis, OSHA preliminarily concludes that the significant risk of carcinogenicity to which workers are currently exposed at the existing PEL would be substantially reduced by promulgation of the proposed PEL.

TABLE C15-7. Multistage Model Estimates of Cancer Risk Associated with Working Lifetime Exposure to o-Toluidine

Exposure Level	Excess Cancer Deaths per 1,000 Workers	
	MLE	UCL
5 ppm[a]	0.137	1.6
2 ppm[b]	0.055	0.64

[a] Current OSHA PEL.
[b] Proposed OSHA PEL.
MLE = Maximum likelihood estimate of risk.
UCL = Upper bound (95 percent) confidence limit on maximum likelihood estimate of risk.

p-TOLUIDINE
CAS: 106-49-0; Chemical Formula: $CH_3C_6H_4NH_2$
H.S. No. 1400

OSHA has no current PEL for p-toluidine. The ACGIH considers this substance a suspected human carcinogen and has given it a classification of A2 (ACGIH 1986) and a TLV-TWA of 2 ppm. There is sufficient evidence for the carcinogenesis of p-toluidine in experimental animals; there is no human epidemiological evidence.

One sludy investigates the carcinogenic potential of lifetime exposure to p-toluidine in experimental animals (Weisburger et al. 1976). Male and female mice were exposed to p-toluidine in the diet for a total of 18 months. During the first 6 months of the experiment, mice were exposed to 1000 and 2000 mg p-toluidine/kg diet. As a result of the weight loss that occurred in mice exposed to the 2000 mg/kg diet dose, the concentrations of p-toluidine were reduced to 500 and 1000 mg/kg diet during the last 12 months of exposure. The rate of food consumption by the animals was not reported and was assumed to be 3 g/day. Thus, the average doses of p-toluidine received during the 18-month exposure were calculated to be 80 and 160 mg/kg body weight per day (Weisburger et al. 1976).

For both the low and high dietary doses of p-toluidine, a significant increase in the incidence of hepatomas was observed. The incidence of tumors in the control, 80, and 160 mg/kg/day groups were 3/38, 10/38, and 12/35, respectively. The same study (Weisburger et al. 1976) showed negative results in male rats exposed to two doses of p-toluidine in the diet for 18 months (1000 and 2000 mg/kg diet).

Risk estimate for p-toluidine. To assess the quantitative risk of p-toluidine's carcinogenicity, OSHA used the Weisburger et al. (1976) data which, despite some limitations, e.g., changes in dose levels during the experiment and the absence of data concerning the amount of food animals consumed during the exposure period, were considered adequate for risk assessment purposes.

The maximum likelihood estimates (MLE) of excess cancers per 1,000 workers over an occupational lifetime and the 95 percent upper-bound estimates were obtained by using a linearized multistage model (GLOBAL83). These values are summarized in Table C15-8. This table shows the number of cancer deaths potentially associated with working lifetime exposure to 20, 5, or 2 ppm p-toluidine.

TABLE C15-8. Multistage Model Estimates of Cancer Risk Associated with Working Lifetime Exposure to p-Toluidine

Exposure Level	Excess Cancer Deaths per 1,000 Workers	
	MLE	UCL
2 ppm[a]	12	19
5 ppm	29	46
20 ppm	112	172

[a] Proposed OSHA PEL.
MLE = Maximum likelihood estimate of risk.
UCL = Upper bound (95 percent) confidence limit on maximum likelihood estimate of risk.

OSHA preliminarily concludes, as Table C15–8 shows, that workers exposed to p-toluidine, which is currently not regulated by OSHA, are at significant risk of developing hepatomas. For example, the MLE at 20 ppm is 112 excess cancer deaths per 1,000 workers exposed over a working lifetime. Promulgating the proposed PEL of 2 ppm will substantially reduce this significant risk. According to this scenario, a 90-percent reduction in excess cancer deaths would be achieved by establishing the 2-ppm limit. The risks existing at the present uncontrolled level are clearly significant and have been determined to be so in several previous OSHA rulemakings. OSHA is proposing a 2-ppm limit as an interim measure. As future priorities permit, the Agency will consider additional rulemaking to investigate whether it is feasible and appropriate to reduce exposures further.

VINYL BROMIDE
CAS: 593–60–2: Chemical Formula: C_2H_3Br
H.S. 1425

OSHA has no current PEL for vinyl bromide. The ACGIH has established an 8-hour TWA of 5 ppm for this substance; NIOSH has no REL for vinyl bromide. The ACGIH places vinyl bromide on its A2 list of industrial substances suspected of having carcinogenic potential in humans. Vinyl bromide is used as an intermediate in organic synthesis and in the manufacture of polymers, copolymers, and flame retardants. Its principal use is as a flame retardant.

Henschler and Hoos (1982) believe that vinyl bromide undergoes the same mechanism of biotransformation as its structural analog, vinyl chloride, a recognized human carcinogen that has been regulated by OSHA in a section 6(b) rulemaking. The microsomal oxidation of vinyl bromide leads to epoxide formation, which results, in turn, in the formation of a reactive intermediate. This intermediate has the potential to form covalent bonds with DNA to produce a mutagenic response. Vinyl bromide has been reported to be mutagenic in *Salmonella typhimurium* and *tradescantia* (IARC 1979; NIOSH/OSHA 1978).

No epidemiological studies have been conducted on populations exposed to vinyl bromide. Benya et al. (1982) reported a positive carcinogenic response in an inhalation study of rats exposed to vinyl bromide vapor; this study is important because inhalation is a major mode of occupational exposure. The results of the Van Duuren (1977) study were equivocal (described below), in that female Swiss albino mice were exposed dermally or by subcutaneous injection either to vinyl bromide in acetone or to polymerized vinyl bromide in an aqueous latex solution.

Benya et al. (1982) exposed male and female Sprague-Dawley rats to 0, 9.7, 52, 247, or 1,235 ppm vinyl bromide by inhalation for 6 hours daily, 5 days per week, for 2 years. The incidence of angiosarcomas, primarily of the liver, was found to be statistically significant in all dose groups tested except controls. The combined incidences of hepatic angiosarcomas in the treated male and female rates were 1/288, 17/240, 86/240, 122/240, and 84/240 for their respective dose levels. One control female rat developed an hepatic angiosarcoma. Table C15–9 summarizes the incidence of angiosarcoma in control and treated rats.

TABLE C15 9. Incidence of Angiosarcomas in Control and Vinyl Bromide Exposed Rats

Group	Exposure level (ppm)	Males			Females		
		No. of animals	No. with angiosarcoma	p	No. of animals	No. with angiosarcoma	p
1	Control	144	0	—	144	1	-
2	10	120	7	<0.025	120	10	<0.01
3	50	120	36	<0.001	120	50	<0.001
4	250	120	61	<0.001	120	61	<0.001
5	1250	120	43	<0.001	120	41	<0.001

Source: Benya et al. (1982)

Van Duuren injected a group of female ICR/Ha Swiss mice once weekly for 48 weeks with 0.05 ml of commercial polymerized vinyl bromide aqueous latex suspension; the animals were observed for 420 days. Nineteen of the 30 mice developed sarcomas at the site of injection. Animals in a positive control group that had been injected with b-propriolactone (0.3 mg/.05 ml trioctanoin) developed 18 sarcomas and 3 squamous cell carcinomas (in 30 mice). No tumors developed in untreated controls or in controls injected with trioctanoin, an organic solvent, alone (Van Duuren 1977).

In another injection study by the same author, a group of female IRC/Ha Swiss mice were treated with 25 mg vinyl bromide per animal in 0.05 ml trioctanoin once weekly for 48 weeks. The mice were observed for 420 days. One control group was given a weekly injection of trioctanoin alone and the other control group was untreated. No local tumors were seen in any of the test groups, although pathological examination of the animals appears to have been incomplete (Van Duuren 1977).

Application of vinyl bromide to the skin of female ICR/Ha Swiss mice at a dose of 15 mg per animal administered in 0.1 ml of acetone 3 times weekly for 420 days resulted in no tumors. When this solution was applied once and was followed by an application of phorbol myristyl acetate (PMA) 3 times weekly, 1 of 30 mice developed a skin papilloma at 412 days, one control treated with PMA developed a tumor after 44 days, and no untreated controls developed tumors (Van Duuren 1977).

In another dermal study, a dose of 0.1 ml of polymerized vinyl bromide in an aqueous latex suspension was applied 3 times weekly to the skin of female ICR/Ha Swiss mice for 420 days. No skin tumors developed, when this solution was applied once, followed by an application of PMA 3 times weekly, 1 of 30 mice developed a skin tumor at 175 days. No untreated controls developed skin tumors (Van Duuren 1977).

Risk estimate for vinyl bromide. The Benya et al. (1982) study was a well-designed and conducted study that yielded sufficient information for quantitative risk estimation. The route of administration used in the study, inhalation, is directly applicable to occupational exposure, and the incidence of hepatic angiosarcoma was significant. Angiosarcoma is a rare and malignant neoplasm that has a very low background incidence in animals and humans. Therefore, its appearance in the exposed rats supports the premise that vinyl bromide is potentially carcinogenic in humans. Also, it is the same tumor that is associated with the exposure of workers and animals to vinyl chloride, a recognized human carcinogen and a compound whose structure is similar to that of vinyl bromide.

To estimate excess cancer risk over background incidence for a chemical, experimental data (experimental doses and corresponding responses) are used to define various parameters of an assumed response model. At low doses, the slope of this dose-response curve is referred to as q_1. The 95 percent upper-bound confidence limit for this slope is referred to as q_1^* or the chemical's potency. q_1 and q_1^* are then used to determine the respective maximum likelihood estimate (MLE) of risk and the 95 percent upper-bound confidence limit (UCL) on risk associated with a given lifetime occupational exposure. A non-threshold, linearized multistage model (GLOBAL83) was chosen to estimate the risk potentially associated with exposure to vinyl bromide because the scientific rationale for this model is biologically the most plausible. Additionally, the choice of a non-threshold model is consistent with current methodologies when positive mutagenicity data are available (EPA 1984).

To choose the appropriate data set to use with the model, the most sensitive species and sex are chosen. In the case of vinyl bromide, both male and female

rats responded equally, and data from the two groups were therefore combined by calculating the geometric means of the risk estimates derived from the male and female response data (Anderson 1983). The high-dose data for each test group were dropped, since their inclusion makes the dose-response curve non-monotonic and precludes proper fitting of the linearized multistage risk model (EPA 1984).

Since cancer risk modeling assumes lifetime exposure, adjustments were made to fit the animal data to this criterion. The adjustments made for the data in Benya et al. (1982) were: Multiplying dose by 5/7 to adjust for days of exposure per week and by 6/24 to adjust for hours of exposure per day. These adjusted doses were then changed to human equivalent doses.

Three hypothetical occupational exposure limits, 5 ppm, 20 ppm, and 250 ppm, were used to calculate the maximum likelihood estimates of risk of developing angiosarcoma of the liver. Five ppm has been the ACGIH limit since 1978. Twenty ppm was chosen as an intermediate exposure level, and 250 ppm was the exposure level before the ACGIH reduced it in 1978. These occupational dose levels were also adjusted for lifetime exposure. The adjustments made were: multiplying dose by 5/7 to adjust for days worked per week, by 50/52 to adjust for vacation time, by 8/24 to adjust for hours of exposure per day, and by 45/70 to adjust for work years per lifetime.

Because inhalation is the primary route of exposure to vinyl bromide in occupational settings, the occupational dose was calculated assuming that air intake in humans is 20 m^3 per 24-hour day (Anderson 1983). The fraction of vinyl bromide absorbed was assumed to be 100 percent, because no absorption rate data were available for vinyl bromide. Because the log p (lipid solubility) value for vinyl bromide (1.52) is similar to that of vinyl chloride (1.38), OSHA assumed that the absorption rates of these two compounds would also be similar. The absorption rate for vinyl chloride used in risk estimations is assumed to be 100 percent (IRIS 1988).

The MLE shown in Table C15–10 for an occupational exposure to 250 ppm is 0.87, or 87 percent. According to the linearized multistage risk model, 870 of 1,000 workers exposed over their working lifetimes to vinyl bromide at 250 ppm are at risk of developing angiosarcoma. The MLE for an occupational exposure to 5 ppm of vinyl bromide is 0.04; this indicates that, at the proposed PEL, 40 workers per 1,000 exposed to this substance over their occupational lifetimes are at risk of developing angiosarcoma.

TABLE C15-10. Multistage Model Estimates of Cancer Risk Associated with Lifetime Exposure to Vinyl Bromide

Exposure Level	Excess Cancer Deaths per 1,000 Workers	
	MLE[a]	UCL[a]
5 ppm[b]	40	48
20 ppm[c]	155	180
250 ppm[d]	870	930

[a] Geometric mean of male and female rats.
[b] Proposed OSHA PEL.
[c] Intermediate exposure level.
[d] ACGIH limit before 1978.
MLE = Maximum likelihood estimate of risk.
UCL = 95 percent upper confidence limit on maximum likelihood estimate on risk.

Table C15–10 shows that workers exposed to this substance, which is currently not regulated by OSHA, are clearly at significant risk of developing hepatic angiosarcomas, the same rare type of tumor associated with exposure to vinyl chloride, a structurally similar substance. Promulgating the proposed PEL of 5 ppm will not eliminate this significant risk, because, as Table C15–10 shows, the upper-bound estimate of residual risk at 5 ppm is 48 excess deaths per 1,000 exposed workers. Thus, residual risk at 5 ppm is clearly significant. OSHA is proposing the 5 ppm limit as an interim measure; as future priorities permit, the Agency will consider additional rulemaking for this potent occupational carcinogen. At the present time. OSHA preliminarily concludes that establishing a PEL of 5 ppm will substantially reduce the significant risk potentially associated with exposure at the uncontrolled levels possible in the absence of an OSHA limit for this substance.

VINYL CYCLOHEXENE DIOXIDE
CAS: 106–87–6; Chemical Formula: $C_8H_{12}O_2$
H.S. No. 1426

OSHA has no PEL for vinyl cyclohexene dioxide (VCD), and NIOSH has no REL for this substance. The ACGIH classifies VCD as a suspected

human carcinogen (A2) and recommends a TLV–TWA of 10 ppm for it. Vinyl cyclohexene dioxide is a colorless liquid used as a chemical intermediate and as a monomer in the manufacture of polyglycols containing unreacted epoxy groups (Hine et al. 1981). It is also used as a reactive diluent for other diepoxides and certain epoxy resins (IARC 1976).

Turchi et al. (1981) assayed the mutagenicity of VCD and several other epoxides using the TA100 strain of *S. typhimurium* and V79 Chinese hamster cells; these authors also investigated the alkylating properties of these chemicals. VCD tested positively in the *S. typhimurium* test (point mutation) and in the V79 Chinese hamster cell test (both point mutation and chromosome aberration) and had an intermediate alkylating capacity relative to other epoxide compounds tested.

There are no data concerning the adverse health effects of VCD in humans. There are no reports as a result of industrial experience that reveal carcinogenic effects in workers caused by VCD exposure (ACGIH 1986).

Four studies have reported the development of skin tumors in mice exposed dermally to VCD (Hendry et al. 1951; Kotkin and Falk 1963; Weil et al. 1963; and Van Duuren et al. 1963). The study of Van Duuren et al. (1963) included controls and is thus particularly well suited for an evaluation of VCD's carcinogenic potential.

These authors painted 30 male Swiss ICR/Ha mice with 0.1 ml of a 10-percent solution of VCD in benzene three times per week (approximately 100 mg of solution per application). Two negative controls were used; one set of 150 mice was treated with benzene alone and another set of 207 mice was not treated with anything. Fourteen of the 30 VCD-treated mice developed skin tumors after an undefined length of time (mean survival time was 326 days). The incidences of skin tumors in the controls were 11/150 and 13/207 for the benzene-treated and untreated mice, respectively. The incidence of skin tumors in the VCD-treated mice was significantly greater than the incidence observed in either of the controls (Van Duuren et al. 1981).

The study of Van Duuren et al. (1963) demonstrates the carcinogenicity of VCD in experimental animals. OSHA considered the possibility of conducting a quantitative risk assessment VCD, and the Agency preliminarily concluded that the dose-response data in this study are unsuitable for quantitative risk assessment purposes because the VCD was administered in a solution of benzene, which is itself regulated as a carcinogen and classified as such by several authorities (IARC, NTP, NIOSH, and ACGIH). Even though the Van Duuren et al. study included a control for the independent carcinogenic effects of benzene, the possibility of a synergistic or additive effect of benzene on VCD cannot be completely ruled out.

Vinyl cyclohexene dioxide has been shown to be carcinogenic by dermal application in mice, and four studies have confirmed these effects. Based on these animal studies showing VCD's carcinogenicity, OSHA preliminarily concludes that exposed employees are at significant risk of cancer potentially associated with exposure to VCD at the uncontrolled levels permitted in the absence of any OSHA limit. The Agency preliminarily concludes that promulgation of a 10-ppm 8-hour-TWA PEL will substantially reduce this significant risk.

Preliminary Conclusions for this Group of Substances

The Supreme Court in *I.U.D.* v. *A.P.I.* (supra, the *Benzene* decision) gave OSHA directions as to its decisional process, and of course that case involved a carcinogen. OSHA is following the Supreme Court's guidance within the context of this present broader rulemaking. In the current rulemaking, OSHA has considered or performed preliminary perform risk assessments for each of the 17 chemicals discussed in this section. The risk assessments follow the approach OSHA has used in prior rulemakings for carcinogens, a process that has repeatedly been upheld by the courts. The risk assessment review process has necessarily been more limited for the hundreds of substances being regulated today than is the case for single-substance rulemakings, and OSHA requests comment on the approach it has taken in the present rulemaking.

OSHA is conducting its significant risk analyses utilizing the principles suggested by the Supreme Court and adopted in its carcinogen rulemakings subsequent to *I.U.D.* v. *A.P.I.* OSHA is proposing to lower its existing exposure limits based on these analyses when significant risk is indicated at the existing PEL. OSHA is preliminarily proposing either the ACGIH or NIOSH numerical level, based on the results of these significant risk analyses.

In some cases (all of the risk assessments have not yet been finalized) it is possible for OSHA to conclude that not enough data are available to quantify risks at the level of detail the Agency has formerly used. In those cases, OSHA will preliminarily decide whether a lower limit is justified; without this latitude, the Agency would indeed be in the "mathematical straitjacket" alluded to by the Court in the *Benzene* decision. In other circumstances, OSHA may decide that not enough evidence is available to propose a lower limit.

OSHA must also demonstrate the feasibility of the level set. In some cases, OSHA believes it has sufficient preliminary data on the feasibility of the proposed limits for these substances. (Of course, all data on feasibility are welcome.) In some cases further analysis might indicate that neither the ACGIH nor the NIOSH-recommended level is the lowest feasible level and that significant risk remains below either level. However, this rulemaking would be inordinately delayed by the amount of effort necessary to make feasibility and significant risk determinations for a greater range of levels than those already established by the ACGIH and NIOSH. In such circumstances, the level proposed here should be considered to be an interim level. As priorities and resources become available, a second-stage rulemaking will be considered to refine these exposure levels, and the interim level should thus be considered a "backstop."

For some substances in this rulemaking other than the 17 discussed in this section, there may be evidence in the literature of carcinogenicity, even though the ACGIH has established limits for these based on other-than-cancer effects. For these few substances, OSHA has not initiated a risk assessment, because this additional effort would have delayed this rulemaking unreasonably.

In sum, where OSHA preliminarily found that there was sufficient evidence of potential carcinogenicity to meet the Agency's legal requirements, the Agency has proposed a level based on the risk of cancer. Where there was not sufficient evidence readily available, OSHA proposed a level based on these substances' non-carcinogenic effects. At a later stage, depending on priorities and resources, OSHA will further review the data to determine whether a second-stage rulemaking based on carcinogenicity is appropriate for these few chemicals.

Overall, OSHA believes its analyses of proposed limits for carcinogenic chemicals meet the Agency's legal requirements. Accordingly, OSHA preliminarily concludes that these limits will lead to substantial reductions in the significant risk currently confronting workers exposed to these substances.

16. Substances for Which Current ACGIH TLVs Are Less Stringent Than Existing OSHA PELS

Introduction

There are 13 substances for which the ACGIH has increased its recommended TLVs since the time that OSHA adopted the 1968 TLVs under the authority of section 6(a) of the Act. These substances are listed in Table C16–1, along with their current OSHA PELs, ACGIH TLVs. NIOSH RELs, CAS numbers, and HS numbers. Evaluating the protectiveness and appropriateness of exposure limits that are less stringent than the corresponding limits on OSHA's Z tables represents a special case in this rulemaking. OSHA has previously stated (see 50 FR 51120, December 13, 1985) the principle to be followed before the Agency raises an exposure limit. This issue is discussed below.

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TABLE C16-1. Substances for Which the ACGIH's Limits are Higher Than the Current PELs

H.S. Number/ Chemical Name	CAS No.	CURRENT PEL*	ACGIH TLV**	NIOSH REL***
1101 Copper (fume)	7440-50-8	0.1 mg/m^3 TWA	0.2 mg/m^3 TWA	--
1126 1,1-Dichloroethane	75-34-3	100 ppm TWA	200 ppm TWA 250 ppm STEL	--
1179 Fluorine	7782-41-4	0.1 ppm TWA	1 ppm TWA 2 ppm STEL	--
1197 Hexachloroethane	67-72-1	1 ppm TWA Skin	10 ppm TWA	Lowest feasible level
1284 Nickel carbonyl	13463-39-3	0.001 ppm TWA	0.05 ppm TWA	1 ppb TWA
1347 Rhodium (metal fume and insoluble salts)	7440-16-6	0.1 mg/m^3 TWA	1 mg/m^3 TWA	--
1348 Rhodium (soluble salts)	7440-16-6	0.001 mg/m^3 TWA	0.01 mg/m^3 TWA	--
1352 Silica, Amorphous-Diatomaceous Earth	68855-54-9	20 mppcf TWA (6 mg/m^3)	10 mg/m^3 TWA	--
1353 Silica, Amorphous-Precipitated and Gel	None	20 mppcf TWA (6 mg/m^3)	10 mg/m^3 TWA	--

TABLE C16-1. Substances for Which the ACGIH's Limits are Higher Than the Current PELs (continued)

H.S. Number/ Chemical Name	CAS No.	CURRENT PEL*	ACGIH TLV**	NIOSH REL***
1362 Silver (metal dust and fume)	7440-22-4	0.01 mg/m^3 TWA	0.1 mg/m^3 TWA	--
1386 Tetraethyl lead	78-00-2	0.075 mg/m^3 TWA Skin	0.1 mg/m^3 TWA Skin	--
1388 Tetramethyl lead	75-74-1	0.075 mg/m^3 TWA Skin	0.15 mg/m^3 TWA Skin	--
1419 Uranium (soluble compounds)	7440-61-1	0.05 mg/m^3 TWA	0.2 $mg.m^3$ TWA 0.6 mg/m^3 STEL	--

* OSHA's TWA limits are for 8-hour exposures; its STELs are for the durations specified; and its ceilings are peaks not to be exceeded for any period of time.

** The ACGIH TWA-TLV is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

*** NIOSH TWA limits are for 10-hour exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

BILLING CODE 4510-26-C

In 1978, OSHA issued a cotton dust standard; this standard did not go into effect in any of the nontextile industries. However, although the new standard's PEL for cotton dust did not apply in these segments, the Z–1 limit for cotton dust continued to apply to them. In 1983, OSHA determined that it would better effectuate the purposes of the Act to exclude the knitting and other nontextile industries from coverage by the Z table limit for cotton dust. In revoking the Z table limit, OSHA stated:

> When it [the Agency] proposes to eliminate a class [of operations or industry sectors] from either a 6(a) or 6(b) standard on health grounds, the evidence must affirmatively indicate that significant risk is unlikely to exist for that class at exposures likely to exist after the standard has been eliminated * * *. OSHA must be able to support with substantial evidence any change it is propounding [50 FR 51120 et seq., Dec. 13, 1985].

Accordingly, the Agency must be able to show that exposed workers will not be placed at increased risk for the health effects at issue even after the limit in question has been raised or revoked. In conformance with this interpretation, OSHA has carefully examined the bases underlying the adoption of increased exposure limits by the ACGIH. After reviewing the available data for these substances, OSHA has made a preliminary determination that adequate evidence exists to increase the permissible exposure limits for only one of these substances (fluorine), and the Agency is now proposing to raise the limit for this chemical to 1 ppm as an 8-hour TWA, with a STEL of 2 ppm to provide additional protection against excessive short-term exposures. For the remaining 11 substances in this group, OSHA believes that the available toxicological data are insufficient to meet the increased burden of proof appropriate when the raising of an exposure limit is under consideration. For these substances, OSHA is not proposing to revise the current PELs at this time, but requests comments and data on the issue.

The following discussion summarizes OSHA's preliminary analyses and findings for each of the 11 substances in this group.

COPPER (FUME)
CAS: 7440–50–8; Chemical Formula: Cu
H.S. No. 1101

The current OSHA limit for copper fume is 0.1 mg/m³ as an 8-hour TWA. Since OSHA adopted this limit in 1971, the ACGIH has increased the recommended TLV to 0.2 mg/m³ as an 8-hour TWA. The ACGIH's previously recommended TLV of 0.1 mg/m³ was based on personal communications (Newton Whitman 1957; 1962) that reported that the taste perception of welders was altered when they were exposed to copper fume at levels ranging from 1 to 3 mg/m³ for short periods but that exposure to 0.02 to 0.4 mg/m³ did not cause such complaints (ACGIH 1966). At the time, the ACGIH judged the 0.1-mg/m³ TLV to be "sufficiently low to provide freedom from irritation from the fume by a reasonable margin" (ACGIH 1966).

In 1972, the ACGIH received a personal communication from a member of the U.K. Industrial Hygiene Unit, Her Majesty's Factory Inspectorate (Luxon 1972) reporting that employees exposed to copper fume at levels up to 0.4 mg/m³ during welding and copper metal refining operations experienced no ill effects from exposure. Based on this additional evidence, the ACGIH increased its TLV for copper fume to 0.2 mg/m³ in 1975.

OSHA preliminarily judges that the evidence cited by the ACGIH (1986) in support of the increase in its TLV for copper fume is not sufficient to provide a basis for OSHA to propose raising the limit for this substance. The basis for this finding is that the ACGIH's action was based largely on a personal communication, making it impossible for the Agency to evaluate the evidence appropriately.

1,1-DICHLOROETHANE
CAS: 75–34–3; Chemical Formula: CH_2ClCH_2Cl
H.S. No. 1126

The current OSHA limit for 1,1-dichloroethane, which is a hepatotoxin, is 100 ppm TWA. The ACGIH recommended TLV is a 200-ppm TWA with a 250-ppm STEL; NIOSH has no REL for this substance. The previous ACGIH TLV of 100 ppm was based on the observation that 1,1-dichloroethane has an acute toxicity approximately half that of carbon tetrachloride and a chronic toxicity somewhat less than that of carbon tetrachloride (for which a TLV of 10 ppm had been set). In 1973, the ACGIH adopted the higher 200-ppm TLV based on unpublished data from the Dow Chemical Company showing that rats, rabbits, guinea pigs, and dogs exhibited no gross or microscopic organ pathology after exposure to 500 or 1000 ppm of 1,1-dichloroethane for 6 months. The ACGIH cited no human data in support of raising the TLV.

Because no human toxicity data are available for 1,1-dichloroethane and because the Dow data are unpublished and thus not available for scrutiny, OSHA preliminarily concludes that the evidence for this substance is insufficient to warrant increasing the PEL at this time.

FLUORINE
CAS: 7782–41–4; Chemical Formula: F
H.S. No. 1179

OSHA's current PEL for fluorine is 0.1 ppm; NIOSH has no REL for fluorine. In 1973, the ACGIH revised its TLV to 1 ppm and, subsequent to that change, adopted a TLV–STEL of 2 ppm. The ACGIH's previous 0.1-ppm TLV, which was adopted by OSHA in 1971, was based on a 30-day inhalation study in rats and dogs (Stokinger 1949) in which no consistent pulmonary, renal, or blood effects were observed following exposure to 0.5 ppm. The ACGIH believed that a TLV of 0.1 ppm would "provide a working environment of probable safety from the effects of F_2" (ACGIH 1966). Subsequently, the ACGIH reviewed a 7-year study (Lyon et al. 1962) of 61 workers exposed to fluorine concentrations "far in excess of 0.1 ppm" (ACGIH 1986), which reported a lack of significant medical findings. This evidence, along with more recent animal evidence (Keplinger and Suissa 1968) suggesting that animals were not as sensitive to fluorine as was reported by Stokinger (1949), led the ACGIH to increase its TLV to 1 ppm. The STEL of 2 ppm was supported by a study (Ricca 1970) in which human volunteers repeatedly exposed to 10 ppm reported only slight irritation.

OSHA believes that the human and animal evidence is adequate to support a proposed increase in the 8-hour TWA for this substance from 0.1 ppm to 1 ppm. OSHA is proposing at this time to revise the PEL for fluorine to 1 ppm as an 8-hour TWA and 2 ppm as a 15-minute STEL.

HEXACHLOROETHANE
CAS: 67–72–1; Chemical Formula: CCl_3CCl_3
H.S. No. 1197

OSHA's current PEL for hexachloroethane is a 1-ppm TWA, with a skin notation, which was adopted from the 1968 ACGIH TLV. The NIOSH REL for this substance is the lowest feasible level, based on hexachloroethane's potential carcinogenicity. The basis for the 1-ppm TLV was to prevent the "serious injury potential to several organ systems" shown by animal studies (ACGIH 1986). Subsequently, the ACGIH revised its TLV upward to 10 ppm based, in part, on a study by Weeks et al. (1979) that reported no adverse effects among several animal species exposed daily to 15- or 48-ppm concentrations of hexachloroethane. The ACGIH also cited an NCI study (NCI 1978b) in which

"extremely heavy dosages * * * administered continuously for a long period of time" resulted in the development of hepatocellular tumors in mice but not in rats. The 10-ppm TLV was further supported by a personal communication of a TLV Committee member who reported that no ill effects occurred among workers "who handled the material with few precautions" during World War II (ACGIH 1986). No exposure data were supplied to support this personal communication.

In 1978, NIOSH reviewed the results of an NCI bioassay in which hexachloroethane was administered by gavage to mice and rats. Both male and female mice exhibited an excess incidence of hepatocellular carcinoma, but rats did not. NCI concluded that early mortality may have obscured detection of a carcinogenic effect in rats. Toxic kidney damage was also found in mice and rats treated with hexachloroethane. Based on this evidence, NIOSH (1978n) has recommended that exposure to hexachloroethane be maintained at the lowest detectable level.

OSHA does not believe that the evidence relied on by the ACGIH is adequate to support raising the PEL at this time. The human evidence cited by the ACGIH is anecdotal and lacks the exposure data necessary to permit OSHA to assess whether significant risk is absent (and likely to remain so) at the 10-ppm exposure level. In addition, OSHA is concerned, as is NIOSH, with the development of tumors in hexachloroethane-exposed mice demonstrated in the NCI study. OSHA therefore retains its PEL of 1 ppm TWA, with a skin notation, and preliminarily concludes that increasing the PEL for hexachloroethane would increase the risk of cancer potentially associated with exposure to this substance.

NICKEL CARBONYL
CAS: 13463–39–3; Chemical Formula: $Ni(CO)_4$
H.S. No. 1284

The current OSHA PEL and the NIOSH recommended limit for nickel carbonyl is 0.001 ppm TWA. In 1976, the ACGIH increased its TLV for nickel carbonyl from 0.001 to 0.05 ppm. The ACGIH's former 0.001-ppm TLV was based primarily on the reported association of nasal and lung cancer among workers exposed to nickel carbonyl during work in nickel refinery operations. In addition, the ACGIH cited evidence (Sunderman et al. 1959) that rats exposed to nickel carbonyl developed lung tumors that metastasized to the kidney. At the time, the ACGIH (1966) noted that these tumors were not of a type generally associated with exposure to environmental agents.

In its 1976 documentation for the 0.05-ppm TLV for nickel carbonyl, the ACGIH cited the work of Doll et al. (1970), who evaluated the exposures of nickel refinery workers in whom cancers had been found. Doll found that there had been no exposures to nickel carbonyl in the facility, and this finding led the ACGIH to conclude that nickel carbonyl was not the causative agent of the cancers reported among the refinery workers in the earlier studies it had relied on to set the 0.001-ppm TLV. A report that no excess nasal or lung tumors had occurred among workers exposed over a 50-year period in a nickel refinery in Wales (Renzoni, personal communication, 1975) appeared to the ACGIH to corroborate Doll's results. The ACGIH concluded that the TLV for nickel carbonyl should be raised based on the acute, systemic effects of this substance and that carcinogenicity was not an appropriate basis for limit-setting (ACGIH 1976). In the 1986 documentation for the 0.05-ppm TLV for nickel carbonyl, the ACGIH concluded that "although the evidence that nickel carbonyl is carcinogenic to humans is inconclusive, this recommended TLV (i.e., one set at 0.05 ppm) is also adequate to minimize any potential carcinogenic effects" (ACGIH 1986).

OSHA finds the evidence discussed by the ACGIH insufficient to warrant an increase in the limit since some of the evidence is in the form of a personal communication, particularly since NIOSH has concluded that occupational exposures should be maintained at the lowest detectable level because of nickel carbonyl's potential carcinogenicity. OSHA therefore retains its existing PEL for nickel carbonyl of 0.001 ppm TWA.

RHODIUM COMPOUNDS (METAL FUME: SOLUBLE AND INSOLUBLE SALTS)
CAS: 7440–16–6; Chemical Formula: Rh
H.S. No. 1347; 1348

The current OSHA PEL for rhodium metal fume and insoluble salts is 0.1 mg/m^3; the current PEL for soluble rhodium compounds is 0.001 mg/m^3. The ACGIH recommends a 1-mg/m^3 TLV for rhodium metal and insoluble salts and a 0.01 mg/m^3 TLV for soluble rhodium salts. The current OSHA PELs for rhodium compounds (i.e., the 1968 ACGIH TLVs) were based on the then-existing TLVs for platinum because of concern that exposure to rhodium might be associated with respiratory sensitization effects. This concern was prevalent because rhodium belongs to the platinum family of metals and because the toxicologic data on rhodium that were available were "meager" (ACGIH 1966).

The ACGIH's decision to increase the TLVs for rhodium compounds was based primarily on a personal communication to the TLV Committee (Johnson 1981). This communication indicated that, in a major precious metals refinery, "procedures which were abandoned for the refining of platinum because of cases of sensitization have been carried out for a year with analogous rhodium compounds without any problems" (ACGIH 1986). In addition, the ACGIH noted that none of the substances in the platinum group was known to produce respiratory effects similar to those of platinum. The ACGIH reported that rhodium exhibited "slight" carcinogenic activity in mice (ACGIH 1986). After considering all of this evidence, the ACGIH judged the previous TLVs to be inappropriate and increased them tenfold.

OSHA preliminarily concludes that the evidence adduced by the ACGIH is not sufficient to meet the standard of proof the Agency must achieve before it can raise an exposure limit. This conclusion is based on the fact that the ACGIH relied heavily on a personal communication when making its decision, and no exposure or other data are available to support the ACGIH's action. Thus OSHA is unable adequately to evaluate the toxicologic evidence pertaining to the rhodium compounds and retains the existing PEL's for rhodium metal fume and insoluble salts (0.1 mg/m^3 TWA) and rhodium soluble salts (0.001 mg/m^3 TWA).

SILICA, AMORPHOUS—DIATOMACEOUS EARTH
CAS: 68855–54–9; Chemical Formula: SiO_2
H.S. No. 1352

OSHA's current limit for amorphous silica is 20 mppcf, equivalent to 6 mg/m^3 (ACGIH 1984), as total dust. The ACGIH has established a limit for this dust, as total dust, of 10 mg/m^2 8-hour TLV–TWA). Amorphous silica (diatomaceous earth) is composed of the skeletons of prehistoric plants known as diatoms. These skeletons are largely non-crystalline, although diatomaceous earth can contain varying amounts of crystalline quartz, which has led, in the opinion of the ACGIH (1986, p. 520) to conflicting results in studies of the pulmonary effects of exposure to this colorless to gray, odorless powder.

Cooper and Cralley (1958) reported "doubtful" linear-nodular changes in the lungs of workers exposed only to

amorphous (noncrystalline) silica for 5 years or more. Other studies (Vigliani and Mottura 1948; 1948; Gardner 1942) also found mild silicosis only or no evidence of serious lung pathology in diatomite workers. Kovalevich (1959) reported silicosis in diatomite workers. However, intratracheal instillation of diatomaceous earth dust in animals showed evidence of fibrosis (Gardner 1942) and silicosis (Kovalevich 1959), although another study (Tebbens and Beard 1957) exposed guinea pigs to this substance at an average concentration of 60 mg/m³ for 37–50 weeks and found both gross and microscopic changes in the lungs but no fibrosis.

In setting its limit for diatomaceous earth, the ACGIH (1986, p. 520) assumed that this substance itself is either "weakly fibrogenic or nonfibrogenic," and thus that those studies discussed above that report adverse pulmonary effects actually involved exposure to diatomaceous earth having an unmeasured but significant crystalline quartz content. Based on this reasoning, the ACGIH considers amorphous silica-diatomaceous earth to be a nuisance dust. However, OSHA does not consider the evidence for diatomaceous earth's lack of fibrogenicity sufficent to demonstrate the absence of significant risk at the ACGIH's revised TLV.

OSHA is proposing to retain an 8-hour TWA of 6 mg/m³ (equivalent to 20 mppcf) for this form of silica. OSHA believes that the health evidence for this substance is not sufficiently persuasive permit an increase in the limit at the present time. The Agency is proposing to change the units in which its permissible exposure limit is expressed; this change is being made to facilitate the accurate monitoring of employee exposures.

SILICA, AMORPHOUS, PRECIPITATED AND GEL
CAS: None; Chemical Formula: SiO_2
H.S. No. 1353

OSHA currently has a limit of 20 mppcf (equivalent to a limit of 6 mg/m³ if expressed in mg/m³ for amorphous silica. The ACGIH recommends a TLV–TWA of 10 mg/m³ measured as total dust containing less than 1 percent quartz. There are numerous methods of producing precipitated silica; those that apply heat to siliceous products produce airborne dusts; these are less toxic than quartz dust, because the particles are generally sheathed in a molecular layer of amorphous silica (ACGIH 1986, p. 521).

Studies of laboratory animals have shown no fibrosis after intratracheal and intraperitoneal injection of precipitated silica or silica gel (Klosterkotter 1954; Klosterkotter 1958). Schepers reported in 1957 that rats exposed for 1 year and guinea pigs and rabbits exposed for 2 years to a concentration of 126 mg/m³ of precipitated amorphous silica displayed no pulmonary fibrosis; the effects of exposure were limited to macrophage accumulations and mild proliferation of reticulin fibers (Schepers 1957).

In a study of human exposures to precipitated amorphous silica, Wilson reported no ill effects in 165 workers exposed for an average of 8.6 years (Wilson, Stevens et al. 1981).

The ACGIH considers the precipitated and gel form of amorphous silica an inert dust, based on the evidence discussed above. However, OSHA notes that effects were seen in animals exposed to this substance for 1 to 2 years and does not find that this evidence demonstrates the absence of significant risk necessary to support an increase in a PEL. Accordingly, OSHA is retaining its current PEL of 6 mg/m³ (equivalent to 20 mppcf) at the present time. The Agency is proposing to change the units in which its permissible exposure limit is expressed to facilitate the accurate monitoring of employee exposures.

SILVER (METAL DUST AND FUME)
CAS: 7440–22–4; Chemical Formula: Ag
H.S. No. 1362

The current OSHA standard for silver metal and soluble compounds (including the metal dust and fume) is 0.01 mg/m³. NIOSH has no REL for this substance, but the ACGIH has established a 0.1-mg/m³ TLV for silver metal dust and fume. The previous TLV of 0.01 mg/m³, which was established for all forms of silver, was designed to protect workers against developing argyria. This condition arises from the accumulation of silver in the body and results in an unsightly, widespread blue-grey discoloration of the skin that can persist for long periods of time. The skin of exposed workers may also become black and have a metallic luster. Argyria may also manifest itself in the conjunctiva of the eye, which may be affected sufficiently to cause lens and visual disturbances.

In arriving at the previous TLV of 0.01 mg/m³ for silver, the ACGIH relied on a publication by Pillsbury and Hill (1939), which stated that an accumulated intake of from 1 to 5 grams of silver would lead to generalized argyria. Assuming a 20-year exposure duration, a 10-m³/day respiratory volume, and a 50-percent body retention, the ACGIH estimated that exposure to 0.05 mg/m³ was sufficient to cause argyria. The former TLV of 0.01 mg/m³ thus appeared to incorporate a safety factor to account for the uncertainties involved in using this approach to develop a TLV. The ACGIH's current TLV of 0.1 mg/m³ for silver metal dust and fume was determined in a similar fashion, except that the ACGIH assumed a lower percent retention and apparently did not incorporate a safety margin (ACGIH 1986). Because the increase in the TLV from 0.01 to 0.1 mg/m³ is based on assumptions regarding the extent to which silver metal is systemically absorbed rather than on human data demonstrating the absence of a significant risk at the revised TLV, OSHA finds the ACGIH's reasoning unpersuasive. Thus DSHA is not at this time proposing to increase the PEL for silver metal dust and fume.

TETRAETHYL LEAD (TEL)
CAS: 78–00–2; Chemical Formula: $(C_2H_5)_4Pb$
H.S. No. 1386

OSHA's current 8-hour limit for tetraethyl lead is 0.075 mg/m³ as lead, with a skin notation; NIOSH has no REL for this substance. The ACGIH is now recommending that worker exposures not exceed 0.1 mg/m³ TWA; ACGIH also recommends a skin notation. The previous TLV of 0.075 mg/m³ was based almost exclusively on a personal communication from the Medical Department of the Ethyl Corporation, which stated that a level of 0.075 mg/m³ "is a good guideline for an allowable air concentration of TEL" (ACGIH 1966). The ACGIH documentation for the 0.075-mg/m³ TLV also pointed out that the ability of tetraethyl lead to penetrate the skin "makes reliance on the airborne concentration impractical in many situations," and that urinary lead levels were a more reliable indicator of exposure than blood lead levels (ACGIH 1966).

In its documentation for the 0.1-mg/m³ TLV, the ACGIH again cited the communication from the Ethyl Corporation. In addition, the organization cited a personal communication from Linch (1968), who reported that an improved analytical procedure for measuring airborne concentrations of tetraethyl lead had been used to determine the relationship between airborne tetraethyl lead levels and urinary lead levels. He reported that urinary lead concentration was not significantly elevated "above a high normal" value (0.15 mg/L) when the airborne TEL level was 121 ug/m³ ACGIH 1986). As a result of this communication, the ACGIH adopted a revised TLV of 0.1 mg/m³ in 1970.

OSHA does not find the evidence presented by the ACGIH to be sufficiently comprehensive or detailed

to permit significant risk to be ruled out at the 0.1-ppm level. The Agency is particularly reluctant to increase the PEL for TEL in light of this substance's ability to be absorbed percutaneously. OSHA is therefore not proposing to raise its PEL for tetraethyl lead.

TETRAMETHYL LEAD (TML)
CAS: 75–74–1; Chemical Formula: $(CH_3)_4Pb$
H.S. No. 1388

The current OSHA limit for tetramethyl lead (TML) is 0.075 mg/m³ with a skin notation, while the ACGIH has recommended a TLV of 0.15 mg/m³, also with a skin notation. There is no NIOSH REL for TML. In establishing the previous TLV of 0.15 mg/m³, the ACGIH cited the work of DeTreville (1962), who reported that tetramethyl lead is about three times more volatile than tetraethyl lead and thus resulted in employee exposures to airborne TML about three times higher than those for TEL. Despite the heavier TML exposure of employees, urinary lead levels were not significantly different from the urinary lead levels of employees exposed to TEL. The ACGIH concluded that a 0.075-mg/m³ TLV for TML, identical to the TLV recommended at the time for TEL, should furnish an adequate margin of safety. The revised TLV of 0.15 mg/m³ was based on a personal communication by Linch (1968), who reported that exposure to 0.179 mg/m³ tetramethyl lead was not associated with a significant increase in urinary lead levels.

Based on the same reasoning as that described above in connection with tetraethyl lead, OSHA is not proposing at this time to increase the existing OSHA limit for TML.

URANIUM (SOLUBLE COMPOUNDS)
CAS: 7440–61–1; Chemical Formula: U
H.S. No. 1419

The current OSHA limit for soluble uranium compounds is 0.05 mg/m³. NIOSH has no REL for soluble uranium compounds. Since 1968, the ACGIH has increased its TLV for soluble uranium from 0.05 mg/m³ to 0.2 mg/m³, with a 0.6-mg/m³ STEL. The previous TLV of 0.05 mg/m³ was based on animal studies relating exposure level and duration to the resulting tissue concentration of uranium and on other chronic animal studies showing the kidney to be the most sensitive target organ. In 1968, the ACGIH List of Intended Changes included a TLV of 0.2 mg/m³ for all forms of uranium, and this value was adopted by the ACGIH in 1969. The basis for adopting the 0.2-mg/m³ TLV for soluble uranium compounds was a study by Wing et al. (1963), reporting no adverse effects from radiation exposure over a 25-year period. Although no data were discussed in the ACGIH (1986) documentation regarding typical exposure levels at the plants studied, the documentation does mention that seven accidental, brief exposures to soluble uranium compounds at levels two- to five-fold the former TLV of 0.05 mg/m³ did not result in physiologic changes or significant body burden.

OSHA does not find that the evidence brought forward by the ACGIH is sufficiently detailed or comprehensive to meet the Agency's increased standard of proof for relaxing an existing standard. In addition, OSHA notes that the 25-year period of observation in the Wing et al. (1963) study is not long enough to rule out the occurrence of some forms of radiation-induced cancer and, further, that the power of the study to detect health effects occurring in a small percentage of the population was very limited. OSHA is accordingly not proposing to raise the current PEL for soluble uranium compounds.

17. Substances for Which OSHA is Proposing Short-Term Exposure Limits; Introduction

OSHA is proposing to add a short-term exposure limit (STEL) to a total of 134 substances; 126 of these STELs are values recommended by the ACGIH, while the remaining eight are NIOSH ceiling values for periods of 15 minutes or more. These substances are listed in Table C17–1.

When OSHA adopted the ACGIH TLVs via the Walsh-Healey Act and the OSH Act's section 6(a) mechanism, the ACGIH had not established the short-term TLV category; as a consequence, none of the substances on OSHA's Z–1 table have STELs. (Some of the substances on OSHA's current Z–2 tables, whose limits derive from standards established by the American National Standards Institute rather than the ACGIH, have "acceptable ceiling concentrations" that act in effect as short-term exposure limits.)

The ACGIH defines a STEL as

> a 15-minute time-weighted average exposure which should not be exceeded at any time during a work day even if the eight-hour time-weighted average is within the TLV. Exposures at the STEL should not be longer than 15 minutes and should not be repeated more than four times per day. There should be at least 60 minutes between successive exposures at the STEL. An averaging period other than 15 minutes may be recommended when this is warranted by observed biological effects (ACGIH 1987).

Basis Under Which ACGIH Established STELs

The ACGIH establishes STELs for substances that cause a wide variety of acute effects; these effects include irritation, narcosis, lung damage, systemic effects, and organic poisoning. The ACGIH first considered adding STELs to the TLV–TWAs for some substances in 1971 when it appointed a subcommittee to study the appropriateness of adding such exposure limits to its TLV list.

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Table C17-1. Substances for Which OSHA is Proposing STELs and Values Being Proposed

H.S. Number/ Chemical Name	CAS No.	ACGIH STEL	NIOSH Ceiling/STEL*
1001 Acetaldehyde	75-07-0	150 ppm	--
1002 Acetic acid	64-19-7	15 ppm	--
1007 Acrolein	107-02-8	0.3 ppm	--
1010 Allyl alcohol	107-18-6	4 ppm	--
1011 Allyl chloride	107-05-1	2 ppm	--
1012 Allyl glycidyl ether (AGE)	106-92-3	10 ppm	--
1013 Allyl propyl disulfide	2179-59-1	3 ppm	--
1021 Ammonia	7664-41-7	35 ppm	--
1022 Ammonium chloride (fume)	12125-02-9	20 mg/m^3	--
1042 Bromine	7726-95-6	0.3 ppm	--
1045 2-Butanone (MEK)	78-93-3	300 ppm	--
1047 n-Butyl acetate	123-86-4	200 ppm	--
1049 sec-Butyl alcohol	78-92-2	150 ppm	--
1050 tert-Butyl alcohol	75-65-0	150 ppm	--
1056 p-tert-Butyltoluene	98-51-1	20 ppm	--
1063 Camphor (Synthetic)	76-22-2	3 ppm	--
1064 Caprolactam (Dust)	105-60-2	3 mg/m^3	--
1065 Caprolactam (Vapor)	105-60-2	40 mg/m^3	--
1069 Carbon dioxide	124-38-9	30,000 ppm	--
1070 Carbon disulfide	75-15-0		10 ppm (15 min)
1072 Carbon tetrabromide	558-13-4	0.3 ppm	--
1074 Carbonyl fluoride	353-50-4	5 ppm	--
1078 Chlorinated camphene	9001-35-2	1 mg/m^3	--
1079 Chlorine	7782-50-5		0.5 ppm (15 min)
1080 Chlorine dioxide	10049-04-4	0.3 ppm	--
1082 2-Chloro-6-trichloromethyl pyridine (nitrapyrin)	1929-82-4	20 mg/m^3	--
1085 Chlorodifluoromethane	75-45-6	1250 ppm	--
1086 Chloroform	67-66-3		2 ppm (60 min)
1089 o-Chlorostyrene	1331-38-8	75 ppm	--
1090 o-Chlorotoluene	95-49-8	75 ppm	--
1091 Chlorpyrifos	2921-88-2	0.6 mg/m^3	--
1095 Clopidol (Coyden)	2971-90-6	20 mg/m^3	--
1103 Crufomate	299-86-5	20 mg/m^3	--
1110 Cyclonite	121-82-4	3 mg/m^3	--
1114 Decaborane	17702-41-9	0.15 ppm	--
1116 Di-sec-octyl-phthalate	117-81-7	10 mg/m^3	--
1119 Dibutyl phosphate	107-66-4	2 ppm	--
1122 1,3-Dichloro-5,5-dimethylhydantoin	118-52-5	0.4 mg/m^3	--

Table C17-1. Substances for Which OSHA is Proposing STELs and Values Being Proposed (continued)

H.S. Number/ Chemical Name	CAS No.	ACGIH STEL	NIOSH Ceiling/STEL*
1125 p-Dichlorobenzene	106-46-7	110 ppm	--
1127 Dichloroethyl ether	111-44-4	10 ppm	--
1137 Diethylamine	109-89-4	25 ppm	--
1143 Dimethylaniline	121-69-7	10 ppm	--
1149 Dipropylene glycol methyl ether	34590-94-8	150 ppm	--
1159 Ethanolamine	141-43-5	6 ppm	--
1161 Ethyl acrylate	140-88-5	25 ppm	--
1162 Ethyl benzene	100-41-4	125 ppm	--
1163 Ethyl bromide	74-96-4	250 ppm	--
1164 Ethyl ether	60-29-7	500 ppm	--
1168 Ethylene dichloride	107-06-2		2 ppm (15 min)
1170 Ethylene glycol dinitrate	628-96-6		0.1 mg/m^3 (20 min)
1177 Ferrovanadium dust	12604-58-9	3 mg/m^3	--
1179 Fluorine	7782-41-4	2 ppm	--
1182 Formamide	75-12-7	30 ppm	--
1184 Furfuryl alcohol	98-00-0	15 ppm, Skin	--
1185 Gasoline	8006-61-9	500 ppm	--
1194 n-Heptane	142-82-5	500 ppm	--
1201 Hexane isomers	- -0	1000 ppm	--
1203 Hexone (Methyl isobutyl ketone)	108-10-1	75 ppm	--
1208 Hydrogen fluoride	7664-39-3		6 ppm (15 min)
1209 Hydrogen sulfide	7783-06-4	15 ppm	--
1216 Iron pentacarbonyl	13463-40-6	0.2 ppm	--
1218 Isoamyl alcohol	123-51-3	125 ppm	--
1224 Isopropyl acetate	108-21-4	310 ppm	--
1225 Isopropyl alcohol	67-63-6	500 ppm	--
1227 Isopropyl glycidyl ether	4016-14-2	75 ppm	--
1228 Isopropylamine	75-31-0	10 ppm	--
1231 Ketene	463-51-4	1.5 ppm	--
1234 Manganese, fume	7439-96-5	3 mg/m^3	--
1242 Mercury, (organo) alkyl compounds	7439-97-6	0.03 mg/m	-
1243 Mesityl oxide	141-79-7	25 ppm	--
1248 Methyl 2-cyanoacrylate	137-05-3	4 ppm	--
1249 Methyl acetate	79-20-9	250 ppm	--
1250 Methyl acetylene/ propadiene mixture	74-99-7	1250 ppm	--
1252 Methyl alcohol	67-56-1	250 ppm, Skin	--
1254 Methyl chloride	74-87-3	100 ppm, Skin	-

Table C17-1. Substances for Which OSHA is Proposing STELs and Values Being Proposed (continued)

H.S. Number/ Chemical Name	CAS No.	ACGIH STEL	NIOSH Ceiling/STEL*
1255 Methyl chloroform (1,1,1-trichloroethane)	71-55-6	450 ppm	--
1258 Methyl formate	107-31-3	150 ppm	--
1261 Methyl isobutyl carbinol	105-30-6	40 ppm, Skin	--
1267 alpha-Methyl styrene	98-83-9	100 ppm	--
1270 o-Methylcyclohexanone	583-60-8	75 ppm, Skin	--
1281 Morpholine	110-91-8	30 ppm, Skin	--
1282 Naphthalene	91-20-3	15 ppm	--
1286 Nitric acid	7697-37-2	4 ppm	--
1289 Nitrogen dioxide	10102-44-0		1 ppm (15 min)
1290 Nitroglycerin	55-63-0		0.1 mg/m^3 (20 min)
1295 Octachloronaphthalene	2234-13-1	0.3 mg/m^3	--
1296 Octane	111-65-9	375 ppm	--
1297 Oil mist (Mineral)	8012-95-1	10 mg/m^3	--
1298 Osmium tetroxide	20816-12-0	0.006 mg/m^3	--
1299 Oxalic acid	144-62-7	2 mg/m^3	--
1301 Ozone	10028-15-6	0.3 ppm	--
1304 Pentaborane	19624-22-7	0.015 ppm	--
1306 Pentane	109-66-0	750 ppm	--
1307 2-Pentanone (Methyl propyl ketone)	107-87-9	250 ppm	--
1308 Perchloroethylene	127-18-4	200 ppm	--
1309 Perchloryl fluoride	7616-94-6	6 ppm	--
1317 Phenylhydrazine	100-63-0	10 ppm	--
1319 Phorate (Thimet)	293-02-2	0.2 mg/m^3	--
1320 Phosdrin (Mevinphos)	7786-34-7	0.3 mg/m^3	--
1321 Phosphine	7803-51-2	1 ppm	--
1322 Phosphoric acid	7664-38-2	3 mg/m^3	--
1323 Phosphorus oxychloride	10025-87-3	0.5 ppm	--
1324 Phosphorus pentasulfide	1314-80-3	3 mg/m^3	--
1324 Phosphorus trichloride	7719-12-1	0.5 ppm	--
1325 Picloram (Tordom)	1918-02-1	20 mg/m^3	--
1328 Picric acid	88-89-1	0.3 mg/m^3	--
1329 Propionic acid	79-09-4	15 ppm	--
1336 n-Propyl acetate	109-60-4	250 ppm	--
1338 Propyl alcohol	71-23-8	250 ppm	--
1340 n-Propyl nitrate	627-13-4	40 ppm	
1341 Propylene dichloride	78-87-5	110 ppm	--
1342 Propylene glycol monomethyl ether	107-98-2	150 ppm	--

Table C17-1. Substances for Which OSHA is Proposing STELs and Values Being Proposed (continued)

H.S. Number/ Chemical Name	CAS No.	ACGIH STEL	NIOSH Ceiling/STEL*
1346 Resorcinol	108-43-3	20 ppm	--
1366 Sodium fluoroacetate	62-74-8	0.15 mg/m^3	--
1372 Styrene (Phenylethylene)	100-42-5	100 ppm	--
1375 Sulfur dioxide	7446-09-5	5 ppm	--
1379 Sulfuryl fluoride	2699-79-8	10 ppm	--
1382 Tantalum	7440-25-7	10 mg/m^3	--
1387 Tetrahydrofuran	109-99-9	250 ppm	--
1397 Toluene	108-88-3	150 ppm	--
1398 Toluene-2,4-diisocyanate	584-84-9	0.02 ppm	--
1403 1,1,2-Trichloro-1,2,2-trifluorethane	76-13-1	1250 ppm	--
1408 Triethylamine	121-44-8	15 ppm	--
1411 Trimethylamine	75-50-3	15 ppm	--
1416 Tungsten & compounds (insoluble)	7440-33-7	10 mg/m^3	--
1417 Tungsten & compounds (soluble)	7440-33-7	3 mg/m^3	--
1418 Uranium (insoluble compounds)	7440-61-1	0.6 mg/m^3	--
1424 Vinyl acetate	108-05-4	20 ppm	--
1428 Vinylidene chloride	75-35-4	20 ppm	--
1431 Xylene (o,m,p-isomers)	1330-20-7	150 ppm	--
1435 Zinc chloride fume	7646-85-7	2 mg/m^3	--
1437 Zinc oxide (fume)	1314-13-2	10 mg/m^3	--
1434 Zinc stearate	557-01-1	20 mg/m^3	--
1435 Zirconium compounds	7440-67-7	10 mg/m^3	--

* NIOSH ceiling limits that are recommended for time periods of 15 minutes or more are treated as short-term exposure limits.

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In 1973, this subcommittee recommended that the ACGIH establish STELs as a third category (along with TLV–TWAs and ceilings) of exposure limits. The STEL was defined as the maximum concentration to which workers can be exposed *for a period up to 15 minutes* continuously without suffering from

1. Intolerable irritation,
2. Chronic or irreversible tissue change, or
3. Narcosis of sufficient degree to increase accident proneness, impair self-rescue, or materially reduce work efficiency (ACGIH 1984).

The ACGIH stipulated that no more than four excursions per day were permissible, with at least 60 minutes between exposure periods, and that the daily TLV–TWA could not be exceeded.

In 1974, the ACGIH agreed by consensus that 425 of the 520 compounds in its 1973 list should be assigned STELs, but these were not in fact published until 1976, when "Tentative Values" for STELs were listed in the organization's annual booklet. In 1978, the ACGIH emphasized that, according to its definition, a STEL was not a TWA value but a maximal allowable concentration, or absolute ceiling, not to be exceeded for any time during the 15-minute excursion period. The TWA–STEL should not be used as an engineering design criterion or considered as an emergency exposure level (ACGIH 1978).

ACGIH redefined the application of STELs in 1979, 1982, and 1984. The 1987–1988 ACGIH TLV booklet states that the TLV–STEL is "the concentration to which workers can be exposed continuously for a short period of time without suffering from (1) irritation, (2) chronic or irreversible tissue damage, or (3) narcosis of sufficient degree to increase the likelihood of accidental injury, impair self-rescue or materially reduce work efficiency * * * provided that the daily TLV–TWA is not exceeded."

In 1982, the ACGIH qualified the conditions under which STELs are recommended to situations "only where toxic effects have been reported from high short-term exposures in either humans or animals." In 1984, the ACGIH proposed the deletion of STELs for 142 substances in its Notice of Intended Changes; most of these STELs were deleted in 1986. Another 53 STELs were proposed for deletion in 1986. These two major revision efforts successfully eliminated many STELs for substances with inadequate toxicological support data. As the introduction to the most recent edition of the *Threshold Limit Values and Biological Exposure Indices* (1986) indicates:

> For the vast majority of substances with a TLV–TWA, there is not enough toxicological data available to warrant a STEL. Nevertheless, excursions above the TLV–TWA should be controlled even where the eight-hour TWA is within recommended limits (ACGIH 1967). At present, a total of 122 STELs remain in the ACGIH's most recent TLV list; the organization has removed its recommended STELs for 297 substances on the ground that insufficient toxicological data existed on which to base a STEL.

The ACGIH has stressed that STELs are set on physiological grounds rather than in response to sampling and analytical limitations (ACGIH 1984).

Separate from the STEL category, the ACGIH in the 1970s established a fourth limit, a general "excursion factor" that should always be observed implicitly but is not specifically assigned to each chemical. The "excursion limit" recommended by the ACGIH is as follows:

> Short-term exposures should exceed three times the TLV–TWA for no more than a total of 30 minutes during a work day and under no circumstances should they exceed five times the TLV–TWA, provided that the TLV–TWA is not exceeded (ACGIH 1987).

The basis for this excursion recommendation is that any process whose emissions display a variability greater than would be permitted by this excursion factor is not under good industrial hygiene control, and the ACGIH believes that in such cases, efforts should be made to restore control (ACGIH 1986). Where specific STELs exist, they take precedence over the general excursion limit (ACGIH 1987). Thus *all* ACGIH TLV–TWAs have implicit excursion limits, but only a few substances (i.e., those for which specific toxicological evidence indicates that a STEL is necessary) have explicit STELs.

Significance of Risk

The STELs being proposed by OSHA in this rulemaking, which parallel those STELs remaining in the ACGIH's most recent list (ACGIH 1987–1988)) are thus limits for substances where there is toxicological evidence of recognized acute effects resulting from short-term exposure. (STELs also are appropriate to reduce significant risk remaining at the 8-hour TWA limit. See *Public Citizen HRG* v. *Tyson*, 796 F.2d 1479, 1505 (D.C. Cir. 1986). That issue does not arise for most of the substances covered in this rulemaking. If evidence is presented or available on this issue, OSHA will consider an appropriate regulatory response.) The health effects associated with short-term exposures for some of these substances are shown in Table C17–2.

TABLE C17–2. HEALTH EFFECTS SUPPORTING PROPOSED ACGIH STELS

H.S. Number/ Chemical Name	ACGIH STEL**	Health Effects
1001 Acetaldehyde.	150 ppm	Eye irritation; narcosis; potential injury to respiratory tract.
1002 Acetic acid.	15 ppm	Irritation.
1007 Acrolein	0.3 ppm	Irritation; lung edema.
1010 Allyl alcohol.	4 ppm	Irritation.
1011 Allyl chloride.	2 ppm	Mucous membrane irritation.
1012 Allyl glycidyl ether (AGE).	10 ppm	Irritation.
1013 Allyl propyl disulfide.	3 ppm	Irritation; lacrimation.
1021 Ammonia	35 ppm	Irritation; temporary blindness.
1022 Ammonium chloride (fume).	20 mg/m³	Irritation.
1042 Bromine	0.3 ppm	Respiratory tract irritation.
1045 2-Butanone (MEK).	300 ppm	Eye and nose irritation.
1047 n-Butyl acetate.	200 ppm	Throat irritation.
1049 sec-Butyl alcohol.	150 ppm	Irritation; narcosis.
1050 tert-Butyl alcohol.	150 ppm	Narcosis.
1063 Camphor (synthetic).	3 ppm	Eye and nose irritation; anosmia.
1064 Caprolactam (dust).	3 mg/m³	Irritation.
1065 Caprolactam (vapor).	40 mg/m³	Irritation.
1072 Carbon tetrabromide.	0.3 ppm	Upper respiratory tract irritation; injury to lungs, liver, and kidney.
1080 Chlorine dioxide.	0.3 ppm	Irritation.
1082 2-Chloro-6-trichloromethyl pyridine (nitrapyrin).	20 mg/m³	Nuisance effects.
1085 Chlorodifluoromethane.	1250 ppm	CNS effects; asphyxiation; narcosis.
1090 o-Chlorotoluene.	75 ppm	Cardiovascular effects.
1091 Chlorpyrifos.	0.6 mg/³	Cholinesterase inhibition.
1103 Crufomate.	20 mg/m³	Cholinesterase inhibition.
1110 Cyclonite	3 mg/m³	CNS effects.
1119 Dibutyl phosphate.	2 ppm	Irritation to respiratory tract; headaches.
1122 1,3-Dichloro-5,-5-dimethyl-hydantoin.	0.4 mg/m³	Respiratory irritation.

TABLE C17-2. HEALTH EFFECTS SUPPORTING PROPOSED ACGIH STELS—Continued

H.S. Number/ Chemical Name	ACGIH STEL**	Health Effects
1125 p-Dichlorobenzene.	110 ppm	Acute poisoning.
1127 Dichloroethyl ether.	10 ppm	Upper respiratory tract and eye irritation.
1137 Diethylamine.	25 ppm	Acute toxicity characterized by strong local irritation.
1149 Dipropylene glycol methyl ether.	150 ppm	Eye, nose, and throat irritation; central nervous system impairment.
1161 Ethyl acrylate.	25 ppm	Irritation.
1162 Ethyl benzene.	125 ppm	Skin and eye irritation.
1163 Ethyl bromide.	250 ppm	Narcosis.
1164 Ethyl ether.	500 ppm	Narcosis; nasal irritation.
1179 Fluorine.....	2 ppm	Eye and skin irritation.
1184 Furfuryl alcohol.	15 ppm	Eye irritation.
1194 n-Heptane.	500 ppm	Narcosis; respiratory irritation.
1201 Hexane isomers.	1000 ppm	Narcotic symptoms; eye and throat irritation; slight nausea, headache.
1203 Hexone (MIBK).	75 ppm	Irritant effects.
1209 Hydrogen sulfide.	15 ppm	Eye irritation.
1216 Iron pentacarbonyl.	0.2 ppm	Headaches; dizziness.
1218 Isoamyl alcohol.	125 ppm	Respiratory and eye irritation.
1224 Isopropyl acetate.	310 ppm	Eye and respiratory irritation.
1225 Isopropyl alcohol.	500 ppm	Narcotic effects and irritation.
1227 Isopropyl glycidyl ether.	75 ppm	Respiratory tract and eye irritation.
1228 Isopropylamine.	10 ppm	Respiratory irritation.
1231 Ketene.....	1.5 ppm	Respiratory irritation.
1236A Manganese, fume.	3 mg/m³	Central nervous system effects.
1243 Mesityl oxide.	25 ppm	Eye and mucous membrane irritation, breathing difficulty, headache and vertigo.
1248 Methyl 2-cyanoacrylate.	4 ppm	Nasal and eye irritation.
1249 Methyl acetate.	250 ppm	Ocular and nervous disturbances; eye, mucous membrane, upper and lower respiratory tract irritation.
1252 Methyl alcohol.	250 ppm	Recurrent headaches; diminution of vision.
1255 Methyl chloroform(1,1,1-trichloroethane).	450 ppm	Anesthesia.
1258 Methyl formate.	150 ppm	Visual disturbances (temporary blindness); narcotic symptoms, mucous membrane irritation; dyspnea.
1261 Methyl isobutyl carbinol.	40 ppm	Eye irritation.
1270 o-Methylcyclohexanone.	75 ppm	Eye and respiratory irritation.
1281 Morpholine.	30 ppm	Irritation and harmful effects to eyes and vision.
1282 Naphthalene.	15 ppm	Ocular effects.
1296 Octane.....	375 ppm	Acute effects on nervous system.
1299 Oxalic acid.	2 mg/m³	Severe local burns to eyes, mucous membranes, and skin.
1304 Pentaborane.	0.015 ppm	Central nervous system effects.
1306 Pentane.....	750 ppm	Narcotic and irritative effects.
1307 2-Pentanone (MPK).	250 ppm	Narcotic effects; irritation.
1308 Perchloroethylene.	200 ppm	Anesthetic effects.
1309 Perchloryl fluoride.	6 ppm	Respiratory irritation; fluorosis.
1323 Phosphorous oxychloride.	0.5 ppm	Irritation.
1324 Phosphorus pentasulfide.	3 mg/m³	Respiratory irritation.
1325 Phosphorus trichloride.	0.5 ppm	Respiratory irritation.
1328 Picloram (Tordom).	20 mg/m³	Systemic effects.
1336 Propionic acid.	15 ppm	Eye and respiratory irritation.
1339 Propyl alcohol.	250 ppm	Possible deep narcosis.
1343 Propylene glycol monomethyl ether.	150 ppm	Odor; eye irritation.
1372 Styrene, monomer.	100 ppm	Tremors with subsequent severe convulsions; pulmonary edema may follow severe single exposure.
1375 Sulfur dioxide.	5 ppm	Respiratory effects.
1387 Tetrahydrofuran.	250 ppm	Narcotic and irritative effects.
1397 Toluene.....	150 ppm	Impairment of coordination, momentary memory loss, anorexia.
1403 1,1,2-Trichloro-1,2,2-trifluoroethane.	1250 ppm	Impairment of psychomotor performance.
1408 Triethylamine.	15 ppm	Acute irritation of eyes, mucous membranes, and lungs.
1424 Vinyl acetate.	20 ppm	Irritation.
1428 Vinylidene chloride.	20 ppm	Overt toxicity.
1431 Xylene (o,m,p-isomers).	150 ppm	Narcosis, irritant effects.
1435 Zinc chloride (fume).	2 mg/m³	Respiratory irritation.

** The ACGIH TWA-TLV is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

Preliminary Conclusions

OSHA is proposing STELs where the Agency believes a short-term limit is needed to supplement the 8-hour TWA to protect workers from the adverse health effects associated with exposure to short-term excursions that would be permitted with the 8-hour limit alone. OSHA preliminarily concludes that, without a STEL, workers remain at risk of experiencing the broad range of recognized acute effects associated with elevated short-term exposures to these chemicals. Compliance with a STEL in addition to an 8-hour limit will substantially reduce the risk of health impairment and functional incapacity potentially faced by workers exposed to these substances during excursions to the levels permitted by the 8-hour limit alone. OSHA believes that, for this

group of substances, the toxicological evidence forms a reasonable basis for proposing to supplement the 8-hour permissible exposure limit with a short-term limit.

At the time of the final rule, OSHA will establish short-term exposure limits for these substances if it determines that STELs will substantially reduce significant risk.

18. Substances for Which OSHA Is Proposing To Add Skin Notations

There are a number of instances in this rulemaking for which OSHA is proposing to add a skin notation to the limit for a substance. In all, a skin notation is being added in 49 cases. Table C18–1 shows all of the substances for which the Agency is proposing to add skin notations.

The ACGIH began to include skin designations for the chemicals in its list for the first time in 1961 (Stokinger 1961). At that time, the organization stated that:

> This notation is to be interpreted simply as an indicator that skin absorption may contribute to the overall intake from exposure in addition to that from inhalation. It refers mainly to absorption from liquid contamination (Stokinger 1961).

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TABLE C18 1. List of Substances for Which OSHA Is Proposing to Add a Skin Notation

H.S. Number/ Chemical Name	CAS No.
1012 Allyl glycidyl ether (AGE)	106-92-3
1051 n-Butyl alcohol	71-36-3
1055 o-sec-Butylphenol	89-72-5
1066 Captafol (Difolatan)	2425-06-1
1084 o Chlorobenzylidene malonitrile	2698-41-1
1091 Chlorpyrifos	2921-88-2
1107 Cyclohexanol	108-93-0
1108 Cyclohexanone	108-94-1
1110 Cyclonite	121-82-4
1118 Diazinon	333-41-5
1120 2-N-Dibutylaminoethanol	102-81-8
1129 1,3-Dichloropropene	542-75-6
1131 Dicrotophos (Bidrin)	141-66-2
1138 Diethylene triamine	111-40-0
1141 Dimethyl 1,2-dibromo-2,2-dichloroethyl phosphate	300-76-5
1146 Dioxathion (Delnav)	78-34-2
1156 Endosulfan	115-29-7
1160 Ethion (Nialate)	563-12-2
1173 Fenamiphos	22224-92-6
1175 Fenthion	55-38-9
1181 Fonofos	944-22-9
1184 Furfuryl alcohol	98-00-0
1195 Hexachlorobutadiene	87-68-3
1198 Hexafluoroacetone	684-16-2
1211 2-Hydroxypropyl acrylate	999-61-1
1220 Isooctyl alcohol	26952-21-6
1229 n-Isopropylaniline	643-28-7
1237 Manganese cyclopentadienyl tricarbonyl	12079-65-1
1241 Mercury (vapor)	7439-97-6
1242 Mercury, (organic) alkyl compounds	7439-97-6
1251 Methyl acrylonitrile	126-98-7
1252 Methyl alcohol	67-56-1
1256 Methyl demeton	8022-00-2
1265 Methyl parathion	298-00-0
1271 Methylcyclopentadienyl manganese tricarbonyl	12108-13-3
1273 4,4'-Methylene bis(2-chloroaniline)	5124-30-1
1313 Phenothiazine	92-84-2
1319 Phorate (Thimet)	298-02-2
1335 Propargyl alcohol	107-19-7
1339 Propyl alcohol	71-23-8
1342 1,2-Propylene glycol dinitrate	6423-43-4
1392 Thioglycolic acid	68-11-1
1394 Tin (organic compounds)	7440-31-5
1400 p-Toluidine	106-49-0
1401 m-Toluidine	108-44-1
1407 1,2,3-Trichloropropane	96-18-4
1414 Triorthocresyl phosphate	78-30-8
1426 Vinyl cyclohexene dioxide	106-87-6
1432 m-Xylene-alpha,alpha'-diamine	1477-55-0

BILLING CODE 4510-26-C

The ACGIH has expanded on its reasoning since the 1960s, and the preface to the most recent *Threshold Limit Values and Biological Exposure Indices* (1987–1988) explains that the skin designation is designed to call attention to the need for "appropriate measures for the prevention of cutaneous absorption so that the threshold limit is not invalidated" (p. 7). Thus a skin notation warns that exposure via the cutaneous route, including absorption through the eyes or mucous membranes, by either inhalation or direct contact, may contribute substantially to an employee's overall exposure and cause systemic toxicity.

In establishing most skin designations, OSHA has relied primarily on animal data, particularly on dermal LD_{50} values in rodents (mice, rats, rabbits, and guinea pigs). This evidence is occasionally supported by findings in humans, and in a few cases, human evidence provides the sole basis. The ACGIH has a policy of using a dermal LD_{50} of 2 g/kg as a general cutoff for determining when to classify a substance as sufficiently absorbable to present a hazard via the percutaneous route; that is, substances having a single-dose dermal LD_{50} of less than 2 g/kg receive a skin notation, while those with dermal LD_{50}s above this cutoff do not (ACGIH 1986, p. 332). The *Documentation* (ACGIH 1986) contains no cutoff value for chronic dermal exposures, i.e., for toxicity resulting from repeated applications of substances to the skin.

The following discussions describe OSHA's reasons for adding or deleting skin notations for some OSHA-regulated chemicals or the regulatory candidates under consideration.

o-sec-Butylphenol. In assigning a skin notation to this substance, the ACGIH relied on an unpublished Dow Chemical Company study showing that the dermal LD_{50} for butylphenol in guinea pigs is between 0.6 and 2.4 g/kg. This range clearly includes the single-dose percutaneous cutoff value of 2 g/kg, and skin absorption of butylphenol thus has the potential to contribute to overall exposure and to cause systemic effects in dermally exposed workers. OSHA preliminarily concludes that the addition of a skin notation is warranted by this avoidance and that the evidence of dermal contact will eliminate the risk of systemic toxicity posed by this chemical.

Phorate. Phorate, an organophosphate insecticide, has been proposed for a skin designation on the basis of its high acute percutaneous toxicity. In male and female rats, the dermal LD_{50} is 6.2 and 2.5 mg/kg, respectively, values substantially below the level used as the criterion for permeability via the percutaneous route (ACGIH 1986, p. 332). OSHA preliminarily concludes that preventing skin contact with this chemical will protect exposed workers from experiencing the neurologic effects associated with overexposure to this cholinesterase inhibitor.

Vinyl cyclohexene dioxide. The skin notation for vinyl cyclohexene dioxide is based on evidence that this substance is highly toxic in rats given a single skin application. The dermal LD_{50} in this species is 0.62 ml/kg. In addition, this chemical has been shown to cause skin tumors and cancers developing late in life in mice receiving dermal applications of a 30-percent solution of vinyl cyclohexene dioxide in acetone. The ACGIH (1986) reports that "extreme caution should be exercised in the use of this diepoxide" because of the "very limited toxicological data and * * * demonstrated carcinogenicity when * * * applied to the skin of the mouse." OSHA preliminarily concludes that the addition of a skin designation to the limit for this chemical is essential to protect workers both from systemic toxicity via the percutaneous route and from the potential risk of developing either systemic or skin cancers.

Substances for Which the ACGIH Has Deleted the Skin Notation

For four substances, the ACGIH has deleted the skin notations that have appeared in the 1968 edition of the *Documentation* and which were subsequently adopted by OSHA under the section 6(a) mechanism in 1971. Table C18–2 shows these chemicals.

OSHA is not proposing to delete the skin notations for these four substances. The Agency believes that deletion of these designations would in effect constitute an increase in the level of exposure permitted and thus a decrease in the extent of worker protection provided by the limits of the current Z tables. In accordance with principles established by OSHA (see the preamble for the final revisions to the cotton dust standard, 50 FR 51120 *et seq.*), the Agency must demonstrate that deleting these skin designations, which were established under the section 6(a) mechanism, will not pose a significant risk to exposed workers. The discussion below describes the ACGIH's reasons for recommending deletion of these notations.

TABLE C18-2. List of Substances for Which the ACGIH Has Deleted the Skin Notation

H.S. Number/ Chemical Name	CAS No.
1113 DDT	50-29-3
1149 Dipropylene glycol methyl ether	34590-94-8
1197 Hexachloroethane	67-72-1
1303 Paraquat, respirable dust	4685-14-7

The evidence on which the ACGIH based its decision to delete skin notations for the four chemicals in question is primarily animal evidence. For DDT and hexachloroethane, the ACGIH deleted the skin designation based on the relatively low dermal toxicity demonstrated by these substances in animal studies. OSHA preliminarily finds, however, that the absence of significant risk via dermal absorption has not been sufficiently shown for DDT and hexachloroethane, and thus OSHA is not proposing deletion at this time.

For the two remaining substances in this group, paraquat and dipropylene glycol methyl ether (DPGME), the skin notation was deleted because the ACGIH believes that the substance does not, in the case of paraquat, "penetrate the unbroken or uninjured skin" or believes, as in the case of DPGME, that the substance is "practically nontoxic * * * by the dermal route for rabbits" (ACGIH 1986). However, OSHA notes that at high doses paraquat does "injure and break down dermal barriers" and gain entry to the body. The *Documentation* records the case of a 44-year-old man who died of respiratory insufficiency after he was poisoned by the percutaneous absorption of an acutely toxic quantity of undiluted paraquat (ACGIH 1986). The ACGIH also reports that there is evidence that, despite DPGME's low single-dose dermal toxicity, repeated applications of the chemical to the skin of rabbits caused death "in a significant number of the exposed rabbits at levels of 3 ml/kg and above" (ACGIH 1986).

In accordance with the principles stated in the cotton dust preamble (50 FR 51120) OSHA does not find the evidence adduced by the ACGIH sufficient to provide a basis for the deletion of the skin notations for this group of substances. The Agency preliminarily concludes that deleting the skin notation from the limits for these four substances will not ensure that workers are protected against the risks potentially posed by percutaneous absorption of these substances.

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V. Summary of Preliminary Feasibility, Regulatory Impact, Regulatory Flexibility and Environmental Impact Analyses

The OSHA Act requires the Agency to consider the feasibility of proposed standards. Executive Order 12291 (46 FR 13197) requires that a regulatory analysis be conducted for any rule having major economic consequences on the national economy, individual industries, geographical regions, or levels of government. The Regulatory Flexibility Act (5 U.S.C. 601 *et seq.*) similarly requires OSHA to consider the impact of the proposed regulation on small entities. Consistent with these requirements, OSHA has prepared a Feasibility Analysis, and a Preliminary Regulatory Impact and Regulatory Flexibility Analysis for this proposed rule. Details supporting this Summary are included in Appendix B of this proposal. Not all of the Supplements to Appendix B are published in this proposal. They are, however, available from OSHA. Instructions on how to obtain the Supplements are given at the end of Appendix B.

Approach

Because this rulemaking involves about 430 chemicals, OSHA has prepared the regulatory impact analysis in two phases. Phase I involved the use of a number of secondary data bases to collect information on the chemicals to be regulated and the industries in which they are used. These data bases provided information on the toxicity and health effects of exposure to the chemicals, and current information on engineering controls in use and emergency response procedures. Two data bases provided information on employee exposures. The 1982 National Occupational Exposure Survey (NOES) was based on a sample of about 4,500 businesses. The data base developed from this survey contains an estimate of the number of persons occupationally exposed to hazardous substances by Standard Industrial Classification (SIC). The second data base was OSHA's Integrated Management Information System (IMIS). The IMIS contains the results of air samples taken since 1979 by OSHA industrial hygienists in the course of compliance inspections. Both the NOES and IMIS data bases provided valuable information on the nature and extent of employee exposures to the substances to be regulated; however, they did not provide complete information on all substances. Supplementary information was obtained from industrial hygienists and engineers. These experts identified exposure controls in use, and the number and size of plants most likely to be affected by this rulemaking. These sources have provided OSHA with a substantial body of information on chemical use, exposures and controls.

Phase II of the data collection effort involved a sampling survey of over 5,300 firms in industries where chemical exposures were believed to pose potential problems. The survey, conducted during the first part of 1988, gathered data on chemicals, processes, exposures and controls currently in use. These additional data have permitted OSHA to refine the Phase I preliminary estimates of technical and economic feasibility. In addition, site visits to over 100 plants are underway to verify the data collected to date on chemicals, processes, controls and employee exposures. The reports covering these site visits will be submitted to the docket prior to the completion of the public hearing in this rulemaking.

Employee Exposure and Benefits

Revising OSHA's Z-Table limits for hazardous substances is expected to result in reduced risk of chemically related disease among exposed employees. Exposure to substances included in the rulemaking has been associated with a variety of adverse health effects, including impairment of organ system functions, mucous membrane irritation, neuropathy, narcosis, allergic sensitization, respiratory disease, cardiovascular disease, and cancer.

Using data from OSHA's IMIS system and information collected from the survey of over 5,300 establishments, OSHA estimates over 17 million employees are potentially exposed to hazardous substances in the workplace. OSHA also estimates that over 3.6 million employees are currently exposed above the proposed exposure limits for these substances. Table V–1 summarizes OSHA's estimates of the number of workers currently at risk of adverse health effects. OSHA estimates that promulgation of the proposed exposure limits will result in a potential reduction of over 55,000 work-related illness cases per year, over 23,600 lost-workday illness cases per year, and over 533,000 lost workdays due to illness per year. OSHA's preliminary estimate is that industry compliance with the proposed exposure limits will result in a reduction of 519 fatalities caused by exposure to substances that cause cancer, respiratory disease, cardiovascular disease, or liver or kidney disease per year.

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TABLE V-1

ESTIMATED NUMBER OF WORKERS POTENTIALLY AT RISK OF EXPERIENCING ADVERSE EFFECTS, BY TYPE OF ADVERSE EFFECT*

ADVERSE HEALTH EFFECT	NO. OF WORKERS POTENTIALLY EXPOSED TO SUBSTANCES ASSOCIATED WITH EFFECT, MINIMUM ESTIMATE	NO. OF WORKERS POTENTIALLY EXPOSED TO SUBSTANCES ASSOCIATED WITH EFFECT, MAXIMUM ESTIMATE	NO. OF WORKERS EXPOSED ABOVE PROPOSED LIMITS FOR SUBSTANCES, MINIMUM ESTIMATE	NO. OF WORKERS EXPOSED ABOVE PROPOSED LIMITS FOR SUBSTANCES, MAXIMUM ESTIMATE
NUISANCE EFFECTS	4,729,417	5,804,846	829,487	892,725
ODOR AND TASTE EFFECTS	718,522	808,537	59,102	59,102
SYSTEMIC TOXICITY	3,233,319	3,818,742	250,282	256,909
MUCOUS MEMBRANE IRRITATION	12,077,315	15,813,663	59,969	1,021,197
METABOLIC INTERFERENCES	2,277,113	2,345,975	746,879	746,879
LIVER/KIDNEY DISEASE	2,321,573	2,488,604	383,581	384,876
OCULAR DISTURBANCES	194	194	0	0
RESPIRATORY DISEASE	3,765,717	4,023,525	639,717	654,319
CARDIOVASCULAR DISEASE	164,576	164,576	44,355	44,355
NEUROPATHY	1,231,744	1,349,421	201,760	210,203
NARCOSIS	4,601,993	6,381,899	526,021	547,731
CANCER	3,663,053	3,784,374	499,716	499,716
ALLERGIC SENSITIZATION	2,710,576	2,903,153	296,444	297,255

*Double counting of employees simultaneously exposed to more than one substance in different adverse health effects categories, prevents the summation of workers exposed to all adverse health effects in this table.

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Nonregulatory Alternatives

OSHA believes that there are no nonregulatory alternatives that adequately protect most workers from the adverse health effects associated with exposure to the chemicals under consideration. OSHA believes that the tort liability laws and Workers' Compensation do not provide adequate worker protection due to market imperfections. Some employers have not complied with the standards recommended by professional organizations. The deleterious health effects resulting from continued high levels of exposure to hazardous substances require a regulatory solution.

Technological Feasibility

Consistent with OSHA regulations and policy, engineering controls and work practices to control employee exposure are preferred over the use of personal protective equipment.

Engineering controls involve the use of local exhaust ventilation, general ventilation, isolation of the worker and enclosure of the source of emissions, process modifications, equipment modifications, and substitution of non-hazardous chemicals. These methods may be used alone or in combination depending upon the industrial processes involved. These controls are widely used and will effectively control exposures either by themselves, or coupled with changes in work practices.

Perhaps the most widely used technique for controlling chemical exposure is the use of ventilation. General ventilation uses the movement of air within the general work space to displace or dilute the contaminant with fresh outside air. General ventilation may not be the preferred control method, however, due to the large volumes of air movement required. Local exhaust ventilation uses much smaller volumes of air, exhausted from the point or source at which contaminants are generated.

Isolation involves placing a physical barrier between the hazardous operation and the worker. Many modern, automated manufacturing processes are now fully enclosed in ventilated cabinets. The effectiveness of such a control technique depends on the frequency with which the workers have to enter the enclosure during normal operations. In other situations, rather than placing the process or machine in an enclosure, the worker is placed in an enclosure. Many processes which involve potential chemical exposures are operated remotely by operators in air-conditioned booths isolated from the hazardous materials.

Substitution refers to the replacement of a toxic chemical in a particular process or work area with another, less toxic product. Properly applied, substitution can be a very effective control technique. However, care must be taken to ensure that the proposed substitute performs in a similar manner to the product being replaced. In addition, it is essential that the substitute be carefully evaluated to ensure that in controlling one hazard, another different hazard is not inadvertently introduced. The substitute must also be compatible with existing manufacturing equipment and processes.

The success of these techniques will depend on the physical properties of the chemicals and emissions encountered (boiling point, vapor pressure, etc.) and the process operating conditions. In some cases, particularly with cleaning solvents, substitution may provide the quickest and most effective means of reducing exposure. In other situations, a major effort may be required to alter processes or install or expand local or general dilution ventilation.

OSHA believes that existing engineering controls are available to reduce exposure levels to the new proposed levels. Standard controls have been adapted in numerous situations to solve situation-specific problems in all of the industry sectors affected. Detailed industry-specific illustrations of this point are presented in the Technological Feasibility Chapter of the Preliminary Regulatory Impact Analysis.

Costs of Compliance

Costs of compliance with the proposed rulemaking would result from industry actions to lower workers' chemical exposure to the levels proposed. The 1988 sample survey of more than 5,300 firms was drawn from a universe of over one million firms potentially affected by the rule. Table V–5 at the end of this section presents a list of industries included in the analysis.

Survey respondents verified the number of work stations and workers related to each process, the process location and configuration, the controls already in place, and potential chemical exposures above new proposed levels. Process controls in place were compared to a list of control designs needed to limit exposures to new lower levels. Where the required controls were not reported to be in place, a compliance cost per work station was assigned. Process control costs were summed per establishment and certain maintenance workers were assigned a respirator cost. Costs for the surveyed establishments were then weighted (by SIC and size) to represent compliance costs for the universe of affected plants.

The survey found that about 500,000 establishments use the chemicals being regulated. Of this number, about 101,200 would incur some costs to comply with the proposed rule. The total estimated annualized capital plus annual operating costs are $927.83 million. Table V–2 presents the annual cost by industry sector, for large and small (fewer than 20 employees) plants.

Among all industry sectors affected by this proposal, about 101,200 establishments are estimated to incur, on average, an annual cost of $9,200.

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TABLE V-2

ANNUAL OPERATING AND ANNUALIZED CAPITAL COST OF COMPLIANCE BY INDUSTRIAL SECTOR (a)

SIC (b)	SIC DESCRIPTION	LARGE PLANTS	SMALL PLANTS	ANNUAL COST
20	FOOD PROD. (c)	$21,704,100	$11,789,000	$33,493,100
21	TOBACCO (c)	$19,700	$0	$19,700
22	TEXT. MILL (c)	$23,308,400	$6,170,000	$29,478,400
23	APPAREL PROD. (c)	$23,604,300	$8,139,900	$31,744,200
24	LUMBER & WOOD	$20,534,800	$175,551,300	$196,086,100
25	FURNITURE	$7,915,300	$6,805,300	$14,720,600
26	PAPER PROD.	$30,966,900	$290,800	$31,257,700
27	PRINTING & PUB.	$15,263,300	$60,425,000	$75,688,300
28	CHEMICAL PROD.	$31,745,500	$8,412,100	$40,157,600
29	PETRO. REFINING	$6,800,700	$414,600	$7,215,300
30	RUBBER & PLASTICS	$53,256,200	$22,225,900	$75,482,100
31	LEATHER PROD.	$1,464,400	$1,115,100 (c)	$2,579,500
32	STONE & CLAY	$15,079,300	$7,463,300	$22,542,600
33	PRIM. METAL	$34,721,200	$5,612,400	$40,333,600
34	FAB. METALS	$50,927,100	$5,010,500	$55,937,600
35	MACHINERY	$43,986,300	$13,754,100	$57,740,400
36	ELEC. MACH.	$24,210,900	$7,570,500	$31,781,400
37	TRANS. EQUIP.	$20,884,600	$26,214,500 (c)	$47,099,100
38	INSTRUMENTS	$10,257,300	$3,207,400	$13,464,700
39	MISC. MANUF.	$13,861,200	$4,334,300	$18,195,500
40	R.R. TRANS.	$532,200	$0	$532,200
45	AIR TRANS.	$1,828,900	$0	$1,828,900
47	TRANS. SERV.	$1,853,100	$0	$1,853,100
49	ELEC. GAS. SAN.	$19,373,900	$3,314,500	$22,688,400
50	WHOLESALE TRADE	$1,416,300	$2,638,300	$4,054,600
51	WHOLESALE, NON-DUR	$3,094,200	$5,764,000	$8,858,200
55	AUTO DEALERS (c)	$9,862,500	$2,092,200	$11,954,700
72	PERSONAL SRV. (c)	$15,648,500	$15,639,300	$31,287,800
73	BUSINESS SRV. (c)	$3,701,700	$5,252,100	$8,953,800
75	AUTO REPAIR (c)	$466,800	$1,044,100	$1,510,900
76	MISC. REPAIR SRV.	$669,500	$4,179,000	$4,848,500
80	HEALTH SERV. (c)	$2,413,000	$2,026,400	$4,439,400
TOTAL		$511,372,100	$416,455,900	$927,828,000

Source: U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.

(a) Costs were calculated by annualizing the capital cost over the projected life of the equipment (10 years) using a 10 percent cost of capital and adding an annual operating and maintenance cost estimated at 10 percent of the capital cost.

(b) Industry sectors not identified in this table include industries with no major cost impact expected, the construction industry, which will be the subject of a separate regulatory analysis, and industries such as mining, over which OSHA has no jurisdiction.

(c) Costs in these sectors were based on expert judgement and secondary data collection. Survey data for SICs 55, 72, 73, 75 and 80 was insufficient to estimate compliance costs.

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Economic Impact

OSHA prepared two estimates of the economic effects of the proposal on potentially affected firms. The two estimates were based upon Zero Cost-Passthrough ("worst case") and Total Cost-Passthrough ("best case") scenarios.

In the first scenario it was assumed that all compliance costs would be absorbed by firms in the form of reduced profits. Table V–3 contains a summary of this "worst case" analysis. Under this scenario, the estimated average percent reduction in profits for all affected firms was less than one percent. The estimated reduction in profit of 8 percent for SIC 24, Lumber and Wood Products Manufacturers, was the highest among all industries.

In the second scenario it was assumed that all compliance costs would be passed on to the consumer in the form of higher prices. The potential price increase for an industry sector at the two-digit SIC level was estimated by dividing the sector's compliance cost by its total sales. In this scenario, there would be little impact on market prices; none of the estimated price increases exceeded one half of one percent (Table V–4).

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Table V-3

ECONOMIC EFFECTS: NO-COST PASSTHROUGH SCENARIO[1]

SIC	Industry	Annual Costs[2] ($ millions)	Total Sales[3] ($ millions)	R.o.R. on Sales (%)[4]	Pre-Reg Profits ($ m)	Post-Reg Profits ($ m)	% Change in Profits
20	FOOD PROD.	33.49	353,780.38	1.9	8,008.04	7,986.29	- 0.2715
21	TOBACCO	0.02	74,030.13	5.3	3,923.60	3,923.59	- 0.0003
22	TEXT. MILL	29.48	60,735.22	2.7	1,765.42	1,747.59	- 1.0100
23	APPAREL PROD.	31.74	74,474.65	2.8	1,813.22	1,793.56	- 1.0845
24	LUMBER & WOOD	196.09	57,994.48	3.9	1,974.51	1,814.21	- 8.1188
25	FURNITURE	14.72	37,648.27	3.5	1,411.02	1,400.96	- 0.7129
26	PAPER PROD.	31.26	103,694.14	3.7	3,778.20	3,761.24	- 0.4489
27	PRINTING & PUB.	75.69	134,830.21	4.8	6,471.85	6,412.25	- 0.9210
28	CHEMICAL PROD.	40.16	272,759.67	3.7	11,738.80	11,714.76	- 0.2048
29	PETRO. REFINING	7.22	196,400.57	2.7	4,964.85	4,960.84	- 0.0808
30	RUBBER & PLASTICS	75.48	86,538.58	4.3	3,423.75	3,376.10	- 1.3918
31	LEATHER PROD.	2.58	15,449.56	2.6	401.69	399.95	- 0.4328
32	STONE & CLAY	22.54	46,094.04	4.1	1,954.99	1,940.73	- 0.7300
33	PRIMARY METALS	40.33	112,564.26	3.3	3,714.62	3,691.27	- 0.6286
34	FAB. METALS	55.94	150,146.41	4.0	6,005.86	5,974.10	- 0.5288
35	MACHINERY	57.74	345,144.89	5.1	17,602.39	17,566.95	- 0.2013
36	ELEC. MACH.	31.78	245,982.70	5.0	12,299.14	12,279.63	- 0.1586
37	TRANS. EQUIP.	47.10	365,427.20	3.9	14,520.25	14,486.69	- 0.2311
38	INSTRUMENTS	13.46	83,359.57	4.9	3,373.26	3,365.00	- 0.2450
39	MISC. MANUF.	18.20	41,870.30	4.4	1,788.56	1,777.39	- 0.6245
40	R.R. TRANS.	.53	43,869.14	10.0	3,969.62	3,969.34	- 0.0072
45	AIR TRANS.	1.83	109,538.08	3.6	3,251.40	3,250.41	- 0.0304
47	TRANS. SERVICES	1.85	12,254.96	2.7	582.18	581.18	- 0.1719
49	ELEC.,GAS & SAN.	22.69	300,254.83	7.0	21,017.84	21,004.06	- 0.0655
50	WHOLESALE TRADE[5]	4.05	13,853.52	2.0	277.07	273.67	- 1.2285
51	WHOLESALE, NON-DUR	8.86	113,848.20	1.5	1,726.26	1,721.48	- 0.2771
55	AUTO DEALERS	11.95	341,574.50	1.9	6,489.92	6,482.81	- 0.1095
72	PERSONAL SERV.	31.29	24,270.74	7.3	1,771.76	1,750.02	- 1.2272
73	BUSINESS SERV.	8.95	22,165.94	6.6	1,462.95	1,455.45	- 0.5126
75	AUTO REPAIR	1.51	45,750.92	5.1	2,492.19	2,491.05	- 0.0457
76	MISC. REPAIR SERV.	4.85	2,665.52	5.5	146.60	142.69	- 2.6696
80	HEALTH SERVICES	4.44	170,234.25	4.5	7,807.72	7,804.54	- 0.0406

Source: U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.

Notes:
1. All values in 1985 dollars.
2. Reproduced from Table VI-1.
3. Dun and Bradstreet, Dun's Marketing Identifiers (DMI) Database.
4. Rate of Return on Sales, Dun and Bradstreet, Industry Norms Database.
5. Consists of SIC 5093 (scrap and waste materials) only.

TABLE V-4

ECONOMIC EFFECTS: TOTAL-COST PASSTHROUGH

SIC	Industry	Annual Costs ($ millions)	Total Sales ($ millions)	Costs as a Percent of Sales
20	FOOD PROD.	33.49	353,780.38	0.0095
21	TOBACCO	0.02	74,030.13	0.0000
22	TEXT. MILL	29.48	60,735.22	0.0485
23	APPAREL PROD.	31.74	74,474.65	0.0426
24	LUMBER & WOOD	196.09	57,994.48	0.3381
25	FURNITURE	14.72	37,648.28	0.0391
26	PAPER PROD.	31.26	103,694.14	0.0301
27	PRINTING & PUB	75.69	134,830.21	0.0561
28	CHEMICAL PROD.	40.16	272,759.67	0.0147
29	PETRO. REFINING	7.22	196,400.57	0.0037
30	RUBBER & PLASTICS	75.48	86,538.58	0.0872
31	LEATHER PRODUCTS	2.58	15,449.56	0.0167
32	STONE & CLAY	22.54	46,094.04	0.0489
33	PRIM. METALS	40.33	112,564.26	0.0358
34	FAB. METALS	55.94	150,146.41	0.0373
35	MACHINERY	57.74	345,144.89	0.0167
36	ELEC. MACH.	31.78	245,982.70	0.0129
37	TRANS. EQUIP.	47.10	365,427.20	0.0129
38	INSTRUMENTS	13.46	83,359.57	0.0162
39	MISC. MANUF.	18.20	41,870.30	0.0435
40	R.R. TRANS.	.53	43,869.14	0.0012
45	AIR TRANS.	1.83	109,538.08	0.0017
47	TRANS. SERVICES	1.85	12,254.96	0.0151
49	ELEC,GAS & SAN.	22.69	300,254.83	0.0076
50	WHOLESALE TRADE[1]	4.05	13,853.52	0.0293
51	WHOLESALE, NON-DUR.	8.86	113,848.20	0.0078
55	AUTO DEALERS	11.95	341,574.50	0.0035
72	PERSONAL SERVICES	31.29	24,270.74	0.1289
73	BUSINESS SERVICES	8.95	22,165.94	0.0404
75	AUTO REPAIRS	1.51	45,750.92	0.0033
76	MISC. REPAIR SERV.	4.85	2,665.52	0.1819
80	HEALTH SERVICES	4.44	170,234.25	0.0026

Source: U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.

Notes: 1. Consists of SIC 5093 (scrap and waste materials) only.

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Based on this analysis, OSHA concludes that the proposed standard is economically feasible for each sector. The impact on prices is slight and even in the worst cases, the reductions in profitability are small.

Regulatory Flexibility Analysis

In accordance with the Regulatory Flexibility Act (Pub. L. 96-353, 94 Stat.1664 (5 U.S.C. 601 *et seq.*), OSHA has made a preliminary assessment of how the proposed rulemaking will affect large and small establishments. The results of this preliminary assessment indicate that some small establishments may experience some adverse impact. The smaller profit margins of some small establishments may make it more difficult for them to absorb increases in compliance costs. OSHA requests comments on approaches to reduce the impact on small establishments. An important ameliorating factor for each affected firm will be its ability to pass through additional costs to the consumer. The ability of individual firms to do this will be dependent upon product demand elasticities. It is expected that all impacted firms will be able to pass through some portion of their increased costs.

Environmental Impact

The proposed standard has been reviewed in accordance with the requirements of the National Environmental Policy Act of 1969 (NEPA), the Council on Environmental Quality NEPA regulations, and the Department of Labor's NEPA compliance procedures and is not anticipated to have a significant impact on the external environment.

TABLE V-5.—SIC GROUPS COVERED IN THE OSHA ANALYSIS

Division D. Manufacturing:
Major Group 20. Food and kindred products
Major Group 21. Tobacco manufactures
Major Group 22. Textile mill products
Major Group 23. Apparel and other finished products, made from fabrics and similar materials
Major Group 24. Lumber and wood products, except furniture
Major Group 25. Furniture
Major Group 26. Paper and allied products
Major Group 27. Printing, publishing, and allied industries
Major Group 28. Chemicals and allied products
Major Group 29. Petroleum refining and related industries
Major Group 30. Rubber and miscellaneous plastics products
Major Group 31. Leather and leather products
Major Group 32. Stone, clay, glass, and concrete products
Major Group 33. Primary metal industries
Major Group 34. Fabricated metal products, except machinery and transportation equipment
Major Group 35. Machinery, except electrical
Major Group 36. Electrical and electronic machinery, equipment, and supplies
Major Group 37. Transportation equipment
Major Group 38. Measuring, analyzing, and controlling instruments; photographic, medical and optical goods; watches and clocks
Major Group 39. Miscellaneous manufacturing industries
Division E. Transportation, Communications, Electric, Gas, and Sanitary Services:
Major Group 40. Railroad transportation
Major Group 44. Water transportation
Major Group 45. Transportation by air
Major Group 47. Transportation services
Major Group 49. Electric, gas, and sanitary services
Division F. Wholeale Trade:
Major Group 50. Wholesale trade—durable goods
Major Group 51. Wholesale trade—nondurable goods
Division G. Retail Trade: Major Group 55. Automotive dealers and gasoline service stations
Division I. Services:
Major Group 72. Personal services
Major Group 73. Business services
Major Group 75. Automotive repair, services, and garages
Major Group 80. Health services

Source: U.S. Department of Labor, OSHA, Office of Regulatory Analysis, as derived from Standard Industrial Classification Manual 1972, Executive Office of the President—Office of Management and Budget (1, pp. 5-7).

The listing excludes the construction industry (SICs 15, 16, and 17) which will be the subject of a separate regulatory analysis.

VI. Clearance of Information Collection Requirements

On March 31, 1983, the Office of Management and Budget (OMB) published a new 5 CFR Part 1320, implementing the information collection provisions of the Paperwork Reduction Act of 1980, 44 U.S.C. 3501 *et seq.* (48 FR 13666). Part 1320, which became effective on April 30, 1983, sets forth procedures for agencies to follow in obtaining OMB clearance not later than the date of publication of the proposal in the **Federal Register** for collection of information requirements contained in proposed rules. It also requires agencies to include a statement in the notice of proposed rulemaking indicating that such information requirements have been submitted to OMB for review under section 3504(h) of the Paperwork Reduction Act.

In addition to the above requirements, applicable federal regulations also provide, 5 CFR 1320 4(a), 1320.5(a), and 1320.5(d), respectively, as follows:

> An agency shall not engage in a collection of information without obtaining Office of Management and Budget (OMB) approval of the collection of information and displaying a currently valid control number and, unless OMB determines it to be inappropriate, an expiration date. * * *
>
> Notwithstanding any other provision of law, no person shall be subject to any penalty for failure to comply with any information collection request if the request does not display a currently valid OMB control number, or, in the case of an information collection request which is submitted to nine or fewer persons, the request fails to state that for this reason it is not subject to OMB review under the Act.* * *
>
> Whenever a member of the public is protected from imposition of a penalty under this section for failure to comply with a collection of information, such penalty may not be imposed by an agency directly, by an agency through judicial process, or by any other person through judicial or administrative process.* * *

The proposed PELs update standard will create no additional recordkeeping requirements.

In accordance with the provisions of the Paperwork Reduction Act and the regulations issued pursuant thereto, OSHA certifies that it will submit the information collection requirements contained in its proposed update of the air contaminants rule on to OMB for review under section 3504(h) of that Act. Comments on these information collection requirements may be submitted by interested persons to the Office of Information and Regulatory Affairs of OMB, Attention: Desk Officer for OSHA.

VII. Summary and Explanation of the Proposed Standard.

Table Z-4 in the standard includes proposed new exposure limits for 428 substances. These include approximately 220 substances for which OSHA has exposure limits and approximately 205 substances for which OSHA has not specified exposure limits prior to this proposal.

OSHA is republishing Tables Z-1, Z-2, and Z-3 which include most existing

OSHA exposure limits. Asterisks have been placed next to the substances which OSHA proposes to remove from those tables. All these substances have been incorporated in Table Z–4 with new exposure limits. Table Z–4 lists the proposed permissible exposure limits for 428 substances. This includes the eleven substances for which increased PELs were considered and two substances for which deleting skin notations were considered, but for which no changes were made. Table VII–C lists 44 substances in which 2 alternate permissible exposure limits, based on the NIOSH–PELs and ACGIH TLVs, were evaluated. Part I–D of the preamble explains the basis for the choice of these two data bases. The OSHA recommended PEL is underlined in this Table. For most substances noted as changes in Table Z–4 the TWA limit is either lower or remains the same but there is a change, or there is an addition or deletion of a STEL or Ceiling limit. For one substance, the TWA has been raised.

The policy reasons for this proposal are explained in Part I of the preamble. The health basis of the new exposure limits are explained in Part IV. The feasibility analysis for the proposal is summarized in part V.

OSHA's preliminary conclusion that the proposed limits noted in Table Z–4 substantially reduce significant risk and are feasible, is based on the analysis in those parts.

Initially, OSHA is proposing these new limits for general industry. Additional consideration and consultation is needed to determine their applicability to other sectors (e.g., construction, maritime, and agriculture). To attempt to consider these sectors in this rulemaking would delay this important proceeding. OSHA is commencing that consultation for the construction sector. See Part I–H of the preamble for further discussion of this subject.

As a matter of form, the final regulation may delete those substances in the Table Z–4 from Tables Z–1, Z–2, and Z–3 rather than republishing them with asterisks. However, Part 1917, Marine Terminals, references the existing Tables Z–1, Z–2 and Z–3 which OSHA is not proposing to change in this proceeding. Therefore, OSHA may publish the final rule in the same format as the proposal so that there will be published current CFR exposure limits for Marine Terminals. In that case the asterisk will refer to limits only applicable to marine terminals. Suitable changes will be made in the language of 29 CFR 1910.1000 to indicate this. An alternative would be to republish the existing Tables Z–1, Z–2 and Z–3 in Part 1917 until such time as a proceeding to update them is completed.

OSHA is adding a new paragraph (d) to § 1910.1000. The old paragraph (d) is redesignated paragraph (f). That paragraph states that employers must not expose employees over the limits specified in Table Z–4. It defines TWA, STEL and Ceiling limits and indicates skin absorption should be prevented for substances so identified in Table Z–4.

OSHA is not considering any changes to paragraphs (a), (b), (c) and (e) in this rulemaking. Certain format changes have been made to redesignated paragraph (f) to clarify that the formulas for multiple exposure applies to substances listed in Table Z–4, substances specified standards listed in Sections 1910.1001–1047 as well as Tables Z–1, Z–2 and Z–3. OSHA interprets the existing language as having the same intent. OSHA is not reopening consideration of the formulas in redesignating paragraph (f).

A slight format change is also made in the introductory text of § 1910.1000 to reflect that Table Z–4 has been added.

The term "material" was used in the standard; it is equivalent to the term "substance" as used in the preamble.

The standard provides an explanation of skin notation and definitions of the following terms used in Table Z–4: Time Weighted Average (TWA). Short Term Exposure Limit (STEL) and Ceiling.

The standard states in paragraph 3.(2):

(2). An employee's skin exposure to materials listed in table Z–4 with an "S" Notation shall be limited through the use of gloves, coveralls, goggles, or other appropriate personal protection equipment or method necessary to prevent possible skin absorption.

The skin notation is used where the substance may be absorbed through the skin. It also may be used where skin contact could damage or irritate the skin. This sub-paragraph indicates for those substances, methods must be used to limit skin exposure. As the language indicates it may be limited through use of appropriate personal protection equipment. Appropriate engineering controls or work practices may also be used. No specific order of priority is stated.

The standard states in paragraph 3.(3):

(3). The following definitions apply to paragraph (d) of this section and Table Z–4:

(i) Time weighted average (TWA) is the average airborne exposure of an employee in any 8-hour work shift of a 40-hour work week which shall not be exceeded.

(ii) Short term exposure limit (STEL) is the employee's 15-minute time-weighted average exposure which shall not be exceeded at any time during a work day. This applies even if the eight hour time-weighted average is within the TWA.

(iii) Ceiling is the employee's exposure which shall not be exceeded during any part of the work day. If instantaneous monitoring is not feasible, then the ceiling shall be assessed by sampling over a 15-minute period as would be done for a STEL, unless a different time period is specifically indicated.

OSHA intends that the effective date of new exposure limits issued in final form based on this proposal shall be 90 days after the date of publication in the Federal Register. This is set forth in section 6(b)(4) of the OSH Act.

In addition, OSHA has set forth start-up dates for most of its health standards. It takes time for employers to evaluate exposures and purchase, install and make operable equipment to control such exposures.

In the case of this proposed standard, start-up dates need to be sufficient to take into account the fact that many employers will have to evaluate and make operable controls for several different chemicals. This may require more time than would be necessary for only one chemical.

OSHA believes that 6 months from the date of publication is a reasonable time to evaluate exposures and come into compliance with any combination of respirators, work practices and engineering controls. OSHA standards generally have had a period of approximately this length or shorter where compliance with an exposure limit was to be achieved with any suitable combination of controls. See, for example, the benzene standard, 29 CFR 1910.1028(m)(2), 52 FR 34460, 345676 (September 11, 1987) and the formaldehyde standard, 29 CFR 1910.1048(p)(2)(iv), 52 FR 46168, 46296 (December 4, 1987). OSHA experience is that the 6 month period is appropriate and sufficient to come into compliance with any combination of controls. Comment is requested.

OSHA has also generally provided a more extended period to come into compliance using the hierarchy of controls contained in 29 CFR 1910.1000(e), with its preference for engineering and work practice controls. It takes more time, in general, to plan, purchase equipment, install and make operational engineering controls than to implement other types of control strategies. Examples of representative phase-in periods include 1 to 10 years (depending on the sector) for the lead standard, 29 CFR 1910.1025(e), 4 years for the cotton dust standard, 29 CFR 1910.1043(m), 2 years for the benzene standard, 29 CFR 1910.1028(m)(2)(ii) and 14 months for the formaldehyde

standard, 29 CFR 1910.1048(p)(2)(v). These dates have varied depending upon OSHA estimates of the difficulties involved. OSHA's experience has been that generally the times proposed here have been sufficient. See the detailed analysis reported in the cotton dust standard, 50 FR 51164, December 13, 1985.

In the case of this proposal, OSHA estimates that compliance can be reasonably achieved by all employers, including those who would have to control exposures for several different chemicals, within 4 years, using the present hierarchy of controls. OSHA requests comment on this estimate.

OSHA will be shortly publishing a Federal Register notice requesting public comment on the hierarchy of controls currently contained in 29 CFR 1910.1000(e). Based on comment, evidence and data received during the course of that rulemaking, it is possible that changes in the hierarchy may be made. If more flexibility were to be permitted in the use of engineering controls, a shorter period to come into compliance with the new exposure limits might be appropriate. On the other hand, during the period of uncertainty about the outcome of the methods of compliance rulemaking, employers seeking to comply with the new exposure limits may install controls under the existing hierarchy that may turn out later not to be required. Accordingly, it may be appropriate to set a start-up time for installing engineering controls that is contingent on the completion date of the methods of compliance rulemaking.

OSHA requests public comment on the approach it should follow and the period it should set for coming into compliance under the hierarchy of controls. OSHA is not requesting comment in this rulemaking on what the hierarchy of controls should be.

For some substances OSHA is tentatively using the 10-hour TWA given in the NIOSH-RELs as a proposed PEL. It should be noted that NIOSH generally refers in its criteria document for airborne concentration of a substance as a "time-weighted average (TWA) exposure for up to a 10-hour work shift in a 40-hour work week." OSHA preliminarily concludes that this is equivalent to the OSHA definition of an 8-hour work shift for a 40-hour work week. OSHA requests comments on this approach.

NIOSH REL ceiling values are based on time intervals which range from instantaneous to 120 minutes. Most of these REL ceilings have been established for a 15-minute period. OSHA proposes that PELs based on REL ceilings of 10, 15, and 20 minutes all be considered as 15-minute PELs in order to achieve greater uniformity and simplicity in the standards. OSHA is also considering treating the 30-minute, 60-minute and 120-minute ceilings in the same way. OSHA invites comments on these two approaches.

OSHA also recognizes that ceiling limits as defined by the ACGIH-TLV committee may represent a permissible exposure based on instantaneous measurement, or an exposure over a 15-minute period, if instantaneous monitoring is not feasible. The ACGIH-TLV committee defined a STEL as a 15-minute measurement. Therefore, some of the ceiling limits can be equivalent to STELs. OSHA will consider adopting the 15-minute period for many of the proposed PELs based on TLV ceiling values in order to achieve greater uniformity and simplicity in the standard. OSHA invites comments on this modification of the definition of a TLV ceiling, and the possible use of two alternate monitoring procedures (instantaneous and 15-minute) for substances having a PEL ceiling.

It should be noted that OSHA is not proposing to change exposure limits for the 24 substances regulated in §§ 1910.1000 to 1910.1047. These limits have previously been issued in section 6(b) rulemakings where their health effects and feasibility have been fully considered. Table VII-A lists those substances. Some of those substances remain listed unchanged in Tables Z-1, Z-2 and Z-3 as well. Those listings apply in limited particular circumstances specified by footnotes.

OSHA is also not proposing to change exposure limits for 9 other substances on Tables Z-1, Z-2 and Z-3 for which ACGIH or NIOSH have recommended different exposure limits. OSHA plans to regulate those substances (such as cadmium) in the near future in individual section 6(b) rulemakings because of the importance of the substance, or the need to fully evaluate the substance individually in determining the level to be set. Those substances are listed in Section VII, Table VII-B.

OSHA is also *not* proposing to change the limits for 160 substances for which the OSHA PEL's and the 1987-88 ACGIH TLV's are identical. Table VII-D lists these substances.

To summarize other sections of the preamble, OSHA reviewed some fourteen (14) data bases in developing an approach to updating the permissible exposure limits (PELs). These data bases included those developed by professional organizations, private-sector corporations, international bodies, governments and government agencies. In making a preliminary selection, the sources of standards were evaluated for the following criteria: Comprehensiveness, currentness, process setting procedures, feasibility, review process, applicability and documentation for each substance.

OSHA has preliminarily concluded that ACGIH-TLV is the most extensive source when compared to a number of other sources, and that it satisfies all the additional criteria established for this effort. OSHA has also preliminarily concluded that NIOSH's recommended exposure limits, while less extensive, should also be considered in the proposal. Thus, the ACGIH-TLVs and NIOSH-RELs will be discussed in order to present the OSHA basis for setting the new proposed PELs.

The ACGIH-TLV's are the most comprehensive of all the examined lists. Currently, over 600 substances are covered and TWA's, STELs, ceilings and skin notations are included where appropriate. The limits developed by the ACGIH TLV committee are applicable to U.S. industry conditions, and feasibility is generally considered.

The ACGIH-TLV process includes multiple professional review, and documentation is included for every recommendation. This documentation includes a discussion of the toxicological data underlying the limit, a description of the exposure limits set for the chemical by NIOSH and other sources, and a statement of the rationale for selecting a particular limit. In some cases, the ACGIH-TLV documentation lacks optimal depth of detail, but in all cases it is possible to determine what AGCIH judges to be the most directly relevant toxicological data. While some documentation is anecdotal or contained in personal communication, this is generally only one component of the total basis for recommending the TLVs. Recently, Castleman and Ziem (1987-1988) and Samuels (1987) have been critical of the TLV's committee's procedures and documentation because a number of references have been based on personal communication and they believe industry representatives on the TLV Committee have had too much influence. It should be noted that voting members of ACGIH must be employed by governments or educational institutions. Industry employees do not vote.

On the basis of all evidence, OSHA makes a preliminary conclusion that the TLVs established by the ACGIH constitute the best available *starting point* for revisions of existing PEL's. This establishes the *bounds* of the substances

to be considered in this proposal as those substances where the 1987–88 TLVs differ from the existing OSHA PELs.

OSHA will also consider the NIOSH–REL's in setting new PEL's in this rulemaking. The NIOSH–RELs are well documented and their RELs are set with U.S. workplaces in mind. NIOSH explains the basis for its limits in criteria documents or in Current Intgelligence Bulletins.

OSHA has considered more than 160 NIOSH–RELs and classified the RELs according to the following preliminary assumptions:

1. OSHA has excluded RELs for substances for which the OSHA comprehensive standards exist. The comprehensive standards had been previously reviewed and analyzed under 6(b) rulemaking process (Tab1e VII–A.).

2. OSHA has excluded RELs for substances which are in various stages of OSHA rulemaking (Table VII–B).

3. OSHA has identified 9 substances for which the existing OSHA–PELs, NIOSH RELs, and ACGIH TLVs are identical. These substances are exempted from the new proposal and are included in Tables Z–1, Z–2, and Z–3 without the asterisk. The 9 substances are: Antimony and compounds, benzoyl peroxide, carbaryl, carbon black, diacetone alcohol, dinitrocresol, fluoride, nitrioc oxide and sulfuric acid.

4. OSHA has identified 11 substances for which the RELs and the TLVs are different, but the TLV and PEL are the same. OSHA has also identified 19 substances which have RELs, but have no corresponding TLVs or PELs. OSHA has preliminarily decided to exclude these 30 substances from this standard. This is in line with OSHA's discussion in Section I–D which limits this proposal to those substances where 1987–88 ACGIH TLVs exist and differ from the existing PEL.

5. OSHA has identified 8 substances for which the RELs equal the TLVs. They are listed in Table Z–4 as proposed PELs and are identified by footnote C.

6. OSHA has identified 44 substances for which significant differences between the RELs and the TLVs exist. Table VII–C lists 44 substances in which two alternate permissible exposure limits are proposed, based on the NIOSH–RELs and the ACGIH–TLVs. OSHA identified 35 substances for which the RELs are lower than the TLVs (Table I–F–A) and 9 substances in which the RELs are higher than TLVs (Table I–F–B). These were evaluated on an individual substance basis and the proposed PEL is listed in Table Z–4. The OSHA proposed PEL is also underlined in Table VII–C.

7. OSHA has identified 15 substances for which the TWA for the RELs and the TLVs are identical, but differences exist in the STEL or Ceiling limit. OSHA has preliminarily concluded that this is a minor difference and therefore is using the TLV limits which are included in Table Z–4. This preliminary approach is based on the fact that the existing PEL's are based on 1967–68 TLV's, and the approach used in this proposal should follow that baseline unless significant differences exist.

8. OSHA has identified 16 substances for which the TWA TLV or TWA REL equals the STEL/Ceiling; the TWA, TLV or REL equals ½ STEL; or the TWA, TLV or REL are within 10%. OSHA has preliminarily concluded that these are minor differences and has selected the available TWA limit. The 16 substances in this group are included in Table Z–4.

OSHA has also identified a number of substances which require special attention because of differences in the allowable exposure guidelines compared to the other sets of limits reviewed or other special circumstances. OSHA has identified several alternatives for dealing with these situations.

For its discussion of health effects OSHA has preliminarily grouped each substance on the basis of the TLV documentation. The substances are divided into 15 generic groups. These are: Neurophatic effects, narcotic effects, sensory irritants, liver and kidney effects, occular effects, adverse respiratory effects, cardiovascular effects, systemic effects, no observed effects, nuisance potential, odor and taste effects, analogy, biochemical and metabolic effects, sensitizers, and carcinogenic effects. The OSHA analysis also considered three special categories concerned with: Change only to the STEL; change regarding skin designation in the TLV; and situations where the TLV is greater than the existing PEL.

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TABLE VII A. Substances Regulated by OSHA Under Section 6(b)

CHEMICAL NAME	STANDARD
2-Acetylaminofluorine	1910.1014
Acrylonitrile	1910.1045
4-Aminodiphenyl	1910.1011
Arsenic (Inorganic)	1910.1018
Asbestos	1910.1001
Benzene	1910.1028
Benzidine	1910.1010
Bis-Chloromethyl Ether	1910.1008
Coke Oven Emissions	1910.1029
Cotton Dust	1910.1043
1,2 Dibromo 3 Dichloropropane	1910.1049
3,3' Dichlorobenzidine	1910.1007

TABLE VII A. Substances Regulated by OSHA Under Section 6(b)

CHEMICAL NAME	STANDARD
4 Dimethylaminoazobenzene	1910.1015
Ethylene Oxide	1910.1047
Ethyleneimine	1910.1012
Formaldehyde	1910.1046
Lead	1910.1025
Methyl Chloromethyl Ether	1910.1006
2 Naphthylamine	1910.1004
3 Naphthylamine	1910 1009
4 Nitrobiphenyl	1910.1003
n Nitrosodimethylamine	1910.1016
b Propiolactone	1910.1013
Vinyl Chloride	1910 1017

TABLE VII B. Substances for Which OSHA Has Initiated 6(b) Rulemaking

CHEMICAL NAME	STANDARD
1,3 Butadiene	
Cadmium Dust and Fume	
2 Ethoxyethanol (Cellosolve)	
2 Ethoxyethyl Acetate	
Ethylene Dibromide	
Methyl Cellosolve	
Methyl Cellosolve Acetate	
Methylene Chloride	
4,4' Methylenedianiline	

TABLE VII-C. Substances for Which OSHA Considered Proposing Either the ACGIH or NIOSH Limit*

H.S. No.	Chemical Name	CAS No.	ACGIH TLV[R]** TWA	ACGIH TLV[R]** STEL	ACGIH TLV[R]** Ceiling	NIOSH REL*** TWA	NIOSH REL*** Ceiling
1004	Acetone	67-64-1	750 ppm	1000 ppm	--	250 ppm	--
1005	Acetonitrile	75-05-8	40 ppm	60 ppm	--	20 ppm	--
1008	Acrylamide	79-06-1	0.03 mg/m^3, Skin	--	--	0.3 mg/m^3	--
1021	Ammonia	7664-41-7	25 ppm	35 ppm	--	--	50 ppm (5 min)
1033	Beryllium and Compounds[+]	7440-41-7	0.002 mg/m^3	--	--	--	0.0005 mg/m^3
1052	n-Butyl Glycidyl Ether	2426-08-6	25 ppm	-	--	--	5.6 ppm (15 min)
1069	Carbon Dioxide	124-38-9	5000 ppm	30,000 ppm	--	10,000 ppm	30,000 ppm (10 min)
1070	Carbon Disulfide	75-15-0	10 ppm	--	--	1 ppm	10 ppm (15 min)

TABLE VII-C. Substances for Which OSHA Considered Proposing Either the ACGIH or NIOSH Limit* (Continued)

H.S. No.	Chemical Name	CAS No.	ACGIH TLV[R]** TWA	ACGIH TLV[R]** STEL	ACGIH TLV[R]** Ceiling	NIOSH REL*** TWA	NIOSH REL*** Ceiling
1071	Carbon Monoxide	630-08-0	50 ppm	400 ppm	—	35 ppm	200 ppm (no defined time)
1073	Carbon Tetrachloride	56-23-5	5 ppm, Skin	--		--	2 ppm (60 min)
1079	Chlorine	7782-50-5	1 ppm	3 ppm	--	--	0.5 ppm (15 min)
1086	Chloroform	67-66-3	10 ppm	--	--	—	2 ppm (60 min)
1088	Chloroprene	126-099-8	10 ppm, Skin	--	--	--	1 ppm (15 min)
1192	Chromic Acid and Chromates+ (Non-carcinogenic)	7738-94-5	0.05 mg/m^3	--	—	0.025 mg/m^3	—
1192	Chromic Acid and Chromates+ (Carcinogenic)	7738-94-5	0.05 mg/m^3	—	--	0.001 mg/m^3	—

TABLE VII-C. Substances for Which OSHA Considered Proposing Either the ACGIH or NIOSH Limit* (Continued)

H.S. No.	Chemical Name	CAS No.	ACGIH TLV[R]** TWA	ACGIH TLV[R]** STEL	ACGIH TLV[R]** Ceiling	NIOSH REL*** TWA	NIOSH REL*** Ceiling
1145	Dioxane	123-91-1	25 ppm, Skin	--	--	--	1 ppm (30 min)
1168	Ethylene Dichloride	107-06-2	10 ppm	--	--	1 ppm	2 ppm (15 min)
1170	Ethylene Glycol Dinitrate	628-96-6	0.05 ppm, Skin	--	--	--	$0.1\ mg/m^3$ (20 min)
1178	Fibrous Glass Dust	--	$10\ mg/m^3$	--	--	$5\ mg/m^3$	--
1184	Furfuryl Alcohol	98-00-0	10 ppm, Skin	15 ppm	--	50 ppm	--
1194	n-Heptane	142-82-5	400 ppm	500 ppm	--	85 ppm	440 ppm (15 min)
1200	n-Hexane	110-54-3	50 ppm	--	--	100 ppm	510 ppm (15 min)
1201	Hexane Isomers	--	500 ppm	1000 ppm	--	100 ppm	510 ppm (15 min)

TABLE VII-C. Substances for Which OSHA Considered Proposing Either the ACGIH or NIOSH Limit* (Continued)

H.S. No.	Chemical Name	CAS No.	ACGIH TLV[R]** TWA	ACGIH TLV[R]** STEL	ACGIH TLV[R]** Ceiling	NIOSH REL*** TWA	NIOSH REL*** Ceiling
1202	2-Hexanone	591-78-6	5 ppm	--	--	1 ppm	--
1205	Hydrazine	302-01-2	0.1 ppm, Skin	-	--	--	0.03 ppm (120 min)
1207	Hydrogen Cyanide	74-90-8	--	--	10 ppm, Skin	--	4.7 ppm (10 min)
1222	Isophorone Diisocyanate	4098-71-9	0.09 mg/m^3	--	--	0.045 mg/m^3	0.18 mg/m^3 (10 min)
1235	Malathion	121-75-5	10 mg/m^3, Skin	--	--	15 mg/m^3	--
1240	Mercury (Aryl and Inorganic Compounds)+	7439-97-6	0.1 mg/m^3, Skin	--	--	0.05 mg/m^3	--
1243	Mesityl Oxide	141-79-7	15 ppm	25 ppm	--	10 ppm	--

TABLE VII-C. Substances for Which OSHA Considered Proposing Either the ACGIH or NIOSH Limit* (Continued)

H.S. No.	Chemical Name	CAS No.	ACGIH TLV^{R}** TWA	ACGIH TLV^{R}** STEL	ACGIH TLV^{R}** Ceiling	NIOSH REL*** TWA	NIOSH REL*** Ceiling
1264	Methyl n-Amyl Ketone+	110-43-0	50 ppm	--	--	100 ppm	—
1283	Nickel (Soluble Compounds)	7440-02-0	0.1 mg/m^3	--	--	0.015 mg/m^3	—
1289	Nitrogen Dioxide	10102-44-0	3 ppm	5 ppm	--	—	1 ppm (15 min)
1290	Nitroglycerin	55-63-0	0.5 mg/m^3, Skin	--	--	—	0.1 mg/m^3 (20 min)
1296	Octane	111-65-9	300 ppm	375 ppm	—	75 ppm	385 ppm (15 min)
1306	Pentane	109-66-0	600 ppm	750 ppm	--	120 ppm	610 ppm (15 min)
1307	2-Pentanone (Methylpropyl ketone)	107-89-9	200 ppm	250 ppm	--	150 ppm	--

TABLE VII-C. Substances for Which OSHA Considered Proposing Either the ACGIH or NIOSH Limit* (Continued)

H.S. No.	Chemical Name	CAS No.	ACGIH TLV^{R}** TWA	ACGIH TLV^{R}** STEL	ACGIH TLV^{R}** Ceiling	NIOSH REL*** TWA	NIOSH REL*** Ceiling
1312	Petroleum Distillates (Naphtha)	--	1600 mg/m^3	--	--	350 mg/m^3	1800 mg/m^3 (15 min)
1317	Phenylhydrazine	100-63-0	5 ppm, Skin	10 ppm	--	--	0.14 ppm (120 min)
1355	Silica, Crystalline Quartz, Respirable	14808-60-7	0.1 mg/m^3	--	--	0.05 mg/m^3	--
1371	Stoddard Solvent	8052-41-3	100 ppm (525 mg/m^3)	--	--	350 mg/m^3	1800 mg/m^3 (15 min)
1375	Sulfur Dioxide	7446-09-5	2 ppm	5 ppm	--	0.5 ppm	--

TABLE VII-C. Substances for Which OSHA Considered Proposing Either the ACGIH or NIOSH Limit* (Continued)

H.S. No.	Chemical Name	CAS No.	ACGIH TLV^{R}**			NIOSH REL***	
			TWA	STEL	Ceiling	TWA	Ceiling
1406	Trichloroethylene	79-01-6	50 ppm	200 ppm	--	<u>25 ppm</u>	—
1424	Vinyl Acetate	108-05-4	<u>10 ppm</u>	<u>20 ppm</u>	--	—	4 ppm (15 min)
1436	Zinc Chromates (CrVI)+	13530-65-9	0.05 mg/m^3	--	—	0.001 mg/m^3	—

* Underlined values indicate the limits being proposed by OSHA.

** The ACGIH TWA-TLV is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

*** NIOSH TWA limits are for 10-hour exposures unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time unless a duration is specified in parentheses.

+ OSHA is not proposing to revise its current limit at the present time.

TABLE VII-D. Substances for Which OSHA Did Not Consider Changing Its Current Limits

CHEMICAL NAME	8-HOUR TWA		15-MINUTE STEL		CEILING		SKIN
	PPM	MG/M3	PPM	MG/M3	PPM	MG/M3	
ACETYLENE TETRABROMIDE	1	-	-	-	-	-	-
ALDRIN	-	0.25	-	-	-	-	S
2-AMINOPYRIDINE	0.5	-	-	-	-	-	-
N-AMYL ACETATE	100	-	-	-	-	-	-
SEC-AMYL ACETATE	125	-	-	-	-	-	-
ANISIDINE (O,P ISOMERS)	-	0.5	-	-	-	-	S
ANTIMONY & COMPOUNDS	-	0.5	-	-	-	-	-
ANTU	-	0.3	-	-	-	-	-
ARSINE	0.05	-	-	-	-	-	-
ARSENIC, ORGANIC COMPOUNDS	0.5	-	-	-	-	-	-
AZINPHOS-METHYL	-	0.2	-	-	-	-	S
BARIUM (SOLUBLE COMPOUNDS)	-	0.5	-	-	-	-	-
BENZOYL PEROXIDE	-	5	-	-	-	-	-
BENZYL CHLORIDE	1	-	-	-	-	-	-
BIPHENYL (DIPHENYL)	0.2	-	-	-	-	-	-
BORON TRIFLUORIDE	-	-	-	-	1	-	-
BROMOFORM	0.5	-	-	-	-	-	S
SEC-BUTYL ACETATE	200	-	-	-	-	-	-
TERT-BUTYL ACETATE	200	-	-	-	-	-	-
TERT-BUTYL CHROMATE	-	-	-	-	-	0.1	S
BUTYLAMINE	-	-	-	-	5	-	S

TABLE VII-D. Substances for Which OSHA Did Not Consider Changing Its Current Limits (Continued)

CHEMICAL NAME	8-HOUR TWA		15-MINUTE STEL		CEILING		SKIN
	PPM	MG/M3	PPM	MG/M3	PPM	MG/M3	
CARBARYL (SEVIN)	-	5	-	-	-	-	-
CARBON BLACK	-	3.5	-	-	-	-	-
CHLORDANE	-	0.5	-	-	-	-	S
CHLORINATED DIPHENYL OXIDE	-	0.5	-	-	-	-	-
CHLORINE TRIFLUORIDE	-	-	-	-	0.1	-	-
CHLOROACETALDEHYDE	-	-	-	-	1	-	-
ALPHA-CHLOROACETOPHENONE	0.05	-	-	-	-	-	-
CHLOROBENZENE	75	-	-	-	-	-	-
CHLOROBROMOMETHANE	200	-	-	-	-	-	-
CHLORODIPHENYL 42% (AROCLOR 1242)	-	1	-	-	-	-	S
CHLORODIPHENYL 54% (AROCLOR 1254)	-	0.5	-	-	-	-	S
CHLOROPICRIN	0.1	-	-	-	-	-	-
CHROMIUM, SOL.CHROMIC, CHROMOUS	-	0.5	-	-	-	-	-
COAL TAR PITCH VOLATILES	-	0.2	-	-	-	-	-
COPPER (DUSTS & MISTS)	-	1	-	-	-	-	-
CRESOL (ALL ISOMERS)	5	-	-	-	-	-	S
CROTONALDEHYDE	2	-	-	-	-	-	-
CUMENE	50	-	-	-	-	-	S
CYANIDES	-	5	-	-	-	-	S
CYCLOHEXANE	300	-	-	-	-	-	-
CYCLOHEXENE	300	-	-	-	-	-	-

TABLE VII-D. Substances for Which OSHA Did Not Consider Changing Its Current Limits (Continued)

CHEMICAL NAME	8-HOUR TWA		15-MINUTE STEL		CEILING		SKIN
	PPM	MG/M3	PPM	MG/M3	PPM	MG/M3	
CYCLOPENTADIENE	75	-	-	-	-	-	-
DEMETON	-	0.1	-	-	-	-	S
2,4-D (2,4-DICHLOROPHENOXYACETIC ACID)	-	10	-	-	-	-	-
DDVP (DICHLORVOS)	-	1	-	-	-	-	S
DIACETONE ALCOHOL	50	-	-	-	-	-	-
DIAZOMETHANE	0.2	-	-	-	-	-	-
DIBORANE	0.1	-	-	-	-	-	-
DIBUTYLPHTHALATE	-	5	-	-	-	-	-
O-DICHLOROBENZENE	-	-	-	-	50	300	-
DICHLORODIFLUOROMETHANE	1000	-	-	-	-	-	-
1,2-DICHLOROETHYLENE	200	-	-	-	-	-	-
DICHLOROTETRAFLUOROETHANE	1000	-	-	-	-	-	-
DIELDRIN	-	0.25	-	-	-	-	S
DIETHYLAMINOETHANOL	10	-	-	-	-	-	S
DIFLUORODIBROMOMETHANE	100	-	-	-	-	-	-
DIISOPROPYLAMINE	5	-	-	-	-	-	S
DIMETHYL ACETAMIDE	10	-	-	-	-	-	S
DIMETHYLAMINE	10	-	-	-	-	-	-
DIMETHYLFORMAMIDE	10	-	-	-	-	-	S
1,1-DIMETHYLHYDRAZINE	0.5	-	-	-	-	-	S

TABLE VII-D. Substances for Which OSHA Did Not Consider Changing Its Current Limits (Continued)

CHEMICAL NAME	8-HOUR TWA		15-MINUTE STEL		CEILING		SKIN
	PPM	MG/M3	PPM	MG/M3	PPM	MG/M3	
DIMETHYLPHTHALATE	-	5	-	-	-	-	-
DINITRO-O-CRESOL	-	0.2	-	-	-	-	S
DINITROBENZENE (ALL ISOMERS)	-	1	-	-	-	-	S
DINITROTOLUENE	-	1.5	-	-	-	-	S
ENDRIN	-	0.1	-	-	-	-	S
EPN	-	0.5	-	-	-	-	S
ETHYL ACETATE	400	-	-	-	-	-	-
ETHYL ALCOHOL	1000	-	-	-	-	-	-
ETHYL BUTYL KETONE	50	-	-	-	-	-	-
ETHYL CHLORIDE	1000	-	-	-	-	-	-
ETHYL FORMATE	100	-	-	-	-	-	-
ETHYL SEC-AMYL KETONE	25	-	-	-	-	-	-
ETHYLAMINE	10	-	-	-	-	-	-
ETHYLENEDIAMINE	10	-	-	-	-	-	-
FLUORIDE (AS F)	-	2.5	-	-	-	-	-
FORMIC ACID	5	-	-	-	-	-	-
HAFNIUM	-	0.5	-	-	-	-	-
HEPTACHLOR	-	0.5	-	-	-	-	S
HEXACHLORONAPHTHALENE	-	0.2	-	-	-	-	S
SEC-HEXYL ACETATE	50	-	-	-	-	-	-
HYDROGEN CHLORIDE	-	-	-	-	5	-	-

TABLE VII-D. Substances for Which OSHA Did Not Consider Changing Its Current Limits (Continued)

CHEMICAL NAME	8-HOUR TWA		15-MINUTE STEL		CEILING		SKIN
	PPM	MG/M3	PPM	MG/M3	PPM	MG/M3	
HYDROGEN PEROXIDE (90%)	1	-	-	-	-	-	-
HYDROGEN SELENIDE	0.05	-	-	-	-	-	-
HYDROQUINONE	-	2	-	-	-	-	-
IODINE	-	-	-	-	0.1	-	-
ISOAMYL ACETATE	100	-	-	-	-	-	-
ISOBUTYL ACETATE	150	-	-	-	-	-	-
L.P.G.	1000	-	-	-	-	-	-
LINDANE	-	0.5	-	-	-	-	S
LITHIUM HYDRIDE	-	0.025	-	-	-	-	-
MALEIC ANHYDRIDE	0.25	-	-	-	-	-	-
MANGANESE DUST AND COMPOUNDS	-	-	-	-	-	5	-
METHYL ACETYLENE (PROPYNE)	1000	-	-	-	-	-	-
METHYL ACRYLATE	10	-	-	-	-	-	S
METHYL ISOCYANATE	0.02	-	-	-	-	-	S
METHYL METHACRYLATE	100	-	-	-	-	-	-
METHYLAL(DIMETHOXYMETHANE)	1000	-	-	-	-	-	-
METHYLENE BISPHENYL ISOCYANATE (MDI)	-	-	-	-	0.02	0.2	-
METHYLAMINE	10	-	-	-	-	-	-
MOLYBDENUM (SOLUBLE COMPOUNDS)	-	5	-	-	-	-	-
MONOMETHYL HYDRAZINE	-	-	-	-	0.2	-	S

TABLE VII-D. Substances for Which OSHA Did Not Consider Changing Its Current Limits (Continued)

CHEMICAL NAME	8-HOUR TWA		15-MINUTE STEL		CEILING		SKIN
	PPM	MG/M3	PPM	MG/M3	PPM	MG/M3	
NAPHTHA (COAL TAR)	100	-	-	-	-	-	-
NICKEL (METAL)	-	1	-	-	-	-	-
NICOTINE	-	0.5	-	-	-	-	S
NITRIC OXIDE	25	-	-	-	-	-	-
NITROBENZENE	1	-	-	-	-	-	S
NITROETHANE	100	-	-	-	-	-	-
NITROGEN TRIFLUORIDE	10	-	-	-	-	-	-
NITROMETHANE	100	-	-	-	-	-	-
1-NITROPROPANE	25	-	-	-	-	-	-
NUISANCE DUST, RESPIRABLE	-	5	-	-	-	-	-
PARATHION	-	0.1	-	-	-	-	S
PENTACHLORONAPHTHALENE	-	0.5	-	-	-	-	S
PENTACHLOROPHENOL	-	0.5	-	-	-	-	S
PERCHLOROMETHYL MERCAPTAN	0.1	-	-	-	-	-	-
PHENOL	5	-	-	-	-	-	S
PHENYL ETHER, BIPHENYL MIXTURE	1	-	-	-	-	-	-
P-PHENYLENE DIAMINE	-	0.1	-	-	-	-	S
PHOSGENE	0.1	-	-	-	-	-	-
PHOSPHORUS (YELLOW)	-	0.1	-	-	-	-	-
PHOSPHORUS PENTACHLORIDE	-	1	-	-	-	-	-
PINDONE	-	0.1	-	-	-	-	-

TABLE VII-D. Substances for Which OSHA Did Not Consider Changing Its Current Limits (Continued)

CHEMICAL NAME	8-HOUR TWA		15-MINUTE STEL		CEILING		SKIN
	PPM	MG/M3	PPM	MG/M3	PPM	MG/M3	
PLATINUM (SOLUBLE SALTS)	-	0.002	-	-	-	-	-
PROPANE	1000	-	-	-	-	-	-
PROPYLENEIMINE	2	-	-	-	-	-	S
PYRETHRUM	-	5	-	-	-	-	-
PYRIDINE	5	15	-	-	-	-	-
QUINONE (P-BENZOQUINONE)	0.1	-	-	-	-	-	-
ROTENONE (COMMERCIAL)	-	5	-	-	-	-	-
SELENIUM AND COMPOUNDS	-	0.2	-	-	-	-	-
SELENIUM HEXAFLUORIDE	0.05	-	-	-	-	-	-
SILICA, CRYSTALLINE QUARTZ, TOTAL DUST	-	4.3	-	-	-	-	
SILVER, SOLUBLE COMPOUNDS	-	0.01	-	-	-	-	-
STIBINE	0.1	-	-	-	-	-	-
STRYCHNINE	-	0.15	-	-	-	-	-
SULFUR HEXAFLUORIDE	1000	-	-	-	-	-	-
SULFURIC ACID	-	1	-	-	-	-	-
2,4,5-T (TRICHLOROPHENOXYACETIC ACID)	-	10	-	-	-	-	-
TEDP (SULFOTEP)	-	0.2	-	-	-	-	S
TELLURIUM	-	0.1	-	-	-	-	-
TELLURIUM HEXAFLUORIDE	0.02	-	-	-	-	-	-

TABLE VII-D. Substances for Which OSHA Did Not Consider Changing Its Current Limits (Continued)

CHEMICAL NAME	8-HOUR TWA		15-MINUTE STEL		CEILING		SKIN
	PPM	MG/M3	PPM	MG/M3	PPM	MG/M3	
TEPP	-	0.05	-	-	-	-	S
1,1,2,2-TETRACHLORO-1, 2-DIFLUOROETHANE	500	-	-	-	-	-	-
1,1,1,2-TETRACHLORO-2, 2-DIFLUOROETHANE	500	-	-	-	-	-	-
TETRACHLORONAPHTHALENE	-	2	-	-	-	-	S
TETRAMETHYL SUCCINONITRILE	0.5	-	-	-	-	-	S
TETRANITROMETHANE	1	-	-	-	-	-	-
TETRYL	-	1.5	-	-	-	-	S
THALLIUM (SOLUBLE COMPOUNDS)	-	0.1	-	-	-	-	S
THIRAM	-	5	-	-	-	-	-
TIN (METAL AND INORGANIC COMPOUND)	-	2	-	-	-	-	-
1,1,2-TRICHLOROETHANE	10	-	-	-	-	-	S
TRICHLORONAPHTHALENE	-	5	-	-	-	-	S
TRIFLUOROMONOBROMOMETHANE	1000	-	-	-	-	-	-
TRIPHENYL PHOSPHATE	-	3	-	-	-	-	-
TURPENTINE	100	-	-	-	-	-	-
WARFARIN	-	0.1	-	-	-	-	-
YTTRIUM	-	1	-	-	-	-	-

BILLING CODE 4510-26-C

VIII. Public Participation—Public Hearings

Interested persons are invited to submit written data, views, and arguments with respect to OSHA'S proposed rule. These comments must be postmarked on or before July 8, 1988, and submitted in quadruplicate to the Docket Officer, Docket H–020, Room N–3670, U.S. Department of Labor, Washington, DC 20210, Tel. (202) 523–7894.

This rulemaking covers a large number of substances and industries. Therefore, to permit the public and OSHA to efficiently review the comments, it is necessary to specify the format of the comments in greater detail than normally required for OSHA rulemakings.

Comments on the general concept of the proposal shall be first and shall begin on a new page with the heading "General Comments"; the name of the commenter, and the commenter's SIC code or codes if a business.

Comments on individual substances shall follow. The comment on each substance shall start on a new page with a heading identifying the substance with the name and code number used in proposed Amended Table Z (Table I–E of the preamble; the HS code number, not the CAS number) and a second line identifying the comment as on "Health Issues" or on "Feasibility Issues." If there are comments on both they shall begin on separate pages with headings that identify the substances, and its code number and area of the comment. For feasibility comments, the heading should contain a third line identifying the SIC codes (preferably 4 digit) that the comment covers.

In addition the first or second page of each comment is to have a table of contents indicating the page number that the general comments begin and the page number that Health and Feasibility comments for each chemical individually discussed begin. Finally, one of the four sets of each comment received should not be stapled or bound, so that it can be easily copied. Written submissions must clearly identify the specific provisions of the proposal which are addressed and the position taken with respect to each issue.

The data, views and arguments that are submitted will be available for public inspection and copying at the above address. All timely submissions received will be made a part of the record of this proceeding. The preliminary regulatory impact assessment, regulatory flexibility assessment, and the exhibits cited in this document will be available for public inspection and copying at the above address.

In addition, the record currently contains many data bases of economic and health information identified in the bibliography. That information is also available for inspection and copying at the Docket Office. (Much of the information is on computer tape. OSHA will supply duplicate tapes for the copying charge).

Notice of Intention To Appear at the Informal Hearings

Pursuant to section 6(b)(3) of the OSHA Act, informal public hearings will be held on this proposal in Washington, DC commencing July 20, 1988, and continuing through August 5, 1988, or until such earlier date as oral presentations are completed. The hearing will commence at 9:30 a.m. in the auditorium of the Frances Perkins Building, U.S. Department of Labor, 3rd Street and Constitution Avenue NW., Washington, DC 20210.

Persons desiring to participate at the informal public hearing must file a notice of intention to appear by July 1, 1988. The notice of intention to appear must contain the following information:

1. The name, address, and telephone number of each person to appear;
2. The capacity in which the person will appear;
3. The approximate amount of time required for the presentation;
4. The issues and substances that will be addressed;
5. A brief statement of the position that will be taken with respect to each issue and substance addressed, and
6. Whether the party intends to submit documentary evidence and, if so, a brief summary of it.

The Notice of Intention to Appear shall be mailed to Mr. Thomas Hall, OSHA Division of Consumer Affairs, Docket No. H–020, U.S. Department of Labor, Room N–3647, 200 Constitution Avenue, NW., Washington, DC 20210, tel. (202) 523–8615, and shall be post-marked no later than July 1 1988.

Note.—This is a different address than that previously listed for docket submissions.

Filing of Testimony and Evidence Before the Hearing

Any party requesting more than ten (10) minutes for presentation at the informal public hearing, or who intends to submit documentary evidence, must provide in quadruplicate the testimony and evidence to be presented at the informal public hearing. The documentary evidence and testimony shall follow the format and include the headings and index required for comments. One copy shall not be stapled or bound and be suitable for copying. These materials must be provided to Mr. Thomas Hall, OSHA Division of Consumer Affairs at the address above and be post-marked no later than July 8, 1988.

Each submission will be reviewed in light of the amount of time requested in the Notice of Intention to Appear. In instances where the information contained in the submission does not justify the amount of time requested, a more appropriate amount of time will be allocated and the participant will be notified of that fact prior to the informal public hearings.

Any party who has not substantially complied with the above requirement may be limited to a ten-minute presentation and may be requested to return for questioning at a later time.

Any party who has not filed a notice of intention to appear may be allowed to testify for no more than 10 minutes as time permits, at the discretion of the Administrative Law Judge, but will not be allowed to question witnesses.

Notices of intention to appear, testimony and evidence will be available for inspection and copying at the Docket Office at the address above.

Conduct of Hearing

The informal public hearing will commence at 9:30 a.m. at the scheduled location with the resolution of any procedural matters relating to the hearing. The informal public hearing will be presided over by an Administrative Law Judge who will have the power necessary and appropriate to conduct a full and fair informal public hearing as provided in 29 CFR Part 1911, including the power to:

1. Regulate the course of the proceedings;
2. To dispose of procedural requests, objections and comparable matters;
3. To confine the presentation to the matters pertinent to the issues raised;
4. To regulate the conduct of those present at the informal public hearing by appropriate means;
5. In the Judge's discretion, to question and permit questioning of any witness; and
6. In the Judge's discretion, to keep the record open for a brief additional period to receive written information and additional data, views, and arguments from any person who has participated in the oral proceedings. It is intended that August 12, 1988 be the deadline for post hearing evidence and August 26, 1988 be the deadline for post hearing briefs.

Following the close of the informal public hearing, the presiding Administrative Law Judge will certify

the record of the informal public hearing to the Assistant Secretary of Labor for Occupational Safety and Health. The notice of proposed rulemaking will be reviewed in light of all testimony and written submissions received as part of the record, and the proposed standard will be modified or a determination will be made not to modify the proposed standard based on the entire record of the proceeding.

State Plan Applicability

The 25 states with their own OSHA-approved occupational safety and health plans must adopt a comparable standard within six months of the publication date of a final standard. These States include: Alaska, Arizona, California, Connecticut (for State and local government employees only), Hawaii, Indiana, Iowa, Kentucky, Maryland, Michigan, Minnesota, Nevada, New Mexico, New York (for State and local government employees only), North Carolina, Oregon, Puerto Rico, South Carolina, Tennessee, Utah, Vermont, Virginia, Virgin Islands, Washington, Wyoming. Until such time as a State standard is promulgated, Federal OSHA will provide interim enforcement assistance, as appropriate.

List of Subjects in 29 CFR Part 1910

Air contaminants, Occupational safety and health, Permissible exposure limits, Health, Risk assessment.

IX. Authority

This document has been prepared under the direction of John A. Pendergrass, Assistant Secretary of Labor for Occupational Safety and Health, U.S. Department of Labor, 200 Constitution Avenue NW., Washington, DC 20210. Pursuant to section 6 of the Occupational Safety and Health Act of 1970 (29 U.S.C. 655), section 4 of the Administrative Procedures Act (5 U.S.C. 551), 29 CFR Part 1911 and Secretary of Labor's Order 9-83 (48 FR 35736), it is proposed to amend 29 CFR Part 1910 as set forth below.

Signed at Washington, DC, this 26th day of May 1988.

John A. Pendergrass,

Assistant Secretary of Labor.

X. Standard

OSHA propose to amend 29 CFR Part 1910 as follows:

PART 1910—[AMENDED]

1. The authority citation for Subpart Z of Part 1910 would be amended by revising the third paragraph, and the first and second paragraphs are republished to read as follows:

Authority: Secs. 6, 8. Occupational Safety and Health Act, 29 U.S.C. 655, 657; Secretary of Labor's Orders 12-71 (36 FR 8754), 8-76 (41 FR 25059), or 9-83 (48 FR 35736) as applicable; and 29 CFR 1911.

Section 1910.1000 Tables Z-1, Z-2, and Z-3 also issued under 5 U.S.C. 553.

Section 1910.1000 paragraphs (a) through (c), (e), (f) and Tables Z-1, Z-2, Z-3, not issued under 29 CFR 1911 except for the arsenic, cotton dust, benzene and formaldehyde listings.

* * * * *

2. It is proposed to amend § 1910.1000 by revising the introductory text, redesignating paragraph (d) as paragraph (f) and revising introductory text of paragraph (f) and the last sentence of paragraph (f)(1)(i); adding a new paragraph (d); republishing Tables Z-1, Z-2 and Z-3 with an asterisk indicating the materials proposed to be removed and adding a new Table Z-4.

§ 1910.1000 [Amended]

An employee's exposure to any material listed in Tables Z-1, Z-2, Z-3 or Z-4 of this section shall be limited in accordance with the requirements of the following paragraphs of this section.

* * * * *

(d) Table Z-4 lists the proposed permissible exposure limits for 428 materials.

(1) An employee's exposure to any material listed in Table Z-4 shall not exceed the Time Weighted Average (TWA), Short Term Exposure Limit (STEL) or Ceiling specified in Table Z-4 for that material.

(2) An employee's skin exposure to materials listed in table Z-4 with an "S" Notation shall be limited through the use of gloves, coveralls, goggles, or other appropriate personal protection equipment necessary to prevent possible skin absorption.

(3) The following definitions apply to paragraph (d) of this section and Table Z-4:

(i) Time weighted average (TWA) is the average airborne exposure of an employee in any 8-hour work shift of a 40-hour work week which shall not be exceeded.

(ii) Short term exposure limit (STEL) is the employee's 15-minute time weighted average exposure which shall not be exceeded at any time during a work day.

(iii) Ceiling is the employee's exposure which shall not be exceeded during any part of the work day. If instantaneous monitoring is not feasible, then the ceiling shall be assessed by sampling over a 15-minute period as would be done for a STEL, unless a different time period is specifically indicated.

* * * * *

(f) Computational formula. The computation formula which shall apply to employee exposure to more than 1 material for which 8 hour time weighted averages are listed in Subpart Z of 29 CFR Part 1910 in order to determine whether an employee is exposed over the regulatory limit is as follows:

(1)(i) * * * *

The value of E shall not exceed the 8-hour time weighted average specified in Subpart Z of 29 CFR Part 1910 for the material involved.

* * * * *

BILLING CODE 4510-26-M

TABLE Z-1

Substance	ppm[a]	mg/m[3][b]
ACETALDEHYDE*	200	360
ACETIC ACID*	10	25
ACETIC ANHYDRIDE*	5	20
ACETONE*	1000	2400
ACETONITRILE*	40	70
ACETYLENE DICHLORIDE, see 1,2-DICHLOROETHYLENE		
ACETYLENE TETRABROMIDE	1	14
ACROLEIN*	0.1	0.25
ACRYLAMIDE-Skin*		0.3
ALDRIN-Skin		0.25
ALLYL ALCOHOL-Skin*	2	5
ALLYL CHLORIDE*	1	3
C ALLYLGLYCIDYL ETHER (AGE)*	10	45
ALLYL PROPYL DISULFIDE*	2	12
2-AMINOETHANOL, see ETHANOLAMINE		
2-AMINOPYRIDINE	0.5	2
AMMONIA*	50	35
AMMONIUM SULFAMATE (AMMATE)*		15
N-AMYL ACETATE	100	525
SEC-AMYL ACETATE	125	650
ANILINE-Skin*	5	19
ANISIDINE (O,P-ISOMERS)-Skin		0.5
ANTIMONY AND COMPOUNDS(as Sb)		0.5
ANTU (ALPHA NAPTHYL THIOUREA)*		0.3
ARSENIC ORGANIC COMPOUNDS (as As)		0.5
ARSINE	0.05	0.2

TABLE Z-1

Substance	ppm[a]	mg/m[3][b]
AZINPHOS-METHYL-Skin*		0.2
BARIUM (SOLUBLE COMPOUNDS)		0.5
p-BENZOQUINONE, see QUINONE		
BENZOYL PEROXIDE		5
BENZYL CHLORIDE	1	5
BIPHENYL, see DIPHENYL		
BORON OXIDE*		15
C BORON TRIFLUORIDE	1	3
BROMINE*	0.1	0.7
BROMOFORM-Skin	0.5	5
BUTADIENE (1,3-BUTADIENE)	1000	2200
BUTANETHIOL, see BUTYL MERCAPTAN		
2-BUTANONE*	200	590
2-BUTOXY ETHANOL(BUTYL CELLOSOLVE)-Skin*	50	240
BUTYL ACETATE (N-BUTYL ACETATE)*	150	710
SEC-BUTYL ACETATE	200	950
TERT-BUTYL ACETATE	200	950
BUTYL ALCOHOL*	100	300
SEC-BUTYL ALCOHOL*	150	450
TERT-BUTYL ALCOHOL*	100	300
C BUTYLAMINE-Skin	5	15
C TERT-BUTYL CHROMATE (as CrO_3)-Skin		0.1
N-BUTYL GLYCIDYL ETHER*	50	270
BUTYL MERCAPTAN*	10	35
P-TERT-BUTYLTOLUENE*	10	60
CALCIUM OXIDE*		5
CAMPHOR*		2

TABLE Z-1

Substance	ppm[a]	mg/m3[b]
CARBARYL (SEVIN)		5
CARBON BLACK		3.5
CARBON DIOXIDE*	5000	9000
CARBON MONOXIDE*	50	55
CHLORDANE-Skin		0.5
CHLORINATED CAMPHENE-Skin*		0.5
CHLORINATED DIPHENYL OXIDE		0.5
C CHLORINE*	1	3
CHLORINE DIOXIDE*	0.1	0.3
C CHLORINE TRIFLUORIDE	0.1	0.4
C CHLOROACETALDEHYDE	1	3
A-CHLOROACETOPHENONE (PHENACYLCHLORIDE)	0.05	0.3
CHLOROBENZENE (MONOCHLOROBENZENE)	75	350
O-CHLOROBENZYLIDENE MALONONITRILE (OCBM)*	0.05	0.4
CHLOROBROMOMETHANE	200	1050
2-CHLORO-1,3-BUTADIENE, see CHLOROPRENE		
CHLORODIPHENYL (42 PERCENT CHLORINE)-Skin		1
CHLORODIPHENYL (54 PERCENT CHLORINE)-Skin		0.5
1-CHLORO, 2,3-EPOXYPROPANE, see EPICHLORHYDRIN		
2-CHLOROETHANOL, see ETHYLENE CHLOROHYDRIN		
CHLOROETHYLENE, see VINYL CHLORIDE		
C CHLOROFORM (TRICHLOROMETHANE)*	50	240
1-CHLORO-1-NITROPROPANE*	20	100
CHLOROPICRIN	0.1	0.7
CHLOROPRENE(2-CHLORO-1,3-BUTADIENE)-Skin*	25	90
CHROMIUM, SOL.CHROMIC, CHROMOUS SALTS AS Cr		0.5
CHROMIUM, METAL AND INSOLUBLE SALTS		1

TABLE Z-1

Substance	ppm[a]	mg/m3[b]
COAL TAR PITCH VOLATILES (BENZENE SOLUBLE FRACTION) ANTHRACENE, BaP, PHENANTHRENE, ACRIDINE, CHRYSENE, PYRENE)		0.2
COBALT, METAL FUME AND DUST*		0.1
COPPER FUME		0.1
COPPER DUSTS AND MISTS		1
COTTON DUST (RAW)		1[e]
CRAG HERBICIDE*		15
CRESOL (ALL ISOMERS)-Skin	5	22
CROTONALDEHYDE	2	6
CUMENE-Skin	50	245
CYANIDE (as Cn)-Skin		5
CYCLOHEXANE	300	1050
CYCLOHEXANOL*	50	200
CYCLOHEXANONE*	50	200
CYCLOHEXENE	300	1015
CYCLOPENTADIENE	75	200
2,4-D		10
DDT-Skin*		1
DDVP -Skin		1
DECABORANE-Skin*	0.05	0.3
DEMETON -Skin		0.1
DIACETONE ALCOHOL (4-HYDROXY-4-METHYL-2-PENTANONE)	50	240
1,2-DIAMINOETHANE, see ETHYLENEDIAMINE		
DIAZOMETHANE	0.2	0.4
DIBORANE	0.1	0.1
DIBUTYL PHOSPHATE*	1	5

TABLE Z-1

Substance	ppm[a]	mg/m3[b]
DIBUTYLPHTHALATE		5
C O-DICHLOROBENZENE*	50	300
P-DICHLOROBENZENE*	75	450
DICHLORODIFLUOROMETHANE	1000	4950
1,3-DICHLORO-5,5-DIMETHYL HYDANTOIN*		0.2
1,1-DICHLOROETHANE*	100	400
1,2-DICHLOROETHYLENE	200	790
C DICHLOROETHYL ETHER-Skin*	15	90
DICHLOROMETHANE, see METHYLENECHLORIDE		
DICHLOROMONOFLUOROMETHANE*	1000	4200
C 1,1-DICHLORO-1-NITROETHANE*	10	60
1,2-DICHLOROPROPANE, see PROPYLENEDICHLORIDE		
DICHLOROTETRAFLUOROETHANE	1000	7000
DIELDRIN-Skin		0.25
DIETHYLAMINE*	25	75
DIETHYLAMINO ETHANOL-Skin	10	50
DIETHYLETHER, see ETHYL ETHER		
DIFLUORODIBROMOMETHANE	100	860
C DIGLYCIDYL ETHER (DGE)*	0.5	2.8
DIHYDROXYBENZENE, see HYDROQUINONE		
DIISOBUTYL KETONE*	50	290
DIISOPROPYLAMINE-Skin	5	20
DIMETHOXYMETHANE, see METHYLAL		
DIMETHYL ACETAMIDE-Skin	10	35
DIMETHYLAMINE	10	18
DIMETHYLAMINOBENZENE, see XYLIDENE		
DIMETHYLANILINE (N-DIMETHYL-ANILINE)-Skin*	5	25

TABLE Z-1

Substance	ppm[a]	mg/m3[b]
DIMETHYLBENZENE, see XYLENE		
DIMETHYL 1,2-DIBROMO-2,2-DICHLOROETHYL PHOSPHATE (DIBROM)		3
DIMETHYLFORMAMIDE-Skin	10	30
2,6-DIMETHYLHEPTANONE, see DIISOBUTYL KETONE		
1,1-DIMETHYLHYDRAZINE-Skin	0.5	1
DIMETHYLPHTHALATE		5
DIMETHYLSULFATE-Skin*	1	5
DINITROBENZENE (ALL ISOMERS)-Skin		1
DINITRO-O-CRESOL-Skin		0.2
DINITROTOLUENE-Skin		1.5
DIOXANE (DIETHYLENE DIOXIDE)-Skin*	100	360
DIPHENYL	0.2	1
DIPHENYLMETHANE DIISOCYANATE (see METHYLENE BIS-PHENYL ISOCYANATE (MDI))		
DIPROPYLENE GLYCOL METHYL ETHER-Skin*	100	600
DI-SEC. OCTYL-PHTHALATE (DI-2-ETHYLHEXYLPHTHALATE)*		5
ENDRIN-Skin		0.1
EPICHLOROHYDRIN-Skin*	5	19
EPN-Skin		0.5
1,2-EPOXYPROPANE, see PROPYLENEOXIDE		
2,3-EPOXY-1-PROPANOL, see GLYCIDOL		
ETHANETHIOL, see ETHYLMERCAPTAN		
ETHANOLAMINE*	3	6
2-ETHOXYETHANOL -Skin	200	740
2-ETHOXYETHYLACETATE-(CELLO-SOLVE ACETATE)-Skin	100	540
ETHYL ACETATE	400	1400

TABLE Z-1

Substance	ppm[a]	mg/m3[b]
ETHYL ACRYLATE-Skin*	25	100
ETHYL ALCOHOL (ETHANOL)	1000	1900
ETHYLAMINE	10	18
ETHYL SEC-AMYL KETONE (5-METHYL-3-HEPTANONE)	25	130
ETHYL BENZENE*	100	435
ETHYL BROMIDE*	200	890
ETHYL BUTYL KETONE (3-HEPTANONE)	50	230
ETHYL CHLORIDE	1000	2600
ETHYL ETHER*	400	1200
ETHYL FORMATE	100	300
C ETHYL MERCAPTAN*	10	25
ETHYL SILICATE*	100	850
ETHYLENE CHLOROHYDRIN-Skin*	5	16
ETHYLENEDIAMINE	10	25
C ETHYLENE GLYCOL DINITRATE AND/OR NITROGLYCERIN -Skin*	0.2[d]	1
ETHYLENE GLYCOL MONOMETHYL ETHER ACETATE, see METHYL CELLOSOLVE ACETATE		
ETHYLENE IMINE-Skin	0.5	1
N-ETHYLMORPHOLINE-Skin*	20	94
FERBAM*		15
FERROVANADIUM DUST*		1
FLUORIDE (AS F)		2.5
FLUORINE*	0.1	0.2
FLUOROTRICHLOROMETHANE*	1000	5600
FORMIC ACID	5	9
FURFURAL-Skin*	5	20

TABLE Z-1

Substance	ppm[a]	mg/m3[b]
FURFURYL ALCOHOL*	50	200
GLYCIDOL (2,3-EPOXY-1-PROPANOL)*	50	150
GLYCOL MONOMETHYL ETHER, see 2-ETHOXYETHANOL		
GLUTHION , see AZINPHOSMETHYL		
HAFNIUM		0.5
HEPTACHLOR-Skin		0.5
HEPTANE (N-HEPTANE)*	500	2000
HEXACHLOROETHANE-Skin*	1	10
HEXACHLORONAPHTHALENE-Skin		0.2
HEXANE (N-HEXANE)*	500	1800
2-HEXANONE*	100	410
HEXONE (METHYL ISOBUTYL KETONE)*	100	410
SEC-HEXYL ACETATE	50	300
HYDRAZINE-Skin*	1	1.3
HYDROGEN BROMIDE*	3	10
C HYDROGEN CHLORIDE	5	7
HYDROGEN CYANIDE-Skin*	10	11
HYDROGEN PEROXIDE (90%)	1	1.4
HYDROGEN SELENIDE	0.05	0.2
HYDROQUINONE		2
C IODINE	0.1	1
IRON OXIDE FUME*		10
ISOAMYL ACETATE	100	525
ISOAMYL ALCOHOL*	100	360
ISOBUTYL ACETATE	150	700
ISOBUTYL ALCOHOL*	100	300

TABLE Z-1

Substance	ppm[a]	mg/m3[b]
ISOPHORONE*	25	140
ISOPROPYL ACETATE*	250	950
ISOPROPYL ALCOHOL*	400	980
ISOPROPYLAMINE*	5	12
ISOPROPYLETHER*	500	2100
ISOPROPYL GLYCIDYL ETHER (IGE)*	50	240
KETENE*	0.5	0.9
LINDANE-Skin		0.5
LITHIUM HYDRIDE		0.025
L.P.G. (LIQIFIED PETROLEUM GAS)	1000	1800
MAGNESIUM OXIDE FUME*		15
MALATHION-Skin*		15
MALEIC ANHYDRIDE	0.25	1
C MANGANESE		5
MESITYL OXIDE*	25	100
METHANETHIOL, see METHYL MERCAPTAN		
METHOXYCHLOR*		15
2-METHOXYETHANOL, see METHYL CELLOSOLVE		
METHYL ACETATE*	200	610
METHYL ACETYLENE (PROPYNE)	1000	1650
METHYL ACETYLENE-PROPADIENE MIXTURE (MAPP)*	1000	1800
METHYL ACRYLATE-Skin	10	35
METHYLAL(DIMETHOXYMETHANE)	1000	3100
METHYL ALCOHOL (METHANOL)*	200	260
METHYLAMINE	10	12
METHYL AMYL ALCOHOL, see ISOBUTYL CARBONOL		
METHYL (N-AMYL) KETONE (2-HEPTANONE)*	100	465

TABLE Z-1

Substance	ppm[a]	mg/m3[b]
C METHYL BROMIDE-Skin*	20	80
METHYL BUTYL KETONE, see 2-HEXANONE		
METHYL CELLOSOLVE-Skin	25	80
METHYL CELLOSOLVE ACETATE-Skin	25	120
METHYL CHLOROFORM*	350	1900
METHYLCYCLOHEXANE*	500	2000
METHYLCYCLOHEXANOL*	100	470
O-METHYLCYCLOHEXANONE-Skin*	100	460
METHYL ETHYL KETONE (MEK), see 2-BUTANONE		
METHYL FORMATE*	100	250
METHYL IODIDE-Skin*	5	28
METHYL ISOBUTYL CARBINOL-Skin*	25	100
METHYL ISOBUTYL KETONE, see HEXONE		
METHYL ISOCYANATE-Skin	0.02	0.05
C METHYL MERCAPTAN*	10	20
METHYL METHACRYLATE	100	410
METHYL PROPYL KETONE, see 2-PENTANONE		
C A-METHYL STYRENE*	100	480
C METHYLENE BISPHENYL ISOCYANATE (MDI)	0.02	0.2
MOLYBDENUM (INSOLUBLE COMPOUNDS)		15
MOLYBDENUM (SOLUBLE COMPOUNDS)		5
MONOMETHYL ANILINE-Skin*	2	9
C MONOMETHYL HYDRAZINE-Skin	0.2	0.35
MORPHOLINE-Skin*	20	70
NAPHTHA (COAL TAR)	100	400
NAPHTHALENE*	10	50
NICKEL CARBONYL*	0.001	0.007

TABLE Z-1

Substance	ppm[a]	mg/m3[b]
NICKEL, (METAL AND SOLUBLE COMPOUNDS AS Ni)		1
NICOTINE-Skin		0.5
NITRIC ACID*	2	5
NITRIC OXIDE	25	30
P-NITROANILINE-Skin*	1	6
NITROBENZENE-Skin	1	5
P-NITROCHLOROBENZENE-Skin*		1
NITROETHANE	100	310
C NITROGEN DIOXIDE*	5	9
NITROGEN TRIFLUORIDE	10	29
C NITROGLYCERIN-Skin*	0.2	2
NITROMETHANE	100	250
1-NITROPROPANE	25	90
2-NITROPROPANE*	25	90
NITROTOLUENE-Skin*	5	30
NITROTRICHLOROMETHANE, see CHLOROPICRINE		
OCTACHLORONAPHTHALENE-Skin*		0.1
OCTANE*	500	2350
OIL MIST, MINERAL*		5
OSMIUM TETROXIDE*		0.002
OXALIC ACID*		1
OXYGEN DIFLUORIDE*	0.05	0.1
OZONE*	0.1	0.2
PARAQUAT-Skin*		0.5
PARATHION-Skin		0.1
PENTABORANE*	0.005	0.01
PENTACHLORONAPHTHALENE-Skin		0.5

TABLE Z-1

Substance	ppm[a]	mg/m3[b]
PENTACHLOROPHENOL-Skin		0.5
PENTANE*	1000	2950
2-PENTANONE*	200	700
PERCHLOROMETHYL MERCAPTAN	0.1	0.8
PERCHLORYL FLUORIDE*	3	13.5
PETROLEUM DISTILLATES (NAPHTHA)*	500	2000
PHENOL-Skin	5	19
P-PHENYLENE DIAMINE-Skin		0.1
PHENYL ETHER (VAPOR)*	1	7
PHENYL ETHER- BIPHENYL MIXTURE (VAPOR)	1	7
PHENYLETHYLENE, see STYRENE		
PHENYL GLYCIDYL ETHER (PGE)*	10	60
PHENYLHYDRAZINE-Skin*	5	22
PHOSDRIN (MEVINPHOS)-Skin*		0.1
PHOSGENE (CARBONYL CHLORIDE)	0.1	0.4
PHOSPHINE*	0.3	0.4
PHOSPHORIC ACID*		1
PHOSPHORUS (YELLOW)		0.1
PHOSPHORUS PENTACHLORIDE		1
PHOSPHORUS PENTASULFIDE*		1
PHOSPHORUS TRICHLORIDE*	0.5	3
PHTHALIC ANHYDRIDE*	2	12
PICRIC ACID-Skin*		0.1
PIVAL (2-PIVALYL-1,3-INDANDIONE)		0.1
PLATINUM (SOLUBLE SALTS) AS Pt		0.002
PROPANE	1000	1800
N-PROPYL ACETATE*	200	840

TABLE Z-1

Substance	ppm[a]	mg/m3[b]
PROPYL ALCOHOL*	200	500
N-PROPYL NITRATE*	25	110
PROPYLENE DICHLORIDE*	75	350
PROPYLENE IMINE-Skin	2	5
PROPYLENE OXIDE*	100	240
PROPYNE, see METHYLACETYLENE		
PYRETHRUM		5
PYRIDINE	5	15
QUINONE	0.1	0.4
RHODIUM, METAL FUME AND DUST, AS Rh*		0.1
RHODIUM (SOLUBLE SALTS)*		0.001
RONNEL*		15
ROTENONE (COMMERCIAL)		5
SELENIUM COMPOUNDS (AS Se)		0.2
SELENIUM HEXAFLUORIDE	0.05	0.4
SILVER, METAL AND SOLUBLE COMPOUNDS*		0.01
SODIUM FLUOROACETATE (1080) -Skin*		0.05
SODIUM HYDROXIDE*		2
STIBINE	0.1	0.5
STODDARD SOLVENT*	500	2900
STRYCHNINE		0.15
SULFUR DIOXIDE	5	13
SULFUR HEXAFLUORIDE	1000	6000
SULFURIC ACID		1
SULFUR MONOCHLORIDE*	1	6
SULFUR PENTAFLUORIDE*	0.025	0.25
SULFURYL FLUORIDE*	5	20

TABLE Z-1

Substance	ppm[a]	mg/m3[b]
SYSTOX, see DEMETON		
2,4,5T		10
TANTALUM*		5
TEDP-Skin		0.2
TELLURIUM		0.1
TELLURIUM HEXAFLUORIDE	0.02	0.2
TEPP-Skin		0.05
C TERPHENYLS*	1	9
1,1,2,2-TETRACHLORO-1,2-DIFLUOROETHANE	500	4170
1,1,1,2-TETRACHLORO-2,2-DIFLUOROETHANE	500	4170
1,1,2,2-TETRACHLOROETHANE-Skin*	5	35
TETRACHLOROMETHANE, see CARBON TETRACHLORIDE		
TETRACHLORONAPHTHALENE-Skin		2
TETRAETHYL LEAD (AS Pb)-Skin*		0.075
TETRAHYDROFURAN*	200	590
TETRAMETHYL LEAD (AS Pb)-Skin*		0.075
TETRAMETHYL SUCCINONITRILE-Skin	0.5	3
TETRANITROMETHANE	1	8
TETRYL (2,4,6-TRINITROPHENOL-METHYL-NITRAMINE)-Skin		1.5
THALLIUM (SOLUBLE COMPOUNDS)-Skin AS Tl*		0.1
THIRAM		5
TIN (INORGANIC COMPOUNDS EXCEPT OXIDES)		2
TIN (ORGANIC COMPOUNDS) *		0.1
C TOLUENE-2,4-DIISOCYANATE*	0.02	0.14
O-TOLUIDINE-Skin*	5	22
TOXAPHENE, see CHLORINATED CAMPHENE		

TABLE Z-1

Substance	ppm[a]	mg/m3[b]
TRIBUTYL PHOSPHATE*		5
1,1,1-TRICHLOROETHANE, see METHYL CHLOROFORM		
1,1,2-TRICHLOROETHANE-Skin*	10	45
TITANIUM DIOXIDE		15
TRICHLOROMETHANE, see CHLOROFORM		
TRICHLORONAPHTHALENE-Skin		5
1,2,3-TRICHLOROPROPANE*	50	300
1,1,2-TRICHLORO-1,2,2-TRIFLUOROETHANE*	1000	7600
TRIETHYLAMINE*	25	100
TRIFLUOROMONOBROMOMETHANE	1000	6100
2,4,6 - TRINITROPHENOL, see PICRIC ACID		
2,4,6 - TRINITROPHENOLMETHYL-NITRAMINE, see TETRYL		
TRINITROTOLUENE-Skin*		1.5
TRIORTHOCRESYL PHOSPHATE*		0.1
TRIPHENYL PHOSPHATE		3
TURPENTINE	100	560
URANIUM (SOLUBLE COMPOUNDS)*		0.05
URANIUM (INSOLUBLE COMPOUNDS)*		0.25
C VANADIUM (V205, DUST)*		0.5
C VANADIUM (V205, FUME)*		0.1
VINYL BENZENE, see STYRENE		
VINYL CYANIDE, see ACRYLONITRILE		
VINYL TOLUENE*	100	480
WARFARIN		0.1
XYLENE (XYLOL)*	100	435
XYLIDINE-Skin	5	25
YTTRIUM		1

TABLE Z-1

Substance	ppm[a]	mg/m3[b]
ZINC CHLORIDE FUME*		1
ZINC OXIDE (FUME)*		5
ZIRCONIUM COMPOUNDS (AS Zr)*		5

[a]Parts of vapor or gas per million parts of contaminated air by volume at 25° C and 760 mm. Hg pressure.

[b]Approximate milligrams of particulate per cubic meter of air.

(No footnote "c" is used to avoid confusion with ceiling value notations.)

[d]An atmospheric concentration of not more than 0.02 p.p.m. or personal protection may be necessary to avoid headache.

[e]This 8-hour time weighted average is for respirable dust as measured by a verticle elutriator cotton dust sampler or equivalent instrument. This time weighted average applies to the cotton waste processing operations of waste recycling (sorting, blending, cleaning, and willowing) and garnetting.

*Substances that are in Table Z-4 in this proposal.

TABLE Z-2

Material	8-hour time weighted average	Acceptable ceiling concentration	Acceptable maximum peak above the acceptance ceiling concentration for an 8-hour shift	
			Concentration	Maximum duration
BENZENE (Z37.40-1969)	10 p.p.m.	25 p.p.m.	50 p.p.m.	10 minutes
BERYLLIUM AND BERYLLIUM COMPOUNDS (Z37.29-1970)*	2 ug/M^3	5 ug/M^3	25 ug/M^3	30 minutes
CADMIUM FUME (Z37.5-1970)	0.1 mg/M^3	0.3 mg/M^3		
CADMIUM DUST (Z37.5-1970)	0.2 mg/M^3	0.6 mg/M^3		
CARBON DISULFIDE (Z37.3-1968)*	20 p.p.m.	30 p.p.m.	100 p.p.m.	30 minutes
CARBON TETRACHLORIDE (Z37.17-1967)*	10 p.p.m.	25 p.p.m.	200 p.p.m.	5 minutes in any 4 hours
CHROMIC ACID AND CHROMATES (Z37.7-1971)*		1 mg/10M^3		
ETHYLENE DIBROMIDE (Z37.31-1970)	20 p.p.m.	30 p.p.m.	50 p.p.m.	5 minutes
ETHYLENE DICHLORIDE (Z37.21 1969)*	50 p.p.m.	100 p.p.m.	200 p.p.m.	5 minutes in any 3 hours

TABLE Z-2

Material	8-hour time weighted average	Acceptable ceiling concentration	Acceptable maximum peak above the acceptance ceiling concentration for an 8-hour shift	
			Concentration	Maximum duration
FORMALDEHYDE (Z37.16-1967)	3 p.p.m.	5 p.p.m.	10 p.p.m.	30 minutes
HYDROGEN FLUORIDE (Z37.28-1969)*	3 p.p.m.			
HYDROGEN SULFIDE (Z37.2-1966)*		20 p.p.m.	50 p.p.m.	10 minutes once only if no other measurable exposure occurs
FLUORIDE AS DUST (Z37.28-1969)	2.5 mg/M^3			
MERCURY (Z37.8-1971)*		0.1 mg/10M^3		
METHYL CHLORIDE(Z37.18-1969)*	100 p.p.m.	200 p.p.m.	300 p.p.m.	5 minutes in any 2 hours
METHYLENE CHLORIDE (Z37.23-1969)	500 p.p.m.	1,000 p.p.m.	2,000 p.p.m.	5 minutes in any 2 hours
ORGANO (ALKYL) MERCURY (Z37.30-1969)*	0.01 mg/M^3	0.04 mg/M^3		

TABLE Z-2

Material	8-hour time weighted average	Acceptable ceiling concentration	Acceptable maximum peak above the acceptance ceiling concentration for an 8-hour shift	
			Concentration	Maximum duration
STYRENE (Z37.15-1969)*	100 p.p.m.	200 p.p.m.	600 p.p.m.	5 minutes in any 3 hours
TETRACHLOROETHYLENE (Z37.22-1967)*	100 p.p.m.	200 p.p.m.	300 p.p.m.	5 minutes in any 3 hours
TOLUENE (Z37.12-1967)*	200 p.p.m.	300 p.p.m.	500 p.p.m.	10 minutes
TRICHLOROETHYLENE (Z37.19-1967)*	100 p.p.m.	200 p.p.m.	300 p.p.m.	5 minutes in any 2 hours

*Substances that are on Table Z-4 in this proposal.

TABLE Z-3

Substance	Mppcf[e]	Mg/M^3
SILICA:		
CRYSTALINE		
QUARTZ (RESPIRABLE)*	$\frac{250}{\%SiO_2+5}$[f]	$\frac{10\ mg/M^3}{\%SiO_2+2}$ [m]
QUARTZ (TOTAL)		$\frac{30\ mg/M^3}{\%SiO_2+2}$
CRISTOBALITE*: Use 1/2 the value calculated from the count or mass formulae for quartz		
TRIDYMITE*: Use 1/2 the value calculated from the formulae for quartz		
AMORPHOUS, including natural diatomaceous earth*	20	$\frac{80\ mg/M^3}{\%SiO_2}$
SILICATES (less than 1% crystalline silica):		
Mica*	20	
Soapstone*	20	
Talc (non-asbestos-form)*	20[n]	

TABLE Z-3

Substance	Mppcf[e]	Mg/M^3
Talc (fibrous). Use asbestos limit		
Tremolite (see talc, fibrous)		
Portland cement*	50	
GRAPHITE (NATURAL)*	15	
COAL DUST (respirable fraction less than 5% SiO_2)*		2.4 mg/M^3 or $\frac{10 \text{ mg/M}^3}{\%SiO_2+2}$
For more than 5% SiO_2*		
INERT OR NUISANCE DUST:		
Respirable fraction*	15	5 mg/M^3
Total dust	50	15 mg/M^3

NOTE: Conversion factors--mppcf X 35.3 = million particles per cubic meter = particles per c.c.

[e]Millions of particles per cubic foot of air, based on impinger samples counted by light field techniques.

[f]The percentage of crystalline silica in the formula is the amount determined from air-borne samples, except in those instances in which other methods have been shown to be applicable.

[m]Both concentration and percent quartz for the application of this limit are to be determined from the fraction passing a size selector with the following characteristics:

[n]Containing less than 1% quartz; if 1% quartz, use quartz limit.

Aerodynamic diameter (unit density sphere)	Percent passing selector
2	90
2.5	75
3.5	50
5.0	25
10	0

The measurements under this note refer to the use of an AEC instrument. The respirable fraction of coal dust is determined with a MRE; the figure corresponding to that of 2.4 Mg/M^3 in the table for coal dust is 4.5 Mg/M^3.

* Substances that are in Table Z-4 in this proposal.

TABLE Z-4. All Substances Considered by OSHA for Proposed Revision or Addition of Limits[a]

H.S. NO./ CHEMICAL NAME	8-HOUR TWA		SHORT-TERM LIMIT*		CEILING**		SKIN NOTATION
	MG/M3	PPM	MG/M3	PPM	MG/M3	PPM	
1001 ACETALDEHYDE	180	100	270	150	—	—	
1002 ACETIC ACID	25	10	37	15	—	—	
1003 ACETIC ANHYDRIDE	—	—	—	—	20	5	
1004 ACETONE[d]	590	250	—	—	—	—	
1005 ACETONITRILE[d]	34	20	—	—	—	—	
1006 ACETYLSALICYLIC ACID (ASPIRIN)	5	—	—	—	—	—	
1007 ACROLEIN	0.25	0.1	0.8	0.3	—	—	
1008 ACRYLAMIDE	0.03	—	—	—	—	—	YES
1009 ACRYLIC ACID	30	10	—	—	—	—	
1010 ALLYL ALCOHOL	5	2	10	4	—	—	YES
1011 ALLYL CHLORIDE	3	1	6	2	—	—	
1012 ALLYL GLYCIDYL ETHER (AGE)	22	5	44	10	—	—	YES
1013 ALLYL PROPYL DISULFIDE	12	2	18	3	—	—	
1014 ALPHA-ALUMINA	10	—	—	—	—	—	
1015 ALUMINUM (ALKYLS)	2	—	—	—	—	—	
1016 ALUMINUM (METAL)	10	—	—	—	—	—	
1017 ALUMINUM (PYRO POWDERS)	5	—	—	—	—	—	
1018 ALUMINUM (SOLUBLE SALTS)	2	—	—	—	—	—	
1019 ALUMINUM (WELDING FUMES)	5	—	—	—	—	—	
1020 AMITROLE (3-AMINO-1,2,4-TRIAZOLE)	0.2	—	—	—	—	—	

TABLE Z-4. All Substances Considered by OSHA for Proposed Revision or Addition of Limits[a]

H.S. NO./ CHEMICAL NAME	8-HOUR TWA		SHORT-TERM LIMIT*		CEILING**		SKIN NOTATION
	MG/M3	PPM	MG/M3	PPM	MG/M3	PPM	
1021 AMMONIA	18	25	27	35	—	—	
1022 AMMONIUM CHLORIDE (FUME)	10	—	20	—	—	—	
1024 AMMONIUM SULFAMATE (AMMATE)	10	—	—	—	—	—	
1025 ANILINE	8	2	—	—	—	—	YES
1028 ASPHALT FUMES[b]	5	—	—	—	—	—	
1029 ATRAZINE	5	—	—	—	—	—	
1031 BARIUM SULFATE	10	—	—	—	—	—	
1032 BENOMYL	10	0.8	—	—	—	—	
1033 BERYLLIUM AND BERYLLIUM COMPOUNDS[e]	0.002	—	0.005 (30 min)	—	0.025	—	
1034 BISMUTH TELLURIDE (SE-DOPED)	5	—	—	—	—	—	
1035 BISMUTH TELLURIDE (UNDOPED)	10	—	—	—	—	—	
1036 BORATES, TETRA, SODIUM (ANHYDROUS)	1	—	—	—	—	—	
1037 BORATES, TETRA, SODIUM (DECAHYDRATE)	5	—	—	—	—	—	
1038 BORATES, TETRA, SODIUM (PENTAHYDRATE)	1	—	—	—	—	—	
1039 BORON OXIDE	10	—	—	—	—	—	
1040 BORON TRIBROMIDE	—	—	—	—	10	1	
1041 BROMACIL	10	1	—	—	—	—	
1042 BROMINE	0.7	0.1	2	0.3	—	—	
1043 BROMINE PENTAFLUORIDE	0.7	0.1	—	—	—	—	

TABLE Z-4. All Substances Considered by OSHA for Proposed Revision or Addition of Limits[a]

H.S. NO./ CHEMICAL NAME	8-HOUR TWA		SHORT-TERM LIMIT*		CEILING**		SKIN NOTATION
	MG/M3	PPM	MG/M3	PPM	MG/M3	PPM	
1044 BUTANE	1900	800	—	—	—	—	
1045 2-BUTANONE (MEK)	590	200	885	300	—	—	
1046 2-BUTOXY ETHANOL	120	25	—	—	—	—	YES
1047 N-BUTYL ACETATE	710	150	950	200	—	—	
1048 BUTYL ACRYLATE	55	10	—	—	—	—	
1049 SEC-BUTYL ALCOHOL	305	100	455	150	—	—	
1050 TERT-BUTYL ALCOHOL	300	100	450	150	—	—	
1051 N-BUTYL ALCOHOL	—	—	—	—	150	50	YES
1052 N-BUTYL GLYCIDYL ETHER	135	25	—	—	—	—	
1053 N-BUTYL LACTATE	25	5	—	—	—	—	
1054 BUTYL MERCAPTAN	1.5	0.5	—	—	—	—	
1055 O-SEC-BUTYLPHENOL	30	5	—	—	—	—	YES
1056 P-TERT-BUTYLTOLUENE	60	10	120	20	—	—	
1057 CALCIUM CARBONATE	10	—	—	—	—	—	
1058 CALCIUM CYANAMIDE	0.5	—	—	—	—	—	
1059 CALCIUM HYDROXIDE	5	—	—	—	—	—	
1060 CALCIUM OXIDE	2	—	—	—	—	—	
1061 CALCIUM SILICATE, TOTAL DUST	10	—	—	—	—	—	
1062 CALCIUM SULFATE	10	—	—	—	—	—	
1063 CAMPHOR (SYNTHETIC)	12	2	18	3	—	—	

TABLE Z-4. All Substances Considered by OSHA for Proposed Revision or Addition of Limits[a]

H.S. NO./ CHEMICAL NAME	8-HOUR TWA		SHORT-TERM LIMIT*		CEILING**		SKIN NOTATION
	MG/M3	PPM	MG/M3	PPM	MG/M3	PPM	
1064 CAPROLACTAM (DUST)	1	--	3	--	--	--	
1065 CAPROLACTAM (VAPOR)	20	5	40	10	--	--	
1066 CAPTAFOL (DIFOLATAN)	0.1	--	--	--	--	--	YES
1067 CAPTAN	5	--	--	--	--	--	
1068 CARBOFURAN (FURADAN)	0.1	--	--	--	--	--	
1069 CARBON DIOXIDE	9000	5000	54,000	30,000	--	--	
1070 CARBON DISULFIDE[d]	3	1	30	10	--	--	
1071 CARBON MONOXIDE[d]	40	35	--	--	229	200(NDT)	
1072 CARBON TETRABROMIDE	1.4	0.1	4.0	0.3	--	--	
1073 CARBON TETRACHLORIDE[d]	--	--	12.6	2 (60 min)	--	--	
1074 CARBONYL FLUORIDE	5	2	15	5	--	--	
1075 CATECHOL (PYROCATECHOL)	20	5	--	--	--	--	
1076 CELLULOSE	10	--	--	--	--	--	
1077 CESIUM HYDROXIDE	2	--	--	--	--	--	
1078 CHLORINATED CAMPHENE	0.5	--	1	--	--	--	YES
1079 CHLORINE[d]	--	--	1.45	0.5	--	--	
1080 CHLORINE DIOXIDE	0.3	0.1	0.9	0.3	--	--	
1081 1-CHLORO-1-NITROPROPANE	10	2	--	--	--	--	
1082 2-CHLORO-6-TRICHLOROMETHYL PYRIDINE (NITRAPYRIN)	10	--	20	--	--	--	

TABLE Z-4. All Substances Considered by OSHA for Proposed Revision or Addition of Limits[a]

H.S. NO./ CHEMICAL NAME	8-HOUR TWA		SHORT-TERM LIMIT*		CEILING**		SKIN NOTATION
	MG/M3	PPM	MG/M3	PPM	MG/M3	PPM	
1083 CHLOROACETYL CHLORIDE	0.2	0.05	--	--	--	--	
1084 O-CHLOROBENZYLIDENE MALONONITRILE	--	--	--	--	0.4	0.05	YES
1085 CHLORODIFLUOROMETHANE	3500	1000	4375	1250	--	--	
1086 CHLOROFORM[d]	--	--	9.78 (60 min)	2 (60 min)	--	--	
1087 CHLOROPENTAFLUOROETHANE	6320	1000	--	--	--	--	
1088 CHLOROPRENE	35	10	--	--	--	--	YES
1089 O-CHLOROSTYRENE	285	50	430	75	--	--	
1090 O-CHLOROTOLUENE	250	50	375	75	--	--	
1091 CHLORPYRIFOS	0.2	--	0.6	--	--	--	YES
1092 CHROMIC ACID AND CHROMATES[e]	--	--	--	--	0.1	--	
1093 CHROMIUM, METAL	0.5	--	--	--	--	--	
1094 CHROMYL CHLORIDE	0.15	0.025	--	--	--	--	
1095 CLOPIDOL(COYDEN)	10	--	20	--	--	--	
1096 COAL DUST, < 5% QUARTZ, RESPIRABLE FRACTION	2	--	--	--	--	--	
1097 COAL DUST, > 5% QUARTZ, RESPIRABLE QUARTZ FRACTION	0.1	--	--	--	--	--	
1098 COBALT CARBONYL	0.1	--	--	--	--	--	
1099 COBALT HYDROCARBONYL	0.1	--	--	--	--	--	
1100 COBALT, METAL, FUME, DUST	0.05	--	--	--	--	--	
1101 COPPER (FUME)[e]	0.1	--	--	--	--	--	

TABLE Z-4. All Substances Considered by OSHA for Proposed Revision or Addition of Limits[a]

H.S. NO./ CHEMICAL NAME	8-HOUR TWA		SHORT-TERM LIMIT*		CEILING**		SKIN NOTATION
	MG/M3	PPM	MG/M3	PPM	MG/M3	PPM	
1102 CRAG HERBICIDE (SESONE)	10	--	--	--	--	--	
1103 CRUFOMATE	5	--	20	--	--	--	
1104 CYANAMIDE	2	--	--	--	--	--	
1105 CYANOGEN	20	10	--	--	--	--	
1106 CYANOGEN CHLORIDE	--	--	--	--	0.6	0.3	
1107 CYCLOHEXANOL	200	50	--	--	--	--	YES
1108 CYCLOHEXANONE[c]	100	25	--	--	--	--	YES
1109 CYCLOHEXYLAMINE	40	10	--	--	--	--	
1110 CYCLONITE	1.5	--	3	--	--	--	YES
1111 CYCLOPENTANE	1720	600	--	--	--	--	
1112 CYHEXATIN	5	--	--	--	--	--	
1113 DDT[e]	1	--	--	--	--	--	YES
1114 DECABORANE	0.3	0.05	0.9	0.15	--	--	YES
1116 DI-SEC-OCTYL-PHTHALATE	5	--	10	--	--	--	
1117 2,6-DI-TERT-BUTYL-P-CRESOL	10	--	--	--	--	--	
1118 DIAZINON	0.1	--	--	--	--	--	YES
1119 DIBUTYL PHOSPHATE	5	1	10	2	--	--	
1120 2-N-DIBUTYLAMINOETHANOL	14	2	--	--	--	--	YES
1121 1,1-DICHLORO-1-NITROETHANE	10	2	--	--	--	--	
1122 1,3-DICHLORO-5,5-DIMETHYLHYDANTOIN	0.2	--	0.4	--	--	--	

TABLE Z-4. All Substances Considered by OSHA for Proposed Revision or Addition of Limits[a]

H.S. NO./ CHEMICAL NAME	8-HOUR TWA		SHORT-TERM LIMIT*		CEILING**		SKIN NOTATION
	MG/M3	PPM	MG/M3	PPM	MG/M3	PPM	
1123 DICHLOROACETYLENE	--	--	--	--	0.4	0.1	
1125 P-DICHLOROBENZENE	450	75	665	110	--	--	
1126 1,1-DICHLOROETHANE[e]	400	100	--	--	--	--	
1127 DICHLOROETHYL ETHER	30	5	60	10	--	--	YES
1128 DICHLOROMONOFLUOROMETHANE	40	10	--	--	--	--	
1129 1,3-DICHLOROPROPENE	5	1	--	--	--	--	YES
1130 2,2-DICHLOROPROPIONIC ACID	6	1	--	--	--	--	
1131 DICROTOPHOS (BIDRIN)	0.25	--	--	--	--	--	YES
1132 DICYCLOPENTADIENE	30	5	--	--	--	--	
1133 DICYCLOPENTADIENYL IRON	10	--	--	--	--	--	
1134 DIETHANOLAMINE	15	3	--	--	--	--	
1135 DIETHYL KETONE	705	200	--	--	--	--	
1136 DIETHYL PHTHALATE	5	--	--	--	--	--	
1137 DIETHYLAMINE	30	10	75	25	--	--	
1138 DIETHYLENE TRIAMINE	4	1	--	--	--	--	YES
1139 DIGLYCIDYL ETHER (DGE)[b]	0.5	0.1	--	--	--	--	
1140 DIISOBUTYL KETONE[c]	150	25	--	--	--	--	
1141 DIMETHYL 1,2-DIBROMO-2,2-DICHLOROETHYL PHOSPHATE	3	--	--	--	--	--	YES
1142 DIMETHYL SULFATE	0.5	0.1	--	--	--	--	YES
1143 DIMETHYLANILINE	25	5	50	10	--	--	YES

TABLE Z-4. All Substances Considered by OSHA for Proposed Revision or Addition of Limits[a]

H.S. NO./ CHEMICAL NAME	8-HOUR TWA		SHORT-TERM LIMIT*		CEILING**		SKIN NOTATION
	MG/M3	PPM	MG/M3	PPM	MG/M3	PPM	
1144 DINITOLMIDE (3,5-DINITRO-O-TOLUAMIDE)	5	--	--	--	--	--	
1145 DIOXANE (DIETHYLENE DIOXIDE)	90	25	--	--	--	--	YES
1146 DIOXATHION (DELNAV)	0.2	--	--	--	--	--	YES
1147 DIPHENYLAMINE	10	--	--	--	--	--	
1148 DIPROPYL KETONE	235	50	--	--	--	--	
1149 DIPROPYLENE GLYCOL METHYL ETHER	600	100	900	150	--	--	YES
1150 DIQUAT	0.5	--	--	--	--	--	
1151 DISULFIRAM	2	--	--	--	--	--	
1152 DISULFOTON	0.1	--	--	--	--	--	
1153 DIURON	10	--	--	--	--	--	
1154 DIVINYL BENZENE	50	10	--	--	--	--	
1155 EMERY	10	--	--	--	--	--	
1156 ENDOSULFAN	0.1	--	--	--	--	--	YES
1158 EPICHLOROHYDRIN	8	2	--	--	--	--	YES
1159 ETHANOLAMINE	8	3	15	6	--	--	
1160 ETHION (NIALATE)	0.4	--	--	--	--	--	YES
1161 ETHYL ACRYLATE	20	5	100	25	--	--	YES
1162 ETHYL BENZENE	435	100	545	125	--	--	
1163 ETHYL BROMIDE	890	200	1110	250	--	--	
1164 ETHYL ETHER	1200	400	1500	500	--	--	

TABLE Z-4. All Substances Considered by OSHA for Proposed Revision or Addition of Limits[a]

H.S. NO./ CHEMICAL NAME	8-HOUR TWA		SHORT-TERM LIMIT*		CEILING**		SKIN NOTATION
	MG/M3	PPM	MG/M3	PPM	MG/M3	PPM	
1165 ETHYL MERCAPTAN	1	0.5	—	—	—	—	
1166 ETHYL SILICATE	85	10	—	—	—	—	
1167 ETHYLENE CHLOROHYDRIN	—	—	—	—	3	1	YES
1168 ETHYLENE DICHLORIDE (1,2-DICHLOROETHANE)[d]	4	1	8	2	—	—	
1169 ETHYLENE GLYCOL	—	—	—	—	125	50	
1170 ETHYLENE GLYCOL DINITRATE[d]	—	—	0.1 (20 min)	—	—	—	YES
1171 ETHYLIDENE NORBORNENE	—	—	—	—	25	5	
1172 N-ETHYLMORPHOLINE	23	5	—	—	—	—	YES
1173 FENAMIPHOS	0.1	—	—	—	—	—	YES
1174 FENSULFOTHION (DASANIT)	0.1	—	—	—	—	—	
1175 FENTHION	0.2	—	—	—	—	—	YES
1176 FERBAM	10	—	—	—	—	—	
1177 FERROVANADIUM DUST	1	—	3	—	—	—	
1178 FIBROUS GLASS DUST[d]	5	—	—	—	—	—	
1179 FLUORINE	2	1	4	2	—	—	
1180 FLUOROTRICHLOROMETHANE	—	—	—	—	5600	1000	
1181 FONOFOS	0.1	—	—	—	—	—	YES
1182 FORMAMIDE	30	20	45	30	—	—	
1183 FURFURAL	8	2	—	—	—	—	YES

TABLE Z-4. All Substances Considered by OSHA for Proposed Revision or Addition of Limits[a]

H.S. NO./ CHEMICAL NAME	8-HOUR TWA		SHORT-TERM LIMIT*		CEILING**		SKIN NOTATION
	MG/M3	PPM	MG/M3	PPM	MG/M3	PPM	
1184 FURFURYL ALCOHOL	40	10	60	15	--	--	YES
1185 GASOLINE	900	300	1500	500	--	--	
1186 GERMANIUM TETRAHYDRIDE	0.6	0.2	--	--	--	--	
1187 GLUTARALDEHYDE	--	--	--	--	0.8	0.2	
1188 GLYCERIN (MIST)	10	--	--	--	--	--	
1189 GLYCIDOL (2,3-EPOXY-1-PROPANOL)	75	25	--	--	--	--	
1190 GRAIN DUST (OAT, WHEAT, BARLEY)	4	--	--	--	--	--	
1191 GRAPHITE, NATURAL, RESPIRABLE	2.5	--	--	--	--	--	
1191A GRAPHITE, SYNTHETIC	10	--	--	--	--	--	
1192 GYPSUM, TOTAL DUST	10	--	--	--	--	--	
1194 N-HEPTANE	1600	400	2000	500	--	--	
1195 HEXACHLOROBUTADIENE	0.24	0.02	--	--	--	--	YES
1196 HEXACHLOROCYCLOPENTADIENE	0.1	0.01	--	--	--	--	
1197 HEXACHLOROETHANE[e]	10	1	--	--	--	--	YES
1198 HEXAFLUOROACETONE	0.7	0.1	--	--	--	--	YES
1200 N-HEXANE	180	50	--	--	--	--	
1201 HEXANE ISOMERS	1800	500	3600	1000	--	--	
1202 2-HEXANONE	20	5	--	--	--	--	
1203 HEXONE (METHYL ISOBUTYL KETONE)	205	50	300	75	--	--	
1204 HEXYLENE GLYCOL	--	--	--	--	125	25	

TABLE Z-4. All Substances Considered by OSHA for Proposed Revision or Addition of Limits[a]

H.S. NO./ CHEMICAL NAME	8-HOUR TWA		SHORT-TERM LIMIT*		CEILING**		SKIN NOTATION
	MG/M3	PPM	MG/M3	PPM	MG/M3	PPM	
1205 HYDRAZINE	0.1	0.1	—	—	—	—	YES
1206 HYDROGEN BROMIDE	—	—	—	—	10	3	
1207 HYDROGEN CYANIDE[d]	—	—	—	—	5 (10 min)	4.7 (10 min)	YES
1208 HYDROGEN FLUORIDE[d]	—	3	—	6	—	—	
1209 HYDROGEN SULFIDE[b]	14	10	21	15	—	—	
1210 HYDROGENATED TERPHENYLS	5	0.5	—	—	—	—	
1211 2-HYDROXYPROPYL ACRYLATE	3	0.5	—	—	—	—	YES
1212 INDENE	45	10	—	—	—	—	
1213 INDIUM & COMPOUNDS	0.1	—	—	—	—	—	
1214 IODOFORM	10	0.6	—	—	—	—	
1215 IRON OXIDE (DUST AND FUME)	5	—	—	—	—	—	
1216 IRON PENTACARBONYL	0.8	0.1	1.6	0.2	—	—	
1217 IRON SALTS (SOLUBLE)	1	—	—	—	—	—	
1218 ISOAMYL ALCOHOL	360	100	450	125	—	—	
1219 ISOBUTYL ALCOHOL	150	50	—	—	—	—	
1220 ISOOCTYL ALCOHOL	270	50	—	—	—	—	YES
1221 ISOPHORONE[d]	23	4	—	—	—	—	
1222 ISOPHORONE DIISOCYANATE[d]	—	0.005	—	—	—	0.02 (10 min)	

TABLE Z-4. All Substances Considered by OSHA for Proposed Revision or Addition of Limits[a]

H.S. NO./ CHEMICAL NAME	8-HOUR TWA		SHORT-TERM LIMIT*		CEILING**		SKIN NOTATION
	MG/M3	PPM	MG/M3	PPM	MG/M3	PPM	
1223 2-ISOPROPOXYETHANOL	105	25	--	--	--	--	
1224 ISOPROPYL ACETATE	950	250	1185	310	--	--	
1225 ISOPROPYL ALCOHOL	980	400	1225	500	--	--	
1226 ISOPROPYL ETHER[e]	2100	500	--	--	--	--	
1227 ISOPROPYL GLYCIDYL ETHER[b]	240	50	360	75	--	--	
1228 ISOPROPYLAMINE	12	5	24	10	--	--	
1229 N-ISOPROPYLANILINE	10	2	--	--	--	--	YES
1230 KAOLIN, TOTAL DUST	10	--	--	--	--	--	
1231 KETENE	0.9	0.5	3	1.5	--	--	
1232 LIMESTONE, TOTAL DUST	10	--	--	--	--	--	
1233 MAGNESITE, TOTAL DUST	10	--	--	--	--	--	
1234 MAGNESIUM OXIDE FUME	10	--	--	--	--	--	
1235 MALATHION	10	--	--	--	--	--	YES
1236A MANGANESE, FUME	1	--	3	--	--	--	
1237 MANGANESE CYCLOPENTADIENYL TRICARBONYL	0.1	--	--	--	--	--	YES
1238 MANGANESE TETROXIDE	1	--	--	--	--	--	
1239 MARBLE, TOTAL DUST	10	--	--	--	--	--	
1240 MERCURY (ARYL AND INORGANIC COMPOUNDS)[e]	--	--	--	--	0.1	--	
1241 MERCURY (VAPOR)	0.05	--	--	--	--	--	YES
1242 MERCURY, (ORGANO) ALKYL COMPOUNDS	0.01	--	0.03	--	--	--	YES

TABLE Z-4. All Substances Considered by OSHA for Proposed Revision or Addition of Limits[a]

H.S. NO./ CHEMICAL NAME	8-HOUR TWA		SHORT-TERM LIMIT*		CEILING**		SKIN NOTATION
	MG/M3	PPM	MG/M3	PPM	MG/M3	PPM	
1243 MESITYL OXIDE	60	15	100	25	—	—	
1244 METHACRYLIC ACID	70	20	—	—	—	—	
1245 METHOMYL (LANNATE)	2.5	—	—	—	—	—	
1246 METHOXYCHLOR	10	—	—	—	—	—	
1247 4-METHOXYPHENOL	5	—	—	—	—	—	
1248 METHYL 2-CYANOACRYLATE	8	2	16	4	—	—	
1249 METHYL ACETATE	610	200	760	250	—	—	
1250 METHYL ACETYLENE/PROPADIENE MIXTURE	1800	1000	2250	1250	—	—	
1251 METHYL ACRYLONITRILE	3	1	—	—	—	—	YES
1252 METHYL ALCOHOL	260	200	310	250	—	—	YES
1253 METHYL BROMIDE	20	5	—	—	—	—	YES
1254 METHYL CHLORIDE	105	50	205	100	—	—	
1255 METHYL CHLOROFORM (1,1,1-TRICHLOROETHANE)	1900	350	2450	450	—	—	
1256 METHYL DEMETON	0.5	—	—	—	—	—	YES
1257 METHYL ETHYL KETONE PEROXIDE	—	—	—	—	1.5	0.2	
1258 METHYL FORMATE	250	100	375	150	—	—	
1259 METHYL IODIDE	10	2	—	—	—	—	YES
1260 METHYL ISOAMYL KETONE	240	50	—	—	—	—	
1261 METHYL ISOBUTYL CARBINOL	100	25	165	40	—	—	YES
1262 METHYL ISOPROPYL KETONE	705	200	—	—	—	—	

TABLE Z-4. All Substances Considered by OSHA for Proposed Revision or Addition of Limits[a]

H.S. NO./ CHEMICAL NAME	8-HOUR TWA		SHORT-TERM LIMIT*		CEILING**		SKIN NOTATION
	MG/M3	PPM	MG/M3	PPM	MG/M3	PPM	
1263 METHYL MERCAPTAN[b]	1	0.5	—	—	—	—	
1264 METHYL N-AMYL KETONE[e]	465	100	—	—	—	—	
1265 METHYL PARATHION[c]	0.2	—	—	—	—	—	YES
1266 METHYL SILICATE	6	1	—	—	—	—	
1267 ALPHA-METHYL STYRENE	240	50	485	100	—	—	
1268 METHYLCYCLOHEXANE	1600	400	—	—	—	—	
1269 METHYLCYCLOHEXANOL	235	50	—	—	—	—	
1270 O-METHYLCYCLOHEXANONE	230	50	345	75	—	—	YES
1271 METHYLCYCLOPENTADIENYL MN TRICARBONYL	0.2	—	—	—	—	—	YES
1272 METHYLENE BIS (4-CYCLOHEXYLISOCYANATE)	—	—	—	—	0.11	0.01	
1273 4,4'-METHYLENE BIS(2-CHLOROANILINE)	0.22	0.02	—	—	—	—	YES
1275 METRIBUZIN	5	—	—	—	—	—	
1276 MICA	3	—	—	—	—	—	
1277 MINERAL WOOL FIBER	10	—	—	—	—	—	
1278 MOLYBDENUM (INSOLUBLE COMPOUNDS)	10	—	—	—	—	—	
1279 MONOCROTOPHOS (AZODRIN)	0.25	—	—	—	—	—	
1280 MONOMETHYL ANILINE	2	0.5	—	—	—	—	YES
1281 MORPHOLINE	70	20	105	30	—	—	YES
1282 NAPHTHALENE	50	10	75	15	—	—	
1283 NICKEL (SOLUBLE COMPOUNDS)	0.1	—	—	—	—	—	

TABLE Z-4. All Substances Considered by OSHA for Proposed Revision or Addition of Limits[a]

H.S. NO./ CHEMICAL NAME	8-HOUR TWA		SHORT-TERM LIMIT*		CEILING**		SKIN NOTATION
	MG/M3	PPM	MG/M3	PPM	MG/M3	PPM	
1284 NICKEL CARBONYL[e]	0.007	0.001	—	—	—	—	
1286 NITRIC ACID	5	2	10	4	—	—	
1287 P-NITROANILINE	3	—	—	—	—	—	YES
1288 P-NITROCHLOROBENZENE[e]	1	—	—	—	—	—	YES
1289 NITROGEN DIOXIDE[d]	—	—	1.8	1	—	—	
1290 NITROGLYCERIN[d]	—	—	0.1 (20 min)	—	—	—	YES
1291 2-NITROPROPANE	35	10	—	—	—	—	
1292 NITROTOLUENE	11	2	—	—	—	—	YES
1293 NONANE	1050	200	—	—	—	—	
1294 NUISANCE PARTICULATES, TOTAL DUST	10	—	—	—	—	—	
1295 OCTACHLORONAPHTHALENE	0.1	—	0.3	—	—	—	YES
1296 OCTANE	1450	300	1800	375	—	—	
1297 OIL MIST (MINERAL)	5	—	10	—	—	—	
1298 OSMIUM TETROXIDE	0.002	0.0002	0.006	0.0006	—	—	
1299 OXALIC ACID	1	—	2	—	—	—	
1300 OXYGEN DIFLUORIDE	—	—	—	—	0.1	0.05	
1301 OZONE	0.2	0.1	0.6	0.3	—	—	
1302 PARAFFIN WAX FUME	2	—	—	—	—	—	
1303 PARAQUAT, RESPIRABLE DUST	0.1	—	—	—	—	—	YES

TABLE Z-4. All Substances Considered by OSHA for Proposed Revision or Addition of Limits[a]

H.S. NO./ CHEMICAL NAME	8-HOUR TWA		SHORT-TERM LIMIT*		CEILING**		SKIN NOTATION
	MG/M3	PPM	MG/M3	PPM	MG/M3	PPM	
1304 PENTABORANE	0.01	0.005	0.03	0.015	--	--	
1305 PENTAERYTHRITOL, TOTAL DUST	10	--	--	--	--	--	
1306 PENTANE	1800	600	2250	750	--	--	
1307 2-PENTANONE (METHYL PROPYL KETONE)	700	200	875	250	--	--	
1308 PERCHLOROETHYLENE	340	50	1340	200	--	--	
1309 PERCHLORYL FLUORIDE	14	3	28	6	--	--	
1310 PERLITE	10	--	--	--	--	--	
1312 PETROLEUM DISTILLATES (NAPHTHA)	1600	400	--	--	--	--	
1313 PHENOTHIAZINE	5	--	--	--	--	--	YES
1314 PHENYL ETHER (VAPOR)[e]	7	1	--	--	--	--	
1315 PHENYL GLYCIDYL ETHER	6	1	--	--	--	--	
1316 PHENYL MERCAPTAN	2	0.5	--	--	--	--	
1317 PHENYLHYDRAZINE	20	5	45	10	--	--	YES
1318 PHENYLPHOSPHINE	--	--	--	--	0.25	0.05	
1319 PHORATE (THIMET)	0.05	--	0.2	--	--	--	YES
1320 PHOSDRIN (MEVINPHOS)	0.1	0.01	0.3	0.03	--	--	YES
1321 PHOSPHINE	0.4	0.3	1	1	--	--	
1322 PHOSPHORIC ACID	1	--	3	--	--	--	
1323 PHOSPHORUS OXYCHLORIDE	0.6	0.1	3	0.5	--	--	
1324 PHOSPHORUS PENTASULFIDE	1	--	3	--	--	--	

TABLE Z-4. All Substances Considered by OSHA for Proposed Revision or Addition of Limits[a]

H.S. NO./ CHEMICAL NAME	8-HOUR TWA		SHORT-TERM LIMIT*		CEILING**		SKIN NOTATION
	MG/M3	PPM	MG/M3	PPM	MG/M3	PPM	
1325 PHOSPHORUS TRICHLORIDE	1.5	0.2	3	0.5	—	—	
1326 PHTHALIC ANHYDRIDE	6	1	—	—	—	—	
1327 M-PHTHALODINITRILE	5	—	—	—	—	—	
1328 PICLORAM	10	—	20	—	—	—	
1329 PICRIC ACID	0.1	—	0.3	—	—	—	YES
1330 PIPERAZINE DIHYDROCHLORIDE	5	—	—	—	—	—	
1331 PLASTER OF PARIS, TOTAL DUST	10	—	—	—	—	—	
1332 PLATINUM, METAL	1	—	—	—	—	—	
1333 PORTLAND CEMENT	10	—	—	—	—	—	
1334 POTASSIUM HYDROXIDE	—	—	—	—	2	—	
1335 PROPARGYL ALCOHOL	2	1	—	—	—	—	YES
1336 PROPIONIC ACID	30	10	45	15	—	—	
1337 PROPOXUR (BAYGON)	0.5	—	—	—	—	—	
1338 N-PROPYL ACETATE	840	200	1050	250	—	—	
1339 PROPYL ALCOHOL	500	200	625	250	—	—	YES
1340 N-PROPYL NITRATE	105	25	170	40	—	—	
1341 PROPYLENE DICHLORIDE	350	75	510	110	—	—	
1342 PROPYLENE GLYCOL DINITRATE	0.3	0.05	—	—	—	—	YES
1343 PROPYLENE GLYCOL MONOMETHYL ETHER	360	100	540	150	—	—	
1344 PROPYLENE OXIDE	50	20	—	—	—	—	

TABLE Z-4. All Substances Considered by OSHA for Proposed Revision or Addition of Limits[a]

H.S. NO./ CHEMICAL NAME	8-HOUR TWA		SHORT-TERM LIMIT*		CEILING**		SKIN NOTATION
	MG/M3	PPM	MG/M3	PPM	MG/M3	PPM	
1346 RESORCINOL	45	10	90	20	--	--	
1347 RHODIUM (METAL FUME & INSOLUBLE COMPOUNDS)[e]	0.1	--	--	--	--	--	
1348 RHODIUM (SOLUBLE SALTS)[e]	0.001	--	--	--	--	--	
1349 RONNEL	10	--	--	--	--	--	
1350 ROSIN CORE SOLDER PYROLYSIS PRODUCT (AS HCHO)	0.1	--	--	--	--	--	
1351 ROUGE, TOTAL DUST	10	--	--	--	--	--	
1352 SILICA, AMORPHOUS, DIATOMACEOUS EARTH	6	--	--	--	--	--	
1353 SILICA, AMORPHOUS, PRECIPITATED OR GEL	6	--	--	--	--	--	
1354 SILICA, CRYSTALLINE - CRISTOBALITE	0.05	--	--	--	--	--	
1355 SILICA, CRYSTALLINE QUARTZ, RESPIRABLE	0.1	--	--	--	--	--	
1356 SILICA, CRYSTALLINE TRIDYMITE	0.05	--	--	--	--	--	
1357 SILICA, CRYSTALLINE TRIPOLI (AS QUARTZ DUST)	0.1	--	--	--	--	--	
1358 SILICA, FUSED	0.1	--	--	--	--	--	
1359 SILICON	10	--	--	--	--	--	
1360 SILICON CARBIDE	10	--	--	--	--	--	
1361 SILICON TETRAHYDRIDE	7	5	--	--	--	--	
1362 SILVER (METAL DUST AND FUME)[e]	0.01	--	--	--	--	--	
1363 SOAPSTONE, TOTAL DUST	6	--	--	--	--	--	
1363A SOAPSTONE, RESPIRABLE DUST	3	--	--	--	--	--	
1364 SODIUM AZIDE	--	--	--	--	0.3	0.1	
1365 SODIUM BISULFITE	5	--	--	--	--	--	

TABLE Z-4. All Substances Considered by OSHA for Proposed Revision or Addition of Limits[a]

H.S. NO./ CHEMICAL NAME	8-HOUR TWA		SHORT-TERM LIMIT*		CEILING**		SKIN NOTATION
	MG/M3	PPM	MG/M3	PPM	MG/M3	PPM	
1366 SODIUM FLUOROACETATE	0.05	--	0.15	--	--	--	YES
1367 SODIUM HYDROXIDE	--	--	--	--	2	--	
1368 SODIUM METABISULFITE	5	--	--	--	--	--	
1369 STARCH, TOTAL DUST	10	--	--	--	--	--	
1371 STODDARD SOLVENT	525	100	--	--	--	--	
1372 STYRENE (PHENYLETHYLENE)[c]	215	50	425	100	--	--	
1373 SUBTILISINS (PROTEOLITIC ENZYMES)	--	--	--	--	0.00006	--	
1374 SUCROSE, TOTAL DUST	10	--	--	--	--	--	
1375 SULFUR DIOXIDE	5	2	10	5	--	--	
1376 SULFUR MONOCHLORIDE	--	--	--	--	6	1	
1377 SULFUR PENTAFLUORIDE	--	--	--	--	0.1	0.01	
1378 SULFUR TETRAFLUORIDE	--	--	--	--	0.4	0.1	
1379 SULFURYL FLUORIDE	20	5	40	10	--	--	
1380 SULPROFOS	1	--	--	--	--	--	
1381 TALC (NON-ASBESTIFORM)	2	--	--	--	--	--	
1382 TANTALUM (METAL AND OXIDE DUSTS)	5	--	10	--	--	--	
1383 TEMEPHOS	10	--	--	--	--	--	
1384 TERPHENYLS	--	--	--	--	5	0.5	
1385 1,1,2,2-TETRACHLOROETHANE	7	1	--	--	--	--	YES
1386 TETRAETHYL LEAD[e]	0.075	--	--	--	--	--	YES
1387 TETRAHYDROFURAN	590	200	735	250	--	--	
1388 TETRAMETHYL LEAD[e]	0.075	--	--	--	--	--	YES

TABLE Z-4. All Substances Considered by OSHA for Proposed Revision or Addition of Limits[a]

H.S. NO./ CHEMICAL NAME	8-HOUR TWA		SHORT-TERM LIMIT*		CEILING**		SKIN NOTATION
	MG/M3	PPM	MG/M3	PPM	MG/M3	PPM	
1389 TETRASODIUM PYROPHOSPHATE	5	--	--	--	--	--	
1391 4,4'-THIOBIS (6-TERT-BUTYL-M-CRESOL)	10	--	--	--	--	--	
1392 THIOGLYCOLIC ACID	4	1	--	--	--	--	YES
1393 THIONYL CHLORIDE	--	--	--	--	5	1	
1394 TIN (ORGANIC COMPOUNDS)	0.1	--	--	--	--	--	YES
1395 TIN OXIDE	2	--	--	--	--	--	
1396 TITANIUM DIOXIDE	10	--	--	--	--	--	
1397 TOLUENE	375	100	560	150	--	--	
1398 TOLUENE 2,4-DIISOCYANATE	0.04	0.005	0.15	0.02	--	--	
1399 O-TOLUIDINE	9	2	--	--	--	--	YES
1400 P-TOLUIDINE	9	2	--	--	--	--	YES
1401 M-TOLUIDINE	9	2	--	--	--	--	YES
1402 TRIBUTYL PHOSPHATE	2.5	0.2	--	--	--	--	
1403 1,1,2-TRICHLORO-1,2,2-TRIFLUOROETHANE	7600	1000	9500	1250	--	--	
1404 TRICHLOROACETIC ACID	7	1	--	--	--	--	
1405 1,2,4-TRICHLOROBENZENE	--	--	--	--	40	5	
1406 TRICHLOROETHYLENE[d]	--	25	--	--	--	--	
1407 1,2,3-TRICHLOROPROPANE	60	10	--	--	--	--	YES
1408 TRIETHYLAMINE	40	10	60	15	--	--	
1409 TRIMELLITIC ANHYDRIDE	0.04	0.005	--	--	--	--	
1410 TRIMETHYL PHOSPHITE	10	2	--	--	--	--	

TABLE Z-4. All Substances Considered by OSHA for Proposed Revision or Addition of Limits[a]

H.S. NO./ CHEMICAL NAME	8-HOUR TWA		SHORT-TERM LIMIT*		CEILING**		SKIN NOTATION
	MG/M3	PPM	MG/M3	PPM	MG/M3	PPM	
1411 TRIMETHYLAMINE	24	10	36	15	--	--	
1412 TRIMETHYLBENZENE	125	25	--	--	--	--	
1413 2,4,6-TRINITROTOLUENE (TNT)	0.5	--	--	--	--	--	YES
1414 TRIORTHOCRESYL PHOSPHATE	0.1	--	--	--	--	--	YES
1415 TRIPHENYL AMINE	5	--	--	--	--	--	
1416 TUNGSTEN & COMPOUNDS (INSOLUBLE)	5	--	10	--	--	--	
1417 TUNGSTEN & COMPOUNDS (SOLUBLE)	1	--	3	--	--	--	
1418 URANIUM (INSOLUBLE COMPOUNDS)	0.2	--	0.6	--	--	--	
1419 URANIUM (SOLUBLE COMPOUNDS)[e]	0.05	--	--	--	--	--	
1420 N-VALERALDEHYDE	175	50	--	--	--	--	
1421 VANADIUM (V2O5, DUST)[b]	0.05	--	--	--	--	--	
1422 VANADIUM (V2O5, FUME)[b]	0.05	--	--	--	--	--	
1423 VEGETABLE OIL MIST	10	--	--	--	--	--	
1424 VINYL ACETATE	30	10	60	20	--	--	
1425 VINYL BROMIDE	20	5	--	--	--	--	
1426 VINYL CYCLOHEXENE DIOXIDE	60	10	--	--	--	--	YES
1427 VINYL TOLUENE[e]	480	100	--	--	--	--	
1428 VINYLIDENE CHLORIDE	20	5	80	20	--	--	
1429 VM & P NAPHTHA	1350	300	--	--	--	--	
1430 WELDING FUMES (TOTAL PARTICULATE)	5	--	--	--	--	--	
1430a WOOD DUST, HARD WOOD	1	--	--	--	--	--	

TABLE Z-4. All Substances Considered by OSHA for Proposed Revision or Addition of Limits[a]

H.S. NO./ CHEMICAL NAME	8-HOUR TWA		SHORT-TERM LIMIT*		CEILING**		SKIN NOTATION
	MG/M3	PPM	MG/M3	PPM	MG/M3	PPM	
1430b WOOD DUST, SOFT WOOD	5	—	10	—	—	—	
1431 XYLENE (O,M,P-ISOMERS)	435	100	655	150	—	—	
1432 M-XYLENE-ALPHA,ALPHA'-DIAMINE	—	—	—	—	0.1	—	YES
1433 XYLIDINE	10	2	—	—	—	—	YES
1434 ZINC STEARATE	10	—	20	—	—	—	
1435 ZINC CHLORIDE FUME	1	—	2	—	—	—	
1436 ZINC CHROMATES (CrVI)[e]	—	—	—	—	0.1	—	
1437 ZINC OXIDE (FUME)	5	—	10	—	—	—	
1438 ZINC OXIDE, TOTAL DUST	10	—	—	—	—	—	
1439 ZIRCONIUM COMPOUNDS	5	—	10	—	—	—	

[a] All proposed limits derive from the 1987-88 ACGIH TLVs, unless otherwise noted.

[b] Proposed limit is based on 1987-88 ACGIH TLV because NIOSH REL for this substance was judged to be essentially equivalent to the TLV.

[c] Proposed limit is based both on 1987-88 ACGIH TLV and the current NIOSH REL, which are identical.

[d] Proposed limit is based on NIOSH REL.

[e] Proposal retains existing PEL.

* Duration is 15 minutes, unless otherwise noted.

** Ceiling is a peak not to be exceeded at any time during the working day.

BILLING CODE 4510-26-C

XI. Appendices

Note.—These appendices will not appear in the Code of Federal Regulations.

Appendix A—Sampling and Analytical Methods

The sampling and analytical methods for the substances listed in Section I–E of this preamble are categorized into three groups: (1) Fully Validated Methods, (2) Other Methods, and (3) No Methods. These methods are indicated in the Table in this Appendix. The first Table details fully validated methods, other methods, substances for which there are no identified methods, and detection limits. The second Table identifies the most recent NIOSH Analytical methods.

A. Fully Validated Methods

Fully Validated methods were developed by either NIOSH or OSHA. The criteria used in validating these procedures were developed independently by each agency. There are some differences in validation protocol, but in general similar testing procedures were followed. These methods are widely accepted by the scientific community.

B. Other Methods

Methods in this category have not been subjected to all of the testing procedures required of fully validated methods. Some of these procedures have been taken directly from the scientific literature and may not have been used by OSHA. Some are methods that were validated for a specific analyte and assumed to be applicable to a similar one.

While the precision and accuracy of these methods has not been determined and may not meet the OSHA accuracy requirement (+25% at the 95% confidence level), they are the best procedures currently available at this time. Work at evaluating these procedures is progressing.

C. No Methods

These analytes to not have an adequate sampling method available at OSHA, nor has an appropriate method been found in the available scientific literature.

D. Detection Limits

The values listed under Detection Limits are the lowest air concentrations that can be monitored, based on recommended sample air volumes. Detection limits for the OSHA validated methods are determined during the evaluation. Detection limits are not routinely determined for OSHA in-house methods, nor were they determined in the validated methods of the NIOSH Standards Completion Program. Therefore, the detection limits listed for these methods are estimated.

SAMPLING AND ANALYTICAL METHODS

Number analyte	Validated method	Other method	No method	Det limit [1]	Comments
1. Acetaldehyde	OSHA 68			0.58	
2. Acetic Acid		In-house[2]		6 ppb	DL based on 240 L air vol.
3. Acetic Anhydride	NIOSH 3506			5 mg/M³	DL based on 100 L air vol.
4. Acetone	NIOSH 1300			0.3 ppm	
5. Acetonitrile	NIOSH 1606			1 ppm	
6. Acetylsalicylic Acid (Aspirin)		In-house		2 µg/M³	DL based on 120 L air vol.
7. Acrolein	OSHA 52			2.7 ppb	
8. Acrylamide	OSHA 21			1.3 ppb	
9. Acrylic Acid	OSHA 28			0.014 ppb	
10. Allyl Alcohol	NIOSH 1402			0.5 ppm	
11. Allyl Chloride	NIOSH 1000			0.2 ppm	
12. Allyl Glycidyl Ether (AGE)	NIOSH S346			0.1 ppm	
13. Allyl Propyl Disulfide		In-house		0.16 ppm	
14. alpha-Alumina	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
15. Aluminum					
Metal & oxide—					
Pyro powders					DL based on 480 L air vol.
Welding fumes	GRAV & In-house			0.1 mg/M³	Method does not differentiate different forms of Al.
Soluble Salts—					
Alkyls			XXXX		
16. Amitrole (3-Amino-1,2,4-triazole).		In-house		0.004 mg/M³	DL based on 60 L air vol.
17. Ammonia		In-house		1 ppm	DL based on 24 L air vol.
18. Ammonium Chloride (fume)	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
19. Ammonium Perfluorooctanoate.			XXXX		
20. Ammonium Sulfamate (Ammate).	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
21. Aniline	NIOSH 2002			0.05 ppm	
22. ANTU (alpha-Naphthyl Thiourea).	NIOSH S276			0.01 mg/M³	DL based on 480 L air vol.
23. Arsenic	OSHA ID105			0.0005 mg/M³	DL based on 480 L air vol.
24. Asphalt (Petroleum) Fumes		OSHA 58		0.006 mg/M	Benzene soluble portion.
25. Atrazine		In-house		0.05 mg/M³	DL based on 300 L air vol.
26. Azinphos-methyl		In-house		0.014 mg/M³	DL based on 300 L air vol.
27. Barium Sulfate	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
28. Benomyl		In-house		0.03 mg/M³	DL based on 60 L air vol.
29. Beryllium & compounds	OSHA ID125			0.04 µg/M³	DL based on 480 L air vol.
30. Bismuth Telluride (Se-doped).		In-house		0.02 mg/M³	DL based on 480 L air vol.

SAMPLING AND ANALYTICAL METHODS—Continued

Number analyte	Validated method	Other method	No method	Det limit [1]	Comments
31. Bismuth Telluride (Undoped).	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
32. Borates, Tetra, Sodium Salts.					
Anhydrous					DL based on 480 L air vol. Method is for total Boron and does not differentiate different forms of Boron.
Decahydrate		In-house		0.01 mg/M³	
Pentahydrate					
33. Boron Oxide	GRAVIMETRIC			0.02 mg/sample	Same as nuisance particulate TLV.
34. Boron Tribromide		In-house		0.013 mg/M³	Based on total Br & 15 L air vol.
35. Bromacil		In-house		0.1 mg/M³	DL based on 50 L air vol.
36. Bromine	OSHA ID108			0.09 ppm	DL based on 30 L air vol.
37. Bromine Pentafluoride		In-house		0.009 mg/M³	Based on total Br & 48 L air vol.
38. Butane		In-house		1 ppm	DL based on 10 L air vol.
39. 2-Butanone (MEK)	OSHA 16			1.5 ppm	
40. 2-Butoxy Ethanol	NIOSH 1403			0.02 ppm	DL based on 10 L air vol.
41. n-Butyl Acetate	NIOSH 1450			0.04 ppm	DL based on 10 L air vol.
42. Butyl Acrylate		In-house		0.3 ppm	DL based on 3 L air vol.
43. sec-Butyl Alcohol	NIOSH 1401			0.5 ppm	DL based on 10 L air vol.
44. tert-Butyl Alcohol	NIOSH 1400			0.5 ppm	DL based on 10 L air vol
45. n-Butyl Alcohol	NIOSH 1401			0.5 ppm	DL based on 10 L air vol.
46. n-Butyl Glycidyl ether (BGE)	NIOSH S81			0.5 ppm	DL based on 10 L air vol.
47. n-Butyl Lactate		OSHA 7		0.2 ppm	Use method for Organic Solvents.
48. Butyl Mercaptan	NIOSH S350			0.2 ppm	DL based on 10 L air vol.
49. o-sec-Butylphenol		In-house		0.1 ppm	DL based on 10 L air vol.
50. p-tert-Butyltoluene	NIOSH 1501			0.1 ppm	DL based on 10 L air vol.
51. Calcium Carbonate (Limestone, Marble).	GRAVIMETRIC			0.02 mg/Sample	Same as nuisance particulate TLV.
52. Calcium Cyanamide		In-house		0.02 mg/M³	DL based on sol. Ca & 480 L air vol.
53. Calcium Hydroxide		OSHA ID121		0.002 mg/M³	DL based on total Ca.
54. Calcium Oxide		OSHA ID121		0.002 mg/M³	DL based on total Ca.
55. Calcium Silicate, Total Dust	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
56. Calcium Sulfate (Plaster of Paris).	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
57. Camphor, Synthetic	NIOSH 1301			0.5 ppm	DL based on 10 L air vol.
58. Caprolactam Vapor & Aerosol.		In-house		0.007 mg/M³	DL based on 100 L air vol.
59. Caprolactam Vapor only		In-house		0.007 mg/M³	DL based on 100 L air vol.
60. Captafol (Difolatan)		In-house		0.025 mg/M³	DL based on 120 L air vol.
61. Captan		In-house		0.02 mg/M³	DL based on 60 L air vol.
62. Carbofuran (Furadan)		In-house		0.02 mg/M³	DL based on 300 L air vol.
63. Carbon Dioxide	OSHA ID172			500 ppm	
64. Carbon Disulfide	OSHA-NIOSH 1600			0.1 ppm	DL based on 5 L air vol.
65. Carbon Monoxide		Field Test		0.5 ppm	Use direct reading instrument.
66. Carbon Tetrabromide		In-house		0.01 ppm	DL based on 10 L air vol.
67. Carbon Tetrachloride (Tetrachloromethane).	NIOSH 1003			0.1 ppm	DL based on 15 L air vol.
68. Carbonyl Fluoride		In-house		0.2 mg/M³	Based on total F & 240 L air vol.
69. Catechol (Pyrocatechol)		OSHA 32		0.5 ppm	Use OSHA method of Cresol.
70. Cellulose (paper fiber)	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
71. Cesium Hydroxide		In-house		0.02 mg/M³	DL based on Total Cs & 480 L air vol.
72. Chlorinated Camphene (Toxaphene).	NIOSH S67			0.05 mg/M³	DL based on 15 L air vol.
73. Chlorine	OSHA ID101			0.13 ppm	
74. Chlorine Dioxide		In-house		0.05 ppm	DL based on 29 L air vol.
75. 1-Chloro-1-nitropropane	NIOSH S211			0.5 ppm	
76. 2-Chloro-6-trichloro-methyl Pyridine (Nitrapyrin).		In-house		0.08 mg/M³	DL based on 60 L air vol.
77. Chloroacetyl Chloride		In-house		0.05 ppm	DL based on 10 L air vol.
78. o-Chlorobenzylidene Malonitrile.		NIOSH 304		0.003 mg/M³	DL based on 90 L air vol.
79. Chlorodifluoromethane		NIOSH 1020		1 ppm	Use NIOSH method for 1,1,2-Trichloro-1,2,2-trifluoroethane.
80. Chloroform	OSHA 5			0.11 ppm	
81. Chloropentafluoroethane		NIOSH 1020		1 ppm	Use NIOSH method for 1,1,2-Trichloro-1,2,2-trifluoroethane.
82. beta-Chloroprene	NIOSH 1002			0.5 ppm	
83. o-Chlorostyrene		NIOSH 1003		0.5 ppm	Use NIOSH method for Chlorobenzene.
84 o-Chorotoluene		NIOSH 1003		0.5 ppm	Use NIOSH method for Chlorobenzene.

SAMPLING AND ANALYTICAL METHODS—Continued

Number analyte	Validated method	Other method	No method	Det limit [1]	Comments
85. Chlorpyrifos (Dursban)	OSHA 62			0.23 ppb	DL based on 480 L air vol.
86. Chromic Acid & Chromates	OSHA ID103			<0.4 µg/M³ as CR^{+6}.	DL based on 480 L air vol.
87. Chromium, metal	ID121, ID125			2 µg/M³	DL based on 480 L air vol.
88. Chromyl Chloride		In-house		0.009 mg/M³	Based on Cr^{+6} & 240 L air vol.
89. Clopidol (Coyden)		In-house		0.08 mg/M³	DL based on 60 L air vol.
90. Coal Dust (<5% Quartz)	GRAV & ID142			0.02 mg/M³	Quartz analysis using 400 L air vol.
91. Coal Dust (>5% Quartz)	GRAV & ID142			0.02 mg/M³	Quartz analysis using 400 L air vol.
92. Cobalt as Co Metal Dust & Fume.	ID121, ID125			2 µg/M³	DL based on 480 L air vol.
93. Cobalt Carbonyl as Co		In-house		0.002 mg/M³	Based on total Co & 480 L air vol.
94. Cobalt Hydrocarbonyl as Co		In-house		0.002 mg/M³	Based on total Co & 480 L air vol.
95. Copper, Fume	ID121, ID125			2 µg/M³	DL based on 480 L air vol.
96. Crag Herbicide (Sesone)	NIOSH S356			1.5 mg/M³	DL based on 90 L air vol.
97. Crufomate		In-house		0.03 mg/M³	DL based on 60 L air vol.
98. Cyanamide			XXXX		
99. Cyanogen		In-house		0.08 ppm	DL based on 10 L air vol.
100. Cyanogen Chloride		In-house		0.05 ppm	DL based on 10 L air vol.
101. Cyclohexanol	NIOSH 1402			0.5 ppm	DL based on 10 L air vol.
102. Cyclohexanone	OSHA 1			0.05 ppm	
103. Cyclohexylamine		In-house		0.5 ppm	DL based on 10 L air vol.
104. Cyclonite (RDX)		In-house		0.08 mg/M³	DL based on 80 L air vol.
105. Cyclopentane		NIOSH 1500		0.5 ppm	Use NIOSH method for pentane.
106. Cyhexatin		In-house		0.008 mg/M³	Solvent extract, Sn anal & 480 L air vol.
107. DDT (Dichlorodiphenyl-trichlorcethane).	NIOSH S274			0.01 mg/M³	DL based on 90 L air vol.
108. Decaborane		In-house		0.002 mg/M³	Hot H_20 extract, B anal & 480 L air vol.
109. Demeton (Systox)	NIOSH 5514			0.006 mg/M³	DL based on 480 L air vol.
110. Di-Sec-Octyl Phthalate		In-house		0.01 mg/M³	DL based on 100 L air vol.
111. 2,6-Di-tert-Butyl-p-cresol		NIOSH 226		0.5 ppm	
112. Diazinon	OSHA 62			0.24 ppb	DL based on 480 L air vol.
113. Dibutyl Phosphate		NIOSH 5017		0.3 mg/M³	DL based on 250 L air vol.
114. 2-N-Dibutylaminoethanol		OSHA-NIOSH 2007.		0.5 ppm	
115. 1,1-Dichloro-1-nitroethane	NIOSH 1601			0.5 ppm	
116. 1,3-Dichloro-5,5-dimethyl-hydantoin.		in-house		0.2 mg/mL	Field anal by Chemiluminescence.
117. Dichloroacetylene		NIOSH 1003		0.05 ppm	Use NIOSH method for 1,1-Dichloroethane.
118. o-Dichlorobenzene	NIOSH 1003			0.5 ppm	
119. p-Dichlorobenzene	NIOSH 1003			0.5 ppm	
120. 1,1-Dichloroethane	NIOSH 1003			0.5 ppm	
121. Dichloroethyl Ether	NIOSH 1004			1 ppm	
122. Dichlorofluoromethane	NIOSH 2516			1 ppm	
123. 1, 3-Dichloropropene		NIOSH 1003		0.5 ppm	Use NIOSH method for 1,1-Dichloroethane.
124. 2,2-Dichloropropionic Acid		In-house		0.5 ppm	DL based on 10 L air vol.
125. Dicrotophos (Bidrin)		In-house		0.008 mg/M³	DL based on 240 L air vol.
126. Dicyclopentadiene		In-house		0.04 ppm	DL based on 10 L air vol.
127. Dicyclopentadienyl Iron		ID121, ID125			
128. Diethanolamine		In-house		0.05 ppm	DL based on 10 L air vol.
129. Diethyl Ketone		In-house		0.5 ppm	DL based on 10 L air vol.
130. Diethyl Phthalate		In-house		0.07 mg/M³	DL based on 180 L air vol.
131. Diethylamine	OSHA 41			0.053 ppm	
132. Diethylenetriamine	OSHA 60			4 ppb	
133. Diglycidyl Ether (DGE)					Not present-Sample for Epichlorohydrin & Bisphenol A.
134. Diisobutyl Ketone	NIOSH 1300			0.5 mg/M³	
135. Dimethyl-1,2-dibromo-2,2-dichloroethyl Phosphate.		In-house		0.01 mg/M³	DL based on 480 L air vol.
136. Dimethyl Sulfate		OSHA-NIOSH 301		0.1 mg/M³	DL based on 20 L air vol.
137. Dimethylaniline (N,N-Dimethylaniline).	NIOSH 2002			0.003 ppm	DL based on 20 L air vol.
138. Dinitolmide (3,5-Dinitro-o-Toluamine).		In-house		0.05 mg/M³	DL based on 240 L air vol.
139. Dioxane (Diethylene Dioxide).	NIOSH 1602			0.5 ppm	
140. Dioxathion (Delnav)		In-house		0.03 mg/M³	DL based on 10 L air vol.

SAMPLING AND ANALYTICAL METHODS—Continued

Number analyte	Validated method	Other method	No method	Det limit [1]	Comments
141. Diphenylamine	OSHA 22			1 ug/M³	
142. Dipropyl Ketone		OSHA 7		0.5 ppm	Use OSHA method for Organic Solvents.
143. Dipropylene Glycol Methyl Ether.	NIOSH S69			0.5 ppm	DL based on 10 L air vol.
144. Diquat		In-house		2.8 mg/M³	DL based on 100 L air vol.
145. Disulfiram		In-house		0.02 mg/M³	DL based on 25 L air vol.
146. Disulfoton (Disyston)		In-house		0.002 mg/M³	DL based on 480 L air vol.
147. Diuron		In-house		0.03 mg/M³	DL based on 60 L air vol.
148. Divinyl Benzene		OSHA 9		0.5 ppm	Use OSHA method for Styrene.
149. Emery	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV
150. Enflurane (Ethrane)	OSHA 29			0.04 ppm	DL based on 10 L air vol.
151. Epichlorohydrin	NIOSH 1010			0.1 ppm	DL based on 20 L air vol.
152. Ethanolamine		In-house		0.08 ppm	DL based on 10 L air vol.
153. Ethion (Nialate)		In-house		0.008 mg/M³	DL based on 240 L air vol.
154. Ethrane (Enflurane)	OSHA 29			0.04 ppm	DL based on 10 L air vol.
155. Ethyl Acrylate	NIOSH 1450			0.5 ppm	
156. Ethyl Benzene	NIOSH 1501			0.05 ppm	DL based on 10 L air vol.
157. Ethyl Bromide	NIOSH 1011			0.5 ppm	
158. Ethyl Ether	NIOSH 1610			0.5 ppm	
159. Ethyl Mercaptan		In-house		0.1 ppm	DL based on 20 L air vol.
160. Ethyl Silicate	NIOSH S264			0.5 ppm	
161. Ethylene Chlorohydrin	NIOSH 2513			0.1 ppm	
162. Ethylene Dichloride (1,2-Dichloroethane).	OSHA 3			0.05 ppm	
163. Ethylene Glycol		NIOSH 5500		0.8 ppm	
164. Ethylene Glycol Dinitrate	OSHA 43			0.043 ppm	DL based on 15 L air vol.
165. Ethylidene Norbornene			XXXX		
166. N-Ethylmorpholine	NIOSH S146			0.1 ppm	
167. Fenamiphos		In-house		0.004 mg/M³	DL based on 480 L air vol.
168. Fensulfothion (Dasanit)		In-house		0.025 mg/M³	DL based on 200 L air vol.
169. Fenthion (Tiguvon)		In-house		0.004 mg/M³	DL based on 480 L air vol.
170. Ferbam		In-house		0.08 mg/M³	DL based on 60 L air vol.
171. Ferrovanadium Dust	ID121, ID125			2 μg/M³	DL based on Fe or V & 480 L air vol.
172. Fibrous Glass Dust	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV
173. Fluorine		In-house		0.1 mg/M³	As F^-, DL based on 240 L air vol.
174. Fluorotrichloromethane	NIOSH 1006			1 pm	DL based on 3 L air vol.
175. Fonofos		In-house		0.004 mg/M³	DL based on 480 L air vol.
176. Formamide		In-house		0.5 ppm	DL based on 10 L air vol.
177. Furfural		In-house		0.1 ppm	DL based on 10 L air vol.
178. Furfuryl Alcohol	NIOSH S365			0.5 ppm	
179. Gasoline		In-house		0.9 mg/M³	DL based on 10 L air vol.
180. Germanium Tetrahydride		In-house		0.0008 mg/M³	As Ge by (HGA AAS) & 240 L air vol.
181. Glutaraldehyde	OSHA 64			4.4 ppb	
182. Glycerin (Mist)	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
183. Glycidol (2,3-Epoxy-1-propanol).	NIOSH 1608			1 ppm	DL based on 10 L air vol.
184. Grain Dust (oat, wheat, barley).	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
185. Graphite (natural, respirable).	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
186. Gypsum, Total Dust	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
187. Halothane	OSHA 29			0.023 ppm	
188. n-Heptane	NIOSH 1500			0.2 ppm	
189. Hexachlorobutadiene	NIOSH 307			0.2 ppm	DL based on 100 L air vol.
190. Hexachlorocyclopentadiene.		NIOSH 2518		1 ppb	
191. Hexachloroethane	NIOSH 1003			0.5 ppm	DL based on 10 L air vol.
192. Hexafluoroacetone			XXXX		
193. Hexamethylene Diisocyanate.	OSHA 42			0.43 ppb	DL based on 15 L air vol.
194. n-Hexane	NIOSH 1500			0.05 ppm	
195. Hexane Isomers		NIOSH 1500		0.5 ppm	Use NIOSH method for Hexane.
196. 2-Hexanone	NIOSH 1300			0.5 ppm	
197. Hexone (Methyl Isobutyl Ketone).	NIOSH 1300			0.05 ppm	DL based on 10 L air vol.
198. Hexylene Glycol		In-house		0.5 ppm	DL based on 10 L air vol.
199. Hydrazine	OSHA 20			1.2 ppb	
200. Hydrogen Bromide		In-house		0.6 ppb	DL based on 96 L air vol.

SAMPLING AND ANALYTICAL METHODS—Continued

Number analyte	Validated method	Other method	No method	Det limit [1]	Comments
201. Hydrogen Cyanide	OSHA ID120			0.03 ppm	DL based on 90 L air vol.
202. Hydrogen Fluoride		In-house		0.1 ppm	DL based on 240 L air vol.
203. Hydrogen Sulfide	OSHA ID141			0.9 ppm	DL based on 2 L air vol.
204. Hydrogenated Terphenyls		NIOSH 5021		0.1 mg/M³	Use NIOSH method of Terphenyls.
205. 2-Hydroxypropyl Acrylate		NIOSH S43		1 ppm	Use NIOSH method for Methyl Methacrylate.
206. Indene		In-house		0.01 ppm	DL based on 10 L air vol.
207. Indium & Compounds		In-house		0.025 mg/M³	As In. DL based on 480 L air vol.
208. Iodoform		In-house		0.005 ppm	DL based on 10 L air vol.
209. Iron Oxide, (Dust & Fume)	ID121, ID125			3 µg/M³	As Fe_2O_3. DL based on 480 L air vol.
210. Iron Pentacarbonyl (as Fe)		COLORIMETRIC		0.04 mg/M³	DL based on 240 L air vol.
211. Iron Salts, Soluble, as Fe	OSHA ID121			2 µg/M³	As Fe water solubie. DL based on 480 L.
212. Isoamyl Alcohol	NIOSH 1402			0.5 ppm	
213. Isobutyl Alcohol	NIOSH 1401			0.03 ppm	
214. Isooctyl Alcohol		NIOSH 1400		0.5 ppm	Use NIOSH method for Isopropanol.
215. Isophorone	NIOSH 2508			0.07 ppm	DL based on 12 L air vol.
216. Isophorone Diisocyanate		OSHA 42		0.02 mg/M³	DL based on 15 L air vol.
217. 2-Isopropoxyethanol		OSHA 53		0.5 ppm	Use OSHA method for Cellosolve.
218. Isopropyl Acetate	NIOSH S50			0.5 ppm	
219. Isopropyl Alcohol	NIOSH 1400			0.2 ppm	
220. Isopropyl Ether	NIOSH S368			0.5 ppm	
221. Isopropyl Glycidyl Ether (IGE).	NIOSH S77			0.5 ppm	
222. Isopropylamine	NIOSH S147			0.5 ppm	
223. N-Isopropylaniline		NIOSH 2002		0.5 ppm	Use NIOSH method for Dimethylaniline.
224. Kaolin, Total Dust	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
225. Ketene	NIOSH S92			0.2 mg/M³	DL based on 50 L air vol.
226. Limestone, Total Dust	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
227. Magnesite, Total Dust	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
228. Magnesium Oxide fume	OSHA ID121			4 ug/M³	As total Mg, DL based on 480 L air vol.
229. Malathion	OSHA 62			2.2 ppb	DL based on 60 L air vol.
230. Manganese as Mn Dust & Compounds Fume.	ID121, ID125			0.07 mg/M³	As Mn, Method does not distinguish fume from dust.
	ID121, ID125			0.004 mg/M³	
231. Manganese Cyclopentadienyl Tricarbonyl as Mn.		In-house		0.004 mg/M³	Total Mn by AAS, 240 L air vol.
232. Manganese Tetroxide	ID121, ID125			0.004 mg/M³	DL based on total Mn & 30 L air vol.
233. Marble, Total Dust	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
234. Mercury as Hg Alkyl Compounds.			XXXX		
All forms except alkyl vapor	In-house			0.001mg/M³	DL based on 15 L air vol.
Aryl & inorganic compounds.	In-house			0.001 mg/M³	DL based on 15 L air vol.
235. Mesityl Oxide	NIOSH 1301			0.5 ppm	
236. Methacrylic Acid		In-house		0.03 ppm	DL based on 20 L air vol.
237. Methomyl (Lannate)		In-house		0.03 mg/M³	DL based on 60 L air vol & OVS-2 Sampler.
238. Methoxychlor		OSHA-NIOSH S371.		0.5 mg/M³	DL based on 100 L air vol.
239. 4-Methoxyphenol		OSHA 32		0.5 ppm	Use OSHA method for Cresol.
240. Methyl-2-cyanoacrylate	OSHA 55			0.01 ppm	
241. Methyl Acetate	NIOSH S42			0.5 ppm	
242. Methyl Acetylene/Propadiene Mixture (MAPP).		In-house		2.5 ppm	DL based on 5 L air vol.
243. Methyl Acrylonitrile		OSHA 37		0.1 ppm	Use OSHA method for Acrylonitrile.
244. Methyl Alcohol	OSHA-NIOSH 2000			1.5 ppm	
245. Methyl Bromide	NIOSH 2520			0.5 ppm	
246. Methyl Chloride	NIOSH 1001			0.5 ppm	DL based on 10 L air vol.
247. Methyl Chloroform (1,1,1-Trichloroethane).	OSHA 14			0.07 ppm	
248. Methyl Demeton		In-house		0.03 mg/M³	DL based on 60 L air vol.
249. Methyl Ethyl Ketone Peroxide.		In-house		0.05 mg/M³	DL based on 15 L air vol.
250. Methyl Formate	NIOSH S291			1 ppm	DL based on 10 L air vol.
251. Methyl Iodide	NIOSH 1014			0.5 ppm	
252. Methyl Isoamyl Ketone		In-house		0.5 ppm	DL based on 10 L air vol.
253. Methyl Isobutyl Carbinol (Methyl Amyl Alcohol).	NIOSH 1402			0.5 ppm	
254. Methyl Isopropyl Ketone		OSHA 7		0.5 ppm	Use OSHA method for Organic Solvents.

SAMPLING AND ANALYTICAL METHODS—Continued

Number analyte	Validated method	Other method	No method	Det limit [1]	Comments
255. Methyl Mercaptan (Methanethiol).	OSHA 26			0.027 ppm	
256. Methyl-(N-amyl) Ketone	NIOSH 1301			0.5 ppm	
257. Methyl Parathion		In-house		0.002 mg/M³	DL based on 480 L air vol.
258. Methyl Silicate		NIOSH S264		0.5 ppm	Use NIOSH method for Ethyl Silicate.
259. alpha-Methyl Styrene	NIOSH 1501			1 ppm	
260. Methylcyclohexane	NIOSH 1500			0.5 ppm	
261. Methylcyclohexanol	NIOSH S374			0.5 ppm	
262. o-Methylcyclohexanone	NIOSH 2521			0.5 ppm	
263. Methylcyclopentadienyl-Mn-Tricarbonyl as Mn.		In-house		0.004 mg/M³	Total Mn by AAS, & 240 L air vol.
264. Methylene Bis (4-cyclohexylisocyanate).		In-house		0.02 mg/M³	DL based on 15 L air vol.
265. 4,4'-Methylene-Bis-(2-chloroaniline).	OSHA 24			3.6 μg/M³	DL based on 100 L air vol.
266. Methylene Bisphenyl Isocyanate (MDI).	OSHA 47			2.6 μg/M³	DL based on 15 L air vol.
267. Metribuzin		In-house		0.1 mg/M³	DL based on 120 L air vol.
268. Mica	GRAV & ID142	In-house		0.02 mg/M³	Regulated as Quartz if >1% Quartz, 400 L.
269. Mineral Wool Fiber	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
270. Molybdenum Insoluble Compounds, as Mo-.	ID121, ID125			4 μg/M³	as Mo, DL based on 480 L air vol.
271. Monocrotophos		In-house		0.008 mg/M³	DL based on 240 L air vol.
272. Monomethyl Aniline	NIOSH S153			0.1 ppm	
273. Morpholine	NIOSH S150			0.5 ppm	
274. Naphthalene	OSHA 35			80 ppb	
275. Nickel Soluble Compounds as Ni-.	ID121, ID125			4 μg/M³	As Ni, DL based on 480 L air vol.
276. Nickel Carbonyl		In-house		0.002 mg/M³	Based on total Ni & 240 L air vol.
277. Nickel Sulfide Roasting, Fume & Dust, as Ni-.		OSHA ID121		4 μg/M³	As Ni, DL based on 480 L air vol.
278. Nitric Acid		ID127		0.5 ppb	DL based on 96 L air vol.
279. p-Nitroaniline	NIOSH S7			0.5 ppm	
280. p-Nitrochlorobenzene	NIOSH 2005			0.1 ppm	
281. Nitrogen Dioxide		ID109, ID182		0.2 ppm	DL based on 3 L air vol.
282. Nitroglycerin (NG)	OSHA 43			2 ppb	DL based on 15 L air vol.
283. 2-Nitropropane	OSHA 46			0.025 ppm	
284. Nitrotoluene	NIOSH 2005			0.1 ppm	
285. Nonane		NIOSH 1500		0.2 ppm	Use NIOSH method for Heptane.
286. Nuisance Particulates, Total Dust-.	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
287. Octachloronaphthalene	NIOSH S97			0.05 mg/M³	
288. Octane	NIOSH 1500			0.5 ppm	
289. Oil Mist, (Mineral)	GRAV & In-house			0.1 mg/M³	DL varies based on oil & 480 L air vol.
290. Osmium Tetroxide		In-house		0.002 mg/M³	Neutron Activation Analysis, Total Os.
291. Oxalic Acid		In-house		0.02 mg/M³	DL based on 480 L air vol.
292. Oxygen Difluoride			XXXX		
293. Ozone		In-house		0.01 ppm	Chemiluminescence, direct read.
294. Paraffin Wax fume		In-house		0.5 mg/M³	DL based on 120 L air vol.
295. Paraquat respirable dust-	NIOSH 5003			0.1 mg/M³	DL based on 90 L air vol.
296. Pentaborane		In-house		0.005 mg/M³	Based on B & 240 L air vol.
297. Pentaerythritol, Total Dust-	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
298. Pentane	NIOSH 1500			0.5 ppm	DL based on 10 L air vol.
299. 2-Pentanone (Methyl Propyl Ketone).	NIOSH 1300			0.5 ppm	DL based on 10 L air vol.
300. Perchloroethylene (Tetrachloroethylene).	NIOSH 1003			0.05 ppm	DL based on 10 L air vol.
301. Perchloryl Fluoride		In-house		0.6 mg/M³	Based on F & 240 L air vol.
302. Perlite	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
303. Persulfates			XXXX		
304. Petroleum Distillates (Naphtha).	OSHA 48			<260 mg/M³	DL based on 3 L air vol.
305. Phenothiazine		In-house		0.1 mg/M³	DL based on 240 L air vol.
306. Phenyl Ether (Vapor)	NIOSH S72			0.1 ppm	
307. Phenyl Glycidyl Ether (PGE)-.	NIOSH S74			0.1 ppm	
308. Phenylmercaptan		OSHA 26		0.1 ppm	Use OSHA method for Methyl Mercaptan.
309. Phenylhydrazine	NIOSH S160			5 mg/M³	DL based on 120 L air vol.

SAMPLING AND ANALYTICAL METHODS—Continued

Number analyte	Validated method	Other method	No method	Det limit [1]	Comments
310. Phenylphosphine			XXXX		
311. Phorate (Thimet)		In-house		0.003 mg/M³	DL based on 480 L air vol.
312. Phosdrin (Mevinphos)	NIOSH 2503			0.001 mg/M³	DL based on 240 L air vol.
313. Phosphine	OSHA ID180			0.015 ppm	DL based on 36 L air vol.
314. Phosphoric Acid		OSHA ID111		0.01 mg/M³	DL based on 480 L air vol.
315. Phosphorus Oxychloride		In-house		0.002 mg/M³	DL based on PO_4^{-3} & 240 L air vol.
316. Phosphorus Pentasulfide		In-house		0.025mg/M³	DL based on 60 L air vol.
317. Phosphorus Trichloride		NIOSH 6402		1.1mg/M³	DL based on 24 L air vol.
318. Phthalic Anhydride		In-house		0.38mg/M³	DL based on 27 L air vol.
319. m-Phthalodinitrile		OSHA 37		0.5 mg/M³	Use OSHA method for Acrylonitrile.
320. Picloram (Tordom)		In-house		0.03 mg/M³	DL based on 60 L air vol.
321. Picric Acid (2,4,6-Trinitrophenol).	NIOSH S228			0.04 mg/M³	DL based on 180 L air vol.
322. Piperazine Dihydrochloride		In-house		1 ppm	DL based on 10 L air vol.
323. Plaster of Paris Total Dust	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
324. Platinum Metal		In-house		0.2 μ/M³	DL based on 480 L air vol.
325. Portland Cement	GRAV & ID142			0.02 mg/M³	Use Quartz std if >1% Quartz.
326. Potassium Hydroxide		OSHA ID121		0.03 mg/M³	Bases on water soluble K^+ & 480 L air.
327. Propargyl Alcohol		NIOSH 1400		0.1 ppm	Use NIOSH method for Isopropanol.
328. Propionic Acid		In-house		0.03 ppm	DL based on 10 L air vol.
329. Propoxur (Baygon)		In-house		0.003 mg/M³	DL based on 60 L air vol.
330. n-Propyl Acetate	NIOSH 1450			0.5 ppm	DL based on 10 L air vol.
331. Propyl Alcohol	NIOSH 1401			0.5 ppm	
332. n-Propyl Nitrate	NIOSH S227			1 ppm	
333. Propylene Dichloride	NIOSH 1003			0.5 ppm	
334. 1,2-Propylene Glycol Dinitrate.		OSHA 43		0.2 mg/M³	Use OSHA 43 for EGDN, & 15 L air vol.
335. Propylene Glycol Monomethyl Ether.		OSHA 53		0.5 ppm	Use OSHA method for Cellosolve.
336. Propylene Oxide	NIOSH 1612			0.1 ppm	
337. Pyridine	NIOSH 1613			0.5 ppm	
338. Resorcinol		In-house		0.02 mg/M³	DL based on 120 L air vol.
339. Rhodium Metal		In-house		0.1 μ/M³	DL based on 480 L air vol.
Insoluble Compounds as Rh.		In-house		0.1 μ/M³	DL based on 480 L air vol.
Soluble Compounds as Rh		In-house		0.1 μ/M³	DL based on 480 L air vol.
340. Ronnel		In-house		0.002 mg/M³	DL based on 120 L air vol.
341. Rosin Core Solder Pyrolysis Products, as Formaldehyde.	OSHA 53	In-house			Sample for Formaldehyde (OSHA 53) and Abietic Acid (OSHA In-house) as per Chemical Information File.
342. Rouge, (Total dust)	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
343. Silica—Amorphous Diatomaceous Earth (uncalcined).	GRAV & ID142			0.02 mg/M³	If >1% Quartz, use Quartz standard.
Percipitated Silica-Silica Gel.					
344. Silica—Crystalline Cristobalite.	OSHA ID142			0.05 mg/M³	DL based on 816 L air vol.
Quartz	OSHA ID142			0.02 mg/M³	DL based on 480 L air vol.
Silica, Fused	GRAVIMETRIC			0.02 mg/M³	If >1% Quartz, use Quartz standard.
Tridymite	OSHA ID142			0.05 mg/M³	
Tripoli	OSHA ID142			0.02 mg/M³	Quartz analysis.
345. Silicon	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
346. Silicon Carbide	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
347. Silicon Tetrahydride (Silane).		In-house		0.002 mg/M³	DL based on Si anal & 240 L air vol.
348. Silver Metal	OSHA ID121			0.5 mg/M³	DL based on 480 L air vol.
Soluble Compounds as Ag	OSHA ID121			0.5 μ/M³	DL based on 480 L air vol.
349. Soapstone, Total	GRAV. & ID142			0.02 mg/M³	If >1% Quartz, use Quartz standard.
350. Sodium Azide		In-house		0.06 mg/M³	DL based on 15 L air vol.
351. Sodium Bisulfite		In-house		0.04 mg/M³	Based on Na & 480 L air vol.
352. Sodium Fluoroacetate		In-house		0.04 mg/M³	Based on Na & 480 L air vol.
353. Sodium Hydroxide		OSHA ID121		0.04 mg/M³	Based on Na & 480 L air vol.
354. Sodium Metabisulfite		In-house		0.04 mg/M³	Based on Na & 480 L air vol.
355. Starch, Total Dust	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
356. Stearates	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
357. Stoddard Solvent	OSHA 48			<260 mg/M³	DL based on 3 L air vol.
358. Styrene Monomer (Phenylethylene, Vinyl Benzene).	OSHA 9			3.1 ppm	
359. Subtilisins (Proteolitic Enzymes).			XXXX		

SAMPLING AND ANALYTICAL METHODS—Continued

Number analyte	Validated method	Other method	No method	Det limit [1]	Comments
360. Sucrose, Total Dust	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
361. Sulfur Dioxide	OSHA ID107			0.07 mg/M³	DL based on 24 L air vol.
362. Sulfur Monochloride		In-house		0.01 mg/M³	Based on Cl^- & 15 L air vol.
363. Sulfur Pentafluoride			XXX		
364. Sulfur Tetrafluoride		In-house		0.05 mg/M³	Based on F^- & 15 L air vol.
365. Sulfuryl Fluoride		Field test		0.1 ppm	Miran 1A & 1B: MIN. Detectable Conc.=0.1 ppm at 11.5 um.
366. Sulprofos		In-house		0.03 mg/M³	DL based on 60 L air vol.
367. Talc (Non-Asbestiform)	GRAVIMETRIC	Particle count		0.02 mg/M³	Same as nuisance particulate TLV.
368. Tantalum, Metal & Oxide Dust.	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV
369. Temephos		In-house		0.02 mg/M³	DL based on 60 L air vol.
370. Terphenyls	NIOSH 5021			0.1 ppm	
371. 1,1,2,2-Tetrachloroethane	NIOSH 1019			0.1 ppm	
372. Tetraethyl Lead (as Pb)		In-house		0.004 mg/M³	DL based on 240 L air vol & total Pb.
373. Tetrahydrofuran	NIOSH 1609			1 ppm	
374. Tetramethyl Lead (as Pb)		In-house		0.004 mg/M³	DL based on 240 L air vol & total Pb.
375. Tetrasodium Pyrophosphate.		In-house		0.015 mg/M³	Based on Na & 480 L air vol.
376. Thallium (Soluble Compounds).		OSHA ID121		0.03 mg/M³	DL based on 480 L air vol.
377 4,4'-Thio-bis(6-tertbutyl-m-cresol).		In-house		0.04 mg/M³	DL based on 120 L air vol.
378. Thioglycolic Acid		In-house		0.07 ppm	DL based on 10 L air vol.
379. Thionyl Chloride		In-house		0.01 mg/M³	Based on Cl^- & 15 L air vol.
380. Tin Oxide		In-house		0.03 mg/M³	DL based on 480 L air vol.
Organic compounds, as Sn		In-house		0.01 mg/M³	Only Org-Sn's listed in Chem Info File.
381. Titanium Dioxide	GRAVIMETRIC			0.02 mg/M³	Same as nuisance particulate TLV.
382. Toluene	NIOSH 4000			0.02 ppm	DL based on 10 L air vol.
383. Toluene-2,4-diisocyanate (TDI).	OSHA 42			0.36 ppb	DL based on 15 L air vol.
384. o-Toluidine	NIOSH 2002			0.1 ppm	
385. p-Toluidine		NIOSH 2002		0.1 ppm	Use NIOSH method for o-Toluidine.
386. m-Toluidine		NIOSH 2002		0.1 ppm	Use NIOSH method for o-Toluidine.
387. Tributyl Phosphate	OSHA-NIOSH S208			0.5 mg/M³	DL based on 100 L air vol.
388. 1,1,2-Trichloro-1,2,2,-trifluoroethane.	NIOSH 1020			1.4 ppm	DL based on 1.5 L air vol.
389. Trichloroacetic Acid		NIOSH 1603		0.1 ppm	Use NIOSH method for Acetic Acid.
390. 1,2,4-Trichlorobenzene		NIOSH 5517		0.5 ppm	DL based on 10 L air vol.
391. Trichloroethylene	NIOSH 1022,3701			0.2 ppm	DL based on 3 L air vol.
392. 1,2,3-Trichloropropane	NIOSH 1003			0.5 ppm	DL based on 10 L air vol.
393. Triethylamine	NIOSH S152			0.5 ppm	
394. Trimellitic Anhydride		In-house		0.001 mg/M³	DL based on 480 L air vol.
395. Trimethyl Phosphate		In-house		0.5 ppm	DL based on 120 L air vol.
396. Trimethylamine		In-house		0.5 ppm	DL based on 10 L air vol.
397. Trimethylbenzene		In-house		0.02 ppm	DL based on 10 L air vol.
398. 2,4,6-Trinitrotoluene (TNT)	OSHA 44			0.021 mg/M³	
399. Triorthocresyl Phosphate	OSHA-NIOSH S209			0.01 mg/M³	DL based on 100 L air vol.
400. Triphenylamine		In-house		0.5 ppm	DL based on 10 L air vol.
401. Tungsten, as W Insoluble Compounds.		In-hosue		0.002 mg/M³	480 L & Neutron Activation Analysis.
Soluble Compounds		In-house		0.002 mg/M³	480 L & Neutron Activation Analysis.
402. Uranium Insoluble Compounds.		In-house		0.8 μg/M³	DL based on 480 L air vol.
Soluble Compounds		In-house		0.8 μg/M³	DL based on 480 L air vol.
403. N-Valeraldehyde		OSHA 68		1 ppm	Use OSHA method for Acetaldehyde
404. Vanadium, as (V_2O_5) Respirable Dust.	OSHA ID125			0.004 mg/M³	Based on total V analysis & 480 L
Fume	OSHA ID125			0.004 mg/M³	Based on total V analysis & 480 L
405. Vegetable Oil Mist	GRAVIMETRIC				Same as nuisance particulate TLV.
406. Vinyl Acetate	NIOSH 278			0.01 ppm	
407. Vinyl Bromide	OSHA 8			0.2 ppm	
408. Vinyl Cyclohexene Dioxide		In-house		0.5 ppm	DL based on 10 L air vol.
409. Vinyl Toluene	NIOSH 1501			0.5 ppm	
410. Vinylidene Chloride	OSHA 19			0.05 ppm	
411. VM&P Naphtha	OSHA 48			<260 mg/M³	DL based on 3 L air vol.
412. Welding Fumes (Total Particulate).	GRAV. & ID125				See Det. Lim. for specific metals
413. Xylene (o-,m-,p-isomers)	NIOSH 1501			0.02 ppm	DL based on 12 L air vol.

SAMPLING AND ANALYTICAL METHODS—Continued

Number analyte	Validated method	Other method	No method	Det limit [1]	Comments
414. m-Xylene-alpha,alpha'-diamine.		In-house		0.1 ppm	DL based on 10 L air vol.
415. Xylidine	NIOSH 2002			2.5 mg/M³	DL based on 20 L air vol.
416. Zinc Chloride fume		OSHA ID121		0.006 mg/M³	Based on Zn & 480 L air vol.
417. Zinc Stearate		OSHA ID121		0.003 mg/M³	Based on Zn & 480 L air vol.
418. Zinc Chromates, as (CrVI)	OSHA ID103			0.4 µg/M³	As Cr^{+6}. DL based on 480 L air vol.
419. Zinc Oxide Fume	GRAV & ID121,			0.003 mg/M³	Total Zn. Does not differentiate fume & dust.
Dust	ID125	ID143		0.003 mg/M³	
420. Zirconium Compounds	GRAV & ID121			1 mg/M³	DL based on 480 L air vol.

1. Detection Limits (DL) are approximate values based on the analytical procedures recommended air volume or the air volume cited in the "comments" section.
2. "In-house" refers to analytical methods used by OSHA that have not been fully validated by either NIOSH or OSHA. These procedures have been developed by OSHA or were taken from the literature. Some literature methods may not have been used by OSHA as yet.

NIOSH ANALYTICAL METHODS FOR PEL UPDATE

No.	Analyte	NIOSH Validated Method	Other NIOSH Method
1.	Acetaldehyde	NIOSH 3507	
2.	Acetic acid	NIOSH 1603	
3.	Acetic anhydride	NIOSH 3506	
4.	Acetone	NIOSH 1400	
5.	Acetonitrile	NIOSH 1606	
7.	Acrolein	NIOSH 2501	
10.	Allyl alcohol	NIOSH 1402	
11.	Allyl chloride	NIOSH 1000	
12.	Allyl glycidyl ether (AGE)	NIOSH S346	
17.	Ammonia	NIOSH 6701	
20.	Ammonium Sulfamate (Ammate)	NIOSH 5348	
21.	Aniline	NIOSH 2002	
22.	ANTU (Alpha-Naphthyl Thiourea)	NIOSH S276	
23.	Arsenic	NIOSH 7900	
29.	Beryllium & compounds	NIOSH 7102	
33.	Boron Oxide	NIOSH 500, 600	
39.	2-Butanone (MEK)	NIOSH 2500	
40.	2-Butoxy ethanol	NIOSH 1403	
41.	n-Butyl acetate	NIOSH 1450	
43.	sec-Butyl alcohol	NIOSH 1401	
44.	tert-Butyl alcohol	NIOSH 1400	
45.	n-Butyl alcohol	NIOSH 1401	
46.	n-Butyl glycidyl ether (BGE)	NIOSH S81	
48.	Butyl mercaptan	NIOSH S350	
50.	p-tert-Butyltoluene	NIOSH 1501	
51.	Calcium carbonate (Limestone, Marble)	NIOSH 500, 600	
53.	Calcium hydroxide	NIOSH 7020	
54.	Calcium oxide	NIOSH 7020	
56.	Calcium Sulfate (Plaster of Paris)	NIOSH 500, 600	
57.	Camphor, synthetic	NIOSH 1301	
63.	Carbon Dioxide	NIOSH 5249	
64.	Carbon Disulfide	NIOSH 1600	
65.	Carbon Monoxide	NIOSH S340	
67.	Carbon Tetrachloride (Tetrachloromethane)	NIOSH 1003	
70.	Cellulose (paper fiber)	NIOSH 500, 600	
72.	Chlorinated Camphene (Toxaphene)	NIOSH S67	
75.	1-Chloro-1-nitropropane	NIOSH S211	
78.	o-Chlorobenzylidene malonitrile		NIOSH 304
79.	Chlorodifluoromethane		NIOSH 1020
80.	Chloroform	NIOSH 1003	
81.	Chloropentafluoroethane		NIOSH 1020
82.	beta-Chloroprene	NIOSH 1002	
83.	o-Chlorostyrene		NIOSH 1003
84.	o-Chlorostyrene		NIOSH 1003
84.	Chlorostyrene		NIOSH 1003

NIOSH ANALYTICAL METHODS FOR PEL UPDATE—Continued

No.	Analyte	NIOSH Validated Method	Other NIOSH Method
86.	Chromic Acid & Chromates	NIOSH 7600	
87.	Chromium, metal	NIOSH 7024, 7300	
92.	Cobalt as Co Metal dust & fume	NIOSH 7027, 7300	
95.	Copper, Fume	NIOSH 7029, 7200, 7300	
96.	Crag Herbicide (Sesone)	NIOSH S356	
101.	Cyclohexanol	NIOSH 1402	
102.	Cyclohexanone	NIOSH 1300	
105.	Cyclopentane		NIOSH 1500
107.	DDT (Dichlorodiphenyl-trichloroethane)	NIOSH S274	
109.	Demeton (Systox)	NIOSH 5514	
111.	2,6-Di-tert-Butyl-p-cresol		NIOSH 226
113.	Dibutyl Phosphate		NIOSH 5017
114.	2-N-Dibutylaminoethanol		NIOSH 2007
115.	1,1-Dichloro-1-nitroethane	NIOSH 1601	
117.	Dichloroacetylene		NIOSH 1003
118.	o-Dichlorobenzene	NIOSH 1003	
119.	p-Dichlorobenzene	NIOSH 1003	
120.	1,1-Dichloroethane	NIOSH 1003	
121.	Dichloroethyl Ether	NIOSH 1004	
122.	Dichlorfluoromethane	NIOSH 2516	
123.	1,3-Dichloropropene		NIOSH 1003
134.	Diisobutyl ketone	NIOSH 1300	
136.	Dimethyl Sulfate		NIOSH 301
137.	Dimethylaniline (N,N-Dimethylaniline)	NIOSH 2002	
139.	Dioxane (Diethylene Dioxide)	NIOSH 1602	
143.	Dipropylene Glycol Methyl Ether	NIOSH S69	
151.	Epichlorohydrin	NIOSH 1010	
152.	Ethanolamine	NIOSH 2007	
155.	Ethyl Acrylate	NIOSH 1450	
156.	Ethyl Benzene	NIOSH 1501	
157.	Ethyl Bromide	NIOSH 1011	
158.	Ethyl Ether	NIOSH 1610	
160.	Ethyl Silicate	NIOSH S264	
161.	Ethylene Cholorhydrin	NIOSH 2513	
162.	Ethylene Dichloride (1,2-Dichloroethane)	NIOSH 1003	
163.	Ethylene Glycol	NIOSH 5500	
164.	Ethylene Glycol Dinitrate	NIOSH 2507	
166.	N-Ethylmoropholine	NIOSH S146	
174.	Fluorotrichloromethane	NIOSH 1006	
177.	Furfural	NIOSH 2529	
178.	Furfuryl Alcohol	NIOSH S365	
183.	Glycido (2,3-Epoxy-1-propanol)	NIOSH 1608	
185.	Graphite (natural, respirable)	NIOSH 500, 600	
186.	Gypsum, Total dust-n-Heptane	NIOSH 500, 600	
188.	n-Heptane	NIOSH 1500	
189.	Hexachlorobutadiene	NIOSH 307	
190.	Hexachlorocyclopentadiene		NIOSH 308
191.	Hexachloroethane	NIOSH 1003	
194.	n-Hexane	NIOSH 1500	
195.	Hexane Isomers		NIOSH 1500
196.	2-Hexanone	NIOSH 1300	
197.	Hexone (Methyl Isobutyl Ketone	NIOSH 1300	
199.	Hydrazine	NIOSH 3503	
200.	Hydrogen Bromide	NIOSH 7903	
201.	Hydrogen Cyanide	NIOSH 7904	
202.	Hydrogen Fluoride	NIOSH 7903	
204.	Hydrogenated Terphenyls		NIOSH 5021
205.	2-Hydroxypropyl Acrylate		NIOSH S43
212.	Isoamyl Alcohol	NIOSH 1402	
213.	Isobutyl Alcohol	NIOSH 1401	

NIOSH ANALYTICAL METHODS FOR PEL UPDATE—Continued

No.	Analyte	NIOSH Validated Method	Other NIOSH Method
214.	isoctyl Alcohol		NIOSH 1400
215.	Isophorone	NIOSH 2508	
218.	Isopropyl Acetate	NIOSH S50	
219.	Isopropyl Alcohol	NIOSH 1400	
220.	Isopropyl Ether	NIOSH S368	
221.	Isopropyl Glycidyl Ether (IGE)	NIOSH S77	
222.	Isopropylamine	NIOSH S147	
223.	N-Isopropylaniline		NIOSH 2002
225.	Ketene	NIOSH S92	
226.	Limestone, Total Dust	NIOSH 500, 600	
227.	Magnesite, Total Dust	NIOSH 500, 600	
233.	Marble, Total Dust	NIOSH 500, 600	
235.	Mesityl Oxide	NIOSH 1301	
238.	Methoxychlor		NIOSH S371
241.	Methyl Acetate	NIOSH S42	
242.	Methyl Acetylene/Propadiene Mixture (MAPP)	NIOSH S85	
244.	Methyl Alcohol	NIOSH S59	
245.	Methyl Bromide	NIOSH 2520	
246.	Methyl Chloride	NIOSH 1001	
247.	Methyl Chloroform (1,1,1-Tricholorethane)	NIOSH 1003	
249.	Methyl Ethyl Ketone Peroxide		NIOSH 3508
250.	Methyl Formate	NIOSH S291	
251.	Methyl Iodide	NIOSH 1014	
253.	Methyl Isobutyl Carbinol (Methyl Amyl Alcohol)	NIOSH 1402	
256.	Methyl-(N-Amy)Ketone	NIOSH 1301	
258.	Metthyl Sililicate		NIOSH S264
259.	alpha-Methyl Styrene	NIOSH 1501	
260.	Methylcyclohexane	NIOSH 1500	
261.	Methylcyclohexanol	NIOSH S374	
262.	o-Methylcyclohexanone	NIOSH 2521	
269.	Mineral Wool Fiber	NIOSH 500, 600	
272.	Monomethyl Aniline	NIOSH S153	
273.	Morpholine	NIOSH S150	
274.	Naphthalene	NIOSH 1501	
276.	Nickel Carbonyl		NIOSH 6007
278.	Nitric Acid	NIOSH 7903	
279.	p-Nitroaniline	NIOSH S7	
280.	p-Nitrochlorobenzene	NIOSH 2005	
281.	Nitrogen Dioxide		NIOSH 6700
282.	Nitroglycerin (NG)	NIOSH 2507	
283.	2-Nitropropane	NIOSH 2528	
284.	Nitrotoluene	NIOSH 2005	
285.	Nonane		NIOSH 1500
286.	Nuisance Particulates, Total dust	NIOSH 500, 600	
287.	Octachloronaphthalene	NIOSH S97	
288.	Octane	NIOSH 1500	
295.	Paraquat respirable dust	NIOSH 5003	
298.	Pentane	NIOSH 1500	
299.	2-Pentanone (Methyl propl ketone)	NIOSH 1300	
300.	Perchloroethylene (Tetrachloroethylene)	NIOSH 1003	
302.	Perlite	NIOSH 500, 600	
304.	Petroleum Distillates (Naphtha)	NIOSH 1500	
306.	Phenyl Ether (Vapor)	NIOSH S72	
307.	Phenyl Glycidyl Ether (PGE)	NIOSH S74	
309.	Phenylhydrazine	NIOSH S160	
312.	Phosdrin (Mevinphos)	NIOSH 2503	
314.	Phosphoric Acid	NIOSH 7903	
317.	Phosphorus Trichloride		NIOSH 305
321.	Picric Acid (2,4,6-Trinitrophenol)	NIOSH S228	

NIOSH ANALYTICAL METHODS FOR PEL UPDATE—Continued

No.	Analyte	NIOSH Validated Method	Other NIOSH Method
323.	Plaster of Paris Total Dust	NIOSH 500, 600	
327.	Propargyl Alcohol		NIOSH S65
330.	n-Propyl Acetate	NIOSH 1450	
331.	Propyl Alcohol	NIOSH 1401	
332.	n-Propyl Nitrate	NIOSH S227	
333.	Propylene Dichloride	NIOSH 1003	
336.	Propylene Oxide	NIOSH 1612	
337.	Pyridine	NIOSH 1613	
342.	Rouge, (Total Dust)	NIOSH 500, 600	
343.	Silica—Amorphous Diatomaceous earth (uncalcined)—Percipitated silica-Silica gel-		NIOSH 7501
344.	Silica—Crystalline		
	Cristobalite	NIOSH 7500	
	Quartz	NIOSH 7500	
	Tridymite	NIOSH 7500	
	Tripoli-	NIOSH 7500	
345.	Silicon	NIOSH 500, 600	
346.	Silicon Carbide	NIOSH 500, 600	
353.	Sodium Hydroxide		NIOSH 7401
355.	Starch, Total Dust	NIOSH 500, 600	
357.	Stoddard Solvent	NIOSH 1550	
360.	Sucrose, Total Dust	NIOSH 500, 600	
361.	Sulfur Dioxide		NIOSH 6004
367.	Talc (Non-asbestiform)	NIOSH 500, 600	
370.	Terphenyls	NIOSH 5021	
371.	1,1,2,2-Tetrachloroethane	NIOSH 1019	
372.	Tetraethyl Lead (as Pb)	NIOSH 2533	
373.	Tetrahydrofuran	NIOSH 1609	
374.	Tetramethyl Lead (as Pb)	NIOSH 2534	
380.	Tin as Sn-		NIOSH 5504
382.	Toluene	NIOSH 1500, 1501	
383.	Toluene-2,4-diisocyanate (TDI)	NIOSH 2535	
384.	o-Toluidine	NIOSH 2002	
385.	p-Toluidine		NIOSH 2002
386.	m-Toluidine		NIOSH 2002
387.	Tributyl Phosphate	NIOSH S208	
388.	1,1,2-Trichloro-1,2,2-trifluoroethane	NIOSH 1020	
389.	Trichloroacetic Acid		NIOSH 1603
390.	1,2,4-Trichlorobenzene		NIOSH 5517
391.	Trichloroethylene	NIOSH 1022, 3701	
392.	1,2,3-Trichloropropane	NIOSH 1033	
393.	Triethylamine	NIOSH S152	
399.	Triorthocresyl Phosphate	NIOSH S209	
401.	Tungsten, as W		
	Insoluble compounds	NIOSH 7074	
	Soluble compounds	NIOSH 7074	
404.	Vanadium, as (V_2O_5)		
	Respirable dust		NIOSH 7504
	fume		NIOSH 7504
406.	Vinyl Acetate	NIOSH 278	
407.	Vinyl Bromide	NIOSH 1009	
409.	Vinyl Toluene	NIOSH 1501	
410.	Vinylidene Chloride	NIOSH 1015	
411.	VM&P Naphtha	NIOSH 1550	
412.	Welding fumes (Total particulate)	NIOSH 7200	
413.	Xylene (o-,m-,p-isomers)	NIOSH 1501	
415.	Xylidine	NIOSH 2002	
419.	Zinc Oxide		
	Fume	NIOSH 7502	

NIOSH ANALYTICAL METHODS FOR PEL UPDATE—Continued

No.	Analyte	NIOSH Validated Method	Other NIOSH Method
	Dust	NIOSH 7502	

Appendix B—Preliminary Regulatory Impact, Regulatory Flexibility Analysis, and Feasibility Analysis

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Supplement 5—Employee Exposures, by Chemical and Industry, Based Upon Data Contained in OSHA's Integrated Management Information System

Supplement 6—Hazardous Substance Exposure Data Linked to Industrial Processes Identified in the 1988 Sample Survey

Copies of Supplements 2 through 6 are available upon request by calling or writing: Ms. Regina Flahie, Office of Regulatory Analysis, Room N3627, U.S. Department of Labor-OSHA, 200 Constitution Ave. N.W., Washington, DC 20210 (202) 523-7283.

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I. Introduction and Executive Summary

Introduction

The Occupational Safety and Health Administration (OSHA) is proposing to amend its existing air contaminant standards 29 CFR 1910.1000, Tables Z-1, Z-2, and Z-3. The amendments would lower the 8-hour time weighted average permissible exposure limits (PELS) for about 100 substances now listed on the "Z" tables, raise the limit for one chemical, set exposure limits for about 190 substances currently not regulated by OSHA, set short term exposure limits for 70 substances, and, in some cases, set limits for ceiling and skin exposures.

Background

Congress enacted the Occupational Safety and Health Act of 1970 (the Act) to achieve several goals, one of which was to protect workers from occupational health hazards. Congress

acknowledged the role of occupational exposure in the development of diseases, and addressed in the Act the need to quickly establish minimum health standards to control exposure to hazardous substances. To accomplish Congress' intent, OSHA adopted initial exposure limits for approximately 430 chemicals. Four hundred of these exposure limits were based on the recommendations of the American Conference of Governmental Industrial Hygienists (ACGIH), and 21 were from the American National Standards Institute. The list of exposure limits was to be updated, improved, and expanded as new knowledge and techniques were developed. To date OSHA has promulgated extensive health standards for only 24 individual chemicals. The rulemaking under consideration here would set exposure limits for about 430 chemicals based on the 1987–88 Threshold Limit Values of the ACGIH, and recommendations of the National Institute for Occupational Safety and Health (NIOSH) of the U.S. Department of Health and Human Services.

The OSH Act requires the Agency to consider the feasibility of proposed standards. Executive Order 12291 (46 FR 13197) requires that a regulatory analysis be conducted for any rule having major economic consequences on the national economy, individual industries, geographical regions, or levels of government. The Regulatory Flexibility Act (5 U.S.C. 601 *et seq.*) similarly requires OSHA to consider the impact of the proposed regulation on small entities. This analysis covers these requirements.

Approach

Because this rulemaking involves about 430 chemicals, OSHA has prepared the regulatory impact analysis in two phases. Phase I involved the use of a number of secondary data bases to collect information on the chemicals to be regulated and the industries in which they are used. These data bases provided information on the toxicity and health effects of exposure to the chemicals, and current information on engineering controls in use and emergency response procedures. Two data bases provided information on employee exposures. The 1982 National Occupational Exposure Survey (NOES) was based on a sample of about 4,500 businesses. The data base developed from this survey contains an estimate of the number of persons occupationally exposed to hazardous substances by Standard Industrial Classification (SIC). The second data base was OSHA's Integrated Management Information System (IMIS). The IMIS contains the results of air samples taken since 1979 by OSHA industrial hygienists in the course of compliance inspections. Both the NOES an IMIS data bases provided valuable information on the nature and extent of employee exposures to the substances to be regulated; however, they did not provide complete information on all substances. Supplementary information was obtained from industrial hygienists and engineers. These experts identified exposure controls in use and the number and size of plants most likely to be affected by this rulemaking. These sources have provided OSHA with a substantial body of information of chemical use, exposures and controls.

Phase II of the data collection effort involved a sampling survey of over 5,300 firms in industries where chemical exposures were believed to pose potential problems. The survey, conducted during the first part of 1988, gathered data on chemicals, processes, exposures and controls currently in use. These additional data have permitted OSHA to refine the Phase I preliminary estimates of technical and economic feasibility. In addition, site visits to over 100 plants are underway to verify the data collected to date on chemicals, processes, controls, and employee exposures. The reports covering these site visits will be submitted to the docket by July 15, 1988.

OSHA has used contractors to assist in these data collection efforts. Three contractors have supplied expert knowledge on the industries affected and the engineering controls needed to reach the proposed exposure levels. These contractors and Kearney/Centaur Division of A.T. Kearney, Meridian Research, and CONSAD. Fu Associates provided datas base management support during all phases of this project. Washington Consulting Group designed the sample for the surveyed firms and KCA Research conducted the telephone interviews of these firms.

Employee Exposure and Benefits

Revising OSHA's Z-Table limits for hazardous substances is expected to result in reduced risk of chemically-related disease among exposed employees. Exposure to substances included in the rulemaking has been associated with a variety of adverse health effects, including impairment of organ system functions, mucous membrane irritation, neuropathy, narcosis, allergic sensitization, respiratory disease, cardiovascular disease, and cancer.

Using data collected from the survey of over 5,300 establishments, OSHA estimates that between 5.8 million and 6.5 million employees are potentially exposed to the 160 hazardous substances identified in the survey. OSHA also estimates over 1.2 million employees are currently exposed above the proposed exposure limits for these substances. Table I–1 summarizes OSHA's estimates of the number of workers currently at risk of adverse health effects. OSHA estimates that promulgation of the proposed exposure limits will result in a potential reduction of over 11,000 work-related illness cases per year, over 8,000 lost-workday illness cases per year, and over 147,000 lost workdays due to illness per year. OSHA's preliminary estimate is that industry compliance with the proposed exposure limits will result in a reduction of 250 fatalities caused by exposure to substances that cause cancer, respiratory disease, cardiovascular disease, or liver or kidney disease.

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TABLE I-1

ESTIMATED NUMBER OF WORKERS POTENTIALLY AT RISK OF EXPERIENCING ADVERSE EFFECTS, BY TYPE OF ADVERSE EFFECT*

ADVERSE HEALTH EFFECT	NO. OF WORKERS POTENTIALLY EXPOSED TO SUBSTANCES ASSOCIATED WITH EFFECT, MINIMUM ESTIMATE	NO. OF WORKERS POTENTIALLY EXPOSED TO SUBSTANCES ASSOCIATED WITH EFFECT, MAXIMUM ESTIMATE	NO. OF WORKERS EXPOSED ABOVE PROPOSED LIMITS FOR SUBSTANCES, MINIMUM ESTIMATE	NO. OF WORKERS EXPOSED ABOVE PROPOSED LIMITS FOR SUBSTANCES, MAXIMUM ESTIMATE
NUISANCE EFFECTS	4,729,417	5,804,846	829,487	892,725
ODOR AND TASTE EFFECTS	718,522	808,537	59,102	59,102
SYSTEMIC TOXICITY	3,233,319	3,818,742	250,282	256,909
MUCOUS MEMBRANE IRRITATION	12,077,315	15,813,663	59,969	1,021,197
METABOLIC INTERFERENCES	2,277,113	2,345,975	746,879	746,879
LIVER/KIDNEY DISEASE	2,321,573	2,488,604	383,581	384,876
OCULAR DISTURBANCES	194	194	0	0
RESPIRATORY DISEASE	3,765,717	4,023,525	639,717	654,319
CARDIOVASCULAR DISEASE	164,576	164,576	44,355	44,355
NEUROPATHY	1,231,744	1,349,421	201,760	210,203
NARCOSIS	4,601,993	6,381,899	526,021	547,731
CANCER	3,663,053	3,784,374	499,716	499,716
ALLERGIC SENSITIZATION	2,710,576	2,903,153	296,444	297,255

*Double counting of employees simultaneously exposed to more than one substance in different adverse health effects categories, prevents the summation of workers exposed to all adverse health effects in this table.

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Nonregulatory Alternatives

OSHA believes that there are no nonregulatory alternatives that adequately protect most workers from the adverse health effects associated with exposure to the chemicals under consideration. OSHA believes that the tort liability laws and Workers' Compensation do not provide adequate worker protection due to market imperfections. Some employers have not complied with the standards recommended by professional organizations. The deleterious health effects resulting from continued high levels of exposure to hazardous substances require a regulatory solution.

Technological Feasibility

Consistent with OSHA regulations and policy, engineering controls and work practices to control employee exposure are preferred over the use of personal protective equipment.

Engineering controls involve the use of local exhaust ventilation, general ventilation, isolation of the worker and enclosure of the source of emissions, process modifications, equipment modifications, and substitution of non-hazardous chemicals. These methods may be used alone or in combination depending upon the industrial processes involved.

These controls are widely used and will effectively control exposures either by themselves, or coupled with changes in work practices.

Perhaps the most widely used technique for controlling chemical exposure is the use of ventilation. General ventilation uses the movement of air within the general work space to displace or dilute the contaminant with fresh outside air. General ventilation may not be the preferred control method, however, due to the large volumes of air movement required. Local exhaust ventilation uses much smaller volumes of air, exhausted from the point or source at which contaminants are generated.

Isolation involves placing a physical barrier between the hazardous operation and the worker. Many modern, automated manufacturing processes are now fully enclosed in ventilated cabinets. The effectiveness of such a control technique depends on the frequency with which the workers have to enter the enclosure during normal operations. In other situations, rather than placing the process or machine in an enclosure, the worker is placed in an enclosure. Many processes which involve potential chemical exposures are operated remotely by operators in air-conditioned booths isolated from the hazardous materials.

Substitution refers to the replacement of a toxic chemical in a particular process or work area with another, less toxic product. Properly applied, substitution can be a very effective control technique. However, care must be taken to ensure that the proposed substitute performs in a similar manner to the product being replaced. In addition, it is essential that the substitute be carefully evaluated to ensure that in controlling one hazard, another different hazard is not inadvertently introduced. The substitute must also be compatible with existing manufacturing equipment and processes.

The success of these techniques will depend on the physical properties of the chemicals and emissions encountered (boiling point, vapor pressure, etc.) and the process operating conditions. In some cases, particularly with cleaning solvents, substitution may provide the quickest and most effective means of reducing exposure. In other situations, a major effort may be required to alter processes or install or expand local or general dilution ventilation.

OSHA believes that existing engineering controls are available to reduce exposure levels to the new proposed levels. Standard controls have been adapted in numerous situations to solve situation-specific problems in all of the industry sectors affected. Detailed industry-specific illustrations of this point are presented in the Technological Feasibility Chapter of this Preliminary Regulatory Impact Analysis.

Costs of Compliance

Costs of compliance with the proposed rulemaking would result from industry actions to lower workers' chemical exposures to the levels proposed. The 1988 sample survey of more than 5,300 firms was drawn from a universe of over one million firms potentially affected by the rule. Table I–5 at the end of this section presents a list of industries included in the analysis.

Survey respondents verified the number of work stations and workers related to each process, the process location and configuration, the controls already in place, and potential chemical exposures above new proposed levels. Process controls in place were compared to a list of control designs needed to limit exposures to new lower levels. Where the required controls were not reported to be in place, a compliance cost per work station was assigned. Process control costs were summed per establishment and certain maintenance workers were assigned a respirator cost. Costs for the surveyed establishments were then weighted (by SIC and size) to represent compliance costs for the universe of affected plants.

The survey found that about 500,000 establishments reported using the chemicals being regulated. Of this number, about 100,500 would incur some costs to comply with the proposed rule. The total estimated annualized capital plus annual operating costs are $927.83 million. Table I–2 presents the annual cost by industry sector, for large and small (fewer than 20 employees) plants.

Among all industry sectors affected by this proposal, about 101,200 establishments are estimated to incur, on average, an annual cost of $9,200.

Economic Impact

OSHA prepared two estimates of the economic effects of the proposal on potentially affected firms. The two estimates were based upon No Cost-Passthrough ("worst case") and Total Cost-Passthrough ("best case") scenarios.

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TABLE I-2

ANNUAL OPERATING AND ANNUALIZED CAPITAL COST OF COMPLIANCE BY INDUSTRIAL SECTOR (a)

SIC (b)	SIC DESCRIPTION	LARGE PLANTS	SMALL PLANTS	ANNUAL COST
20	FOOD PROD. (c)	$21,704,100	$11,789,000	$33,493,100
21	TOBACCO (c)	$19,700	$0	$19,700
22	TEXT. MILL (c)	$23,308,400	$6,170,000	$29,478,400
23	APPAREL PROD. (c)	$23,604,300	$8,139,900	$31,744,200
24	LUMBER & WOOD	$20,534,800	$175,551,300	$196,086,100
25	FURNITURE	$7,915,300	$6,805,300	$14,720,600
26	PAPER PROD.	$30,966,900	$290,800	$31,257,700
27	PRINTING & PUB.	$15,263,300	$60,425,000	$75,688,300
28	CHEMICAL PROD.	$31,745,500	$8,412,100	$40,157,600
29	PETRO. REFINING	$6,800,700	$414,600	$7,215,300
30	RUBBER & PLASTICS	$53,256,200	$22,225,900	$75,482,100
31	LEATHER PROD.	$1,464,400	$1,115,100 (c)	$2,579,500
32	STONE & CLAY	$15,079,300	$7,463,300	$22,542,600
33	PRIM. METAL	$34,721,200	$5,612,400	$40,333,600
34	FAB. METALS	$50,927,100	$5,010,500	$55,937,600
35	MACHINERY	$43,986,300	$13,754,100	$57,740,400
36	ELEC. MACH.	$24,210,900	$7,570,500	$31,781,400
37	TRANS. EQUIP.	$20,884,600	$26,214,500 (c)	$47,099,100
38	INSTRUMENTS	$10,257,300	$3,207,400	$13,464,700
39	MISC. MANUF.	$13,861,200	$4,334,300	$18,195,500
40	R.R. TRANS.	$532,200	$0	$532,200
45	AIR TRANS.	$1,828,900	$0	$1,828,900
47	TRANS. SERV.	$1,853,100	$0	$1,853,100
49	ELEC. GAS. SAN.	$19,373,900	$3,314,500	$22,688,400
50	WHOLESALE TRADE	$1,416,300	$2,638,300	$4,054,600
51	WHOLESALE, NON-DUR	$3,094,200	$5,764,000	$8,858,200
55	AUTO DEALERS (c)	$9,862,500	$2,092,200	$11,954,700
72	PERSONAL SRV. (c)	$15,648,500	$15,639,300	$31,287,800
73	BUSINESS SRV. (c)	$3,701,700	$5,252,100	$8,953,800
75	AUTO REPAIR (c)	$466,800	$1,044,100	$1,510,900
76	MISC. REPAIR SRV.	$669,500	$4,179,000	$4,848,500
80	HEALTH SERV. (c)	$2,413,000	$2,026,400	$4,439,400
TOTAL		$511,372,100	$416,455,900	$927,828,000

Source: U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.

(a) Costs were calculated by annualizing the capital cost over the projected life of the equipment (10 years) using a 10 percent cost of capital and adding an annual operating and maintenance cost estimated at 10 percent of the capital cost.

(b) Industry sectors not identified in this table include industries with no major cost impact expected, the construction industry, which will be the subject of a separate regulatory analysis, and industries such as mining, over which OSHA has no jurisdiction.

(c) Costs in these sectors were based on expert judgement and secondary data collection. Survey data for SICs 55, 72, 73, 75 and 80 was insufficient to estimate compliance costs.

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In the first scenario it was assumed that all compliance costs would be absorbed by firms in the form of reduced profits. Table I–3 contains a summary of this "worst case" analysis. Under this scenario, the estimated average percent reduction in profits for all affected firms was less than one percent. The estimated reduction in profit of 8 percent for SIC 24, Lumber and Wood Products Manufacturers, was the highest among all industries.

In the second scenario it was assumed that all compliance costs would be passed on to the consumer in the form of higher prices. The potential price increase for an industry sector at the two-digit SIC level was estimated by dividing the sector's compliance cost by its total sales. In this scenario, there would be little impact on market prices; none of the estimated price increases exceeded one-half of one percent (see Table I–4).

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TABLE I-3

ECONOMIC EFFECTS: NO-COST PASSTHROUGH SCENARIO[1]

SIC	Industry	Annual Costs[2] ($ millions)	Total Sales[3] ($ millions)	R.o.R. on Sales (%)[4]	Pre-Reg Profits ($ m)	Post-Reg Profits ($ m)	% Change in Profits
20	FOOD PROD.	33.49	353,780.38	1.9	8,008.04	7,986.29	- 0.2715
21	TOBACCO	0.02	74,030.13	5.3	3,923.60	3,923.59	- 0.0003
22	TEXT. MILL	29.48	60,735.22	2.7	1,765.42	1,747.59	- 1.0100
23	APPAREL PROD.	31.74	74,474.65	2.8	1,813.22	1,793.56	- 1.0845
24	LUMBER & WOOD	196.09	57,994.48	3.9	1,974.51	1,814.21	- 8.1188
25	FURNITURE	14.72	37,648.27	3.5	1,411.02	1,400.96	- 0.7129
26	PAPER PROD.	31.26	103,694.14	3.7	3,778.20	3,761.24	- 0.4489
27	PRINTING & PUB.	75.69	134,830.21	4.8	6,471.85	6,412.25	- 0.9210
28	CHEMICAL PROD.	40.16	272,759.67	3.7	11,738.80	11,714.76	- 0.2048
29	PETRO. REFINING	7.22	196,400.57	2.7	4,964.85	4,960.84	- 0.0808
30	RUBBER & PLASTICS	75.48	86,538.58	4.3	3,423.75	3,376.10	- 1.3918
31	LEATHER PROD.	2.58	15,449.56	2.6	401.69	399.95	- 0.4328
32	STONE & CLAY	22.54	46,094.04	4.1	1,954.99	1,940.73	- 0.7300
33	PRIMARY METALS	40.33	112,564.26	3.3	3,714.62	3,691.27	- 0.6286
34	FAB. METALS	55.94	150,146.41	4.0	6,005.86	5,974.10	- 0.5288
35	MACHINERY	57.74	345,144.89	5.1	17,602.39	17,566.95	- 0.2013
36	ELEC. MACH.	31.78	245,982.70	5.0	12,299.14	12,279.63	- 0.1586
37	TRANS. EQUIP.	47.10	365,427.20	3.9	14,520.25	14,486.69	- 0.2311
38	INSTRUMENTS	13.46	83,359.57	4.9	3,373.26	3,365.00	- 0.2450
39	MISC. MANUF.	18.20	41,870.30	4.4	1,788.56	1,777.39	- 0.6245
40	R.R. TRANS.	.53	43,869.14	10.0	3,969.62	3,969.34	- 0.0072
45	AIR TRANS.	1.83	109,538.08	3.6	3,251.40	3,250.41	- 0.0304
47	TRANS. SERVICES	1.85	12,254.96	2.7	582.18	581.18	- 0.1719
49	ELEC.,GAS & SAN.	22.69	300,254.83	7.0	21,017.84	21,004.06	- 0.0655
50	WHOLESALE TRADE[5]	4.05	13,853.52	2.0	277.07	273.67	- 1.2285
51	WHOLESALE, NON-DUR	8.86	113,848.20	1.5	1,726.26	1,721.48	- 0.2771
55	AUTO DEALERS	11.95	341,574.50	1.9	6,489.92	6,482.81	- 0.1095
72	PERSONAL SERV.	31.29	24,270.74	7.3	1,771.76	1,750.02	- 1.2272
73	BUSINESS SERV.	8.95	22,165.94	6.6	1,462.95	1,455.45	- 0.5126
75	AUTO REPAIR	1.51	45,750.92	5.1	2,492.19	2,491.05	- 0.0457
76	MISC. REPAIR SERV.	4.85	2,665.52	5.5	146.60	142.69	- 2.6696
80	HEALTH SERVICES	4.44	170,234.25	4.5	7,807.72	7,804.54	- 0.0406

Source: U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.

Notes:
1. All values in 1985 dollars.
2. Reproduced from Table VI-1.
3. Dun and Bradstreet, Dun's Marketing Identifiers (DMI) Database.
4. Rate of Return on Sales, Dun and Bradstreet, Industry Norms Database.
5. Consists of SIC 5093 (scrap and waste materials) only.

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Based on this analysis, OHSA concludes that the proposed standard is economically feasible for each sector. The impact on prices is slight and even in the worst cases, the reductions in profitability are small.

Regulatory Flexibility Analysis

In accordance with the Regulatory Flexibility Act (Pub. L. 96–353, 94 Stat. 1664 (5 U.S.C. 601 *et seq.*), OSHA has made a preliminary assessment of how the proposed rulemaking will affect large and small establishments. The results of this preliminary assessment indicate that some small establishments may experience some adverse impact. The smaller profit margins of some small establishments may make it difficult for them to absorb increases in compliance costs. OSHA requests comments on approaches to reduce the impact on small establishments. An important ameliorating factor for each affected firm will be its ability to pass through additional costs to the consumer. The ability of individual firms to do this will be dependent upon product demand elasticities. It is expected that all impacted firms will be able to pass through some portion of their increased costs.

Environmental Impact

The proposed standard has been reviewed in accordance with the requirements of the National Environmental Policy Act of 1969 (NEPA), the Council on Environmental Quality NEPA regulations, and the Department of Labor's NEPA compliance procedures and is not anticipated to have a significant impact on the external environmental.

TABLE I-4.—ECONOMIC EFFECTS: TOTAL-COST PASSTHROUGH

[Dollars in millions]

SIC/industry	Annual costs	Total sales	Costs as a percent of sales
20—Food products	$33.49	$353,780.38	.0095
21—Tobacco	0.02	74,030.13	.0000
22—Text. mill	29.48	60,735.22	.0485
23—Apparel products	31.74	74,474.65	.0426
24—Lumber and wood	196.09	57,994.48	.3381
25—Furniture	14.72	37,648.28	.0391
26—Paper products	31.26	103,694.14	.0301
27—Printing and publishing	75.69	134,830.21	.0561
28—Chemical products	40.16	272,759.67	.0147
29—Petro. refining	7.22	196,400.57	.0037
30—Rubber and plastics	75.48	86,538.58	.0872
31—Leather products	2.58	15,449.56	.0167
32—Stone and clay	22.54	46,094.04	.0489
33—Prim. metals	40.33	112,564.26	.0358
34—Fab. metals	55.94	150,146.41	.0373
35—Machinery	57.74	345,144.89	.0167
36—Elec. mach	31.78	245,982.70	.0129
37—Trans. equip	47.10	365,427.20	.0129
38—Instruments	13.46	83,359.57	.0162
39—Misc. manuf	18.20	41,870.30	.0435
40—R.R. trans	.53	43,869.14	.0012
45—Air trans	1.83	109,538.08	.0017
47—Trans. services	1.85	12,254.96	.0151
49—Elec. gas and san	22.69	300,254.83	.0076
50—Wholesale trade [1]	4.05	13,853.52	.0293
51—Wholesale, non-dur	8.86	113,848.20	.0078
55—Auto dealers	11.95	341,574.50	.0035
72—Personal services	31.29	24,270.74	1289
73—Business services	8.95	22,165.94	.0404
75—Auto repairs	1.51	45,750.92	.0033
76—Misc. repair serv	4.85	2,665.52	1819
80—Health services	4.44	170,234.25	.0026

Source: U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.

[1] Consists of SIC 5093 (scrap and waste materials) only.

Table I-5.—SIC Groups Covered in the OSHA Analysis

Division D. Manufacturing
- Major Group 20. Food and kindred products
- Major Group 21. Tobacco manufactures
- Major Group 22. Textile mill products
- Major Group 23. Apparel and other finished products, made from fabrics and similar materials
- Major Group 24. Lumber and wood products, except furniture
- Major Group 25. Furniture
- Major Group 26. Paper and allied products
- Major Group 27. Printing, publishing, and allied industries
- Major Group 28. Chemicals and allied products
- Major Group 29. Petroleum refining and related industries
- Major Group 30. Rubber and miscellaneous plastics products
- Major Group 31. Leather and leather products
- Major Group 32. Stone, clay, glass, and concrete products
- Major Group 33. Primary metal industries
- Major Group 34. Fabricated metal products, except machinery and transportation equipment
- Major Group 35. Machinery, except electrical
- Major Group 36. Electrical and electronic machinery, equipment, and supplies
- Major Group 37. Transportation equipment
- Major Group 38. Measuring, analyzing, and controlling instruments; photographic, medical and optical goods; watches and clocks
- Major Group 39. Miscellaneous manufacturing industries

Division E. Transportation, Communications, Electric, Gas, and Sanitary Services
- Major Group 40. Railroad transportation
- Major Group 44. Water transportation
- Major Group 45. Transportation by air
- Major Group 47. Transportation services
- Major Group 49. Electric, gas, and sanitary services

Division F. Wholesale Trade
- Major Group 50. Wholesale trade—durable goods
- Major Group 51. Wholesale trade—nondurable goods

Division G. Retail Trade
- Major Group 55. Automotive dealers and gasoline service stations

Division I. Services
- Major Group 72. Personal services
- Major Group 73. Business services
- Major Group 75. Automotive repair, services, and garages
- Major Group 80. Health services

Source: U.S. Department of Labor, OSHA, Office of Regulatory Analysis, as derived from Standard Industrial Classification Manual 1972, Executive Office of the President—Office of Management and Budget [1, pp. 5–7].

The listing excludes the construction industry (SICs 15, 16, and 17) which will be the subject of a separate regulatory analysis.

Respondents:

II. Survey of Affected Industries

Chemicals and other hazardous substances are present to some degree in all industries. However, some industry sectors use chemicals more extensively than others and have controls in place which do not always reduce workers' exposures below permissible exposure levels. This chapter presents an overview of those industries which OSHA believes may experience costs and benefits as a result of this rulemaking. In order to estimate and quantify the potential impact of the rule, a sample survey of over 5,300 establishments was conducted during the first part of 1988. The results of the survey provided the basis for the cost and benefit estimates presented in this preliminary Regulatory Impact Analysis (PRIA). A technical description of the survey is presented in Supplement 1.

In order to determine which industries to include in the sample survey, OSHA relied primarily on two data sources: (1) The NIOSH National Occupational

Exposure Survey (NOES) of 1982 and supplementary information from the NIOSH 1972 survey; and (2) data in the OSHA Integrated Management Information System (IMIS). The 1982 NOES database contains a sample of the number of persons exposed by substance and industry from almost 4500 businesses in 98 different geographic areas in the United States. OSHA's IMIS contains the results of exposure samples taken since 1979 by industrial hygienists during the course of compliance inspections. Using these two databases, industries which are likely to use the substances in this rulemaking at levels which might exceed the proposed exposure limits were identified.

As a check on this list of industries, OSHA contracted with about one dozen industrial hygienists and chemical engineers to review the list. Based on their professional knowledge, these experts verified the industries with potential exposure problems. The final list of industries selected for the sample survey included over 30 two-digit and three-digit SICs where it is believed that chemical exposure potentially exceed the proposed levels.

Industry sectors not included in the survey are those where OSHA believes there is little potential chemical exposure or where existing exposures are well controlled. Industries which were not surveyed for these reasons included finance, real estate, insurance and most services and retail trade sectors. The construction industry was also excluded and will be the subject of a separate rulemaking action. Industries such as mining and certain transportation sectors were not included since other agencies have safety and health enforcement jurisdiction. Certain industry sectors including textile, apparel, food and tobacco products are expected to incur some costs as a result of this rulemaking, but these were not included in the sample survey. The reasons for not including these sectors were a combination of the limits imposed by the sample size, relatively low hazardous substance exposure levels and the availability of adequate information on the engineering controls currently in use in these industries. (Supplement 1 contains a list of the industry sectors included in the sample survey.)

Industrial hygienists and engineers under contract to OSHA also identified the processes used in the industries surveyed, and the chemicals used in those processes. Expected levels of exposure and the number of employees potentially exposed were estimated. The list of processes and chemicals determined to be in common use in each industry sector was subsequently verified in the sample survey. Supplement 2 identifies the processes and chemicals found at the four-digit level for all industries surveyed.

Establishments to be surveyed were selected based on a statistical sample of all establishments in the surveyed industry sectors. For each SIC, establishments were selected from four size categories:

(a) 0–19 employees
(b) 20–29 employees
(c) 100–249 employees
(d) 250 or more employees

This permitted analysis of the effects of the rulemaking by establishment employment size.

About 5,000 completed responses were required to obtain statistically valid results. The field survey was conducted by KCA Research using Computer-Assisted Telephone Interviewing (CATI). Trained interviewers requested data from each establishment regarding production employment, chemical usage, and exposure guidelines in use. Respondents were asked to verify the presence or absence of chemicals and processes believed to be found in establishments in their industry, and were asked to volunteer information on other chemicals not included on the interviewers' "prompt" list of chemicals in use. For each chemical present, the respondent was asked about amounts used, employee exposure levels, and processes where used. For each process, the respondent was asked questions concerning its configuration, frequency of use, and the types of controls and personal protective equipment in use. This information was used to develop the estimates of costs and benefits presented in this PRIA.

The survey results generally corroborated the preliminary assessments of potential industry exposure and overexposure to chemicals. In the sample of over 5,300 firms, about two-thirds reported chemicals being used in the workplace. Most of the firms which reported no chemical usage were small administrative or distribution units of multi-plant companies. Supplement 2 contains a list of the chemicals and processes found in the survey. Based on the survey, OSHA estimates that over 60 percent of production workers in most of the industries surveyed are potentially exposed to chemicals and about 10–15 percent of these would be overexposed at the levels proposed in this rulemaking. Chapter III presents OSHA's estimates of the benefits occuring from a reduction in the number of employees exposed to these chemicals.

The industry profiles that follows present economic information on industry sectors expected to be affected by the rulemaking. Most but not all of these industries were included in the sample survey. Table II–1 at the end of the chapter, contains employment and establishment data for each industry profiled.

SIC 20—Food and Kindred Products

This major industry group includes establishments that manufacture or process food and beverages for human consumption as well as certain related products such as ice, chewing gum, vegetable and animal fats and oils, and prepared animal feeds [1, pp. 59 to 69]. Increased disposable income, low commodity prices, changing demographics, and the 1986 tax reforms are expected to benefit this industry in the next few years. [2, p. 39–1].

Employment and establishment data are shown in Table II–1. The total 1985 value of SIC 20 shipments ($301.6 billion) was 13 percent of the value of all manufacturing industry shipments; this represented the largest share of any two-digit manufacturing industry. The most important industry within SIC 20 is meat products; accounting for 22 percent of the value of shipments, followed by beverages and dairy products, accounting for 14 percent each [3, Vol 1:8].

In 1985, 1.6 million workers in over 29,000 establishments were employed in SIC 20. About 70 percent of these are production workers [Table II–1]. Employment has declined since 1979. The largest employer is the meat products industry, with 23 percent of the workforce in 1986, followed by preserved fruits and vegetables (15 percent) and beverages (13 percent). Meat and miscellaneous food products both experienced 1986 employment levels slightly above the 1979 peak [4]. The largest number of food products establishments are in the manufacturing or processing of miscellaneous foods and meat products (17 percent and 16 percent, respectively).

Establishments in SIC 20 are similar in size to those in the manufacturing industry as a whole, although there is a smaller concentration of very large establishments. Mean establishment size is 55 workers.

Most recent growth by larger food processors has been through business acquisitions rather than internal expansion. The food and beverage sector is becoming more concentrated

and efficient. In most food industries for which data are available, concentration is moderate, with the largest four firms having a 30 percent share of sales. Exceptions can be found in cereal breakfast foods, where the four-firm concentration ratio is just over 75 percent, and in soft drinks, where it is 88 percent [2, pp. 39–1 to 39–39].

In the next few years, most food and beverage producers will benefit from increases in disposable income, favorable trends in consumer purchasing patterns, and continued low commodity prices. Decreased operating costs and expenses have resulted in a 6 percent increase in income in 1985–86 for large food and beverage processors, despite sales gains of only a little more than 1 percent [2, p. 39–1].

In 1985, the median rate of return on assets in the food and kindred products industry was 5.1 percent; this was the third lowest for the 20 two-digit manufacturing industry group. The highest rates of return were registered by the cookie and cracker industry and the blended and prepared flour industry (11.8 percent and 11.1 percent, respectively), followed by the flavoring extracts industry (9.2 percent). At the other extreme, the wine and brandy industry registered a −0.9 percent rate of return on assets in 1985, with an average rate of under 0.1 percent for the 1984–86 period. The cheese and rice milling industries also have very low rates of return on assets (2.2 percent) [5].

SIC 21—Tobacco Manufactures

Establishments in the tobacco manufactures industry produce cigarettes (SIC 211), cigars (SIC 212), chewing and smoking tobacco, and snuff (SIC 213), or they engage in tobacco stemming and redrying (SIC 214) [1, p. 70]. The major worker exposures in these industries are to nuisance dusts generated during the initial handling of tobacco and to chemicals that have been used to treat the tobacco.

Data on employment and establishments for SIC 21 are shown in Table II–1. In 1985, the value of tobacco manufacturing shipments was $18.5 billion, slightly more than 6 percent of the value of shipments for all manufacturing [3, Vol. 1: 8]. SIC 21 has less than 0.3 percent of the total employment or establishments in manufacturing [6, pp. 10, 15]. Three-quarters of the employees in this industry are production workers. The cigarette industry is the most important component of SIC 21, accounting for more than 80 percent of the value of shipments [3, Vol. 1: 8] and 70 percent of employment for this sector, but only 9 percent of establishments [6, p. 15].

Establishments in SIC 21 are large, with a mean size of 296 employees, compared to 55 for all manufacturing. More than half of the establishments in this two-digit SIC have fewer than 20 employees [Table II–1]; and more than 17 percent have 250 or more employees. These large establishments account for more than 85 percent of all employees. The cigarette industry is especially highly concentrated, with a mean establishment size of 2,430 employees. Eleven establishments in the cigarette industry employ 1,000 or more workers, and 99.8 percent of all cigarette manufacturing employees work in these large establishments. Mean establishment sizes in other tobacco industries range from 80 to 135 employees [6, pp. 10, 15]. Employment in the tobacco products industry has declined every year since 1976 (except in 1981), with a total decline in employment of more than 23 percent over the last decade [4].

Most tobacco firms remain profitable because input costs have been relatively stable and prices have increased faster than consumption has declined. The major tobacco companies are continuing to reduce their vulnerability through mergers and diversification [2, pp. 40–1 to 40–7]. Thus, profitablility in the tobacco manufactures industry is good. The 1985 median rate of return on assets (7.7 percent) was the fifth highest median rate of return on assets among firms in the 20 manufacturing industry groups [5].

SIC 22—Textile Mill Products

SIC 22 includes those establishments that perform any of the following six operations: (1) Preparation of fiber and subsequent manufacturing of yarn, thread, braids, twine and cordage; (2) manufacturing broadwoven fabrics, narrow woven fabrics, knit fabrics, and carpets and rugs from yarn; (3) dyeing and finishing fiber, yarn, fabrics, and knit apparel; (4) coating, waterproofing, or otherwise treating fabrics; (5) the integrated manufacturing of knit apparel and other finished articles from yarn; and (6) the manufacture of felt goods, lace goods, nonwoven fabrics, and miscellaneous textiles [1, p. 85].

According to the Department of Commerce, in 1986 shipments for the textile industry increased 4 percent. The value of shipments in 1985 ($53.3 billion) has increased 6 percent since 1981. Employment, however, remained on a long-term downward trend, although the 1986 drop was marginal. An upward trend in output and relatively high operating rates helped to keep the drop in employment to a minimum. Also, average hours worked, which increased in the second half of 1985, continued to rise relatively strongly in 1986 [2, p. 41–1].

Table II–1 presents data on the number of establishments and employment in SIC 22. Similar to other manufacturing industries, the mean establishment size in SIC 22 was 64 employees. Between 1981 and 1985, SIC 22 experienced a 15 percent decrease in employment. In 1985, almost 86 percent of the number of employees were production workers [4]. The median rate of return on assets in the textile mill products industry was 5.6 percent in 1985 [5].

SIC 23—Apparel and Other Products

SIC 23 is referred to as the "cutting-up and needle trades," and includes establishments producing clothing and fabricating products by cutting and sewing purchased woven or knit textile fabrics and related materials. These materials may include leather, rubberized fabrics, plastics, and furs. In addition, establishments that manufacture clothing by cutting and joining materials are included [1, p. 97].

SIC 23 includes three types of apparel establishments: (1) The regular or inside factories, which perform the usual manufacturing functions within their own plant; (2) contract factories, which manufacture apparel from materials owned by others; and (3) apparel jobbers, which buy raw materials, design and prepare samples, arrange for the manufacture of clothing from their materials, and sell the finished product [1, p. 97]. According to U.S. Department of Commerce estimates, the 1986 value of shipments for SIC 23 experienced a growth rate of almost 2 percent over 1985 values [2, p. 42–2].

Between 1980 and 1985, SIC 23 was among the top ten SICs to experience the greatest employment decline. Due to large inventories at both retail and wholesale levels and low consumer demand, there were decreases in both shipments and employment in 1985. In several geographic areas, plants were forced to close. The drop in employment has been attributed to the recent rise of imports into the U.S. market and to improvements in industry efficiency through streamlined operations and increased productivity [2, p. 42–2].

The apparel industry is a major employer of women and minorities, employing more than 6 percent of the manufacturing workforce in plants. Due to intense competition in the industry, profits and wages are lower in this industry than in most other manufacturing industries. The price of labor is the single most important cost

component in the industry, which accounts for the sensitivity that employment levels have to industry growth levels. Production workers make up 85 percent of the apparel work force. Typically, as inventory levels grow, production slows down and employment drops [2, p. 42–2].

In 1986, current-dollar shipments in the apparel industry expanded in value by 3 percent. An increase in consumer demand was the major factor contributing to the upturn. Output levels began to regain former levels of output, and the falling rate of employment of about 1 percent was well below the 3.1 percent annual rate of decline during the 1980–1985 period [2, p. 42–2].

Table II–1 presents employment and establishment for SIC 23 for 1985. During the period of 1981 through 1985, SIC 23 experienced a 10 percent decrease in employment. Almost 84 percent of the total number of employees were production workers [4]. In 1985, the median rate of return on assets in this SIC was 6.3 percent [5].

SIC 24—Lumber and Wood Products

This industry produces logs, pickets and fences, mining timbers, railroad ties, poles and pulpwood. SIC 24 includes establishments that cut timber and pulpwood, merchant sawmills, lath mills, shingle mills, cooperage stock mills, planing mills, and plywood mills and veneer mills engaged in producing lumber and wood basic materials; and establishments that manufacture finished articles made entirely or mainly of wood or related materials [1, p. 107]. According to U.S. Department of Commerce estimates, the logging industry's timber harvest in 1986 was an estimated $8.5 billion, compared with $8.3 billion in 1985 (constant 1982 dollars) [2, p. 4–1].

The Department of Commerce reports that a strong expansion of the market for wood products took place in 1985 due to gains in housing and nonresidential construction activities. Although domestic demand for softwood lumber was strong, Canadian imports displaced American products and contributed to an oversupply, depressing prices. These lower prices lowered U.S. lumber producer profit margins and induced industrywide efforts to restrict imports of lower priced Canadian softwood lumber. In addition to the oversupply caused by the competitive prices in Canada, preventive harvesting to avoid pest damage forced inventories to go up and prices to fall further [2, p. 4–1].

In 1986, similar trends continued in the domestic market for wood products. This was due to a 6 percent rise in housing starts, continued growth in home remodeling and renovation, and strong demand from furniture makers and other end users. However, lower-priced softwood lumber imports from Canada continued to squeeze profits in 1986 [2, p. 4–1].

The Canadian softwood lumber prices brought about a trade agreement in March 1987 between the United States and Canada, in which Canada agreed to set a 15 percent export tax on its softwood lumber. This action has proved to be favorable towards the domestic industry for softwood lumber in the United States. Canadian softwood lumber prices in the United States have risen 3 to 4 percent and imports have decreased about 3 to 4 percent. Since the agreement, Canada's market share has dropped from 33 percent to 28 percent. Market earnings in the domestic industry have increased despite a drop in housing starts, a strong suggestion that the U.S. is benefiting from the higher Canadian prices. It is expected that the trade agreement will keep Canadian softwood prices up and continue to aid the domestic softwood lumber market [7].

Table II–1 presents employment and establishment data form for SIC 24 for 1985, as well as for three of the individual three-digit industry groups. In 1985, the mean establishment size in SIC 24 was 19 employees, which is significantly smaller than the average size in other manufacturing sectors. The median rate of return on assets in this SIC was 7.3 percent [5].

SIC 243—Millwork, Veneer and Plywood

This SIC includes establishments that manufacture fabricated wood millwork, covered with materials such as metal and plastics. According to U.S. Department of Commerce, the value of shipments for SIC 243 was $16.7 billion in 1985, which represents 31 percent of the value of shipments for SIC 24. The value of shipments in SIC 243 increased 21 percent since 1981. In 1985, the number of employees in SIC 243 was about 37 percent of SIC 24. The number of employees in SIC 243 increased by 18 percent in 1985, of which about 83 percent of this total was production workers. Average hourly earnings dropped about 1 percent during that same time period. The number of establishments in SIC 243 in 1985 was about 38 percent of all establishments in SIC 24 [4].

SIC 245—Wood Buildings and Mobile Homes

This SIC includes manufacturers of wood buildings and mobile homes. The 1985 value of shipments for SIC 245 ($6.0 billion) represents 11 percent of the value of shipments for SIC 24 in 1985. The value of shipments in SIC 245 increased 6 percent since 1981. In 1985, the number of employees in SIC 245 was 10.5 percent of SIC 24. The number of employees in SIC 245 increased by 8 percent during this period, of which almost 77 percent of these total employees were production workers. The number of establishments in SIC 245 in 1985 was 4.4 percent of all establishments in SIC 24 [4].

SIC 249

This SIC covers miscellaneous wood products, and includes four four-digit SICs. SIC 249 represents 12 percent of the value of shipments for SIC 24 in 1985. The value of shipments in SIC 249 ($6.6 billion) increased almost 19 percent over 1981. In 1985, the number of employees in SIC 249 was about 15 percent of SIC 24. From 1981 to 1985, the number of employees in SIC 249 decreased by 4 percent, of which almost 84 percent of these employees were production workers. The number of establishments in SIC 249 in 1985 was about 14 percent of all establishments in SIC 24 [4].

SIC 2491 includes establishments that treat wood, sawed or planed in other establishments, with creosote or other preservatives to prevent decay and to protect against fire and insects. This industry also includes the cutting, treating, and selling of poles, posts, and pilings. The Department of Commerce reports that during 1985, there was increased use of treated wood for home improvement projects, such as new decks and all-weather wood foundations. Markets for treated lumber and plywood have been expanding, while markets for treated telephone poles, marine pilings, and railroad ties tend to be cyclical and grow more slowly over the long term. The market for railroad ties in 1985 was strong, as railroads were able to generate enough cash flow to maintain their track systems by replacing worn out ties. In 1986, however, the market for railroad ties declined. About 30 percent of total treated wood shipments are lumber and plywood, of which only about 2 percent has been used for wood foundations [2, p. 4–14]. The Department of Commerce estimated that in 1986, the value of shipments in this industry increased by 5 percent [2, p. 4–14]. SIC 2491 represents 23 percent of the value of shipments for SIC 249 [5]. Employment rose in 1986 by 2.7 percent [2, p. 4–14]. The number of employees in SIC 2491 was almost 16 percent of SIC 249 and

SIC 2491 represents almost 11 percent of all establishments in SIC 249 [5].

SIC 25—Furniture and Fixtures

Manufacturers of household, office, public building, and restaurant furniture and office and store fixtures are included in SIC 25 [1, p. 114]. The U.S. Department of Commerce states that producers of furniture and fixtures recently have been benefiting from lower real interest rates, reduction in the value of the dollar versus other major currencies, and changes in the tax laws. In addition, the U.S. furniture industry is undergoing consolidation, so that big firms are becoming larger and dominating a greater share of the market. The remaining smaller firms are finding it more difficult to compete, given the rapid increase in low-priced imports. Moreover, new manufacturing technologies require large capital investments and large volume, neither of which are readily available to small firms [2, p. 44–2].

For the industry, the value of shipments in 1985 increased by 24 percent since 1981. Although furniture manufacturers anticipate stronger demand in the future, these manufacturers remain uncertain as to the duration and extent of increased demand. Therefore, rather than hiring additional workers, producers have increased the average number of hours worked by current employees. This trend was evident in the wood and metal furniture plants, where average overtime hours increased 16 percent and 24 percent, respectively, in the first half of 1986 [2, p. 44–2].

Table II–1 presents employment and establishment data for SIC 25 for 1985. During this period, SIC 25 experienced a 6 percent growth in employment. Almost 80 percent of the total number of employees working in SIC 25 were production workers. In 1985, the median rate of return on assets in the furniture industry was 7.3 percent.

SIC 26—Paper and Allied Products

Establishments in this industry process fiber from trees, wastepaper, and other fibrous materials into end products that are used by both consumers and industry [1, p. 100]. Based on U.S. Department of Commerce estimates, the paper and allied products industry experienced an increase of 14 percent in the value of shipments from 1981 to 1985, and almost 10 percent between 1985 and 1986. The 1985 value of shipments was $93.4 billion [2, p. 5–1].

Growth patterns in the industry vary, depending on the sector. Some sectors of the industry are more closely related to changes in industrial activity, while others are more directly affected by changes in real personal income or demographic factors. The industry's overall demand patterns are closely linked to rates of change in GNP. In 1985, for example, real growth for the industry was judged to be flat, trailing that of GNP. The largest fluctuations in the industry's shipments have occurred in products geared specifically for commercial-industrial use, which are tied to the annual rate of business activity [2, p. 5–1].

Table II–1 presents employment and establishment data for SIC 26 for 1985. From 1981 to 1985, employment declined by approximately 2 percent. Almost 76 percent of the total number of employees were production workers [4]. In 1985, the median rate of return on assets was 7.4 percent [5].

Within SIC 26, there are six, three-digit SIC groups, which are described below. SIC 261 includes manufacturers of pulp from wood or other materials. The Department of Commerce reports that U.S. market pulp prices dropped nearly 10 percent in the first six months of 1985. The main reasons for the decline included a leveling of demand, high operating rates at market pulp mills, large pulp inventories, and new capacity coming into production. By the end of 1985, however, producers' pulp mill inventories had dropped, helping to stabilize pulp prices. About one-fourth of all market pulp companies either shut down some of their mills in 1985 or curtailed production to reduce the oversupply in the market. In 1986, the industry experienced increased productivity, higher prices and improved worldwide demand. For SIC 261, the value of shipments in 1986 increased by 2.5 percent over 1985. SIC 261 represents 3.4 percent of the value of shipments for SIC 26 [2, p. 5–2].

SIC 262 includes manufacturers of paper from wood pulp and other fiber pulp, and manufacturers of converted paper products. SIC 263 includes manufacturers of paperboard. SIC 263 represents 11 percent of the value of shipments for SIC 26. The value of shipments decreased by 3.6 percent. The number of employees in SIC 263 was less than 1 percent of SIC 26 [4].

SIC 264 includes manufacturers of coated or laminated flexible materials used for packaging purposes. In this sector, the value of shipments, which represents 36 percent of the value of shipments for SIC 26, increased by 17 percent during the same period. The number of employees in SIC 264 was 34 percent of SIC 26 [4].

SIC 265 includes manufacturers of setup paperboard boxes from purchased paperboard. Corrugated boxes have taken the place of wooden shipping containers, pallets, and metal drums in the U.S. packaging market in recent years [2, p. 5–6]. Similarly, consumption of folding boxes continued steadily in 1985. This pattern continued in 1986 with shipments of corrugated boxes increasing 5.5 percent and 3 percent for folding boxes. Several important nondurable end users of folding cartons, such as producers of beverages, dry foods, textiles, sporting goods and toys, hardware, candy, and cosmetics, showed significant declines in real growth in 1985, while the market for boxed paper goods either grew slightly or remained fairly level, [2, p. 5–9].

Manufacturers of sanitary food containers, such as paperboard milk cartons and paper serving and eating utensils, are also included in SIC 265. This industry has been strongly influenced by the shift to plastic containers. Having experienced two successive years of decline, in 1986 the industry increased the value of shipments by 2 percent. Since 1983, the most rapid growth area within the sanitary food container industry has been aseptic packaging. This is specially treated paperboard combined with plastic film and aluminum foil. Sanitary paper products have undergone radical changes in manufacturing in recent years; however, due to the non-discretionary nature of most of this industry's relatively high-priced product line, it has developed a stable base from which to expand.

The value of shipments for SIC 265 increased by 16 percent from 1981 to 1985. This three-digit SIC represents 24 percent of the value of shipments for all of SIC 26. In 1985, the number of employees in SIC 265 was 29 percent of SIC 26 [3].

SIC 266 includes manufacturers of building paper and building board from wood pulp and other fibrous materials. Trends in employment and value of shipments have followed overall trends in SIC 26.

SIC 27—Printing, Publishing, and Allied Industries

This industry is divided into a publishing sector and a printing sector. The publishing sector includes newspaper publishing (SIC 271), periodical publishing (SIC 272), book publishing (SIC 2731) and greeting card publishing (SIC 277). The printing sector includes commercial printing (SIC 275), book printing (SIC 2732), and printing trade services (SIC 279) [1, pp. 106–110].

There were approximately 84,279 establishments in the printing and publishing business in 1985. The

majority of these firms (84.1 percent) had fewer than 20 employees, and the mean establishment size was 17 workers. The firms in SIC 27 employed 1.4 million people [Table II–1]. According to the U.S. Department of Commerce, the value of shipments for all printing and publishing establishments in 1985 was 4.9 percent of the value of shipments for all manufacturing industries. Most of the value of shipments in SIC 27 is from the commercial printing sector (32.4 percent). In 1985, the median rate of return on assets was 8.2 percent for the printing and publishing industry.

Foreign trade has not been a major concern for this industry in the past, but imports are starting to increase at a steady rate. The value of imports and exports was fairly equal in 1986 at $1.3 billion each [2, p. 27–2].

The newspaper industry has maintained slow growth in circulation and advertising over the last several years. Most of the growth in circulation has come from the Sunday edition, while daily circulation fell by 0.6 percent. Sales revenues increased by 8.9 percent, from $14.8 billion in 1986 to $16.2 billion in 1987. Advertising revenues rose slightly, but most of this gain was due to rate increases. Total net worth increased by 17.4 percent from 1986 to 1987 [8].

The periodical industry has experienced growth in both advertising receipts and circulation. Advertising revenue increased over 4 percent in 1986, while circulation revenues increased mainly due to the increase in subscriptions for consumer magazines. There was a large increase in the number of new publications entering the market; over 238 new periodicals were published in 1986 [1, p. 27–6].

Both book publishing and printing showed strong gains over the last several years. Sales and employment increased by 3.5 percent and 2.7 percent, respectively. Spurred by the increase in school enrollment, sales of textbooks accounted for 30 percent of total industry sales. Book printing usually follows the path of book publishing, increasing substantially when book publishing has a strong year [1, pp. 27–9 to 27–13].

Miscellaneous publishing and printing consists of newsletters, catalogs, directories, greeting cards, and business forms. This industry has seen steady gains due in part to the success of mail-order catalogs, telephone directories, and newsletters [1, pp. 27–13 to 27–20].

SIC 28—Chemicals and Allied Products

SIC 28 includes establishments that produce basic chemicals, and establishments that manufacture products using chemical processes. There are three general classes of products: (1) Basic chemicals, such as acids, alkalies, salts, and organic chemicals; (2) chemical products to be used in further manufacturing, such as synthetic fibers, plastics materials, dry colors, and pigments; and (3) finished chemical products to be used for consumption, such as drugs, cosmetics, and soaps; or to be used as materials or supplies in other industries, such as paints, fertilizers, and explosives [1, p. 132].

The chemical and allied products industries have experienced small but steady growth over the recent past. Total shipments by the chemical industry increased approximately 1.2 percent in 1986, following a 1.2 percent gain in 1985. Chemical prices have been somewhat stable in the industry since 1982, due to declining energy costs, which are major factors associated with manufacturing costs. Like many other U.S. industries, various sectors within the chemical industry are undergoing structural changes, such as mergers, plant closings, sales of plants, and other adjustments. This industry employs approximately 5 percent of all industrial workers, but more than 10 percent of all U.S. scientists and engineers. As seen in Table II–1, SIC 28 experienced a 6 percent decline in employment between 1981 and 1985. In 1985, 55.4 percent of the total number of employees in SIC 28 were production workers. The value of shipments increased 8.9 percent during the 1981 to 1985 time period. The median rate of return on assets in the chemical industry was 6.3 percent [5].

Within SIC 28, there are eight, three-digit SICs, which are described below. Most known chemicals that are either produced to be used as an end product, by-product, or effluent, or are used as an input in the production of other substances, can be found in SIC 28. In the following descriptions of four-digit SIC groups, examples of chemicals produced are listed.

SIC 281

This SIC includes establishments that manufacture basic industrial inorganic chemicals. SIC 281 represents 10.3 percent of the value of shipments of SIC 28 in 1985. The value of shipments increased 11.4 percent since 1981, and employment declined by 12 percent. Production workers equaled almost 51 percent of all workers. The number of establishments in SIC 281 was 14.5 percent of all establishments in SIC 28 [Table II–1].

SIC 281 is subdivided into four groups. Examples of the products of each four-digit SIC are given below.

SIC 2812 Products—Chlorine, soda ash, caustic potash, caustic soda, washing soda, and sodium bicarbonate.

SIC 2813 Products—Oxygen, acetylene, argon, carbon dioxide, and hydrogen.

SIC 2816 Products—Color pigments, iron colors, iron oxide, lead oxide pigments, mineral colors, titanium pigments, and zinc oxide pigments.

SIC 2819 Products—Sulfuric, hydrochloric, and hydrofluoric acids.

SIC 282

This SIC includes manufacturers of plastics materials and synthetic resins, synthetic rubbers, and cellulosic and other manmade fibers. Plastics make up a variety of products which are used in diverse markets. Packaging and construction account for over 50 percent of consumption, with the remainder going into the transportation, electronics, and medical industries. SIC 282 represents almost 17 percent of the value of shipments of SIC 28. The value of shipments in SIC 282 increased 8.5 percent over the period 1981 to 1985. Industry shipments of plastics in 1986 gained 6.3 percent as volume rose in response to slightly increased demand for materials. However, declining prices of plastic materials held shipments to a 2 percent increase [2, p. 14–1]

Table II–1 gives employment and establishment data for this segment. The number of employees in SIC 282 in 1985 was almost 16 percent of SIC 28 and the number of establishments was 8 percent of all establishments in that SIC. In 1985, employment in SIC 282 declined by 12 percent, and production workers equaled 66.5 percent of all workers [4].

SIC 282 is subdivided into four groups. Examples of the products from each of these four-digit SICs are given below.

SIC 2821 Products—Cellulose plastics materials, phenolic and other tar acid resins, acrylic resins, polyethylene resins, coumarone-indene and petroleum polymer resins, and casein plastics.

SIC 2822 Products—Copolymers of butadiene and styrene, or butadiene and acrylonitrile, and polybutadienes.

SIC 2823 Products—Cellulose, rayon, and triacetate fibers.

SIC 2824 Products—Fibers of acrylic, acrylonitrile, polyvinyl ester, and nylon.

SIC 283

This group includes establishments that manufacture, fabricate, or process medicinal chemicals and pharmaceutical products. The value of shipments in SIC 283 has increased almost 29 percent since 1981. SIC 283 represents 16 percent of the value of shipments of SIC 28 and almost 20 percent of the number of employees. The U.S. Department of Commerce estimated that the pharmaceutical industry experienced a 6.3 percent increase in the value of shipments in 1986. However, after adjusting for price changes, this growth rate was closer to 1.8 percent. Productivity also increased in 1986, growing by approximately 2.6 percent [2, p. 17–1].

As seen in Table II–1, the number of establishments in SIC 283 was almost 12 percent of all establishments in SIC 28. Employment increased by 3 percent since 1981, and production workers equaled approximately 46 percent of all workers in SIC 283. Agar, vitamins, antibiotics, vaccines, and viruses are examples of the products of this SIC.

SIC 284

This SIC includes manufacturers of detergents, emulsifiers, cosmetic, and producers of glycerin. SIC 284 represents 15 percent of the value of shipments of SIC 28. The value of shipments in SIC 284 increased almost 17 percent from 1981 to 1985. In 1986 the value was estimated at $31 billion, which represents about a 2 percent increase after adjusting for price changes over 1985 values [2, p. 16–1].

The number of employees in this SIC was almost 15 percent of SIC 28 and the number of establishments was almost 22 percent. In 1985, employment in SIC 284 had increased by 1 percent since 1981, and production workers equaled approximately 63 percent of all workers in SIC 284 [4].

There are four subgroups within SIC 284. Examples of the products produced by each four-digit SIC are given below.

SIC 2841 Products—Soap, synthetic organic detergents, inorganic alkaline detergents, and crude and refined glycerin from vegetable and animal fats and oils.

SIC 2842 Products—Household, institutional, and industrial plant disinfectants, non-personal deodorants, dry cleaning preparations, household bleaches, and other sanitation products.

SIC 2843 Products—Textile and leather finishing agents, soluble oils and greases.

SIC 2844 Products—Perfumes, cosmetics, home permanent kits, shampoos, shaving products, and talcum powder.

SIC 285

This SIC includes manufacturers of paints and allied paint products such as varnishes, shellacs, and paint removers. The paint industry grew by about 5.3 percent in 1986, a vast improvement over 1985's decline of 2.9 percent. Estimated shipments for 1986 were $11.1 billion, of which architectural coatings accounted for about 41 percent, followed by product coatings (35 percent) and specialty products (24 percent) [2, p. 15–2].

SIC 285 represents about 6 percent of the value of shipments of SIC 28. The value of shipments increased almost 21 percent from 1981 to 1985. The number of employees in SIC 285 was 6 percent of SIC 28 and the number of establishments was 9 percent.

SIC 286

This SIC includes manufacturers of a variety of industrial organic chemicals. Industry shipments of organic chemicals increased approximately 3 percent over 1985, which was the same level of growth experienced in the previous year. In 1985, the value of shipments for SIC 286 was $41.8 billion, representing 21 percent of the value of shipments of SIC 28. The value of shipments in SIC 286 decreased 11.3 percent over the previous year [2, p. 12–6]. The number of employees in SIC 286 was almost 11 percent of SIC 28 and the number of establishments was approximately 7 percent. Employment in SIC 286 increased by 10 percent, and production workers equaled 51 percent of all workers [Table II–1].

There are three subgroups in SIC 286. Examples of products for each four-digit SIC are given below.

SIC 2861 Products—Hardwood and softwood distillation products, wood and gum naval stores, charcoal, natural dyestuffs and natural tanning materials.

SIC 2865 Products—Toluene, benzene, synthetic organic dyes and pigments.

SIC 2869 Products—Alcohols, caprolactum, and ethylene glycol.

SIC 287

This SIC includes establishments that manufacture agricultural chemicals and pesticides. According to the U.S. Department of Commerce, the 1985 value of shipments of SIC 287 ($14.8 billion) represents 7.5 percent of the value of shipments of SIC 28. The value of shipments in SIC 287 decreased 9.6 percent from 1981 to 1985. Employment in SIC 287 represented 5 percent of SIC 28, but has declined by 16 percent since 1981. The number of establishments in SIC 287 was approximately 9 percent of all establishments in SIC 28 and production workers equaled approximately 62 percent [Table II–1].

SIC 2873 includes manufacturers of nitrogenous and mixed fertilizers. The value of shipments of nitrogenous fertilizers in 1986 was $3.43 billion, which represents a decrease of approximately 8.2 percent over 1985 shipments [2, p. 13–1].

SIC 2874 includes manufacturers of phosphatic fertilizers, such as phosphoric acid, made from phosphate rock. The value of shipments of phosphatic fertilizers in 1986 was $4.67 billion, which represents a decrease of approximately 13 percent over 1985 shipments [2, p. 13–3]. Ammonia and phosphoric acid are two substances with potential exposure problems that are produced and or used in SIC 2874.

SIC 2875 includes establishments that mix fertilizers from purchased fertilizer materials. SIC 2879 includes formulators and preparers of ready-to-use agricultural and household pest control chemicals, such as fungicides, insecticides, and herbicides.

SIC 289

This group includes manufacturers of miscellaneous chemical products. For 1985, SIC 289 represents 7 percent ($14.6 billion) of the value of shipments of SIC 28. From 1981 to 1985, the value of shipments in SIC 289 increased 15.5 percent. The number of employees in SIC 289 was almost 10 percent of SIC 28 and has remained unchanged since 1981. The number of establishments in SIC 289 was approximately 19 percent of all establishments in SIC 28. Production workers equaled approximately 62 percent of all workers [Table II–1].

SIC 2891 includes manufacturers of industrial and household adhesives and sealants. Industry shipments for adhesives and sealants in 1986 amounted to $4.2 billion, of which about 60 percent were by synthetic resins and rubber-based adhesives; 20 percent by sealant and caulking compounds; and the remaining 20 percent by natural-based adhesives and miscellaneous compounds [2, p. 15–3].

SIC 2892 includes manufacturers of explosives, such as TNT (Trinitrotoluene). Ethylene glycol dinitrate is one of the products of this SIC which may have potential exposure problems. SIC 2893 includes manufacturers of printing ink, whereas SIC 2895 includes manufacturers of carbon black. SIC 2899 includes manufacturers of miscellaneous chemical products, not elsewhere

classified. Among these three SICs, ethylene glycol, nitrotoluene, hexylene glycol, trimellitic anhydride, and coal dust are all substances with suspected exposure problems that are either produced or used in these sectors.

SIC 29—Petroleum and Related Industries

This industry is divided into petroleum refiners and producers of other related products. Petroleum refineries (SIC 2911) produce fuels (such as gasoline, kerosene, and distillate and residual fuel oils) as well as lubricants and chemical feedstocks. These products are produced through straight distillation of crude oil, redistillation of unfinished petroleum derivatives, cracking, or other processes. Other producers in this sector manufacture asphalt and tar products for paving and roofing (SIC 295) and other lubricating oils, greases, and petroleum and coal products (SIC 299) [1, pp. 127–128].

The 1985 value of shipments for SIC 29 ($179.1 billion) was 7.9 percent of the value of shipments for all manufacturing industries. Petroleum refining dominates SIC 29, accounting for 94 percent of this sector's value of shipments [2, pp. 10–8 to 10–14].

The number and size distribution of establishments in SIC 29 are shown in Table II–1, as is total employment. Relative to value of output, SIC 29 has few establishments and low employment, accounting for less than 1 percent of all manufacturing establishments and employment [6, pp. 10, 30].

About 40 percent of the establishments in SIC 29 are petroleum refineries [9], which are large and extremely capital intensive. Production is highly automated; enclosed processes are used throughout. Mean employment size is 105 employees. By contrast, plants in the other industries within SIC 29 are relatively small and less capital intensive, and processes are generally not automated. Mean establishment size in the rest of SIC 29 is 19 employees.

The general pattern of firms in SIC 29 in the 1980s has been one of decline. The real value of petroleum product shipments, consumption of petroleum products, petroleum refining capacity, and employment in SIC 29 all peaked between 1977 and 1981. There has been an upturn since 1985, resulting principally from a sharp decline in crude oil prices in the first half of 1986, which stimulated demand for refinery products [4; 2, pp. 10–1 and 10–2]. Demand for petroleum products is expected to grow only slightly in the short run and to show little long-run growth. In the past, trends have been strongly influenced by sharp fluctuations in the price of crude oil [2, pp. 10–3 and 10–4].

In 1985, imports of refined petroleum products were $18.2 billion, which reflects both the price of crude oil and the volume of imports. The volume of imports is itself strongly influenced by the price of crude oil, since low prices of crude oil tend to reduce imports of refined petroleum products by making domestic refineries more competitive in the production of gasoline. Import penetration has been erratic [2, pp. 10–1 and 10–3].

The profitability of firms in SIC 29 is low. The median 1985 rate of return on assets (4.4 percent) is the second lowest median return on assets of all 20 two-digit manufacturing industries [5].

SIC 30—Rubber and Miscellaneous Plastics Products Industry

This industry sector consists of establishments that manufacture a variety of products from plastic resins and from natural, synthetic, and reclaimed rubber. Although plastic products account for the largest share of the value of shipments of this industry group, the industry also manufactures a variety of rubber products, including tires, inner tubes, footwear, and belting [1, pp. 129–132]. The value of shipments for 1985 was $71.3 billion. This industry is dominated by the miscellaneous plastic products sector (SIC 307 until 1987 and now SIC 308), which accounts for 81 percent of the establishments, 66 percent of the value of shipments, and 70 percent of the employment for the entire industry group [9]. The tire and inner tube (SIC 301) sector and miscellaneous rubber products (SIC 306) sector are the other major components of this industry.

Similar processes are used in manufacturing plastic and rubber products, with the nature and form of the final product determining the process more than the product's components. A product's components, however, determine the types of chemical exposures employees experience. Examples of particularly serious types of exposures are those to the foaming agents that are used in the production of foam rubber or plastic foams and to the styrene used to produce polystyrene or for lamination processes.

As shown in Table II–1, the industry sector has relatively small establishments, 61 percent of which have fewer than 20 employees, with an average of 43 employees per establishment. Employment in this industry grew by 7 percent between 1981 and 1985, with growth in the tire and inner tube and miscellaneous plastic product sectors balancing declines in other sectors [4].

Firms in this industry have above-average profits for manufacturing industries, with a 7.7 percent median rate of return on assets compared with a 7.0 percent median for all manufacturing firms [5].

SIC 31—Leather and Leather Products

The leather and leather products industry (SIC 31) consists of several sectors such as leather tanning (SIC 311), non-rubber footwear (SIC 314), and luggage and leather goods (SIC 315–319), [1, pp. 133–135]. Shipments of leather products and employment in the leather industry have been declining steadily over the past several years, due mainly to the worsening import situation [2, p. 43–1].

According to the U.S. Department of Commerce, the 1985 value of shipments for leather and leather products ($8.6 billion) was down 8.2 percent from 1984. The total represents 0.4 percent of the value of shipments for all manufacturing industries. Non-rubber footwear (SIC 314) makes up most of the value of shipments in this industry with 51.9 percent of the total value [3]. The median return on assets in 1985 for the leather and leather product industry was 6.3 percent [5].

The number of establishments in the leather tanning and finishing industry (SIC 311) has decreased by over 244 establishments, from 384 establishments in 1982 to 140 establishments in 1986. Employment and shipments have also decreased significantly. Since the leather tanning industry is highly dependent on the demand from the non-rubber footwear industry, it is not likely that the situation will improve in the near future [2, pp. 43–1 and 43–2].

The footwear, luggage, and handbag industry (SIC 31, other than 311) has had reductions in sales over the past few years. The non-rubber footwear industry has suffered substantially since 1981 when an import restraint agreement with South Korea and Taiwan expired. Since then, import's share of the domestic market has increased to over 80 percent. In the last few years, production, employment, and domestic shipments have declined substantially [2, p. 43–5].

The luggage and leather goods industry has also seen declines in production, employment, and shipments over the past several years. Imports reached over 52 percent of the domestic market in 1986. Employment has also been declining over the past few years, from 42,000 total employees in 1984 to

37,400 total employees in 1986 [2, pp. 43–12 and 13].

SIC 32—Stone, Clay, Glass and Concrete Products

This industry is made up of products such as cement (SIC 324), concrete (SIC 327), pottery (SIC 326), stone (SIC 328), glass (SIC 321–323), and clay brick (SIC 325). Since these products are primarily used as construction materials, the industry is heavily dependent on the amount of new construction activity in a given year.

There were 21,054 establishments in the stone, clay and glass industry (SIC 32) in 1985. Most of these firms (73.7) percent) employed fewer than 20 people in 1985. The mean establishment size was 28 workers. Total employment was 588,000 in 1985, a decrease of 0.8 percent over the 1984 total employment figure of 593,000 [6].

In 1985, the value of shipments in SIC 32 increased 3.1 percent over 1984. The total value was 2.4 percent of the value of shipments for all manufacturing industries. The value of shipments is evenly distributed over the entire industry, except for the concrete sector (SIC 327) with 35.4 percent of shipments [3]. The median rate of return on assets for SIC 32 was 6.5 percent in 1985 [5].

The concrete industry (SIC 327) has seen considerable improvement in production, employment, and demand in the past several years. The demand for concrete has increased substantially since 1982, when shipments were 23 percent below their current figure. Imports are not a significant factor when dealing with concrete. Since concrete demand depends mainly on non-residential building construction, the demand for concrete should decrease slightly in the near future [2, pp. 2–4 to 2–9].

The glass industry (SICs 321–323) has experienced steady growth over the past two years, mainly in production and shipments. New product introductions have allowed the glass industry to make substantial gains in winning market share. The outlook for continued growth for the glass industry is good [2, pp. 2–9 to 2–12].

Shipments of clay bricks (SIC 325–326) have increased substantially over the past few years, from 6.2 billion bricks in 1983 to 7.3 billion bricks in 1986. The outlook for the industry is for slow growth in the near future [2, pp. 2–12, 13].

SIC 33—Primary Metal Industries

The primary metal industry (SIC 33) is divided into two different sectors: Nonferrous metals and foundries (SIC 333–336) and ferrous metals and foundries (SIC 331–332) [1, pp. 145–152]. This includes the basic iron and steel industry, and the metals industry. Both sectors have been hurt in the recent past by a decline in domestic consumption and the growing number of imports into the United States. The future for these industries, however, looks brighter due to an increase in orders, slowing imports, and a decrease in capacity [10]. These industries have had increases in prices, shipments, and profits in 1987 and 1988, helped by the fall in the dollar.

As seen in Table II–1, the number of establishments in SIC 33 in 1985 totaled 10,101. The majority of these had fewer than 20 employees in 1985. Total employment (808,000 employees in 1985) and production employment (612,000 in 1985) have declined over the last several years, while the average hourly wage of production workers has increased by 2.2 percent from 1984 to 1985 [6]. The mean establishment size was 80 workers.

Production in the steel mill products industry has declined over the past few years, from 88.3 million tons in 1985 to 84.0 million tons in 1986, a decline of 4.9 percent. The 1985 value of shipments ($110.3 billion) in SIC 33 was 4.8 percent of the value of shipments for all manufacturing industries [3]. The median rate of return on assets in 1985 was 5.5 percent for the primary metal industry [5].

The outlook for the steel industry is brighter than just a few years ago. In 1987, the industry is expected to have its first profitable year since 1981. This is due mainly to the large decrease in capacity and employment that the industry has implemented over the last several years. The industry has cut costs of production while prices have remained steady.

The import situation has also improved for the steel industry, due in part to the falling value of the dollar against major competitors such as Japan and Europe. Over the past few years, imports of steel mill products took a large share of the domestic market, an increase of 7.7 percent in the market share from 1979 to 1984. Exports declined during the same time period by 13.7 percent [2, pp. 19–1 to 19–9].

The ferrous castings industry (SIC 332) has shown a poor performance over the past few years. The value of shipments has been steadily decreasing, from $10.8 billion in 1985 to $10.1 billion in 1986, a decline of 7.3 percent. The value of shipments for SIC 332 is forecast to increase 7.1 percent in 1987, although this trend is not likely to continue in the future. Total employment and the number of production workers has also fallen since the early 1980's, by 9.7 percent and 10.0 percent, respectively, from 1979 to 1984 [2, pp. 19–1 to 19–9].

Nonferrous metals can be classified as four primary metals: aluminum, zinc, lead, and copper. The aluminum industry has had mixed progress when it comes to improving their industry. Shipments have increased steadily in the past few years, with a 3.3 percent increase in 1986. The aluminum industry does experience a cost disadvantage due in part to its high electrical costs. Most aluminum producers use very high levels of electricity in their production processes; costs of production fluctuate with electrical utility costs. The industry faces potentially higher electricity rates related to legislative bills designed to reduce acid rain depositions produced by coal burning utilities. The enactment of such legislation is projected to increase power rates to aluminum smelters by over 10 percent.

The zinc industry should have steady growth over the next few years, due mainly to a decline in capacity and an increase in consumption. This has caused the price of zinc to rise, although rather slowly. Domestic consumption was still expected to increase to 960,000 tons in 1987. The value of shipments declined by 18.8 percent in 1986, but it is expected to increase by 7.7 percent in 1987. Total employment and the number of production workers has remained steady for the past several years.

The lead industry has had slow growth over the past few years, due mainly to a decline in demand from products such as gasoline and automobile batteries. Consumption is expected to increase slightly by a modest 2.1 percent in 1987, but to continue increasing into the 1990's. Industry production should also increase slowly, about 2 percent a year for the next few years [2, pp. 20–6 to 20–18].

The copper industry has been undergoing restructuring to remain competitive in the world market. This has forced the industry to decrease capacity and reduce employment [2, pp. 20–6 to 20–18]. The price of copper has been driven up recently due to a decline in inventories. This should allow the industry to turn a significant profit for the first time in several years.

SIC 34—Fabricated Metal Products

The fabricated metal products industry (SIC 34) consists of several different groups: Metal cans and shipping containers (SIC 341); cutlery and hand tools (SIC 342); heating equipment (SIC 343); fabricated structural metal products (SIC 344); screw machine products, bolts, and washers (SIC 345); forgings and

stampings (SIC 346); plating and coating (SIC 347); small arms and ordinance (SIC 348); and miscellaneous wire and fabricated products (SIC 349). SIC 34 excludes machinery and transportation equipment [1, pp. 153–166].

The total number of establishments in the fabricated metal products industry in 1985 was 46,322. The majority of these firms (67.0 percent) employ fewer than 20 people, a change of 0.2 percent since 1984. Total employment in this industry has reached 1.465 million employees, an increase of 0.1 percent since 1984 [6].

The 1985 value of shipments for SIC 34 represents a 2.7 percent increase over 1984. This was 6.1 percent of the value of shipments for all manufacturing industries [3]. The median return on assets for the fabricated metal products industry in 1985 was 7.1 percent [5].

Metal cans (SIC 3411) shipments have been increasing steadily in the past few years, from 92.3 billion units in 1983 to 105.0 billion units in 1986, an increase of over 13.75 percent. This was due mainly to the increase in soft drink and beer cans being shipped. The value of shipments has also increased with a compound annual increase of 3.3 percent from 1979 to 1984. Total employment in the metal cans industry has remained steady with only a slight decrease expected in 1987. The number of production workers has increased slightly with an increase of 0.3 percent from 1985 to 1986. Exports of metal cans have decreased substantially since 1984 when they reached an all-time high of $56.5 million. Since that time they have decreased to $37.5 million in 1986 [2, pp. 6–1 to 6–4].

The fabricated structural metal industry (SIC 3441) produces structural metal components used primarily in the construction industry. Shipments of fabricated structural metal increased 8.0 percent from 1984 to 1985, from $7.5 billion to $8.1 billion in 1985. Total employment has been increasing slowly at an annual rate of 1.0 percent, while the number of production workers has been increasing at an annual rate of 1.4 percent [2, pp. 2–3, 4].

The value of shipments in the screw machine products, bolts, and washers industry (SIC 345) decreased slightly from 1984 to 1987, from $7.5 billion to $7.3 billion. Total employment also decreased from 96,200 in 1984 to 95,600 in 1986. Since the automotive industry is the major customer for this industry, stable automotive sales are the key to economic health for this industry sector [2, pp. 24–1 to 24–6].

SIC 35—Non-Electrical Machinery

The non-electrical machinery industry (SIC 35) is made up of several different sectors: Miscellaneous machinery (SIC 351–356); computer and office equipment (SIC 357); refrigeration and service industry machinery (SIC 358); and miscellaneous machinery and equipment (SIC 359 [1, pp. 167–183].

As seen in Table II–1, the number of establishments in 1985 totaled 77,748. The majority of these (77.1 percent) had fewer than 20 employees in 1985. Total employment and production employment have decreased over the last several years. The 1985 value of shipments ($215.1 billion) in SIC 35 was 9.4 percent of the value of shipments for all manufacturing industries [3]. In 1985, the median rate of return on assets for SIC 35 was 7.5 percent [5].

Miscellaneous machinery (SICs 351–356) has been experiencing an increase in shipment over the past few years. In 1984, the value of shipments was $113.5 billion and since then it has increased to $114.0 billion in 1985. The majority of this increase was from metalworking machinery, which increased from $18.6 billion in 1984 to $19.7 billion in 1985, an increase of 5.7 percent. Total employment in this sector has been declining from 1,153,500 workers in 1984 to 1,117,400 workers in 1985 [2, pp. 23–1 to 23–17, 25–1 to 25–5, and 21–1 to 21–17].

The computer industry (SIC 357) has been facing stagnant demand for its products in the U.S. market [11]. The value of shipments of electronic computing equipment (SIC 3573) has decreased from $53.5 billion in 1984 to $49.2 billion in 1986, a decline of 8.0 percent. Total employment and the number of production workers have also declined since 1984 by 15.5 percent and 27.8 percent, respectively. Imported computer equipment have made significant inroads into the domestic market, due mainly to the standardization of products and the fall in price of computer equipment [2, pp. 28–1 to 28–10].

The refrigeration and service machinery industry (SIC 358) has had exceptional performance since 1984, with a compound annual rate of growth of 5.1 percent from 1979 to 1984. This is due mainly to the increase in new residential construction. Total employment and the number of production workers have also been increasing substantially, although this trend slowed in 1986, probably due to the trade situation. Imports have been steadily increasing, while exports have been decreasing at a steady rate [2, pp. 22–9 to 22–11].

SIC 36—Electric and Electronic Equipment

This industry is made up of several distinct sectors: Transformers and switchgear (SIC 361); electrical industrial apparatus (SIC 362); household appliances (SIC 363); electrical lighting and wiring (SIC 364); consumer electronics and communications equipment (SIC 365–366); electronic components and accessories (SIC 367); and miscellaneous electrical equipment and machinery (SIC 369) [1, pp. 184–195].

There were approximately 28,478 establishments in the electric and electronic equipment industry in 1985, employing over 2 million workers. The majority of these firms (62.4 percent) had fewer than 20 employees. The value of shipments for all electric and electronic equipment establishments in 1986 was $192.7 billion. This was 8.4 percent of the value of shipments for all manufacturing industries. Most of the value of shipments in SIC 36 is from the communication equipment sector (33.9 percent) [3]. The median return on assets for the electric and electronic equipment industry was 7.9 percent in 1985 [5].

The transformer and switchgear industry (SIC 361) has had mixed performance in the last year. While the value of shipments increased for switchgear by 2.5 percent, the value of shipments for transformers decreased by 1.9 percent from 1985 to 1986. Total employment and the number of production workers has remained fairly steady since the early 1980's [2, pp. 26–1 to 26–4].

The electrical industrial apparatus industry (SIC 362) is facing an uncertain future. The value of shipments for this industry has remained fairly steady over the past few years, but shipments in the future are predicted to decline due to weak demand from the automotive industry. Total employment has already begun to decline from 190,300 in 1984 to 178,900 in 1985, a decreases of 6.0 percent. The majority of the industry has been forced to cut operating costs to compensate for the reduced demand [2, pp. 26–4 to 26–6].

The household appliance industry (SIC 363) has had a steady increase in sales since the early 1980's, from $12.6 billion in 1982 to $15.2 billion in 1986, an increase of 19.98 percent [12]. The industry is optimistic about its future, due mainly to the increased residential construction and the increase in disposable income. Imports have not been a substantial burden on this industry, although exports have not

increased substantially either. Total employment and the number of production workers have declined from 1984 to 1986 by 9.1 percent and 9.9 percent, respectively. This decline in employment is due to the recent number of acquisitions within the industry and the need to cut costs of production [2, pp. 44–5 to 44–9].

The value of shipments for the electrical lighting and wiring industry (SIC 364) has been increasing steadily over the last decade, from 11,321 in 1980 to 15,606 in 1985, an increase of 39.6 percent. Total employment and the number of production workers have decreased, although at a slow rate. Performance in this industry is due, in part, to the good performance of the construction industry. Since the electrical lighting and wiring industry depends on both residential and non-residential construction, they are able to withstand a slowdown in one sector as long as the other sector is still profitable [2, pp. 3–2 to 3–5].

The consumer electronics and communication equipment industry (SICs 365–366) has had mixed performance over the past few years. While the communication equipment industry has performed well in the past, the consumer electronics industry has not performed as well, due to the large import volume in this industry. Overall, the value of industry shipments has remained fairly stable, with shipments increasing in the communication equipment industry and shipments decreasing in the consumer electronics industry. Total employment and the number of production workers also follow this pattern, decreasing for consumer electronics and increasing for communication equipment [2, pp. 29–1 to 29–5 and 44–9 to 44–15].

The electronic components and accessories industry (SIC 367) is expected to show record growth over the next few years. Industry shipments were up 8.1 percent, from $39.7 billion in 1986 to $43.0 billion in 1987. This was due, in part, to the strong performance of the defense electronics industry. The number of production workers and total employment has remained fairly steady in 1986 and 1987. Imports are still increasing, but may be slowed due to the fall in the value of the dollar [2, pp. 32–1 to 32–4].

SIC 37—Transportation Equipment

This industry sector includes establishments engaged in manufacturing equipment for land, sea, air, space transportation and includes manufacturers of parts and accessories as well as complete vehicles.

The major subdivisions within this sector are motor vehicles and motor vehicle equipment (SIC 371), aircraft and parts (SIC 372), ship and boat building and repair (SIC 373), railroad equipment (SIC 374), motorcycles, bicycles and parts (SIC 375), guided missiles, space vehicles and parts (SIC 376), and miscellaneous transportation equipment (SIC 379). Establishments in the miscellaneous subdivision manufacture a broad range of products (e.g., from tanks to wheelbarrows) [1, pp. 196–201]. Because the manufacture of transportation equipment involves a wide range of industrial processes, establishments in this sector often include or involve foundries, electroplating operations, various types of hot metal work, welding, laminating, plastic molding, and painting and coating. Thus workers may be exposed to the chemicals generated by a broad variety of processes.

Although the transportation equipment industry includes both very small and very large establishments, it has an unusual number of very large establishments that have thousands of employees. These very large establishments are most likely to be found in plants that produce final equipment on a mass-production basis (e.g., automobile plants, aircraft plants, or tank assembly lines). There is still a place in this industry, however, for smaller establishments, and, as shown in Table II–1, 68 percent of all establishments have fewer than 20 employees.

Because foreign competition plays an important role in this sector, the prosperity of the industry fluctuates with business cycles and with the value of the dollar. Employment in this industry declined between 1981 and 1982 but had recovered to the 1981 level by 1984 and had increased another 4 percent by 1985 [4].

The value of shipments for SIC 37 was $301.4 billion in 1985. The average firm in this industry had a 7.3 percent return on assets in 1985, which is slightly above the median of 7.0 for firms in all manufacturing industries. Some sectors within the two-digit industries had significantly lower returns on assets, although no major component of the industry had a return on assets below 4 percent in 1985 [5].

SIC 38—Measuring, Analyzing and Controlling Instruments

SIC 38 includes manufacturers of instruments used to measure, test, analyze and control. It also includes optical instruments and lenses; surveying and drafting instruments; hydrological, hydrographic, meteorological, and geophysical equipment; search, detection, navigation, and guidance systems and equipment; surgical, medical, and dental instruments, equipment, and supplies; ophthalmic goods; photographic equipment and supplies; and watches and clocks [1, p. 243].

The industries in this SIC rely heavily on research and development activities (R&D) of other industries for sales of their products. According to the U.S. Department of Commerce, increases in research and development expenditures by industry and government in 1986 caused increases in sales of scientific and industrial instruments. High tech firms, which represent a large portion of SIC 38's product market, are the largest investors in research and development, where R&D expenditures are measured as a percentage of gross sales. Firms producing semiconductors, computers and related equipment, office equipment, and software, among others, were major sources of R&D funds in 1986. The pharmaceutical and chemical industries also have relied on R&D to a large extent. In addition, the decline in the price of oil, which raises profits by lowering production costs, is expected to further stimulate R&D expenditures by the chemical industry [2, p. 33–1].

Similarly, government outlays for R&D increased in 1986 by more than 9 percent in current dollars. Most of the R&D expenditures, however, were for defense-related research. In addition, the National Aeronautics and Space Administration (NASA) is expected to invest in new instrumentation for the redesign of the space shuttle and other rocket systems [2, p. 33–4].

According to the U.S. Department of Commerce, the value of shipments in 1985 ($61 billion) increased almost 26 percent since 1981. Between 1981 and 1985, SIC 38 experienced a 1 percent loss in employment. Of all employees, 54.4 percent working in SIC 38 were production workers [4]. In 1985, the median rate of return on assets in this SIC was 7.3 percent [5].

From 1981 to 1985, the value of shipments for SICs 383 and 384 experienced growth, rising 60 and 54.3 percent, respectively. SIC 383 comprises 8 percent of the total value of shipments in SIC 38, while SIC 384 represents 23 percent. In contrast, SIC 387 experienced a drop of 36 percent in the value of shipments, representing only 1.5 percent of the total value of shipments in SIC 38 [5].

SIC 39—Miscellaneous Manufacturing Industries

In any classification scheme, some items inevitably fall outside the scheme. In manufacturing, these miscellaneous industries are included in SIC 39, which contains five three-digit industries that are as dissimilar from one another as is usually the case at the two-digit level in other manufacturing sectors. Most of the industries in SIC 39 produce discretionary durable consumer goods, some of which are luxury goods. Establishments that cannot be grouped together even at the three-digit level are included in SIC 399. At the three-digit level, miscellaneous manufacturing industries include producers of jewelry, silverware, and plated ware (SIC 391); musical instruments (SIC 393); toys and sporting goods (SIC 394); pens, pencils, office and art supplies (SIC 394); and costume jewelry and notions (SIC 396). A sixth category, miscellaneous manufactures (SIC 399), includes producers of brooms and brushes, signs and advertising displays, burial caskets, hard surface floor coverings, and manufacturing industries "not elsewhere classified" [1, pp. 211–218].

The number of establishments and employment in SIC 39 are shown in Table II–1. Nearly three-quarters (72 percent) of these employees are production workers.

Establishments in SIC 39 are generally far smaller than those in manufacturing as a whole, with higher proportions of employees concentrated in small establishments. The mean size of establishments is 11 employees, with 85 percent of establishments having fewer than 20 employees, compared with less than 65 percent for manufacturing establishments as a whole. Relatively few establishments in SIC 39 have 100 or more employees [6].

Miscellaneous manufactures (SIC 399) has the largest share (more than one-third) of the value of shipments for SIC 39 ($26.5 billion in 1985) [3, vol. 1: 8, 22, 24]. The SIC 39 industries were generally hard hit by the 1982 recession. Substantial import competition, aided by the strength of the dollar, has impeded the recovery of many of these industries since 1982, and the import share of new supply doubled between 1980 and 1985 in many industries. Imports account for nearly 60 percent of the new supply of sporting and athletic goods and between one-quarter and three-eighths of new supply in many other industries. The recent decline of the dollar has tended to halt or reverse import penetration to varying degrees [2, pp. 45–2 to 45–11; 46–10 to 46–13]; however, domestic production in SIC 39 will also be affected by the tendency of the doll and toy sector to move offshore [2, pp. 45–2 to 45–11; 46–120 to 46–12].

Generally, this cyclical pattern also is found in this industry at the more disaggregated level. In the latest cycle, the peak-to-trough decline of production, employment, and real value of shipments in many of these industries was between 10 and 25 percent. In some cases (particularly in musical instruments and toys and sporting goods), production and/or employment declined by as much as half. Exceptions to this pattern, however, can be found in the manufacture of dolls, sporting and athletic goods, and costume jewelry, where the real value of shipments grew steadily despite the business cycle. (The apparent contradiction between from this growth and decline in production and employment results from the movement offshore of production facilities of domestic companies.) In miscellaneous manufacturing (SIC 399), 1986 employment was 15 percent above the previous peak, making this the only three-digit industry to regain the peak level [5, 2, pp. 45–3 to 45–11; 46–11 to 46–13].

In terms of profitability, the majority of industries in SIC 39 are more profitable than most manufacturing industries. The median 1985 rate of return on assets (8.0 percent) is the second highest median return on assets of all two-digit manufacturing industries. Median rates of return for four-digit industries within this sector range from 3.4 percent to 9.5 percent [5].

SIC 40—Railroad Transportation

SIC 40 includes establishments that provide line-haul railroad transportation, and switching and terminal establishments. General authority for the working conditions at railroad operations is vested in the Federal Railroad Administration. For the most part, OSHA's standards apply only to off-track operations such as shops and servicing areas. The U.S. Department of Commerce estimates that in 1986, there were 23 individual Class I railroads (those with operating revenues of $50 million or more in 1987 dollars), which accounted for over 95 percent of the freight tonnage handled by the railroad industry. The industry also includes about 480 smaller carriers, including shortlines and switching and terminal companies. The 1986 operating revenue for the railroad industry was estimated as $26.5 billion (1982 dollars), representing an annual loss of about 4 percent. Revenue ton miles were estimated as 880 billion, which represents less than a 1 percent rate of growth [2, p. 55–8]. Between 1980 and 1985, the industries in SIC 40 experienced a serious economic decline, as indicated by the fact that it was the second slowest growing SIC (behind SIC 10, metal mining), and third highest in terms of employment losses (behind SIC 33, primary metals and SIC 35, heavy machinery). During this period, employment declined by approximately 27 percent [2, pp. 13–14]. The median rate of return on assets in 1985 was 4.4 percent [5].

SIC 45—Air Transportation

This SIC includes establishments that provide domestic and foreign transportation by air and also those that operate airports and flying fields and provide terminal services. The Federal Aviation Administration (FAA), U.S. Department of Transportation, enforces rules and regulations governing the safety and health of flight and cabin crew of aircraft in flight. In general, the FAA also has jurisdiction over airline maintenance and ground support personnel. According to the U.S. Department of Commerce, the U.S. airline industry consists of approximately 250 individual commercial air carriers operating over 4,500 aircraft and employing over 355,000 people. In 1985, the industry served 380 million passengers and operated 6 billion cargo ton-miles. Twelve major carriers account for 84 percent of all revenue passenger miles. (The U.S. Department of Commerce defines a major carrier as having at least $1 billion in annual revenue, in 1982 dollars.) The remaining passenger revenue is shared by 16 carriers classified as nationals (each with annual revenues between $75 million and $1 billion in 1982 dollars), which account for about 12 percent, and by the regionals/commuters, which account for 4 percent. The U.S. Department of Commerce estimated the 1986 operating revenue for the airlines industry as $49 billion (1982 dollars), representing an annual growth rate of about 4.9 percent. Revenue passenger miles were estimated as 363 billion, which represents an 8 percent rate of growth [2, p. 55–1]. In 1985, the median rate of return on assets in this sector was 4.3 percent [5].

SIC 47—Transportation Services

SIC 47 includes establishments that furnish services related to transportation. Activities classified in SIC 47 include freight forwarding, arranging transportation for passengers and freight, renting railroad cars, inspection and weighing services; and freight car loading [1, pp. 280–281].

According to the U.S. Department of Commerce, between 1980 and 1985, SIC 47 was the third-fastest growing industry group behind SIC 62 (Securities) and SIC 73 (Business Services) [2, pp. 13–14]. Between 1981 and 1985, SIC 47 experienced a 31 percent increase in employment. Table II–1 presents employment and establishment data for SIC 47. The median return on assets in this SIC was 7.1 percent [5].

SIC 49—Electric, Gas, and Sanitary Services

SIC 49 includes establishments that generate, transmit, and/or distribute electricity, gas, or steam. These establishments may be combinations of any of these services, but also may include other types of services, such as transportation, communications, refrigeration and pipelines for natural gas. Water and irrigation systems, and sanitary systems that collect and dispose of garbage, sewage, and other wastes, also are included in this SIC [1, p. 284].

The utilities covered in SIC 49 have been undergoing many changes in the past few years. The utilities have been in a state of transition due to ongoing changes in regulations regarding utility rates and competition. Some industrial customers have begun producing their own energy and utilities are now competing for customers outside their service areas. This competition has forced structural changes in the industry, especially diversification. Utilities have been forced to upgrade their overall efficiency. With declining interest rates, regulators have been decreasing the allowed rate of return for utilities. This, too, has led to intensified pressures on competition [13, p. 56]. The Federal Energy Regulatory Commission is currently considering whether to allow utilities to open their power lines to other competing utilities. Users would be given the choice of suppliers. With the decreasing rate of return and the increasing competition, utilities have stepped up efficiency in order to offset the impending drop in their profit margins [14, p. 48].

Table II–1 presents employment and establishment data for SIC 49 for 1985. Between 1981 and 1985, SIC 49 experienced a 6 percent growth in employment. In 1985, almost 80 percent of all employees were production workers [4]. The median return on assets was 4.0 percent [5].

Within this SIC, there are seven three-digit SICs, including establishments that generate, transmit, or distribute electrical energy for sale and that operate crude petroleum and natural gas field properties; establishments that transmit and or store natural gas for sale; establishments that provide electric or gas services in combination with other services, only if one service does not constitute 95 percent or more of revenues; establishments that distribute water for sale for domestic, commercial, and industrial use; establishments that collect and dispose of wastes conducted through a sewer system, including such treatment processes as may be provided; establishments that produce and/or distribute steam and heated or cooled air for sale; and establishments that operate water supply systems for the purpose of irrigation [1, pp. 284–286].

SIC 50 and SIC 51—Wholesale Trade

The wholesale trade sector includes establishments engaged in the wholesale selling of merchandise to retailers; industrial, commercial, institutional, farm, or business users; or to other wholesalers or firms that act as agents or brokers in the wholesale buying or selling of merchandise. Wholesale trade is divided into trade in durable goods (SIC 50) and in nondurable goods (SIC 51). This analysis focuses only on a few of the wholesale trade industries (e.g., dealers in scrap and waste materials, SIC 5093; grain, SIC 5153; chemicals and allied products, SIC 5161; farm supplies, SIC 5191; and paints, varnishes, and supplies, SIC 5198 [1, pp. 241, 250, 255–257]. In addition to the types of worker exposures associated with materials handling and receiving, some industries engage in other activities that can lead to significant chemical exposures. For example, assembling, breaking up, and sorting scrap and waste materials (SIC 5093) and storing grain (SIC 5153) are activities that can lead to exposures.

Wholesale trade sales ($1,375 billion in 1985) were fairly equally divided between durable goods and nondurable goods—46 percent and 54 percent, respectively [2, p. 56–1]. Of the approximately 425,000 establishments in wholesale trade, about five-eighths were in durable goods, and three-eighths were in nondurable goods. The specific four-digit industries studied for this analysis include about 11 percent of all wholesale trade establishments [3, pp. 59, 62, 64–65].

Table II–1 shows employment data at the four-digit level. Somewhat less than 60 percent of total employment in wholesale trade is in durable goods, while a little more than 40 percent is in nondurable goods. The specific four-digit industries being analyzed here account for less than 9 percent of all employment in wholesale trade [6].

SIC 55—Automotive Dealers and Service Stations

This industry sector includes retailers of transportation equipment for personal use (new and used automobiles) as well as recreational vehicles (boats, motor homes, and dune buggies); sellers of automobile parts and accessories; and gasoline stations. Although it does not include establishments whose primary business is automotive repair, it does include repair operations that are part of automobile dealerships or service stations. Only those retail outlets that earn more than 50 percent of their revenues from gasoline or lubricating oil sales are included. Many car washes and convenience stores that sell gasoline are excluded, as are traditional full-service gas stations that earn more than 50 percent of their revenues from such activities as repairs, towing, or the sale of auto accessories [1, pp. 265–266]. According to one estimate, this sector includes only 55 percent of all retail motor fuel outlets [15, pp. 6–13]. Although many employees are involved in selling, some are exposed to chemicals during painting or stripping or as a result of the indoor operation of engines or the use of solvents.

As shown in Table II–1, most establishments are relatively small (80 percent have fewer than 20 employees). Only in one sector (i.e., new and used automobile dealerships) do more than half of the establishments have more than 19 employees [9]. Even in this sector, however, 90 percent of the establishments have fewer than 100 employees [4]. Although the typical operation is relatively small, total employment is substantial because of the large number of establishments. New and used automobile dealerships account for 48 percent of total employment, gasoline service stations for 31 percent, and automobile and home supply stores for 16 percent.

Although many firms own only a single establishment, large firms own a significant portion of all establishments, which are operated as chains under leasing or franchising agreements.

The profitability of firms in SIC 55 is below the national average, with a medium return on assets of 5.9 percent in 1985; however, this rate of return improved in 1986 as gasoline prices declined, and new car sales increased [5].

SIC 72—Personal Services and SIC 73—Business Services

The personal services industry consists primarily of consumer services. SIC 721, laundry, cleaning and garment

services has the highest potential for overexposure to chemicals. Other segments of SIC 72 include photographic studios (SIC 722); beauty shops, barber shops and shoe repair (SIC 723–725); and funeral service and crematories (SIC 726) [1, pp. 298–300].

As seen in Table II–1, the number of establishments in 1985 totaled 161,004. Almost all of these (96.9 percent) had fewer than 20 employees in 1985. The mean establishment size was 7 employees. The largest single segment of this industry is SIC 7231, beauty shops, which totaled 53,165 firms in 1986 [6]. Total employment (1,056,000 employees in 1985) has increased over the last several years. In 1986, the value of sales was $39.4 billion in the personal services industry, a 6.6 percent increase over 1985 [16]. The median rate of return on assets for the personal services industry was 10.5 percent in 1985 [5].

The business services industry consists of several different sectors. Among the sectors included are mailing, reproduction, and commercial art and photography (SIC 733); building cleaning and maintenance services (SIC 734); and miscellaneous business services (SIC 739), such as photofinishing laboratories and commercial testing laboratories [1, pp. 301–308].

The number of establishments in 1985 totaled 382,626. Almost all of these (90.5 percent) had fewer than 20 employees in 1985. The mean establishment size was 12 workers. Total employment (4,457,000 employees in 1985) has increased over the last several years (4,057,000 employees in 1984) [4]. In 1986, the value of sales was $198.7 billion in the business services industry, a 9.2 percent increase over 1985 [16]. The median rate of return of assets SIC 73 was 11.1 percent in 1985 [5].

SIC 75—Automotive Repair, Services, and Garages

This sector includes establishments that provide automotive repair, rental, leasing, and parking services to the general public, but excludes gasoline stations (SIC 55) and repair shops that are part of automobile dealerships or that service commercial fleets [1, p. 309]. Employees may be exposed to engine emissions in parking garages or repair shops, to a variety of chemical solvents (particularly in painting and stripping), and to dust from body work.

Eighty-five percent of the establishments are automotive repair shops, which is the sector most likely to have significant chemical exposure, and they employ 61 percent of all industry workers [9]. As shown in Table II–1, SIC 75 is dominated by businesses employing fewer than 20 workers (97 percent) with a median return on assets of 9.2 percent in 1985. The profitability of automotive repair and service firms is high, although it varies by size and industry sector. Small firms (under $100,000 in assets) had returns of 18.3 percent in 1985, while large businesses (over $1,000,000) had returns of 3.9 percent. Paint shops (SIC 7535) were the most profitable type of operation, while parking lots (SIC 7523) and parking structures (SIC 7525) registered significantly lower rates of return [5].

SIC 76—Miscellaneous Repair

This industry group includes a wide variety of repair services, differentiated by object repaired and processes used. Industries of particular concern include reupholstery and furniture repair (SIC 7641) and welding (SIC 7692) [1, pp. 312–314]. Reupholstery and furniture repair workers may be exposed to nuisance dusts during wood working, and to solvents; welders may be exposed to fumes.

Nineteen percent of the 56,000 industry establishments in SIC 76 are in SIC 7641 and SIC 7692. These two industries account for approximately 14 percent of all SIC 76 employment [6, pp. 81–82].

The industry is made up almost entirely of very small firms, and the sector has extremely low concentration. Mean business size is 5.5 employees: more than 95 percent of all establishments have fewer than 20 employees, and 65 percent of all workers are employed by establishments of this size. Only 0.2 percent of all miscellaneous repair establishments (with about 6 percent of total employment) have 100 or more employees, and only 17 establishments have 250 or more. The four-digit industries of concern are even more completely dominated by small establishments, with a mean size of 4.8 employees in SIC 7641 and 3.4 employees in SIC 7692 [6, pp. 81–82].

Despite a slight decline in 1981 and 1982, employment in SIC 76 has grown fairly steadily since the 1975 recession, increasing by 23 percent between 1979 and 1984 and by 7 percent between 1984 and 1986. More than 98 percent of all workers are neither administrative nor clerical [4].

Miscellaneous repair firms have high profit rates. The median 1985 rate of return on assets in SIC 76 is 10.0 percent. This rate of return is higher than that of any two-digit manufacturing industry. The median rates of return on assets in SIC 7641 and SIC 7692 are over 11 percent [5].

SIC 80—Health Services

The health services industry encompasses a broad range of medical, surgical, and other health services, both public and commercially owned. These services are provided by a variety of practitioners (e.g., physicians, dentists, osteopathic physicians, chiropractors, optometrists) at a variety of facilities (e.g., hospitals, nursing facilities, outpatient care facilities, medical laboratories) [1, pp. 321–323].

Total expenditures on health care and medical services ($425 billion in 1985) are very large, with 40 percent of this amount going to hospital care and 20 percent to physicians' services. Expenditures on nursing home care, drugs and medical sundries, and dentists' services each accounted for 6 to 8 percent of all health and medical services expenditures [2, p. 54–1].

Data on health care establishments are shown in Table II–1. Although the number of health service establishments (313,000) is very large, 85 percent of these are offices of licensed practitioners. No other three-digit sector within the health services industry accounts for more than 4 percent of health service establishments, and only about 2.7 percent (i.e., 1,500 establishments) are hospitals.

Total health services employment is very large (6.3 million), with hospitals accounting for almost half (i.e., 48 percent) of this workforce. Because of their large numbers, practitioners' offices are next in percentage of workforce employed (24 percent), followed by nursing and personal care homes (18 percent). Mean establishment sizes range from six or fewer employees in practitioners' offices to 250 or more employees in hospitals. The overall mean size of establishments in this industry is 20 employees, with more than 91 percent of these establishments having fewer than 20 employees, and approximately 22 percent of all SIC 80 employees working in establishment of this size. SIC 80 facilities with more than 250 employees employ more than 50 percent of the workforce in this sector, and facilities with more than 100 employees employ more than 60 percent [6; 4].

The health and medical services industry has been expanding rapidly for more than a decade. A variety of factors have caused this increase, including the expansion of the elderly population, the increasing use of sophisticated high-technology equipment, the expanded treatment of expensive diseases, and the increasing costs of malpractice insurance. In addition, between 1985

and 1986, the price for most medical services rose between 6 and 9 percent, compared with 1.5 percent increase in consumer prices. The implementation of Medicare's prospective payment system is also causing major changes in the health care industry [2, pp. 54–1,2].

Hospital care costs have been a major target of cost-cutting measures, resulting in a decline in hospital admissions, a shortening of hospital stays, and substantial industry restructuring, including increased mergers and acquisitions by large chains, vertical integration, diversification of services offered, expanded professional peer review, and more businesslike operations. Major investor-owned nursing home chains also have experienced rapid expansion and acquisition [2, pp. 54–1, 2].

For SIC 80 as a whole, the growth rate in expenditures averaged 12.6 percent per year from 1979 to 1984 and more than 9 percent for the next 3 years [2, p. 54–1]. Employment grew by 31 percent between 1979 and 1986, rising by 2 to 5 percent in each year [4]. The growth picture is fairly consistent across three-digit industries, although expenditures on "other professional services" have shown the most rapid growth of any health service (16.3 percent annually from 1979 to 1984). Expansion has been especially rapid in health maintenance organizations and home health care, both of which have the potential for reducing health costs and substituting, to some degree, for hospital care [2, pp. 54–1 to 54–4].

The median rate of return on assets in health services (5.0 percent in 1985) is relatively low compared with that in manufacturing industries, and hospitals have somewhat lower median rates of return than is the case for health services as a whole. Several "offices" industries, on the other hand, have median rates of return higher than 13 percent. Medical and dental laboratories have median rates of return that are above the median for two-digit manufacturing industries [5].

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TABLE II - 1

Industries With Potential Hazardous Exposures,
Number of Establishments and Employment
(1985)

SIC	Description	Establishments[a] Total Number	Percent Large[b]	Percent Small[c]	Employment[d] Total (1,000)	Production Workers (1,000)
20	FOOD AND KINDRED PRODUCTS	29,043	37.14	62.86	1,603	1,118
21	TOBACCO MANUFACTURES	216	46.76	53.24	64	48
22	TEXTILE MILL PRODUCTS	11,023	39.40	60.60	702	607
23	APPAREL PRODUCTS	30,032	33.33	66.67	1,121	945
24	LUMBER & WOOD PRODUCTS, EXCEPT FURNITURE	36,710	19.73	80.27	697	584
243	MILLWORK, VENEER & PLYWOOD	13,921	17.87	82.13	288	190
245	BUILDING & MOBILE HOMES	1,618	40.05	59.95	72	56
249	MISCELLANEOUS WOOD PRODUCTS	5,666	18.73	81.27	77	64
25	FURNITURE AND FIXTURES	16,791	27.16	72.84	494	394
26	PAPER AND ALLIED PRODUCTS	8,750	53.86	46.14	678	512
27	PRINTING, PUBLISHING & ALLIED INDUSTRIES	84,279	15.87	84.13	1,428	789
28	CHEMICAL AND ALLIED PRODUCTS	20,823	32.59	67.41	1,044	578
281	INDUSTRIAL INORGANIC CHEMICALS	3,024	35.42	64.58	142	72
282	PLASTICS & SYNTHETICS	1,666	51.50	48.50	172	114
283	DRUGS	2,454	37.82	62.18	206	95
284	SOAP, CLEANERS, & COSMETICS	4,498	24.59	75.41	148	94
285	PAINTS, VARNISHES, LACQUERS	1,880	36.54	63.46	64	31
286	INDUSTRIAL ORGANIC CHEMICALS	1,528	34.88	65.12	160	82
287	AGRICULTURAL CHEMICALS	1,843	23.77	76.23	59	37
289	MISCELLANEOUS CHEMICAL PRODUCTS	3,930	29.64	70.36	94	54
29	PETROLEUM REFINING & RELATED INDUSTRIES	3,334	28.40	71.60	179	109
291	PETROLEUM REFINING	1,332	33.18	66.82	141	82
295	PAVING & ROOFING MATERIALS	1,222	23.81	76.19	26	20
299	MISCELLANEOUS PETROLEUM & COAL PRODUCTS	780	27.44	72.56	-	-
30	RUBBER & PLASTICS PRODUCTS	18,002	38.85	61.15	786	607
307	MISCELLANEOUS PLASTIC PRODUCTS	14,638	39.62	60.38	550	435
31	LEATHER AND LEATHER PRODUCTS	3,940	29.85	70.15	165	137
311	LEATHER TANNING & FINISHING	480	35.42	64.58	15	12
32	STONE, CLAY, GLASS, & CONCRETE PRODUCTS	21,054	26.26	73.74	588	451

[a] Dun and Bradstreet

[b] 20 or more employees

[c] Fewer than 20 employees

[d] Labstat, U.S. Department of Labor (Database)

TABLE II - 1

Industries With Potential Hazardous Exposures,
Number of Establishments and Employment
(1985)
(continued)

SIC	Description	Establishments[a] Total Number	Percent Large[b]	Percent Small[c]	Employment[d] Total (1,000)	Production Workers (1,000)
33	PRIMARY METAL INDUSTRIES	10,101	44.75	55.25	808	612
34	FABRICATED METAL PRODUCTS	46,322	32.96	67.04	1,465	1,084
35	MACHINERY, EXCEPT ELECTRICAL	77,748	22.90	77.10	2,174	1,307
36	ELECTRICAL & ELECTRONIC MACHINERY, EQUIPMENT & SUPPLIES	28,478	37.64	62.36	2,197	1,300
37	TRANSPORTATION EQUIPMENT	16,132	31.58	68.42	1,980	1,257
38	INSTRUMENTS	16,814	29.42	70.58	720	391
39	MISCELLANEOUS MANUFACTURING INDUSTRIES	32,212	15.82	84.18	367	264
40	RAILROAD TRANSPORTATION	2,645	27.30	72.70	359	-
45	TRANSPORTATION BY AIR	11,832	19.46	80.54	522	-
47	TRANSPORTATION SERVICES	35,626	7.56	92.44	276	-
49	ELECTRICAL GAS, & SANITARY SERVICES	21,115	25.71	74.29	915	729
5093	SCRAP & WASTE MATERIALS	7,556	12.61	87.39	92	-
5153	GRAIN	7,523	5.84	94.16	-	-
5161	CHEMICALS & ALLIED PRODUCTS	13,045	8.51	91.49	-	-
5191	FARM SUPPLIES	20,392	4.55	95.45	151	-
5198	PAINTS, VARNISHES, & SUPPLIES	4,033	6.89	93.11	-	-
55	AUTO DEALERS & SERVICE STATIONS	189,214	9.77	90.23	1,890	1,886
72	PERSONAL SERVICES	161,004	3.13	96.87	1,056	-
73	BUSINESS SERVICES	382,626	9.46	90.54	4,457	3,863
75	AUTO REPAIR, SERVICES, & GARAGES	149,260	2.64	97.36	731	614
7641	REUPHOLSTERY & FURNITURE REPAIR	10,655	0.92	99.08	-	-
7692	WELDING REPAIR	9,413	2.21	97.79	-	-
80	HEALTH SERVICES	313,076	8.71	91.29	6,299	5,607

Source: U. S. Department of Labor Occupational Safety and Health Administration, Office of Regulatory Analysis.

[a] Dun and Bradstreet

[b] Labstat, U.S. Department of Labor (Database)

[c] 20 or more employees

[d] Fewer than 20 employees

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III. Employee Exposures and Benefits

Employee exposures to the substances included in the scope of this rulemaking are associated with a wide variety of acute and chronic illnesses. These include sensory irritation, narcosis, organs system dysfunction, chronic respiratory disease, neurological impairment, allergic sensitization, and cancer. Since OSHA's adoption of existing Federal and consensus standard limits in 1971, toxicologic evidence has become available that shows that adverse health effects can occur as a consequence of exposure to many of the substances listed in OSHA's Z tables, and that such health effects occur even when exposures are maintained at the current Z table limits. In addition, many substances that have come into widespread use or been introduced since 1971 have been shown to be potentially hazardous in the workplace environment. OSHA thus believes that reducing worker exposures to such substances by lowering existing exposure limits or by adding limits for previously unregulated substances will result in a significantly reduced risk of illness to workers.

This chapter describes the methodology used to identify workers potentially exposed to the hazardous substances included in this rulemaking and the expected benefits to those workers resulting from lowering permissible exposure levels. An important existing data base for identifying employees potentially exposed to hazardous substances was OSHA's Integrated Management Information System. The IMIS data were used to project expected benefits resulting from lowering permissible exposure levels to the substances being regulated.

The IMIS data base does not include information on all substances and has more information on some substances than others. IMIS contained research information on about 160 substances among the approximately 430 substances covered by the proposal. While the IMIS data base contains the results for over 100,000 samples of substances currently regulated by OSHA, no plant specific information was available for about 200 of the substances included in this rulemaking but currently not regulated by OSHA. To obtain additional information on employee exposures and on substances not covered by IMIS, a nationwide survey was begun in January 1988, which was designed to collect worker exposure data at over 5,300 establishments nationwide that are believed to be affected by this proposal. The survey results include industry-sector-specific data on the extent of employee exposures to hazardous materials and, in addition, provide specific information on the industrial processes in which these substances are used.

To assess the benefits of the proposed revision to OSHA's Z-Tables, OSHA relied both on the survey and IMIS data and used two basic approaches. The first approach relies on IMIS data combined with raw survey data to estimate the extent to which employees are currently exposed to substances included in this rulemaking. From this analysis, OSHA estimates the reduction in illness cases and disease-related fatalities associated with reducing exposure limits for these substances. The second approach relies solely on the use of the imputed survey data base to generate an exposure profile (the imputed survey file was created using statistical procedures to fill in missing responses to particular questions asked in the survey).

Description of Data Sources Used

To assess the quantitative benefits associated with this rulemaking, the following data were used:

- The proposed exposure limits for substances included in the rulemaking;
- Employee exposure data for these substances;
- Employment data for four-digit SIC code for the base year 1985;
- Annual illness and lost workday rates for the base year 1985; and
- Health effects information on the substances included in the rulemaking.

Employee exposure data for about 160 substances were obtained from OSHA's Integrated Management Information System (IMIS). This data base contains exposure measurements obtained by OSHA compliance officers during the conduct of thousands of health inspections. For each facility inspected, the IMIS file includes information on the number of employees at the facility, results of employee air monitoring for specific substances, and the number of employees potentially exposed to each substance monitored. To perform the benefits asssessment, a summary IMIS file was created that contained the following information:

- A list of substances for which personal 8-hour TWA samples were taken, by four-digit SIC and facility inspected
- The number of workers potentially exposed to each substance monitored, by four-digit SIC and facility
- The number of employees at each facility inspected
- The total number of personal 8-hour TWA samples obtained for each substance, by four-digit SIC and facility
- The number of samples taken at each facility that showed concentrations exceeding OSHA's proposed limits.

Only those substances for which OSHA is proposing to reduce an existing 8-hour TWA limit or to add a new 8-hour TWA limit were included in the analysis. A total of approximately 37,500 personal air sample results for about 160 substances were appropriate for use in this analysis. This analysis does not estimate the benefits associated with reducing current ceiling limits or adding new short-term exposure limits (STELs) because the data obtained from the IMIS did not include information on sample duration for ceiling or peak measurements, or OSHA was not able to relate the IMIS data on ceiling or

peak measurements to the proposed short-term or ceiling limits.

In addition to the IMIS exposure data, OSHA has completed a telephone interview survey of over 5,300 workplaces that are potentially affected by the revision of OSHA's Z Tables. Data from this survey provide information on substances that are used in a variety of industrial processes at the facilities surveyed, the number of workers involved in these processes, and whether personal exposure measurements taken at the processes exceeded OSHA, ACGIH, or NIOSH limits.

Employment data by four-digit SIC code were obtained from three data sources. For each four-digit SIC represented in the IMIS file, OSHA first relied on 1985 data from the BLS LABSTAT data base [1]. Where data were unavailable for this source at the four-digit SIC level, OSHA relied on Dun & Bradstreet's *Market Identifiers* file for 1985 [2]. Data from 1985 *County Business Patterns* [3] were used to obtain employment data for four-digit SIC groups not represented in either the LABSTAT or Dun & Bradstreet file.

Data on illness and lost workday rates were obtained from the 1985 LABSTAT file for all industries (at the three- and four-digit level) represented in the IMIS file. These data included rates per 100 employees for total illness cases, lost-workday illness cases, and total number of lost workdays.

Estimates of the Number of Potentially Exposed Employees

Estimates of the number of employees potentially exposed to the substances included in this analysis were derived from the IMIS data, OSHA's survey data, and employment data bases. To conduct the analysis, OSHA used the IMIS and survey data separately to derive independent estimates of the number of workers potentially exposed and the number of workers exposed above the proposed limits for each substance. The estimates derived from these two data sources were then combined to yield an overall assessment of the extent of employee exposure, by four-digit SIC, to substances included in this rulemaking. The following sections describe how each of the data bases was used to develop estimates of employee exposures, and how these estimates were then combined.

Estimates Derived From OSHA's IMIS Data Base

For each facility inspected, the IMIS contained information on the number of employees at the facility and the number of employees observed to be potentially exposed to each substance for which personal air samples were collected. For each substance sampled within an industry (at the four-digit level), the estimated number of employees potentially exposed to that substance in the industry was determined by the following formula:

$$\sum \frac{P_f}{E_f} * W = P$$

where
P_f = number of employees observed to be potentially exposed to the substance at a facility;
E_f = total number of employees at the facility;
W = number of production workers in the industry in 1985; and
P = estimated number of employees potentially exposed to the substance in the industry.

The estimated number of workers currently exposed above the proposed limits for each substance was calculated using the following formula:

$$\sum \frac{S_f}{T_f} * P = Z$$

where
S_f = number of samples that exceeded the proposed limit for the substance at all facilities in an industry sector;
T_f = total number of personal samples taken for the substance at all facilities in the industry sector.
P = estimated number of employees potentially exposed to the substance in the industry; and
Z = estimated number of workers in an industry sector currently exposed above the proposed limits for the substance.

Estimates Derived From OSHA's Survey Data

Facilities participating in OSHA's telephone survey provided the following information that was useful for estimating the extent of employee exposure to chemical substances:

- The facility's four-digit SIC code;
- The total number of production employees at the facility;
- The number of employees involved in each process used at the facility;
- The substances used or present in each process;
- The exposure limits used as internal targets or goals at the facility (i.e., OSHA's current limits, ACGIH limits, NIOSH limits, or "Other" limits such as those from material safety data sheets or insurance carriers); and
- Whether employee exposures exceeded the targeted limits for each process/chemical combination present at the facility.

To estimate the number of employees potentially exposed to a given substance in a 4-digit SIC industry group, OSHA assumed that all employees who are involved with processes in which the substance was used or present are potentially exposed. Thus, the formula for estimating the number of employees who are potentially exposed to a substance in a given industry sector is

$$\sum \frac{X_f}{T_f} * W = P$$

where
X_f = number of employees at the facility who are involved in processes using a given substance;
Tf = total production workforce at the facility;
W = the number of production workers in the industry sector in 1985; and
P = estimated number of employees potentially exposed to the substance in the industry sector.

To estimate the number of employees currently exposed above the proposed limits, OSHA relied on survey responses that indicated whether exposure measurements associated with a process exceeded the facility's internal exposure limits. If a facility responded that exposure measurements taken at a process area did not exceed ACGIH, NIOSH, or some "other" set of limits, OSHA assumed that no potentially exposed employee is currently exposed above the proposed limit for any substance associated with the process. On the other hand, if a facility responded that exposure measurements taken at a process did not exceed current OSHA, ACGIH, or "Other" set of limits, OSHA assumed that all potentially exposed employees are currently exposed above the proposed limits for all substances associated with the process. In addition, if the process had certain characteristics that suggested that the proposed limits were not being achieved (i.e., lack of ventilation system or open process used indoors) all employees involved in the process were assumed to be overexposed. The decision logic for determining when employees at a particular process may be overexposed was the same as that used for estimating costs associated with achieving the

proposed limits (see Chapter VI, Costs of Compliance).

Approach for Combining Estimates Derived From the IMIS Data and Survey Data

To obtain an overall estimate of the extent of employee exposures to substances used in each four-digit SIC industry group, OSHA combined the estimates derived separately from the IMIS and survey data. Table III–1 illustrates how these estimates were combined to yield an overall estimate of the extent of employee exposures in SIC 2851. Where estimates for a given substance could be derived from one data set but not the other, the combined assessment uses the available estimates without adjustment. Where estimates could be derived from both data sets for the same substance, the combined assessment is based on the average of the available estimates; this approach has the effect of giving equal weight to estimates derived from either the IMIS or survey data.

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TABLE III-1

ANALYSIS OF EMPLOYEE EXPOSURES IN SIC 2851 DERIVED FROM IMIS DATA, SURVEY DATA, AND BOTH IMIS AND SURVEY DATA COMBINED

NAME	ASSESSMENT FROM IMIS		ASSESSMENT FROM SURVEY		COMBINED ASSESSMENT	
	WORKERS POTENTIALLY EXPOSED	WORKERS ABOVE LIMITS	WORKERS POTENTIALLY EXPOSED	WORKERS ABOVE LIMITS	WORKERS POTENTIALLY EXPOSED	WORKERS ABOVE LIMITS
2-HEXANONE	6,547	727	26,751	0	16,649	364
ACETONE	2,875	96	5,912	*	4,393	147
ALPHA-ALUMINA	1,286	0			1,286	0
BUTOXYETHANOL	2,121	0			2,121	0
BUTYL ACRYLATE	13,302	0			13,302	0
CARBON MONOXIDE	672	672			672	672
CARBON TETRACHLORIDE	5,912	0			5,912	0
COBALT AS CO	4,604	0			4,604	0
CYCLOHEXANONE	6,821	0			6,821	0
DIISOBUTYL KETONE	4,434	0			4,434	0
ETHYLENE GLYCOL			53,429	19,876	53,429	19,876
FURFURAL	2,956	0			2,956	0
HEPTANE	5,454	0			5,454	0
HEXAFLUOROACETONE	41,383	41,383			41,383	41,383
HEXANE	4,678	0			4,678	0
HEXONE	7,131	319			7,131	319
IRON OXIDE DUST AND FUME, AS F	5,173	0			5,173	0
ISOBUTYL ALCOHOL	10,996	0			10,996	0
ISOPHORONE			17,440	0	17,440	0
MAGNESIUM OXIDE FUME, AS MG	10,560	0			10,560	0
METHYL N-AMYL KETONE	9,038	0			9,038	0
MOLYBDENUM, INSOLUBLE COMPOUND	11,853	0			11,853	0
N-BUTYL ALCOHOL			51,803	12,329	51,803	12,329
N-BUTYL GLYCIDYL ETHER	28,030	0			28,030	0
NAPHTHA			296	*	296	*
NUISANCE PARTICULATES	5,040	1,217			5,040	1,217
PERCHLOROETHYLENE	1,973	0			1,973	0
PETROLEUM DISTILLATES, RUBBER	7,885	0			7,885	0
PHTHALIC ANHYDRIDE	4,427	0			4,427	0
STODDARD SOLVENT	6,961	194	28,821	13,113	17,891	6,654
STYRENE	1,508	0	7,464	5,598	4,486	2,799
TIN METAL AND OXIDE	1,286	0			1,286	0
TITANIUM DIOXIDE	2,668	0			2,668	0
TOLUENE	7,538	187	11,750	*	9,644	239

TABLE III-1

(continued)

ANALYSIS OF EMPLOYEE EXPOSURES IN SIC 2851 DERIVED FROM IMIS DATA, SURVEY DATA, AND BOTH IMIS AND SURVEY DATA COMBINED

NAME	ASSESSMENT FROM IMIS		ASSESSMENT FROM SURVEY		COMBINED ASSESSMENT	
	WORKERS POTENTIALLY EXPOSED	WORKERS ABOVE LIMITS	WORKERS POTENTIALLY EXPOSED	WORKERS ABOVE LIMITS	WORKERS POTENTIALLY EXPOSED	WORKERS ABOVE LIMITS
TRIBUTYL PHOSPHATE	244	0			244	0
TRICHLOROETHYLENE	3,695	0			3,695	0
TRIETHYLAMINE	244	0			244	0
TRIMELLITIC ANHYDRIDE	1,626	813			1,626	813
TRIMETHYL BENZENE	8,099	0			8,099	0
VINYL ACETATE	13,302	0			13,302	0
VM & P NAPHTHA	25,909	0			25,909	0
ZINC OXIDE, FUME			3,178	*	3,178	*

*Insufficient data from survey responses to permit an assesment of exposure levels.

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Estimates of the number of employees potentially exposed and the number exposed above the proposed limits are available in Supplement ι2, ι3, ι4, ι5, and ι6 to this regulatory analysis. In addition to exposure estimate these Supplements also contain process and industry use information by substance. (Copies of these Supplements are available, upon request, from the office of Regulatory Analysis, Room N3627, OSHA.)

Aggregate estimates of the number of employees potentially exposed or exposed above the proposed limits to any substance considered in the analysis are presented by two-digit SIC code in Table III–2. Because an employee may be exposed to more than one substance in a given industry, aggregate estimates of the size of the exposed population are presented as minimum and maximum estimates. Maximum estimates of the size of the exposed population assume that no employee is exposed to more than one substance; minimum estimates assume the greatest possible extent of multiple chemical exposure. For example, if 200 employees are estimated to be exposed to acetone and 300 employees are estimated to be exposed to toluene in a given industry, a minimum of 300 employees is estimated to be exposed to either substance in the industry, and a maximum of 500 employees is estimated to be exposed to either substance in the industry.

Employees exposed above the proposed limits are considered to be "at risk" of adverse health effects. It should be noted that this presentation shows risk reduction in employee equivalent terms; while all (100 percent) of the workers currently exposed above the new proposed limits would benefit from reduced risk, the new lower limits would not eliminate all chemical exposure risk. An estimated five, ten, or twenty percent residual risk equivalent would remain at the new lower limits. Although not quantified, all employees currently exposed to hazardous substances at or below the recommended new levels would experience this residual risk. To obtain an approximation of risk reduction at the lower exposure levels being proposed, OSHA estimated that 95, 80, or 90 percent of the workers currently exposed above the proposed limits (i.e., the midpoint between the minimum and maximum estimates) will benefit from reduced risk after their exposures are lowered to or below the proposed limits. The results of this analysis are also presented by two-digit SIC codes in Table III–2.

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TABLE III-2. NUMBER OF WORKERS EXPOSED AND NUMBER OF WORKERS FOR WHOM RISK IS REDUCED, BY 2-DIGIT SIC

SIC Code	Number of Production Workers	No. Workers Potentially Exposed, Minimum Estimate	No. Workers Potentially Exposed, Maximum Estimate	No. Workers Exposed Above Limits, Minimum Estimate	No. Workers Exposed Above Limits, Maximum Estimate	No. Workers With Reduced Risk, (80% Risk Reduction)	No. Workers With Reduced Risk, (90% Risk Reduction)	No. workers With Reduced Risk (95% Risk Reduction)
20	1,149,971	218,638	275,837	109,289	161,000	108,115	121,630	128,387
21	48,625	1,298	1,298	62	62	50	56	59
22	244,367	147,842	170,842	13,730	16,909	12,256	13,788	14,554
23	994,363	271,934	353,225	13,494	15,894	11,755	13,225	13,959
24	543,695	201,869	318,642	81,184	99,437	72,248	81,279	85,794
25	496,760	189,194	429,107	77,910	96,802	69,885	78,620	82,988
26	667,082	367,943	606,256	66,302	108,383	69,874	78,608	82,975
27	1,255,639	1,069,582	1,198,072	137,557	180,397	127,182	143,079	151,028
28	866,859	788,571	866,570	300,562	426,653	290,886	327,247	345,428
29	178,202	177,058	178,202	133,282	134,096	106,951	120,320	127,005
30	817,652	607,900	810,873	156,191	591,627	299,127	336,518	355,214
31	152,241	41,525	76,274	4,347	5,907	4,102	4,614	4,871
32	587,946	307,033	493,751	56,274	93,588	59,945	67,438	71,184
33	754,180	451,813	719,699	114,179	311,416	170,238	191,518	202,158
34	1,228,572	606,058	1,104,519	138,744	262,805	160,619	180,697	190,735
35	1,508,032	661,605	1,186,259	139,409	197,843	134,901	151,763	160,195
36	1,319,939	693,114	996,305	139,421	169,876	123,718	139,183	146,916
37	1,270,731	708,119	1,015,308	90,357	182,918	109,310	122,974	129,806
38	606,978	374,905	482,051	34,649	35,131	27,912	31,401	33,146
39	417,988	232,354	294,590	53,671	67,160	48,333	54,374	57,395
40	193,605	174,308	180,573	15,421	19,430	13,941	15,683	16,555
45	346,071	127,515	159,158	45,756	45,756	36,605	41,180	43,468
47	85,386	31,917	32,596	1,731	2,382	1,645	1,850	1,953
49	804,200	759,231	775,063	477,380	481,333	383,485	431,420	455,388
50	2,759,479	545,417	1,065,910	193,893	211,291	162,074	182,333	192,462
51	1,147,688	592,777	748,500	226,780	288,088	205,947	231,691	244,562
55	1,470,816	1,017,352	1,470,816	249,123	325,852	229,990	258,739	273,114
72	765,244	551,441	604,813	41,497	46,935	35,373	39,794	42,005
73	4,179,928	2,113,228	2,266,266	347,718	569,457	366,870	412,729	435,659
75	675,834	461,366	593,967	54,146	75,771	51,966	58,462	61,710
76	331,435	252,837	268,243	76,296	139,331	86,251	97,033	102,423
80	6,237,985	2,399,295	2,749,195	46,599	54,400	40,400	45,450	47,975
TOTAL	34,107,493	17,168,039	22,469,780	3,668,060	5,386,824	3,621,954	4,074,696	4,301,071

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Estimates of the Reduction in Illness Cases and Lost Workdays

The BLS LABSTAT data base contains illness and lost workdays rates by SIC code. These rates are expressed as the annual number of illness cases or number of lost workdays per 100 full-time-equivalent employees. Reducing employee exposures to hazardous substances to a level below that associated with adverse health effects will result in a decrease in the number of illness cases and lost workdays.

To assess the impact on illness and lost workday rates of reducing employee exposures, OSHA first examined the relationship between the percentage of workers estimated to be exposed above the proposed exposure limits for the 160 substances included in the study and current illness and lost workday rates. This analysis was conducted at the three-digit SIC code level because of the lack of illness rate data for some of the four-digit SIC code groups. The results of this analysis are presented graphically in Figure III–1. Among three-digit industries for which OSHA has found that no employees are currently exposed above the proposed limits, total illness case rates reported by the BLS for the same industry group are usually less than 0.2 cases per 100 employees per year, and frequently are reported to be zero. In contrast, where OSHA has found that industry groups have more than 2 percent of their workforce exposed above the proposed limits, total illness case rates above 0.2 cases per 100 employees are frequently reported. In no instance does an industry group having 10 percent or more of its workforce exposed above the proposed limits report a total illness case rate of zero. Among three-digit SIC code industry groups for which OSHA has not found employee exposures above the proposed limit, 37 percent of the groups reported an illness rate of zero, 45 percent reported an illness rate of 0.1 to 0.2 cases per 100 employees, and only 18 percent of the industry groups reported an illness rate greater than 0.2 cases per 100 employees (but none above 0.5 cases per 100 employees). Given this distribution of illness rates across these particular industry groups, it is concluded that industry groups in which employee exposures have been controlled to or below the proposed limits will have an illness rate approximating 0.1 cases per 100 employees. It is believed that total illness cases at the three-digit level will be reduced to no more than 0.1 cases per 100 employees after employee exposures are reduced to or below the proposed limits.

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Figure III-1. Relationship Between Employee Exposures to Hazardous Substances and Industry Illness Rates

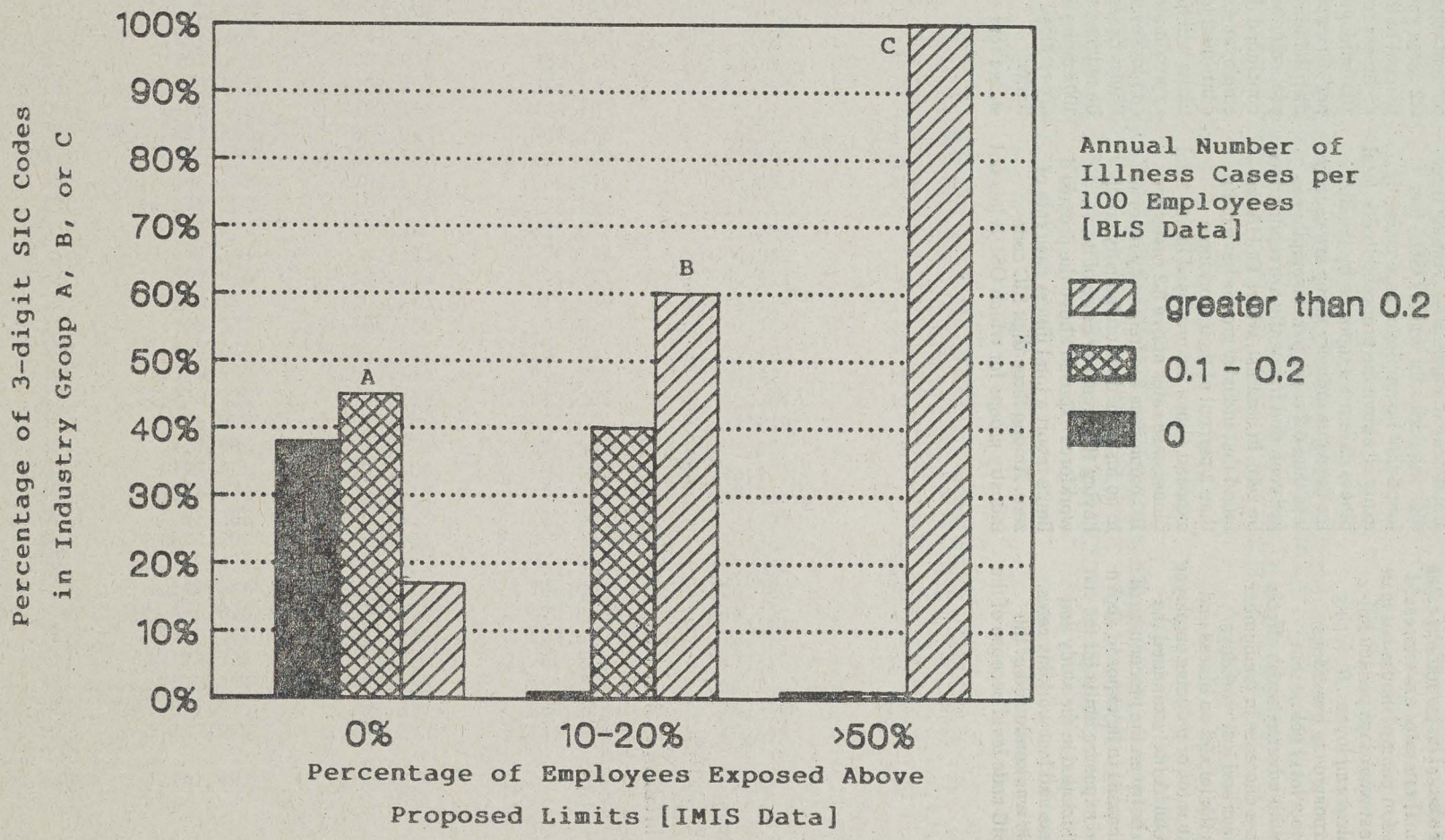

Figure III-1

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OSHA performed a similar analysis that also indicates that the rate of lost-workday illness cases will decline to a base rate of 0.05 cases per 100 employee and the annual rate of lost workdays will decline to 1 per 100 employees after employees exposures are reduced to or below the proposed limits.

OSHA estimated the number of illness cases and lost workdays potentially avoided annually by applying current illness rates to the estimated number of production workers per three-digit SIC group; this yielded an estimate of the annual number of illness cases and lost workdays reported by each three-digit SIC code industry group. It was assumed that, after promulgation of the proposed limits, these industries would experience illness rates of 0.1 cases per 100 employees, 0.05 lost-workday illness cases per 100 employees, and 1 lost workday per 100 employees per year. Using this approach, OSHA estimated that promulgation of the proposed limits will potentially avoid 55,216 illness cases per year, 23,608 lost workday illness cases per year, and 533,197 lost workdays caused by illnesses per year.

The movement to a 0.1 illness rate is presented as a best estimate supported by OSHA's interpretation of the relationship between chemical exposure levels and current industry illness rates. It may be argued, however, that if a 0.1 illness rate were achieved, the reduction in illnesses could not be credited exclusively to OSHA's rulemaking initiative, since some portion of the current BLS illness rate is made up of illnesses associated with exposures to hazardous agents or physical stress (e.g., radiation, noise, ergonomic stress).

While no claim is made that this rulemaking action will reduce illnesses related to these causes, OSHA believes that the benefit estimates related to this proposal of over 55,000 illnesses, over 23,600 lost workday illnesses, and over 533,000 lost workdays avoided each year are reasonable. This is based on the belief that company records, upon which the BLS data are based, rarely show chronic illnesses caused by exposures to toxic substances [4, 6]. The potential level of underreported illnesses in the BLS series is illustrated in a recent report by Landrigan and Markowitz. Using California physicians' reports of occupational illnesses, these authors estimated an occupational illness rate among New York State employees that was more than twice the BLS illness rate [8].

Estimates of the Number of Employees Potentially at Risk by Type of Hazard

In addition to estimating the number of employees exposed to the substances included in this analysis, OSHA also estimated the number of employees who are at risk of experiencing particular types of adverse health effects. To conduct this analysis, each substance included in the rulemaking was assigned to a health hazard category; these assignments were based on the primary health effects that provided the impetus for reducing an existing limit or proposing a new limit for a particular substance. (The assignment of substances to health effect categories is described in detail in Section IV–C of the preamble.) It should be noted that, in some instances, substances included in this rulemaking were grouped together in the preamble according to some basis other than a particular health effect; for example, several substances were grouped together because the ACGIH-recommended limits were derived based on the structural analogy of the grouped substances with that of other substances. For the benefits analysis described here, these substances were re-classified according to the primary health effect associated with exposure to the analogous chemical.

The number of employees estimated to be exposed to substances causing a particular health effect in an industry group was calculated by summing the number of employees exposed to all substances causing the same effect. Aggregate estimates across all affected industry sectors are presented in Table III–3. This table provides estimates of employees potentially exposed to substances in each health group, as well as estimates of employees exposed above the proposed limits for substances in each health group. Employees are frequently at risk from a variety of adverse health effects as a result of concurrent exposure to more than one toxic substance. Thus, the total number of employees considered to be at risk from any type of illness (as estimated in Table III–3) cannot be summed because the sum would result in doublecounting.

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TABLE III-3. ESTIMATED NUMBER OF WORKERS POTENTIALLY AT RISK OF EXPERIENCING ADVERSE EFFECTS, BY TYPE OF ADVERSE EFFECT

ADVERSE HEALTH EFFECT	NO. OF WORKERS POTENTIALLY EXPOSED TO SUBSTANCES ASSOCIATED WITH EFFECT, MINIMUM ESTIMATE	NO. OF WORKERS POTENTIALLY EXPOSED TO SUBSTANCES ASSOCIATED WITH EFFECT, MAXIMUM ESTIMATE	NO. OF WORKERS EXPOSED ABOVE PROPOSED LIMITS FOR SUBSTANCES, MINIMUM ESTIMATE	NO. OF WORKERS EXPOSED ABOVE PROPOSED LIMITS FOR SUBSTANCES, MAXIMUM ESTIMATE
NUISANCE EFFECTS	4,729,417	5,804,846	829,487	892,725
ODOR AND TASTE EFFECTS	718,522	808,537	59,102	59,102
SYSTEMIC TOXICITY	3,233,319	3,818,742	250,282	256,909
MUCOUS MEMBRANE IRRITATION	12,077,315	15,813,663	59,969	1,021,197
METABOLIC INTERFERENCES	2,277,113	2,345,975	746,879	746,879
LIVER/KIDNEY DISEASE	2,321,573	2,488,604	383,581	384,876
OCULAR DISTURBANCES	194	194	0	0
RESPIRATORY DISEASE	3,765,717	4,023,525	639,717	654,319
CARDIOVASCULAR DISEASE	164,576	164,576	44,355	44,355
NEUROPATHY	1,231,744	1,349,421	201,760	210,203
NARCOSIS	4,601,993	6,381,899	526,021	547,731
CANCER	3,663,053	3,784,374	499,716	499,716
ALLERGIC SENSITIZATION	2,710,576	2,903,153	296,444	297,255

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Estimates of the Number of Illness-Related Fatalities Avoided

As discussed in the preceding section, OSHA has estimated the number of employees currently at risk of experiencing a variety of adverse health effects brought about by over-exposures to the substances included in this rulemaking. Many of these adverse effects result in lethal outcomes, in particular cancer, cardiovascular effects, chronic respiratory disease, and chronic liver and kidney damage. OSHA also believes that employees who are excessively exposed to substances causing systemic organ damage, neurological impairment, or metabolic effects (i.e., cardiovascular disease through excessive formation of methemoglobin or carboxyhemoglobin, and neurological impairment through cholinesterase inhibition) are at excess risk of incurring a fatal condition.

To estimate the number of fatalities associated with excessive exposure to the 160 substances included in this analysis, OSHA relied on standard U.S. mortality rates and on published estimates of the proportion of fatalities that are believed to be associated with occupational illnesses. These data allowed OSHA to calculate cause-specific mortality rates that are attributable to occupational illnesses (i.e., mortality rates that represent the excess risk of mortality from occupational disease). OSHA then applied these occupationally-related mortality rates to its estimates of the number of employees exposed to the 160 substances of concern at levels above the proposed limits. OSHA's methodology and estimates are presented in Table III–4, and are described in detail below.

TABLE III-4

Estimated Annual Number of Fatalities Caused By Occupational Illness Among Workers Currently Exposed Above Proposed Limits, Using Alternative Assumptions

Cause of Death	U.S. Annual Death Rate Per 100,000 Residents (1985), by Cause[a]	Total Number of Deaths Per Year in U.S., by Cause[b]	Number of Deaths Attributed to Occupational Illnesses, by Cause	Annual Death Rate Per 100,000 Attributed to Occupational Illnesses	Number of Workers Exposed Above Proposed Limits	Annual Number of Fatalities Among This Group of Workers
Cancer	193.3	461,484	46,148[c]	55.3	499,716[g]	276
			23,074[d]	27.7	499,716	138
Chronic Pulmonary Disease	31.3	74,726	2,242[e]	2.7	647,018[h]	17
			747[f]	0.9	647,018	4
Chronic Liver Disease	11.2	26,739	802[e]	1.0	384,229[i]	4
			267[f]	0.3	384,229	1
Cardiovascular, Neurological, and Renal	418.5	999,127	29,974[e]	35.9	1,250,811[j]	449
			9,991[f]	12.0	1,250,811	150
TOTAL, All Causes						293–746

[a] Source: National Center for Health Statistics [5, Table II].
[b] Based on a total residential population in 1985 of 238,740,000. [7, p. 18].
[c] Assumes 10 percent of all cancer deaths are of occupational origin (Landrigan and Markowitz, 1987).
[d] Assumes 5 percent of all cancer deaths are of occupational origin (Landrigan and Markowitz, 1987).
[e] Assumes 3 percent of all deaths are of occupational origin (Landrigan and Markowitz, 1987).
[f] Assumes 1 percent of all deaths are of occupational origin (Landrigan and Markowitz, 1987).
[g] From Table III–3, midpoint estimate of number of workers exposed above proposed limits for potential carcinogens.
[h] From Table III–3, midpoint estimate of number of workers exposed above proposed limits for respiratory toxins.
[i] From Table III–3, midpoint estimate of number of workers exposed above proposed limits for liver toxins.
[j] From Table III–3, midpoint estimate of number of workers exposed above proposed limits for systemic toxins, metabolic toxins, cardiovascular toxins, and neuropathic agents.

Estimate of the Number of Cancer Fatalities

The U.S. National Center for Health Statistics has published cause-specific U.S. mortality rates for 1985 (the most recent data available) [5]. This source reported that the annual U.S. cancer death rate in 1985 was 193.3 per 100,000 residents. Based on a total resident U.S. population of 238,740,000 in 1985 [7, p. 18], the number of cancer deaths that occurred in 1985 was 461,484. Landrigan and Markowitz [8] reviewed several published estimates of the percentage of cancer deaths that are attributable to occupationally-related disease. These estimates range from less than 5 percent to 33 percent of all cancer deaths. Landrigan and Markowitz believe that, as a best estimate, 10 percent of all cancer deaths have an occupational origin.

Using an occupational cancer death estimate of 10 percent and applying it to the estimated number of cancer deaths in 1985, OSHA estimates that 46,148 occupationally-related cancer deaths occurred in the United States in that year. Table III–4 also shows an alternative assessment of occupationally-induced mortality that is based on an assumption that only 5 percent of all cancer deaths are of occupational origin.

As the next step, OSHA estimated the overall cancer death rate among the population which is occupationally exposed to chemicals and the remainder of the population. In 1985, there were an average of 108,856,000 persons employed [9, p. 8]. However, 25,469,200 of these were employed in industries or occupations where there is a low risk of exposure to toxic substances, such as finance, insurance, real estate, and private households [9, pp. 30, 84–88]. The remaining 83,386,800 persons are considered to be occupationally exposed to chemicals in varying degrees. Many would have only intermittent exposure at very low levels. Using the two alternative assumptions of the percentage of all cancer deaths which are of occupational origin (10 percent and 5 percent), OSHA calculated the annual cancer death rate attributed to occupational exposure by dividing the number of cancer deaths attributable to occupational illness by the population exposed, and multiplying by 100,000. If the 10 percent assumption is used, OSHA estimates that the annual cancer mortality rate attributable to occupational exposure to toxic substances is 55.3 per 100,000; with the 5 percent assumption, the rate would be 27.7. OSHA then estimated that there are 499,716 workers currently exposed above the proposed limits to the potential carcinogens included in this rulemaking. Applying the work-related cancer death rates to this population, OSHA estimates that between 138 and 276 cancer fatalities occur each year among these workers and that these fatalities will be prevented by the proposed rule.

In arriving at this estimate, two important offsetting arguments were considered. Because some of these workers may also be exposed to occupational carcinogens that are not covered in this rulemaking (such as asbestos or benzene), the number of occupational cancer deaths attributed to the substances included in this rulemaking may be overestimated. Offsetting this potential overestimate is the fact that the excess mortality rates of 27.7–55.3 per 100,000 workers were developed on the basis of occupational exposures among all exposed workers. However, the excess mortality rate experienced among workers with high average exposures to hazardous chemicals typically runs at least two or three times higher than the national average rate. In consideration of this, OSHA believes that any overestimate of cancer fatalities avoided attributed to regulated chemicals not covered under this rulemaking is offset by the use of a mortality rate which understates the true excess mortality rate among workers with very high exposures to toxic chemicals. (Additional comments on excess mortality rate estimates are included in the final section of this chapter.)

An alternative analysis of the reduction in cancer mortality was conducted using OSHA's quantitative risk assessments for the potential human carcinogens included in this rulemaking (the results of OSHA's risk assessments are presented in the preamble to the proposal). This analysis is presented in table III–5. Using the combined IMIS and survey data, OSHA found that employees are currently exposed above the proposed limits to four of these potential carcinogens (acrylamide, carbon tetrachloride, chloroform, and styrene). Applying OSHA's risk estimates to the estimated number of workers currently overexposed to thse substances, OSHA estimates that compliance with the proposed limits will avoid 5,005 cancer fatalities over the working lifetime of the population (i.e., 45 years). The average annual reduction in the number of cancer fatalities avoided over 45 years is estimated to be 111.

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TABLE III-5

ESTIMATES OF CANCER DEATHS POTENTIALLY AVOIDED, BASED ON QUANTITATIVE RISK ASSESSMENTS[a]

Substance	Number of Workers Above Proposed Limit	Estimated Number of Cancer Deaths		Estimated Number of Potentially Avoidable Cancer Deaths over a Working lifetime
		Current PEL	Proposed PEL	
Acrylamide	7,784	75	8	67
Carbon Tetrachloride	86,682	1,554	798	756
Chloroform	152,170	1,852	31	1,821
Styrene[b]	248,603	4,723	2,362	2,361
TOTAL		8,204	3,199	5,005

[a]Risk assessments are presented in Section IV-C of the preamble.

[b]This figure reflects an average estimate resulting from the combined IMIS and survey data. The 1988 sample survey alone identified just over 30,000 employees overexposed to this chemical. The Styrene Information and Research Center estimates that 224,250 workers make products with styrene.

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Although OSHA has evaluated the cancer risk for 17 potential carcinogens, there were IMIS and survey data for only four of these. Lack of IMIS or survey data means that the substance has not been sampled by an OSHA compliance officer or that none of the survey participants indicated that the substance was used at their facilities. This does not mean that no workers are currently exposed to these substances. Lacking a basis for estimating the extent of employee exposure, OSHA could not estimate the extent of reduction in cancer deaths attributable to the proposed reduction in exposure limits for these substances. To the extent that employee exposure to these carcinogens is reduced, further reductions in the number of cancer deaths will occur.

Estimated Reduction in Occupational Deaths From Causes Other Than Cancer

As shown in Table III–4, OSHA also estimated the number of occupationally-related fatalities that are expected to occur annually among employees exposed to substances associated with adverse health effects other than cancer. To perform this analysis, OSHA relied on an estimate made by Landrigan and Markowitz [7] that between 1 and 3 percent of all non-malignant disease is of occupational origin. Using the 1 and 3 percent figures as alternative assumptions and using the same methodology as that described above for cancer deaths, Table III–4 shows the following:

—Between 4 and 17 deaths caused by respiratory disease are estimated to occur each year among workers exposed to respiratory toxins covered in this rulemaking;
—Between 1 and 4 deaths are estimated to occur each year among workers exposed to liver toxins covered in this rulemaking;
—Between 150 and 449 deaths are estimated to occur each year among workers exposed to systemic toxins, cardiovascular toxins, metabolic toxins, and neurological toxins covered in this rulemaking.

Summing these estimates, OSHA believes that between 155 and 470 non-cancer-related occupational fatalities occur each year. The same offsetting considerations discussed in the analysis of the cancer fatalities avoided under this rule, also apply here. While some substances are being controlled by activity outside of this rulemaking, any overestimate effect is balanced by an underestimate of the real excess mortality rate for workers with high exposure levels to the chemicals under consideration.

In sum, the combined estimate for the number of cancer and non-cancer deaths potentially avoided each year by compliance with the proposed new limits, is between 293 and 746 or an average of 519 fatalities avoided each year.

Additional Comments and an Alternative Method for Estimating Excess Mortality Rates

The analysis described above to estimate the number of fatalities that are potentially preventable relies on published estimates of the proportion of all U.S. fatalities that are believed to result from occupational illnesses. These estimates were used with U.S. cause-specific mortality rate figures to estimate the excess mortality rate among all U.S. workers, by cause of death (shown in Table III–4).

In making these excess mortality rate estimates, OSHA applied the excess number of fatalities across the U.S. working population occupationally exposed to chemicals. Implicit in this approach is an assumption that all workers are at some risk of fatality from all causes of death. In fact, only a portion of the workforce are at risk of fatality from each type of occupational illness. Deaths will occur only among workers who are potentially exposed to carcinogens; no excess deaths will occur among workers who are not so exposed. Similarly, not all workers are at risk of dying from occupationally-related cardiovascular illnesses; only some portion of the workforce are at excess risk and all fatalities resulting from occupationally-related cardiovascular disease will occur among this subset of workers. Because OSHA's excess mortality rate estimates presented earlier were derived by applying the estimated number of work-related fatalities across the exposed U.S. workforce, excess mortality rate figures are likely to be substantially understated.

To assess the magnitude of this bias, OSHA conducted an alternative analysis to estimate the number of work-related fatalities that are expected to occur among workers exposed above the proposed limits. This alternative assessment relied on judgments regarding the general increase in mortality rates that are frequently observed in epidemiologic studies that demonstrate a causal relationship between exposure to toxic substances and excess disease mortality. The alternative assessment is presented in Table III–6.

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TABLE III-6

ALTERNATIVE ASSESSMENT OF NUMBER OF FATALITIES EXPECTED TO OCCUR AMONG WORKERS CURRENTLY EXPOSED ABOVE PROPOSED LIMITS

Cause of Death	U.S. Cause-Specific Mortality Rate, Per 100,000 Residents[a] (1985)	Estimated Excess Mortality Rate Per 100,000 Workers at Risk From Hazard	Number of Workers Exposed Above Proposed Limits	Annual Number of Fatalities Among This Group of Workers
Cancer	193.3	193.3[b]	499,716[c]	966
Chronic Pulmonary Disease	31.3	9.4[d]	647,018[c]	61
Chronic Liver Disease	11.2	3.3[d]	384 229[c]	13
Cardiovascular,	418.5	125.6[d]	1,250,811[c]	1,571
TOTAL				2,611

[a] Source: National Center for Health Statistics [5, Table 11].

[b] Assumes that overall cancer mortality rate among workers at risk is twice the U.S. rate (i.e., a 100 percent excess rate).

[c] From Table III-4.

[d] Assumes that overall disease mortality rate among workers at risk is 1 3 times the U.S rate (i.e., a 30 percent excess risk).

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The overall U.S. cancer mortality rate for 1985 is 193.3 deaths per 100,000 residents (Table III–6). Typically, when causal relationships between exposure and excess lung cancer mortality are found in epidemiologic investigations, the exposed cohort frequently shows a cancer mortality rate of 1.1 to 10 times higher than the general population. For cancers that are more rare than lung cancer, mortality rates among working populations may be 50 times higher than for the general population. An alternative estimate of the number of cancer fatalities expected to occur among the estimated 499,716 workers exposed above the proposed limits for the potential carcinogens could be developed based on the assumption that the overall cancer fatality rate among these workers is twice that of the U.S. population (i.e., 386.6 per 100,000 workers vs. 193.3 per 100,000 residents). The *excess* cancer mortality rate among these workers is therefore assumed to be 193.3 per 100,000 workers (386.6–193.3). Applying this estimated excess cancer mortality rate to the 499,716 workers exposed above the proposed limits yields as estimated 966 cancer deaths occurring annually that are attributable to occupational exposure. This same approach could be used for estimating non-cancer-related fatalities assuming that the overall fatality rate among workers at risk from these illnesses is 1.3 times the corresponding U.S. mortality rate (mortality rates of 1.1 to 1.5 are frequently observed in epidemiologic studies demonstrating causal relationships between exposure and excess fatalities). This amounts to an *excess* mortality rate of 30 percent above the overall U.S. rate.

Applying these excess mortality rate figures to the estimated worker populations exposed above the proposed limits, OSHA estimates that, among these workers, 61 deaths occur annually due to chronic pulmonary disease, 13 deaths occur annually due to liver disease, and 1,571 deaths occur annually due to cardiovascular, neurological, and renal disease. In total, including cancer, OSHA estimates that 2,611 work-related fatalities may be occurring each year among employees who are exposed above the proposed limits for substances included in this rulemaking.

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IV. Assessment of Nonregulatory Alternatives

Introduction

The declared purpose of the Occupational Safety and Health (OSH) Act of 1970 is "* * * to assure so far as possible every working man and woman in the Nation safe and healthful working conditions and to preserve our human resources * * *." Thus, the Act requires the Secretary of Labor, when promulgating occupational safety and health standards for toxic materials or harmful physical agents, to set the standard "* * * that most adequately assures, to the extent feasible, on the basis of the best available evidence, that no employee will suffer material impairment of health or functional capacity * * *." It is on the basis of this congressional directive that OSHA has initiated regulatory actions to reduce the adverse health effects associated with occupational exposure to hazardous substances.

Market Failure

Economic theory suggests that the need for government regulation is greatly reduced where private markets work efficiently and effectively to allocate health and safety resources. The theory typically assumes perfectly competitive labor markets where workers, having perfect knowledge of job risks and being perfectly mobile among jobs, command wage premiums that fully compensate for any risk of future harm. Thus, theoretically, the costs of occupational injury and illness are borne initially by the firms responsible for the hazardous workplace conditions and, ultimately, by the consumers who pay higher prices for the final goods and services produced by these firms. With all costs internalized, private employers have an incentive to reduce hazards wherever the cost of hazard abatement is less than the cost of the expected injury or illness. The resultant level of safety and health is considered "efficient" in the sense that it minimizes the sum of the costs of hazard prevention and of injury or illness. Perfectly competitive labor markets, however, do not exist for many industrial markets. OSHA, therefore, believes that it must take appropriate actions to provide greater health protection for workers exposed to toxic substances.

Evidence indicates that market forces have not been effective in reducing excessive occupational exposure to hazardous substances, thereby contributing to the consequent development of occupational diseases. In spite of the danger associated with the inhalation or other exposure to hazardous substances, the social costs of production have not been internalized, in part, because of market imperfections and the existence of externalities. Consequently, the amount of protection that the private market will offer to workers differs from the socially desired level.

First, evidence on occupational health hazards in general suggests that in the absence of immediate or clear-cut danger, employees and employers have little incentive to seek or provide information on the potential long-term effects of exposure. Employers faced with potentially high compensatory payments may, in fact, have a disincentive to provide information to employees. When relevant information is provided, however, employers and employees might still find informed decisionmaking a difficult task, especially where long latency periods precede the development of chronic disabling disease. Morever, if signs and symptoms are nonspecific—that is, if an illness could be job-related or could have other causes—employees and employers may not link disease with such occupational exposure.

Second, even if workers were fully informed of the health risks associated with exposure to hazardous substances, many face limited employment options. Nontransferability of occupational skills and high national unemployment rates sharply reduce a worker's expectation of obtaining alternative employment quickly or easily. A worker employed in a foundry, for example, could find it difficult to apply occupational skills to a new job in searching for a safer workplace.

In many regions of the country, the practical choice for workers is not between a safe job and a better paying

but more hazardous position, but simply between employment and unemployment at the prevailing rates of pay and risk. In addition to the fear of substantial income loss from prolonged periods of unemployment, the high costs of relocation, the reluctance to break family and community ties, and the growth of institutional factors such as pension plans and seniority rights serve to elevate the cost of job transfer. Thus, especially where wages are more responsive to the demands of more mobile workers who tend to be younger and perhaps less aware of job risks, hazard premiums for the average worker will not fully compensate. Where this is the case, labor market negotiations are unlikely to reflect accurately the value that workers place on health.

In addition to the market imperfections, externalities occur if employers and employees settle for an inefficiently low level of protection from hazardous substances. For the competitive market to function efficiently, only workers and their employers should be affected by the level of safety and health provided in market transactions. In the case of occupational safety and health, however, society shares part of the financial burden of occupationally induced diseases, including the costs of premature death, chronic illness, and disability. Those individuals who suffer from occupationally related illness are cared for and compensated by society through taxpayer support of social programs, including welfare, Social Security, and Medicare. These combined factors of labor market imperfections and the existence of externalities contribute to the failure of the market to supply healthful working conditions in industries where hazardous substances exist.

Tort Liability

The use of liability under tort law is one nonregulatory alternative that has been increasingly used in litigation concerning occupationally related illnesses. Prosser [1] describes a tort, in part, as a "civil wrong, other than a breach of contract, for which the court will provide a remedy in the form of an action for damages," although he says that "a really satisfactory definition has yet to be found."

If the tort system applies, it would allow a worker whose health has been adversely affected by occupational exposure to a hazardous substance to sue and recover damages from the employer. Thus, if the tort system is effectively applied, it might shift the liability of direct costs of occupational disease from the worker to the firm under certain specific circumstances.

With very limited exceptions, however, the tort system is not a viable alternative in dealings between employees and their employers. All states have legislation providing that Workers' Compensation is either the exclusive or principal remedy available to employees against their employers. Thus, under tort law, workers with an occupational disease caused by exposure to a hazardous substance can only file a product liability suit against a third party manufacturer (e.g., Johns Manville), processor, distributor, sales firm, installer, agency, or contractor. It is often difficult, however, to demonstrate a direct link between an exposure to a hazardous substance and the illness.

In order to pursue litigation successfully, there must be specific knowledge of the magnitude and duration of a worker's exposure to a hazardous substance, as well as the causal link between the disease and the occupational exposure. Usually, it is extremely difficult to isolate the role of occupational exposures in causing the disease, especially if workers are exposed to many toxic substances. This difficulty is further compounded by the long latency periods that are frequently involved. In addition, the liable party must be identifiable, but workers may have several employers over a working lifetime. The burden of proof that an occupational exposure to a hazardous substance occurred, that a specific employer is the liable party, and that the exposure level was significant may prohibit the individual from initiating the suit.

The costs associated with producing information and with litigation itself may be quite substantial. First, information is a public good, which means that once produced it can be transmitted inexpensively to any number of individuals without diminishing the quality or quantity of the information. It is, therefore, difficult to control distribution once the information is produced. A producer of information may find that information produced at great expense can be acquired freely by potential customers, and that consequently, the market for the information has virtually disappeared. As a result, public goods are typically underproduced relative to what is considered economically efficient. This general undersupply of information adversely affects the workers' awareness of the cause of their illness and thus reduces the likelihood that they will pursue tort liability suits.

Second, legal proceedings impose costs on both plaintiffs and defendants. In deciding whether to sue, the tort victim must be sure that the size of the claim will be large enough to cover legal expenses. In effect, the plaintiff is likely to face substantial transaction costs in the form of a contingency fee, commonly 33 percent, plus additional legal expenses. The accused firm must also pay for its defense.

The majority of occupational disease tort activity has involved workers exposed to asbestos. To date, about 25,000 individual plaintiffs have filed asbestos lawsuits in the country. These employees avoided the exclusive remedy of Workers' Compensation by suing suppliers of asbestos instead of employers. A report prepared by the Research Triangle Institute entitled, *Tort Liability and Worker Health: An Examination of the Economic, Legal, and Scientific Issues Surrounding the Occupational Disease Protection Afforded by Tort Law* [2], contains some data pertaining to legal costs and the size of awards. One investigator, for example, found that an average ratio of legal costs to proceeds was 37 percent for a sample of cases. The data, however, do not separate legal fees paid by the defendants and plaintiffs.

Insurance and liability costs are not borne in full by the specific employer responsible for the risk involved. For firms that are insured, the premium determination process is such that premiums only partially reflect changes in risk associated with changes in exposure to hazardous substances. This lack of complete adjustment is the so-called "moral hazard problem," which is the risk that arises from the possible dishonesty or imprudence of the insured. As the insured firm has paid an insurance company to assume some of the risks, that firm has less reason to exercise the diligence necessary to avoid losses. Transfer of risk is a fundamental source of imperfection in markets.[1]

For firms that self-insure or carry liability insurance with a large deductible, the costs of a single claim may be fully borne by the firm. Very small firms, and large firms with a large number of claims, however, may fail to meet the full costs by declaring bankruptcy. For example, the Johns

[1] For a general discussion of moral hazard as a source of market failure, see Arrow [4] and Spence and Zeckhauser [5]. For applications of this concept to employee health and safety, see Chelius [6], Rea [7], and Consad and General Research Corporation [8, Section 5.1].

Manville Corporation [2] declared bankruptcy to avoid massive claims associated with asbestos-related disease. Although the firm experienced a sharp decline in the value of its stock, it is still in business, while its obligation to pay asbestos-related claims is in considerable doubt. Other asbestos producers including U.N.R. Industries, Inc. and Amatex Corporation have followed the example of the Manville Corporation by filing for bankruptcy [9], further reducing the chances that their workers or others who contract asbestos-related diseases will collect Workers' Compensation or tort liability awards.

Workers' Compensation

The Workers' Compensation system is a result of the perceived inadequacies in liability or insurance systems to compel employers to prevent occupational disease or compensate workers fully for their losses. The system was designed to internalize some of the social costs of production, but in reality, it has fallen short of compensating workers adequately for occupationally related disease. Thus, society shares the burden of occupationally related adverse health effects, premature mortality, excess morbidity, and disability through taxpayer support of social programs such as welfare, Social Security disability payments, and Medicare.

Compensation tends to be inadequate, especially in permanent disability cases, in view of the expiration of benefit entitlements and the failure to adjust benefits for changes in a worker's expected earnings over time. As of January 1987, 8 states still restricted permanent disability benefits either by specifying a maximum number of weeks for which benefits could be paid or by imposing a ceiling on dollar payments [10].

At present, time and dollar restrictions on benefit payments are even more prevalent in the area of survivor benefits. The duration of survivor benefits is often restricted to 10 years, and dollar maximums on survivor payments range from $7,000 to $60,000. In addition, it should be noted that if the employee dies quickly from the occupational illness and has no dependents, the employer need pay only nominal damages under Workers' Compensation (i.e., a $1,000 death benefit).

Finally, in spite of current statutory protection, disability from occupational diseases represents a continuing, complex problem for Workers' Compensation programs. Occupational diseases may take years to develop, and more than one causal agent may be involved in their onset. Consequently, disabilities resulting from occupationally induced illness often are less clearly defined than those from occupationally induced injury. As a result, Workers' Compensation is often a weak remedy in the case of occupational disease. For example, as recently as April 1983, the U.S. Supreme Court refused to hear an occupational disease case *(Richard D. Bunker* v. *National Gypsum Co.)* involving a worker who was diagnosed as having asbestosis 23 years after the expiration of the 3-year time limit allowed by Indiana law for filing a compensation claim [11]. Indeed, there is some evidence indicating that the great majority of occupationally induced illnesses are never reported or compensated [12].

The insurance premiums paid by a firm under the Workers' Compensation system are generally not experience rated—that is, they do not reflect the individual firm's job safety and health record. About 80 percent of all firms are ineligible for experience rating because of their small size. Such firms are class rated, and rate reductions are granted only if the experience of the entire class improves. Even when firms have an experience rating, the premiums paid may not accurately reflect the true economic losses. Segregation of loss experience into classes is somewhat arbitrary, and an individual firm may be classified with other firms that have substantially different normal accident rates. An experience rating is generally based on the benefits paid to workers, not on the firm's safety record. Thus, employers may have a greater incentive to reduce premiums by contesting claims than by initiating safety measures.

In summary, the Workers' Compensation system suffers from several defects that seriously reduce its effectiveness in providing incentives for firms to create safe and healthful workplaces. The scheduled benefits are significantly less than the actual losses to the injured workers, and recovery is often very difficult in the case of occupational diseases. Thus, the existence of a Workers' Compensation system limits an employer's liability significantly below the actual costs of the injury. In addition, premiums for individual firms are unlikely to be specifically related to that firm's risk environment. The firm, therefore, does not receive the proper "signals" and consequently fails to invest sufficient resources in reducing workplace injuries and illnesses. The economic costs not borne by the employer are borne by the employee or, as is often the case, by society through public insurance and welfare programs.

Standards of Other Organizations

Traditionally, representatives of professional organizations have collectively developed voluntary guidelines to assist members in maintaining safe and healthful working conditions for their employees. These guidelines are widely disseminated among members of the organizations and, at times, have been adopted as guidelines by organizations beyond the initiating one as well as by industry groups. In some cases they have become the *de facto* industry standard. Three professional organizations have developed voluntary guidelines in the form of exposure limits for chemical substances: The American National Standards Institute (ANSI); the American Industrial Hygiene Association (AIHA); and the American Conference of Governmental Industrial Hygienists (ACGIH). ANSI has withdrawn its earlier hazardous substance standards and has stated it does not intend to publish any others. The AIHA has a rather limited list of recommended limits. However, the ACGIH has published an extensive list of threshold limit values (TLVs) for many years. The ACGIH is recognized throughout the world for its members' expertise and contribution to industrial hygiene.

In May 1971, OSHA adopted as Federal health standards the exposure limits recommended by ANSI and ACGIH for 425 chemicals. Since that time, advances in scientific knowledge have demonstrated that those limits are not always adequate to protect employee health. Consequently, the ACGIH, the professional organization which continues to develop TLVs, has changed its recommendations yearly to reflect later information. However, adherence to the TLVs developed after 1971 is purely voluntary. Except for imminent hazards, there is no sanction for failure to comply with the limits and many employers have not adopted practices which would control employee exposure to these new levels.

In addition to professional organizations, international bodies such

[2] Johns Manville Corporation, formerly the world's largest asbestos manufacturer, filed for Chapter XI protection under the Federal Bankruptcy Law in August 1982. The company was financially solvent when it filed for bankruptcy but estimated that it would ultimately face a cost of more than $2 billion to settle 52,000 asbestos-related claims. In the meantime, the company's assets have been frozen and successful plaintiffs cannot collect awards [9].

as the European Economic Community, the International Labor Organization, and the World Health Organization have recommended exposure limits for some hazardous substances. While these limits may not be as widely known in the United States as those of U.S. professional organizations, they are made available to the industrial hygiene community through professional journals and meetings. Within the U.S., the National Institute for Occupational Safety and Health (NIOSH) of the Department of Health and Human Services has published recommended exposure limits (RELs) for a number of chemicals. These are publicized through NIOSH Current Intelligence Bulletins and other publications which are widely disseminated.

Although the ACGIH TLVs and the NIOSH RELs are widely recognized by health professionals and employers alike, OSHA has found that some employers are not complying voluntarily with the newer TLVs, the RELs, or the standards of other bodies. Chapter III discussed OSHA's estimates of the extent of exposures in excess of the TLVs, and the adverse health effects resulting from the exposure. OSHA believes that significant numbers of employees are exposed to chemicals at levels exceeding those recommended by other organizations, and that OSHA cannot rely on employers to comply voluntarily with the recommendations. Therefore, OSHA concluded that this nonregulatory alternative is not generating the optimal level of occupational health.

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V. Technological Feasibility

Feasibility Determination

This chapter presents a technological feasibility analysis of industry's ability to meet OSHA's proposed permissible exposure limits (PELs) for a wide range of occupational health hazards. These PELs would include limits on airborne concentrations of substances, and in some instances, direct contact of the skin with the substance.

The control of workplace exposures to toxic chemicals involves combining a variety of standard techniques to solve a situation-specific problem. OSHA believes that existing engineering controls are available to reduce exposure levels to new proposed levels. Standard controls have been adapted in numerous situations to solve situation-specific problems.

In this chapter, a number of process situations where airborne exposures have been controlled by the use of engineering controls are described. OSHA has found examples of successful engineering controls for each of the processes identified in the sample survey and presented in Supplement 2.

Types of Controls

In general, three basic types of controls may be employed to reduce employee exposures:

- Engineering controls
- Work practices and administrative reforms
- Personal protective equipment

Consistent with OSHA regulations and policy, this chapter examines the feasibility of engineering controls and work practices to control employee exposure, in preference to personal protective equipment.

Engineering Controls

Engineering controls involve the use of: Local exhaust ventilation; general ventilation; isolation of the worker and enclosure of the source of emissions; process modifications; equipment modifications; and substitution of non-hazardous chemicals. These methods may be used alone or in combinations of any two or more controls depending upon the needs of a specific situation. Variations in situations usually result from the type of process being used and the number of chemicals in the air. However, these controls are considered standard techniques which will effectively control these variables either by themselves, or coupled with changes in work practices.

Ventilation

Perhaps the most widely used technique for controlling chemical exposures is the use of ventilation. General ventilation uses the movement of air within the general work space to displace or dilute the contaminant with fresh outside air. General ventilation is not typically the preferred control method in most operations due to the large volumes of air movement required. Local exhaust ventilation uses much smaller volumes of air, exhausted from the point at which contaminants are generated to remove the contaminant at the source.

Isolation

Isolation involves placing a physical barrier between the hazardous operation and the worker. Many modern, automated manufacturing processes are now fully enclosed in ventilated cabinets. The effectiveness of such a control technique depends on the frequency with which the workers have to enter the enclosure during normal operations. In other situations, rather than placing the process or machine in an enclosure, the worker may be put into a controlled atmosphere enclosure. Many processes which involve potential chemical exposures are operated remotely by operators in air conditioned booths.

Substitution

Substitution refers to the replacement of a toxic chemical in a particular process or work area with another, less toxic product. Properly applied, substitution can be a very effective control technique. However, care must be taken to ensure that the proposed substitution performs in a similar manner to the product being replaced. In addition, it is essential that the substitute be carefully evaluated to

ensure that in controlling one hazard, that another, different hazard is not inadvertently introduced. The substitute must also be compatible with existing manufacturing equipment and processes.

The success of these techniques will depend on the physical properties of the chemicals and emissions encountered (boiling point, vapor pressure, etc.) and the process operating conditions (temperature, pressure, etc.). In some cases, particularly with cleaning solvents, substitution may provide the quickest and most effective means of reducing exposure. In other situations where particular physical or chemical properties are required, major effort may be required to alter processes or install or expand local or general dilution ventilation. The extent to which engineering controls may be effectively used will vary from industry to industry, as well as plant to plant within an industry.

Work Practices and Administrative Reforms

Work practice controls include housekeeping procedures, material handling or transfer procedures, leak detection program, training and personal hygiene. In many cases, it is possible to bring about substantial reduction in employee exposures by applying work practice controls.

Personal Protective Equipment

Where it is impractical to apply engineering or work practice controls, or where their application will not consistently reduce employee exposures below the proposed PELs, personal protective equipment may be used to prevent and reduce exposures.

Industry Engineering Controls

To determine whether engineering controls and work practices can reduce employee exposures to the proposed PELs, OSHA, through its contractors, examined typical work processes found in a cross section of industries. Supplement 2 contains a list of processes identified in the nationwide sample survey of over 5,300 firms. Using this list, industry experts identified which major processes had potential hazardous exposures and may require additional engineering controls or different work practices in order to achieve the proposed PELs. To assess whether these would be feasible for the processes within the industry group, records maintained by OSHA and NIOSH were searched to identify examples of the successful application of controls to these processes. Based upon the judgments of the industry experts, a determination was made as to the probable feasibility of achieving the proposed PELs.

The following are examples of feasible methods of controlling exposure to hazardous substances encountered in processes used in the SICs for which costs and benefits have been identified. Unit costs for these similar controls were used as the basis for the cost projections in Chapter V.

SIC 20—Food and Kindred Products

The levels of ammonia gas encountered in poultry processing (SICs 2016 and 2017) were controlled by the appropriate placement of cut-off valves to freezer coils and by the use of an alarm detection system to monitor ambient air conditions.

In beet sugar refining (SIC 202), exposure to calcium oxide was controlled by replacing slide gates with rotary feeders. A milk products plant (SIC 2023) controlled carbon dioxide exposures by using a hood which fully enclosed the chiller-conveyor line and exhausted air from the system to an exterior baghouse.

Carbon dioxide levels resulting from the use of dry ice were controlled at a meat packing plant (SICs 2011 and 2013) by a stainless steel exhaust hood. Similarly, a poultry dressing plant controlled carbon dioxide emissions by using a slotted blood exhaust ventilation system.

A food processing plant (SIC 202) controlled carbon dioxide exposure by increasing the number of air changes in the packaging room.

SIC 21—Tobacco Products

Tobacco dust and residual pesticide dusts created during cutting and shredding operations are reduced through the use of local exhaust ventilation. This is also used to control emissions of ethyl alcohol-based chemical flavorings during blending operations.

SIC 22—Textile Mill Products

Textiles are dyed at various stages in their manufacture, including unspun fibers, unwoven yarn, and finished fabric. Workers who prepare fabrics from unspun fibers are of particular concern, since they could be potentially exposed to dyes contained on dusts generated during manufacture. In addition, some dyes possess much poorer fastness to wet treatment than do others; persons who launder such clothing are potentially exposed to dyes. Stringent control measures and work practices can prevent such exposure.

Several generally acceptable practices for the control of hazardous materials can be used wherever there is potential for exposure. For example, pressure failure alarms for closed systems and exhaust ventilation can rapidly indicate a system failure that might result in the release of substantial quantities of dyes. Continuous flow indicators, such as water or oil manometers properly mounted at the juncture of a fume hood and duct throat and marked to indicate acceptable airflow, will give a readily observable indication of decreased efficiency in the ventilation system for the hood. Wet methods, vacuum cleaning, or other methods that do not lead to redispersion of settled dust should be used for plant maintenance and sanitation. Dry sweeping or blowing with compressed air should be prohibited.

SIC 23—Apparel

Chemical exposures in the apparel industry occur principally as a result of three exposure sources: Spot cleaning, dry cleaning and contact with treated fabrics.

Spot cleaning and dry cleaning operations can be controlled with the use of local exhaust ventilation and general ventilation. Work practice improvements help reduce solvent exposure during transfer operations. Routine scheduled maintenance is used to detect and control leaks from door gaskets and seals. Contact dermatitis is reduced through the use of disposable gloves and adherence to a personal hygiene program.

SIC 24—Lumber and Wood

The primary worker exposure in the lumber and wood industry is wood dust. For the operation of large equipment (e.g. in debarking and sawmill activities), the operator can be placed in an enclosed control booth, or in the case of moving equipment (e.g. cherry pickers, loaders and cranes), the operator can be located in an enclosed cab. In both cases, air would be filtered and conditioned. In the case of felting or matting process lines, or such equipment as belt sanders, the equipment can be enclosed or hooded and vented to a baghouse. For smaller equipment, such as variety saws, tenoners, and dovetailers, hoods or various types of negative pressure (or combinations of positive and negative pressure) local ventilation devices can be used to control wood dust. In the case of hand-held sanders, a vacuum system can sometimes be applied to the process. Some other wood dust generating equipment can also be enclosed (e.g. planers), but this is generally done for noise control.

SIC 25—Furniture

In the metal office furniture manufacturing sector (SIC 2522), air contaminants from the coating process have been controlled. Prefabricated sheet components for file cabinets are prewashed and coated with polyester or acrylic on a high speed conveyor line. The application process includes manual spraying of cabinets with airless atomizing sprayers and electrostatic spray guns on reciprocators. Manual spraying operations are performed in downdraft booths. Filtered fresh air is supplied through the open top of the booths and removed at the bottom through a water curtain by exhaust fans mounted on the roof of the booth. Spray headers in exhaust plenums clear paint mist from the air stream. Automatic spray booths contain electrostatic spray guns and side draft ventilation. Furthermore, organic solvent vapors in the paint mixing and storage room are controlled by equipping each drum with a heavy barrel cover, an integral adgitator, sealed pipe openings, and a closeable access line.

SIC 26—Paper and Allied Products

Pulp mills occur primarily in SIC 2611, but can also be present as part of the operations in SICs 2621 and 2631. High control costs could potentially be incurred because of the large quantities of chemicals used in breaking down pulp to form cellulose and the reactions that occur in the digesting process between these chemicals and the substances contained in the wood fiber. Large quantities of chemicals are also used in the bleaching operations. The digesting and bleaching operations are also very extensive. Large quantities of pulp are generally produced from wood in these mills either for captive use or for shipment to paper and paperboard mills. The type of controls that would be used include ventilation, enclosure and/or process change, but less likely the latter. Various engineering controls have been used by the paper mill industry to prevent the mixing of toxic chemicals in sewer lines. Tanks containing the hazardous chemicals have been isolated and surrounded by dikes. Discharge lines have been re-routed to prevent accidental mixing.

SIC 27—Printing and Publishing

Exposure to solvent vapors in the silk-screening sector of a printing plant (SIC 2751) was controlled by installing a new ventilation system and an exhaust fan.

SIC 28—Chemicals and Allied Products

The plastic materials and resins manufacturing sector (SIC 2821) used a tank with a hinged cover and fixed ductwork as an exhaust when dumping dye and additives into hot methanol.

Dust exposure during the bag opening operation in paint manufacturing (SIC 2851) was controlled by modifying the hood to increase dust capture. Likewise, a new dust collection system (collection hoods) with increased capture velocity was installed for use in the bagging and packaging of pesticides (SIC 287).

Pharmaceutical manufacturers (SIC 2824) addressed the problem of nuisance dust particles by fitting vacuum crescents and elephant trunks on point sources, by fitting chutes with covers, and by placing vacuum attachments on receiving drum covers. Additionally, monitoring was performed from an outside room.

In order to reduce employee exposure to sulfur dioxide while producing sulfur dioxide gas (SIC 2819), sample collection units were enclosed and attached to a fume collection system. Sample waste was recycled to prevent open exposure in process areas. Electronic spent acid interface detectors were installed to eliminate the need for employee visual inspection of intermittently pulled samples. To control TDI exposure in urethane foam manufacturing (SIC 2822), the bun conveyor was enclosed and exhausted. Employee exposure was limited to the startup and finish procedures when installing and removing bun support. A mechanism was designed to support the bun, which eliminated the need for it to be done manually.

The production of paints (SIC 2851) is a batch procedure which involves the following steps: Prebatching, mixing, dispersing, tinting and shading, filling, and storage or shipping. When prebatching or mixing, an employee will slit a bag of dry pigment with a knife and either scoop out the contents for weighing or dump the pigment into the mixer. In some cases, pigments are received in a slurry form and are piped directly into the mixer. Solvents and other raw materials are added into the mixer. Once combined, the mixture is in a past or slurry form. This mixture is then thoroughly dispersed in a roller, ball, or sand mill or a high-speed disperser all of which are generally closed processes. The paste is transferred to a storage tank where thinning or other agents are added. The paint is later drawn off, filtered and packaged in cans or drums. Airborne dust exposures to components in dry pigments occur during the prebatching and mixing operations when the bags of pigments are opened and dumped. Exposure to chemicals in dry pigments can also occur from pigment spillage and empty bag flattening and disposal. Once the batch is in solution in the mixer, there are no further dust emission points. Exposure to solvents can occur during addition of these ingredients to the mixing tanks, during any leaks or spills, and during packaging.

Local exhaust ventilation would be used to control exposures to dusts and fumes in the paint production processes. Pigment dust exposures at the dumping station can be controlled with the use of a vented enclosure kept under negative pressure by a ventilation system. Empty bags would be manually ejected through a side opening into a large plastic disposal bag to minimize dust generation during bag flattening and disposal.

Exposures to solvents would be minimized with the use of portable hoods attached to flexible ductwork. These ventilation hoods could be placed over the liquid dumping process and also the packaging operation if the percentage and volatility of the solvents would result in exposures.

SIC 29—Petroleum Refining

In order to assure the quality of petroleum products and determine quality of waste streams, petroleum refiners must sample their process streams periodically. As with maintenance, workers that sample process streams are at risk of being in close contact with a variety of chemicals. Controls for this operation involve sampling boxes that vent gases and vapors away from the operator and/or shield the operator from accidentally splashed or spilled material.

Process stream samples are taken to the laboratory to determine if their qualities lie within acceptable limits. As laboratory workers perform analyses, they can be exposed to various organic and inorganic chemicals if appropriate engineering controls are not in place or if proper procedures are not used. Exposure controls include exhaust fans and laboratory ventilation hoods.

SIC 30—Rubber and Miscellaneous Plastics Products

To control dust while cutting reinforced plastic, a local exhaust fan was installed at the back of the cutting instrument.

The materials to be mixed in a Banbury mixer used in tire production (SIC 3011) are charged into a hopper and then into a chamber which contains the mixing rotors. The rotors mix the materials into a homogeneous mixture, and then are discharged through a drop

door or slide gate. When the hot rubber mixture is discharged, it emits an aerosol which looks like smoke. These aerosols are typically well controlled. The operations and equipment used to charge the mixer can be sources of dust exposures. The powdered chemicals in the operation are weighed in rigid plastic containers or paper bags, all of which are either emptied or dropped into the mixer by workers. This dropping, the handling of these contaminated containers or the crushing of the bags which contained chemicals, all expose the workers to dust. When the rubber and chemicals are charged using a charging conveyor, a dust exposure can result from the use of kaolin as an anti-tack agent. The conveyor belt becomes contaminated by the kaolin. When workers place materials on the conveyor, dust puffs into the worker's breathing zone.

One design which minimizes dust dispersion is the use of an automated batching system to isolate the worker from the dust. Other techniques include the use of rubber bins on a tilt stand to charge carbon black rather than the use of screw conveyors. The kaolin can be replaced by a soap anti-tack agent. To capture the aerosol fumes, a local exhaust hood can be placed by the drop gate between the legs of the mixer. Exhaust hoods may be placed by the hopper vents, on the carbon black charge chute, about the charging door and surrounding the dust ring seal on the mixer ram.

SIC 31—Leather and Leather Products

Toluene exposure in the shoe manufacturing industry (SIC 314) was decreased by revising standard work practices to reduce the contact time between leather and toluene. This also reduced the generation of vapors.

SIC 32—Stone, Clay and Glass Products

In batch mixing of raw materials for glass production (SICs 321 and 322) drysweeping and/or the use of compressed air for cleaning may contribute substantially to the employees overall exposures. By substituting vacuum cleaning systems, worker exposure can be reduced.

SIC 33—Primary Metals Industries

In the primary metal industries (including SICs 331, 332, 333, and 334), the overall size of the process equipment and buildings makes contaminant control difficult. Local exhaust and general ventilation systems used in this industry group must move large volumes of air as compared to similar systems in other industries. Baghouses and other air cleaning devices used in such systems must also be proportionally larger. This effect of scale translates to higher than average control costs, but use of ventilation can control exposures to the proposed levels.

SIC 331—Basic Steel Products

Primary metal products manufacturing (SIC 331) controlled dust with a side draft enclosure exhaust hood and a circular backdraft booth, both driven by a blower-dust collector. Ventilation was provided by a freely suspended hood and conical hood.

The replacement of slide gates on transfer hoppers with butterfly-type valves assisted in dust control in the material handling of electrometallurgical products (SIC 3313).

SIC 332—Iron and Steel Foundries

The arc air process in steel foundries (SICs 3324, 3325) was used during the processing of steel castings to control fumes. In order to ventilate the arc air booths, fumes were exhausted through the back of the booth and fresh air was supplied from above and behind the operator.

Steel foundries (SICs 3324, 3325) used an overhead canopy hood during the induction melting of steel to control fumes. The hood consisted of sheet metal barriers extending down from the roof to the top of the hot metal ladle monorail. Thermal drafts carried the fumes upward into the hood where they were exhausted by ventilators. Mancooler fans behind the workers pushed some fumes under the hood.

Emissions during the oxy-acetylene torch cutoff of risers from steel castings was encountered in iron and steel foundries (SIC 332). Castings were cut in a specially designed booth with a rear exhaust flow and a frontal air supply flow. Air pressure from the cutting nozzle of the torch was directed toward the rear exhaust port for effective dust and fume control.

Fume control of a sandwich-type inoculation in iron foundries (SICs 3321, 3322) was achieved through the use of a commercially available canopy hood. The fume-laden air was exhausted through mobile duct work and cleaned by a fabric collector before being discharged into the surrounding environment. The hood tilted with the furnace so that it always was directly over the ladle for fume capture.

Fume, dust, and gas control from the melting of iron (SICs 3321, 3322) in an arc furnace was achieved by the installation of a hood. The exhausts collected by the hood were filtered by cloth filters before being released into the external environment.

Control of dust and gas emissions from phenolic urethane cold box coremaking in iron foundries (SIC 3321, 3322) included local exhaust ventilation which provided negative pressure at the core box. Parting line gaskets, blow seals, and stripper pin o-rings were regularly maintained for emission control. Exhaust outlets captured excessive dust.

In an iron foundry (SICs 3321, 3322), hot combustion gases were exhausted and flowed through an after burner, cooled, and then passed through a dust collector. Tapping emissions were captured by a canopy hood. General ventilation was provided by mancooler fans.

SIC 333/334—Primary and Secondary Non-Ferrous Metals

Control of emissions from aluminum ore handling and storage (SIC 3334) was addressed with an unloader which uses movable vacuum nozzles to remove alumina and coke from barges. The ore was moved on an enclosed conveyor which was equipped with air exhaust hoods at loading and transfer points. The operator can be situated in an air conditioned cab.

Reduction of alumina dust emissions during ship unloading (SIC 3334) was achieved by automating and controlling operations from an enclosed control booth. Furthermore, mixing operations were hooded and exhausted.

During anode rodding in prebake plants during primary aluminum production (SIC 3334), spent butt remover, butt crushers, cast iron remover, and shot cleaner were exhausted to a bag filter dust collector. Use of induction furnaces and exhaust hooding reduced metal fume exposure during melting. Hoods and slotted hoods were also used. The operator can maintain controls from an enclosed console.

Control of air emissions during potline operations of aluminum smelters (SIC 3334) was achieved through the use of potroom ventilation and automated processes such as the use of hooding which consists of curved and ribbed shields, the employment of a dual draft system, and an exhaust system which leads to a dry scrubber. Other control methods included hooding with rigid air-operated doors which exhausted the emissions through air takeoffs to an expanding duct exhaust manifold which, in turn, was exhausted by a fan. Furthermore, computer-controlled systems existed which could automatically perform production functions without requiring workers to open pots or hood shields above pots.

The mercury cell process may be used in aluminum smeltering (SIC 3334) to produce chlorine gas from brine water. To reduce chlorine gas exposure as a result of this production, the diameter of the brine header was increased to accommodate the gas phase above the liquid phase; the number of cells in the system was increased; the pH of the brine was adjusted; the compressor controls were modified to accommodate surges in pressure; inlet box covers were replaced with better covers; and the brine feed nozzle flange was modified.

Several engineering controls have been recommended for copper smelting locations (SICs 3331, 3341). A preventive maintenance program can be developed and implemented to insure that ventilation and conveyor systems are operating properly. Dead beds can be installed in chutes to break the fall of material and reduce the level of dust generated. Pneumatic aerators can be installed to eliminate the need for manual air lancing in bins and chutes. Industrial vacuum systems can be used.

Collection hoods can be installed at each conveyor transfer point at copper smelter sites (SICs 3331, 3341) to control copper particulate. Primary copper smelting conveyor skirting can be properly adjusted, and fingers installed at discharge points. Inspection doors should not be left open, and the lunchroom/breakroom should be located outside of the reclaim building. General measures throughout copper smelting plants (SICs 3331, 3341) to control copper dust emissions included: Using local exhaust ventilation for localized sources, and general exhaust ventilation for areas with unidentifiable sources; enclosing conveyor belts and transfer points; enclosing the air conveying system for the transfer of flue dusts; enclosing workers' operating vehicles; installing secondary hoods on converters; prohibiting the blowing out of converters while on stack; performing preventive maintenance on balloon flues; not allowing converters to remain rolled out for extended periods of time; and providing cleaning rooms with filtered, tempered, positive pressure air. When hauling slag from the metal smelting operation, slag can be granulated after skimming with high velocity water; a chemical dust suppression system can be used when crushing any cooled slag; and the slag crew can ride downwind from fumes. Further engineering controls include constructing pulpits for operators; close-coupling the ventilation system to the Larry car; using dead beds in calcine loading; enclosing a portion of the building to block wind; and vacuuming the superstructure of the Larry car and any spills.

Controls used to decrease exposures to arsenic, dust and sulfur dioxide at primary copper and lead smelters (SICs 3331, 3332) included upgrading the present ventilation systems; operating electric furnaces at negative pressure; eliminating air lancing as a method of removing concentrates from receiving hoppers; using pneumatic aerators or belt wipes; using wet techniques in storage; reclaiming concentrates; and improving general housekeeping.

Exposures to lead, cadmium, and arsenic at lead and copper smelters (SICs 3331, 3332) were reduced by the replacement of old sintering machines with ones equipped with dust and fume controls and by placing a cover over the charge hole when slag was not being charged into the reverberatory furnace.

Use of a multipurpose crane with an enclosed cab reduced operator exposures to air emissions at carbon bake plants (SIC 3334). The cab was supplied with filtered conditioned air. The crane was equipped with a vacuum system which could aspirate cake from ovens and separate fines.

Controls for exposure to soluble platinum salts in precious metal refining (SIC 3339) included local exhaust ventilation used in jaw crusher and recovery sampling, maintenance of a closed system in refinery through use of glove box filters, the use of borohydrate solution to wash down spills and reduce salts to insoluble platinum metal, and mandatory showers and daily clothing changes.

Controls for the primary non-ferrous metals industry (SIC 333) included local exhaust ventilation systems; general dilution ventilation; covers, hoods and exhaust systems for belts, material handling and transfer systems; enclosure and exhaust of sinter machine area; local exhaust and dilution ventilation for the reverberatory and refinery areas.

The reduction of exposures to inert cadmium and silver dust during a ball mill operation was accomplished by building and equipment process changes such as local exhaust ventilation, hood enclosure of process or worker, and air cleaning equipment.

In the secondary smelting and refining of non-ferrous metals (SIC 334), particulate emissions from a dross mill were reduced by making modifications to the dust collection system and to air volumes drawn through the baghouse. Engineering controls used include increasing fan efficiency through the use of sheaves and belts, installing water sprays on crusher infeeds, running new pipe to localized dust areas, installing additional cleanout ports, and replacing the top of the baghouse.

Employee exposure to nuisance dust from zinc smelters (SIC 3333) was controlled by replacing the dross handling operation with a dross mill. The crusher was replaced with a rotating mixer, thus eliminating fugitive dust from this part of the process.

SIC 336—Non-Ferrous Foundries

Fumes were controlled during the casting of bronze in foundries (SIC 3362) through the use of enclosing hoods. A mobile hood exhausted the ladle at all hot metal transfer points. Flexible ducting connected the hood to a traveling exhaust carriage.

SIC 339—Miscellaneous Primary Metals Products

Manufacturers improved dust control using closed screw conveyors in the transport and manufacture of iron powder (SIC 3399). Open conveyor belts were changed to a closed screw conveyor system. Duct work was totally replaced. Local exhaust was provided for the rotary screens. New baghouses and electrostatic precipitators were also installed.

SIC 34—Fabricated Metal Products

Control of copper dust at a cookware manufacturing plant (SIC 3469) was addressed by unclogging the ventilation system, repositioning cooling fans, and instituting weekly ventilation system inspection and maintenance programs.

A plating shop (SIC 3471) uses extensive local exhaust ventilation to control worker exposure. Each part to the plated undergoes some surface pretreatment. This can consist of shot-peening, abrasive blasting, degreasing, wax or tape masking and other treatments. Parts are manually placed into the tank using an overhead hoist for large parts.

The tanks are set on top of concrete ducts. The floors of the shop and the aisles between the tanks are reinforced concrete, however the area around the perimeter of the tank is open to the basement and covered by steel grating. The ducts are connected to a fan on the roof of the building.

The largest of the hard chrome tanks, holding over 1000 gallons of plating solution, has a two sided lateral exhaust ventilation system. The slot on each side consists of a series of seven slots. The slots are set back from the edge of the tank but an overhanging hood extends to the edge of the tank. A second tank has both a two sided slot ventilation system and a cover. This two piece

cover is hinged to a ventilation manifold and extends beyond the front and rear edges of the tank.

Arc welding is performed in many SICs as an auxilliary process and in several industries such as fabricated structural metals (SIC 3441), as the principal process requiring engineering control. During the welding process, temperatures are sufficiently high to vaporize some of the base material of the electrode and produce large quantities of fumes containing the elements in the electrode and the base metal. Thus welders and other workers in the vicinity are exposed to mixtures of fume-sized particulates and both irritant and toxic gases which in combination may have additive or synergistic physiological effects.

Differences in worker exposure are attributable to a variety of factors including type of welding helmet worn, position of the welding operator, the work environment, arc time, and the availability and performance of ventilation equipment.

Arc time varies greatly due to differences in work schedules, set up times, and the sizes, shapes and types of tasks. Tasks can vary from short-term repairs conducted irregularly to full time production welding.

During arc time the fume is generated within or close to the worker's breathing zone. Background fume concentrations could also be significant if a large number of welders are working or the work is being performed in a relatively confined space.

Because of the numerous factors that can influence exposure levels during welding, four different types of controls can be used for various welding situations. The controls include: (1) Local exhaust ventilation for welding in shops; (2) ambient air cleaning devices to minimize background fume concentrations; and (3) a portable blower for use in confined areas.

Local exhaust ventilation configurations include: A welding bench with a backdraft hood for small to medium work pieces; a fixed close-capture hood placed at the back of a work rest table; a portable close-capture system including electrostatic precipitator; or an exhaust hose incorporated into the structure of the welding gun.

Ambient air cleaning devices are designed to lower background welding fumes which escape collection by the local exhaust system. The ambient air cleaner is expected to surpass general dilution ventilation systems in terms of both fume removal and cost.

A portable blower system works by exhausting fumes from a confined space through a large flexible tube.

SIC 35—Machinery

Employee exposure to welding fumes was controlled in the manufacture of pumps (SIC 3561) through the use of an air lux fume eliminator.

In the milling of tungsten carbide tools (SIC 354), the placement of local exhaust ventilation controlled cobalt exposures during the transfer of carbide.

In farm equipment manufacturing and repair (SIC 3523), paint mist was controlled through sophisticated application techniques as applied to downdraft spray booths. The use of heated paint in the painting of hay stack wagons allowed the airless atomization to take place at relatively low paint pressures. This resulted in low droplet velocity with little rebound.

SIC 36—Electric and Electronic Equipment

Electric lamp manufacturers (SIC 3641) have reduced mercury vapor in lighting plants. Glass pellets used as starters for fluorescent lamps were flame sealed after mercury had been injected into them. Overhead suction velocity of the exhaust system was increased to reduce mercury overexposure. Also, a special vacuum cleaner was employed to clean the turntable.

SIC 37—Transportation Equipment

A four-sided enclosure with electrostatic precipitator ventilation was used on the welding table in the manufacture of travel trailers (SIC 3792).

Engineering controls for styrene exposure in the fiberglass boat works industry (SIC 3732) included flexible duct exhaust ventilation that is transportable to the employee's point of operation.

General recommendations in the reinforced plastic boat making industry included local exhaust, the use of styrene-suppressed resins, and good work practices.

In the manufacturing of aircraft engine parts (SIC 3724), a hood was installed above an abrasive cutoff saw to capture the cobalt dust produced.

In the repairing and rebuilding of railroad cars (SIC 3743), present exhaust fans were relocated, cleaned and serviced and "upblast" roof ventilator fans were installed, thus changing the air every 15 minutes.

SIC 38—Instruments

Many fluxing agents are used in soldering and brazing operations during instrument manufacture. In most cases, these fluxes give of acid or alkali fumes when heated that can irritate the skin. Conducting soldering and brazing operations in well-ventilated areas and use of protective clothing and gloves is recommended.

For many soldering and brazing operations, control of fumes and vapors generated by dilution ventilation will be sufficient. That is, enough fresh air is added to the contaminated air that hazardous concentrations do not develop.

Local exhaust ventilation is the most effective means of control for airborne contaminants produced by the soldering or brazing process. Local exhaust ventilation can be provided by several types of equipment: freely movable hoods, fixed enclosures (booths), and down-draft benches.

A freely movable hood consists of a movable hood attached to a fan. The fan draws air from the work space and exhausts it outdoors, either directly or through a dust collection system. The hoods are normally constructed so that they can be moved into place by the solderer. The air handling system should move air at least 100 feet per minute across the soldering site at even the most remote point from the exhaust opening. It is important that the exhaust hood be placed as near as possible to the work being done. As such, the proper functioning of a freely movable hood is dependent upon good work practices of the solderer.

In some instances soldering or brazing operations carried out in a fixed location can be provided with a fixed enclosure. This is a structure built around the soldering or brazing operation which has a top and at least two sides. A means for drawing air through the work area is provided so that the work space is flushed continuously with fresh air.

Within such an enclosure, work should be arranged and conducted in such a way that the fresh air enters in the enclosure through the worker's breathing zone and then through the work space in which the contaminants are produced. For most fixed enclosures, the air should move at least 100 feet per minute across the entrance to the enclosure.

A third type of local exhaust ventilation system is the down-draft bench or table. The soldering or brazing is performed on a bench or table which has an open grid as the work surface. Air is drawn downward through the grid, into the duct work, and then exhausted.

SIC 39—Miscellaneous Manufacturing

In the manufacturing of hard surface floor coverings (SIC 3996), processes include pre-weighing and blending raw materials, followed by mixing and gelling of the composition in internal batch mixers of the Banbury type or by continuous mixing operations carried out in mixers of the extruder type.

Potential worker exposures may result from dusts of the raw materials as they are handled (automatically or manually) prior to and during charging of the mixer. Fumes and dusts can emanate from leaks on the mixer and from hot, freshly mixed material as it is discharged.

The types of exposures depend on the substances used, though dusts of any type can constitute exposure to nuisance particulates. Applicable exposure controls include local exhaust ventilation at the mixer doors and over conveyor transfer points. The use of good working practices is extremely effective in controlling exposures during the opening of the mixers and the pouring of materials.

SICs 40, 45, 47—Transportation

Cleaning and coating operations are conducted in rail (SIC 40), and air transport industries (SIC 45), as well as in transportation services (SIC 47). These operations require the application of cleaning agents and/or the sandblasting of particles prior to the application of paints or coating. Spraying processes are required for the application of both the cleaning agents and the paints and coatings.

Rail car applications, for example, are generally performed within a large facility, part of which is established as a spray room. The cars are rolled into an enclosed spray area. In manual spray painting rooms, the operator is required to enter and move about the enclosure during spraying. Automatic spray rooms (or booths) are similar but the pressurized spray guns are automatically operated.

Three major spray techniques are used to apply cleaning agents, coatings or paints. These are: Compressed air spraying (low pressure spraying); airless spraying (high-pressure spraying); and electrostatic spraying. The compressed air spray gun atomizes a stream of liquid by impaction with a jet of air. Atomization may take place inside or external to the gun. The air stream and paint droplets intersect the prepared surface. The airless spray gun atomizes the liquid by forcing it through a small orifice under high pressure. The resulting particulate cloud is impelled by the pressure-created momentum toward the surface. Electrostatic spray equipment is based upon the attractive force between two oppositely charged objects. The liquid is atomized by compressed air, airless, or electrostatic techniques. The particles are given either positive or negative charge and the conductive surface to be sprayed is grounded. In general, electrostatic spray techniques result in the lowest exposure levels, followed by airless and then compressed air spraying.

In enclosed spray rooms, particulates enter the operator's breathing zone due to backspray. Exhaust ventilation to control exposure can be designed using down draft or a multiple sidedraft system. Worker positioning in relation to the spray plume is also critical in minimizing exposures. These include minimized line pressure, changing and cleaning of filter banks, enclosure integrity and ventilation maintenance. Personal protective equipment is also generally worn to insure the worker protection.

SIC 49—Electric, Gas and Sanitary Services

Coal-fed power plants present the potential for exposure to coal dust as well as a number of other substances. Coal dust exposure potentially occur in the area where coal is fed into the furnaces. The coal is generally fed into large hoppers off conveyors. Conveyors are filled by front-end loaders from the coal storage area. The operators of the front-end loaders are protected from coal dust exposure with the use of closed, air-conditioned cabs which provide purified breathing air.

SICs 50 and 51—Wholesale Trade

Some firms in this classification receive liquid chemicals in bulk quantities from a tank truck, store them and then redistribute them in smaller containers.

Solvents, for example, emit considerable vapor when poured from one container to another or when a container is being filled, displacing the air in it. Pouring and filling operations are often enclosed to minimize vapor losses (this helps to reduce product loss as well as prevent exposures). In addition, secondary vapor recovery is often incorporated, whereby vapors emitted at the transfer points are captured and returned through a separate circuit to the storage tank from which the volatile liquid is being removed.

SIC 72—Personal Services

To control dry cleaning emissions (SICs 7216, 7217), louvered wall fans and grilled ducts were installed to provide ventilation. Ceiling exhaust fans provided general ventilation. Natural ventilation was provided by through doors in the production area and by louvered panels along walls in the plant. Forced ventilation was provided by ceiling mounted exhaust fans and evaporative coolers. A local exhaust system with a standard single floor pickup exhausted air through a carbon absorption unit to the outside. Gaskets in machinery doors and ductwork needed routine maintenance to prevent deterioration. Various cleaning machines, pressure filter extractors and dryers were used. Dryers and drying cabinets were provided with local exhaust ventilation.

SIC 73—Business Services

Blueprinting and photocopying firms (7332) control ammonia fumes from blueprint duplication machines through use of local exhaust ventilation. The exhaust system is often built into large, high volume machines. Improvements in work practices control exposures during transfer.

SIC 55, 75—Automotive Repair Shops, Dealers

In automobile engine reconditioning lines (SIC 7538), exhaust fans and flexible ducts which extend directly over the engines were installed in order to control carbon monoxide exposure.

SIC 76—Miscelleanous Repair Services

Control techniques for welding fumes (SIC 7692) included a "smoke exhaust" welding gun which captures and removes fumes. These guns have some limitations and are applicable to continuous or semicontinuous flux core or metal inert gas welding operations. Crossdraft airflow was also suggested. The use of a portable fan is not recommended.

SIC 80—Health Services

Many medical and dental practitioners perform surgery in outpatient clinics and private offices outfitted for the procedure. Air contamination in an operating room may consist of waste anesthetic, the propellants of different sprays, scrubbing agents, cleansing agents, methylmethacrylate (released from surgical cement) and the possible decomposition products of the volatile or gaseous agents. The magnitude of gas flow, type of flow circuit and scavenging of waste gases significantly influence the levels of waste gases in the room air. Exposures are usually controlled by general dilution ventilation. Some clinics and offices, which are specifically

designed for surgical use, may have local exhaust systems installed.

Personal Protective Equipment

In the operations and processes included in Supplement 2 reductions in exposure limits can be achieved through engineering controls and work practice notifications. However, certain generic work activities are more problematical and may require the use of personal protection equipment.

Examples of these are:

Maintenance Activities in all SIC codes. It may be more difficult to control exposures of plant maintenance personnel by using engineering controls except for activities taking place in the maintenance shops. These maintenance employees may work in areas not normally accessed by production workers or may work under conditions which normal production workers do not. Respiratory protection is sometimes the appropriate control technology.

Painting and Coating Activities in all SIC codes. Although spray painting operations are performed in exhausted paint booths, the painting of many larger non-production items, such as construction equipment and heavy machinery, requires that the operator enter the booth. The booth is then primarily a control to prevent migration of the paint spray into other areas of the plant. In these circumstances it is usually necessary to provide respiratory protection to the workers painting.

Exterminating. Exposure of pesticide applicators cannot be controlled through engineering controls because their work does not take place in a fixed place of employment, but rather at a customer's facility. Personal protective equipment and/or work practice controls would therefore be required. EPA has jurisdiction in most situations.

Welding In certain situations, such as in confined spaces, or where the welder must be positioned directly above the fume plume, welders cannot be sufficiently protected by local exhaust ventilation. Personal protective equipment would be required.

In addition to the above examples, a number of the substances included in this rulemaking carry the designation "Skin." This refers to potential exposure through the skin. Table V–1 presents a list of chemicals for which skin protection would be required. Employees exposed to substances with the "Skin" notation would be required to wear protective equipment, including gloves, long sleeved shirts and coveralls.

Products are commercially available to adequately protect workers from dermal exposure. In some cases the permeability of currently used materials may be inadequate and firms will have to change the specific product now used to one offering greater protection.

TABLE V–1.—INDUSTRIES AND PROCESSES WHERE SKIN PROTECTION HAS BEEN ADDED

SIC No.	Chemical name	Process name
20	CAPTAFOL (DIFOLATAN)	Food Storage and Preservation
	CARBON DISULFIDE	Food Storage and Preservation
24	2-HEXANONE	Coating and Finishing
25	N-BUTYL ALCOHOL	Coating
	2-HEXANONE	Coating and Finishing
	STYRENE (PHENYLETHYLENE)	Lay-up, Molding
26	METHYL ALCOHOL	Chemical Recovery
27	CYCLOHEXANONE	Plate cleaning
	FURFURYL ALCOHOL	Plate cleaning
	2-HEXANONE	Plate cleaning
	HYDROGEN CYANIDE	Plate making/Engraving
	METHYL ALCOHOL	Plate cleaning
28	N-BUTYL ALCOHOL	Blending, Packaging
	CARBON DISULFIDE	Blending, Packaging
	DIAZINON	Blending, Packaging
	DISULFOTON	Blending, Packaging
	2-HEXANONE	Blending, Packaging
	METHYL ALCOHOL	Blending, Packaging
	METHYL PARATHION	Blending, Packaging
	STYRENE (PHENYLETHYLENE)	Lay-up, Molding
30	N-BUTYL ALCOHOL	Finishing, Trimming, Painting
	CARBON DISULFIDE	Compounding, Mixing and Blending
	2-HEXANONE	Solvent Mixing, Molding, Mold cleaning
	STYRENE (PHENYLETHYLENE)	Lamination, Foam Processing
31	CARBON TETRACHLORIDE	Finishing/Degrading
	DDT	Defestation/Disinfestion
	2-HEXANONE	Finishing/Degreasing
	HYDROGEN CYANIDE	Beamhouse
	METHYL ALCOHOL	Finishing/Degreasing
	TRIORTHOCRESYL PHOSPHATE	Finishing/Degreasing
32	CARBON DISULFIDE	Batch preparation
	CARBON TETRACHLORIDE	Batch preparation
	METHYL ALCOHOL	Batch preparation
	STYRENE (PHENYLETHYLENE)	Packaging
	THALLIUM (SOLUBLE)	Batch preparation
	TIN	Float Process
33	HYDROGEN CYANIDE	Coremaking
34	N-BUTYL ALCOHOL	Coating/Painting
	CARBON TETRACHLORIDE	Degreasing/Cleaning
	STYRENE (PHENYLETHYLENE)	Coating/Packing
35	N-BUTYL ALCOHOL	Coating/Painting
	HYDROGEN CYANIDE	Soldering/Brazing
	1,2-PROPYLENE GLYCOL DINITRATE	Coating
	STYRENE (PHENYLETHYLENE)	Packing/Coating
36	N-BUTYL ALCOHOL	Coating/Painting
	HYDROGEN CYANIDE	Soldering/Brazing

TABLE V-1.—INDUSTRIES AND PROCESSES WHERE SKIN PROTECTION HAS BEEN ADDED—Continued

SIC No.	Chemical name	Process name
	MERCURY (VAPOR)	Soldering/Brazing
	METHYL ALCOHOL	Cleaning
	STYRENE (PHENYLETHYLENE)	Packing/Coating
37	2-HEXANONE	Degreasing
	STYRENE (PHENYLETHYLENE)	Painting, Coating
38	2-HEXANONE	Injection Molding, Foaming
	MERCURY (VAPOR)	Handling of measurement liquids
		Preparation of Special Tubes
		Assembling
	METHYL ALCOHOL	Blending/Packaging
39	N-BUTYL ALCOHOL	Painting, Coating
	2-HEXANONE	Degreasing, Coating
	STYRENE (PHENYLETHYLENE)	Painting, Coating
40	2-HEXANONE	Coating
44	2-HEXANONE	Coating
45	N-BUTYL ALCOHOL	Cleaning/Spraying
	2-HEXANONE	Cleaning/Spraying
	METHYL ALCOHOL	Cleaning/Spraying
47	2-HEXANONE	Coating/Spraying
50	2-HEXANONE	Materials Recev./Packaging
	STYRENE (PHENYLETHYLENE)	Materials Recev./Packaging
51	CARBON TETRACHLORIDE	Materials Recev./Packaging
55	N-BUTYL ALCOHOL	Painting/Coating
72	MERCURY (VAPOR)	Embalming
	METHYL ALCOHOL	Embalming
73	DIAZINON	Exterminating
	DIOXATHION	Exterminating
	PHENOTHIAZINE	Exterminating
75	N-BUTYL ALCOHOL	Painting/Coating
80	N-BUTYL ALCOHOL	Disinfectant and solvent use
	MERCURY (VAPOR)	Preparation of amalgams

VI. Costs of Compliance

Costs of compliance result from the purchase, installation, operation and maintenance of equipment to maintain workers' exposures at or below the levels specified in the proposed standard. Costs are related to the engineering controls needed for specific processes which involve the use of hazardous substances. Existing data sources and expert judgement were used to sort the approximately 430 substances being regulated, by industry and by process within industry segments. Given the large number of substances being regulated, a process orientation rather than a chemical specific focus, was recommended, since prescribed engineering controls can address worker exposure problems to several chemicals, involving the same general process, simultaneously. The approach has proven to be efficient analytically and reduces the problem of double counting the costs of similar or the same engineering controls for separate chemicals involved in the same process or operation.

OSHA has a large amount of exposure data in its Integrated Management Information System and from NIOSH and other sources. But to improve the available information on the use of substances, OSHA decided to engage in a nationwide field survey of affected establishments. This survey, involving over 5,300 establishments in both manufacturing and nonmanufacturing sectors, has provided valuable information on chemical usage by industry process and potential worker exposures to these chemicals. Supplement 1 contains a description of the sample survey design and a statistical evaluation of the data collected.

In order to maximize the efficiency of this nationwide sample survey and limit the number of required sample observations per SIC category, a considerable effort was made to verify chemical by industry usage from existing data sources and to make best estimates of where likely or potential workers exposure problems (and consequently engineering costs) existed. For the purposes of the statistical survey being conducted, the larger the suspected potential exposure/cost problem in a particular industry sector, the more important it was to insure a large enough sample of firms in that sector so as to reduce the standard error of the cost estimates.

The following sections of this chapter outline the methodology adopted to identify:

- Chemicals by their industrial usage and employee exposures
- Processes involving known or suspected schemical exposures and control costs
- Industry costs for the controls needed to reduce industry exposure levels

Linking Hazardous Substances by Industry Use and Employee Exposure

Figure VI-1 presents a flow chart of the methodology used for identifying chemicals by industry use and employee exposure. The first step in the methodology was an analysis of the chemicals for which OSHA proposes new exposure limits. The 1982 NIOSH National Occupational Exposure Survey (NOES) and the OSHA IMIS data files was searched to determine the potential for worker exposure to each of the chemicals on the proposed list. The objective of this analysis was to create a subset of chemicals which are known to be present in specific industries at exposure levels above the proposed limits. These chemicals would then be considered to generate potential compliance costs within a specific industry sector.

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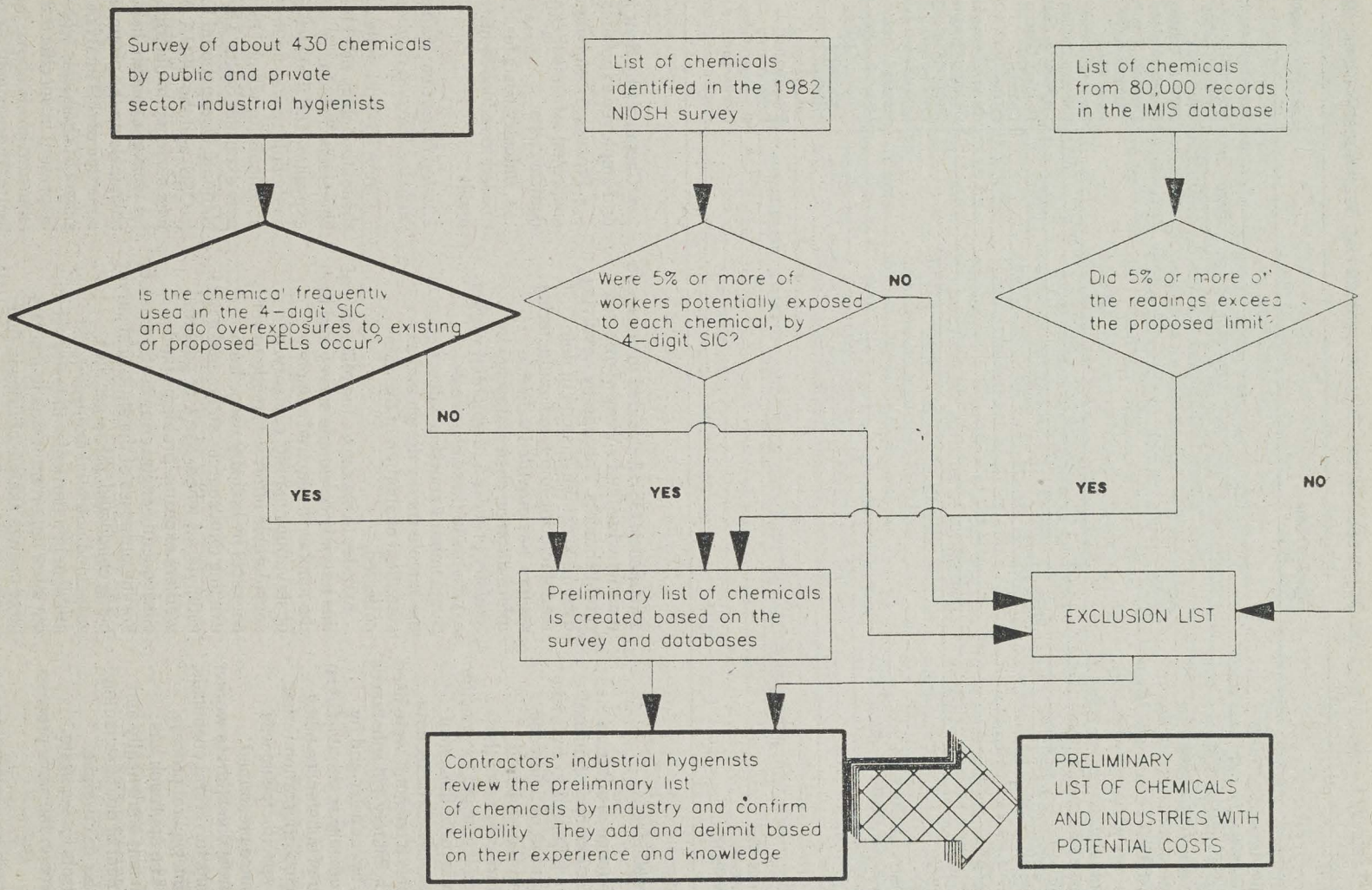
FIGURE VI-1

METHODOLOGY FOR IDENTIFYING CHEMICALS WITH POTENTIAL COST IN EACH 4-DIGIT SIC



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The 1982 NIOSH National Occupational Exposure Survey (NOES) data (supplemented by results from NIOSH's 1972 survey) provided an estimate of the number of workers potentially exposed to a specific chemical in a four-digit SIC. OSHA divided this estimate by the total number of employees in an industry segment to get a percentage of workers potentially exposed to that chemical. If 5 percent or more of the workers were potentially exposed, that chemical was considered to present a potential cost within the four-digit SIC. For example, in SIC 3011, Tires and Innertubes Manufacturing, 1,532 persons were potentially at risk of exposure to n-hexane at the sample of plants included in the NOES database. This represented 21.9 percent of all workers sampled in the four digit industry sector and this chemical would have a potential cost impact depending upon current exposure levels.

From the OSHA IMIS data, the severity of exposure within a four-digit SIC was estimated. OSHA compared the total number of monitored readings for each chemical with the number of readings which exceeded the proposed limits and calculated the percentage of all sample monitor readings which were above the proposed limits. If there were no readings which exceeded the proposed limits, the chemical was not considered to have a compliance cost within the four digit SIC. If 5 percent or more of the readings exceeded the proposed limits, then the chemical was identified as having a potential compliance cost within the four-digit SIC. For example, in SIC 2641, Paper Coating and Glazing, 22 samples were taken for n-hexane. Thirteen of these, or 59 percent, were above the proposed standard for n-hexane. This chemical, therefore, was believed to have a potential cost impact and questions regarding its use were included in the field survey. Chemicals with non-compliance percentages between zero and 5 percent were evaluated individually by industrial hygienists to determine whether or not specific survey questions needed to be asked about their industrial usage.

In addition to the IMIS and NOES databases, a survey of about one dozen industrial hygienists was conducted (under personal services contracts). The purpose of this survey was to identify any additional hazardous substances or industry sectors not identified in the IMIS or NOES databases with potential exposure problems at new recommended levels. For example, in SIC 2891, Adhesives and Sealants Manufacturing, the surveyed industrial hygienists reported that n-hexane overexposures could exist under the proposed standard. (Overexposures in SIC 2891 were not previously identified in the IMIS or NOES databases.)

The information from all sources was combined to compile a preliminary list of substances with potential compliance costs by four-digit SIC classification. To further refine the list of chemicals, a second group of six industrial hygienists, using personal industry knowledge and the information gathered from the survey of the initial group of industrial hygienists, reviewed once again the chemicals which appeared in the NOES and IMIS datasets. They also made chemicals by industry use linkages when particular chemicals were known to be present in certain SICs, but had not been identified in the NOES and IMIS data bases.

Upon completion of the two-tier industrial hygienist review, a list of chemicals believed to be present at exposure levels above the proposed standard, within specific four-digit SIC industry sectors was finalized. This list identified those industry segments with potential compliance costs needed to comply with the proposed standards. This chemical by industry listing is presented in Supplement 2 of this Appendix.

Industrial Processes and Control Costs

The number of industrial processes, exposure levels and exposure controls in place varies greatly within industry segments. In order to efficiently structure the statistical sample of surveyed firms, it was necessary to make a best estimate of which industry segments were likely to experience compliance costs. As noted above, the survey was designed to limit the standard error for potential high cost industry sectors. To concentrate the survey on the potential high cost sectors, a process orientation was adopted which supplemented and refined the chemical use information. Industry sectors with very low incidence of processes and chemicals with low potential exposure levels (and consequently low potential compliance costs) were included in the sample survey.

A team of engineers and industrial hygienists analyzed each four digit SIC to assess the processes in which worker exposure to listed chemicals occur. Examples of industrial processes included grinding, mixing, spraying, degreasing, separation, bagging and loading. A list of potential cost chemicals and related processes was then developed to identify potentially high impact (cost) industries. Supplement 2 presents the chemical by process listing for each four digit SIC covered by the sample survey. In general, an industry segment with a relatively large number of processes using chemicals with suspected high exposure levels was sampled at the three digit industry level. Industries with fewer processes and low chemical exposures were sampled at the two digit level. (See Supplement 1 for a more detailed explanation of the survey design.)

Each of the over 5,300 respondents in the survey was asked to verify the chemicals used, manufactured or generated by process within the establishment. Thus, chemicals were linked to specific processes, process controls and workers exposed at the process in the surveyed industries. Control methods and costs were then assigned for each process where employee exposures would exceed the proposed PELs.

Controls were assigned to protect workers exposed to all chemicals in total at a process. The controls were designed and costed to lower exposure to the chemical(s) with the greatest change in the permissible exposure limit (PEL). It was the judgment of the experts involved that by assigning controls for the "major" chemicals, exposures for all other chemicals would be controlled. Chemicals and/or processes not included in the proposed standard (e.g., those covered by separate 6(b) rulemaking) were excluded from the survey. Examples of chemicals not included in the survey are asbestos, formaldehyde and benzene.

Survey information collected from each respondent include:

- Type of processes at the establishment;
- Type and amount of chemical used, manufactured, or generated in each process;
- Number of work stations and workers related to the process;
- Potential chemical exposure above the proposed standards (monitoring data, recorded overexposures) at the process;
- Process location (indoors/outdoors), and configuration (size, full enclosure, partial enclosure);
- Ventilation or other controls in place; and
- Economic and other characteristics of the plant.

A computer algorithm was developed to assess survey data to determine if potential worker overexposure and therefore compliance costs occur for each process at an establishment. Figure

VI–2 presents a general diagram of the computer logic adopted for use in the survey. The logic assesses potential overexposures on the basis of: Actual reported monitoring data; statements that overexposures occur; and the particular process location, configuration, type and amount of chemical use and existing controls in place.

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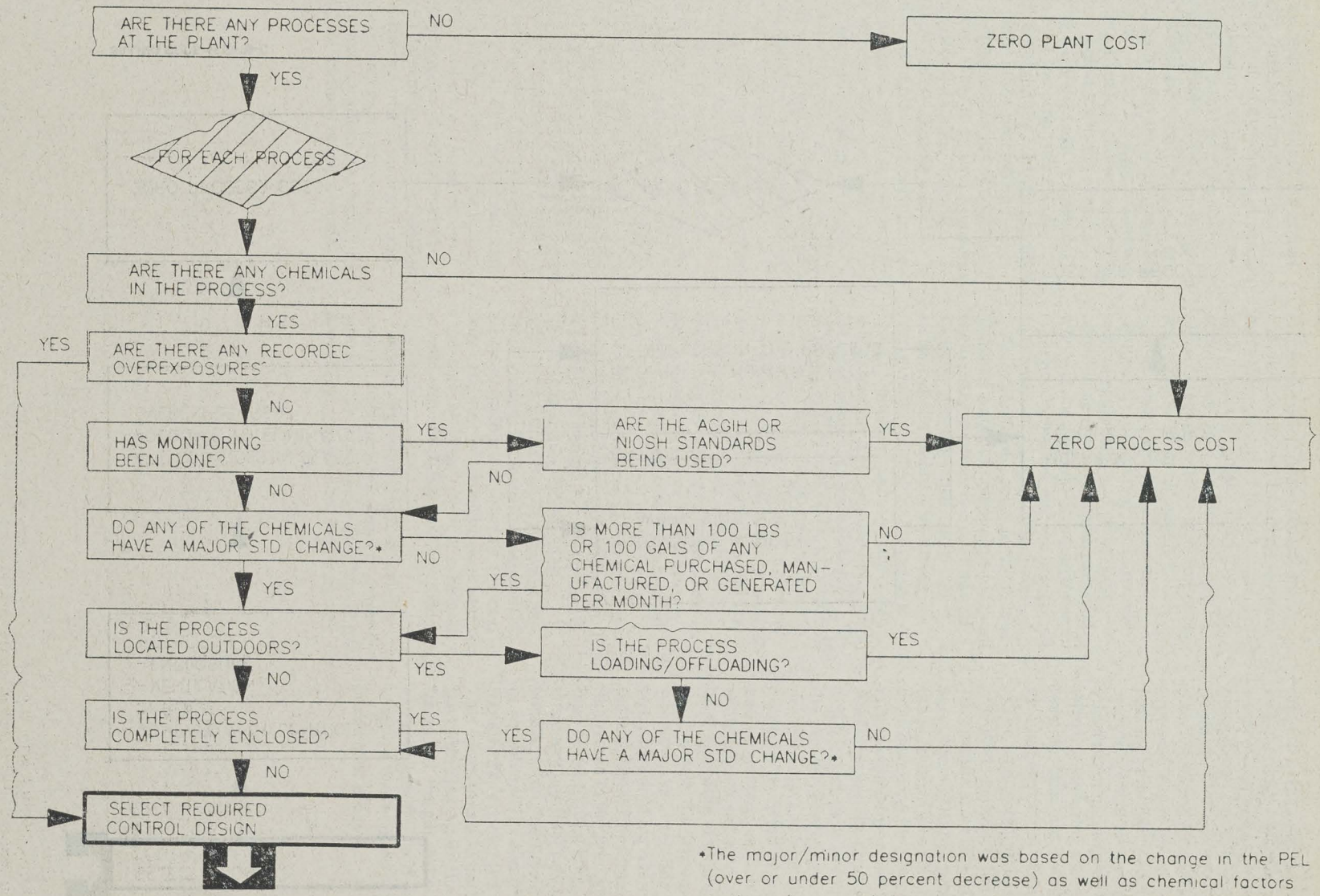
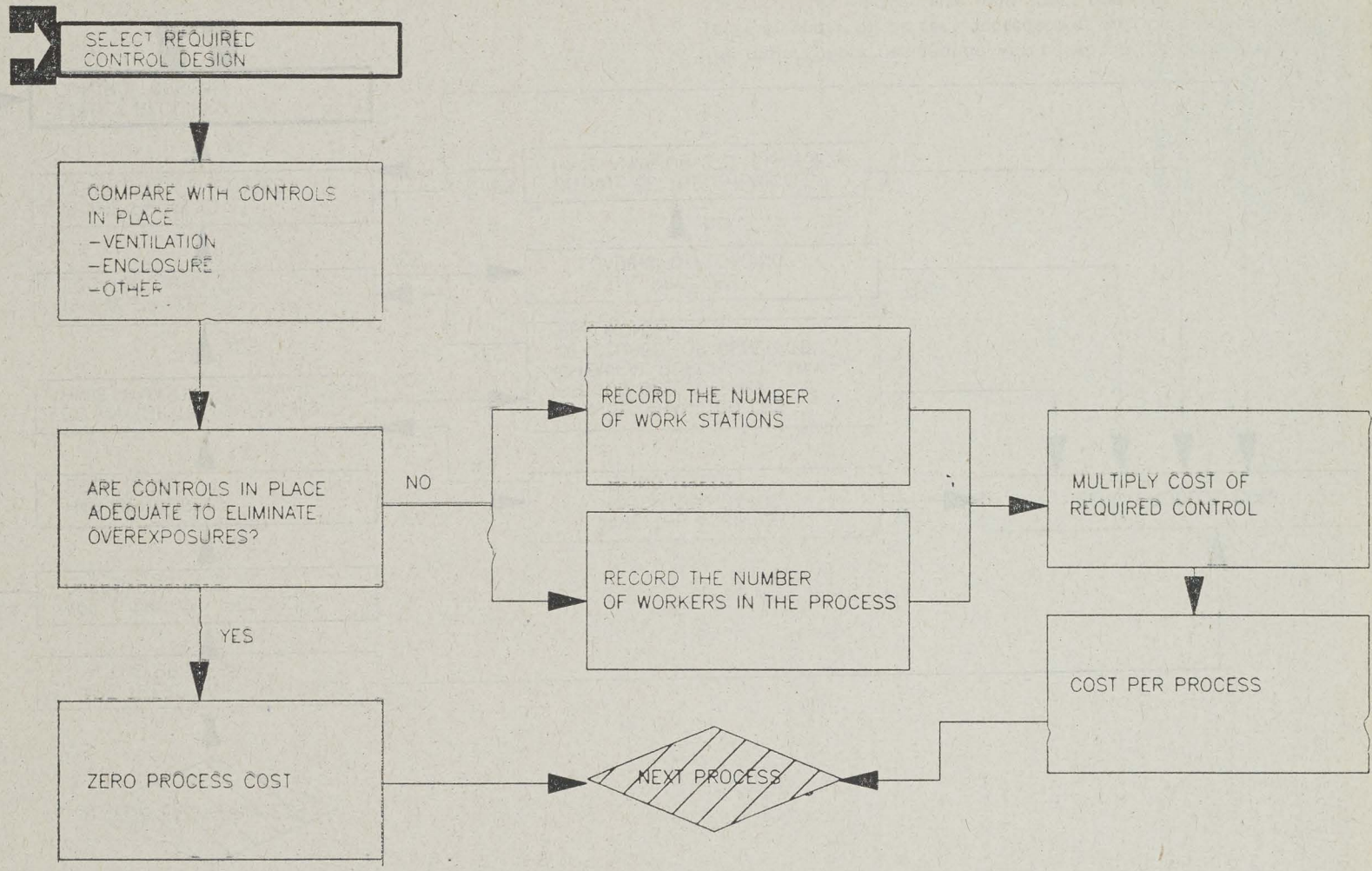
FIGURE VI-2
COMPUTER LOGIC FOR DERIVING INDUSTRY COST OF COMPLIANCE



*The major/minor designation was based on the change in the PEL (over or under 50 percent decrease) as well as chemical factors such as form, particle size, and vapor pressure

FIGURE VI-2 (CONTINUED)
COMPUTER LOGIC FOR DERIVING INDUSTRY COST OF COMPLIANCE



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When a respondent provided actual monitoring data for a process that indicated chemical exposures above the proposed standard, compliance costs were assigned to that process on the basis of prescribed controls for the given process. Where no monitoring data or reports of overexposure were available, the computer algorithm logic examined process and chemical characteristics to determine if workers at the process were potentially exposed to chemicals at levels over the proposed standard. The logic assessed the controls reported to be in place at the process and compared them with a list of controls throught necessary to control exposures in that process within the industry. When the requried controls were reported to be in place, no compliance cost was assigned. When the required controls were not reported to be in place, a compliance cost per work station was assigned.

The computer algorithm determined that some processes within plants had no potential overexposures and consequently no compliance costs. Zero compliance costs resulted where no proceses and/or chemicals were reported to occur at the establishment. Zero compliance costs also resulted when the respondent had monitored a process using ACGIH or NIOSH standards and found no overexposures. Where only very small quantities of chemicals, none of which were designed as "major" proposed exposure limit changes were present in a process, no overexposure was determined and zero compliance costs assigned. The major/minor designation was based on the proposed change in the PEL (over or under a 50 percent decrease) as well as chemical characteristics such as form, particle size, and vapor pressure.

Process configurations and location also were indications of compliance. Processes which were reported as completely enclosed with no worker entry were assumed to be in complaince with the proposed standard (have no compliance cost). Outdoor loading/offloading processes or other outdoor processes with no chemicals with "major" proposed exposure limit changes were assumed not to require control equipment and costs. Zero compliance costs were also assessed where processes which required control equipment reported that the prescribed equipment was currently in place.

An example of a process which was assigned a cost of compliance to install engineering controls is a coating and spraying process in SIC 2511, Wood Household Furniture. The survey respondent reported that toluene, n-butyl alcohol and xylene were used in this operation. The proposed standard for toluene reduces the existing PEL by 50 percent. This reduction is considered to require concerted exposure control and is considered a "major" proposed exposure limit change. Because workers were involved in the process and the process was reported to be neither located outdoors nor fully enclosed, controls were assumed necessary to insure compliance with the proposed standard. The control required for controlling exposures at this process was determined as local ventilation. The type of local ventilation prescribed in this case is a spray booth at an estimated cost of $3,070 annually per work station. Because the respondent reported no local ventilation, the cost was assigned for the eight work stations reported, resulting in a total estimated annual cost of $24,560 for this process at this site.

Expert engineering and industrial hygiene judgment was used to determine which of the various controls would be necessary to control for exposures by process in the affected industries. Engineering controls identified included exhaust ventilation (local and general); process enclosure; and process change. Some or all of these will be required by affected plants for compliance with the proposed exposure levels. In addition, personal protective equipment such as respirators will be needed for intermittent maintenance activities where engineering controls are not feasible. Skin and eye protection may also be required in certain situations.

The engineers and industrial hygienists classified the approximately 180 specific processes identified in the survey into about 20 process groups for the purpose of assessing required controls and estimating costs. These process groupings were based on similarities in the processes and levels and types of exposures resulting from the process. Factors used to group processes include the chemicals generally invovled in the process, type and usual configuration of the equipment, usual work station design, level and route of exposure, industry group where the process exists and worker tasks in relation to the equipment and exposure route. The process similarities translated into likenesses in required controls such as type of ventilation hood, booth or enclosure, air flow rates, duct configuration and type and size of filters or scrubbers. The compliance costs framework is presented in Figure VI–3. This figure presents the process groups, the industries where the processes were identified, the general classification of controls specified and work station unit costs for the required controls assigned.

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FIGURE VI-3.
COMPLIANCE COST FRAMEWORK AND WORK STATION UNIT COSTS

PROCESS GROUP (1)	INDUSTRY GROUP (SICs)	REQUIRED CONTROL CONFIGURATION (2)	ANNUAL COST PER WORK STATION
Leather Processing, major	31	Local Ventilation	$ 2,510
Leather Processing, minor	31	General Ventilation	$ 720
Electrical & Electronics Manufacture	36	Local Ventilation	$ 2,520
Printing Processes, minor	27, 38, 73, 80	Local Ventilation	$ 1,240
Printing Processes, major	27, 39	Local Ventilation	$ 3,510
Glass Processing, major	32	Local Ventilation	$ 3,890
Glass Processing, minor	32, 36	Enclosure	$ 90
Resource Recovery & Water Treatment, major	28, 29, 33, 49	Enclosure & Local Ventilation	$ 21,900
Resource Recovery & Water Treatment, major	26	Enclosure & Local Ventilation	$ 14,000
Resource Recovery & Water Treatment, minor	26, 29, 49, 50	Enclosure & Local Ventilation	$ 14,000
Foundry Operations, major	33	Local Ventilation	$ 2,520
Foundry operations, minor	33, 39	Local Ventilation	$ 1,820
Grinding, Blasting, & Metalworking, major	25, 33, 36, 39	Local Ventilation	$ 7,200
Metalworking & Welding	All SICs	Local Ventilation	$ 1,140
Coke Ovens	29 (3)	Enclosures, Local Ventilation & Air Purifiers	$150,000

1 The "major" and "minor" designation of process groups refers to the level of the exposure change and consequently the extent of required control configuration costs within a given control and process configuration. For example, leather processing is the general process group and processes within that group are classified based on whether the employee exposure control requires major or minor control costs.

2 The specific required control configuration cost was estimated including all necessary components, such as ductwork, fans, hoods, baghouses, etc.

3 Coke ovens in SIC 33 are not included as they are covered by OSHA's Coke Oven Standard

FIGURE VI-3 (Cont.)
COMPLIANCE COST FRAMEWORK AND WORK STATION UNIT COSTS

PROCESS GROUP	INDUSTRY GROUP (SICs)	REQUIRED CONTROL CONFIGURATION	ANNUAL COST PER WORK STATION
Paper Manufacturing, major	26, 30, 39	Ventilation & Air Purification in Control Rooms	$ 2,900
Paper Manufacturing, minor	All SICs	Local Ventilation	$ 180
High Temperature Drying	All SICs	Local Ventilation	$ 4,740
Layup	3632,3715,3732 3792,3995	Local Ventilation	$ 16,550
Coating, Spraying, & Adhesive Application	All SICs	Local Ventilation	$ 3,070
Chemical Handling & Formulation	All SICs	Local Ventilation	$ 1,760
Material Handling & Inspection, major	All SICs	Local Ventilation & Partial Enclosure	$ 1,120
Material Handling & Inspection, minor	All SICs	General Ventilation	$ 560
Cleaning & General Solvent Use, major	All SICs	Local Ventilation	$ 1,500
Cleaning & General Solvent Use, minor	All SICs	Local Ventilation	$ 710
Waste Collection & Transport	4953, 5093	Respirators (4)	$520 per worker
Painting, Maintenance	All SICs	Respirators (4)	$520 per worker
Welding, Maintenance	All SICs	Respirators (4)	$520 per worker
Zero Cost Processes:			
Laundering	72		
Embalming	72		
Permanents	72		
Anesthesia	80		

4 Use of respirators is considered the only feasible control for these processes due to their intermittent performance and because they are generally not performed at a fixed site.

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The development of unit costs for each control configuration required the development of "model" control designs. Model configurations were selected to provide exposure control at "typical" process/work stations with the specified process group. This costing approach ("model" configurations for "typical" work stations) required the differentiation of some process groupings as major or minor. The major/minor differentiation addresses the expected level of control required.

The control designs were developed by engineers based on their experience in industry and extensive secondary research on operations and exposure situations in each industry sector. This research included an examination of industry and industrial hygiene journals, engineering process reports, and texts. Included in the detailed cost calculations for the control configurations were costs for enclosure construction, baffles, fans, ductwork, filters, scrubbers, baghouses, and all other equipment required for exposure control. All of the costs were developed on a per work station basis so that an average size did not need to be estimated for the process. Investment costs were assigned to each control design on the basis of engineering handbooks and supplier catalogs. Investment costs were annualized over the projected life of equipment (10 years) using a 10 percent cost of capital and adding annual operating and maintenance costs estimated at 10 percent of the capital cost. Respirator costs for use by maintenance workers for intermittent activities were considered annual costs and include the respirator purchase as well as an estimated year's work of cartridges and canisters.

Process control costs were summed per establishment and any maintenance worker respirator requirements cost included. A total annualized capital cost and annual operating cost was developed for each establishment. Costs for the survey establishment were then weighted (by SIC and size) to represent compliance costs for the universe of affected plants.

Projected Impact by Industry Sector

Following the methodology described in the preceding section of this chapter, annual compliance costs were estimated by industry sector. The costs presented are based on the data collected from the over 5,300 survey respondents. (For industries not included in the survey, expert judgment and secondary sources were used for estimating costs.) A small percentage of respondents (less than 5 percent) actually provided monitoring data during the survey. However, based on survey data it was determined that about 86.1 percent of all establishments in the surveyed industries have no exposures in excess of the proposed standard and will not incur any costs to comply with the proposed standard. Only 11.1 percent of all establishments in the surveyed industries will incur costs to provide engineering controls for processes within the plant. However, over 20 percent of the firms which reported using the chemicals being regulated, will incur some engineering control costs. About 2.8 percent of the establishments in the surveyed industries will be required to provide personal protective equipment only for maintenance workers whose intermittent operations cannot be controlled with engineering controls.

Table VI–I presents the total annualized capital and annual operating cost for compliance with the proposed standard by industry. As shown, annual compliance costs are estimated to total $927.8 million. These costs represent an estimate of compliance costs for large and small plants in the universe of establishments to be affected by the proposed standard. Industries with some anticipated cost impact are identified below in order of the total annual compliance costs estimated.

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TABLE VI-1

ANNUAL OPERATING AND ANNUALIZED CAPITAL COST OF COMPLIANCE BY INDUSTRIAL SECTOR (a)

SIC (b)	SIC DESCRIPTION	LARGE PLANTS	SMALL PLANTS	ANNUAL COST
20	FOOD PROD. (c)	$21,704,100	$11,789,000	$33,493,100
21	TOBACCO (c)	$19,700	$0	$19,700
22	TEXT. MILL (c)	$23,308,400	$6,170,000	$29,478,400
23	APPAREL PROD. (c)	$23,604,300	$8,139,900	$31,744,200
24	LUMBER & WOOD	$20,534,800	$175,551,300	$196,086,100
25	FURNITURE	$7,915,300	$6,805,300	$14,720,600
26	PAPER PROD.	$30,966,900	$290,800	$31,257,700
27	PRINTING & PUB.	$15,263,300	$60,425,000	$75,688,300
28	CHEMICAL PROD.	$31,745,500	$8,412,100	$40,157,600
29	PETRO. REFINING	$6,800,700	$414,600	$7,215,300
30	RUBBER & PLASTICS	$53,256,200	$22,225,900	$75,482,100
31	LEATHER PROD.	$1,464,400	$1,115,100 (c)	$2,579,500
32	STONE & CLAY	$15,079,300	$7,463,300	$22,542,600
33	PRIM. METAL	$34,721,200	$5,612,400	$40,333,600
34	FAB. METALS	$50,927,100	$5,010,500	$55,937,600
35	MACHINERY	$43,986,300	$13,754,100	$57,740,400
36	ELEC. MACH.	$24,210,900	$7,570,500	$31,781,400
37	TRANS. EQUIP.	$20,884,600	$26,214,500 (c)	$47,099,100
38	INSTRUMENTS	$10,257,300	$3,207,400	$13,464,700
39	MISC. MANUF.	$13,861,200	$4,334,300	$18,195,500
40	R.R. TRANS.	$532,200	$0	$532,200
45	AIR TRANS.	$1,828,900	$0	$1,828,900
47	TRANS. SERV.	$1,853,100	$0	$1,853,100
49	ELEC. GAS. SAN.	$19,373,900	$3,314,500	$22,688,400
50	WHOLESALE TRADE	$1,416,300	$2,638,300	$4,054,600
51	WHOLESALE, NON-DUR	$3,094,200	$5,764,000	$8,858,200
55	AUTO DEALERS (c)	$9,862,500	$2,092,200	$11,954,700
72	PERSONAL SRV. (c)	$15,648,500	$15,639,300	$31,287,800
73	BUSINESS SRV. (c)	$3,701,700	$5,252,100	$8,953,800
75	AUTO REPAIR (c)	$466,800	$1,044,100	$1,510,900
76	MISC. REPAIR SRV.	$669,500	$4,179,000	$4,848,500
80	HEALTH SERV. (c)	$2,413,000	$2,026,400	$4,439,400
TOTAL		$511,372,100	$416,455,900	$927,828,000

Source: U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.

(a) Costs were calculated by annualizing the capital cost over the projected life of the equipment (10 years) using a 10 percent cost of capital and adding an annual operating and maintenance cost estimated at 10 percent of the capital cost.

(b) Industry sectors not identified in this table include industries with no major cost impact expected, the construction industry, which will be the subject of a separate regulatory analysis, and industries such as mining, over which OSHA has no jurisdiction.

(c) Costs in these sectors were based on expert judgement and secondary data collection. Survey data for SICs 55, 72, 73, 75 and 80 was insufficient to estimate compliance costs.

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Manufacturing

Lumber and Wood Products (SIC 24). The annual costs of compliance in the lumber and wood products industry are estimated to total $196.1 million. The compliance costs for this sector are heavily weighted by the cost of controls required to lower exposures to the proposed limits for wood dust. The survey indicated that sanding and other "dusty" processes would require controls to lower wood dust exposure. The large number of establishments that must engineer ventilation systems for wood dust control account for the substantial proportion of compliance costs to be incurred by small establishments in this sector.

In addition to wood dust, controls for exposures to solvents and other chemicals in coating processes and exposures to wood preserving chemicals are estimated to result in compliance costs in SIC 24. Overall, about a fourth of all establishments is SIC 24 are estimated to incur compliance costs.

Printing and Allied Industries (SIC 27). Compliance costs in the printing industry sectors (an estimated $75.7 million) would result from ventilation requirements to control exposures to cleaning solvents and ink spray generated within the printing process. A very large number of small establishments are involved in printing and over 4,600 of them would be affected by the proposed standards. The survey indicated that a large number of small establishments currently lack exposure controls and provision of these controls account for the high control costs in this sector.

Rubber and Miscellaneous Plastics Products (SIC 30). Annual costs of compliance in this industry sector are estimated to total about $75.5 million. Controls were required for processes such as molding, and vulcanizing. Worker exposure to chemical vapors require the addition of local ventilation to many processes. The miscellaneous plastic products industry (SIC 3079) accounts for over 20 percent of the annual costs in this sector. The costs in SIC 3079 result from the high proportion of small plants in this sector which will incur costs of compliance. Controls are required in SIC 3079 for many crushing and grinding operations used to prepare plastic material for hot processes.

Machinery Except Electrical (SIC 35) and Electrical Machinery (SIC 36). The machinery manufacturing sectors together are estimated to incur total annual compliance costs of $89.5 million. Machinery except electrical accounts for $57.7 million of this total. The electrical machinery sector is estimated to require $31.8 million in annual compliance costs. Controls in these sectors would be required for exposures to metals, solvents and nuisance dusts.

Fabricated Metal Products Manufacturing (SIC 34). Plating and coating establishments (SIC 347) and miscellaneous fabricated products (SIC 349) would account for a major portion of the $55.9 million annual costs in SIC 34. Worker exposures in this industry sector result from chemicals used in plating processes, solvents and coatings, metals and dusts. The survey indicated that ventilation systems are not now present at many of the processes with chemical exposure.

Transportation Equipment Manufacturing (SIC 37). Annual costs of compliance for SIC 37 are estimated at $47.1 million. Costs in the truck and car body and motor vehicle parts sectors (SICs 3711, 3713, 3714) would account for a large percentage of the costs in SIC 37. Controls may be needed in order to control exposures to heavy metals, solvents, welding fumes and a large variety of other chemicals at large scale hot processes. Additionally, costs in small plants in this sector will include compliance activities to control exposures to styrene and other chemicals in small boat construction, as well as trailer and recreational vehicle insulation.

Primary Metal Manufacturing (SIC 33). The annual costs of compliance in primary metal manufacturing are estimated to total $40.3 million. This estimate derived from the survey may somewhat undercount the compliance costs required in this sector. The cost algorithm assigned costs where prescribed controls were not in place. While many establishments in SIC 33 had controls in place, it is possible that the controls were not all operationally sufficient to control missions to the levels of the proposed standard.

The compliance costs for this sector are heavily weighted by the cost of controls required in large establishments in this segment. Blast furnace establishments, and primary foundries contain large numbers of hot processes which require controls. Control of emissions from hot metal processes to the levels indicated in the proposed standard will require large increases in the amount of air being moved through the ventilation systems. Additionally, costs will be required to enlarge the capacity of scrubbers and baghouse operations.

Chemicals and Allied Products (SIC 28). Annual compliance costs in SIC 28 are estimated to total $40.2 million. Over 25 percent of the costs in SIC 28 are estimated to occur in paints and allied products manufacturing (SIC 2851). The survey indicated that a large proportion of plants will require additional controls for the number and type of substances used in paint and paint product manufacturing. There are many chemicals in this industry segment which present exposure problems in a variety of wet and dry processes, including reaction, separation, crushing, mixing, drying and bagging.

Industry group SIC 282, Plastics Materials, Synthetic Resins, Synthetic Rubber also accounts for a major portion (about 22 percent) of compliance costs in this sector. Compliance costs are related to ventilation and other requirements to control carbon disulfide and other emissions in the manufacture of plastics materials and synthetic rubber.

Food and Kindred Products (SIC 20). Costs are projected for a large number of establishments in this sector. The prepared feeds and feed ingredients, not elsewhere classified (SIC 2048) are estimated to account for a large percentage of the $33.5 million annual costs in SIC 20. Controls may be necessary for dust exposures and chemical fumigants.

Paper and Allied Products (SIC 26). Annual costs in the paper and allied products industry are estimated to be $31.3 million. Much of the estimated costs in SIC 26 will be associated with the cost of controls in large pulp mill and associated operations. Pulp mills are operated separately (those listed in SIC 2611) or as part of paper or paperboard mills (SIC 2621 and SIC 2631 respectively). The cost of compliance in these operations would result from controlling the large quantities of chemicals used in breaking down the pulp to form cellulose and the reactions that occur in the digesting process. The digesting and bleaching operations required further ventilation or enclosure.

Apparel and Other Finished Products (SIC 23) and Textile Mill Products (SIC 22). These sectors have a large number of establishments which may incur compliance costs. The apparel industry is estimated to incur about $31.7 million in annual compliance costs. Many of the affected establishments in SIC 23 may require controls for cleaning solvents such as perchloroethylene as well as nuisance dusts. The $24.5 million annual costs in the textile industry are estimated to result from control of exposures to dusts, solvents, dyes and other substances.

Stone, Clay, Glass and Concrete Product Manufacturing (SIC 32). The stone, clay, glass and concrete product industry is estimated to incur

compliance costs of about $22.5 million. A major part of the annual costs in this industry segment may occur in the concrete, gypsum and plaster products (SIC 327) industries. According to the survey, controls in this sector are primarily expected to control nuisance particulates generated during large scale crushing, grinding and sizing operations.

Miscellaneous Manufacturing (SIC 39). This industry accounts for a wide range of products, processes and chemical exposures. About half of the establishments that would incur the $18.2 million annual cost in the industry are believed to be included in SIC 3999, miscellaneous manufacturing not elsewhere classified.

Furniture and Fixtures (SIC 25). Annual costs of compliance in the furniture and fixtures industries are estimated to total $14.7 million. Costs to control wood dust exposures during sanding, cutting and other dusting processes are the major components of compliance costs in this sector. Establishments would also incur costs for control of coatings and solvents. The survey indicated that the furniture sectors which include metal working (SICs 2514, 2515, 2522, 2542, 2591 and 2599) would also require controls for welding fumes and various metal particulates resulting from grinding and other processes.

Instruments Manufacturing (SIC 38). Annual control costs in SIC 38 are estimated to total $13.5 million. Exposures in this sector are to a large number of chemicals used within instruments and to dust, metals and solvents.

Petroleum Refining and Related Products (SIC 29). Petroleum refining may account for a large percentage of the $7.2 million annual costs in SIC 29, even though a relatively limited number of establishments in the petroleum refining industry would require additional controls. This industry has extensive control technology in place as well as many closed processes with few exposed workers.

Other Manufacturing. The lowest costs of compliance in the manufacturing sectors are expected to occur in SIC 21, Tobacco Manufacturers, ($0.02 million) and SIC 31, Leather and Leather Products ($2.6 million). It is estimated that very few plants will incur costs in the tobacco manufacturing industry. In the leather and leather products industry sector, most of the affected establishments produce manufactured leather goods.

Transportation, Communication, Utilities

The transportation and utilities sectors (SICs 40, 45, 47, and 49) include a large number of establishments. However, operations at Railroad (SIC 40), and Air Transport establishments (SIC 45) are subject to regulation by other Federal agencies in addition to OSHA. Consequently, the number of establishments which would incur costs to comply with the proposed standard are limited. For railroads, OSHA's standards normally apply to off-track operations.

Electric, Gas and Sanitary Service Utilities (SIC 49). Annual cost in the utilities sectors are estimated to total $22.7 million. Costs would result from installation and improvement of controls necessary for activities such as boiler/furnace feed preparation in electric services, odorant addition by natural gas companies, and water treatment and purification of water supplies.

Transportation Services Sector (SIC 47). The $1.8 million annual costs in the SIC 47 may primarily be incurred in SIC 4789, transportation services not elsewhere classified. This sector includes establishments which provide incidental services such as cleaning railroad ballast and other rail car maintenance.

Wholesale and Retail Trade

Costs in the wholesale trade sectors (SICs 50, 51), are estimated to total about $12.9 million annually. A large percentage of the total number of establishments which would incur costs to comply with the proposed rule are in SIC 5093, scrap and waste materials, wholesale.

The only retail trade sector expected to incur compliance costs, Auto Dealers (SIC 55) is estimated to incur $12.0 million annually. These costs result from the potentially large number of motor vehicle dealers (SIC 5511) which may incur compliance costs to control exposures to paints, coatings and solvents during vehicle spray and coating operations. The costs result from the installation of paint spray booths.

Services

The services sectors, SICs 72, 73, 75, 76 and 80 are estimated to total about $51.0 in annual compliance costs. The major costs (61.3 percent) in these sectors would result from potential compliance activities in SIC 721, laundry, cleaning and garment services. Establishments in SIC 721 would incur annual operating and annualized capital costs to control exposures for dry cleaning operations.

Additional costs in the service sectors may result from control of solvent chemicals in SIC 734, building services, control of welding fumes at welding repair operations (SIC 7692), control of solvent and photographic chemicals in mailing, reproduction, commercial art and photography and stenographic services (SIC 733), and local ventilation for exposure control in SIC 8071, medical laboratories.

Per Plant Average Costs

Table VI–2 presents the estimated average per plant annual cost of compliance by industry sector. Costs shown in this Table are calculated only for those establishments in a sector which would incur costs. Average per plant annual operating and annualized capital costs for all affected establishments across industry sectors is estimated at $9,200. The per plant cost for large plants is $14,400 and for small plants with fewer than 20 employees, $6,300.

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TABLE VI-2

AVERAGE PER PLANT ANNUAL COSTS AND NUMBERS OF AFFECTED PLANTS

SIC (a)	SIC DESCRIPTION	ANNUAL COST OF	TOTAL # PLANTS	# OF AFFECTED PLANTS	% AFFECTED	AVERAGE COST PER AFFTED PLANT	AVERAGE COST PER LARGE AFFTED PLANT	AVERAGE COST PER SMALL AFFTED PLANT
20	FOOD PROD. (b)	$33,493,100	29,043	4,932	16.98%	$6,800	$13,000	$3,600
21	TOBACCO (b)	$19,700	216	3	1.39%	$6,600	$6,600	$0
22	TEXT. MILL (b)	$29,478,400	11,023	2,765	25.08%	$10,700	$21,400	$3,700
23	APPAREL PROD. (b)	$31,744,200	30,032	6,179	20.57%	$5,100	$11,500	$2,000
24	LUMBER & WOOD	$196,086,100	17,383	3,715	21.37%	$52,800	$14,200	$77,400
25	FURNITURE	$14,720,600	13,072	2,001	15.31%	$7,400	$9,900	$5,700
26	PAPER PROD.	$31,257,700	6,583	2,060	31.29%	$15,200	$20,400	$500
27	PRINTING & PUB.	$75,688,300	59,107	6,077	10.28%	$12,500	$10,500	$13,100
28	CHEMICAL PROD.	$40,157,600	15,345	3,620	23.59%	$11,100	$14,500	$5,900
29	PETRO. REFINING	$7,215,300	2,271	378	16.64%	$19,100	$25,700	$3,700
30	RUBBER & PLASTICS	$75,482,100	14,964	4,229	28.26%	$17,800	$24,700	$10,700
31	LEATHER PROD.	$2,579,500	2,196	822	37.43%	$3,100	$11,200	$1,600
32	STONE & CLAY	$22,542,600	13,980	3,958	28.31%	$5,700	$7,900	$3,600
33	PRIM. METAL	$40,333,600	7,640	2,744	35.92%	$14,700	$20,100	$5,500
34	FAB. METALS	$55,937,600	33,946	6,285	18.51%	$8,900	$14,800	$1,800
35	MACHINERY	$57,740,400	62,443	8,059	12.91%	$7,200	$13,300	$2,900
36	ELEC. MACH.	$31,781,400	34,370	4,436	12.91%	$7,200	$13,300	$2,900
37	TRANS. EQUIP.	$47,099,100	12,834	4,444	34.63%	$10,600	$14,400	$8,800
38	INSTRUMENTS	$13,464,700	14,561	1,879	12.91%	$7,200	$13,300	$2,900
39	MISC. MANUF.	$18,195,500	19,677	2,540	12.91%	$7,200	$13,300	$2,900
40	R.R. TRANS.	$532,200	4,798	81	1.68%	$6,600	$6,600	$0
45	AIR TRANS.	$1,828,900	16,487	277	1.68%	$6,600	$6,600	$0
47	TRANS. SERV.	$1,853,100	16,705	281	1.68%	$6,600	$6,600	$0
49	ELEC. GAS. SAN.	$22,688,400	15,935	1,409	8.84%	$16,100	$16,400	$14,400
50	WHOLESALE TRADE	$4,054,600	12,222	1,228	10.05%	$3,300	$7,700	$2,500
51	WHOLESALE, NON-DUR	$8,858,200	26,701	2,683	10.05%	$3,300	$7,700	$2,500
55	AUTO DEALERS (b)	$11,954,700	189,214	3,091	1.63%	$3,900	$6,600	$1,300
72	PERSONAL SRV. (b)	$31,287,800	161,004	12,453	7.73%	$2,500	$27,900	$1,300
73	BUSINESS SRV. (b)	$8,953,800	382,626	4,557	1.19%	$2,000	$6,600	$1,300
75	AUTO REPAIR (b)	$1,510,900	149,260	528	0.35%	$2,900	$14,600	$2,100
76	MISC. REPAIR SRV.	$4,848,500	13,856	2,388	17.23%	$2,000	$2,200	$5,000
80	HEALTH SERV. (b)	$4,439,400	313,076	1,156	0.37%	$3,800	$12,500	$2,100
TOTAL		$927,828,000	1,702,569	101,258	5.95%	$9,200	$14,400	$6,300

(a) Industry sectors not identified in this table include industries with no major cost impact expected, the construction industry, which will be the subject of a separate regulatory analysis, and industries such as mining, for which OSHA does not have jurisdiction.

(b) All data shown for these industries was derived from secondary sources and expert judgement.

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The highest costs on an average per plant basis are expected to occur in SIC 24. Average per plant costs in SIC 24 may total $52,800 in annual operating and annualized capital costs. This per plant cost is heavily weighted by the above average costs which may be incurred by small establishments to control worker exposure to wood dust. Per plant costs in SIC 24 are substantially higher than those in the next highest industry, SIC 30, Rubber and Plastics. The $17,800 per plant costs in this industry result from above average compliance costs estimated for exposure control in molding and vulcanizing in large plants and crushing and grinding operations in small plants.

Although small establishments, account for about 65.0 percent of the 101,200 affected establishments, compliance costs for small establishments are expected to account for roughly 44.9 percent of total industry compliance costs.

References

1. Dun and Bradstreet, Inc., 1985 Count of Establishments. (Database)

VII. Economic Impact, Regulatory Flexibility Analysis and Environmental Impact Assessment

Economic Impact

The economic impacts discussed in this chapter have been estimated following an analysis of data collected through a nationwide sample survey of over 5,300 affected establishments. Two alternative polar assumptions were used in this analysis.

- *Perfectly Elastic Demand or Zero Cost-Passthrough:* All compliance costs are absorbed by the firm in the form of reduced profits. This assumption is the "worst case" scenario, where the maximum reduction in profits to the firm (and industry) results.
- *Perfectly Inelastic Demand or Total Cost-Passthrough:* All compliance costs are passed on to the consumer sector in the form of higher prices. From the perspective of the firm, this is the "best case" scenario. The resulting price increase would be the maximum theoretically possible.

Two points should be noted. First, for the majority of industry sectors, neither assumed market structure would be accurate. In practice, the impacts will almost always produce a price increase smaller than the inelastic demand projection and a reduction in profits smaller than that predicted under perfectly elastic demand conditions. Second, increased firm productivity would mitigate any adverse economic effects of the proposed standard. Productivity effects would be related to reduced worker illness, absence and turnover. In addition, knowledge of improved workplace health conditions could result in higher workforce morale and productivity . The firm would enjoy lower employee training costs (due to the reduced turnover rate) and lower medical benefit and worker compensation claims. Overall productivity increases would be realized by firms that use a relatively fixed-factor production process (i.e., low elasticities of substitution between labor and other factors of production). It is difficult to estimate the magnitude of these productivity and cost reducing effects. Any estimated economic costs of compliance would have to be adjusted downward to reflect these effects. Since data were not available to make any offset estimates, the economic effects of the proposal identified in this chapter, are overstated.

For this analysis, OSHA used a percentage reduction in profits approach to obtain estimates of the short-run economic impacts under the assumption of perfect demand elasticity. These estimates were obtained by using the following formula:

$$\text{Percentage Reduction} = \frac{\text{New Profits—Old Profits}}{\text{Old Profits,}}$$

where New Profits = Old Profits—Compliance Costs, and
Old Profits = (Return on Sales)*(Total Sales).

These calculations were performed at the two-digit SIC level for firms in large and small size-class stratifications (above and below 20 employees). The data used to obtain these estimates was based on Dun and Bradstreet company files [1;2].

The potential impact on prices was used to estimate the market consequences under the second assumption of inelastic demand. This price increase was estimated for each industry at the four-digit SIC level. Total sales values for 1985 were used, the year for which the compliance costs were estimated. (Total sales represent the totality of production that leaves the establishment, whether it is sold to customers or sent to a parent company in a captive transaction. For industries in the service and trade sectors, total sales data were used. The rate of return percentage for each industry sector corrected and transformed gross sales data into more accurate and relevant industry profit estimates.)

For a given firm-size class, the potential price increase was estimated by dividing the total estimated compliance costs for a firm by the sales of that firm. These estimated price effects were then compared to recent industry price series. The intent of this comparison was to evaluate the impact of the compliance cost-generated price increase in light of recent industry price increase experience.

In this scenario, the potential for international trade implications of the proposed standard was explored. It is anticipated that any international trade effects will not be significant given the small value of domestically produced goods and services which are exported (about seven percent of GDP). Also, in recent months, the U.S. dollar has experienced a sharp decline in value relative to the yen and European currencies. Between February 1985 and December 1987, the trade-weighted value of the U.S. dollar fell 46 percent [3]. This depreciation overwhelms any potential adverse international economic effect of the standard.

In Table VII–1 and VII–2, the estimated domestic economic impacts are reported for the two polar methodologies. To derive the percentage change in profits and the costs as a percent of sales, industry sales and rate of return (R.o.R.) on sales data were obtained from Dun and Bradstreet. The total sales data are best estimates for industry sectors potentially impacted by the proposed rulemaking.

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TABLE VII-1

ECONOMIC EFFECTS: NO-COST PASSTHROUGH SCENARIO[1]

SIC	Industry	Annual Costs[2] ($ millions)	Total Sales[3] ($ millions)	R.o.R. on Sales (%)[4]	Pre-Reg Profits ($ m)	Post-Reg Profits ($ m)	% Change in Profits
20	FOOD PROD.	33.49	353,780.38	1.9	8,008.04	7,986.29	- 0.2715
21	TOBACCO	0.02	74,030.13	5.3	3,923.60	3,923.59	- 0.0003
22	TEXT. MILL	29.48	60,735.22	2.7	1,765.42	1,747.59	- 1.0100
23	APPAREL PROD.	31.74	74,474.65	2.8	1,813.22	1,793.56	- 1.0845
24	LUMBER & WOOD	196.09	57,994.48	3.9	1,974.51	1,814.21	- 8.1188
25	FURNITURE	14.72	37,648.27	3.5	1,411.02	1,400.96	- 0.7129
26	PAPER PROD.	31.26	103,694.14	3.7	3,778.20	3,761.24	- 0.4489
27	PRINTING & PUB.	75.69	134,830.21	4.8	6,471.85	6,412.25	- 0.9210
28	CHEMICAL PROD.	40.16	272,759.67	3.7	11,738.80	11,714.76	- 0.2048
29	PETRO. REFINING	7.22	196,400.57	2.7	4,964.85	4,960.84	- 0.0808
30	RUBBER & PLASTICS	75.48	86,538.58	4.3	3,423.75	3,376.10	- 1.3918
31	LEATHER PROD.	2.58	15,449.56	2.6	401.69	399.95	- 0.4328
32	STONE & CLAY	22.54	46,094.04	4.1	1,954.99	1,940.73	- 0.7300
33	PRIMARY METALS	40.33	112,564.26	3.3	3,714.62	3,691.27	- 0.6286
34	FAB. METALS	55.94	150,146.41	4.0	6,005.86	5,974.10	- 0.5288
35	MACHINERY	57.74	345,144.89	5.1	17,602.39	17,566.95	- 0.2013
36	ELEC. MACH.	31.78	245,982.70	5.0	12,299.14	12,279.63	- 0.1586
37	TRANS. EQUIP.	47.10	365,427.20	3.9	14,520.25	14,486.69	- 0.2311
38	INSTRUMENTS	13.46	83,359.57	4.9	3,373.26	3,365.00	- 0.2450
39	MISC. MANUF.	18.20	41,870.30	4.4	1,788.56	1,777.39	- 0.6245
40	R.R. TRANS.	.53	43,869.14	10.0	3,969.62	3,969.34	- 0.0072
45	AIR TRANS.	1.83	109,538.08	3.6	3,251.40	3,250.41	- 0.0304
47	TRANS. SERVICES	1.85	12,254.96	2.7	582.18	581.18	- 0.1719
49	ELEC.,GAS & SAN.	22.69	300,254.83	7.0	21,017.84	21,004.06	- 0.0655
50	WHOLESALE TRADE[5]	4.05	13,853.52	2.0	277.07	273.67	- 1.2285
51	WHOLESALE, NON-DUR	8.86	113,848.20	1.5	1,726.26	1,721.48	- 0.2771
55	AUTO DEALERS	11.95	341,574.50	1.9	6,489.92	6,482.81	- 0.1095
72	PERSONAL SERV.	31.29	24,270.74	7.3	1,771.76	1,750.02	- 1.2272
73	BUSINESS SERV.	8.95	22,165.94	6.6	1,462.95	1,455.45	- 0.5126
75	AUTO REPAIR	1.51	45,750.92	5.1	2,492.19	2,491.05	- 0.0457
76	MISC. REPAIR SERV.	4.85	2,665.52	5.5	146.60	142.69	- 2.6696
80	HEALTH SERVICES	4.44	170,234.25	4.5	7,807.72	7,804.54	- 0.0406

Source: U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.

Notes:
1. All values in 1985 dollars.
2. Reproduced from Table VI-1.
3. Dun and Bradstreet, Dun's Marketing Identifiers (DMI) Database.
4. Rate of Return on Sales, Dun and Bradstreet, Industry Norms Database.
5. Consists of SIC 5093 (scrap and waste materials) only.

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TABLE VII-2.—ECONOMIC EFFECTS: TOTAL-COST PASSTHROUGH

SIC	Industry	Annual Costs (dollars millions)	Total Sales (dollars millions)	Costs as a percent of sales
20	Food prod	33.49	353,780.38	0.0095
21	Tobacco	0.02	74,030.13	.0000
22	Text. mill	29.48	60,735.22	.0485
23	Apparel prod	31.74	74,474.65	.0426
24	Lumber & wood	196.09	57,994.48	.3381
25	Furniture	14.72	37,648.28	.0391
26	Paper prod	31.26	103,694.14	.0301
27	Printing & pub	75.69	134,830.21	.0561
28	Chemical prod	40.16	272,759.67	.0147
29	Petro. refining	7.22	196,400.57	.0037
30	Rubber & plastics	75.48	86,538.58	.0872
31	Leather products	2.58	15,449.56	.0167
32	Stone & clay	22.54	46,094.04	.0489
33	Prim. metals	40.33	112,564.26	.0358
34	Fab. metals S	55.94	150,146.41	.0373
35	Machinery	57.74	345,144.89	.0167
36	Elec. mach	31.78	245,982.70	.0129
37	Trans. equip	47.10	365,427.20	.0129
38	Instruments	13.46	83,359.57	.0162
39	Misc. manuf	18.20	41,870.30	.0435
40	R.R. trans	.53	43,869.14	.0012
45	Air trans	1.83	109,538.08	.0017
47	Trans. services	1.85	12,254.96	.0151
49	Elec, gas & san	22.69	300,254.83	.0076
50	Wholesale trade [1]	4.05	13,853.52	.0293
51	Wholesale, non-dur	8.86	113,848.20	.0078
55	Auto dealers	11.95	341,574.50	.0035
72	Personal services	31.29	24,270.74	.1289
73	Business service	8.95	22,165.94	.0404
75	Auto repairs	1.51	45,750.92	.0033
76	Misc. repair serv	4.85	2,665.52	.1819
80	Health services	4.44	170,234.25	.0026

Source: U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.
NOTE.—1. Consists of SIC 5093 (scrap and waste materials) only.

Dun and Bradstreet provided OSHA with this information. The R.O.R. on sales were obtained from summary statistics found in the Dun and Bradstreet Industry Norms Database.

Industry Effects

The estmiated economic impact of the proposed rule from firms potentially affected is summarized in Table VII-1. These estimates represent the maximum industry impact within a market scenario where none of the costs can be passed onto consumers, and there is no productivity offset to costs.

Data in Table VII-1 indicate that the proposed rule will not have a significant impact on profits in most industry sectors. The estimated average change in profits is less than one percent; this amount of profit reduction should not represent a significant economic burden.

The most adversely affected industry sector is SIC 24 with an estimated 8 percent reduction in profits. The only other industry with an impact greater than 2 percent is SIC 76, Miscellaneous Repair Services (2.7 percent). However, even in the worst case, OSHA believes the proposal is economically feasible. In reality, the reduction in profits will be less because part of the costs will be passed on to consumers.

Consumer effects were estimated using a "full cost passthrough" scenario. As demonstrated by the estimates summarized in Table VII-2, the impacts on market prices will not be significant. No price increase would exceed one half of one percent. Changes of this magnitude are within general price movements recorded by producer price and other price indicies.

Regulatory Flexibility Analysis

In accordance with the Regulatory Flexibility Act (Pub. L. 96-353, 94 Stat. 1164 (5 U.S.C. 601 *et seq.*)), OSHA has assessed the impact of the proposed rulemaking on large and small establishments. For this assessment, large establishments are defined as those with 20 or more employees and small establishments as those with 19 or fewer employees. The results of this assessment are summarized in Table VII-3.

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TABLE VII-3

ECONOMIC IMPACTS BY ESTABLISHMENT SIZE

SIC	Industry	Percentage Change in Profits	
		Large	Small
20	FOOD PROD.	- 0.1526	- 3.0770
21	TOBACCO	- 0.0003	- 0.0000
22	TEXT. MILL	- 0.7442	- 7.0804
23	APPAREL PROD.	- 0.7916	- 3.4067
24	LUMBER & WOOD	- 0.6683	-47.3160
25	FURNITURE	- 0.3306	- 4.9038
26	PAPER PRODUCTS	- 0.4613	- 0.1559
27	PRINTING & PUB.	- 0.1579	- 4.1037
28	CHEMICAL PROD.	- 0.1533	- 1.2443
29	PETRO. REFINING	- 0.0754	- 0.3721
30	RUBBER & PLASTICS	- 0.8900	- 9.8232
31	LEATHER PROD.	- 0.2115	- 3.4147
32	STONE & CLAY	- 0.5342	- 1.4208
33	PRIMARY METALS	- 0.5330	- 2.3396
34	FAB. METALS	- 0.5079	- 0.7201
35	MACHINERY	- 0.1402	- 1.7854
36	ELEC. MACH.	- 0.1087	- 2.3924
37	TRANS. EQUIP.	- 0.0782	-21.2970
38	INSTRUMENTS	- 0.1704	- 2.2245
39	MISC. MANUF.	- 0.4920	- 1.3785
40	R.R. TRANS.[1]	n/a	n/a
45	AIR TRANS.[1]	n/a	n/a
47	TRANS. SERVICES[1]	n/a	n/a
49	ELEC., GAS & SAN.[1]	n/a	n/a
50	WHOLESALE, TRADE[2]	- 0.7355	- 1.8818
51	WHOLESALE, NON-DUR	- 0.1776	- 0.3964
55	AUTO DEALERS	- 0.1267	- 0.0778
72	PERSONAL SERV.	- 1.0749	- 1.3488
73	BUSINESS SERV.	- 0.3186	- 0.8749
75	AUTO REPAIR	- 0.0527	- 0.0441
76	MISC. REPAIR SERV.	- 0.8702	- 3.3812
80	HEALTH SERVICES	- 0.0736	- 0.0295

Source: U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.

Notes: 1. Percent of profit attributable to small firms was not available.

2. Consists of SIC 5093 (scrap and waste materials) only.

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Industry sales and profit estimates were based on data from Dun and Bradstreet and the Department of Commerce 1982 Census of Manufacturers [5], Wholesalers [6], Retailers [7], and Services [8]. Sales and profit data for selected transportation sector industries (SIC 40, 45, 47, and 49) were not available for use in this preliminary assessment.

The information summarized in Table VII–3 indicates that some small establishments will experience some adverse impact. The smaller profit margins of some small establishments make it difficult for them to absorb increases in compliance costs. In particular, small establishments in SIC 24 (Lumber and Wood), SIC 37 (Transportation Equipment Manufacturers), and SIC 30 (Rubber and Plastics) show high potential changes in profits. Some of these industries are capital-intensive and the costs are not high in proportion to the capital invested. OSHA requests comments on approaches to reduce the impact on these establishments.

It should be noted that these negative effects result in part from the extreme assumption of perfectly elastic demand. An important ameliorating factor for each firm will be its ability to pass through additional costs to the consumer. The ability of individual firms to do this will be dependent upon product demand elasticities. It is expected that most impacted firms will be able to pass through some portion of their increased costs.

Environmental Impact Assessment

This assessment has been prepared in accordance with provisions of the National Environmental Policy Act (NEPA) (42 U.S.C. 4325 *et seq.*) as well as the regulations of the Council on Environmental Quality (40 CFR Part 1500), and DOL–NEPA Compliance Procedures (29 CFR Part 11).

OSHA has reviewed the proposal and the information contained in the secondary data bases as well as the information submitted by the contractors' industry experts and has concluded that no significant environmental impacts are likely to occur as a result of this action.

Two environments may be affected by an OSHA regulatory action: (1) The workplace environment; and (2) the general human environment external to the workplace, including impacts on air and water pollution, solid waste, energy, and land use. Usually OSHA regulations have their most significant impacts on the workplace environment since this environment is under the Agency's jurisdiction. Lower and new PELs would benefit the workplace environment because they would reduce worker exposure to toxic substances.

In most cases, the effects of previous OSHA regulations on the external environment have been negligible because of their limited scope and application. Similarly, there is no evidence to indicate that there would be any significant adverse impacts to the external environment as a result of this proposal. As with other OSHA regulations in the past, however, there may be a potential benefit to the environment, The potential benefits and other impacts are briefly summarized here.

Air Pollution

Because of the nature of the emission standards of the Environmental Protection Agency (EPA) (40 CFR Part 61), many industry operations already use engineering controls to reduce the amount of emissions to the atmosphere. This practice is not expected to change as a result of the proposal. OSHA anticipates that controls already in place will continue to operate effectively in reducing emissions under the proposed revisions. Fourteen of the chemicals addressed in this proposal have been recognized by EPA as air pollutants. These are listed below:

- Beryllium
- Carbon Monoxide
- Epichlorhydrin
- Ethylene dichloride
- Hexachlorocyclopentadiene
- Mercury
- Methyl chloroform
- Nitrogen dioxide
- Ozone
- Perchloroethylene
- Sulfur dioxide
- Toluene
- Trichloroethylene

Water Pollution

EPA regulates over 100 of the chemicals addressed in this proposal under the Clean Water Act of 1977 (33 U.S.C. 1251 *et seq.*). EPA's effluent limitation guidelines (40 CFR Part 427) include (1) standards of performance for all new point sources within specified categories and (2) pretreatment standards for new plants discharging to municipal sewer systems. These limitations would serve to prevent the discharge of effluents into the environment without prior treatment. Moreover, the Fedral Water Pollution Control Act Amendments of 1972 required that wastewater effluents be treated by the best practicable technology (BPT) by December 31, 1977 and that the best available technology (BAT) economically achievable be used by December 31, 1983. The EPA effluent limitations establish the degree of effluent quality necessary to meet the BPT and BAT requirements. The BAT and pretreatment standards would essentially mean no discharge of process wastewater to navigable waters and no discharge of incompatible pollutants. These requirements will not change as the result of this proposal and where they continue to be met, effluent quality will not be altered.

Solid Waste Disposal

It does not appear that there would be any significant change in present waste disposal practices for over 80 chemicals addressed by this proposal, or in the maintenance of waste disposal sites. EPA's national emissions standards will continue to provide for the control and maintenance of active and inactive disposals as well as require no visible emissions from these sites.

Energy and Land Use

The implementation of required engineering controls could result in an increase in total energy requirements or costs for general industry. This would be particularly true where controls are not in place. Where general exhaust ventilation is used, there is the expense of heating or cooling the replacement air brought in from the outside. These costs, plus the cost of vacuuming, where necessary, have been included in the annual costs estimated in Chapter VI. In terms of land use, OSHA does not project any significant impact on land use plans, policies or controls. OSHA does not anticipate any significant impact on the short term uses of man's environment or upon the maintenance of long-term productivity.

Other Impacts

The proposal could also have other impacts that may affect the external environment. The proposal could encourage the further use, research, and development of suitable substitutes for hazardous chemicals. This, in turn, would result in a positive environmental effect because fewer hazardous chemicals would be used, emitted to the air, discharged as wastewater effluent, or disposed of as solid waste. The magnitude or probability of these impacts, however, is impossible to quantify.

Overall, the projected impacts of the proposed standard on the external environment are not expected to be significant in view of EPA's regulation of air emissions, water effluents, and solid waste disposal methods.

Summary

Based on the data summarized in Tables VII–1 and VII–2 and historical information, OSHA has initially concluded that the economic impacts of the proposed rulemaking will not unduly burdensome for firms in the industries potentially affected. However, some industry sub-sectors may experience an adverse economic impact. In addition, the estimates indicate that some small establishments in SICs 24, 30, and 37 may experience a greater impact than larger entities. OSHA requests comments on approaches to reduce the impact on those small establishments. It should be emphasized that these estimates are preliminary. The proposed rule is not expected to have an adverse effect on the environment.

References

1. Dun and Bradstreet, Inc. Industry Norms and Key Business Ratios. 1985. (Database)
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6. U.S. Department of Commerce, Bureau of the Census. 1982 Census of Wholesale Trade, Establishment and Firm Size (Including Legal Form of Organization). Washington D.C.: U.S. Government Printing Office.
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Supplement 1 to Appendix B—Technical Description of the Sample Survey

1. Introduction

This appendix contains a description of the statistical methodology employed to design and implement the PEL survey. The following topics will be discussed:

- Survey objectives;
- Sampling frame selection;
- Stratification;
- Sample size determination;
- Estimation procedures;
- Data collection method;
- Variance estimation;
- Treatment of non-sampling errors; and
- Survey treatment.

Survey Objectives

Surveys are frequently designed to produce a set of estimates at a predefined level of accuracy. This requires defining the set of quantities to be estimated and specifying their levels of accuracy. Since many variables may ultimately be estimated from the survey, and since no single design can be optimal for all estimates simultaneously, it is customary to define the most important variables for estimation. For this survey, the following variables were identified as those motivating the survey design:

- Cost to industry of the proposed set of new permissible exposure limits (as a group);
- Number of workers potentially exposed to toxic substances; and
- Number of workers affected by the proposed regulations.

Statistical theory dictates that responses be concentrated both in groups which have the highest variability with respect to these variables and in groups representing the majority of establishments in the population. No hard information relating to the variability of the variables mentioned above by industry sector or other relevant breakdown was available at the outset of the survey. Hence, the variability in the number of employees was used as a variability measure. Consistent with the notion that the variability of numbers exposed as well as the variability of cost required to remedy an overexposure are highest in the largest companies, the sample was designed to include a higher proportion of larger establishments.

The sample was drawn so as to insure that the coefficient of variation of estimates (the ratio of the sample standard error to the mean) was within predetermined bounds. The coefficient of variation is a measure of the accuracy of each estimate. A coefficient of variation of.5 percent means that the standard error of the estimate is equal to 5 percent of that estimate. This can be interpreted as saying that the estimate is within two standard errors or 10 percent of the true value with 95 percent probability. Since risks were judged to be different in different sectors, OSHA selected a 5 percent coefficient of variation in the industries using the most chemicals, 7.5 percent in industries with moderate use of chemicals and 10 percent in the service sectors. A table of design specifications in included in Section 5 below.

3. Sampling Frame Selection

The Dun and Bradstreet (D&B) listing was chosen for the PEL survey sampling frame (a listing of establishments from which sample units are selected). This is a nationally based list, containing establishment names as well as each establishment's address, telephone number, SIC code, and number of employees. The Dun and Bradstreet database is regularly refined (every six months) thus minimizing the probability of obtaining out of business or out of scope (e.g., wrong SIC code) establishments when using the frame. The D&B is a commercial listing and its use does not violate any confidentially requirement associated with other frames available to particular agencies in the government.

4. Stratification

Thirty-four groupings of industries (estimation cells) were chosen to be examined for the PEL study. The cell definitions were determined by grouping together industry sectors defined by Standard Industrial Classifications (SICs) which share similar processes and procedures. The cell definitions used for the PEL survey are given in TABLE 1–1.

TABLE 1–1.—DEFINITIONS OF ESTIMATION CELLS

Cell No.	SIC codes included	Description
1	243	Millwork, Veneer, Plywood.
2	245	Wood Bldgs & Mobile Homes.
3	249	Misc. Wood Products.
4	25	Furniture.
5	26	Paper Products.
6	27	Printing & Publishing.
7	281	Indust. Inorganic Chems.
8	282	Plastics & Syn. Resins.
9	283	Drugs.
10	284	Soaps, Detergents & Cleaning.
11	285	Paints, Varnishes, Lacquers.
12	286	Indust. Organic Chems.
13	287	Agricultural Chemicals.
14	289	Misc. Chemical Products.
15	291	Petroleum Refining.
16	295	Paving & Roofing Materials.
17	299	Misc. Petroleum Products.
18	308	Misc. Plastic Products.
19	30 (not 308)	Plastics & Rubber.
20	311	Leather Tanning.
21	31 (not 311)	Leather & Leather Products.
22	32	Stone & Clay.
23	33	Primary Metals.
24	34	Fabricated Metals.
25	35	Machinery.
	36	Electrical Machinery.
	38	Instruments.
	39	Misc. Manufacturing.
26	40	R.R. Transportation.
	44	Water Transportation.
	45	Air Transportation.
	47	Transportation Services.
27	46	Pipelines.
28	49	Electrical, Gas & Sanitary.
30	5093	Misc. Durable Goods.
	5153	Grain.
	5161	Chemicals & Allied Products.

TABLE 1-1.—DEFINITIONS OF ESTIMATION CELLS—Continued

Cell No.	SIC codes included	Description
	5191	Misc. Farm Supplies.
	5198	Misc. Paints, Varnishes.
31	55	Auto Dealers.
	75	Auto Repair.
32	7211	Power Laundries, Family & Commercial.
	7213	Linen Supply.
	7215	Coin-operated Laundries & Cleaning.
	7216	Drycleaning Plants, except Rug.
	7218	Industrial Launderers.
	7219	Laundry & Garment Services, nec.
	7221	Photographic Studios, Portrait.
	7231	Beauty Shops.
	7241	Barber Shops.
	7251	Shoe Repair & Shoeshine Parlors.
	7261	Funeral Service & Crematories.
	7299	Miscellaneous Services, nec.
	7332	Blueprinting & Photocopying Services.
	7342	Disinfecting & Pest Control Services.
	7395	Photofinishing Laboratories.
33	7641	Furniture Repair.
	7692	Welding Repair.
34	80	Health Services.
99	37	Transportation Equipment.

For each estimation cell, units on the Dun and Bradstreet sampling frame were classified into one of the four size classes listed below:

Size	Number of Employees
1	0 to 19.
2	20 to 99.
3	100 to 249.
4	250 and above.

For each size class stratum within a cell, the establishments on the frame were further identified by their four digit SIC classification (within the two or three digit sample cell). A separate systematic sample was then selected in each estimation cell/size class stratum. This procedure was accomplished by first selecting one case at random in the size class for the first K units on the frame—where K is the reciprocal of the sampling fraction—and then selecting every Kth unit in the stratum thereafter. Note, from the size class definitions that establishments having zero employees were not included in the survey. Such units were assumed to be out of the scope of the survey.

5. Sample Size Determination and Allocation Within Strata

The total number of establishments selected from the Dun and Bradstreet sampling frame was determined using two stages. The first stage was to compute the target number of respondents for each estimation cell using the standard sample size formula. The formula requires the specification of a target coefficient of variation for estimates.

The coefficients of variation for this survey were set at the following levels:

SIC's	Coefficient of Variation (percent)
24 through 29	5
30 through 39	7.5
40 through 80	10

The units were then allocated to size classes within the estimation cells using Neyman allocation. This method allocates based on the number of establishments in each stratum and on the stratum variability in the key design variable (in this case employment). Size class strata having a large number of establishments on the frame or a high variability in employment (as defined by the population variance) received a greater number of sample units than other strata in the sample. Because the larger size classes often have a high variability in employment, this allocation resulted in "oversampling" the larger size classes in a cell. The required number of cases for each stratum are shown in TABLE 1-2 in the column labeled "Target Number of Respondents."

The number of units actually selected from the D&B frame in each stratum was based on the number of completed cases required for the stratum and on the expected response rate. Almost all sample surveys, especially voluntary surveys, select some number of cases which do not result in a completed interview. In some instances, these will be establishments which have gone out of business, are duplicate cases, or are companies not in the SIC category shown on the frame. Such cases are "Out of Scope". Other establishments, though in scope, refuse to participate or are not reached in the sampling protocol, defined here as a total of five telephone attempts. Experience of surveys similar to the PEL survey indicated that a completion ratio of 50–60% was expected for this survey (the ratio of completed questionnaires to total cases which must be drawn—both in and out of scope). However, to be safe, a larger number of cases were selected and held in reserve from the D&B frame so that, if additional sample units needed to be included to reach the target sample sizes, the cases could be easily obtained.

In fact, for the vast majority of cells, a 60 percent completion ratio was realized. The total number of establishments called in each of the sample strata are shown in TABLE 1-2 in the column labeled "Total Cases Called." In general, this number is equal to the target sample divided by 0.60. The number of completed responses as of May 2, 1988 is shown in TABLE 1-2

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TABLE 1-2

Number of Firms, Required Sample Sizes, Calls Made and Completes

SIC GROUPS	Size	Total Plants	Target Number Respondents	Total Cases Called	Number Completed May 1988
243	0-19	10,986	39	78	53
	20-99	1,995	32	64	34
	100-249	346	12	24	17
	>250	147	48	98	63
	Total	13,474	131	264	167
245	0-19	864	15	25	9
	20-99	385	15	40	28
	100-249	220	17	45	17
	>250	43	30	43	8
	Total	1,512	77	153	62
249	0-19	4,301	37	74	27
	20-99	888	35	70	25
	100-249	129	11	22	8
	>250	44	12	24	8
	Total	5,362	95	190	68
25	0-19	11,505	20	40	23
	20-99	3,254	26	52	35
	100-249	858	13	26	13
	>250	449	73	146	78
	Total	16,066	132	264	149
26	0-19	3,485	20	44	16
	20-99	2,830	30	62	33
	100-249	1,307	30	54	34
	>250	576	184	384	221
	Total	8,198	264	544	304
27	0-19	64,922	45	60	31
	20-99	10,656	34	122	77
	100-249	1,850	12	126	87
	>250	869	88	50	37
	Total	78,297	179	358	232
281	0-19	1,721	20	52	30
	20-99	735	20	52	34
	100-249	189	20	52	29
	>250	157	96	157	75
	Total	2,802	156	313	168

TABLE 1-2, cont'd

SIC GROUPS	Size	Total Plants	Target Number Respondents	Total Cases Called	Number Completed May 1988
282	0-19	700	20	40	26
	20-99	499	20	40	26
	100-249	184	20	40	25
	>250	175	58	116	52
	Total	1,558	118	236	129
283	0-19	1,289	25	50	28
	20-99	544	25	50	28
	100-249	179	25	50	31
	>250	205	92	184	90
	Total	2,217	167	334	177
284	0-19	3,065	20	40	23
	20-99	767	20	40	28
	100-249	184	20	40	22
	>250	155	70	140	59
	Total	4,171	130	260	132
285	0-19	1,092	20	50	37
	20-99	549	15	40	21
	100-249	100	10	30	15
	>250	45	37	45	30
	Total	1,786	82	165	103
286	0-19	860	20	54	28
	20-99	346	15	44	27
	100-249	95	15	44	26
	>250	92	50	62	35
	Total	1,393	100	204	116
287	0-19	1,306	8	31	15
	20-99	338	9	33	22
	100-249	57	4	23	15
	>250	43	44	44	18
	Total	1,744	65	131	70
289	0-19	2,562	16	38	25
	20-99	918	23	52	31
	100-249	162	9	24	11
	>250	85	51	85	37
	Total	3,727	99	199	104

TABLE 1-2, cont'd

SIC GROUPS	Size	Total Plants	Target Number Respondents	Total Cases Called	Number Completed May 1988
291	0-19	606	20	50	28
	20-99	227	20	50	25
	100-249	85	20	50	28
	>250	130	59	90	47
	Total	1,048	119	240	128
295	0-19	862	21	46	28
	20-99	237	26	56	39
	100-249	45	10	24	19
	>250	9	8	9	3
	Total	1,153	65	135	89
299	0-19	516	15	32	23
	20-99	186	22	46	31
	100-249	23	4	11	10
	>250	5	5	5	3
	Total	730	46	94	67
308	0-19	8,062	16	32	21
	20-99	4,249	13	26	19
	100-249	1,162	7	14	12
	>250	388	18	36	22
	Total	13,861	54	108	74
30 (not 308)	0-19	1,983	25	50	33
	20-99	722	25	50	37
	100-249	239	25	50	29
	>250	234	48	96	59
	Total	3,178	123	246	158
311	0-19	283	5	24	2
	20-99	125	9	21	8
	100-249	29	4	16	2
	>250	16	5	10	5
	Total	453	23	71	17
31 (not 311)	0-19	2,232	8	16	6
	20-99	610	13	26	21
	100-249	220	13	26	17
	>250	176	13	26	17
	Total	3,238	47	94	61

TABLE 1-2, cont'd

SIC GROUPS	Size	Total Plants	Target Number Respondents	Total Cases Called	Number Completed May 1988
32	0-19	14,499	15	28	9
	20-99	4,207	34	62	40
	100-249	873	35	64	45
	>250	448	29	53	34
	Total	20,027	113	207	128
33	0-19	4,983	67	201	87
	20-99	2,803	67	201	127
	100-249	1,006	42	126	83
	>250	711	25	75	48
	Total	9,503	201	603	345
34	0-19	29,005	62	113	51
	20-99	11,849	110	200	113
	100-249	2,394	86	157	77
	>250	1,080	62	113	61
	Total	44,328	320	583	302
35,36, 38,39	0-19	117,005	100	200	65
	20-99	30,820	126	188	122
	100-249	7,468	93	137	98
	>250	5,657	80	133	72
	Total	160,950	399	658	357
40,44, & 45	0-19	45,323	20	37	20
	20-99	5,612	20	37	16
	100-249	799	20	37	15
	>250	533	50	91	32
	Total	52,267	110	202	83
46	0-19	439	15	28	23
	20-99	162	15	28	20
	100-249	18	8	18	14
	>250	5	5	5	5
	Total	624	43	79	62
49	0-19	12,982	40	73	48
	20-99	4,046	40	73	57
	100-249	844	40	73	58
	>250	558	150	273	198
	Total	18,430	270	492	361

TABLE 1-2, cont'd

SIC GROUPS	Size	Total Plants	Target Number Respondents	Total Cases Called	Number Completed May 1988
50 & 51~	0-19	45,422	200	364	237
	20-99	3,464	65	142	101
	100-249	205	30	79	48
	>250	57	57	57	30
	Total	49,148	352	642	416
55 & 75	0-19	284,632	10	30	13
	20-99	20,846	10	30	16
	100-249	1,523	10	30	15
	>250	116	20	60	22
	Total	307,117	50	150	66
72 & 73~~	0-19	139,889	120	240	22
	20-99	5,511	30	60	6
	100-249	527	20	40	9
	>250	108	25	50	13
	Total	146,035	195	390	50
7641 & 7692	0-19	18,098	60	110	67
	20-99	289	20	48	32
	100-249	12	10	9	8
	>250	1	1	1	1
	Total	18,400	91	168	108
80	1	233,984	50	91	49
	2	17,174	30	55	36
	3	6,310	30	55	37
	4	3,912	220	400	272
	Total	261,380	330	601	394
37	1	9,863	10	19	6
	2	2,997	10	19	12
	3	1,026	10	19	12
	4	1,072	70	128	72
	Total	14,958	100	185	102

~ Refers to SIC Codes: 5093, 5193, 5161, 5191, 5198
~~Specifically Sic Codes: 7211,7213,7215,7216,7218,7231, 7241,7251,7261,7299,7732,7342, and 7395

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6. Data Collection Methodology

The data collection method chosen for the survey was Computer Assisted Telephone Interviewing (CATI). In this method the interviewer talks to the respondent on the telephone while sitting in front of a computer screen. Each question to be asked appears on the screen in the proper sequence. CATI systems allow for the responses to be examined during the data collection process. Answers that are out of the possible range of responses or which are not consistent with other answers received earlier in the questionnaire can be immediately identified. Another advantage is that it frees the interviewer up from using a hard copy questionnaire which requires skipping manually to different parts of the questionnaire based upon the responses. Finally, this method saves resources by creating a machine readable record of the responses at the conclusion of the interview, thereby eliminating the need for keypunching.

7. Estimation Procedures

Cost estimates for the PEL study take the form of a combined ratio estimator and can be expressed in the following manner:

$$\hat{cost}_i = \frac{\sum_j \sum_k wgt_{ij} * NRAF1_{ij} * cost_{ijk}}{\sum_j \sum_k wgt_{ij} * NRAF2_{ij} * empl_{ijk}} * (Empl/BLS202_i)$$

where

wgt_{ij} = the weight for cell i and size class j

$NRAF1_{ij}$ = nonresponse adjustment factor

$NRAF2_{ij}$ = nonresponse adjustment factor

$cost_{ijk}$ = cost associated with unit k in the cell i and class j

$empl_{ijk}$ = number of employees at unit k in cell i and class j

$Emp/BLS202_i$ = total employment in cell i from the BLS 202 database.

This estimator benchmarks the estimate obtained from the Dun and Bradstreet sample to Bureau of Labor Statistics ES–202 employment counts for each cell. The benchmarking helps to correct for any deficiencies in the D&B sampling frame. Such a procedure is particularly important in the non-manufacturing industries where establishments are frequently under-represented on the D&B sampling frame.

8. Variance Estimation

As with any sample survey, quantification of sampling error of estimates is an important function. Errors are quantified by computing the standard error of each estimate produced from the survey. Under certain assumptions, the standard error can be used to make probability statements about estimates. For example, an interval equal to two standard errors on either side of an estimate is a 95 percent confidence interval. This means that one can be 95 percent sure that the true value of the quantity being estimated lies somewhere inside that interval.

A replication technique will be used to determine standard errors for the PEL survey. For this method one resamples the original data multiple times to compute standard errors. A replication method was chosen because of two characteristics of the survey. First, some of the estimates which are planned to be produced are nonlinear such as ratio estimates. Second, we are using a nonresponse adjustment factor to adjust the final weights. In both of these situations, replication-type variance estimators are particularly useful.

9. Treatment of Non-Sampling Errors

An important component to any survey effort is the treatment of nonsampling errors. Examples of such errors are:

- Nonresponse bias—error introduced because some selected respondents either do not respond at all (unit nonresponse) or do not respond to a particular question (item nonresponse);
- Response bias—error introduced due the way questions are phrased or the way respondents interpret what is being asked (this also includes error due to deliberate misrepresentation of the answers to questions by respondents).

In the PEL survey, the nonresponse problem was dealt with using two standard methodologies. For unit nonresponse, a mean imputation procedure was used. This procedure assumes that there is no fundamental difference between respondents and nonrespondents and, therefore, usable cases can be reweighted to represent the entire universe. For item nonresponse, an imputation scheme which uses related cases in the respondent group to estimate the missing data was used.

The situation for response bias will be handled by obtaining information from site visits. OSHA will conduct 100 site visits in a cross section of industries. A large portion of these visits will be done on establishments which were also in the telephone survey. Data on key variables collected during the telephone survey will be compared with information obtained from the site visits. This analysis must be undertaken when site visits have been completed.

9.1. Unit Nonresponse Adjustment

In order to adjust the sample for those cases selected from the D&B frame which where called but were out of scope (OOS), out of business (OOB), or in scope but unwilling to participate in the survey, the following approach was used.

- All solicited sample units were assigned a response code based on the following categories:

03 Non-working telephone number
04 Incorrect SIC—out of scope
05 Out of Business (OOB)
06 Not a business or wrong business
07 Duplicate record
08 Could not reach respondent after five attempts
09 Communication barrier
10 Initial refusal
11 Mid-interview refusal (did not answer initial chemical and process questions)
12 Completed interview (completed both initial chemical and process questions)
13 Other nonresponse.

- All units having a response code equal to 08, 09, 10, 11, 12, or 13 were classified as viable sample units (in

scope, in business). Sample units having a response code equal to 12 were classified as both viable and usable. Two nonresponse adjustment weights were assigned to each usable record in the database, based on the ratio of viable to usable sample units in the record's cell and size stratum:

$$NRAF1_{ij} = \sum_{k=1}^{n_i} I(V_k) \Big/ \sum_{k=1}^{n_i} I(U_k)$$

$$NRAF2_{ij} = \sum_{k=1}^{n_i} I(V'_k) \Big/ \sum_{k=1}^{n_i} I(U'_k)$$

where,

i = number of estimation cell

j = number of the size class

$I(V_k)$ = 1 if the kth sample unit is viable,
= 0 otherwise;

$I(U_k)$ = 1 if the kth sample unit is usable,
= 0 otherwise;

V' and U' refer to the precoded employment from the D&B frame for viable and usable units.

NRAF1 is the ratio of viable to usable sample units in the cell/size class stratum. NRAF2 is a weighted ratio of viable to usable units, using the pre-coded employment on the D&B to weight the indicator variable. NRAF2 was used for estimates directly related to employment (e.g., the number of employees in a particular estimation cell). All other estimates used NRAF1 for the nonresponse adjustment weight.

As of May 2, 1988 the response rate experience was as follows:

Response rate=73.2%
Completion rate=61.5%

The response rate is defined as the number of usable cases divided by the number of viable cases. The completion rate is defined as the number of usable cases divided by the total number of cases contacted.

9.2 Item Nonresponse Adjustment/ Imputation

Often survey respondents do not know the answers to some questions or refuse to answer particular questions. In such cases, it is possible to fill in missing values using an imputation scheme. The idea is to use information from both the respondent (answers to other questions which they did supply) and information from other respondents (those answering the missing question) in order to estimate a reasonable response to the missing datum.

The imputation method chosen for the PEL survey is a hybrid method which combines the concepts of a mean imputation and a "hot-deck" imputation. A mean imputation method replaces the missing values on a certain question with the mean value from those respondents answering that question. A hot deck method attempts to find a respondent who matches the respondent having a missing value (in terms of other survey characteristics) and uses the value of the "twin" to replace the missing value. The method used here is a hybrid in the sense that it employs a mean imputation, but only over a small segment of the population which obviously matches the respondent having a missing value.

In particular, the procedure examines three or four digit SIC subgroups within the estimation cell by size class. The mean values of the responses to a particular question of interest in such sample subgroupings were used to impute the missing values in that grouping. In the case of categorical variables (for example, YES/NO questions), a randomization scheme was used which randomly supplied the appropriate set of responses to missing questions based on a probability distribution determined from those who responded.

It should be noted that the values which were placed on the database were not intended to be estimates of the missing reponses. Rather, they are meant to be substitute responses which allow the case to be used in the generation of survey estimates. In the aggregate, estimates produced using imputed data make sense, but may not for the individual establishment. Care was taken in the imputation program to be sure that imputed responses were consistent with other answers for the establishment of interest. Original responses to all questions were retained on the sample record and all responses representing imputed values were identified. One set of questions which was not imputed for was whether monitoring for the presence of certain toxic chemicals was done at the establishment. The data collected as of May 2, 1988 produced an estimate, for those establishments where chemicals or processes were found, that 14.5 percent did monitoring, 65.7 percent did not do monitoring, and 19.8 percent of respondents did not know or refused to answer the question. Of those establishments that did monitor, 25.6 percent provided the requested data.

10. Survey Instrument

As mentioned earlier, data collection for PEL survey was accomplished by Computer Assisted Telephone Interviewing. Prior to calling, a letter was sent to each selected establishment. This letter is shown in Exhibit 1–1. Also,

a hard copy version of the PEL questionnaire is given in Exhibit 1–2.

Exhibit 1–1

U.S. Department of Labor

Assistant Secretary for
Occupational Safety and Health
Washington, DC 20210

SIC Code 3479
Metal Coating & Allied Serv.
OMB Approval No. 1218–0142
February 25, 1988.

Mr. John Q. Sample,
Chairman, Anycompany, 123 Sample St., Anytown, US 12345

Dear Mr. Sample: The Occupational Safety and Health Administration (OSHA) of the U.S. Department of Labor is required by law to set permissible exposure limits for chemical substances in the workplace. Current exposure limits were set 17 years ago using values established by the American Conference of Governmental Industrial Hygienists (ACGIH) and the American National Standards Institute (ANSI).

OSHA has begun a process for revising out-of-date permissible exposure limits. To ensure that any new exposure limits take into account actual workplace conditions, we are conducting a voluntary survey of U.S. business establishments. Included will be questions about specific processes which we believe are performed in your industry and a limited (no more than 10 per process) list of chemicals which we believe are involved in those processes. Your facility was selected to be included in the study.

Decisions regarding new permissible exposure limits will be improved significantly if we have input from as many firms as possible. The interview will take about 30 minutes. Names of responding firms will not be associated with their answers, and all data will be treated as confidential by our contractor.

Please help us expedite the survey process by returning to us, within one week, the enclosed postage paid card with the name and phone number of the person in your organization our contract interviewer should contact. If this card is not received, a representative of our contractor, KCA Research, Inc., will call your office directly to conduct the interview or be directed to the company official designated by you.

Enclosed is a list of the topic areas for the survey. This may help in preparing for the interview.

We appreciate your cooperation and look forward to receiving the information we need from your designated representative.

Sincerely,

John A. Pendergrass,
Assistant Secretary for OSHA.

Enclosure

Topics Covered by Survey

I. *General Firm Characteristics*
- —Primary activity at this location
- —Approximate numbers of production & maintenance workers
- —Number of shifts per day and length of shift

II. *Identification of General Processes Performed by Firm*
- —Chemicals used in specific processes or operations and estimated quantities involved
- —Approximate number of work stations or assembly lines used and number of workers at each
- —Description of process engineering controls such as ventilation and enclosures
- —Estimated frequency of process or operation performance
- —Description of personal protective equipment used, including respirators, eye, face, and skin protection
- —Information regarding exposure monitoring

Exhibit 1–2

_____, we are conducting a survey on behalf of OSHA to assess the current practices of all types of businesses in the handling of toxic and hazardous chemicals. A letter was sent to you informing you of this survey.

1. Did you receive our letter?

1=Yes
2=No

If answer "Yes", begin next paragraph with "As you know,"

If answer "No", begin next paragraph with "I'm sorry. Let me summarize what the letter said about the survey".

We are interested in understanding all significant operations or processes in your firm that generate dust, mist, fumes, gases or vapor that your employees might potentially encounter. *Of course, all responses and trade or technological secrets will be kept strictly confidential and no company-specific information will be released to OSHA.*

2. Should I direct my questions to you, or is there someone else in the firm who you feel would be better qualified to answer?

1=Yes, this person will answer survey
2=No, call:
Name _____
Title _____
Phone _____
C=Call back (Set up time for recontact)
R=Refused to answer (Terminate interview)
D=Don't Know/No Response

Let me begin by asking some general questions about your facility.

3. Our records show your firm to be engaged in _____.? Is this correct? (Interviewer will read title or brief description for this SIC code.)

1=Yes
2=No, our function here is _____.
C
R
D

4. How many production workers do you have at this location?

1=_____ production workers
C=Call back
R=Refused to answer
D=Don't know

5. How many maintenance workers (for example; painters, welders & cleaning staff) do you employ?

1=_____ maintenance workers
2=Production workers do maintenance functions
3=None, only clerical, managerial, or sales personnel
C
R
D

5a. Of these maintenance workers, how many do painting as their primary work activity?

1=_____ do painting as primary activity
2=None
C
R
D

5b. Of these maintenance workers, how many do welding as their primary work activity?

1=_____ do welding as primary activity
2=None
C
R
D

6. How many shifts per day (24 hr. period) do you have at this location?

1=_____ shifts/24 hr.
C
R
D

I now want to ask you some questions about chemicals which we believe are common among firms in your industry. [These chemicals would be selected on the basis of large volume usage, known toxicity, or known exposure problems in excess of permissable limits as identified from NOES or IMIS or from industry expert opinion].

7. Which of the following chemicals are used, processed, or emitted at your facility?

Chemical A
1=Yes
2=No
C
R
D

[The interviewer will read chemical list specified for this 4-digit SIC. If "Don't Know" (D) is the response, the interviewer will then attempt to clarify the question by reading a list of common synonyms for the chemical. The subsequent answer can then be reassessed as "Yes" or "No"]

8. Are there any other chemicals in major use in your operations that I did not list?

1=Yes (Skip to #8 and add to list)
2=No
C
R
D

9. What is the approximate quantity of chemical A that your facility purchases each week or month?

1=_____ lbs. per week purchased
2=_____ gals. per week purchased
3=_____ lbs. per month purchased
4=_____ gals. per month purchased
C
R
D

Repeat Question #9 until all identified chemicals are quantified.

10. Have exposure limits been adopted by your firm for these chemicals?
1=Yes
2=No
C
R (Skip to #12)
D

11. What exposure limits have been adopted?
1=OSHA PEL's
2=NIOSH REL's
3=ACGIH TLV's
4=Other _____
C
R
D

The next questions are about processes/operations which we believe are common among firms in your industry.

12. Are any of the following processes/operations performed in your facility?
Operation #1
1=Yes
2=No
C
R
D

[Interviewer would read list of up to 6 processes or operations specified for this 4-digit SIC code. This list would be identified from secondary data sources and industry experts. If information regarding relevant processes was not available or sufficient, then this question would be rephrased to elicit process/operation identification from the respondent]

13. Are there any other processes/operations at your facility that I did not list?
1=Yes (Skip to #12 and add to list)
2=No
C
R
D

For each identified process/operation, ask questions 14–26.

14. In Process/Operation 1:
Is Chemical A used?
1=Yes
2=No
C
R
D

REPEAT UNTIL ALL IDENTIFIED CHEMICALS HAVE BEEN ASKED ABOUT USAGE IN THIS PROCESS.

15. How many work stations (or assembly lines) are involved in this process/operation?
1=_____ work stations
2=_____ assembly lines
C
R
D

16. On average, how many workers are directly involved in this process/operation at each work station (or assembly line)?
1=_____ workers/work station
2=_____ workers/assembly line
C
R
D

17. Of these workers, what percent work exclusively at this process/operation?
1=100% (Go to #18)
2=_____%
C
R (Go to #18)
D

17a. For those workers who do not work exclusively at this process/operation, in what other processes/operations are they also employed?
1=_____
C
R
D

18. Is this process/operation a completely enclosed activity?
1=Yes (Skip to #14)
2=No
C
R
D

19. Is this process/operation located outdoors?
1=Yes (Skip to #21)
2=No
C
R
D

20. Is this process/operation ventilated?
1=Yes
2=No
C
R (Skip to #21)

20a. What is the type of ventilation?
1=Local exhaust 1=Yes 2=No
C R D
2=General dilution
3=Natural ventilation
4=Other (specify type)

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21. How often is this process/operation performed during each shift?
1=Continuously over entire shift, every shift
2=Daily (specify #/day) _____
3=Weekly (specify #/week) _____
4=Monthly (specify #/month) _____
5=Yearly (specify #/year) _____
6=Other (specify #/period) _____
C
R
D

22. Are respirators routinely used by workers?
1=Yes
2=No (Skip to #23)
C
R
D

22a. What type of respirator?
1=Single use
2=Half-mask cartridge
3=Half-mask canister
4=Full-face cartridge
5=Full-face canister
6=Powered air purifying respirator
7=Air supplied respirator
8=Self-contained breathing apparatus
9=Escape respirator
10=Other _____
C
R
D

23. Do you provide maintenance workers who have exposure to this process with respirators?
1=Yes
2=No
C
R
D

24. Is skin, face, or eye protection used?
1=Yes
2=No (Skip to #25)
C
R
D

24a. What type(s), of skin, face, or eye protection?
1=Long sleeve shirt
2=Coverall
3=Apron
4=Gloves
5=Chemical Protective Clothing
6=Goggles
7=Face Shield
8=Other _____
C
R
D

25. Do you have a hazard communications training program for these workers?
1=Yes
2=No
C
R
D

26. Has environmental monitoring been done at or near this process/operation?
1=Yes
2=No
C
R (Skip to #14 until all processes surveyed)
D

26a. Has this monitoring been designed to evaluate control of:
1=potential short term (15 min.) exposures? (STEL)
2=potential 15 minute—4 hour exposures?
3=potential 4–8 hour exposures? (TWA)
C
R
D

26b. During this monitoring, were any chemicals found to be in excess of your adopted exposure guidelines?
1=Yes
2=No
C
R (Skip to #27)
D

26c. Which chemical(s) were found to exceed adopted guidelines?
1=_____
C
R (skip to #27)
D

26d. What activity, work process or operation do you feel is most responsible for the exposures above your adopted guidelines?

1=_____
2=Not able to specify
C
R
D

27. Can you give us your monitoring data for Process 1?

1=Yes
2=No
C
R (Skip to #14)
D

27a. What is the name of the first [next] chemical for which you have monitoring data?

1=_____
C
R
D

27b. Is the data for the work area or for the person (worker)?

1=Area
2=Person
C
R
D

27c. Is the data recorded for the individual worker or the work process?

1=Worker
2=Process
C
R
D

27d. Is the data reported for a short-term peak or an 8 hour TWA (Time Weighted Average)?

1=Short-Term Peak
2=TWA
C
R
D

27e. Is the unit of measurement parts per million or milligrams per cubic meter?

1=PPM
2=Mg/M^3
C
R
D

27f. What is the exposure data for this chemical?

1=_____
C
R
D

27g. Do you have exposure estimates for other chemicals used in this process?

1=Yes (Skip to 27a)
2=No
C
R (Skip to #14 until all processes surveyed)
D

28. What do you estimate to be the market value of plant and equipment at your facility?

1=Less than $50,000
2=$50,000–$500,000
3=$501,000–$1,000,000
4=$1 to $5 million
5=$5 to $50 million
6=More than $50 million
C
R
D

29. Can you estimate the annual value of shipments from your facility?

1=Less than $50,000
2=$50,000–$500,000
3=$500,000–$1,000,000
4=$1–$5 million
5=$5–$50 million
6=More than $50 million
C
R
D

Thank you for cooperating with us in our survey.

Supplements Nos. 2, 3, 4, 5, and 6

Copies of these Supplements are available upon request by calling or writing: Ms. Regina Flahie, Office of Regulatory Analysis, Room N3627, U.S. Department of Labor-OSHA, 200 Constitution Avenue NW., Washington, DC 20210, (202) 523–7283.

No. 2—Industry Processes and Chemicals, by Four Digit SIC, Identified in the 1988 Sample Survey

No. 3—Employee Exposures by Chemical and Industry, Based on Combined Data from OSHA's Integrated Management Information System and the 1988 Sample Survey

No. 4—Employee Exposures, by Chemical and Industry, Based Upon Data Collected in the 1988 Sample Survey

No. 5—Employee Exposures, by Chemical and Industry, Based Upon Data Contained in OSHA's Integrated Management Information System

No. 6—Hazardous Substance Exposure Data Linked to Industrial Processes Identified in the 1988 Sample Survey.

[FR Doc. 88–12213 Filed 6–6–88; 8:45 am]
BILLING CODE 4510–26–M