be considered in a closed meeting by authority of subsections (c)(2), (c)(8), and (c)(9)(B) of the "Government in the Sunshine Act" (5 U.S.C. 552b (c)(2), (c)(8), and (c)(9)(B)).

The meeting was held in the Board Room of the FDIC Building located at 550 17th Street, N.W., Washington, D.C.

Dated: February 1, 1990.

John M. Buckley, Jr.,

Executive Secretary.

[FR Doc. 90-2759 Filed 2-1-90; 5:10 pm.]

BILLING CODE 6714-01-M

Corrections

Federal Register
Vol. 55, No. 25
Tuesday, February 6, 1990

This section of the FEDERAL REGISTER contains editorial corrections of previously published Presidential, Rule, Proposed Rule, and Notice documents. 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 COMMERCE

National Oceanic and Atmospheric Administration

50 CFR Part 628

[Docket No. 900110-0010]

RIN 0648-AC51

Atlantic Bluefish Fishery

Correction

In proposed rule document 90-1960 beginning on page 2853 in the issue of Monday, January 29, 1990 make the following corrections:

1. On page 2853, in the first column, under **DATE**, in the second and third lines, "March 15, 1990" should read "March 12, 1990".

2. On page 2854, in the second column, in the third paragraph, in the fifth line, "10" should read "0".

BILLING CODE 1505-01-D

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Office of Human Development Services

Federal Council on the Aging; Meeting

Correction

In notice document 90-2360 appearing on page 3489 in the issue of Thursday, February 1, 1990, make the following correction:

On page 3489, in the second column, the signature and title were inadvertently omitted. They should read as set forth below;

Ingrid Azvedo,

Chairperson, Federal Council on the Aging.

BILLING CODE 1505-01-D

NUCLEAR REGULATORY COMMISSION

10 CFR Part 35

RIN 3150-AC65

Basic Quality Assurance Program, Records and Reports of Misadministrations or Events Relating to the Medical Use of Byproduct Material

Correction

In proposed rule document 90-821 beginning on page 1439 in the issue of Tuesday, January 16, 1990, make the following corrections:

1. On page 1439, in the second column, in footnote one, in the fourth line, "the human beings" should read "to human beings".

2. On page 1440, in the first column, in the heading for table 1, in the first line, "Therapy" was misspelled.

3. On page 1441, in the first column, in the last complete paragraph, in the next to last line, "voluntary" was misspelled.

4. On the same page, in the third column, in the first complete paragraph, the ninth line should read, "impact and efficacy of the proposed".

5. On page 1443, in the first column, in the second paragraph, in the fourth line "misadministration" should read "misadministrations".

6. On the same page, in the 2nd column, in the 13th line, "implies" was misspelled.

7. On the same page, in the same column, in the last complete paragraph, in the 9th line, "fractional" was misspelled, and in the 11th line "doze" should read "dose".

8. On the same page, in the same column, in the same paragraph, in the 17th line, "(§ 35.34(b)(i))" should read "(§ 35.34(b)(3)(i))", and in the next to last line "fractions" was misspelled.

9. On page 1444, in the first column, in the last complete paragraph, in the ninth line, "administration" should read "misadministration".

10. On page 1445, in the first column, in the next to last line, "also"should read "already".

11. On page 1446, in the first column, in the last paragraph, in the sixth line, after "per" insert "year per".

12. On page 1447, in the first column, under **IX. Text of Proposed Regulation**, in the fifth line, "5 U.S.C. 533"should read "5 U.S.C. 553".

BILLING CODE 1505-01-D

DEPARTMENT OF THE TREASURY

Internal Revenue Service

26 CFR Parts 1 and 602

[T.D. 8282]

RIN-1545-A023

Election of Reduced Research Credit; Income Taxes

Correction

In rule document 90-1520 beginning on page 2374 in the issue of Wednesday, January 24, 1990 make the following corrections:

1. On page 2375, in the second column, in the first complete paragraph, in the 10th line, "280(c)(3)" should read "280C (c)(3)".

§1.280C-4 [Corrected]

2. On page 2376, in the second column, under § 1.280C-4(b)(2), in the second line, "41(b)" should read "41(h)".

BILLING CODE 1505-01-D

Corrections

federal register

Tuesday
February 6, 1990

Part II

Department of Labor

Occupational Safety and Health Administration

29 CFR Part 1910

Occupational Exposure to Cadmium; Proposed Rule

DEPARTMENT OF LABOR

Occupational Safety and Health Administration

29 CFR Part 1910

[Docket No. H-057a]

RIN 1218-AB16

Occupational Exposure to Cadmium

AGENCY: Occupational Safety and Health Administration (OSHA), DOL.

ACTION: Proposed rule and notice of hearing.

SUMMARY: The Occupational Safety and Health Administration (OSHA) proposes to amend its existing regulation for occupational exposure to cadmium in the general, construction, agriculture and maritime industries at 29 CFR 1910.1000, Table Z-2, 29 CFR part 1926, 29 CFR part 1928, and 29 CFR 1910.252 (f)(1)(v) and (f)(9). The basis for issuance of this proposal is a preliminary determination by the Assistant Secretary that employees exposed to cadmium face a significant risk to their health at the current permissible exposure limits and that promulgating this proposed standard will substantially reduce that risk. The information gathered so far in this rulemaking demonstrates that employees chronically exposed to levels of cadmium well below the permissible exposure limits are at increased risk of developing kidney dysfunction and cancer. This notice proposes two 8-hour time-weighted average permissible exposure limits (TWA PEL) of 5 and 1 micrograms of cadmium per cubic meter of air as alternatives for all cadmium compounds. OSHA also proposes an excursion limit (EL), measured over a fifteen minute period for all cadmium compounds, of five times the TWA PEL. For a TWA PEL of 5 $\mu g/m^3$ the EL is 25 $\mu g/m^3$. For a TWA PEL of 1 $\mu g/m^3$, the EL is 5 $\mu g/m^3$. In addition, OSHA proposes to set an action level (TWA) of 2.5 $\mu g/m^3$ for a TWA PEL of 5 $\mu g/m^3$ and an action level of 0.5 $\mu g/m^3$ for a TWA PEL of 1 $\mu g/m^3$.

OSHA proposes to require other ancillary provisions for employee protection such as exposure monitoring, medical surveillance, recordkeeping, regulated areas, emergency procedures, preferred methods to control exposure, hazard communication, and proper selection and maintenance of personal protective equipment. OSHA proposes to regulate occupational exposure in general industry, agriculture, the maritime industry and the construction industry.

DATES: Written comments on the proposed standard must be postmarked on or before April 27, 1990. Notices of Intention to Appear at one of the informal hearings must be postmarked on or before April 4, 1990.

Parties requesting more than 10 minutes for their presentation at the hearings and parties submitting documentary evidence at the hearing must submit the full text of their testimony and all documentary evidence no later than April 27, 1990.

The informal hearing will begin at 9:30 a.m. on the first day of the hearing. The informal hearing will begin on June 5, 1990, in Washington, DC, and will continue on July 17, 1990, in Denver, Colorado.

ADDRESSES: Four copies of the notice of intention to appear, testimony and documentary evidence which will be introduced into the hearing record must be sent to Mr. Tom Hall, Division of Consumer Affairs, Occupational Safety and Health Administration, Room N-3647, U.S. Department of Labor, 200 Constitution Ave., NW., Washington, DC 20210. For complete instructions on filing a Notice of Intention to Appear, see below, Section XII, Public Participation—Notice of Hearing.

The hearing beginning on June 5, 1990 will be held in the Departmental Auditorium in the Frances Perkins Building, U.S. Department of Labor, 200 Constitution Ave., NW., Washington, DC 20210. The hearing continuing on July 17, 1990, will be held in the Cripple Creek/Silver Heels Room, Holiday Inn, 1450 Glen Arm Place, Denver, Colorado 80202.

Written comments must be submitted in quadruplicate to the Docket Officer, Docket No. H-057a, Room N-2625, U.S. Department of Labor, 200 Constitution Ave., NW., Washington, DC 20210, (202) 523-7075.

FOR FURTHER INFORMATION CONTACT: Hearing: Mr. Tom Hall, Division of Consumer Affairs, Occupational Safety and Health Administration, U.S. Department of Labor, Room N-3647, 200 Constitution Ave., NW., Washington, DC 20210, (202) 523-8615.

Proposal and Hearing Issues: Mr. James F. Foster, Occupational Safety and Health Administration, U.S. Department of Labor, Room N3647, 200 Constitution Ave., NW., Washington, DC 20210. (202) 523-8151.

SUPPLEMENTARY INFORMATION:

I. Introduction

A. The Format of this Document (The Preamble)

The preamble and the accompanying proposed standard are divided into 14 parts, numbered I-XIV. The following is a table of contents.

Table of Contents

I. Introduction
II. History of the Regulation
III. Pertinent Legal Authority
IV. Chemical Identification, Production, and Use of Cadmium
V. Health Effects
VI. Preliminary Quantitative Risk Assessment
VII. Significance of Risk
VIII. Summary of the Regulatory Impact and Flexibility Analysis
IX. Environmental Impact Assessment
X. Summary and Explanation of the Proposed Standard
XI. Clearance of Information Collection Requirements
XII. Public Participation—Notice of Hearing
XIII. Authority and Signature
XIV. Proposed Standard

B. Summary

This preamble to the proposed standard on occupational exposure to cadmium discusses the events leading to the proposal, the physical properties, manufacture and use of cadmium, the health effects of exposure, and the degree and the significance of the risk. In addition, an analysis of the regulatory impact and technological and economic feasibility of the proposed standard and the rationale behind the specific provisions set forth in the regulatory text are also presented. Public comment on all matters discussed in this notice and all other relevant issues is requested for the purpose of assisting OSHA in the development of a final standard for occupational exposure to cadmium.

C. Issues

Comment is requested on all relevant issues, including health effects, technological and economic feasibility and provisions that should be included in a final cadmium standard. OSHA is proposing, as one alternative, a time-weighted average permissible exposure limit (TWA PEL) of 5 $\mu g/m^3$, with an action level of 2.5 $\mu g/m^3$. Alternatively, OSHA is proposing a TWA PEL of 1 $\mu g/m^3$, with an action level of 0.5 $\mu g/m^3$. Comment is requested on these TWA PELs and on other possible TWA PELs ranging from 0.5 $\mu g/m^3$ to 40 $\mu g/m^3$. In proposing a TWA PEL of 1 $\mu g/m^3$, OSHA acknowledges that respirator usage could be required, at least part-time, for up to 37% of workers in industries and occupations with cadmium exposures. This anticipated reliance on respirators is greater than in previous OSHA rulemakings. OSHA has specifically addressed the issue of respirator usage in the questions below.

In proposing two alternative PELs, OSHA acknowledges that either PEL would be difficult to achieve in some sectors through engineering controls alone. In these latter industry and occupational sectors, reliance upon respirators, as stated above, would be considerable in order to achieve a PEL of 1 $\mu g/m^3$. Much less respirator use would be required to achieve a PEL of 5 $\mu g/m^3$. Whether or not the additional burden of respirator use needed to achieve a PEL of 1 $\mu g/m^3$ is justified depends, in part, upon the accuracy of the data used in the risk assessments. As discussed in the Significance of Risk section of the preamble, the Agency recognizes the uncertainty inherent in quantitative risk assessments and in the extrapolation of risks from animals to humans for estimating carcinogenic potential. Given these uncertainties, OSHA requests comments on whether a PEL of 5 $\mu g/m^3$ may be low enough to appropriately mitigate the risk of cancer.

OSHA seeks comment on the alternatives. Hereinafter, OSHA will refer to the alternative TWA PELs as simply 1(5) $\mu g/m^3$. OSHA will refer to the alternative excursion limits (ELs) of 5 $\mu g/m^3$ for a TWA PEL of 1 $\mu g/m^3$ and 25 $\mu g/m^3$ for a TWA PEL of 5 $\mu g/m^3$ as 5(25) $\mu g/m^3$, respectively. OSHA will refer to the alternative action levels as 0.5(2.5) $\mu g/m^3$ for TWA PELs of 1 and 5 $\mu g/m^3$, respectively.

OSHA is especially interested in answers, supported by evidence and reasons, to the following questions.

1. Do OSHA's proposed TWA PELs of 1 $\mu g/m^3$ and 5 $\mu g/m^3$ substantially reduce a significant risk?

2. Are the proposed TWA PELs of 1 $\mu g/m^3$ and 5 $\mu g/m^3$ technologically and economically feasible? Is there evidence other than that presented by OSHA regarding economic and technological feasibility of the proposed PELs?

3. Should a TWA PEL other than 1 $\mu g/m^3$ or 5 $\mu g/m^3$ be adopted? If so, what level, for example 0.5 $\mu g/m^3$, 10 $\mu g/m^3$, 20 $\mu g/m^3$, or 40 $\mu g/m^3$, should be established? Please provide evidence for establishing a lower or higher TWA PEL.

4. Should OSHA revoke the ceiling limits in 29 CFR 1910.1000, Table Z-2 and replace them with a single excursion limit (EL)? If so, should the EL be a 15-minute limit of 5 $\mu g/m^3$, 25 $\mu g/m^3$ or some other alternative?

5. Are the proposed action levels of 0.5 $\mu g/m^3$ and 2.5 $\mu g/m^3$ (as an 8 hour TWA) the appropriate levels for the TWA PELs under consideration? If not, at what level should the action level be set? For instance, although OSHA is proposing an action level of 2.5 $\mu g/m^3$ for a TWA PEL of 5 $\mu g/m^3$, OSHA is also considering an action level of 1 $\mu g/m^3$ for a TWA PEL of 5 $\mu g/m^3$ due to concerns for workers with high past exposures to cadmium. OSHA is also considering special provisions for medical surveillance for these veteran workers. If there were no action level, which provisions currently proposed to be triggered by an action level should be made applicable to cadmium-exposed employees?

6. What is the risk of developing cancer, kidney dysfunction and other adverse health effects that might arise from exposure to cadmium at OSHA's current PELs? What is the risk from exposure to TWA PELs of 0.5 $\mu g/m^3$, 10 $\mu g/m^3$, 20 $\mu g/m^3$ and 40 $\mu g/m^3$? Are there different estimates of risk of adverse health effects, in specific industries or occupations? Are there estimates of risk from quantitative risk assessments other than those used by OSHA, using the same or alternate TWA PELs?

7. How many workers are exposed to cadmium? What are the jobs being performed and their current exposure levels? Please provide individual air cadmium monitoring results for the past 3 years by job classification on a plant-by-plant basis (or, if not available by job classification, by operation). Please accompany the provision of industrial monitoring results with a description of associated engineering and work practice controls, organized on a plant-by-plant, job-by-job, or operation-by-operation, basis.

8. Where exposures are intermittent, what is the duration, frequency and level of exposure? What jobs involve intermittent exposures?

9. What industries (provide Standard Industrial Classification or SIC Codes and descriptions) and processes use cadmium?

10. Should this standard cover the construction industry? If so, should it differ from the current proposal? If so, how? Should the standard cover the agriculture and maritime industries? If so, should it differ and how?

11. What is the lowest level of cadmium exposure achievable by engineering and work practice controls? Please support your answer with a discussion of current exposure levels, current controls, other available controls, efficiency of various controls in reducing exposure levels, and the costs and the time needed for implementation of those controls. For example, can cadmium exposures be reduced by present technologies and work practices to a TWA PEL of 0.5 $\mu g/m^3$?

12. What would be the capital and operating costs required to achieve the proposed TWA PELs and the proposed ELs? Are these costs economically feasible for the affected industries? How, if at all, would extending compliance deadlines affect costs and feasibility?

13. How, if at all, would the costs and economic feasibility of achieving the TWA PEL be affected if the TWA PEL were 0.5, 10, 20 or 40 $\mu g/m^3$ rather than 1 $\mu g/m^3$ or 5 $\mu g/m^3$? To what extent should the degree of respirator usage required to achieve the TWA PEL be considered in determining feasibility?

14. Are there conditions under which respirator use should be permitted in addition to those proposed? What respirator fit testing requirements should be included in a final standard and when should such testing be required? Should respirators be used at all times in regulated areas?

15. Are there any unique conditions in work settings where cadmium is produced or used that make engineering controls infeasible?

16. Have there been any recent technological improvements or changes in the production or use of cadmium for the purpose of improving productivity or product quality that have also resulted in reduction in cadmium exposures?

17. What measurement and analytical methods, in addition to the methods in Appendix E, are available for use in determining compliance with a cadmium exposure limit or action level of less than 0.5 $\mu g/m^3$? Are there sufficient laboratories available to accurately and precisely determine cadmium in air levels? What recommendations, if any, are there for standardizing or otherwise assuring the quality of laboratories that perform these determinations? Is the NIOSH system for standardizing laboratories adequate?

18. Under this proposal, industry has the option of relying upon "objective data" instead of air cadmium monitoring results to document the fact that employees are not exposed at or above the action level. Is this appropriate? What evidence, other than air cadmium monitoring results, should qualify as "objective data" and what, if any, limitations should be put on the use of such data?

19. Under this proposal, two consecutive air monitoring measurements taken at least 7 days apart are required to demonstrate the lowered exposure levels necessary to reduce the frequency of monitoring. Should this time be changed to a longer length of time? If so, how long should this time period be? Should the same minimum length of time between consecutive exposure monitorings apply

to workplaces independently of past exposures?

20. The proposed standard includes requirements for medical surveillance, respirators, personal protective clothing and equipment, hygiene facilities and practices, regulated areas, maintenance of records, housekeeping, employee information and training, and labels and signs. What form should they take? Should these requirements be included in a final standard? To what extent are these provisions currently being employed by industry and what are their costs?

21. Are the proposed medical surveillance provisions inadequate, adequate, or too extensive? Please make specific recommendations and provide rationale. Should the schedule for biological monitoring of urine and blood be more frequent than the schedule for the full medical examination? Are annual pulmonary function tests necessary or can they be conducted less frequently and still provide appropriate protection?

22. What is the most accurate and earliest biological indicator of overexposure to cadmium that can be used to detect preclinical manifestations of kidney disease?

23. Is it reasonable and feasible to use a quantitative measurement of low molecular-weight proteins as a workplace screening method for early detection of cadmium-related kidney dysfunction? If so, is one method preferred over another (e.g. retinol-binding proteins vs. B–2 microglobulins)? What are feasible testing schedules? Please address specifically:

a. Sensitivity and specificity of tests;
b. Any logistical problems associated with collecting, transporting, or analyzing specimens;
c. Issues of feasibility and practicability, including cost;
d. Means for standardizing these tests such that results may be meaningfully interpreted.

24. What are the existing biological indicators of overexposure to cadmium? Which of these are useful in determining overexposure prior to the development of cadmium related kidney dysfunction? Are there sufficient laboratories available with the technology to quantify these indicators for all workers exposed to cadmium at or above the proposed action level? Are these indicators sensitive and specific for cadmium-related dysfunction?

25. Are cadmium levels in the blood and/or urine, commonly used at present, useful indicators of overexposure to cadmium? If so, what levels indicate overexposure to cadmium? Are there sufficient laboratories available with the technology to precisely and accurately determine biological levels of cadmium in blood and urine? Are there recommendations for standardizing or otherwise assuring the quality of the laboratories that perform these determinations? Should specific concentrations of cadmium in the blood and/or in the urine be used to trigger medical removal? If so, what trigger levels for removal are recommended and under what circumstances and on what basis, if any, should the worker be returned?

26. Regarding current work practices for medical removal:

a. What are the current practices for removing workers overexposed to cadmium?
b. What specific biological indicators and what levels are currently being used?
c. For workers who have been removed from cadmium exposed jobs because of overexposure to cadmium, what types of jobs were they given and how long did they remain off the job from which they were removed?
d. Under what circumstances are removed workers returned?
e. On what basis should determinations for returning employees to work be made?
f. What material benefits are these employees receiving, if any, while they are medically removed from work?

27. Are the proposed provisions for temporary medical removal of any employee from exposure at or above the proposed action level when that employee has proteinuria indicative of cadmium toxicity necessary and appropriate? Please address specifically:

a. What advice should be given to workers who manifest tubular proteinuria?
b. Is tubular proteinuria an indication for medical removal?
c. If so, at what specific levels of what specific proteins should workers be removed?
d. If a recommendation is made for medical removal based on tubular proteinuria, under what circumstances, if any, should the worker be returned?
e. On what basis should the determination to return an employee be made?

28. Regarding medical removal:

a. Are the proposed time periods for medical removal adequate?
b. If benefits are needed, what benefits should an employee have while medically removed from work due to adverse health effects of cadmium overexposures?

29. Are the provisions for medical removal of employees who have difficulty breathing while wearing a respirator or during the fit test for respirator usage necessary and appropriate? Under what circumstances and at what levels of lung function loss should a worker be removed for inability to wear respirators? How long should a worker be removed? Can the worker return to work and if so, when? Who should make these decisions and on what criteria?

30. Should employees who have one or more years of occupational exposure to cadmium be treated differently from new employees? If so, in what ways should they be treated differently in order to protect them? Should there be differences in medical surveillance and removal?

31. Laboratory testing with cadmium has demonstrated adverse reproductive effects in animals. Please address specifically:

a. Are there implications for human reproductive effects from workplace exposures?
b. If so, should policies be adopted to address these concerns?
c. What are current practices in considering placement of workers of reproductive age, male and female, in jobs with relatively high exposure to cadmium?

32. Provisions have been included in the proposed standard for medical evaluations at termination of employment of all employees who have been eligible for annual evaluations. OSHA requests comments on all aspects of these provisions, including the potential uses and abuses of such examinations by employers or employees.

33. For the last five years in your plant and industry:

a. What were the total annual volumes and dollar values of production, shipments, and inventories?
b. What was the total annual investment categorized as replacement, expansion, modernization, environmental and health and safety?
c. What were the retained earnings, after tax income, total assets, stockholders' equity, net worth, depreciation charges, and debt-equity, ratios?
d. What were the total annual employment levels and labor turnover for the industries with

cadmium exposures?

OSHA and JACA have performed detailed feasibility analyses for the industry sectors where the impact of this standard would be significant. OSHA believes that the impact in other industrial segments would not be substantial. Comments are requested from all industry segments that may be significantly affected.

34. The cadmium record includes copies of the preliminary Regulatory Impact Analysis, which presents OSHA's feasibility analyses, and the JACA report. Comments are requested on these analyses and on the feasibility and cost effectiveness of alternative PELs, for example, 0.5, 10, 20 or 40 $\mu g/m^3$.

35. The following information is requested from small businesses so that OSHA can better evaluate the impacts of the proposed standard on these organizations and, where appropriate, adapt proposed requirements to take into account their circumstances:

a. What kinds of small businesses or organizations and how many of them would be affected by this proposal?
b. Which, if any, federal rules may duplicate, overlap, or conflict with this proposal?
c. What difficulties will be encountered by small entities when attempting to comply with requirements of the proposed standard? Can some of the requirements be deleted or simplified for small entities, while still achieving comparable protection for their employees?
d. What timetable would be appropriate to allow small entities sufficient time to comply?

36. Please submit any information, data or comments pertaining to possible environmental impacts of this proposal, such as the following:

a. Any positive or negative environmental effects that could result should the proposal be adopted;
b. Beneficial or adverse relationships between the human environment and productivity;
c. Any irreversible commitments of natural resources which could be involved should the proposal be adopted; and
d. Estimates of the degree of reduction of cadmium in the environment effected by the proposed OSHA standard.

In particular, consideration should be given to the potential direct or indirect impacts of the proposal on water and air pollution, energy usage, solid waste disposal, or land use.

37. For which industrial processes are there substitutes for cadmium that are less toxic?

38. OSHA understands that several factors may mean that delay in implementation of the standard is warranted and requests comments on how much time should be allowed before compliance is required. The relevant factors may include time to allow laboratories to standardize their environmental and biological testing and time needed to improve engineering controls.

II. History of the Regulation

A. OSHA's Current PELS

OSHA's present permissible exposure limits were originally developed by the American National Standards Institute. In 1941 the American Standards Association (now American National Standards Institute, or ANSI) set as guidelines an American Defense Emergency Standard of 1000 $\mu g/m^3$ for cadmium and its compounds. This was done to reduce discomfort from exposures to cadmium and to reduce the incidence of acute health effects. ANSI revised its standard to current levels (ANSI Z37.5, 1970) which OSHA adopted in 1971 as a national consensus standard under section 6(a) of the Occupational Safety and Health Act of 1970 (29 U.S.C. 655). OSHA's current PELs, as specified in 29 CFR 1910.1000, Table 2–2 are an 8-hour time-weighted average (TWA PEL) of 100 $\mu g/m^3$ for cadmium fume with a ceiling concentration of 300 $\mu g/m^3$ and an 8-hour TWA of 200 $\mu g/m^3$ for cadmium dust with a ceiling concentration of 600 $\mu g/m^3$. OSHA's existing TWA PEL in the construction industry is 100 $\mu g/m^3$ for cadmium oxide fumes (29 CFR 1910.1926.55).

B. Other Recommendations

In preparing this document, OSHA reviewed the existing regulations for occupational exposures to cadmium in other countries worldwide. The range of existing permissible exposure limits runs from the ban of all non-essential uses of cadmium in Sweden to OSHA's existing TWA PEL of 200 $\mu g/m^3$ for cadmium dust.

Agencies and institutions other than OSHA have revised their air quality standards for cadmium. In 1976, the National Institute for Occupational Safety and Health (NIOSH) recommended that exposures to any form of cadmium should not exceed a concentration greater than 40 $\mu g/m^3$ as a 10-hour TWA or a concentration greater than 200 $\mu g/m^3$ for any 15-minute period. This recommended limit was intended to protect against renal damage and pulmonary disease. In 1984, NIOSH issued a Current Intelligence Bulletin (CIB), which recommended that cadmium and its compounds be regarded as potential occupational carcinogens based on evidence of lung cancer in workers exposed to cadmium in a smelter.

The Environmental Protection Agency (EPA) issued a Health Assessment Document (HAD) for cadmium in 1981 which presented the health effects and potential risk to human health associated with environmental exposure to cadmium. An update of the HAD in 1985 concluded that the epidemiologic evidence is suggestive of a significant risk of lung cancer from exposure to cadmium. According to the EPA's 1984 Proposed Guidelines for Carcinogenic Risk Assessment, cadmium is classified as a Group B1 substance and is thus considered to be a "probable" human carcinogen (Ex. 4–04).

In 1987, the International Agency for Research on Cancer (IARC) of the World Health Organization (WHO) summarized the results from tests for genetic and related effects of a large number of compounds thought to be potentially carcinogenic. The IARC working group of experts evaluated these data as well as epidemiologic and animal studies and concluded that cadmium and cadmium compounds should be classified in Group 2A—"probably carcinogenic to humans" (Ex. 8–681).

Since 1946, the American Conference of Governmental Industrial Hygienists (ACGIH) has recommended that exposures to cadmium be controlled. In 1946, ACGIH recommended a Maximum Allowable Concentration (MAC) of 100 $\mu g/m^3$ for cadmium. After 1948, the MAC was called the Threshold Limit Value (TLV). In 1956, a TLV of 100 $\mu g/m^3$ was assigned to cadmium oxide fume. In 1965, a value of 200 $\mu g/m^3$ for cadmium (metal dusts and soluble salts) was proposed; it was adopted as a recommended value in 1967. In 1970, the ACGIH TLV of 200 $\mu g/m^3$ for cadmium dust and salts remained unchanged, but the TLV for cadmium fume was changed to a ceiling. In 1973, the ACGIH announced its intent to change the TLV for cadmium fume to 50 $\mu g/m^3$ and in 1974 announced its intent to extend this TLV to cadmium dusts and salts. A note was added in 1975 indicating that cadmium oxide production involved carcinogenic or co-carcinogenic potential.

Recently, the ACGIH has recommended further changes in air quality standards for cadmium. They have classified cadmium as a potential human carcinogen and published a Notice of Intent to lower the TLV to 10 $\mu g/m^3$ to protect workers from lung cancer. ACGIH justified this latest change by noting:

> In consideration of the strength of the white rat inhalation studies and with some additional support from the retrospective human mortality study by Thun et al., an $A_2$2 designation as an industrial substance of carcinogenic potential for man is given to cadmium and its compounds (Ex. 8–664).

ACGIH's TLVs are used as guidelines universally. For instance, OSHA used many of the ACGIH TLVs as starting points for the recent Air Contaminants Standards, (54 PA 2332, January 19, 1989), which updated some of the Agency's permissible exposure limits. OSHA has used the ACGIH TLV on cadmium in its current guidelines on interim exposure limits for occupational exposures to cadmium (OSHA Instruction PUB 8–1.4A, 9/26/88, Ex. 8–676).

Unlike ACGIH, however, OSHA must, as part of the overall significant risk determination under section 6(b) rulemaking, consider other factors including all relevant health information, the underlying data, the reasonableness of its risk assessment, and the statistical significance of the findings and the significance of the risk. OSHA's cancer risk assessment, based on human and animal data, indicates that at a TWA PEL of 10 $\mu g/m$ or 1 $\mu g/m$, a significant risk of excess cancer deaths may remain although, as will be discussed, there are uncertainties as to the true risk at these levels.

The ACGIH has for several years been in the process of lowering its TLV of 50 $\mu g/m$ to 10 $\mu g/m^3$ in order to protect workers from lung cancer and kidney damage. It reached this decision partially on the basis of results from a mortality study of cadmium smelter workers (Thun et al., Ex. 4–68). OSHA understands that most recently, ACGIH has been considering levels below 10 $\mu g/m^3$.

The Mine Safety and Health Administration (MSHA) publishes air quality standards which include cadmium. MSHA frequently incorporates, by reference, the ACGIH TLVs as permissible exposure levels. Currently, MSHA is in the process of revising these levels to take account of proposed ACGIH changes in the TLVs. MSHA recently published a Notice of Proposed Rulemaking (54 PA 35760; August 29, 1989), proposing alternative TWA PELs of 10 $\mu g/m$ and 5 $\mu g/m^3$ for cadmium. Under this process, MSHA is seeking comments on the applicability of the ACGIH TLVs for cadmium.

Since 1987, the National Center for Health Statistics (NCHS), Department of Health and Human Services, Center for Disease Control, has included cadmium-in-urine measurements in its current third National Health and Nutrition Examination Survey (NHANES III). This survey, originally started in 1974, provides national estimates of diagnosed and undiagnosed medical conditions, as well as information on normal and abnormal conditions in the general population of the U.S. Such information is used by government agencies to obtain a more complete picture of national health and medical needs (Ex. 8–679). OSHA considers the inclusion of cadmium by NCHS to indicate a high level of concern regarding cadmium-related health effects among the general population, which experiences lower cadmium exposures than most occupational groups.

C. OSHA's Current Proposal

OSHA's current proposal to reduce OSHA's PELs for cadmium exposures is in response to a petition, in 1986, by the Public Citizen Health Research Group (HRG) joined by the International Chemical Workers Union (ICWU). HRG and ICWU petitioned OSHA to issue an Emergency Temporary Standard (ETS) for cadmium providing for a permissible exposure limit (PEL) of 1 $\mu g/m^3$ as an 8-hour TWA and a 5 $\mu g/m^3$ ceiling limit. In support of their position the petitioners cited several studies which they believed provided evidence that workers were in grave danger from occupational exposure to cadmium at and below current PELs. The major human study cited was that by Thun *et al.* (Ex. 4–68), which found significant increases in lung cancer in cadmium smelter workers. The petitioners also cited several animal studies that demonstrate the carcinogenic potential of cadmium. The most notable of these was an inhalation study cited in which rats exposed to cadmium chloride at levels below OSHA's PEL, developed lung cancer while the unexposed controls developed none (Ex. 4–67). Other human studies cited by the petitioners showed statistically significant increases in prostate cancer among battery factory, smelter, and alloy factory workers exposed to cadmium. Other human studies cited by the petitioners also showed evidence of renal damage and non-malignant respiratory disease among workers exposed to cadmium at levels below the PEL. The exposure limits requested by HRG and ICWU were aimed at ensuring that workers would not be at excess risk of cancer and kidney disease.

On July 1, 1987, OSHA denied the Public Citizen HRG and ICWU petition for an ETS, based on it's determination that the record did not support findings that cadmium posed a grave danger as defined by the courts. However, OSHA determined that the current PELs were not sufficiently protective and that the Agency would proceed with permanent rulemaking under section 6(b) of the Act to reduce cadmium exposure.

Under this Act, as part of the overall significant risk determination, 05HA must consider many factors. OSHA's risk assessment indicates a significant cancer risk may exist in the range from 10 $\mu g/m^3$ to 1 $\mu g/m^3$. Based on the animal data, the TWA PEL proposed by 0SHA should be at least as low as 1 $\mu g/m^3$. Over 45 years, this amounts to an overall lifetime occupational exposure of 45 $\mu g/m^3$-years. Although the human data on lung cancer in cadmium smelter workers lead to a lower estimate of cancer risk than does the animal data, analysis of the human data suggests that a significant risk may remain at a PEL of 5 $\mu g/m^3$.

In addition to the carcinogenic potential, cadmium exposure is associated with adverse kidney effects. Based on Kjellstrom's study published in 1977 (Ex. 8–664), preclinical kidney dysfunction (defined as urinary B_2-microglobulin concentrations greater than 290 $\mu g/L$) was observed in 19% of employees with average exposures of 50 $\mu g/m^3$ to cadmium oxide dust for an average of 9 years (or 450 $\mu g/m^3$-years). Only three percent of controls had this level of kidney dysfunction, which gives a relative risk (RR) of 6.3 for kidney dysfunction at exposures of 450 $\mu g/m^3$-years (19% divided by 3% times 100). Kjellstrom's findings are within the range of risks predicted by 0S in its risk assessment.

The ACGIB has characterized this level of kidney dysfunction as follows:

> "Persons excreting 290 $\mu g/L$ B2-microglobulin per liter of urine are not disabled; indeed they will not experience any symptoms. However, the lesion is irreversible and represents a permanent loss of functional reserve. An infection or other condition which compromises renal function, but which would not normally lead to serious illness, could overwhelm the remaining kidney capacity (Ex. 8–664)."

OSHA considers this dysfunction to represent material impairment of health. OSHA's risk assessment predicts an unacceptably high level of kidney dysfunction among workers exposed to

a lifetime occupational cadmium exposure of 10 $\mu g/m^3$. In its quantitative risk assessment, OSHA presents its quantitative risk assessment of both cancer and kidney effects and requests comments on all aspects of this assessment.

In keeping with the recommendations of other federal agencies (e.g., NIOSH and EPA) and the ACGIH' this proposal does not differentiate between fumes and dust. In earlier recommendations, a distinction was made between exposures to fumes and dusts. Since the early 1940's, acute inhalation of cadmium fumes from soldering or welding was known to cause severe health effects such as chemical pneumonitis and death (Ex. 8–678). These properties led researchers to readily e,cept the possibility of adverse health effects associated with exposure to fumes. Now, however, it is generally accepted that overexposures to cadmium in any form results in the same final chronic endpoints, cancer and kidney dysfunction (Exs. 4–27, 4–28, 4–68, and 4–19). By 1970, when ANSI republished their original guidelines, it acknowledged that exposures to cadmium fumes *or* dusts cause irreversible lung damage, proteinuria, and kidney damage. In the mid-1970's the ACGIH announced an intent to change the TLV for all cadmium compounds (fumes, dust, and salts) to 50 $\mu g/m^3$, and the differentiation between fumes and dusts was set aside.

III. Pertinent Legal Authority

This proposed standard and the issuance of a final standard are authorized by sections 6(b), 8(c), and 8(g) (2) of the Occupational Safety and Health Act of 1970 (the Act), (84 stat. 1593; 29 U.S.C. 655(b), 657(g) (2)). 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 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.

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 that under the Act 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" (488 U.S. at 642). The Court also stated "that the Act does limit the Secretary's power to requiring the elimination of significant risks" (448 U.S. at 644, n. 49).

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 overprotection rather than underprotection" (448 U.S. at 655, 656). The court also stated that "while that 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." (448 U.S. at 655, 656, n. 62).

After OSHA has determined that a significant risk exists and that such risk can be reduced by the proposed standard, it must set a 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 * * * " (Section 6(b)(5) of the Act). The Supreme Court has interpreted this section to mean that OSHA must enact the most protective standard possible to eliminate a significant risk of material health impairment, subject to the constraints of technological and economic feasibility. *American Textile Manufacturers Institute, Inc.* v *Donovan*, 452 U.S. 490 (1981). The court held that "cost-benefit analysis is not required by the statute because feasibility analysis is." (452 U.S. at 509). The Court stated that the Agency could use cost-effective analysis and choose the least costly of two equally effective standards. (452 U.S. 531, n. 32).

Authority to issue this proposed standard is also found in section 8(c) and (g) of the Act. Section 8(c)(3) gives the secretary authority to require employers to "maintain accurate records of employee exposures to potentially toxic materials or harmful physical agents which are required to be monitored or measured under section 6." Section 8(g)(2) gives the Secretary authority to "prescribe such rules and regulations as he may deem necessary to carry out [his] * * * responsibilities under this Act."

In addition, the Secretary's responsibilities under the Act are amplified by its enumerated purposes (Section 2(b)), which include:

> encouraging employers and employees in their efforts to reduce the number of occupational safety and health hazards at their places of employment, and to stimulate employers and employees to institute new and to perfect existing programs for providing safe and health working conditions; * * *
>
> authorizing the Secretary of Labor to set mandatory occupational safety and health standards applicable to business affecting interstate commerce, and by creating an Occupational Safety and Health Review Commission for carrying out adjudicatory functions under the Act; * * *
>
> building upon advances already made through employer and employee initiative for providing safe and healthful working conditions; * * *
>
> providing for the developing and promulgation of occupational safety and health standards; * * *;
>
> providing for appropriate reporting procedures with respect to occupational safety and health which procedures will help achieve the objectives of the Act and accurately describe the nature of the occupational safety and health problem; * * *;
>
> exploring ways to discover latent diseases * * *; * * *;
>
> establishing causal connections between diseases and work in environmental conditions * * *;
>
> encouraging joint labor-management efforts to reduce injuries and disease arising out of employment * * *;
>
> and * * * developing innovative methods, techniques, and approaches for dealing with

occupational safety and health problems * * *.

Because the proposed cadmium standard is reasonably related to these statutory goals and because the Agency's preliminary judgment is that the evidence satisfies the statutory requirements and that the proposed standard is feasible and substantially reduces a significant risk of cancer and other adverse health effects, the Secretary preliminarily finds that the proposed standard is necessary and appropriate to carry out her responsibilities under the Act.

IV. Chemical Identification, Production, and Use of Cadmium

Cadmium (Chemical Services Registry Number 7740-43-9) is a soft, blue-white, malleable, lustrous metal or a grayish-white powder. Cadmium is a biologically non-essential metal. It occurs in nature in lead, copper, and zinc sulfide ores, and is obtained as a by-product from the extraction, separation and recovery of those metals in refinery plants. World production in 1985 amounted to 18,660 metric tons.

Cadmium metal is produced by three basic processes: fractional precipitation and distillation of roasted zinc ores; direct distillation of cadmium-bearing zinc; and, electrolytic zinc processing. A primary use for cadmium metal is as an anticorrosive, electroplated onto steel. Cadmium may serve as an electrode component in alkaline batteries and may be used in alloys, silver solders, and welding.

Cadmium occurs in one valence state, +2, and does not form stable alkyl compounds or other organometallic compounds of known toxicologic significance. However, cadmium compounds commonly associated with industrial processes such as cadmium oxide, cadmium chloride, and cadmium sulfide, are occupational exposures with potentially serious health effects.

A substantial amount of cadmium sulfide and cadmium sulfoselenide is used in pigments to yield colors ranging from yellow to deep red. These pigments have a high tolerance to heat and to light and are used primarily in coloring plastics, ceramics and paints. Cadmium stearate is used as a stabilizer in plastics because it inhibits the deterioration of the product. Cadmium compounds are also used in smaller amounts in electric batteries and electronic components. Of the many inorganic cadmium compounds, several are quite soluble in water.

Cadmium exposures may also occur in refining and smelting operations. Relative to the metals with which it is found, cadmium volatilizes readily during these processes because of its low boiling point (765 °C) and high vapor pressure. The cadmium then condenses to form fine airborne particles that react almost immediately with oxygen to form respirable cadmium oxide. Other industry groups where exposure to cadmium may occur include electroplating, battery manufacturing, and pigment and plastics manufacturing. In addition, cadmium exposure is associated with welding, brazing, and painting operations in many other industries.

V. Health Effects

A vast amount of literature exists which documents the various non-cancer and cancer health effects in both man and animals from acute and chronic exposure to cadmium. This section will not attempt to refer to all of these studies but will present instead a selective review of the pertinent literature in order to present a condensation of the knowledge and opinion concerning the health effects of cadmium. For greater detail, reviews and cited original articles should be consulted.

83A. Metabolism

Occupational exposure to cadmium occurs primarily through inhalation. However, cadmium may also be ingested either directly (from contaminated hands when workers eat or smoke at the workplace) or indirectly from inhaled material that is deposited in the respiratory tract, cleared by mucociliary transport and then swallowed. Other environmental sources of cadmium, such as food and cigarette smoke, may add to a worker's total cadmium exposure. Exposure by inhalation is either in the form of small particles of cadmium fume or larger particles of cadmium dust. The extent of deposition depends on the particle size. It is estimated that ten percent of the particles of approximately 5.0 micrometers mean mass diameter (MMD) are deposited in the lung, whereas 50 percent of the particles of 1.0 micrometer MMD are deposited in the lung.

Of the amount deposited, 20 to 25 percent is systemically absorbed. (Exs. 8-619, 8-086a, p. 107). After absorption, cadmium is distributed to various organs throughout the body, particularly to the liver, kidney and muscles. Approximately one half to one third of the body burden of cadmium is found in the kidneys after chronic low-level exposure, with the highest concentrations found in the renal cortex. (Ex. 8-086a, p. 168). One sixth and one fifth of the body burden are found in the liver and muscles, respectively, after long term exposure. As exposure level increases, a greater proportion of the body burden of cadmium will be found in the liver relative to the kidney. Also, upon the onset of renal dysfunction, the level of cadmium in the kidney will decrease. The half-life of cadmium in the liver, kidney and muscles is 5 to 15, 10 to 30, and more than 30 years, respectively. (Ex. 8-086a, p. 168).

After initial exposure and absorption, cadmium is transported by the blood to the liver where it induces the synthesis of metallothionein, a low molecular weight metal-binding protein. Cadmium becomes bound to this protein forming a metal-protein complex which is then released back to the blood and transported to the kidney. In the kidney, the cadmium-metallothionein complex passes through the glomeruli and is reabsorbed by the proximal tubules. This complex can then be broken down by lysosomes, releasing unbound cadmium which can induce renal synthesis of metallothionein. In workers with only short-term low levels of cadmium exposure, the cadmium will be bound again to the locally produced metallothionein, providing a protective effect from cadmium. However, after prolonged exposure the binding process in the kidney becomes saturated, leading to an increase in unbound cadmium which can result in toxic effects.

B. Non-Carcinogenic Health Effects

11. Acute Effects

a. *Humans.* A variety of adverse health effects may result from acute exposure to cadmium compounds. For man, the most widely recognized effects are seen in the respiratory system from the inhalation of cadmium fumes and dust.

Symptoms first appear 10 to 24 hours after initial acute inhalation exposures to cadmium fumes. These signs are similar to metal fume fever with irritation and dryness of the throat and nose, cough, headache, dizziness, weakness, chills, fever, and chest pain. (Ex. 8-086b, p. 4). In extreme exposure cases pulmonary edema may develop and cause death several days after exposure. Such symptoms have been commonly reported among workers exposed to high concentrations of cadmium. For example, several cases of cadmium fume poisoning were observed among workers cutting cadmium plated metal (Ex. 8-41). After a day's exposure to cadmium fumes, workers developed severe weakness, dyspnea, coughing and tightness of the chest. Chest

radiographs showed signs of pulmonary edema.

In many investigations, as in the case above, the exposure levels at which the adverse effects occurred were not recorded. Attempts have been made to estimate the exposure levels associated with acute respiratory effects. For example, the actual exposure levels responsible for fatal cadmium fume poisoning have been estimated by first measuring the amount of cadmium found in the lung after death and then modifying this measurement by the amount of cadmium fumes assumed to be retained by the lungs. In the case study cited above, it was assumed that there was 11% retention of cadmium fumes in the lungs. Given an exposure time of 5 hours, the average concentration was estimated to be 8.6 mg/m³ over 5 hours. In terms of an 8-hour TWA exposure, the concentration would equal approximately 5 mg/m³. However, due to the assumptions used to derive this exposure level there is some uncertainty as to the accuracy of the estimate. Also, the amount of cadmium measured in the lung of the fatal cases may have been higher than the amount necessary to cause death. It should also be noted that this type of estimate is for lethal concentrations, and that lower concentrations may give rise to acute symptoms and significant lung damage without resulting in death.

b. *Animals.* Inhalation studies of animals exposed to both fumes and cadmium dust have confirmed the above mentioned resporatory effects. In addition, animals injected with cadmium compounds have exhibited acute effects in the testes, ovaries, liver and blood. Teratogenic effects have also been observed in animals after short term exposure.

Administration of cadmium compounds through various routes of exposure to experimental animals has also induced acute pulmonary effects. Several different species, in a number of studies reviewed by Friberg *et al.* (Ex. 8–086b, p. 3), have been exposed to various cadmium compounds. In the studies, exposures ranging from 5 to 10 mg/m³ over 15 to 120 minute periods were sufficient to induce significant increases in lung weights indicative of pulmonary edema. Also, rats exposed to cadmium aerosol at 60 mg/m³ for 30 minutes died within 3 days from pulmonary edema (Ex. 8–402). Multiple experimental studies confirm these findings of acute pulmonary effects and are reviewed by Friberg (Ex. 8–086b, p. 2), NIOSH (Ex. 4–02) and EPA (Ex. 8–619).

2. Chronic Effects

a. *Humans—i. Renal Effects.* Early evidence of adverse health effects of chronic low-dose exposure to cadmium can be measured in the renal system. In the majority of studies, the kidney is considered to be one of the target organs. Friberg (Ex.4–29) conducted one of the first studies on the prevalence of renal dysfunction among workers chronically exposed to cadmium. In this study, workers exposed to cadmium dust in alkaline accumulator factories over a period of 9 to 34 years exhibited a high prevalence of proteinuria, a condition in which there is an excess of serum proteins in the urine. In urine specimens examined to characterize the type of protein, it was found to consist primarily of low molecular weight proteins. Investigators have since identified this disorder as "tubular proteinuria," referring to the abnormally high urinary levels of low molecular weight proteins such as beta-2-microglobulin, retinol binding protein, and lysozyme that result from dysfunction of the proximal tubule of the nephron.

Normally, as blood passes through the kidney, the small size of low molecular weight proteins allows them to cross the intact glomerular basement membrane into the kidney tubule. However, only very small quantities of low molecular weight proteins are excreted in the urine because they are routinely reabsorbed by the proximal tubule of the nephron. The presence of excess low molecular weight proteins in the urine is an indication that kidney function is impaired due to damage to the cells lining the proximal tubules (tubular proteinuria). In cadmium associated renal disease, tubular proteinuria is considered to be one of the earliest signs of renal dysfunction.

In contrast, high molecular weight proteins (albumin, immunoglobulin G, and a variety of glycoproteins) do not cross the intact glomerular basement membrane into the kidney tubule. Glomerular proteinuria refers to the presence of high molecular weight proteins in the urine due to the increased permeability of the glomerulus (a "leaky" glomerulus) which allows the passage of the high molecular weight proteins into the tubule. High molecular weight proteins are not reabsorbed by the proximal tubule and therefore, the proteins are excreted in the urine. Glomerular proteinuria is considered to the indicative of a more progressive state of kidney dysfunction (Exs. 8–086b, p 63, 4–54).

After prolonged exposure to cadmium, tubular proteinuria may progress to glomerular proteinuria and possibly evolve to glycosuria, aminoaciduria, phosphaturia, and hypercalciuria (excess glucose, amino acids, phosphate, or calcium, respectively, in the urine, Exs. 8–086b, 4–28). The altered levels of excreted calcium may be associated with increased incidence of renal stones. Friberg in his early study of cadmium workers noted cases of renal stones as a common finding among cadmium exposed workers (Ex. 4–29). Hypercalciuria with renal stone formation was also observed in a follow-up study of workers exposed to cadmium dust/fume for 28 to 45 year (Ex. 9–9).

Many studies subsequent to Friberg's examination have similarly documented the high prevalence of proteinuria among workers exposed to cadmium dust and fumes. For example, workers manufacturing copper-cadmium alloys who were exposed to cadmium fumes over a period of 2 to 28 years developed tubular proteinuria indicated by excess of low molecular weight proteins in urine (Ex. 4–22). All cases observed had greater than 5 years exposure and in some cases greater than 15 years exposure. In this study no cadmium air concentrations above 270 μg/m³ were reported for any 12-hour period. A follow-up of this study four years later reported further cases of proteinuria despite the cessation of external exposure to cadmium (Ex. 4–23). Workers exposed to cadmium fume at levels below 100 μg/m³ during the brazing of wire and who were employed at least 21 years showed a higher prevalence of proteinuria compared to non-exposed controls (Ex. 4–28). A higher prevalence of proteinuria was also found among workers exposed to cadmium dust in a battery production factor (Ex. 4–47). For example, a 19% prevalence of tubular proteinuria was observed among workers in the battery factory who were employed from 6 to 12 years and were exposed to cadmium in air levels of approximately 50 μg/m³, whereas a control population of lumbermen and shipyard workers belonging to the same occupational health clinic showed only a 3% prevalence of proteinuria. Cadmium smelter workers exposed to cadmium dust over a 25 year period at estimated average exposure concentrations of 63 μg/m³ exhibited a reduction in tubular reabsorption and increased protein excretion compared to non-exposed workers in the same plant (Ex. 4–47). In this study, exposure estimates based on area samples were corrected to account for respirator usage.

Renal tubular dysfunction may be measured by the elevated urinary activity of N-acetyl-beta-d-glucosaminidase (NAG) or alanine aminopeptidase (AAP), indicating a disruption of renal tubular cell membranes or lysosomes. Mueller (Ex. 8–686) found urinary NAG and AAP were elevated and displayed a dose response to urinary cadmium in cadmium exposed workers. The investigators estimated a 10% chance of an elevated AAP at a urine cadmium level of 5.0 μg/g creatinine, the action level of the 1980 WHO study group.

In an examination of workers exposed to cadmium dust, Lauwerys (Ex. 4–50) observed that glomerular proteinuria was found in all exposed workers. Only workers with greater than 20 years exposure showed mixed-type proteinuria. Based on this result Lauwerys postulated that glomerular proteinuria might precede tubular proteinuria. These studies have been criticized because the methods used to measure the proteins in the urine were not sensitive enough to fully detect beta-2-microglobulin (Ex. 8–086b, p. 62). Therefore, the conditions diagnosed as glomerular proteinuria may have been preceded by tubular proteinuria.

Cumulative blood cadmium dose was used by Jarup (Ex. 8–685) and determined to be a more sensitive predictor of cadmium induced renal damage (as indicated by beta-2-microglobulin) than cumulative cadmium in air. Cadmium battery workers with tubular proteinuria had a proportionately high serial blood cadmium dose than their fellow workers without renal dysfunction but with the same cumulative air cadmium dose.

On the basis of autopsy studies, the World Health Organization task group concluded that the critical level in the renal cortex for the appearance of proteinuria (low and high molecular weight) ranged from 100 to 300 μg cadmium with the likely estimate being 200 μg cadmium/g wet weight. (Exs. 4–12, 8–440). Similarly, a review of autopsies and *in vivo* measurements (through neutron activation analysis) of human kidney tissues, shows that adverse effects first occur in the range of 170 to 200 μg cadmium/g. (Ex. 8–086b, p. 99). In animals, the concentration of cadmium in the renal cortex at which dysfunction first appears ranges from 100 to 300 μg cadmium/g wet weight, with most species showing proteinuria at 200 μg cadmium/g (Ex. 8–086b, p. 97).

In vivo measurements have also indicated that, upon prolonged exposure to cadmium, renal damage may occur. Neutron activation analyses conducted on workers in zinc-cadmium production plants (Ex. 4–58), in cadmium production plants (Ex. 4–26), and in cadmium smelters (Ex. 4–32) noted decreases in cadmium levels in the renal cortex with increasing levels of beta-2-microglobulin and cadmium in the urine. These findings indicate that as proteinuria progresses, damage to the kidney cells occurs leading to a loss of cadmium from the renal cortex.

Furthermore, evidence has shown that once tubular dysfunction is established, it may progress with little or no subsequent external exposure. For example, Piscator (Ex. 4–54) conducted follow-up studies on several groups of cadmium workers who had been previously exposed to cadmium. Some workers showed an increase in excretion of total proteins several years after the cessation or reduction of cadmium exposure. In none of the cases was there a return to normal protein excretion. Thus it was concluded that cadmium induced proteinuria is irreversible (Ex. 12–38). Furthermore, if tests for cadmium in urine are conducted, a low cadmium level could mean no disease or disease which has already caused irreversible damage. The use of a low molecular weight protein, such as beta-2-microglobulin, is a better test for identification of disease because beta-2-microglobulin levels still increase despite loss of cadmium from the kidney after damage. These findings, according to Friberg's review of the available data (Ex. 8–086b, p. 72), indicate that cadmium-induced renal damage is permanent.

The gravity of cadmium-induced renal damage is compounded by the fact that there is no medical treatment to prevent or reduce the accumulation of cadmium in the kidney (Ex. 8–619). In contrast to other heavy metals, current chelation therapy does not reduce the body burden of cadmium without producing significant renal damage. When chelated cadmium arrives in the kidneys, the cadmium may still be toxic to renal cells. Thus, large amounts of cadmium may move from the liver or muscle storage sites, overwhelm the kidney's usual attempts to store cadmium in a less toxic form, and accelerate deterioration of renal function. With the presently available chelating agents, it is essential that no worker be treated for elevated blood or urine cadmium levels by chelation therapy.

ii. Pulmonary Effects. In addition to chronic renal effects, long term exposure to cadmium may induce adverse effects on the respiratory system. Reduced pulmonary function and chronic lung disease indicative of emphysema have been observed in workers who have had prolonged exposure to cadmium dust or fumes. In Friberg's study at an alkaline accumulator factory (Ex. 4–29), workers exposed to cadmium dust at estimated concentrations of 3 to 15 mg/m³ for 9 to 34 years exhibited impaired olfactory sensation, shortness of breath, and impaired lung function with associated poor physical working capacity. Further evidence of these clinical observations comes from studies in which rabbits exposed to cadmium dust, taken from the alkaline accumulator factory, exhibited chronic inflammatory changes in the nasal mucosa and signs of emphysema in the lung tissue (Ex. 4–29). Subsequent studies have confirmed the findings of these initial clinical and experimental studies. Bonnell (Ex. 4–22) and Kazantzis (Ex. 4–42) studied workers exposed from 5 to 15 years to cadmium fume at copper-cadmium alloy factories. The average concentration of cadmium over an 8-hour period was reported not to have exceeded 270 μg/m³. The workers exhibited shortness of breath and impairment of pulmonary function, which were suggested to have been the result of emphysema. Similarly, a study of workers in three different factories exposed to cadmium dust at concentrations below 200 μg/m³ for greater than 20 years showed significantly lower pulmonary function compared to within plant non-exposed controls (Ex. 4–50). No correlations between symptoms and lung damage, or between cadmium air levels and symptoms/lung damage as evidenced by radiographic data were presented in this study. Smith (Ex. 4–63) examined workers who were exposed to airborne cadmium at 0.2 mg/m³ or greater for 6 years or more at a cadmium producing plant. Workers were found to have decreased pulmonary function and mild to moderate interstitial fibrosis. Findings in this study suggested that the lung damage was due to prolonged exposure rather than repeated acute exposures. No worker's medical records showed evidence of acute illnesses which would have occurred if cadmium air levels were 5 mg/m³. Furthermore, a dose-response relationship between reduced pulmonary function and months of cadmium exposure was observed (i.e. pulmonary function decreased as the months of exposure increased). It should be noted that in many of these studies proteinuria was observed in a number of the workers who experienced adverse respiratory effects, thus indicating that both chronic systemic effects and damage at site of contact result from inhalation of cadmium dusts and fumes.

The potential hazard to the respiratory tract of cadmium in inhaled

air depends in part upon the particle size. According to their diameter, particles can be inspirable or respirable. A particle is considered to be inspirable if it can be deposited anywhere in the respiratory tract. Respirable particulates are only those that are small enough to be transported to the alveolar region of the lungs where the gas-blood exchange occurs. Opinions differ on how small a particle must be in order to be respirable. Most experts, however, would classify any particle less than 8 um in mean mass diameter as respirable (Ex. 8–692). Inspirable particles up to 30 um can reach the tracheobronchial region of the lung (bronchus), but not the alveoli. The bronchi and the alveolar portion of the lungs are considered the thoracic portion of the respiratory tract. Still larger particles can reach the extra-thoracic portion of the respiratory tract. This portion includes the nose, maxillary sinuses, throat, oral cavity and pharynx.

Workers are exposed to a wide range of cadmium particles of various sizes. The distribution of cadmium particle size for any process is specific to the particular process. For example, the cadmium sulfide pigments used in the electronics industry and cadmium oxide fumes produced by welding operations are almost entirely respirable.

Not all workers exposed to cadmium are exposed to particles that are respirable. However, the risks from cadmium exposures are not limited to exposures that are respirable. Inspirable cadmium particles that are too large to be respirable but still small enough to enter the tracheobroncial region of the lung can lead to bronchoconstriction, chronic pulmonary disease, and cancer of that portion of the lung. Similarly, particles that are constrained by their size to the extra-thoracic regions of the respiratory system such as the nose and maxillary sinuses can lead to loss of smell. This condition also is commonly reported among cadmium-exposed workers.

Because cadmium particles that are not respirable but can be inspired can lead to all of the serious diseases mentioned above, regulation of respirable particles alone is insufficient to reduce risk of all diseases caused by cadmium.

iii. Skeletal Effects. Workers with progressive forms of proteinuria have also exhibited adverse effects on the skeletal system associated with improper bone mineralization such as osteoporosis and osteomalacia. It is possible that cadmium-induced disturbances in the kidney are associated with these adverse effects (Ex. 8–086b, pp. 111–158). For example, the active metabolite of vitamin D, 1,25-dihydrocalciferol (1,25 DHCC), forms in the kidney and stimulates intestinal absorption of calcium which is required for normal bone mineralization. As cadmium accumulates in the renal cortex it may inhibit the metabolism of vitamin D to its active metabolite. Additionally, cadmium induced renal damage may decrease the tubular reabsorption of calcium, thereby increasing the urinary excretion and loss of calcium from the body. Recent studies of patients with cadmium induced bone defects have also shown reduced concentrations of vitamin D metabolites in their blood (Ex. 8–189).

Bone mineralization may also be inhibited when there is interference with collagen metabolism. Cadmium may inhibit the formation of collagen fibers by interfering with the copper-dependent enzymes responsible for the cross linking of collagen molecules into fibrils. These fibrils form collagen fibers which in turn provide the fiber structure necessary for proper mineralization of bone. Improper bone mineralization results in a decreased density and softening of bone, conditions associated with osteoporosis and osteomalacia.

In humans, adverse bone effects have been observed after long-term exposure to cadmium. In a follow-up study of workers exposed to cadmium dust for 28 to 45 years, several workers showed hypercalciuria (an excess of calcium in the urine) with one case advancing to osteomalacia (Ex. 8–9). A case study of a battery plate worker exposed to cadmium for 36 years documented the development of renal tubular dysfunction and severe osteomalacia (Ex. 8–170). However, Friberg notes that relative to the number of workers with reported severe renal tubular damage the reported number of cases of adverse bone effects is low (Ex. 8–086b, p. 140). One reason may be that the bone has a reserve of calcium to maintain an adequate level in the body and thus it may take a long period of time for cadmium to induce bone disease. A second reason is that diet deficiencies, in addition to cadmium exposure, may also be necessary to induce bone effects. For example, in cadmium-polluted areas of Japan, cases of Itai-Itai disease, (a condition characterized by osteomalacia and renal tubular dysfunction), have been causally related to cadmium exposure from contaminated rice. However, among the cases there was also a dietary deficiency of calcium and vitamin–D, leading to the possibility that the inadequate consumption of essential food elements and vitamins may have been a contributing factor to the disease (Ex. 8–086b, p. 151–153).

iv. Other Information. There is a lack of data on reproductive effects in humans, despite evidence in animals. There is no evidence of cadmium-induced testicular necrosis in humans, most likely because extremely high doses would be required to induce such an effect. Friberg suggests that if the absorbed oral dose required to produce a testicular effect is proportional to the doses administered in the injection studies, a dose of 70 mg to a 70 kg man would be required to elicit the same response as the 1 mg/kg dose studies in animals (Ex. 8–086b, p. 185). The lack of data on testicular function following cadmium exposure in humans makes it diffcult to draw any conclusions on possible acute testicular effects in man. There is also a lack of evidence on human teratogenic effects, as epidemiological studies have not been conducted. It is possible, however, that high exposures to cadmium might influence zinc metabolism and induce zinc deficiencies that could alter fetal growth and development in humans as it does in animals.

Data submitted to OSHA (Ex. 12–10) indicate that some cadmium compounds (e.g., pigments) may not be as readily absorbed as others and, therefore, may not be as toxic. However, OSHA preliminarily concludes that these data do not provide adequate evidence to show that such compounds are not as toxic. Human studies on pigments indicate low urinary-cadmium and beta-2-microglobulin levels among cadmium pigment workers (Ex. 12–10 e, f, and g). Exposure levels for these workers, however, are poorly characterized, and it is difficult, therefore, to determine whether the results are due to low solubility of cadmium pigments or low exposure. These studies, some of which appear not to have been peer-reviewed or published in professional journals, are summarized in the following paragraphs.

The effect of cadmium exposure on health was evaluated by Mikche (Ex. 12–10e) for a group of workers involved in cadmium pigment production and cadmium pigment application. Among a group of 36 workers, with an average length of employment of 11.75 years in a cadmium pigment production plant, no correlation between cadmium air concentrations and cadmium or beta-2-microglobulin concentrations in urine was found. However, the only exposure information given in this study was the average air concentrations reported in 1977, 1979 and 1980. These concentrations were 50 $\mu g/m^3$,

30 μg/m³, and 30 μg/m³ respectively. These reported values are averages for only three years of the period for which workers were potentially exposed. Even for these years, the data do not allow one to determine the exposure levels for the workers whose body cadmium levels were measured. Without adequate exposure information, one cannot conclude that low cadmium body burdens are a result of the low absorbability of cadmium pigments since a low exposure might give the same result.

Also in this study, among 21 workers engaged in the application of cadmium pigments, the average cadmium concentrations in blood and urine were in the same range as those reported for non-exposed persons. The average length of employment among these workers was 11.3 years. However, no exposure levels at all were reported.

Low cadmium urine levels and low beta-2-microglobulin levels in urine were also found among other workers engaged in cadmium pigment manufacturing and processing (Ex. 12–10f). In this study by Fietz et al. workers were placed into five different categories based on the nature of their exposure. Groups I through III were involved in pigment manufacturing (e.g. raw material mixing, combustion, washing, drying and finishing). Groups IV and V were involved in pigment processing (e.g. paint formulation and pigment mixing). The average exposures were higher for groups I–III ranging from 14 to 201 μg/m³, with the levels decreasing over time. For groups IV and V exposures were only reported for 1981 and 1982. These values were lower than those found in pigment manufacturing. Correspondingly, the cadmium urine levels and beta-2-microglobulin levels were higher among those workers with greater exposure. These urinary levels decreased over time as cadmium air concentrations decreased. The authors concluded that the study showed that the use of technical measures (e.g. exhaust ventilation, sealing of machines, enclosure of sources of dusts and consistent use of respirators) can reduce the cadmium air levels and the harmful effects from cadmium (i.e. elevated urinary cadmium and beta-2-microglobulin levels). However, results from the study are not useful in evaluating whether the observed low levels of cadmium and beta-2-microglobulin in the urine were the result of the lower absorbability of cadmium pigments or low levels of cadmium air concentrations.

Health studies were also conducted by Greenburg et al. for a group of workers exposed to both lead and cadmium during the manufacturing of pigments (Ex. 12–10g). In this study of 38 men, the average length of employment was 20.7 years. Cadmium air levels were reported as "single measurements" with a range of 0 to 384 μg/m³ and mean values of 5μg/m³ in maintenance and 229 μg/m³ in the cadmium departments. The authors did not state whether or not these values were time weighted averages. However, the authors did state that 31% of the values, among all workers measured, exceeded the NIOSH recommended level of 40 μg/m³ which is a time weighted average. Worker exposure was characterized as either light (no elevated blood or urine levels/worked briefly in exposure areas), moderate (normal or moderately elevated levels, more than half of the work time in exposure areas/smelter operator) or heavy (levels known to have been high, removed from job site due to elevated levels/prolonged exposure). Workers who were unable to recall warnings about blood, urine or cadmium levels were classified as moderate. Workers were also classified according to smoking status.

Among the smokers there was a statistically significant increase in cadmium urine levels in workers with high or moderate cadmium exposure compared to workers with low exposure. There was also an increase in liver and kidney cadmium levels among high and moderately exposed workers. This increase was not statistically significant but this may have been due to the small number of subjects analyzed. A similar analysis was not possible for non-smokers because most of these workers were classified as having moderate exposure. Among all workers, 22% of non-smokers and 40% of smokers had kidney cadmium levels above what the authors consider to be "normal." For liver cadmium levels (CdL), 22% of non-smokers and 25% of smokers were above "normal" but below 40 μg/gm (CdL). In both cases it was not stated what was considered "normal". Excretion of beta-2-microglobulin in the urine was increased for 3 workers. The authors stated that there was a low prevalence of renal disease as evidenced by the fact that no worker had a decreased glomerular filtration rate. They pointed out that this low prevalence was unexpected given the fact that 31% of the exposure values were above NIOSH recommended limits for exposure to cadmium. Above this limit, it is believed by the authors, adverse effects are likely to occur. The authors conclude, because there was no evidence of renal disease after exposure to levels of cadmium pigments considered high enough to cause adverse effects, that cadmium pigments do not cause the same effects as more soluble cadmium compounds at similar exposure levels.

However, glomerular damage is generally considered to be a more advanced stage of kidney damage. It may not be surprising, therefore, to find that workers with histories of low exposure to cadmium do not have glomerular damage. Using a more advanced stage of kidney damage, such as reduced glomerular filtration rate, as evidence of renal disease may be inappropriate because the disease may exist without having progressed to the stage where glomerular damage has occurred. Earlier indicators of irreversible kidney damage, such as increased beta-2-microglobulin excretion, may be more appropriate to measure the presence of kidney disease. Also there is some uncertainty as to whether the "single measurements" of exposure that were reported were time weighted averages(TWA's). If these values were not TWAs, and if they were subsequently averaged as eight hour exposure values, these values may not have been above NIOSH recommended levels. In which case, a low prevalence of renal disease would be expected.

b. Animals—i. Renal Effects. Experimental animal studies support the finding of cadmium-induced proteinuria in humans, in particular regarding the critical concentration level of cadmium in the target organ, and the finding that increased concentrations of beta-2-microglobulin in the urine constitutes a biological marker of cadmium-induced tubular proteinuria. Friberg induced proteinuria in rabbits by exposing the animals by inhalation to cadmium oxide (CdO) dust at 8 mg/m³ for 5 hours/day for 8 months (Ex. 4–29). In the same study, rabbits exposed by injection to 0.65 mg/kg cadmium sulfate ($CdSO_4$) developed proteinuria after 2 months of exposure. A number of experimental studies in which animals were exposed by injection or oral exposure have also shown cadmium-induced proteinuria (Exs. 8–086b, p. 29, 8–402).

Some studies have observed the presence of proteins in the urine with higher molecular weight than beta-2-microglobulin and thus diagnosed the cadmium-induced proteinuria as glomerular proteinuria or "mixed-typed" proteinuria (both high and low molecular weight proteins present). For example, Bernard (Ex. 4–20) injected rats with 1 mg/m³ cadmium chloride ($CdCl_2$) 5 days/week for 2 months and induced proteinuria. The cadmium-

induced proteinuria was characterized not only by increased excretion of low molecular weight proteins but also by high molecular weight proteins indicative of glomerular dysfunction. In a similar study (Ex. 4–49), rats injected with cadmium also showed mixed-type proteinuria. After prolonged oral exposure rats developed glomerular proteinuria.

ii. Skeletal Effects. Experimental animal evidence has shown that by either injection or ingestion, exposure to cadmium can induce disturbances in cadmium metabolism with osteoporotic and osteomalacic conditions. For example, chicks which were administered cadmium in their feed for 3 weeks showed a decrease in calcium absorption from the intestine suggesting a possible effect on the formation of 1,25–DHCC (Ex. 8–3). The calcium absorption in this study further decreased with increased doses of cadmium. Also, osteomalacia was induced in rats fed dietary concentrations of 10, 50 or 100 ppm cadmium for 19 months (Ex. 8–112).

In rats, osteoporotic changes increased with increased doses of cadmium. The rats fed cadmium developed osteoporotic changes in bone before the onset of kidney damage indicating that cadmium may possibly have a direct effect on bone rather than an indirect effect through renal damage (Ex. 8–55). However, Friberg (Ex. 8–086b.p. 115–139) presents a review of experimental studies in which the preponderance of data seem to suggest that chronic exposure to cadmium induces osteoporosis and osteomalacia subsequent to, and perhaps associated with, renal tubular damage.

iii. Other Information. In addition to the major effects on the kidneys, lungs and bones, other adverse effects have been reported in experimental animals chronically exposed to cadmium. There are scattered reports of chronic effects on the gastrointestinal tract, peripheral nervous system and endocrine organs. More commonly documented effects in animals include anemia, changes in liver morphology, immunosuppression, and hypertension. For example, various experimental animals fed or injected with cadmium have commonly exhibited anemia, possibly due to cadmium's influence on the absorption and distribution of such metals as zinc and iron (Ex. 8–086b, p.167). Similarly, rats chronically exposed to cadmium oxide dust by inhalation developed anemia (Ex. 4–29). Animals exposed to cadmium by various routes of administration have shown morphological changes in the liver as well as disturbances in hepatic enzyme concentrations (Ex. 8–086b, p.161). Chronic oral exposure of mice to cadmium through drinking water decreased antibody synthesis (Ex. 8–24) and induced immunosuppression (Ex. 8–35).

There is conflicting evidence with respect to cadmium induced hypertension. Several studies have shown an increase in blood pressure after exposure to cadmium. Hypertension has been induced in rats orally exposed from 3 to 24 months to 0.1 to 10 mg cadmium/liter drinking water (EX. 8–14). In this study, levels as low as 0.1 mg/1 for 3 months increased systolic blood pressure. The renal cortical level was 5 to 30 μg cadmium/g wet weight, which is below the critical concentration at which proteinuria is commonly detected. There are also studies, under similar experimental conditions, which have shown no hypertensive effects (Ex. 8–086b, p. 170–173). It has been suggested that dietary differences may have caused the different responses, because rats on rye-based diets exhibited increased blood pressure whereas rats on other non-rye-based diets did not.

Testicular necrosis has been induced in animals after short term exposure to cadmium. For example, male mice and rats injected with a .02 mmol/kg dose of cadmium chloride (2.2 mg Cd/kg body weight) exhibited acute destruction of the testes, with destruction of the seminiferous epithelium and interstitial tissue within 24 to 48 hours (Ex. 8–107). After one subcutaneous injection with 1 mg cadmium chloride/100 g body weight, rats showed vascular alterations of the testes within 6 hours. Within 48 hours the seminiferous epithelium was destroyed (Ex. 8–139). Male rats injected with a 0.25 mg cadmium/kg dose of cadmium chloride ($CdC1_2$) for 5 days/week over 24 weeks, however, showed no change in testicular damage. Kidney damage, however, had occurred. Several studies have also shown acute effects on the ovaries of animals injected with cadmium chloride at doses ranging from 2.3 mg to 10 mg cadmium/kg body weight. Such effects included hemorrhage, endothelial damage, and morphological changes of the blood vessels of the ovaries. (Exs. 8–086b, p. 184, 8–157). Female rats injected with 0.036 and 0.18 mg cadmium/kg for 8–60 weeks showed an increase in the thickness of the basal lamina of the uterus. (Ex. 8–086b, p. 183–4).

Teratogenic and embryotoxic effects also have been observed in animals. When experimental animals were exposed to high doses of cadmium early in pregnancy, severe malformations and fetal death occurred. For example, the injection of rats with 4 to 12 mg cadmium chloride/g body weight on days 13–16 of gestation resulted in a rise in the fetal death rate, a decrease in fetal weight, and malformations such as cleft palate, clubfoot and small lungs (Ex. 8–202). Pregnant rats injected with a 1.8 mg cadmium/kg body weight dose of cadmium chloride produced offspring exhibiting malformations of the eyes, ears, and abdominal wall. (Ex. 8–204). Also, exposure of pregnant rats exposed later in pregnancy (e.g. days 17–20 gestation) to 2.5–4.5 mg cadmium/kg led to damage of the placenta and fetal death (Ex. 8–086b, p. 188). Less severe reproductive effects were observed in pregnant rats exposed by inhalation. For example, rats exposed to a 3 mg/m^3 dose of cadmium sulfate ($CdSO_4$) during pregnancy showed a reduction in fetal weight. Rats exposed to $CdC1_2$ at 0.2, 0.4 and 0.6 mg/m^3 for 21 days also resulted in fetal weight reduction, but only at the highest dose level (Ex. 8–086b). It is believed by the authors that cadmium influences the metabolism of zinc, possibly inducing a zinc deficiency which may cause teratogenic effects. This belief is in part due to the fact that little cadmium is transported across the placenta after the closure of the vitellin duct. Also, experimental data on rats have shown a decrease in fetal zinc concentrations after maternal cadmium exposures of .25 to 1.25 mg cadmium/kg (Ex. 8–157). In addition, data have shown that maternal exposure to cadmium alone induces fetal anomalies and zinc deficiencies, whereas co-administration of cadmium and zinc prevented fetotoxicity and fetal zinc deficiencies (Ex. 8–152).

As stated previously in the section on other human effects, data submitted to OSHA (Ex. 12–10) indicate that some cadmium compounds (e.g., pigments) may not be as readily absorbed as others and, therefore, may not be as toxic. OSHA preliminarily concludes, however, that these data do not provide adequate evidence to show that such compounds are not as toxic. (See for example Ex. 12–10.) The animal studies included in these data were of short exposure periods and presented conflicting results that do not indicate a simple relationship between solubility and bioavailability. A brief summary of these animal studies follows.

Hazelton Laboratories conducted a short term rat feeding study to determine whether or not there was a positive correlation between cadmium solubility and cadmium absorption through the gastrointestinal tract (Ex. 12–10b). In this study, extraction tests

were conducted with distilled water and with acid to determine the solubility of 12 different cadmium pigments. These same pigments were then fed to rats for one week at levels of 10,000 ppm and 50,000 ppm in the diet to evaluate the level of absorption of cadmium from the pigment. For purposes of comparison, rats were also fed a highly soluble compound, $CdCl_2$, at a concentration of 10 and 50 ppm in the diet. The percentage of cadmium absorbed was determined by measuring the amount of cadmium found in the urine, kidneys and liver and dividing by the amount of cadmium found in the feces and GI tract contents. The percent solubility of the pigments was much lower than the percent solubility of $CdCl_2$. The percent solubility for $CdCl_2$ was 61% whereas for the pigments, the solubility ranged from 0.06 to 1.38%. Correspondingly, the percentage of cadmium absorbed from the pigments was also much lower than for $CdCl_2$. The percentage of cadmium absorbed from $CdCl_2$ was 0.65% compared to .0004 to .0060 percent cadmium absorption from the cadmium pigments. From the data the authors concluded that there was a positive correlation between solubility and absorption, that is, the greater the solubility the greater the amount absorbed by the body. One should keep in mind that this feeding study was for only one week. While the percent of cadmium absorbed from the pigments after one week's exposure is relatively low compared to $CdCl_2$, the total percentage absorbed after chronic exposure to cadmium pigments (e.g. 18 months) is not known and may be more substantial.

In an acute inhalation study by Rusch et al (Ex. 12–10d), male and female Sprague-Dawley rats were exposed to dusts of cadmium carbonate ($CdCO_3$), cadmium yellow pigment, cadmium red pigment and cadmium fume for two hours at 100 mg/m^3 for one day in order to determine if there were differences in uptake and distribution with compounds of different solubilities. No mortality was observed among rats exposed to either cadmium pigment after 30 days follow-up. However, 3 out of 52 rats died from exposure to $CdCO_3$ and 25 out to 52 rats died from exposure to cadmium fume. In the cadmium pigment exposed groups, greater amounts of cadmium were eliminated by the feces at faster rates than for the $CdCO_3$ exposed rats. The $CdCO_3$ exposed rats also showed higher kidney cadmium levels. The authors stated that $CdCO_3$ followed predicted patterns of uptake, distribution and retention, whereas, the pigments showed only minimal uptake and tissue deposition. Therefore, it appeared that inhalation exposures to soluble compounds resulted in more rapid uptake and higher body burdens than did exposure to less soluble cadmium compounds. However, as in the feeding study above, this inhalation exposure was for a short time period. Rats were exposed for only two hours. Therefore, it is difficult to draw conclusions about the cadmium body burdens which might result from long term chronic exposure to cadmium pigments.

Longer periods of exposure were examined in a subacute inhalation animal study by Glaser et al (Ex. 12–10c). In this study male Wistar rats were continuously exposed for 30 days to aerosols of cadmium chloride and cadmium oxide at 0.1 mg/m^3 and aerosols of cadmium sulfide (CdS) at 1 mg/m^3. CdS was administered at a higher dose because of its lower solubility. In this study no clinical signs of intoxication were observed among any of the cadmium exposed groups. Cadmium was retained in the lung, liver and kidneys for all three compounds tested. Lesser amounts of cadmium were retained in the lung among $CdCl_2$ exposed rates compared to CdO and cadmium sulfide (CdS) exposed rats. After one month's exposure approximately 25 μg of cadmium were retained in the whole lung of $CdCl_2$ exposed rats whereas approximately 50 μg of cadmium and 140 μg of cadmium were retained in the lung for CdO and CdS exposed rats respectively. The authors note that a 10 times greater exposure in the form of CdS did not result in a 10 times greater amount of cadmium in the whole lung. Therefore they suggested that there must be a difference in toxicokinetics (i.e. deposition, dissolution, clearance or toxicity) for CdS. In addition they noted that for the $CdCl_2$ and CdO exposed rats more of the cadmium was distributed to the cytosol fractions of the lung compared to the CdS exposed rats, indicating that more of the CdS was retained in the extracellular fractions and was not absorbed into the cell. However for a site-of-contact carcinogen, which some evidence suggests cadmium may be, it is entirely possible that the more insoluble the compound, the greater the carcinogenic potential. In fact there was evidence of a cytotoxic effect to the alveolar macrophages from exposure to CdS equal to that observed from exposure to CdO. Each of these cytotoxic effects were greater than the effect observed from exposure to $CdCl_2$. In addition, the lung metallothionein-cadmium content for rats exposed to CdS and CdO were similar to one another and greater than the metallothionein-cadmium content in $CdCl_2$ exposed rats. Metallothionein is produced in response to cadmium ions and, according to the authors, is an indication of cadmium bioavailability. In the liver and kidney, cadmium burdens were significantly higher for the CdO and for the CdS exposed rats than for the $CdCl_2$ exposed rats. After one months's exposure approximately 15 μg of cadmium accumulated in the liver and kidney of $CdCl_2$ exposed rats compared to 70 μg of cadmium and 60 μg of cadmium which accumulated in CdO and CdS exposed rats. The authors state that it was unexpected that cadmium accumulation in the liver and kidney would be lower for $CdCl_2$ exposed rats than for CdO and CdS exposed rats because of $CdCl_2$'s higher solubility. It had been previously thought by the authors that cadmium accumulation was correlated to the solubility of the compound. Thus, the results of this study seem to suggest that absorption and bioavailability may not be simply equated to the compound's solubility. For example the body burdens of cadmium in the kidney and liver for CdO and CdS exposed rats are similar despite the fact that ten times more CdS was administered, thus implying that the lower solubility of CdS may be responsible for the lower accumulation of cadmium. However the body burdens of cadmium in the kidney and liver are higher for CdO exposed rats than $CdCl_2$ exposed rats despite the fact the $CdCl_2$ is more soluble. Thus it appears that there may be other factors besides solubility which may influence the systemic absorption and bioavailability, factors which could be further influenced by long term exposure (i.e. greater than one month). Furthermore this study shows that for rats exposed to less soluble cadmium compounds, more cadmium is retained in the lung. This may be important when site-of-contact tumors are involved, which may be the case where there is evidence of lung carcinogenicity from cadmium exposure.

Among the studies that have examined cadmium pigments there is some evidence to suggest that cadmium pigments are less soluble than other cadmium compounds such as cadmium chloride. It is possible that due to their relative insolubility the pigments generally are also less available to the body tissues. However the evidence is equivocal with respect to the observable toxic effects. The short term animal tests seem to show fewer adverse effects (e.g. lower mortality and cadmium body

burdens) among animals exposed to cadmium pigments. However the animals were only exposed for short periods of time. Yet even in these short term exposure studies there is evidence of accumulation of cadmium in the lung, liver and kidney. There is also positive evidence of tumor formation in rats exposed to a cadmium pigment compound. In the human studies, low urinary cadmium and beta-2-microglobulin levels were observed among cadmium pigment workers but, in most cases, the level of exposure was poorly reported raising the possibility that the lack of effect seen among these pigment exposed workers was simply a result of low exposure. Thus although there is some evidence to suggest that cadmium pigments are less soluble than other cadmium compounds, there is not sufficient data to show that this reduced solubility correlates with a reduced toxicity, especially after long term exposure. One study even suggests an increased bioavailability with a less soluble cadmium compound. Furthermore if the site-of-contact is a key factor in a toxic effect, the solubility and subsequent bioavailability of a compound to other systemic sites may make no difference to the adverse effects which might be induced at the initial point of entry. In any event, after long term exposure to cadmium pigments, cadmium may be retained or may accumulate in body tissues and result in adverse health effects similar to the adverse effects which have been observed after long term exposure to other cadmium compounds.

OSHA concludes, given the inadequacy of these data and the severe health effects that can result from cadmium exposures, that OSHA should not regulate these cadmium compounds differently from other cadmium compounds. OSHA seeks comment on this issue.

3. Conclusions about Non-Carcinogenic Effects

There is an abundance of data for several adverse health effects, clearly indicating that exposures to cadmium in the industrial environment can cause serious toxic effects in human beings. Not only are there many experimental studies showing the acute and chronic effects of cadmium exposure, but there is also a great amount of human evidence among cadmium exposed workers indicating adverse effects from chronic exposure to cadmium.

In humans, one of the earliest observable adverse effects from chronic exposure to cadmium is the presence of an excess amount of low-molecular weight proteins in the urine. When the excess for B_2-microglobulin, for example, reaches 200 $\mu g/gr$ Creatinine to 400 $\mu g/gr$ Creatinine, it probably indicates tubular proteinuria Exs. 12–07, 4–27, 4–28). This proteinuria is in itself an indicator of the impairment or loss of kidney function. Because of the body's ability to accumulate and store cadmium over long periods of time this condition may develop even after a reduction in or cessation of external cadmium exposure. Upon prolonged exposure tubular proteinuria may progress to more severe forms of renal dysfunction such as glycosuria, aminoaciduria, phosphaturia, and glomerular proteinuria or chronic nephrotoxicity.

The major functions of the kidneys are to remove the end products of metabolism and control the constituents of body fluids. With an impairment of kidney function, the body loses its ability to maintain a balance of chemical constituents which are carried by the blood and used throughout the body. Furthermore, once cadmium induced proteinuria has developed it is essentially irreversible, creating a permanent loss of kidney function. There is no specific treatment for chronic cadmium poisoning or a treatment to restore kidney function. Persons with cadmium induced renal disease are at increased riak for developing kidney failure if additional renal insults occur (e.g. exposure to other nephrotoxins including medications, infections of the renal-urinary system, obstruction of the urinary system, or reduced volume of blood flow to the kidneys due to reduced blood volume or vascular disease). In cases of cadmium-induced kidney damage, rigid control of diet, water intake and electrolyte balance in addition to medical treatment is required. In addition, since other environmental sources of cadmium (e.g. water, food, and ambient air) may contribute to the total body burden, it is necessary to avert additional adverse health effects to minimize all exposure to cadmium.

The major adverse health effects associated with long term occupational exposure to cadmium are on the kidneys, lungs and bones. In many cases it was observed that renal effects preceded or occurred simultaneously with other effects often at exposures below 100 $\mu g/m^3$, the current OS PEL for cadmium. In fact some effects, particularly those associated with disturbances in calcium metabolism, may be secondary manifestations of renal damage. Thus, the kidney appears to be a critical organ with regard to many adverse non-carcinogenic health effects associated with cadmium exposure.

As noted in the lead standard (43 PA 52952), diseases resulting from exposures to heavy metals proceed in five stages: (1) Normal, (2) physiological change of uncertain significance, (3) pathophysiological change, (4) overt symptoms (morbidity), and (5) mortality. within this process there are no sharp distinctions, but rather there is a continuum of effects. Categories overlap due to the variation in individual susceptibilities and exposures in the working population. While step 2 remains uncertain as to incidence of disease, by step 3 (pathophysiologic changes) important adverse health effects have occurred.

OSHA has designed this standard to prevent illness, or in the case of veteran workers already exposed to cadmium at higher levels over a period of years to minimize the extent of illness, by lowering worker exposure to cadmium and mandating medical surveillance. The provisions in this standard are specifically designed to detect early physiological and pathophysiological changes in the status of worker health so that future ill-health may be avoided or at least minimized. Evidence indicates that there is a progression of non-carcinogenic health effects that result from cadmium exposure. The effects start with a decrease in tubular reabsorption and/or a decrease in pulmonary function, and continue through more progressive forms of kidney and pulmonary dysfunction. Eventually, continued exposure results in more severe disorders in the kidneys, lungs and bones. Biological variability among individuals will determine the cadmium level at which a particular person will move through each stage in the disease continuum. However, these levels may be predicted with some degree of accuracy for most workers exposed to cadmium. As the level of cadmium exposure increases a greater proportion of the population will manifest each ill effect.

Given this understanding of the progressive stages of cadmium effects, OSHA has concluded that tubular proteinuria, indicative of the disruption of tubular reabsorption and of irreversible renal damage, is a pathophysiologic change and represents material impairment. There is a close correlation between observed and predicted levels of proteinuria associated with specific cadmium in air levels (Ex. 4–26). OSHA believes that the early stages of cadmium poisoning cannot be considered merely as an attempt by the body to adjust and

stabilize the internal environment to cadmium exposure. They are early indications of significant physiological disruption, which must be considered as material impairment of the health of the worker.

C. Mutagenicity

A wide range of tests, ranging from bacteria to human cells, have been conducted to determine the mutagenic effects of cadmium. For example, the mutagenicity of cadmium has been tested in bacteria, plants, insects, and mammalian cells, including human cells, *in vitro* and *in vivo*. Comprehensive reviews of these various investigations have been provided by Friberg (Ex. 8–086b, p. 223), Degraeve (Ex. 4–24), and EPA (Ex. 4–04). Both positive and negative results have been reported from these studies. This has lead to a somewhat confusing picture as to the mutagenicity of cadmium. The following section will give an overview of the more pertinent studies covered in the above reviews.

Cadmium has been shown to modify the metabolism of both RNA and DNA. Evidence has been obtained both *in vitro* and *in vivo* in microorganisms, plants, and mammalian cells showing enhancement and inhibition of RNA synthesis, degradation of DNA repair, inhibition of DNA synthesis, and inhibition of thymidine incorporation into DNA.

Gene mutation studies on microorganisms, yeasts, and mammalian cells have given mixed results on cadmium's mutagenic effects. For example, positive and negative mutagenic responses were observed in histidine reverse mutation assays using the bacteria *Salmonella typhimurium*. Some of these studies were considered inconclusive because several protocols were used in the assays. For example, different strains of *S. typhimurium* were tested using different dose regimens (e.g. single doses and doses with other chemicals). Conflicting and inconclusive results were also observed in gene mutation studies using yeast. For example, in a test for the induction of petite mutations, p-mutants were induced at the high and low doses but not at the middle dose. In a similar yeast assay, no p-mutants were induced at all, however; the dose was so toxic that only 1% of the yeast cells survived. Gene mutation assays using mammalian cell cultures of mouse lymphoma and chinese hamster cells have shown increased mutation frequencies with cadmium treatment.

Conflicting results were also reported in mutagenicity tests on fruit flies. Negative results were observed in sex-linked recessive lethal mutation tests, but positive results were observed in -dominant lethal mutation tests. However, among the negative results it was noted that in one case too few chromosomes were tested while in another case the number of chromosomes tested was not reported. Thus, the scope of the tests may have been too small to detect a positive response.

In higher order plants, the mutagenicity tests have been mostly positive. Aberrations such as chromosomal lesions and breaks were induced in several different species of plants.

In mammalian cells, *in vitro* studies on human lymphocytes, have shown increased incidences of structural chromosomal aberrations after treatment with cadmium. Among the observed aberrations were chromatid breaks, symmetrical and asymmetrical translocations, and deletions. *In vitro* tests on other mammalian cells in culture, such as Chinese hamster cells, displayed no increase in structural chromosomal aberrations with cadmium treatment but did show an increase in numerical chromosomal aberrations (e.g. hyperploidy and diploidy).

Numerical aberrations were also observed *in vivo* in the oocytes of mice and hamsters treated with cadmium. In these studies no structural chromosomal aberrations were noted. Numerical aberrations were also observed in the blastocytes of cadmium treated mice, indicating that aberrations induced in the oocytes may be transferred to the embryo. Other *in vivo* tests on mice have shown negative responses. For example, in micronucleus assays, the frequency of micronuclei in experimental groups did not increase compared to control groups. Also, in dominant lethal assays no increase in mutants was observed in mice injected with cadmium chloride compared to controls. Heritable translocation assays revealed no observable translocations in the spermatocytes of the F_1 progeny of mice injected with cadmium chloride.

As in other test systems, studies on humans have produced conflicting results. For example, lymphocytes from the blood samples of some patients suffering from Itai-Itai disease showed a high rate of chromosomal aberrations such as chromatid breaks and translocations; however, a similar examination of other Itai-Itai patients showed no aberrations. Similarly, positive and negative results were observed *in vivo* among cadmium exposed workers in two different smelter plants. It was noted that for the positive effects these workers may also have been exposed to other metals such as lead and zinc which might have induced or contributed to the observed aberrations.

Thus, although a number of positive mutagenic responses have been observed, there are also a number of conflicting negative responses. It is difficult to make comparisons or to make conclusions about these conflicting results since the studies investigated different endpoints, and often used different protocols. Thus, until more conclusive mutagenicity studies are conducted and reported, cadmium may be considered to be a potential mutagenic agent.

D. Carcinogenic Health Effects

Cadmium has been shown to induce cancers in laboratory animals and is associated with lung and prostate cancer in man. Cancer is the second most common cause of death in the U.S. today. Lung cancer claims the largest share of cancer deaths among males and the second largest share of cancer deaths among females. The National Center for Health Statistics reports that in 1980, the lung cancer death rate was 68.8 per 100,000 for males and 24.4 per 100,000 for females.

Few cases of lung cancer are curable, despite advances in medical and surgical oncology. Survival rates for lung cancer patients are poor with about 10% surviving five years or more after diagnosis (Ex. 8–62). Because lung cancer is almost certainly fatal, OSHA considers this disease to represent the gravest material impairment of health.

Prostate cancer does not always lead to death. Males may have prostate cancer for some time without any clinical manifestation of the disease. Some of these tumors lack the capacity for rapid growth, while others invade surrounding tissue and metastasize to distant organs and cause death. In 1980, 22,881 men died of prostate cancer; the prostate cancer death rate was 20.8 per 100,000 men. Because workers who work with cadmium are found to be at higher risk (Ex. 8–683) of prostate cancer and because so many men die of this disease, OSHA considers prostate cancer to also represent the gravest material impairment of health.

1. Animal Studies

Cadmium has been shown to be a carcinogen in animals when administered by inhalation. The strongest evidence of carcinogenicity comes from a rat bioassay by Takenaka et al (Ex. 4–67). In this well conducted

study, cadmium was found to induce lung carcinomas in exposed Wistar rats. Incidence in the exposed groups was statistically significantly elevated over the incidence in controls, and a statistically significant dose-response was observed.

Takenaka exposed three groups of male rats continuously for 18 months to cadmium chloride aerosols with nominal cadmium concentrations of 12.5, 25, and 50 $\mu g/m^3$. An additional group of 41 rats served as controls. The animals received water ad libitum during the experiment but were fed only 8 hours per day to minimize food contamination. The rats were observed for 13 months after the last exposure, at which time all surviving rats were sacrificed. There was no statistically significant difference in mean survival times among the four groups of rats, although the mean survival time for the high dose group was slightly shorter than the mean survival time for the other groups.

A histopathological examination was given to all rats surviving the exposure phase of the study unless their bodies were too autolyzed to allow such an exam. Cadmium concentrations were measured in the lungs, liver, and kidneys of a subgroup of each exposure group. Concentrations in the lung were nearly as high as in the liver. In all organs concentrations were observed to increase with dose except that only the low dose rats were found to have a slightly higher concentration in the lung than was found in the middle dose rats.

The incidence of lung carcinomas was 0/38 (0%) in the controls, 6/39 (15.4%) in the low dose group, 20/38 (52.6%) in the middle dose group, and 25/35 (71.4%) in the high dose group. The majority of carcinomas were adenocarcinomas; however, epidermoid carcinomas, mucoepidermoid carcinomas, and combined epidermoid carcinomas and adenocarcinomas were observed. The incidence of each of these tumors is presented in Table V–A.

TABLE V–A.—INCIDENCE OF LUNG CARCINOMAS IN MALE WISTAR RATS EXPOSED TO CADMIUM CHLORIDE AEROSOLS[*]

Tumor type	Controls (percent)	12.5 $\mu g/m^3$ (percent)	25 $\mu g/m^3$ (percent)	50 $\mu g/m^3$ (percent)
Adenocarcinoma	0/38 (0)	4/39 (10)	15/38 (39)	14/35 (40)
Epidermoid carcinoma	0/38 (0)	2/39 (5)	4/38 (11)	7/35 (20)
Mucoepidermoid carcinoma	0/38 (0)	0/39 (0)	0/38 (0)	3/35 (9)
Combined epidermoid carcinoma and adenocarcinoma	0/38 (0)	0/39 (0)	1/38 (3)	1/35 (3)
Total carcinomas	0/38 (0)	6/39 (15)	20/38 (53)	25/35 (71)

[*] From Takenaka et al. (Ex. 4–67)

The Takenaka study appears to have been the first animal study to conclusively document a lung cancer response from inhaled cadmium. Takenaka noted that a number of prior experimental study results had only raised the possibility of lung cancer being induced by cadmium inhalation. Other studies, however, have shown the induction of lung cancer and other cancers as a result of either inhalation or subcutaneous injection of several different cadmium compounds.

The Risk Assessment Guidelines published by the Office of Science and Technology (OSTP) call for taking account of negative as well as positive studies in assessing the weight of evidence on carcinogenicity (Ex. 8–693). Since 1980, OSHA has not published guidelines nor a standard concerning how it will assign weight of evidence in the qualitative evaluation of carcinogenicity in experimental animals. Other agencies have published guidelines, however, including OSTP and EPA. In EPA's guidelines, (51 FR 33992; Sept. 24, 1986), five conditions are identified that, if present, may lead to a relatively high degree of confidence in the results of animal bioassays for determining carcinogenicity: (1) Biologically independent tumors were found at a large number of sites; (2) independent experiments have demonstrated carcinogenic responses in both genders and in multiple species or strains of animals; (3) there is a clear-cut and statistically significant dose-response relationship; (4) there is a dose-related shortening of time-to-tumor occurrence; and (5) there is a dose related increase in the proportion of tumors that are malignant. Of these five conditions, four appear to exist for cadmium. OSHA requests comments concerning the degree of confidence that should be placed on the experimental study results related to cadmium in light of these five criteria.

The Takenaka study grew out of a pilot study by Heering et al. (Ex. 4–04). In that study, 10 rats were exposed for 18 months to cadmium chloride aerosols with a nominal cadmium concentration of 20 $\mu g/m^3$. The animals were sacrificed when exposure ended and four adenomas and one adenocarcinoma were observed.

Results from a study of intratracheal instillations of cadmium oxide are more equivocal. In a study of male Fisher-44 rats, Sanders and Mahaffey found no evidence of cadmium-induced lung carcinomas, but they did observe an increased incidence of mammary fibroadenomas (Ex. 4–61). In that study, three groups of rats were given intratracheal instillations of 25 μg cadmium oxide. Forty-eight rats received one treatment at 70 days of age; 46 rats received two treatments at 70 and 100 days of age for a total dose of 50 μg cadmium oxide; and 50 rats received three treatments at 70, 100, and 130 days for a total dose of 75 μg cadmium oxide. Forty-six rats serving as controls received one intratracheal instillation of 0.9% sodium chloride solution.

The observed incidence of mammary fibroadenomas was 3/45 (7%) in the controls, 7/44 (16%) in the low dose group, 5/41 (12%) in the middle dose group, and 11/48 (23%) in the high dose group. Using the Fisher Exact Test, only the high dose group had a statistically significantly elevated incidence over incidence in the controls ($p = .027$). Two (5%) adenocarcinomas of the lung were observed in the middle dose group. The average number of tumors per tumor bearing rat were 1.4, 1.5, 1.6, and 1.8 for the control, low dose, middle dose, and high dose groups respectively. The authors reported that this difference was significant ($p = .044$) in a chi-square test for independence between number of tumors and treatment groups. Slightly more rats in the control group were found to have no tumors (16%) than treated rats (5 to 7%).

Additional evidence of the carcinogenicity of inhaled cadmium is provided by the results from a long term bioassay by Oldiges et al. (Exs. 12–10i, 12–10h, and 12–35). In this study, groups of 20 male and female Wistar rats were

exposed to cadmium chloride concentrations at 30 $\mu g/m^3$ or 90 $\mu g/m^3$, cadmium oxide dust at concentrations of 30 $\mu g/m^3$ or 90 $\mu g/m^3$, cadmium oxide fumes at concentrations of 10 $\mu g/m^3$ or 30 $\mu g/m^3$, cadmium sulfate at a concentration of 90 $\mu g/m^3$, cadmium sulfide at concentrations of 90 $\mu g/m^3$, 270 $\mu g/m^3$, 810 $\mu g/m^3$, or 2430 $\mu g/m^3$, or a combination of cadmium oxide and zinc oxide dust at concentrations of 30 and 300 $\mu g/m^3$ respectively or 90 and 900 $\mu g/m^3$ respectively. Twenty male rats and 20 female rats served as controls.

Most groups of animals were exposed for 22 hours per day for 7 days per week. For each of these groups, exposure continued for 18 months or until 25% of that group had died. Other groups of animals were exposed to their cadmium compound for 40 hours per week for 6 months. This shorter exposure protocol was chosen to determine whether a brief exposure period would induce primary lung tumors. Animal groups were followed through month 31 of the study or until 75% of a group had died. At many of the exposure concentrations, doses proved to be too toxic and many animals did not survive the 31 months of study.

Preliminary results from this study are presented in Table V-B. The primary tumors observed in these rats were bronchio-alveolar adenomas, adenocarcinomas, and squamous cell tumors. The extremely high mortality rates seem to make this study unsuitable for quantitatively assessing the risk associated with each of the cadmium compounds studied or for assessing their relative carcinogenic potency. The study results indicate, however, that while zinc oxide dust may mitigate the carcinogenic potential of lower doses of cadmium oxide, each of the cadmium compounds alone is carcinogenic in animals exposed to these levels through inhalation.

TABLE V-B.—INCIDENCE OF PRIMARY LUNG TUMORS IN MALE AND FEMALE WISTAR RATS EXPOSED TO FOUR CADMIUM COMPOUNDS [a]

Exposure	Dose ($\mu g/m^3$)	Sex	Months of exposure [b]	Months of study [c]	Lung tumor incidence [d]
Controls		M		31	0/20
		F		31	0/20
Cadmium chloride	30	M	18	30	15/20
	30	F	18	31	13/18
Cadmium chloride	90	M	6	30	11/20
	90	F	6	29	3/18
Cadmium sulfate	90	M	14	31	11/20
	90	F	18	29	18/20
Cadmium sulfide	90	M	18	30	17/20
	90	F	18	31	15/20
Cadmium sulfide	270	M	16	30	14/19
	270	F	16	30	16/19
Cadmium sulfide	810	M	7	30	11/20
	810	F	10	29	13/20
Cadmium sulfide	2430	M	4	30	7/16
	2430	F	3	31	6/19
Cadmium sulfide	[e] 270	M	6	27	3/20
	[e] 270	F	6	29	3/20
Cadmium oxide dust	30	M	18	31	15/20
	30	F	18	31	15/20
Cadmium oxide dust	90	M	7	31	9/17
	90	F	1	31	11/16
Cadmium oxide dust	[e] 90	M	6	31	4/20
	[e] 90	F	6	31	3/20
Cadmium oxide dust	[f] 30	M	18	29	13/18
	[f] 30	F	18	29	12/20
Cadmium oxide fume	10	M	18	31	0/19
	10	F	18	31	0/19
Cadmium oxide fume	30	M	18	31	3/19
	30	F	18	31	4/17
Cadmium oxide and zinc oxide dust		M	18	31	0/20
	[g] 30/300	F	18	31	0/20
Cadmium oxide and zinc oxide dust		M	18	31	8/20
	[g] 90/900	F	18	31	7/20

[a] From Oldiges et al (Exs. 12-10i, 12-10h, and 12-35).
[b] Study protocol called for 6 or 18 months of exposure, but exposure was terminated when 25% of an animal group died.
[c] Months of study includes months of exposure. All animals in a group were sacrificed when mortality in that group exceeded 75%.
[d] Incidence is number of animals with at least one primary tumor divided by the number of animals at risk. Primary lung tumors are bronchio-alvoelar adenomas, adenocarcinomas, and squamous cell tumors.
[e] Exposure was for 40 hours per week.
[f] Rats were fed a zinc-reduced diet.
[g] Dose was 30 $\mu g/m^3$ of cadmium and 300 $\mu g/m^3$ of zinc or 90 $\mu g/m^3$ of cadmium and 900 $\mu g/m^3$ of zinc.

Heinrich et al (Ex. 8-694) exposed male and female Syrian golden hamsters and female mice to aerosols of cadmium sulfide, cadmium sulfate, cadmium chloride and cadmium oxide (as dust and fume) in various concentrations ranging from 30 to 1000 μg Cd/m^3. The animals were exposed for either 19 hours a day or 8 hours a day, 5 days a week for up to 14 months. After completion of the exposure period the animals were observed for another 6 to 12 months prior to sacrifice. In hamsters and mice, all cadmium compounds showed toxic effects in the respiratory tract leading to high mortality rates even in those groups where exposure was 30 $\mu g/m^3$ (mice) or 90 $\mu g/m^3$ (hamsters). For example, in mice exposed to cadmium oxide only 5 out of 14 treatment groups did not have shortened lifespans. Out of these 5 groups, 3 had a significant increase in the incidence of lung tumors, but this finding may be distorted by the high mortality rates seen in these groups and the high lung tumor incidence in the control groups. In

mice, all cadmium compounds caused an increased incidence of alveolar lipoproteinosis, interstitial fibrosis and bronchio-alveolar hyperplasia.

In hamsters, Heinrich et al claim to have observed no carcinogenic effect from any of the cadmium compounds, but tabular data were not provided on hamsters. It was observed that all the compounds caused a dose-dependent increase of bronchio-alveolar hyperplasia, thickening of the septa and proliferation of connective tissue, and the incidences of these conditions were statistically significantly increased.

There have been numerous studies involving the subcutaneous or intramuscular injection of cadmium into both rats and mice. The U.S. Environmental Protection Agency's "Updated Mutagenicity and Carcinogenicity Assessment of Cadmium" presents a summary of many of these studies (Ex. 4–4, p. 62–64).

A short summary of several of these studies is provided in the following section. Several studies have failed to demonstrate a carcinogenic effect from cadmium. In a series of studies, rats and mice were given 5 ppm cadmium acetate or oxalate in drinking water throughout their lives (Exs. 8–308, 8–121, 8–196). Compared to controls, there were no significant differences in the incidence of tumors in animals treated with cadmium, althoμgh mortality was increased in rats and male mice. In a study of prostatic changes due to cadmium, Levy et al. (Ex. 8–194) treated rats by subcutaneous injection of cadmium sulphate into the flank once weekly for two years in doses of 0.2, 0.1, and 0.05 mg. A low incidence of sarcomata at the injection site was seen in the treated groups. Levy stated that this finding was not unexpected, having been previously reported by Haddow et al. in 1964 (Ex. 4–34), Kazantzis in 1963 (Ex. 8–576), and Health et al., in 1962 (Ex. 3–117). No neoplastic changes were seen in the prostate gland, and there was no treatment-related increase in the incidence of neoplasms at other sites.

In two further studies of the effect of cadmium on the prostate gland by Levy et al. (Ex. 8–034 and 8–117), mice and rats were treated with cadmium sulphate by gastric instillation. Dosing regimens were 0.35, 0.18, and 0.087 mg/kg body weight once weekly for two years for rats, and 1.75, 0.88, and 0.44 mg/kg body weight once weekly for 18 months for mice. Concurrent dosing regimens of mice and rat controls were run using gastric instillation of equivalent amounts of distilled water. In both studies, no neoplastic lesions of the prostate or urinary tract were seen. Tumors seen in other organs could not be related to cadmium treatment.

Loser (Ex. 8–643) treated rats with cadmium chloride in the diet for two years at doses of 1, 3, 10, and 50 ppm. Fifty male and fifty female rats were used for each level; 100 rats of each sex served as concurrent controls. Cadmium treatment was not associated with an increased incidence of total numbers of tumors or any specific type of neoplasia.

Other studies (Exs. 4–55, 4–57, and 8–253) show that the injection of cadmium metal or certain salts of cadmium produce sarcomas at the site of injection as well as interstitial and Leydig cell tumors of the testes in experimental animals. The simultaneous administration of zinc and cadmium has been found to reduce the incidence of cadmium-induced testicular tumors (Ex. 8–253). For a discussion of these studies, please see Elinder (Ex. 8–086B p. 206).

OSHA has not relied upon the injection and peroral studies for assessing carcinogenic risk, nor upon the preliminary data on inhalation. The reasons for this are set forth below in the Significance of Risk section of the preamble.

OSHA relied, in part, upon the review by the International Agency for Research on Cancer [(IARC) Ex. 8–656] using IARC's criteria for categorizing animal data. IARC states that cadmium chloride, cadmium oxide, cadmium sulfate, and cadmium sulfide produced local sarcomas in rats following injection. Cadmium chloride and cadmium sulfate produced testicular tumors in mice and rats after subcutaneous administration. IARC concluded that the animal data are "sufficient", that is, a causal relationship has been established between exposures to cadmium and an increased incidence of malignant neoplasms or a combination of benign and malignant neoplasms in 2 or more species or in 2 or more independent studies in one species. IARC classifies cadmium as a probable human carcinogen because it is biologically plausible and prudent to regard agents for which there is "sufficient" animal evidence of carcinogenicity as if they presented a carcinogenic risk to humans.

Epidemiological Studies

Strong supportive evidence of the carcinogenicity of cadmium in humans comes from a mortality study of cadmium smelter workers by Thun et al (Ex. 4–68). Thun observed an excess of lung cancer deaths which was dependent upon intensity of cadmium exposure. 602 white males were selected for study. Each had spent at least 6 months in a production area of the smelter between 1940 and 1969. Workers were followed through 1978. The mortality status of all but 12 workers (2%) was determined; 411 were still alive (69%) and 179 had died (29%). Twenty-six workers who met the inclusion criteria were omitted from most of the analysis because these workers were hired prior to 1926 when the smelter functioned as an arsenic smelter. Arsenic is a known risk factor in lung cancer.

Worker exposures were estimated by Smith et al who based his estimates on historical area monitoring data adjusted to reflect the actual exposures of workers wearing respirators (Ex. 4–64). Using Smith's exposure estimates and company personnel records, Thun calculated cumulative dose estimates for each worker in his cohort.

Thun analyzed his data using a modified life-table method developed by NIOSH. Expected rates were calculated from the U.S. population and were adjusted for age, sex, race, and calendar time. Both standardized mortality ratios (SMRs) and standardized risk ratios (SRRs) were examined. To analyze his data by cumulative exposure, Thun divided his cohort into three groups. The low dose group had cumulative exposures less than or equal to 584 mg/m^3-days; the middle dose group had cumulative exposures between 585 and 2920 mg/m^3-days; and the high dose group had cumulative exposures greater than or equal to 2921 mg/m^3-days. These exposures correspond to 40 years of exposure at less than or equal to 40 μg/m^3 for the low dose group; 40 years of exposure between 41 and 200 μg/m^3 for the middle dose group; and 40 years of exposure at greater than 200 μg/m^3 for the high dose group. Thun also identified for separate analysis a subset of the low exposure group of his cohort in which the 40-year TWA equivalent exposures ranged from 21–40μg/m^3. These calculations are based on the assumption that 10 years of exposure at 1 μg/m^3 has the same carcinogenic effect as 1 year of exposure at 10 μg/m^3.

Forty-three percent of the workers had less than 2 years of employment. Follow-up time was long; 82.5% had more than 20 years of follow-up and 66.3% had more than 30 years of follow-up. Among the entire cohort of 602 workers, a statistically significant excess of deaths due to respiratory cancer (Obs=20; Exp=12.5; SMR=165; CI=101–254) and deaths due to non-malignant gastrointestinal disease (Obs=9; Exp=2.35; SMR=383; CI=175–727) were observed. All deaths due to lung cancer occurred in workers with more than two years of employment.

When the analysis was restricted to the 576 workers hired after 1926, the excess of lung cancer death was no longer statistically significant, but when the analysis was further restricted to those workers with two or more years of employment, the observed excess was statistically significant (Obs=16; Exp=7.00; SMR=229; CI=131–371).

Analysis of the 576 workers hired after 1926 indicates that the incidence of lung cancer death increases with dose. Among the low dose group, there was a deficit, i.e. lower than expected, of lung cancer deaths (Obs=2; Exp=3.77; SMR=53; SRR=.48). Among the subset of the low exposure group, the lung cancer SMR was 100 and the SRR was 96. For the middle dose group, there was no such deficit (Obs=7; Exp=4.61; SMR=152; SRR=1.55), but the observed excess was not statistically significant. For the high dose group, the excess lung cancer deaths was statistically significant (Obs=7; Exp=2.5; SMR=280; CI=113–577; SRR=3.45). Thun reported that this dose-response trend was also observed when the analysis was restricted to workers with more than 20 years since first exposure. The regression slope of the SRR for lung cancer was statistically significant indicating that an increase in cadmium exposure is producing a real increase in the risk of lung cancer.

OSHA notes that for many reasons, the finding of a deficit in lung cancer in the low dose group may not demonstrate an absence of lung cancer risk at low doses. For example, workers tend to be healthier than the general population. One would therefore expect a lower incidence of lung cancer among workers than in the general population if cadmium exposure posed no carcinogenic risk. This "healthy worker effect" is evidenced by studies which show that active workers experience a mortality risk of 60% to 90% that of the general population which includes sick, disabled, and institutionalized persons (Ex. 8–677).

Thun et al. also observed a significant increase in death from non-malignant gastrointestinal disease (NMGID), 9 observed versus 2.35 expected. The death certificates for six of these individuals suggested peptic ulcer disease. For those hired after 1926, there was a significant linear trend between increased cadmium exposure and the SRR from NMGID. The authors thought this observation was noteworthy in light of previously reported associations between cadmium exposure and severe gastrointestinal irritation in humans.

A non-statistically significant excess of genitourinary cancer was observed for the entire cohort (Obs=6; Exp=4.45; SMR=135; CI=49-293). Three of these deaths were from prostate cancer. The observed mortality from prostate cancer exceeded the expected, but the excess was not statistically significant (Obs=3; Exp=2.2; SMR=136). There were two other cases of prostate cancer, however, which Thun did not include in his analysis. One of these was a death from prostate cancer which occurred in a guard who had not spent 6 months in a production area of the smelter. The second case was not included because prostate cancer was not the underlying cause of death.

Thun et al. also evaluated the potential for arsenic exposure and cigarette smoking to confound the relationship between cadmium exposure and lung cancer. With regard to arsenic exposuree the authors separated their cohort into those hired prior to and subsequent to 1926, since arsenic smelting operations ceased in 1925. Thun *et al.* also had information from company records that the percent of arsenic in ore used at the cadmium smelter subsequent to 1925 was about 5% or lower and that potential for arsenic exposure was limited to only a few operations. They then took into account arsenic exposure to workers based on area and personal sampling data for atmospheric arsenic exposure, respirator use and urinary arsenic excretion. They estimated that there had been an average of 25 ug/m^3 of arsenic exposure for a total of 1,728 person-years of exposure. This estimate was considered biased on the high side. They then calculated the impact of this exposure on the role of lung cancer among employees in the cohort using the risk assessment model preferred by OSHA during its arsenic rulemaking. They concluded that the arsenic exposure received by the cohort would result in no more than 0.77 lung cancer deaths over the entire lifetime of the cohort. Thus, arsenic exposure did not seem to have any major impact on the lung cancer risk observed among the cadmium exposed workers.

Thun et al. also evaluated the role of cigarette smoking on lung cancer among the cohort members (Ex. 8-658). While it is difficult to know the smoking habits of workers in 1965, it is known that a sizable proportion of the cohort consisted of Hispanics who have a lower frequency of cigarette use. Hispanics are known to have a lower rate of lung cancer than the general U.S. white male population partially as a result of this (Ex.8-658). Use of U.S. white male population statistics to generate expected death rates, therefore, would overestimate the expected rates thus lowering the SMR.

OSHA is of the opinion that confounding from arsenic exposure and cigarette smoking is not likely to account for the increased lung cancer risk observed among the cadmium exposed workers. Furthermore, since the majority of this cohort is comprised of Hispanic workers, who have a lower rate of lung cancer mortality than the general U.S. white male population that was used to calculate the expected mortality, the lung cancer risk in the cadmium cohort may have been underestimated.

Varner conducted an earlier study of workers at the same cadmium smelter (Ex. 8–649). His cohort consisted of 644 workers with at least six months employment between 1940 and 1969. The cohort was followed through 1981. Mortality data was analyzed using Standardized Cause Ratios (SCRs). The preliminary findings of the study were statistically significant excesses of mortality due to lung cancer, urinary tract cancer, specific bladder cancers, and total cancers. Mortality due to prostrate cancer was elevated, but the excess was not statistically significant.

Varner attributed the observed excess of lung cancer deaths to arsenic exposure and cigarette smoking. Nonetheless, a dose-response relationship was observed between cadmium exposure and lung cancer and between cadmium exposure and total cancers. Cumulative cadmium exposures were estimated for each member of the cohort using personal monitor measurements made from 1973 through 1976. Exposures measured during this period were assumed to be constant for the entire period of study. The cohort was divided into a low exposure group (0–4 mg/m^3-years), a middle exposure group (5–15 mg/m^3-years), and a high exposure group (16+ mg/m^3-years). The observed SCRs for lung cancer deaths for each exposure group were: 95 for the low dose group, 159 for the middle dose group, and 332 for the high dose group. The observed SCRs for all cancer deaths for each exposure group were: 108 for the low dose group, 123 for the middle dose group, and 168 for the high dose group. If arsenic exposure and smoking were the cause of the excess cancer deaths, one would not expect to see such clear dose-response relationships between cadmium exposures and lung cancer.

Both the Thun study and the Varner study were follow-ups to an earlier study of workers at the same smelter by Lemen et al (Ex. 4–61). Lemen defined his study population differently than Thun. Lemen's study population consisted of 292 white males with a

mind of 2 years employment between 1940 and 1969. A statistically significant excess of deaths due to malignant neoplasms was observed (Obs=27; Exp=17.57; SMR=154;). Twelve of these deaths were due to respiratory cancer which was also a statistically significant excess (Obs=12; Exp=5.11; SMR=235;). Lemen reported that the risk of lung cancer increased with time since first exposure and that the greatest risk was observed among workers with more than 30 years of follow-up. Lemen also reported an excess of deaths due to prostate cancer (Obs=4; Exp=1.15; SMR=347; p>.05) which was significant when the analysis was restricted to workers with more than 20 years since first exposure (Obs=4; Exp=.88; SMR=452).

There have been numerous studies of workers in cadmium battery factories which suggest a link between cadmium exposure and prostate cancer. One of the earliest of these was by Kipling and Waterhouse who observed four prostate cancer cases among a cohort of 248 men employed in a British nickel-cadmium battery factory with cadmium oxide dust exposure (Ex. 4–45). Using incidence rates from a regional cancer registry to calculate the expected number of cases, these authors reported that the observed incidence of prostate cancer was more than seven times greater than the expected 0.58 cases (p=.003).

In a further study of workers from the same factory, Sorahan and Waterhouse observed a statistically significant excess of respiratory cancer (Obs=89; Exp=70.2; SMR=127; p<.05)(Ex. 4–65). An excess of prostate cancer was also observed, but this was not statistically significant (Obs=8; Exp=6.6; SMR=121).

To assess the effect of dose on mortality, the authors devised two measures of cadmium exposure. The first exposure measure was "cumulative duration of employment in high exposure jobs", and the second exposure measure was "cumulative duration of employment in high or moderate exposure jobs." Using the method of regression models in life tables, the authors found that cumulative duration of employment in high cadmium exposure jobs was significantly related to prostate cancer mortality but only when the four original cases described by Kipling and Waterhouse were included in the analysis. When cadmium exposure was measured by cumulative duration of employment in high cadmium exposure jobs, exposure was not statistically significantly associated with lung cancer mortality, but when cadmium exposure was measured by cumulative duration of employment in high or moderate cadmium exposure jobs, a statistically significant association was observed. The authors caution, however, that this observed effect could be confounded by oxyacetylene fume exposure.

Workers at this factory were studied once again by Armstrong and Kazantzis, who conducted a case-control study of workers who had died of prostate cancer, renal cancer, bronchitis or emphysema, or nephritis or nephrosis (Ex. 4–19). Cases were selected from three cohorts of British workers exposed to cadmium. All of the cohorts had been studied previously. Cohort C1 was comprised of workers from a lead-zinc-cadmium smelter previously studied by Armstrong and Kazantzis (Ex. 8–565). Cohort C2 was comprised of workers from the nickel-cadmium battery factory studied by Sorahan and Waterhouse (Ex. 4–65). Cohort C3 was comprised of workers from a copper-cadmium alloy plant previously studied by Holden who had found statistically significant excess of prostate cancers (Ex. 4–40). Cases consisted of workers who died of prostate cancer, chronic respiratory disease or renal disease. Only men born before 1940 with at least one year employment before 1970 were included. For each case, three controls were selected matched by plant, age, and, as nearly as possible, date of birth.

The authors divided these cohorts into three groups: always low cadmium exposure; ever medium cadmium exposure; and ever high cadmium exposure. They found that the odds of prostate cancer for the ever medium or ever high exposure groups were elevated relative to the always low exposure groups (1.55 and 1.35 respectively), but neither of these odds ratios were statistically significant. The authors note, however, that the small number of prostate cancer cases makes interpretation of this finding difficult.

In 1987, Sorahan updated his study of the nickel cadmium battery workers (Ex. 12–12A). Twenty-two additional deaths from lung cancer were reported. According to the author, there was some evidence of an association between risk of death from lung cancer and duration of employment in high or moderate (or slight) exposure jobs for "early workers", (i.e. first employed before 1946), but none for "late workers" (i.e. first employed after 1946). A significant increase in lung cancer was observed for the entire cohort of workers (110 Obs., 84.5 Exp., p<.01). Sorahan did not report a statistically significant increase in lung cancer for his cohort when workers were divided into "early workers" and "late workers", but OSHA's analysis shows that there was a significant excess of lung cancers for the "late workers" (45 Obs., 33 Exp., p<.05—one tail).

Among "late workers", the SMRs for lung cancer were observed to increase with years from first employment. Because this trend was not observed for "early workers", Sorahan suggested that there might be selection bias for the "early workers" and that this sub-cohort may be incomplete. The study's inability to demonstrate a significant relationship between duration of employment and lung cancer risk, however, does not mean that there is no association between cadmium exposure and lung cancer risk. Duration of exposure may not be a surrogate for dose, particularly when the length of exposure periods are not adjusted for the particular years in which the exposure occurs. The observed excess of lung cancer deaths among the "late workers" supports an association between cadmium exposure and lung cancer.

Ades and Kazantzis conducted a study of lung cancer in non-ferrous smelter workers (Ex. 12–14C). This cohort of men employed in a lead-zinc-cadmium smelter was part of Cohort C1 in the Armstrong and Kazantzis study described above (Ex. 4–19). The authors found a significant excess of lung cancer deaths among the entire cohort (182 Obs., 146.2 Exp., p<.005). In subcohorts of workers, a significant excess of lung cancer deaths was observed for workers with 20 to 29 years of employment (44 Obs., 23.1 Exp., p<.005) and for workers with 40 or more years of employment (8 Obs., 2.74 Exp., p<.02).

SMRs for lung cancer death were observed to increase with duration of employment for the cohort. This linear trend was statistically significant. The risk of lung cancer for workers with more than five years of employment relative to the risk for workers with less than five years of employment was also observed to increase with duration of employment. Using a matched logistic regression analysis, the authors were able to associate this increasing risk with exposure to arsenic and lead but not cadmium. This finding, however, could be due in part to the study protocol for choosing controls. Cases and controls were matched by date of hire, but because controls were required to have ten years of follow-up and to survive the matched case, cases and controls may have been inadvertently matched on cadmium exposure as well.

The entire Armstrong and Kazantzis cohort was studied again by Kazantzis and associates (Ex. 8–684). In this

update, the authors followed the workers for an additional five years. Seventy-five additional cases of lung cancer were observed, resulting in a significant excess of mortality due to lung cancer for both the additional five year period (SMR=134; 95% CI=103–164) and the entire study period (Obs=277; Exp=240.9; SMR=115; 95% CI=101–129).

The increased lung cancer risk occurred mainly among those first employed before 1940, and the risk increased with length of employment and length of follow-up. The majority of lung cancer deaths were among workers employed in the non-ferrous smelter studied by Ades and Kazantzis. This worksite provided over 60% of the total study population, but its workers' exposures were characterized only as low or medium. No exposures in the smelter were characterized as high.

Over the entire study period, there was a statistically significant excess of mortality due to stomach cancer (Obs=98; Exp=70.6; SMR=139; 95% CI=111–166). Of the 98 deaths observed, 22 occurred during the five years of added follow-up, giving a statistically significant excess of stomach cancer mortality for that five year period (SMR=179; 95% CI=112–271).

In an update of an earlier study by Kjellstrom *et al* (Ex. 4–48) Elinder *et al* analyzed mortality data on a cohort of 545 male workers at a Swedish cadmium-nickel battery factory (Ex. 4–25). While no statistically significant excess of mortality due to any type of cancer was observed, the authors reported that the SMRs for cancers of the lung, prostate, and bladder increased with time since initial exposure (i.e. latency) among workers with at least 5 years of exposure. Thus, for lung cancer, the SMR, was 133 for the entire cohort, but for workers with at least five years of exposure, the SMR was 163 after 10 years latency and 175 after 20 years latency. For prostate cancer, the SMR was 108 for the entire cohort, but for workers with at least 5 years of exposure, the SMR was 125 after 10 years latency and 148 after 20 years latency. For bladder cancer, the SMR was 181 for the entire cohort, but for workers with at least 5 years of exposure, the SMR was 222 after 10 years latency and 250 after 20 years latency.

In the paper, Elinder summarized the results of 13 studies of occupational cadmium exposure and prostate cancer. Twelve of the 13 studies reported excess cancers of the prostate, and 4 of these excesses were statistically significant. Elinder noted that the median SMR of the combined studies was 167, and when the number of observed and expected cases are combined for the most recent updates of the six independent studies, (7 of the 13 studies were updates of earlier studies), the statistically significant SMR for prostate cancer for all cohorts is 162 (28 obs., 17.2 exps., $p<.02$).

Elinder also summarized the lung cancer mortality observed in these 13 studies. Twelve of these excesses were statistically significant. The SMR for data pooled from the five most recent independent studies was 121, and this too was statistically significant (195 obs., 161.4 exp., $p<.01$).

What is most compelling about all these studies is the consistency of association between lung and prostate cancer and exposure to cadmium among workers in different industries located in different countries. Elinder et al. concluded, "Our interpretation is that the accumulating data on the mortality of cadmium workers with high exposure levels in the past (above 0.3 mg Cd/m^3 support an association between lung cancer and cancer of the prostate and exposure to cadmium." (Ex. 4–25). OSHA agrees with this conclusion.

Some of the cadmium exposed cohort members in some studies had potential for exposure to other potential lung carcinogens. For example, workers in the Elinder *et al* study also had exposure to nickel hydroxide as well as to cadmium oxide. Nickel exposure may also have contributed to the excess of lung cancer seen in the British battery plant. Nickel exposure, however, is not likely to have occurred in the British copper alloy plant nor in the U.S. plant studied by Thun *et al.* Although some workers in the Thun et al. study had potential for exposure to arsenic, the study demonstrated a dose-response for lung cancer in relation to cadmium exposure. If background contamination of arsenic was responsible for the increase in lung cancer observed among the employees in the cohort, one would not expect to see a dose-response in relation to the cadmium exposure unless arsenic and cadmium exposures were correlated. Furthermore, analyses by Thun *et al.* estimated that arsenic contamination could have accounted for less than one lung cancer death in his study.

While there is the potential for some cohort members in some of the studies to have been exposed to other potential lung carcinogens, it is OSA's opinion that the epidemiologic data taken as a whole demonstrate a significant association between cadmium exposure and lung cancer. The data also demonstrate a significant association between cadmium exposure and prostate cancer. These epidemiologic findings are consistent with the results of cancer bioassays demonstrating the carcinogencity of cadmium in experimental animals. Thus, OSHA agrees with IARC (Ex. 8–656) that cadmium is a probable human carcinogen.

VI. Preliminary Quantitative Risk Assessment

A. Introduction

The United States Supreme Court, in the "benzene" decision, (*Industrial Union Department, AFL–CIO* v. *American Petroleum Institute*, 448 U.S. 607 (1980)) has ruled that the OSH Act requires that, prior to the issuance of a new standard, a determination must be made that there is a significant risk of health impairment at existing permissible exposure limits and that issuance of a new standard will substantially reduce or eliminate that risk. 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. 642]. The Court also stated "that the Act does limit the Secretary's power to require the elimination of significant risks" [448 U.S. 644].

Although the Court in the cotton dust case (*American Textile Manufacturers Institute* v. *Donovan*, 452 U.S. 490 (1981)) rejected the use of cost-benefit analysis in setting OSHA standards, it reaffirmed its previous position in "benzene" that a risk assessment is not only appropriate, but also required to identify significant health risk to workers and to determine if a proposed standard will achieve a reduction in that risk. Although the Court did not require OSHA to perform a quantitative risk assessment in every case, the Court implied, and OSHA as a matter of policy agrees, that assessments should be put into quantitative terms to the extent possible.

The determining factor in the decision to perform a quantitative risk assessment is the availability of suitable data for use in such an assessment. In the case of cadmium, OSHA has determined that data are available to quantify two types of risk. The first of these is cancer risk. Data from both the Takenaka rat bioassay (Ex. 4–67) and the Thun human mortality study (Ex. 4–68) have been used by others to quantify the risk associated with cadmium. OSHA used both of these data sets for

its quantitative risk assessment. The Agency believes, however, that the exposure measurements are more accurate in the rat study than in the human study. Limited evidence exists concerning the exposure rate and duration of exposure for members of Thun's cohort, and no exposure estimates exist for individuals in the cohort. Furthermore, the Thun data can be used to predict excess lung cancer deaths only, whereas the rat data may be used to predict excess deaths from all types of cancer.

The second type of risk associated with cadmium which OSHA has determined can be quantified is the risk of kidney dysfunction. As discussed in a previous section, a number of studies have shown a dose-response relationship between cadmium exposure and kidney dysfunction. The authors of one study, Ellis, Cohn, and Smith, (Ex. 4–27), used their data to model the observed dose-response relationship. It is this model (a logistic regression model) which OSHA has used to derive its best estimate of risk of kidney dysfunction.

There is uncertainty associated with the quantification of any kind of risk. In this risk assessment, OSHA has tried to describe many of the sources of uncertainty and to address their implications on OSHA's estimates of risk. Additional discussion of the uncertainty in OSHA's cadmium risk assessment is provided below in the Significance of Risk section of this preamble.

B. Estimates of Cancer Risk Derived from Animal Data

1. Choice of Data Base for Quantitative Risk Assessment

The inhalation bioassay conducted by Takenaka et al (Ex. 4–67) provides the best available data for quantifying the carcinogenic risk associated with cadmium exposure. OSHA requests comments concerning how and whether the additional positive and negative experimental studies should be used in developing OSHA's final risk assessment based upon the toxicologic studies. The study, described above, entailed the continuous exposure of three groups of 40 male Wistar rats to cadmium chloride aerosols at nominal cadmium concentrations of 12.5, 25, and 50 $\mu g/m^3$. Forty-one male Wistar rats served as controls. The rats were continuously exposed to the test article for 18 months. They were then followed for an additional 13 months, when all surviving rats were sacrificed. Mean survival was 121.9 weeks for the controls, 119.2 weeks for the low dose group, 124.5 weeks for the middle dose group, and 116.1 weeks for the high dose group.

The Takenaka study is particularly suitable for quantitative risk assessment for several reasons. First of all, the exposure levels are well documented. The study was run with concurrent controls, and a statistically significant excess of malignant neoplasms in the exposed rats and a statistically significant dose-response relationship were observed. Finally, the route of exposure used in this study (i.e. inhalation) is the same as is found in most occupational settings.

The carcinogenic response observed in the rats was carcinoma of the lung. Three different types of carcinoma were observed: adenocarcinoma, epidermoid carcinoma, and mucoepidermoid carcinoma. The majority of tumors were adenocarcinomas. For the purpose of quantifying risk, these tumor types were combined to obtain an overall measure of carcinogenic response. The number of rats at risk in each group is the number of rats examined histologically. Rats were not examined if they died during the 18 months of exposure or if they were too autolyzed to be examined. The observed incidence of lung carcinomas was 0/38 (0%) for controls, 6/39 (15.4%) for the low dose group, 20/38 (52.6%) for the middle dose group, and 25/35 (71.4%) for the high dose group.

2. Measure of Dose

The extrapolation of carcinogenic risk across species rests on the assumption that when dose is measured in equivalent units for both species, then the risk associated with lifetime exposure to a substance is the same for each species at each dose. It does not follow from this assumption, however, that the observed carcinogenic response will be the same across species. Indeed, the cancers associated with exposure to a substance often differ across species and may differ between sexes of the same species. For example, ethylene oxide exposure is associated with peritoneal mesothelioma in male Fischer 344 rats, mononuclear cell leukemia in female Fischer 344 rats, and leukemia in humans. In rulemaking for ethylene oxide, OSHA based its risk assessment of human cancer risk on the rat data despite differences in observed tumor types (Apr. 21, 1983; 48 FR 17284).

Takenaka found that cadmium induced lung cancer in rats. Lung cancers have been reported in association with human exposure to cadmium, but other tumors (e.g., prostate cancer) have also been reported in association with human exposure to cadmium. Thus, OSHA's risk assessment uses data from the rat bioassay to predict the excess human risk of death from all types of cancers associated with occupational exposure to cadmium. This is consistent with OSHAs practice of estimating total excess cancer deaths as was done in the Arsenic, Ethylene Oxide, and Asbestos Standards. Exposure levels are scaled to equivalent doses for rats and man by measuring dose in units of inhaled micrograms per kilogram of body weight per day($\mu g/kg/day$). This conversion may adjust for differences in rates of inhalation, metabolism, and absorption between species.

In the case of an inhaled particulate like cadmium, the dose received by exposed animals or humans is a function of three factors: particulate levels in ambient air; volume of air inspired; and fraction of inhaled particles deposited in the lungs and upper airways. In its cadmium risk assessment, the Office of Health and Environmental Assessment, U.S. Environmental Protection Agency (EPA) raised the question of whether this last factor, fraction of inhaled particles deposited in the lungs and upper airways, can and should be taken into account in estimating internal dose (Ex. 4–04). After noting that particle deposition varied with species, particle size, and depth and rate of inhalation, EPA concluded that it was not possible to adjust internal dose for this factor.

OSHA agrees with EPA's conclusion. Clearly, particle size can not be used to adjust for particle deposition because there is no precise data available on the size of cadmium particles to which workers are exposed. Alveolar deposition of cadmium particles is believed to range from 50% for 0.1 um particles to 5% for 10 um particles (Ex. 8–086A, page 107). Furthermore, as noted by EPA, considerable variability in the fraction of cadmium particles deposited in the lungs was observed not only between studies but also among individuals within each study and within individuals themselves. This last source of variability was attributed to changes in breathing patterns. EPA reported that the fraction of particle deposition in human alveoli exposed to cadmium particles 0.5 um in diameter was observed to vary from approximately 9% to 21%. This range covers the 10% fraction of particle deposition observed in rats exposed to particles 0.55 um in diameter, approximately the size of the particles used in the Takenaka study. Thus, like EPA, OSHA believes that given the available data, it is reasonable to assume that cadmium particle

deposition in the alveoli is similar for rats and humans for cadmium particles of similar size, since the range for humans covers the range for rats, and that no adjustment for species differences in particle deposition is required.

As mentioned in the section of the preamble dealing with chronic pulmonary effects, OSHA does not believe it is possible to quantify the risk of cancer or other diseases of the lung based on differences in particle size. Furthermore, if it were possible to quantify risk based on the particle size distribution of cadmium compounds in various occupational settings, OSHA does not have data concerning the distribution of particle sizes to which workers are exposed in different occupational settings. OSHA therefore requests comments and empirical data concerning the size distribution of actual particles encountered in occupational settings and any information that would improve the ability to assess risks based on particle size. In particular, would different lung disease risks be posed by different particle sizes? Is regulation warranted according to particle size? If distribution of particle size should be measured, what type of sampling and analytical methodology should be used? Is there a need to distinguish the respirable portion of total airborne cadmium particles?

Data for converting experimental dose into units of microgram per kilogram per day (μg/kg/day) are presented in Table VI–A. From the Federation of American Societies for Experimental Biology it is reported that a rat which weighs .113 kg breaths .105 m^3/day. Using surface area proportionality, the volume of air inhaled per day (I m^3) by a rat which weighs W kg may be estimated using the equation:

$$I = .105 \times (W/.113)^{2/3}.$$

To estimate the volume of air inhaled by rats in each of the bioassay exposure groups, the average weight of each group of rats at 18 months was used.

Takenaka reported that the average measured concentrations of cadmium received by the rats was 13.4 μg/m^3 for the low dose group, 25.7 μg/m^3 for the middle dose group, and 50.8 μg/m^3 for the high dose group. For each group of rats, let MC μg/m^3 represent the measured cadmium concentration: let I m^3=the volume of air inhaled per day; and let W kg=the average weight of the rats at 18 months. The daily dose of cadmium received by that group of rats may be converted into units of μg/kg/day using the equality

$$\text{Dose}(\mu g/kg/day) = \frac{MC(\mu g/m^3) \times I(m^3)}{W(kg)}$$

Once dose has been converted into units of μg/kg/day, it must be adjusted to an equivalent continuous lifetime dose as required by most quantitative risk assessment computer programs. The rats in the Takenaka bioassay were exposed to cadmium for 18 months. If it is assumed that average survival was two years, then the rats were exposed continuously for 75% of their lives. On the assumption that exposure at level Y for 18 months has the same effect as exposure at 75% of level Y for two years, the dose received by each group of rats is multiplied by .75 to arrive at an equivalt continuous lifetime dose. Mean survival ranged from 116.1 weeks to 124.5 weeks for exposed rats. The mean survival control rats was 121.9 weeks. These doses are estimated as 6.01 μg/kg/day for the low dose group, 11.41 μg/kg/day for the middle dose group, and 22.78 μg/kg/day for the high dose group.

TABLE VI–A.—DATA FOR CONVERTING DOSE MEASURED IN μg/m^3 to Units of μg/kg/day for the Takenaka Rat Bioassay

	Low	Middle	High
Nominal dose (μg/m^3)	12.5	25	50
Measured dose [a] (μg/m^3)	13.4	25.7	50.8
Average weight at [b] 18 Months (Kg)	.4246	.4376	.4243
Volume air inhaled [c] per day (m^3)	.2538	.2589	.2537
Experimental dose [d] (μg/kg/day)	8.01	15.21	30.41
Continuous lifetime [e] dose (μg/kg/day)	6.01	11.41	22.78

[a] Measured concentrations reported by Takenaka.
[b] Average Weight for each rat group at 18 months.
[c] Calculated as 1=.105×(W/.113) 2/3, where W is the average weight of the rats.
[d] Calculated as (Measured Dose×Volume Air Inhaled)/Weight.
[e] Calculated as Dose (μg/kg/day)×.75.

Like the experimental dose received by the rats, the occupational doses received by humans must be converted into units of μg/kg/day and adjusted to equivalent continuous lifetime doses. Assuming that a worker weighing 70 kg inhales approximately 10 $m^3$of air during an eight hour shift, then for any exposure level Y of cadmium measured in μg/m^3, the worker's dose measured in μg/kg/day is given by

$$\text{Dose}(\mu g/kg/day) = \frac{Y(\mu g/m^3) \times 10(m^3)}{70(kg)}$$

This dose is converted to an equivalent continuous lifetime dose assuming exposure occurs for 250 days per year for 45 years per 74 year lifetime. Doses in units of μg/kg/day and corresponding continuous lifetime doses are presented in Table VI–B for various occupational exposure levels measured in μg/m^3.

TABLE VI–B—DAILY DOSE IN sG/KG/DAY AND EQUIVALENT CONTINUOUS LIFETIME DOSE FOR VARIOUS OCCUPATIONAL EXPOSURE LEVELS OF CADMIUM

Occupational exposure level (μg/m^3)	Daily dose [a] (μg/kg/day)	Continuous dose [b] (μg/kg/day)
1	.1429	.0595
5	.7143	.2975
10	1.4286	.5950
20	2.8571	1.1900
40	5.7143	2.3801
50	7.1429	2.9751
100	14.2857	5.9502
200	28.5714	11.9004

[a] Assumes worker weighs 70 kg and inhales approximately 10 m^3 per 8-hour work shift.
[b] Assumes worker is exposed 250 days per year for 45 out of 74 years.

3. Statistical Models for Low Dose Extrapolation of Risk

While OSHA has consistently evaluated a variety of models for quantitative risk assessment, it has relied primarily upon the multistage model of carcinogenesis to provide its "best estimate" of risk from experimental animal data. This model, from a theory proposed by Armitage and Doll in 1961, is a mechanistic model based on the biological assumption that cancer is induced by carcinogens through a series of stages. It is generally considered to be a conservative model because it assumes no threshold, (i.e. any exposure to a carcinogen is associated with some excess risk), and because it is approximately linear at low doses.

A special case of the multistage model is the one-hit model. It, too, is a mechanistic model, but it is based on the assumption that there is only one stage in the carcinogenic process. Like the multistage model, the one-hit model is linear at low doses, but at moderate and high doses the model is concave. Consequently, the one-hit model does not provide a good fit to many sets of empirical data. At low doses, the one-hit model will, in general, predict risks which are larger than those predicted by a multistage model of two or more stages.

Another type of model is the tolerance distribution model. This type of model is

based on the assumption that for each individual in a population, some critical level of exposure to a carcinogen is required before a tumor will develop. It is these individual thresholds which are modeled by the tolerance distribution models. The probit, logit, and Weibull models are all tolerance distribution models. All predict dose-response curves which are sigmoid in shape. At low doses, these models are not linear. Rather, they approach zero more quickly than does the multistage model, so each will predict smaller risks at low doses than the risks predicted by the multistage model. The probit model will predict the smallest risks of all because it approaches zero the fastest.

While OSHA has examined these models, the Agency prefers to rely on the multistage model for its "best" estimate of risk. OSHA believes that the multistage model has the best empirical and theoretical justification of all low-dose extrapolation models, and because it is a conservative, non-threshold model, OSHA believes that the use of the multistage model in quantitative risk assessment is prudent public health practice.

OSHA has received the following comment:

> * * * [T]here is considerable scientific dispute concerning dose-response models, and * * * no particular model is likely to be preferred in all cases. The Office of Science and Technology Policy (OSTP) has cautioned that '[n]o single mathematical procedure is recognized as the most appropriate for low-dose extrapolation in carcinogensis.' (50 FR 10378.) [sic] Moreover, 'if background additivity is assumed * * * then all models are essentially linear in the low-dose region.' (50 FR 10439). [sic] The multistage model has the additional attribute of approximate linearity across the entire range of doses from zero to the current PELs. OSHA * * * [should request] comments as to whether this peculiar feature of the multistage model is desirable or undesirable in the context of estimating low-dose risks from occupational cadmium exposure.
>
> OSHA has analyzed five alternative dose-response models to the Takenaka rat data. At the exposure levels contemplated by OSHA's alternative proposed PELs of 1 $\mu g/m^3$ and 5 $\mu g/m^3$, predicted lifetime excess cancer risks vary by a factor of more than 100 depending on the model used to derive the estimate. Thus, OSHA's determination of significant risk is acutely sensitive to the mathematical model selected to estimate low-dose risks. OSHA * * * [should request] comments as to whether there is a biological basis for preferring any particular model for estimating low-dose cancer risks from inhaled cadmium. In the absence of any such biological basis, OSHA must rely heavily on statistical criteria. Thus, OSHA also * * * [should request] comments as to what statistical criteria are appropriate for comparing dose-response models.

4. Estimates of Risk

Table VI–C presents estimates of the number of deaths from cancer due to occupational exposure to cadmium at a variety of levels. These estimates were derived from the Takenaka rat data using the various models described in the previous section. Both the maximum likelihood estimate (MLE) and the 95% upper confidence limit (UCL) are presented. The MLE is a point estimate which represents that value which maximizes the likelihood of risk. The 95% UCL represents a plausible upper bound below which the true risk is likely to be. Estimates are presented as deaths per 10,000 workers.

The probit, logit, and Weibull models were fit to the data using the computer program Risk 81 developed by J. Kovar and D. Krewski. The multistage and one-hit models were fit to the data using a version of R.B. Howe and K.S. Crump's computer program Global 83 adapted for the microcomputer by M.S. Cohn of the U.S. Consumer Product Safety Commission. The multistage model was fit to the data by constraining the number of stages to be no greater than the number of nonzero dose levels (i.e. three). The model predicted a two stage process; the dose coefficients Q(O) and Q(3) were both zero.

In its publication "Chemical Carcinogens: A Review of the Science and its Associated Principles," the Office of Science and Technology Policy (OSTP) wrote "[n]o single mathematical procedure is recognized as the most appropriate for low-dose extrapolation in carcinogenesis" (Ex. 8–693). OSHA agrees with this position and recognizes that there is debate within the scientific community concerning dose-response models. OSTP has held, however, that "when data and information are limited, and when much uncertainty exists regarding the mechanisms of carcinogenic action, models or procedures which incorporate low-dose linearity are preferred when compatible with limited information." A close examination of Table VI–C shows that only the multistage model and the one-hit model, a special case of the multistage model, are linear at low doses. Thus, OSHA's preference for the multistage model is supported by OSTP. OSTP has noted that "if background additivity is assumed, i.e., if it is presumed that there is a common mechanism of tumor induction, then all models are essentially linear in the low-dose region," but in this case, however, background additivity can not be assumed because there was no background incidence of lung cancers among Takenaka's rats. OSHA seeks comment on the importance of low-dose linearity in selection of low-dose extrapolation models in general and for cadmium in particular.

Table VI–C demonstrates the range of risks predicted by the various models. For occupational doses less than 100 $\mu g/m^3$, the one-hit model gives the largest estimates of risk while the probit model gives the smallest estimates of risk. At an occupational dose of 200 $\mu g/m^3$, however, only the logit model gives higher estimates of risk than the probit model. Regardless of which model one chooses as "best", it is clear that the risk of cancer at the current OSHA PEL of 100 $\mu g/m^3$ for cadmium fume is unacceptably high. At an exposure level of 100 $\mu g/m^3$, these models predict risks ranging from 1862/10,000 to 2660/10,000. Each model shows that a reduction of the PEL to 5 $\mu g/m^3$ will lead to a significant reduction in risk.

At the two levels OSHA is proposing as its TWA PEL,5 $\mu g/m^3$ and 1 $\mu g/m^3$, the estimates of risk vary by a factor of more than 100 across the models considered. The estimates in Table VI–C show that the TWA PEL selected by OSHA for its final rule will depend upon the model OSHA selects for its "best"estimate of risk. Statistically, there is no way to determinewhich model fits best because the goodness-of-fit chi-square may be used only to determine whether or not a model fits the data, and it can not be used to determine whether one model fits the data better than another. Therefore, OSHA must rely on some other criteria for preferring one model over others. As stated above, OSHA prefers the multistage model because it is abiologically based model and it is linear at low doses. The Agency seeks comment on its preference for the multistage model on these grounds and on what criteria should be used to select a low-dose extrapolation model for its cadmium risk assessment.

The estimates presented in Table VI–C are risks associated with occupational exposure to cadmium particles approximately 0.5 μm in diameter. Particle deposition and thus internal dose depends upon particle size and particle size distribution. At any given dose, larger particles may be associated with lower risks of lung cancer because fewer particles are deposited in the lung, and smaller particles may be associated with higher risks because more particles are deposited in the lung.

In addition, various cadmium compounds (e.g. cadmium oxide, cadmium chloride, etc.) also differ in their solubility and may therefore differ in the bioavailability of cadmium.

Nonetheless, since the carcinogenic agent is cadmium and not the ion to which it is bound, it is not clear that less soluble compounds are less toxic. Without specific human absorption data on various cadmium compounds, no adjustment can be made to dose for varying solubility.

In any event, if cadmium is a site-of-contact carcinogen, asindicated by the induction of lung cancer following inhalationof several cadmium compounds, relative insolubility may not reduce the potential of a carcinogenic response. Indeed, one could speculate that the more insoluble forms of cadmium may result in a greater lung cancer risk because they are in contact with lung tissue for a longer period. Conversely, the moresoluble forms may remain at the portal of entry for a shorter period of time but interact to a greater degree with tissues of more distant organs. OSHA requests comment and empirical data concerning differences in particle absorption, toxicity, and carcinogenicity for the major cadmium compounds.

TABLE VI-C.—ESTIMATES OF EXCESS CANCER DEATHS PER 10,000 WORKER WITH 45 YEARS OCCUPATIONAL EXPOSURE TO CADMIUM [a,b]

Dose (μg/m³)	Multistage model [c]	One-hit model [d]	Probit model [e]	Logit model [f]	Weibull model [g]
200	4385 (5276)	4612 (5410)	4684 (5520)	4694 (5569)	4449 (5285)
100	2213 (3122)	2660 (3225)	1862 (2778)	1898 (2803)	2097 (2991)
50	1091 (1707)	1433 (1769)	441 (990)	584 (1143)	898 (1570)
40	868 (1390)	1164 (1442)	246 (635)	389 (824)	676 (1256)
20	429 (721)	598 (749)	27 (106)	106 (277)	276 (605)
10	213 (367)	304 (382)	2 (9)	28 (88)	111 (281)
5	106 (185)	153 (193)	0 (0)	7 (27)	45 (128)
1	21 (37)	31 (39)	0 (0)	0 (2)	5 (19)
X^2	3.00	3.63	1.52	1.59	2.58
Degrees of freedom	2	3	1	1	1
P-value	>.25	>.50	.22	.21	.11

[a] Estimates derived using data from the Takenaka rat bioassay.
[b] Numbers in parentheses are the 95% Upper Confidence Limits.
[c] Parameters given as q(0)=0; q(1)=3.559E-2; q(2)=1.065E-3; q(3)=0.
[d] Parameters given as q(0)=0; q(1)=5.197E-2.
[e] Parameters given as A=2.983; B=1.173; C0.
[f] Parameters given as A=4.871; B=1.917; C0.
[g] Parameters given as A=3.806; B=1.323; C0.

C. Estimates of Cancer Risk Derived from Human Data

1. Choice of Data Base for Quantitative Risk Assessment

The best available human data for quantifying the lung cancer risk associated with cadmium exposure is found in the mortality study of a cohort of cadmium smelter workers conducted by Thun et al (Ex. 4–68). This study provides the strongest evidence of a cadmium-induced carcinogenic response in humans, and it has sufficient exposure data to demonstrate a dose-response relationship. The study, described above, is a historical prospective study of 602 white men employed in a production area of the smelter for at least six months between 1940 and 1969. Follow-up continued through 1973.

Prior to 1926, the cadmium smelter functioned as an arsenic smelter. Because arsenic is a known risk factor in lung cancer, a sub-cohort of 26 men hired prior to 1926 were examined and found to have a statistically significantly elevated incidence of death due to lung cancer (Obs=4:Exp=.56: SMR=714). This and all expected incidences are based on calendar time, age-specific respiratory cancer death rates for U.S. white males. To control the influence of this potential confounder, the sub-cohort of 26 men hired prior to 1926 were excluded from further analyses.

Among the 576 workers hired after 1926, an elevated incidence of death due to lung cancer was observed, (Obs=16; Exp=10.87; SMR=147), but it was not statistically significant. When the analysis was restricted to workers with two or more years employment at the smelter, however, the elevated incidence of lung cancer deaths was statistically significant (Obs=16; Exp=7.0; SMR=229).

Thun divided his post-1926 cohort into three groups. The low dose group consisted of workers whose cumulative exposure was less than or equal to an equivalent 8 hour TWA exposure of 40 μg/m³ for 40 years. The middle dose group consisted of workers whose cumulative exposure was greater than the low dose group but less than an equivalent 8 hour TWA exposure of 200 μg/m³ for 40 years. The high dose group consisted of workers whose cumulative dose was in excess of an equivalent 8 hour TWA exposure of 200 μg/m³ for 40 years.

With the cohort divided into three dose groups, a dose-response relationship between cadmium and lung cancer became apparent. For the low dose group, 2 deaths due to lung cancer were observed while 3.77 were expected (SMR=53). For the middle dose group, 7 deaths due to lung cancer were observed while 4.61 were expected (SMR=152). For the high dose group, 7 deaths due to lung cancer were observed while 2.50 were expected (SMR=280). Workers in the low dose group had fewer lung cancer deaths than expected, but the middle and high dose groups had more than expected, and the ratio of observed to expected increased with dose. These data, along with other data relevant to quantifying risk, are presented in Table VI–D.

TABLE VI-D.—DATA USED FOR ESTIMATING RISKS FROM A MORTALITY STUDY OF CADMIUM SMELTER WORKERS BY THUN ET AL.

Cumulative Exposure (mg/m³-days)	Person Years At Risk	# Lung Cancers Observed	# Lung Cancers Expected [a]	SMR
< 584	7005	2	3.77	53
585–2920	5825	7	4.61	152
>2921	2214	7	2.50	280

Cumulative exposure (mg/m³-days)	TWA equivalent (μg/m³)		Median dose [d] (mg/m³-days)	Continuous dose [e] (μg/m³-years)
	40-year [b]	45-year [c]		
< 584	>40	>36	280	168
585–2920	40–200	36–178	1210	727
>2921	>200	>178	4200	2522

[a] Expected incidence based on calendar time, age-specific death rates for U.S. white males.
[b] Calculated as (cumulative dose × 1000)/(365 × 40).
[c] Calculated as (cumulative dose × 1000)/(365 × 45).
[d] As provided by Thun to EPA.
[e] Calculated as median dose × 1000 × (8/24) × (1/365) × (240/365).

2. Measure of Dose

Thun arrived at an estimate of cumulative dose for each member of his cohort using the industrial hygiene data from the smelter provided by Smith et al (Ex. 4–64). Thun describes his methods for estimating cumulative dose in his paper (Ex. 4–68) and in an addendum to that paper (Ex 4–68a). Below is a brief description of those methods.

For five time periods, pre-1950, 1950–1954, 1955–1959, 1960–1964, and 1965–1976, Smith estimated airborne cadmium concentrations measured as 8 hour TWAs for nine departments in the smelter and for office and laboratories combined (i.e. non-production work areas). Thun classified each of the nine departments into either high or low exposure categories. Then, for each time period, he calculated a weighted average "high exposure dose" from estimates for the high exposure departments, and a weighted average "low exposure dose" from estimates for the low exposure departments. He used the non-production work area exposure estimates as Smith reported them. This resulted in three exposure vectors: one for high exposure, one for low exposure, and one for "non-production work" exposure estimates corresponding to the time periods provided by Smith.

Thun estimated individual cumulative cadmium exposure first by assigning all employment into seven broad work categories and then by determining which exposure vector applied to each work category. High exposure production work and plant maintenance work were the work categories assumed to have high exposures. Low exposure production work, shop maintenance work, and supervisory work in production areas (i.e. foremen) were the work categories assumed to have low exposures. Office work and other work (e.g. guard, laboratory technician, etc.) were the work categories assumed to have "non-production work" exposures.

For each worker, Thun recorded the number of days a worker was employed in each work category during each time period. Dose for that time period and work category was then multiplied by the number of days and summed across all work categories and time periods to calculate cumulative dose in milligram per cubic meter-days (mg/m³-days). So, for example, if a worker spent 100 days in a high exposure production job between 1955 and 1959 where exposure was estimated to be A mg/m³, 100 days in a low exposure production job between 1955 and 1959 where exposure was estimated to be B mg/m³, and 200 days in an office job between 1960 and 1964 where exposure was estimated to be C mg/m³, then that workers cumulative dose would be given by:
$[(100\times A)+(100\times B)+(200\times C)]$mg/m³-days.

Once cumulative dose was estimated for each worker, each worker was assigned to the high, medium, or low exposure group. Thun provided EPA with the median observation of exposure within each group. Following EPA, the median dose, in mg/m³-days, is converted into units of μg/m³-years by multiplying dose by 1000 and dividing by 365. Dose is then converted into a 24 hour continuous dose by multiplying it by 8/24 (exposure was for 8/24 hours) and 240/365 (an employee worked 240 days/years). This last adjustment must be made because Thun computed exposure days on the basis of elapsed calendar time in a work category, not on the basis of working days (i.e. Thun assumed 1 month in a work category meant 30 days of exposure). The median dose for each group is given in Table VI–D. For the purpose of this risk assessment, the 24 hour continuous median dose for each exposure group will be taken as the dose received by the entire exposure group.

3. Statistical Models for Estimation of Risk

The methods used by OSHA to quantify risks from the Thun data closely follow those used by EPA. In addition to the absolute risk model used by EPA, however, OSHA has examined the relative risk model. EPA chose the absolute risk model because it was the simplest model that could be used with Thun's data. OSHA chose to examine both the absolute risk model and the relative risk model because although both are linear models, the two models are based on different assumptions which lead to different estimates of risk.

The absolute risk model starts from the assumption that the absolute risk of lung cancer death attributable to cadmium exposure, $h_e(t)$, is proportional to cumulative dose up to time t, or

$$h_e(t)=\beta X,$$

where X represents cumulative dose up to time t. An individual's total risk of lung cancer death at time t, h(t), is a function of that individual's background

risk of lung cancer at time t, $h_o(t)$, plus the risk due to cadmium exposure, or

$$h(t)=h_o(t)+h_e(t)$$
$$=h_o(t)+\beta X.$$

This model, also known as the additive model, is of the form

$$Y=a+bX.$$

Here, $h_o(t)$ is the intercept (i.e. the risk of lung cancer death with no exposure), and β is the slope of the dose-response line representing the change in absolute risk per unit dose.

If we consider each person year of observation to be an independent Bernoulli trial, (i.e. an event with only two possible outcomes), and sum over all person years of observation for the jth exposure group, then the expected number of lung cancer deaths for the period of observation, $E(O_j)$, is given by

$$E(O_j)=E_j+\beta X_j W_j,$$

where E_j is the expected number of cases assuming no cadmium exposure, (i.e. background), X_j is the median cumulative dose for exposure group j, and W_j is the number of person years of observation. If we assume that the observed number of deaths is a Poisson random variable with expectation given above, then the likelihood of observed results is given by

$$LIK=\prod_{j=1}^{3} [\exp-(E_j+\beta X_j W_j)]\,[EG5J+\beta X_j W_j]^{O_j} / O_j$$

The maximum likelihood estimate of the unknown parameter β is obtained by maximizing the first derivative of the log likelihood with respect to β. The variance of β is given by the inverse of the observed information. (The Fisher's information may also be used; the estimates of the variance given by the observed information and the Fisher's information are very close.) We solve for the parameter β using a Newton-Raphson algorithm, a computing algorithm for finding the root of a polynomial. Estimates of β and its variance derived from the Thun data using the absolute risk model are given in Table VI–E.

The absolute risk model rests on the assumption that the risk attributable to cadmium exposure is dependent only on cumulative dose. This means that for any given dose, the risk of lung cancer attributable to cadmium exposure is constant regardless of age. However, we know that the background risk of lung cancer death increases with age, so an assumption of constant absolute risk implies that the relative risk of lung cancer death for cadmium-exposed individuals decreases with age.

The relative risk model is based on the assumption that the increase in the relative risk of lung cancer death due to cadmium exposure is the product of an individual's background risk at the time t, $h_o(t)$, and the risk attributable to cadmium exposure, $h_e(t)$. As with the absolute risk model, the risk attributed to cadmium exposure is given by

$$h_e(t)=\beta X,$$

where X represents cumulative dose up to time t. An individual's total risk of lung cancer death at time t, h(t), is given by

$$h(t)=h_o(t)+(h_o(t)\,h_e(t))$$
$$=h_o(t)+(h_o(t)\,\beta X).$$

This model can also be written as a linear model where

$$\frac{h(t)}{h_o(t)}=1+\beta X.$$

Here, 1 is the intercept (i.e. the relative risk assuming no exposure), and β is the slope of the dose-response line representing the change in relative risk per unit dose.

If we consider each individual to be an independent Bernoulli trial and sum over all individuals in the jth exposure group, we obtain the expected number of lung cancer deaths for the period of observation, $E(O_j)$, which is given by

$$E(O_j)=E_j+(E_j\beta X_j).$$

Again, E_j is the expected number of cases assuming no cadmium exposure, (i.e. background), and X_j is the median dose for exposure group j. Assuming that the observed number of deaths is distributed as a Poisson random variable with expectation given above, we obtain the likelihood of observed results

$$LIK=\prod_{j=1}^{3} [\exp-(E_j+(E_j\beta X_j)]\,[E_j+(E_j\beta_j)]^{O_j} / O_j$$

As with the absolute risk model, the maximum likelihood estimate of the unknown parameter β is obtained by maximizing the first derivative of the log likelihood with respect to β. The variance of β is given by the inverse of the observed information. We solve for the parameter β using a Newton-Raphson algorithm. Estimates of β and its variance derived from the Thun data using the relative risk model are given in Table VI–E.

TABLE VI–E.—PARAMETER ESTIMATES FROM THE ABSOLUTE AND RELATIVE RISK MODELS USING THE THUN MORTALITY DATA

Parameter	Absolute risk model	Relative risk model
#	6.4118E-7	6.6971E-4
Var (β)	1.1936E-13	1.2696E-7

* Variance is estimated by the observed information.

The relative risk model rests on the assumption that the ratio of the risk of lung cancer death for cadmium-exposed individuals to the risk of lung cancer death for individuals with no cadmium exposure depends only on dose and is constant across age groups. In other words, for any given cumulative dose, the risk of death for a twenty year old exposed individual relative to a non-exposed twenty year old exposed individual relative to a non-exposed twenty year old individual is the same

as the risk of death for a fifty year old exposed individual relative to a non-exposed individual of the same age.

OSHA recognizes that this difference in assumptions may be important in estimating true occupational risk. Thus, OSHA specifically requests comments concerning the significance of assumptions between these models and their applicability for estimating risks from exposure to inhaled cadmium.

After fitting both models to the Thun data, it is reasonable to ask whether or not the fits are good. The standard approach for measuring goodness-of-fit is to perform a chi-square test, in this case calculating the deviation of the number of lung cancers predicted for each exposure group from the number of lung cancers observed in each group. For the absolute risk model, the number of lung cancers predicted for the jth exposure group, $_{oj}$, is given by

$$_{oj}=E_j + \{\beta X_j W_j\},$$

Where E_j is the expected number of lung cancers for the jth exposure group (given in Table VI-D), X_j is the median continuous dose expressed in $\mu g/m^3$-years for the jth exposure group (given in Table VI-D), W_j is the number of person years at risk for the jth exposure group (given in Table VI-D), and β is the estimated parameter for the absolute risk model (given in Table VI-E). for the relative risk model, the number of lung cancers predicted for the jth exposure group, $^{o}_{j}$, is given by

$$_{oj}=E_j + \{E_j\beta X_j\}.$$

Where E_j and X_j are as defined above, and β is the estimated parameter for the relative risk model (given in Table VI-E).

The numbers of lung cancers predicted by each model for each exposure group are presented in Table VI-F. In addition, the predicted SMRs (calculated as the predicted number of lung cancers divided by the expected number of lung cancers times 100) are also presented. Using the numbers presented in Table VI-F, the goodness-of-fit chi-square for the absolute risk model is 1.53 on two degrees of freedom ($.5 > p > .25$). For the relative risk model, the goodness-of-fit chi-square is 1.17 on two degrees of freedom ($.75 > p > .5$). Neither of these chi-square is statistically significant at the .05 level, therefore OSHA concludes that both models provide good fits to the observed Thun data.

TABLE VI-F.—OBSERVED AND PREDICTED LUNG CANCER DEATHS AND SMRS FROM THE ABSOLUT AND RELATIVE RISK MODELS USING THE THUN MORTALITY DATA

Exposure group [a]	#Lung cancers observed	#Lung cancers predicted [b]	
		Absolute risk model	Relative risk model
Low	2	4.5	4.2
Medium	7	7.3	6.9
High	7	6.1	6.7

Predicted SMR c	Exposure group [a]	Observed *SMR*	
		Obsolute risk model	Relative *risk model*
Low	53	120	111
Medium	152	159	149
High	280	243	269

[a] The low exposure group is the group with exposures less than or equal to 584 mg/m³-day. The medium exposure group is the group with exposures between 585 and 2920 mg/m³-days.

b The numbers of lung cancers predictedc by the models are calculated as # lung cancers expected + 16.4118E-7 × continuous dose ($\mu g/m^3$-years) × person years at risk] for the absolute risk model and as # lung cancers expected + [6.6971E-4 x Continuous Dose ($\mu g/m3$ × # lung cancers expected] for the relative risk model.

c Calculated as (# lung cancers predicted)/(# lung cancers expected) for each exposure group and model.

4. Estimates of Risk

Gail describes an approach for estimating the excess risk of cancer death due to constant exposure to environmental carcinogens in the presence of competing risks (Ex. 8–651). This method is easily adapted to estimate the excess risk of lung cancer death due to occupational exposure to cadmium.

Occupational dose was first converted to continuous dose on the assumption that exposure occurs for 8/24 hours and 240/365 days. OSHA assumed further that exposure begins at age 20 and continues at a constant level for 45 years, and that life expectancy is 74 years. OSHA used 1984 U.S. male age-specific death rates for all causes, and the 1982 U.S. male age-specific lung cancer deaths rates for all races. The lung cancer death rates, which were given for five-year age intervals, were assumed to be constant throughout each interval.

Let d_i = the cumulative dose received at the midpoint of the ith age interval. Thus, for example, if an individual is exposed at a constant level X from age 20 on, then at age 24, d_{24} would equal 4.5X. From age 65 on, cumulative exposure would be 45X. Let $q_x(i)$ = the probability of death from all causes at age i, and let $q_L(i)$ = the probability of lung cancer death at age i. Using the absolute risk model and the MLE of β derived from this model above, the lifetime excess risk of lung cancer due to 45 years of occupational exposure to cadmium is given by

$$\sum_{i=20}^{74} \beta d_i \exp[-\sum_{j=20}^{i} \beta d_i + q_x(j).$$

Using the relative risk model and the MLE of β derived from this model above, the lifetime excess risk of lung cancer due to 45 years of occupational exposure to cadmium is given by

$$\sum_{i=20}^{74} \beta d_i q_L(i) \exp[-\sum_{j=20}^{i} \beta d_j q_L(j) + q_x(j).$$

Table VI-G presents estimates of excess deaths derived from the Thun data using the method described here. In addition, 95% upper confidence limits and 5% lower confidence limits were constructed for each of the MLEs. This was done by replacing β by $\beta \pm K\ 1.645\ \Sigma E(\beta)$ and using the formulas above.

The estimates of excess lung cancer death from the relative risk model are nearly twice as large as those from the absolute risk model, but both models predict significant risk at the current OSHA PEL. At 100 $\mu g/m^3$, these models predict between 16 and 30 excess lung cancer deaths per 1000 exposed workers. At exposure levels as low as 5 $\mu g/m^3$, the excess risk of lung cancer death estimated by these models is about 1 per 1000 exposed workers.

TABLE VI-G.—ESTIMATES OF EXCESS LUNG CANCER DEATHS PER 10,000 WORKERS WITH 45 YEARS OCCUPATIONAL EXPOSURE TO CADMIUM [a,b]

Dose (μg/m³)		# Excess deaths	
TWA	Continuous [c]	Absolute risk model	Relative risk model
200	43.84	323 (37,599)	602 (78,1089)
100	21.92	163 (19,305)	307 (39,566)
50	10.96	82 (9,154)	155 (20,289)
40	8.77	66 (7,123)	125 (16,232)
20	4.38	33 (4,62)	63 (8,117)
10	2.19	16 (2,31)	31 (4,59)
5	1.10	8 (1,15)	16 (2,29)
1	.22	2 (0,3)	3 (0,6)

[a] Estimates derived using data from the Thun mortality study of cadmium smelter workers.
[b] Numbers in parentheses are 5% lower and 95% upper confidence limits.
[c] Assumes exposure occurs for 8/24 hours and 240/365 days.

There are some issues which arise in applying these estimates of risk to populations other than the Thun cohort. Even after 1926, some arsenic exposure continued, but estimates of the exposure among the workers in the cadmium cohort suggest that such exposure made little contribution to the excess lung cancer risk. Thun also reported that there is some evidence that the smoking rate for these workers was less than that for the general white male population that was used to calculate the expected number of deaths in his study (Ex. 8–673).

Estimates of cancer risk due to cadmium exposure have been calculated using both human and animal data. OSHA has presented the methodology used to derive the risk estimates from these data and has indicated the strengths and weaknesses of the data sets and of each estimation technique. OSHA's approach to its quantitative risk assessment is in accord with the Office of Science and Technology Policy's position that "the risk assessment process should not be viewed as strictly 'scientific' in the usual sense of the word. Instead, risk assessment involves a complex blend of current scientific data, reasonable assumptions and scientific judgements that permit decisions to be made in the absence of complete information" (Ex. 8–693).

On the basis of the risk assessment using the Takenaka study and the multistage model, OSHA proposes a PEL of 1 μg/m³. However, there is support for the use of the Thun study of cadmium smelter workers as the basis for establishing an exposure level since no extrapolation across species is required. The estimates of risk derived from the Thun data are lower than those derived from the Takenaka data. OSHA's estimate from the Thun data at 5 μg/m³ (1 to 2 per 1000) is approximately equal to OSHA's best estimate from the Takenaka data at 1 μg/m³ (2 per 1000). OSHA is therefore proposing alternate PELs of 1 μg/m³ and 5 μg/m³ based in part upon these estimates and in part upon the concerns for the technological feasibility of achieving a PEL of 1 μg/m³.

OSHA solicits comments on the quality of the Takenaka and Thun studies, the appropriate risk assessment model to use for each data set, and its choice of the Takenaka study for its best estimate. OSHA also solicits comments (as noted in the list of questions in the introductory portion of this preamble) on the appropriate level for the PEL.

D. Estimates of Risk of Kidney Dysfunction

1. Choice of Data Base for Quantitative Risk Assessment

The effects of cadmium on the kidney are well documented. As discussed above, there are many studies which show a relationship between cadmium exposure and kidney dysfunction. Dysfunction is most commonly manifested as proteinuria, a condition characterized by excess serum proteins in the urine. Proteinuria indicates that damage has occurred to the proximal tubules and/or glomerulus, and because this damage is irreversible and can lead to still more serious health effects, OSHA considers such dysfunction to represent material impairment of health.

OSHA has attempted to quantify the risk of kidney dysfunction due to occupational exposure to cadmium. Two studies of cadmium-exposed workers provide adequate exposure data for such an assessment. The first of these is a study of cadmium smelter workers conducted by Ellis et al (Ex. 4–27). The second is a study of workers at a refrigeration compressor production plant conducted by Falck et al (Ex. 4–28). In both studies, kidney dysfunction is defined as the presence of excess proteins in the urine.

Ellis studied 82 male workers at the same smelter as was studied by Thun. The cohort was comprised of 51 active workers and 31 retired workers with experience in production, non-production, office, and laboratory work. Cumulative exposure estimates were made for each member of the cohort using industrial hygiene data provided by Smith (Ex. 4–64). The chronological record of each worker's job assignments was obtained from personnel files at the smelter. For each worker, the time-weighted inhalation exposure (TWE) was calculated by multiplying the duration of exposure in a given work area (t_i) by the estimated inhalation exposure for that area and year (E_i) and then summing these values to obtain cumulative exposure or

$$TWE = \frac{\Sigma}{i} E_i t_i.$$

Each cohort member completed a health history questionnaire, took a physical exam, gave specimens for blood and urine tests, and provided 24-hour urine samples. The 24-hour urine samples were used to determine whether a worker had abnormal kidney function. Kidney function was judged to be abnormal if urinary levels of the low molecular weight protein β_2-microglobulin exceeded 200 μg/g creatinine or if total urinary protein levels exceeded 250 mg/g creatinine. Eighteen active workers and twenty-three retired workers were classified as having abnormal kidney function. Descriptive statistics for the entire cohort are presented in Table VI–H.

TABLE VI-H.—DESCRIPTIVE STATISTICS FOR A COHORT OF 82 ACTIVE AND RETIRED CADMIUM SMELTER EMPLOYEES [a]

	Normal kidney function	Abnormal kidney function
	Mean (SD) [b]	Mean (SD)
Active Workers		
N	33	18
Age (yrs)	42.6 (13.3)	53.6 (6.8)
Duration of exposure (mos)	141 (118)	264 (105)
TWE [c] (μg/m³-years)	105 (9.0)	1690 (2.7)
Renal cadmium [d] (μg/g)	125 (2.8)	230 (2.0)
Liver cadmium (ppm)	11.3 (2.8)	63.9 (1.5)
Retired Workers		
N	8	23
Age (yrs)	69.0 (8.3)	67.9 (6.9)
Duration of exposure (mos)	342 (75)	329 (103)
TWE [c] (μg/m³-years)	379 (3.3)	3143 (3.6)
Renal cadmium [d] (μg/g)	148 (2.1)	169 (1.7)
Liver cadmium (ppm)	14.0 (3.1)	33.6 (2.9)

[a] Data taken from Ellis et al. (Ex. 4-27).
[b] Mean (Standard Deviation) presented. Means and SDs for age and duration of exposure are arithmetic means and SDs. All others are geometric means and SDs.
[c] Time-weighted inhalation exposure estimate (i.e., dose).
[d] Renal cortex cadmium concentration; assumes 145 g weight for the total kidney and a 1.5 ratio between cortex and total kidney concentration.

Falck studied 33 male workers at a plant which produces refrigeration compressors with silver brazed copper fittings. The silver brazing contained between 18% and 24% cadmium, and compressors were brazed either manually or by an automated process. Estimates of cumulative exposure were made for each worker using data from air monitoring done by the Michigan Department of Industrial Health. Air monitoring had been done at the plant since 1961. The mean estimated cadium exposure on the automated brazing line was $39 \pm 7.8\ \mu g/m^3$ for the 11 year period of operation for which sampling data was available. The mean estimated cadmium exposure on the manual brazing line was $110 \pm 25.5\ \mu g/m^3$ for a 21 year period of operation. Work history records were obtained for each employee in the study, and a time-weighted exposure for each worker was calculated by multiplying the length of time on each brazing line (t_i) by the mean estimated exposure for that brazing line (E_i) or

$$TWE = \sum_i E_i t_i.$$

Each of the 33 workers provided medical histories and spot blood and urine samples. Three workers were dropped from further analysis because of health conditions which affect kidney function. Of the remaining 30 workers, 8 were asked to provide 24-hour urine samples because their urinary glucose, protein, and/or β_2-microglobulin levels exceeded the 95% tolerance limits constructed for these variables from the spot urine samples of 41 unexposed workers who served as controls. Glucose, protein, β_2-microglobulin, and creatinine levels were measured in the 24-hour urine samples of the eight workers and in the 24-hour urine samples of seven age-matched male controls. Seven of the eight workers were found to have urinary protein levels in excess of the 95% tolerance limit constructed for urinary protein from the controls, and these workers were judged to have abnormal kidney function. Descriptive statistics for the cohort are presented in Table VI-I.

TABLE VI-I.—DESCRIPTIVE STATISTICS FOR A COHORT OF 30 EMPLOYEES AT A REFRIGERATION COMPRESSOR PRODUCTION PLANT [a]

	Normal kidney function	Abnormal kidney function	P-Value [b]
	Mean (95% CI) [c]	Mean (95% CI)	
N	23	7	
Age (yrs)	49 (47,51)	53 (51,55)	.13
TWE [d] (μg/m³-years)	459 (332,634)	1137 (741,1737)	.02
Smoking Habits (pack-years)	14 (9,19)	24 (14,34)	.07
Urine Ratios:			
Protein/Creatinine (mg/g)	34 (26,43)	246 (132,456)	<.001
β_2-M/Creatinine (μg/g) [e]	53 (31,90)	6375 (1115,36463)	<.001
Cadmium/Creatinine (μg/g)	11 (10,13)	16 (8,36)	.07
Serum Ratios:			
Creatinine/Serum (mg/100 ml)	1.1 (1,1.2)	1.4 (1.2, 1.7)	.003
β_2-M/Serum (μg/ml)	2 (1.6,2.4)	2.3 (1.8,2.8)	.32

[a] Data taken from Falck et al (Ex. 4-23).
[b] P-value associated with a test of differences between group means.
[c] Mean and 95% confidence intervals are presented. Means for age and smoking habits are arithmetic means; all others are geometric means. Confidence intervals are constructed from arithmetic standard deviations for age and smoking; all others from the geometric standard deviations.
[d] Time-weighted inhalation exposure estimate (i.e. dose).
[e] β_2-M = β_1-microglobulin.

2. Statistical Models for Estimation of Risk

Logistic regression may be used to model the relationship between cadmium exposure and the presence or absence of kidney dysfunction, a dichotomous outcome variable. Logistic regression models are based on the assumption that the probability (p) of an event is distributed as a binomial random variable and that the logic function is linear, or

$$\log(p/1\text{-}p) = \alpha + \beta x.$$

Ellis used this technique to analyze his data. Regressing kidney dysfunction expressed as a (0,1) variable, (0=normal kidney function; 1=abnormal kidney function), on the log of cumulative dose, he obtained the model

$$\log(p/1\text{-}p) = -8.34 + 1.24 \log(\text{dose}), \text{ or}$$
$$p = \text{dose}^{1.24}/[e^{8.34} + \text{dose}^{1.24}].$$

Here, p represents the probability of kidney dysfunction for any given cumulative dose.

Falck did not perform a logistic regression analysis, but he provided the cumulative dose data so that such an analysis could be done. Using Falck's data, OSHA obtained parameter estimates for the logistic model

$$\log(p/1\text{-}p) = -19.75 + 2.78 \log(\text{dose}), \text{ or}$$
$$p = \text{dose}^{2.78}/[e^{19.75} + \text{dose}^{2.78}],$$

where again, p is the probability of kidney dysfunction for any given dose.

3. Estimates of Risk

Using the logistic regression models above, OSHA estimated the risk of kidney dysfunction from 45 years of exposure to a variety of occupational doses. For any hypothetical 8-hour time-weighted average exposure level Y, the cumulative dose measured in $\mu g/m^3$-years was calculated as

Cumulative Dose = Y × 45.

Estimates of risk derived from the Ellis and Falck models for a variety of occupational doses are presented in Table VI–J.

TABLE VI–J.—ESTIMATES OF KIDNEY DYSFUNCTION PER 10,000 WORKERS WITH 45 YEARS OF OCCUPATIONAL EXPOSURE TO CADMIUM

8-hour TWA dose ($\mu g/m^3$)	Cumulative dose ($\mu g/m^3$-yrs)	Incidence of kidney dysfunction	
		Ellis model	Falck model
1	45	261	1
5	225	1646	90
10	450	3177	589
20	900	5237	3005
40	1800	7220	7467
50	2250	7740	8457
100	4500	8900	9741

These logistic regression models are simple and do not take into account the role of other factors which may predict dysfunction. Age and smoking are two such factors. Age is a potential confounder in relating kidney dysfunction to cadmium exposure when dysfunction is measured by urinary protein levels because for men, urinary protein levels increase with age (Ex. 8–618). Cigarettes represent an additional source of cadmium exposure because each cigarette contains approximately 2 μg of cadmium (Ex. 8–668). In order to examine the importance of age and smoking for predicting kidney dysfunction, Falck has provided OSHA with data on these variables for each member of his cohort (Ex. 4–28A).

OSHA analyzed these data using a forward stepwise logistic regression procedure. This procedure allows an investigator to determine which independent variables, (e.g. age, dose, etc.), alone or in combination with one another, best predict some outcome, in this case the probability of kidney dysfunction. As each term is added to the model, a statistic is calculated reflecting the contribution of that term to the predictive value of the model. When no additional term will make a significant contribution, then the model is considered "best".

In its analysis, OSHA considered four independent variables: age, smoking status (current, past, or never), pack-years smoked, and occupational cadmium dose measured as the log of the time-weighted exposures (TWEs). Dose was the only independent variable to make a significant contribution to the predictive value of the model ($p=.0019$). Pack-years made the smallest contribution ($p=.1133$), while the contribution of age and smoking status approached significance but did not achieve it ($p=.0692$ and $p=.0568$ respectively).

The three remaining independent variables, age, smoking status, and pack-years smoked, were added one at a time to the model

$$\log(p/1-p)=a+\beta \log(\text{dose}).$$

No additional term was found to make a significant contribution to the predictive value of the model. This means that once dose is in the model, none of the other independent variables considered make a meaningful contribution to predicting the probability of kidney dysfunction. The finding that this is the best model for predicting dysfunction is consistent with Falck's report that there was no statistically significant difference in age and smoking history between workers with normal kidney function and workers with abnormal kidney functions.

The additional data provided by Falck helps answer some questions about other factors which could possibly affect kidney function in the Falck cohort. In addition, there are other variables which may play some role in kidney dysfunction and which may have been overlooked. Duration of exposure, for example, is one such factor. In a study of 37 cadmium smelter workers, Gompertz et al found that a small group of workers with an average of 4.6 years of exposure had high liver cadmium concentrations but no evidence of renal dysfunction (Ex. 4–32). For workers with more than 10 years of cadmium exposure, elevated liver cadmium concentrations were associated with kidney dysfunction. Duration of exposure, however, will be closely correlated with cumulative dose, and indeed, this covariate may be in these models by proxy.

Table VI–J shows that up to a dose of approximately 35 $\mu g/m^3$ (1600 $\mu g/m^3$-years), the Ellis model predicts risks which are higher than those predicted by the Falck model. For doses greater than 35 $\mu g/m^3$, the Falck model predicts higher risks. It is possible that the Ellis model may be over predicting risks at low doses. One member of the Ellis cohort with a cumulative exposure of only 51 $\mu g/m^3$-years had abnormal kidney function, and, as acknowledged by Ellis, this worker perhaps should have been excluded from the analysis. The worker was an 82 year old retired office worker who had not worked for fifteen years. His level of urinary β_2-microglobulin was just slightly elevated over the 200 mg/g creatinine limit. This worker was the only member of the cohort with abnormal kidney function at a cumulative exposure level of less than 400 $\mu g/m^3$-years.

Because one observation may be very influential in a logistic regression, OSHA attempted to reproduce Ellis's analysis excluding this one case. Although the Agency was unable to obtain the raw data used by Ellis, by visual review of the graphs presented in the Ellis paper OSHA was able to reproduce dose data for each member of the cohort (Ex. 4–27A). To measure the accuracy of the eyeballed estimates, a logistic regression was run with all of the reproduced data. The Agency obtained the parameter estimates: $a=-8.29$ and $\beta=1.24$. These are very close to the parameter estimates of $a=-8.34$ and $\beta=1.24$ reported by Ellis.

A second logistic regression was run with the reproduced data excluding the case described above. Without this case, OSHA obtained the model

$$\log(p/1-p)=-10.83+1.59 \log(\text{dose}), \text{ or}$$
$$p=\text{dose}^{1.59}/[e^{10.83}+\text{dose}^{1.59}].$$

where, p represents the probability of kidney dysfunction for any given cumulative dose. This model will be referred to as the Ellis/OSHA model. Estimates of risk derived from this model for a variety of occupational doses are presented in Table VI–K.

TABLE VI–K.—ESTIMATES OF KIDNEY DYSFUNCTION PER 10,000 WORKERS WITH 45 YEARS OF OCCUPATIONAL EXPOSURE TO CADMIUM DERIVED FROM THE ELLIS/OSHA MODEL

8-hour TWA dose ($\mu g/m^3$)	Cumulative dose ($\mu g/m^3$-yrs)	Dysfunction incidence
1	45	83
5	225	981
10	450	2467
20	900	4965
40	1800	7480
50	2250	8089
100	4500	9272

At low doses, the estimates derived from the Ellis/OSHA model are much closer to those derived from the Falck model than are the estimates derived from the original Ellis model. The results in Table VI–K indicate that the one case

was an influential observation. OSHA is unwilling, however, to rely upon the Ellis/OSHA model instead of the original Ellis model for its risk estimates. The Ellis/OSHA model was obtained using crude estimates and not the actual data. Furthermore, because a case is unusual does not mean it should be excluded. Rather, its role as an influential observation should be examined and acknowledged. OSHA notes that Ellis was aware that this case was unusual, yet he did not exclude it from his analysis. Therefore, OSHA does not believe it should be excluded.

While the logistic regression technique is useful for regressing dichotomous variables such as normal/abnormal kidney function against continuous variables such as dose, the models derived from the Ellis data and the Falck data can not reliably predict the risk of kidney dysfunction at low doses. Part of the reason for this is that both data sets have very small sample sizes. As sample size increases, the uncertainty associated with any logistic regression estimates decreases.

The most important reason that these models can not reliably predict the risk of kidney dysfunction at low doses, however, is that these are non-threshold models whereas cadmium-induced kidney dysfunction is known to be a threshold effect. For any level of cadmium exposure, regardless how small, the models derived from the Ellis and Falck data will predict some risk. This is contrary to the evidence which indicates that there must be a minimum cadmium burden in the kidneys before cadmium-induced dysfunction can occur. The cadmium burden necessary to induce dysfunction may vary from person to person, and the cumulative exposure level necessary to achieve that burden may also depend upon individual sensitivity.

Ellis acknowledged the limitation of his logistic regression model when he wrote that the model was used "to investigate the concept of an allowable limit for the inhalation exposure estimate such that exposures beyond this value would ultimately be associated with renal dysfunction." Thus, Ellis chose this model to determine the air concentration level below which workers would be safe from kidney dysfunction. OSHA seeks comment on the use of the logistic regression for estimating the risk of kidney dysfunction.

An alternative model for determining the air concentration level of cadmium associated with the kidney dysfunction threshold is the two phase linear model used by Mason et. al., (Ex. 8–669). This allows two linear models with two different slopes to be fit to the same data set at the same time. The advantage of this model is that dose is related to various biological indicators (e.g., β_2-microglobulin, albumin, etc.) instead of the dichotomous outcome variable normal/abnormal kidney function. Therefore, a medical doctor does not have to make an *a priori* determination of who is normal and who is not, as both Ellis and Falck did for their respective cohorts.

Interpretation of the two phase linear model, however, is difficult. The model can only tell us at what dose the relationship between exposure and the biological indicators changes significantly. It can not tell us at what dose the risk of illness is unacceptably high.

OSHA has not been delegated the responsibility of performing original research to determine the biological basis for threshold effects of kidney dysfunction. OSHA relies upon the research of others to assess the damage resulting from cadmium diffused in work environments. Any inferences OSHA makes are deducible from the experiments of others by classical, statistical methodology. The experiments upon which OSHA's inferences are based are clearly set forth in the proposed standard. OSHA seeks comment on the use of the above mentioned models or any other model for the estimation of risk of kidney dysfunction due to occupational exposure to cadmium, including a discussion of advantages and drawbacks to the models.

OSHA believes that the logistic regression models derived from the data from the Ellis and Falck cohorts are adequate for quantifying the risk of kidney dysfunction due to occupational exposure to cadmium. OSHA is impressed by the consistency of these risk estimates derived from workers in two different industries by two independent investigators. Although the models predict varying risks at very low doses, at doses as low as 5 $\mu g/m^3$, (225 $\mu g/m^3$ years) both models predict risks in excess of 1 per 1000. For cumulative doses greater than 300 $\mu g/m^3$-years, (approximately 7 $\mu g/m^3$ as an 8-hour TWA for 45 years), the risks predicted by these models differ by less than a factor of ten. At the current OSHA PEL of 100 $\mu g/m^3$, both models predict unacceptably high risks for 45 years of occupational exposure.

OSHA has received the following comment:

OSHA [should request] comment on the appropriate threshold model to use to estimate the risk of renal dysfunction. If the underlying health effects in fact behave according to a threshold, any non-threshold probability model will invariably overestimate risks at low doses and underestimate risks at high doses. Given that two of the epidemiologic studies of cadmium-related renal dysfunction [Falck *et al.* (1985); Mason *et al.* (1988)] suggest that cadmium-related renal dysfunction is a threshold event, is there a biological basis for estimating renal dysfunction risks using non-threshold models? What is the magnitude of bias introduced by using a non-threshold model?

Mason *et al.* (1988) found that analysis of several relevant biochemical variables (urinary total protein, albumin, beta-2 microglobulin, and retinol binding protein) indicated that the excess risk threshold for cadmium was approximately 1,100 $\mu g/m^3$-years. This value corresponds to a 45-years 8-hour PEL of 16 $\mu g/m^3$. Are there any epidemiologic studies that argue for locating the threshold at a point lower than 16 $\mu g/m^3$? Is there a scientific basis for concluding that cadmium exposures below 16 $\mu g/m^3$ constitute a significant risk of renal dysfunction?

Logistic regression is one many statistical methods normally reserved for qualitative dependent variables. Observations may fall in only one of two (or more) categories. Generally, these categories correspond to objectively observable phenomena. In OSHA's analysis of renal dysfunction risks, the dependent variable is a derived index of several continuously measured variables (e.g., beta-2 microglobulin, albumin). To classify workers into the alternative states of function and dysfunction, OSHA established subjective thresholds for each criterion variable [sic] used to construct the index (e.g., beta-2 microglobulin > X, albumin > Y). OSHA * * * [should request] comment on the statistical validity of subjectively transforming continuously measured variables into an index, then using the index as a qualitative and meaningful dependent variable. How do the indices used by Ellis differ from those used by other researchers, such as Falck *et al.* (1985) and Kjellstrom *et al.* (1977)? Is there a scientific consensus or a divergence of opinion as to what thresholds constitute renal dysfunction? How would OSHA's estimates of renal dysfunction risk vary depending on these thresholds?

Mason *et al.* (1988) uses a two phase linear model (i.e., kinked) [sic] to identify the most likely location of the threshold. What other estimation methods exist for threshold-related phenomena?

In each of the epidemiologic studies concerning renal dysfunction there were confounding factors, including occupational exposures to other substances and cigarette smoking. Kjellstrom *et al.* (1977) reported an average 19 percent incidence of renal dysfunction associated with exposures of approximately 50 $\mu g/m^3$ for 6–12 years. However, this figure masks a statistically significant difference in incidence between smokers and non-smokers. For an exposure range of 10 to 122 $\mu g/m^3$, Kjellstrom reported an incidence of 100 percent for two cohorts of non-smokers and 0 percent for a third. For similarly exposed smokers, however,

Kjellstrom reported incidence rates of 22 percent for two cohorts and 29 percent for the third. [See Table 6 in Kjellstrom *et al.* (1977).] Smoking also was a confounding factor in Falck *et al.* (1985). Falck reported that workers classified as having abnormal renal function smoked an average of 24 pack-years, whereas workers classified as having normal renal function smoked an average of 14 pack-years. Falck dismissed the confounding effects of smoking by noting that this difference was not statistically significant using a two-tailed test and alpha =.05. Would a one-tailed test have been more appropriate? What do these results suggest as to the effect of cadmium exposure *independent* of smoking?

Another confounding factor in Kjellstrom is that workers were simultaneously exposed to nickel hydroxide [$Ni(OH)_2$] dust as well as cadmium. According to Kjellstrom, nickel hydroxide also causes proteinuria, and nickel hydroxide concentrations were typically two to ten times *greater* than cadmium oxide levels. What do these results suggest as to the effect of cadmium exposure *independent* of nickel hydroxide? Does OSHA's risk model appropriately capture the *independent* effects of cadmium exposure by controlling for confounding factors, such as smoking and exposure to other substances?

E. Other Estimates of Risk

Under contract to OSHA, two quantitative assessments of the risks associated with occupational exposure to cadmium were prepared jointly by Meridian Research and Roth Associates (Ex. 16–A and Ex. 16–B). The first of these deals with cancer risks. For occupational exposure at the current OSHA PEL of 100 $\mu g/m^3$ for 45 years, Meridian and Roth predicted 2130 excess cancer deaths per 10,000 exposed workers based on the rat data and a range from 167 to 339 excess cancer deaths per 10,000 exposed workers based on the Thun data. In their assessment of kidney dysfunction risks, Meridian and Roth predicted a range from 1292 to 9743 cases of kidney dysfunction per 10,000 workers exposed at 100 $\mu g/m^3$ for 45 years.

OSHA is in the process of reviewing these risk assessments. They have been placed in the OSHA cadmium docket and are available for public review and comment.

VII. Significance of Risk

In the 1980 benzene decision, the Supreme Court, in its discussion of the level of risk that Congress authorized OSHA to regulate, indicated when a reasonable person might consider a risk significant and take steps to decrease it. The court stated:

> It is the Agency's responsibility to determine in the first instance what it considers to be a "significant" risk. Some risks are plainly acceptable and others are plainly unacceptable. If, for example, the odds are one in a billion that a person will die from cancer by taking a drink of chlorinated water, the risk clearly could not be considered significant. On the other hand, if the odds are one in a thousand that regular inhalation of gasoline vapors that are 2 percent benzene will be fatal a reasonable person might well consider the risk significant and take the appropriate steps to decrease or eliminate it. (*I.U.D.* v. A.P.I., 448 U.S. 607, 655).

The Court further stated that "while the Agency must support its findings 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." The Court added that the significant risk determination required by the OSH Act is "not a mathematical straitjacket," and that "OSHA is not required to support its findings 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 overprotection rather than underprotection" (448 U.S. at 655, 656).

As part of the overall significant risk determination, OSHA considers a number of factors. These include the type of risk presented, the quality of the underlying data, the reasonableness of the risk assessments, the statistical significance of the findings and the significance of risk (48 FR 1864; January 14, 1983).

Cadmium exposure causes a number of extremely serious adverse health effects. In 1971 OSHA adopted the ANSI standard with a TWA PEL of 100 $\mu g/m^3$ for cadmium fume and a TWA PEL of 200 $\mu g/m^3$ for cadmium dust to prevent the acute effects caused by exposure to cadmium at levels higher than these. Since 1971, however, a body of evidence has developed which shows that exposure to any cadmium, dust or fumes, at levels below these TWA PELs can also lead to very serious health effects such as kidney dysfunction and cancer. Because current occupational cadmium exposure levels generally are below 100 $\mu g/m^3$, this discussion of the significance of risk focuses exclusively on these non-acute effects.

As indicated in Section V, the health effects section of this preamble, exposure to cadmium causes cancer, kidney dysfunction, reduced pulmonary function, and chronic lung disease indicative of emphysema. Other health effects, such as improper bone mineralization have been reported. In addition to these major effects in humans, studies of experimental animals suggest that exposure to cadmium may also cause anemia, change in liver morphology, decrease in immunosuppression, and hypertension.

As discussed in Section V, there are numerous epidemiologic studies which show an elevated risk of lung cancer among cadmium exposed workers. Because lung cancer is almost certainly fatal, OSHA considers this disease to represent the greatest material impairment to health. A number of studies of workers also suggest an association between occupational cadmium exposures and increased deaths from other cancer, most notably prostate cancer. The relationship between cadmium exposure at low levels and prostate cancer, however, is difficult to establish. Most epidemiological investigations use mortality rates to estimate incidence rates, but because prostate cancer does not always lead to death, most studies probably underestimate the true incidence of the disease. Although prostate cancer is not always fatal, OSHA nonetheless considers it to be a very serious material impairment to health.

Chronic exposure to cadmium is also known to cause renal dysfunction. This impairment of kidney function typically is manifested as proteinuria, a condition characterized by an excess of serum proteins in the urine. The damage to the proximal tubules and/or glomerulus in the kidney indicated by proteinuria is irreversible. Because of the body's ability to accumulate and store cadmium over long periods of time, the loss of kidney function may develop even after a reduction or cessation of external cadmium exposure. Upon prolonged exposure, tubular proteinuria may progress to more severe forms of renel dysfunction such as glycosuria, aminoaciduria, phosphaturia and glomular proteinuria. OSHA therefore also considers tubular proteinuria to be a material impairment of health.

Long term exposure to cadmium appears to cause other adverse effects on the respiratory system in addition to lung cancer. Workers with prolonged exposure to cadmium dust or fumes have exhibited shortness of breath, impaired pulmonary function associated with poor physical working capacity, and chronic lung disease indicative of emphysema. Workers with progressive forms of proteinuria have exhibited adverse bone effects associated with improper bone mineralization, such as osteoporosis and osteomalacia.

These other disease risks are serious, though not usually fatal. However, this discussion of significant risk concentrates on the cancer risk and concludes that that risk is significant in and of itself. The risk of permanent impairment of the kidney, which is quantifiable and is included in this discussion, also poses a significant risk. The other risks, though not as readily quantifiable, add to the significance of risk presented.

The underlying epidemiologic and experimental animal studies that provide the basis for this quantification of risk are of reasonable quality and demonstrate a relationship between cadmium exposure, on the one hand, and cancer and kidney dysfunction, on the other. There is a reasonable basis for determining the exposed population, estimating dose, and excluding other potentially causal agents of the observed diseases. The Environmental Protection Agency (EPA) has concluded that the available data are adequate to quantify the risk of cancer due to cadmium exposure. This is OSHA's preliminary conclusion as well.

OSHA has received the following comment:

> The epidemiologic study by Thun *et al.* (1985), however, provides the strongest evidence of a carcinogenic response in humans, and has sufficient exposure data to demonstrate a dose-response relationship. Given that Takenaka obtained only lung cancers, is the Takenaka study in fact superior than [sic] the available epidemiologic studies for estimating cancer risks at other sites? Should the Takenaka animal bioassay be judged superior to the Thun epidemiologic study for assessing human risk because of weaknesses in the exposure data in the Thun study?"

OSHA used two data sets for its quantitative assessment of the risk of death from cancer. One set was from the rat bioassay by Takenaka and others (Ex. 4–67). The other is from the human mortality study by Thun and others (Ex. 4–68). For its preliminary quantitative risk assessment, the Agency has relied on the rat data for its best estimate of total risk of cancer death because OSHA believes the measures of exposure are more accurate in the rat study and because the rat study can be used to predict all cancer deaths attributable to occupational exposure to cadmium. By contrast, the Thun data can be used to predict only lung cancer deaths attributable to occupational exposure to cadmium. This use of animal data to predict total cancer deaths is consistent with risk assessments conducted for other standards and upheld in the Courts (e.g. ethylene oxide).

OSHA requests comment on its preliminary preference for the Takenaka study, and on the criteria that OSHA should use in the final rule to determine its preference, if a preference is appropriate, for any particular animal or epidemiological study. OSHA is interested in comments regarding the tradeoff in strengths and weaknesses in the uncertainties in quantitative risk assessment using the various studies.

In Section VI, OSHA discusses at length its risk assessments for cadmium, including the bases and criticisms of those assessments. Although OSHA prefers the multistage model for its best estimates of risk, the Agency has also fit several other models to the experimental animal data to obtain a range of estimates of risk of cancer death attributable to occupational exposure to cadmium over a working lifetime of 45 years. At 100 $\mu g/m^3$, the current OSHA TWA PEL for cadmium fume, the excess risk of death from cancer ranges under the various models from 186.2 to 266 per 1,000 workers. Even at the lowest point in this range, the estimate of excess risk of cancer death is significant and unacceptably high. The Agency's best estimate of total cancer risk is 221.3 per 1,000 (Table VI–C, above).

OSHA requests comments on whether the estimates based on the multistage model and reflected in Table VI–C, above, are the best estimates of risk. The multistage model is a mechanistic model based on the biological assumption that cancer is induced by carcinogens through a series of stages. The model generally is considered conservative in the sense that it risks error on the side of over protection rather than under protection, because it assumes no threshold for carcinogenesis and because it is approximately linear at low doses. OSHA believes that the use of such a model is prudent public health practice.

Using the multistage model, OSHA projects that under current employment conditions at current exposure levels, 824 cancer deaths attributable to cadmium exposure will occur among cadmium-exposed workers over their working lives. Using the same model, OSHA estimates that a reduction of exposure levels to meet a TWA PEL of 5 $\mu g/m^3$ would reduce the number of cancer deaths attributable to cadmium exposure to 652, a reduction of 21%. Reducing exposure levels further to meet a TWA PEL of 1 $\mu g/m^3$ would reduce the number of cancer deaths attributable to cadmium exposure to 186, a reduction of 77% (for details of these calculations, see OSHA's Preliminary Regulatory Impact Analysis, Exs.15–A and 15–B). It is important to note that while reducing exposures to meet a TWA PEL of 1 $\mu g/m^3$ greatly reduces the number of cancer deaths attributable to cadmium exposure, the estimate of risk at 1 $\mu g/m^3$ using this methodology, 2.1 per 1,000 workers, is still significant.

In addition to its risk assessment based on the animal data, OSHA also performed a risk assessment using data from a mortality study of a cohort of cadmium smelter workers conducted by Thun and others (Ex. 4–68). This study, which is an historical prospective study of 602 white men employed in production areas of the smelter for at least six months between 1940 and 1969, provides the strongest evidence of the carcinogencity of cadmium in humans. For workers with two or more years of employment at the smelter, the incidence of lung cancer was statistically significantly elevated (SMR=229). Dividing the cohort of workers into those with low, middle and high cumulative exposures to cadmium, a dose-response relationship between cadmium and lung cancer was observed.

The methods used to quantify risk from the Thun data closely follow those used by EPA (Ex. 4–04). Unlike EPA, however, OSHA fit a relative risk model as well as an absolute risk model to the data. At an exposure level of 100 $\mu g/m^3$, the absolute risk model predicts 16.3 lung cancer deaths and the relative risk model predicts 30.7 lung cancer deaths attributable to occupational exposure to cadmium per 1000 workers with 45 years of exposure. At a proposed TWA PEL of 5 $\mu g/m^3$, the risk of lung cancer death attributable to cadmium exposure per 1000 workers with 45 years exposure is 0.8 using the absolute risk model and 1.6 using the relative risk model. At a proposed TWA PEL of 1 $\mu g/m^3$, the same risks are 0.2 using the absolute risk model and 0.3 using the relative risk model (see Table VI–G above). Although the estimates of risk derived from the relative risk model are twice as large as those derived from the absolute risk model, both estimates at the current OSHA TWA PEL of 100 $\mu g/m^3$ are indisputably significant.

OSHA relied upon two studies of workers to quantify the risk of kidney dysfunction associated with occupational exposure to cadmium. These studies were chosen because they provided adequate exposure data to perform such an assessment. One study, by Ellis and others (Ex. 4–27), examined 82 male workers at the same cadmium smelter studied by Thun. The other, by Falck and others (Ex. 4–28), examined 33 workers at a plant that produces refrigeration compressors with silver

brazed copper fittings containing between 18% and 24% cadmium. Ellis used a logistic regression model to describe the relationship between kidney dysfunction and cadmium exposure. OSHA performed a logistic regression on Falck's data as well. Although, as explained in Section VI, the Ellis' model may over estimate risk at low doses, and although both models may omit variables that may be associated with kidney dysfunction, such as age and duration of exposure, OSHA nonetheless believes that these logistic regression models are adequate for quantifying the risk of kidney dysfunction due to occupational exposure to cadmium.

At the current TWA PEL of 100 $\mu g/m^3$, the incidence of kidney dysfunction per 1000 workers with 45 years of occupational cadmium exposure is predicted to be 890 from the Ellis model and 974 from the Falck model. At the proposed TWA PEL of 5 $\mu g/m^3$, the estimated incidence of kidney dysfunction ranges from 9 per 1000 workers (Falck) to 165 per 1000 workers (Ellis), and at a proposed TWA PEL of 1 $\mu g/m^3$, the estimated incidence of kidney dysfunction ranges from 0.1 per 1000 workers (Falck) to 26.1 per 1000 workers (Ellis). (See Table VI–J.) Although the Ellis and Falck models predict different risks at low doses, at the current OSHA TWA PEL of 100 $\mu g/m^3$ both models predict unacceptably high risks and even at doses as low as 5 $\mu g/m^3$ both models predict a risk well in excess of 1 per 1,000 workers.

Using both the Ellis and Falck models, OSHA projects that between 576 (Falck) and 10,273 (Ellis) of the approximately 512,000 cadmium-exposed workers will develop occupationally related kidney dysfunction if exposed at current levels over their working lives. Thus, under both models, the number of workers who will suffer kidney dysfunction due to occupational exposure to cadmium at current levels is unacceptably high. OSHA anticipates that reducing the TWA PEL to 5 $\mu g/m^3$ will prevent between 477 (Falck) and 1853 (Ellis) of these cases of kidney dysfunction, a reduction which ranges from 18% to 83%, while reducing the TWA PEL to 1 $\mu g/m^3$ will prevent between 574 (Falck) and 9127 (Ellis) of these cases of kidney dysfunction, a reduction which ranges from 89% to 99%. (For details of these calculation, see OSHA's Preliminary Regulatory Impact Analysis, Exs.15–A and 15–B).

Thus, rodent and human studies support OSHA's conclusion that cadmium is a potential occupational carcinogen. Other health science organizations that have considered this issue in recent years, such as IARC, NIOSH, EPA, and ACGIH, have also concluded that cadmium is a potential human carcinogen. Cadmium has been shown to cause lung cancer in male rats by inhalation and in rats, hamsters and mice by injection. In addition, preliminary results from an inhalation study in male and female Wistar rats using four cadmium compounds show formation of primary lung tumors.

However, not all the studies point to an association between exposure to cadmium and cancer. Studies involving the oral administration and intratracheal instillation of cadmium and preliminary results from some long-term inhalation studies do not show a positive association in mice and hamsters. While a number of these studies may have flaws that make interpretation of their data difficult (Ex. 12–41), as discussed in the carcinogenicity section, above, collectively they may raise some questions about the association across species.

Although OSHA believes that the evidence supports its conclusion that cadmium is a potential occupational carcinogen, the Agency has less confidence in the specific quantitative estimate of risk of cancer associated with particular levels of exposure to cadmium. This reduction in confidence has two sources: first, the inherent uncertainties in using quantitative analysis to describe the real world; and, second, the limitations on the scientific studies that are available.

The Takenaka and Thun studies are amenable to quantitative risk analysis. Both of these studies provide measures of dose and response that allow their use for such analysis. Other studies, both positive and negative, that do not provide such measures can be considered in qualitative assessments, but there is no way to determine what weight to give such studies in a quantitative risk assessment.

OSHA believes the Takenaka rat study is suitable for quantitative risk assessment because exposure levels were well documented, the study was run with concurrent controls, there was no opportunity for confounding exposures, and the route of exposure, inhalation, is the same as the primary route of exposure in occupational settings. The drawbacks to this study, however, are that the animals were dosed continuously and the test material was cadmium chloride. By contrast, workers generally are exposed only eight hours a day, and their exposure is mostly to cadmium and cadmium compounds other than cadmium chloride. In addition, deriving estimates of human risk from rat data requires cross species extrapolation, which introduces uncertainty into any estimate of risk.

OSHA recognizes these drawbacks to reliance upon the Takenaka study for its preferred estimate of risk. Nevertheless, the Agency feels there are reasons to rely upon this study. Cadmium chloride may be more soluble than other cadmium compounds, but OSHA believes that the active agent in carcinogenesis is cadmium, and not the ion to which it is bound. Rats in the study were dosed continuously, whereas workers are not, but cancer risk assessments have always been based upon total dose. Scientists do not know how to account for differences in dose regimen.

As indicated above, to quantify risk from cadmium exposure using the Takenaka rat data, OSHA has examined five low-dose extrapolation models. Each of these models—the multistage model, the one-hit model, the probit model, the logit model, and the Weibull model—provides a good fit to the data. At the current OSHA PEL of 100 $\mu g/m^3$, all of the models predict risks well in excess of 1 per 1,000.

The lowest risks are predicted by the probit, the logit, and the Weibull models, which are tolerance distribution models. These models are based on "growth" curves found in bioassays and predict dose-response curves that are generally sigmoid in shape. Because these curves are not necessarily linear at low doses, they tend to approach zero more rapidly than does the dose-response curve derived from the multistage model. Thus, for example, the risks they predict at low doses are lower than those predicted by the multistage model. Among these tolerance distribution models, the lowest risks are predicated by the probit model. If one were to select that model as one's choice of best model, the risk of lung cancer from cadmium exposure at 10 $\mu g/m^3$ would drop below 1 per 1,000.

The choice of model is very important. It involves scientific judgment. There is no certain way to determine which model is correct. The statistics that allow us to measure goodness of fit cannot provide help in judging "best" fit. Consequently, the best (correct) model must be chosen on the basis of some other criterion.

OSHA prefers the multistage model as its best model because the Agency believes the multistage model has the best empirical and theoretical justification of all the models for

estimating carcinogenic dose-response. The multistage model is a nonthreshold model that is linear at low doses. The Agency believes that this model conforms most closely to what we know of the etiology of cancer. OSHA's preference is consistent with the position of the Office of Science and Technology Policy, which recommends that "when data and information are limited, and when much uncertainty exists regarding the mechanisms of carcinogenic action, models or procedures that incorporate low-dose linearity are preferred when compatible with limited information (Ex. 8-693).

At doses of 10 $\mu g/m^3$ or lower the models differ widely in their estimates of risk. The fact that all the models fit the rat data so well and yet predict a wide range of risks at low doses, adds some uncertainty to OSHA's estimates of risk. Some confidence is added, however, by the fact that the risk estimates from OSHA's preferred model, the multistage model, differ by less than a factor of 10 from the risk estimates derived from the Thun epidemiological study.

OSHA has also performed a quantitative risk assessment using the epidemiological data reported by Thun et. al. This study has strengths and weaknesses for use in a quantitative cancer risk assessment. Its major strengths are that it is based upon observation of workers, it has individual dose measurements, and vital status was determined for 98% of the cohort members. The study also provides extensive follow-up of cohort members, most having been followed for at least 20 years. It is the only epidemiological study of cadmium exposed workers that has dose estimates adequate for dose-response analysis.

However, questions have been raised about the methods used to determine historical cadmium exposure levels and about the role of arsenic contamination and cigarette smoking in the excess lung cancer risk observed by Thun et al. Concern also has been raised about confounding from the fact that the cohort is comprised of about 40% Hispanics, who have about one-third the rate of lung cancer as compared to the U.S. white male population that was used to derive the expected lung cancer rates in the study. Nor was any adjustment made for the healthy worker effect. The authors evaluated all but the latter two of these potentially confounding factors and concluded that the former factors played little role in the excess lung cancer risk. The amount of confidence one can place in the cadmium exposure estimates and in the resolution of the issues related to confounding as mentioned above will ultimately be a factor in OSHA's determination of how much the Agency should rely on the Thun study in its quantitative estimates of cancer risk.

If OSHA were to rely on the Thun data, the estimated excess lung cancer risk would be 30.7 per 1,000 workers exposed to cadmium at 100 $\mu g/m^3$ over a working lifetime. The estimated risk for workers exposed to cadmium at 5 $\mu g/m^3$ and 1 $\mu g/m^3$ would be 1.6 and 0.3 per 1,000, respectively. This risk is approximately one-seventh the risk estimated using the Takenaka data.

OSHA has received the following comment:

> Using on [sic] the Takenaka results, each of the five low-dose extrapolation models predicts excess lifetime lung cancer risks greater than one in six (2×10^{-1}) at exposure levels equivalent to 100 $\mu g/m^3$, and excess lifetime lung cancer risks greater than one in two (7×10^{-1}) for exposures exceeding 400 $\mu g/m^3$. Given the fact that exposures historically equalled or exceeded these levels in some occupational settings, it is useful to compare high-dose risk estimates derived from the Takenaka data with the available epidemiological evidence from equivalently high-dose exposure.
>
> This is important for two reasons. First, it provides a real-world test of whether similarly high risks might be expected to arise in human populations. All risk assessments are uncertain as to the degree to which the dose-response observed in animals is an accurate indicator of the dose-response in humans. Confidence in animal bioassay results is enhanced when dose-equivalent cancer rates predicted in human populations are roughly equivalent to those obtained from bioassay. However, confidence in animal bioassay results is diminished if cancer rates observed differ substantially from dose-equivalent cancer rates predicted in human populations.
>
> Second, estimates of low-dose cancer risks derived from animal bioassays are influenced by tumor incidence observed at high doses. If tumor incidence observed in animals at high doses is substantially less than incidence predicted to arise from equivalent doses in humans, then estimated low-dose risks derived from animal data are likely to understate actual human cancer risks at low doses. Conversely, if high-dose animal risks substantially exceed predicted high-dose human risks, then estimates of risk at low doses are likely to overstate actual human cancer risks.
>
> * * * [M]ore than a dozen epidemiological studies * * * have examined the carcinogenic effects of inhaled cadmium compounds. None of these studies, however, shows lung cancer rates among cadmium-exposed workers as great as observed in the Takenaka rat data. For example, total lung cancer incidence was 14 percent (based on person-years at risk) in the high exposure cohort of the study by Thun *et al.* This exposure cohort is interesting because it is roughly equivalent to the high-dose rat cohort examined by Takenaka; median exposure in this cohort was estimated to be 4,200 mg/m^3-days, or 369 $\mu g/m^3$ [sic] based on a 45-year 8-hour TWA. As is the case for all retrospective mortality studies, of course, this cancer rate is preliminary in the sense that a substantial fraction of the high-exposure cohort was still alive when the study was conducted. Thus, the "ultimate" lung cancer incidence for this cohort will not be known for many years. It can be shown, however, that for this "ultimate" cancer rate to reach 70% (the rate observed by Takenaka at the rat-equivalent dose), *virtually every living member* of the cohort must contract cadmium-induced lung cancer.
>
> * * * [T]his apparent discrepancy between the animal and human data at equivalently high doses * * * [has] implications for the estimation of low-dose cancer risks. OSHA * * * [should request] comment and analysis as to the significance of this problem and what bearing it should have in the development of a final risk assessment.
>
> To see this result, first note that the baseline mortality rate was no less than 35 percent of the workers in the high-exposure cohort. The highest plausible exposure scenario involves workers exposed prior to 1960 in any one of three plant departments at a level of 1.5 mg/m^3. Since the lower bound of Thun's high-exposure cohort is (2,921) mg/m^3-days, the minimum length of exposure in the cohort must be (2,921 mg/m^3-days)/<[1.5 $mg/m^3\times$(240/12) days/months]>[sic]=93.6 months, or 7.8 years. This means that all members of the high-exposure cohort must have been exposed for at least two years. According to Thun (Table 2), 119 of 345 (35%) workers in this group had died when the study was conducted.
>
> Using this 35 percent mortality rate as a lower-bound for the high-exposure cohort provides a conservative test of the plausibility of observing a 70 percent lung cancer rate once the entire cohort has died. The question to answer is: What proportion of the living cohort members must contract cancer to obtain a total cancer incidence equal to that observed by Takenaka in the high-dose rat cohort? If this proportion is reasonable, then Takenaka's results would appear to be a credible proxy for human experience. If it is unreasonable, however, then the Takenaka results would appear to be an inaccurate indicator of human cancer risks at equivalent doses. Inconsistencies in risk estimates that arise at high doses imply similar discrepancies at low doses, thus making this time an important source of uncertainty that must be reckoned with by regulatory decision makers.
>
> The observed total cancer rate in the high-exposure cohort is 14.2% [7 cancer deaths/(2214 person-years at risk/45 years per working lifetime)]. Determining the proportion of the 222 living workers that must contract cancer for the 'ultimate' cancer rate to reach 70% involves solving for x in the following equation:
>
> $(119*0.14)+(222*x)=344*0.70.$
>
> $x=1.01.$

Thus, even if every living member of the high-exposure cohort contracted lung cancer, the total cancer rate for the cohort still would not equal the 70 percent rate observed by Takenaka in rats at equivalent doses. Furthermore, any total cancer rate predicted in humans reflects baseline lung cancer risks arising from other factors (e.g., smoking), not just the *incremental* effect attributed to cadmium exposure, which is the interpretation of the 70 percent incidence rate observed by Takenaka.

This result is robust with respect to the baseline mortality rate of high-exposure cohort. The baseline mortality rate of 35 percent for workers exposed more than two years is likely to understate the mortality rate of the high-exposure cohort, which was not reported in the study. This 35 percent figure is a weighted average of workers belonging to the low-, medium-, and high-exposure cohorts. Given the observed dose-response relationship, it is almost certain that the mortality rate in the high-exposure cohort exceeds 35 percent. Hence, the proportion from this cohort still alive when the study was conducted is likely to be lower than 64 percent. A higher baseline mortality rate increases the magnitude of the first term in the equation above and makes it even less plausible that sufficient cancers could arise in the remaining members of the cohort to observe in humans the high cancer rate found in rats.

With regard to the science of quantitative risk assessment, OSHA believes the risks derived from the animal and human data are not incompatible. However, the Agency would like to explore the possible reasons for the differences in the estimated cancer risks based on the animal and epidemiological data. On the one hand, if one accepts the quantitative risk assessment results from the epidemiologic study as representing the "true" dose-response relationship, then the dose-response relationship based on the animal data may have overestimated the risk, and the difference in the quantification of risk may be a reflection of an incorrect assumption in extrapolating risks to humans from the Takenaka study. On the other hand, if the estimate of risk from the animal data represents the "true" dose-response relationship, then the dose-response relationship based upon the epidemiologic data may have underestimated the risk, and the difference may be due to error in dose estimation, confounding from cigarette smoking, Hispanic composition of the cohort, as well as lack of adjustment for the healthy worker effect.

OSHA requests public comment on all these points and on the uncertainties involved in using the Takenaka rat data or the epidemiological data from the Thun study to perform its quantitative assessment of the risk of cancer associated with occupational exposure to cadmium. OSHA further requests public comment on how the Agency might better resolve these issues.

OSHA has also assessed the risk of kidney dysfunction associated with occupational cadmium exposure. The Agency relied upon the studies by Ellis et al. and by Falck et al. to model this relationship. A logistic regression technique was used to estimate risk at a variety of exposure levels. This technique was chosen because it allows one to relate a continuous independent variable (dose) to a dichotomous dependent variable (sickness or nonsickness).

The drawback of this model, however, is that it is a nonthreshold model: Any exposure is associated with some risk. In fact, the scientific evidence indicates that kidney dysfunction has a threshold. Some cadmium must be accumulated in the kidney before dysfunction occurs. This means that estimates of risk derived from this model at doses lower than the threshold are not reliable. What that threshold is, however, is uncertain. There are established ranges of cumulative air cadmium levels and of kidney cadmium burdens associated with kidney damage. But within those ranges, OSHA does not know the location of the threshold level for kidney damage. Additional uncertainty is caused by the small size of the cohorts studied by Ellis and Falck.

OSHA requests public comment on its use of this logistic regression model and on other uncertainties involved in using the Ellis and Falck data to perform its quantitative risk assessment for kidney dysfunction. OSHA further requests public input on other techniques for modeling this relationship.

OSHA's conclusion that the risk of death from cancer and the risk of kidney dysfunction resulting from exposure to cadmium at 100 $\mu g/m^3$ over a working lifetime are both significant is consistent with OSHA's determination of significance of risk at the previously existing TWA PELs for two carcinogens recently subject to rulemaking. The two carcinogens are inorganic arsenic (Jan. 14, 1983; 48 FR 1864, 1986); and ethylene oxide (Apr. 21, 1983; 48 FR 17284). The risk estimates per 1000 employees for a working lifetime exposure to each of these carcinogens ranged from 148 to 425 lung cancer deaths from inorganic arsenic and from 63 to 109 cancer deaths from ethlyene oxide.

In addition, for both carcinogens, OSHA concluded that, if it were feasible, OSHA would seek to further reduce the predicted remaining risk at the new proposed or set TWA PELs. That remaining excess risk of death for a working lifetime exposure per 1,000 workers was 8 for inorganic arsenic and 1 to 2 for ethylene oxide.

Further guidance for the Agency in evaluating significant risk is provided by an examination of occupational risk rates, legislative intent, and language of the Supreme Court of the United States. For example, in the high risk occupations of mining and quarrying (Division B), the average risk of death from an occupational injury or an acute occupationally-related illness over a lifetime of employment (45 years) is 15.1 per 1,000 workers. Typical occuptional risks of deaths for all manufacturing (Division D) are 1.98 per 1,000. Typical lifetime occupational risk of death in an occupation of relatively low risk, like retail trade, is 0.82 per 1,000 (Division G). (These rates are averages derived from 1984–1986 Bureau of Labor Statistics data for employers with 11 or more employees, adjusted to 45 years of employment, for 50 weeks per year.)

There are relatively few data on risk rates for occupational cancer, as distinguished from occupational injury and acute illness. The estimated cancer fatality rate from the maximum permissible occupational exposure to ionizing radiation is 17 to 29 per 1,000 (47 years at 5 rems; Committee on Biological Effects of Ionizing Radiation (BEIR) III predictions). However, most radiation standards require that exposure limits be reduced to the lowest level reasonably achievable below the exposure limit (the ALARA principle). Consequently, approximately 95% of radiation workers have exposures less than one-tenth the maximum permitted level. The risk at one-tenth the permitted level is 1.7 to 2.9 per 1,000 exposed employees.

Congress passed the Occupational Safety and Health Act of 1970 because of a determination that occupational safety and health risks were too high. Congress therefore gave OSHA authority to reduce above-average or average risks when feasible. In discussing the level of risk that Congress authorized OSHA to reduce, the Supreme Court stated that "if the odds are one in a thousand that regular inhalation of gasoline vapors that are 2% benzene will be fatal a reasonable person might well consider the risk significant and take the appropriate steps to decrease or eliminate it." (*I.U.D.* v. *A.P.I.*, 448 U.S. 607, 655).

Within this context, OSHA's preliminary best estimates of risk from occupational exposure to cadmium at the current TWA PELs are substantially higher than other risks that OSHA has concluded are significant, are substantially higher than the risk of

fatality in high-risk occupations, and are substantially higher than the example presented by the Supreme Court. Consequently, OSHA preliminarily concludes that its best estimate of risk, 221 cancer deaths per 1,000 workers, associated with the current TWA PEL of 100 $\mu g/m^3$ presents a significant risk. OSHA's estimate of risk, derived from the same data and model, shows that at an exposure level of 5 $\mu g/m^3$, the risk is 10.6 per thousand, and at an exposure level of 1 $\mu g/m^3$, the risk is 2.1 deaths per thousand, both of which would also be significant based on the above reasoning.

OSHA further notes that a large number of employees are exposed to cadmium well under the current exposure limit. Many employers appear to be keeping exposures relatively low. Consequently, most currently cadmium-exposed employees are exposed to risks below the risk levels presented.

OSHA preliminarily concludes that either the proposed TWA PEL of 5 $\mu g/m^3$ or the proposed TWA PEL of 1 $\mu g/m^3$ will substantially reduce significant risk of cancer. Compared to the risk of cancer death attributable to occupational exposure to cadmium at the current TWA PEL of 100 $\mu g/m^3$, using the same data and model, a 5 $\mu g/m^3$ standard will result in a reduction of at least 211 cancer deaths per 1000 workers exposed, a 95% reduction in risk, while a 1 $\mu g/m^3$ standard will result in a reduction of at least 219 cancer deaths per 1000 workers, a 99% reduction in risk.

The actual reduction is likely to be greater at either TWA PEL and the remaining risk is likely to be smaller than estimated for a number of reasons. First, the proposed action levels, which would trigger medical surveillance and other measures to protect employees from the adverse effects of cadmium exposure, are set at 2.5 $\mu g/m^3$ for a TWA PEL of 5 $\mu g/m^3$ and at .5 $\mu g/m^3$ for a TWA PEL of 1 $\mu g/m^3$. This means that employers will be required to implement the medical surveillance program and other ancillary provisions for workers exposed at or above the action level. For workers exposed over the action level, illness and injury may be identified at an early enough stage to prevent irreversible damage. Consequently, as discussed in the medical surveillance portion of the Summary and Explanation section of this preamble, the program triggered by the action level will further decrease the incidence of disease beyond the predicted reductions attributable merely to a lower TWA PEL.

Many employers may be motivated to reduce exposures below the action level, if it is feasible, to avoid the obligations of medical surveillance and other ancillary provisions of the standard. This would further reduce risk as well as reduce industrial hygiene costs. In addition, since cadmium accumulates in the body thereby increasing the risk of death from lung and prostrate cancer and increasing the excess risk of kidney dysfunction, it will accumulate more slowly in the body below the action level. At the lower proposed action level of .5 $\mu g/m^3$, the Takenaka rat data applied to the multistage model predicts a risk of excess total cancer deaths just above 1 death per 1000 workers. The risk of kidney dysfunction at this level could be as high as 26 per 1000. The rat data applied to the multistage model predict that cancer risks at the alternative action level of 2.5 $\mu g/m^3$ are higher, as is the risk of kidney dysfunction. Thus, the action level provides added employee protection while increasing the cost effectiveness and performance orientation of the standard.

OSHA is also proposing an excursion limit (EL) of 25 $\mu g/m^3$ for a TWA PEL of 5 $\mu g/m^3$ and an EL of 5 $\mu g/m^3$, for a TWA PEL of 1 $\mu g/m^3$. This should further limit employee exposures and therefore risk in certain circumstances.

Although OSHA cannot quantify the reductions in risk that may be expected from these and other similar provisions in the proposed standard, OSHA believes that the effect of including these provisions in a final standard will further reduce the remaining risks estimated at the proposed TWA PELs. Therefore, OSHA's preliminary conclusion is that either of the proposed TWA PELs of 5 $\mu g/m^3$ or 1 $\mu g/m^3$ will substantially reduce a significant risk in areas where the reduction is quantifiable and in addition, will result in very real further substantial reductions in risk.

OSHA, therefore, preliminarily concludes that both of the proposed TWA PELs reduce risk within the limits of feasibility.

As just discussed, OSHA expects the action level, the EL, the medical surveillance provisions and other industrial hygiene requirements of the final standard to substantially reduce the risk remaining at the proposed TWA PELs, although the additional reduction cannot be quantified. As a result, OSHA preliminarily concludes that its proposed TWA PELs of 5 $\mu g/m^3$ or 1 $\mu g/m^3$ will protect employees and that employers who comply with the provisions of the standard will be taking reasonable steps to protect their employees from the hazards of cadmium.

VIII. Summary of the Regulatory Impact and Flexibility Analysis

A. Introduction

Executive Order 12291 (46 FR 13197, Feb. 19, 1981) 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 the Occupational Safety and Health Administration (OSHA) to consider the impact of the proposed regulation on small entities.

Consistent with these requirements, OSHA has prepared a Preliminary Regulatory Impact and Regulatory Flexibility Analysis (PRIA) for the proposed cadmium standard with 5 $\mu g/m^3$ and 1 $\mu g/m^3$ eight hour time weighted average permissible exposure limits (TWA PELs). This analysis describes the industries affected by the standard, the regulatory alternatives considered, some of the potential benefits that will accrue to employees exposed to cadmium at their places of work, the costs of compliance with the proposed standard, and the technological and economic feasibility of the proposed provisions. The following is a summary of this analysis.

B. Industry Profiles

This section provides information on those industries and occupations most likely to be affected by a change in OSHA's current cadmium standard. Employees are potentially exposed to cadmium in industries which are involved in refining cadmium and cadmium-bearing ores, and in industries and occupations which make or use cadmium and cadmium compounds. Production processes include cadmium and zinc refining, nickel-cadmium battery production, electroplating, the production and use of cadmium pigments, the production and use of cadmium stabilizers, and the production and use of cadmium-containing alloys. Occupations where cadmium is found include brazers and solderers of metals and furnace operators.

The principal source of information for this profile is JACA Corporation's final report, "Economic Impact Analysis of the Proposed Revision to the Cadmium Standard," Chapter 2, March 15, 1988 [Ex. H-057a]. References to original material may be found in the JACA Report.

Cadmium Production

Cadmium is a silver-white, malleable metal with a variety of industrial applications that range from electroplating to the stabilization of plastics. Cadmium is marketed as a metal and as a compound, depending on the application. Cadmium metal (grades of 99.95% purity) is sold in balls or sticks for use in electroplating and alloys production while cadmium compounds, such as cadmium sulfide and cadmium selenide, are used to produce plastic stabilizers and additives.

JACA Corporation [Ex. H–057a] reports that in 1985, world cadmium production amounted to 18,660 metric tons, down slightly from the 1984 level of 19,170 metric tons. The leading world producers of cadmium metal include Canada, the Soviet Union, West Germany, Belgium, the United States, Japan, and Australia. These nations accounted for 64 percent of the total cadmium metal production in 1985.

International trade is an important aspect of the cadmium industry. Canada, Australia, Finland, and the Netherlands are all major cadmium exporters, while the United States, the United Kingdom, France, Belgium, and West Germany are all major importers.

JACA found that in 1985, domestic cadmium production amounted to 1,600 metric tons [Ex. H–057a]. U.S. production accounted for nine percent of world cadmium metal production. Presently, four refining facilities are producing cadmium metal in the United States. In addition to cadmium metal, three of these companies also produce one or more of the compounds cadmium oxide, cadmium sulfide, and cadmium selenide.

Cadmium does not exist in sufficient quantities to warrant mining it exclusively. It is generally found in the ores of other metals—primarily zinc, copper, and lead. The majority of cadmium is recovered from zinc ores as by-products of zinc metal refining. Three of the four cadmium production facilities are primary zinc producers.

Cadmium (metal and/or oxide) can be produced as a by-product of roaster calcine leachate at zinc smelters or from lead smelter baghouse dusts. Exposures occur during the melting of cadmium sponge or sheet (intermediate products) and can be expected when the molten cadmium is cast into various shapes. Exposures can also occur when bags containing lead smelter baghouse dust, which can be up to 60 to 65 percent cadmium, are handled and emptied. If cadmium oxide or cadmium metal powder are produced, exposures are likely to occur during the reheating of cast cadmium in the retort furnace and during the packaging of the powdered material.

Cadmium and Zinc Refining

Zinc is a metal which is surpassed only by iron, aluminum, and copper in industrial usage. It is used mainly as a protective coating on steel and iron (galvanizing), in alloys for diecastings, as an alloying metal with copper, and as a chemical compound in rubber and paints. Zinc oxide is the primary zinc-based chemical compound.

After extraction, zinc ores are treated at a mill for separation of mineral constituents. The zinc product (zinc concentrate) is then refined at a smelter by either electrolytic deposition (electrolysis) or distillation in retorts or furnaces.

According to JACA, domestic production of zinc metal reached an all time high in 1969 at 944,000 metric tons. By 1985, output was 67 percent lower. Unlike domestic production, world production has grown over the decade. In 1984 the leading zinc producing countries were Canada, the Soviet Union, Australia, and Peru, while the U.S. accounted for 4 percent of world mine output and 5 percent of world smelter output. There are currently four primary zinc smelters in the U.S.

In zinc refining, cadmium exposures can be expected during the handling of zinc ores, which contain from 0.3 to 0.4 percent cadmium, and during routine baghouse maintenance. Storage and handling of a cadmium-containing leach residue produced during sintering can also expose workers to cadmium, as can the production of zinc oxide.

Electroplating

Electroplating is the electrodeposition of a metal (typically zinc, chromium, copper, nickel, or cadmium) onto a surface in order to import characteristics of the plating material. Plating is done across many manufacturing industries with the heaviest use in the automotive, electronics, industrial hardware, and aerospace industries.

JACA reports that electroplating in the U.S. consumed 1,270 metric tons of cadmium in 1985. This represented 34 percent of the total cadmium consumed domestically. Since 1973, the U.S. has been dedicating approximately one third of its cadmium consumption to electroplating operation.

Electroplating is done captively and on a job shop basis. Captive electroplating includes companies which electroplate parts or materials that go into their products. Job shop electroplaters are companies which, on a contract basis, electroplate parts or materials for another company. Since a great deal of electroplating is done captively and many job shops are quite small, reliable data on the number of electroplating operations are difficult to find. JACA reviewed data published by the Census Bureau and by the National Association of Metal Finishers. Their report indicates that there are approximately 5,300 plating operations in the U.S., including 3,400 job shops and 1,900 captives. "Finisher's Management" estimates that there are currently 1,166 plants which electroplate predominantly with cadmium. Electroplating with cadmium is usually conducted in a cyanide bath. The solution is prepared from cadmium oxide in sodium cyanide. Cadmium exposure due to this type of bath is expected to be minimal since the cadmium is used in a liquid solution that is kept well below the boiling point. In addition to the baths, employees could also be exposed during the handling of the cadmium before the plating solution is prepared.

Battery Manufacture

Cadmium is used in the negative plate of nickel-cadmium, silver-cadmium, and mercury-cadmium batteries. Of these types, nickel-cadmium (Ni-Cd) batteries are the most common.

Ni-Cd batteries can be divided into two main groups. The first consists of open or vented cell batteries, which are used mainly in industrial applications. The vented cells are constructed using either a pocket plate design or a sintered design, depending on the application and power requirements. The second group consists of sealed cell batteries, which are used primarily in consumer applications. Sealed cells are smaller than their vented counterparts and have lower power capabilities.

The JACA study states that in 1985, the Ni-Cd battery industry consumed just over 1,000 metric tons of cadmium. The demand for Ni-Cd batteries grew rapidly through the 1960's and early 1970's. By 1985 domestic consumption for battery production reached 27 percent of total cadmium demand. Worldwide demand for cadmium in Ni-Cd production has continued to grow and it is estimated that 37 percent of the cadmium consumed in Japan, West Germany, and Great Britain is used to produce batteries.

The manufacturers involved in the production of Ni-Cd batteries in the U.S. comprise a diverse group. They include large, highly diversified corporations as well as smaller, more specialized companies. Several of the Ni-Cd producers also manufacture lead-acid

batteries or other energy storage systems.

Vented cell batteries are produced using either the pocket plate design or the sintered plate design. Exposures can occur in pocket plate battery production during preparation of cadmium oxide (the pocket plate starting material) or during routine maintenance operations. Exposure can occur in the sintered cell process when handling the cadmium metal before it is put into solution, as well as during abrasion and cutting activities.

Cadmium Pigments

Cadmium pigments are inorganic compounds ranging in color from yellow to red which are used to color plastics, paints, ceramics, and printing inks. They are usually produced as powders but are also produced in other forms such as pastes and liquids. For applications in the plastics industry, cadmium compounds are available in master batch pellets. These are compounded polymer resins into which pigments have been incorporated.

As reported by JACA, worldwide use of cadmium for pigment production has been relatively stable since 1960, but slight declines have been seen in recent years. At present, approximately 25 percent of cadmium consumption in the major consuming nations of Japan, West Germany, the U.S., and the U.K. is used in the production of pigments. In 1985, U.S. producers used approximately 595 metric tons of cadmium in the production of cadmium-based pigments. Production in the U.S. is dominated by five companies.

Cadmium emissions from cadmium pigments production occur as particulate matter from solid cadmium-containing raw materials and powders. The major potential sources of emmissions are the calcining operations, cadmium sulfate production, drying operations, grinding operations, blending operations, and packaging operations.

Plastic Stabilizers

Cadmium stabilizers are cadmium-based compounds which are added to plastic resins to provide heat stability and protection from ultraviolet light. These compounds are used primarily in the production of polyvinyl chloride (PVC) and are usually carboxylates of barium-cadmium. Stabilizers are used in both liquid and solid forms and can be added to either the flexible or rigid types of PVC.

The U.S. consumed 520 metric tons of cadmium in the production of plastic stabilizers in 1985. The use of cadmium stabilizers was at its peak in 1970, when 1,340 metric tons of cadmium were used in stabilizer production. At that time, stabilizers accounted for one-third of domestic cadmium consumption. By 1976 cadmium consumption in stabilizer manufacture slipped to about 13 percent of total domestic consumption. Cadmium consumption in this industry has remained relatively stable since 1976, with current consumption at approximately 15 percent of total.

The combined use of cadmium by Japan, West Germany, the U.S., and the U.K. for stabilizer production in 1985 represented 12 percent of total consumption. Similar to the pattern in the U.S., consumption increased in the 1960's and peaked in the early 70's. Environmental factors and the high price of cadmium-based stabilizers have led to the slow decline of cadmium use in stabilizers.

Two-thirds of the cadmium stabilizers produced in the United States are in liquid form. The liquids contain barium, cadmium, and zinc soaps combined with materials such as antioxidants and phosphates. These liquid stabilizers generally contain one to four percent cadmium. The powdered stabilizers are soaps of barium and cadmium and fatty acids. The powders are more expensive than the liquids because they contain 7 to 15 percent cadmium.

The potential source of cadmium emissions in liquid stabilizer production is in the handling of cadmium metal or cadmium oxide before it is dissolved in an acid and solvent. In solid stabilizer manufacture, emissions can occur when cadmium oxide is added to acid to produce a cadmium chloride solution or during drying, handling, and packaging of the final product.

OSHA estimates there are approximately 5,200 workers exposed to cadmium in these industry sectors. Table VIII–A contains a frequency distribution of workers at various exposure levels based upon sample data obtained from JACA site visits, the cadmium docket, OSHA's IMIS and NIOSH's NOS computerized data base.

TABLE VIII–A.—EMPLOYMENT AND FREQUENCY DISTRIBUTION OF CADMIUM EXPOSURE OBSERVATIONS IN AFFECTED INDUSTRIES

Industry	Percent distribution of exposure observations and distribution of employees exposed to cadmium at exposure levels (μg/m)—									
	0–5	6–9	10–14	15–19	20–49	50–99	100–249	250–499	500+	Total
Cadmium Refining:										
Observations	55.42%	4.82%	2.41%	0.00%	13.25%	2.41%	7.23%	3.61%	10.84%	100.00%
(Employees)	(81)	(7)	(4)	(0)	(19)	(4)	(11)	(5)	(16)	(147)
Dry Process Stabilizer Production:										
Observations	49.98%	6.12%	0.00%	0.00%	12.24%	4.08%	12.24%	2.04%	14.29%	100.00%
(Employees)	(44)	(5)	(0)	(0)	(11)	(4)	(11)	(2)	(13)	(89)
Wet Process Stabilizer Production:										
Observations	50.00%	3.13%	0.00%	0.00%	15.63%	3.13%	12.50%	3.13%	12.50%	100.00%
(Employees)	(55)	(3)	(0)	(0)	(17)	(3)	(14)	(3)	(14)	(110)
Pigment Production:										
Observations	47.83%	10.14%	1.45%	2.90%	11.59	2.90%	8.70%	4.35%	10.14%	100.00%
(Employees)	(38)	(8)	(1)	(2)	(9)	(2)	(7)	(3)	(8)	(79)
NI–CD Battery Production:										
Observations	36.47%	7.06%	10.59%	2.35%	15.29%	8.24%	9.41%	5.88%	4.71%	100.00%
(Employees)	(105)	(20)	(31)	(7)	(44)	(24)	(27)	(17)	(14)	(289)
Electro Plating:										
Observations	97.62%	0.00%	0.00%	2.38%	0.00%	0.00%	0.00%	0.00%	0.00%	100.00%
(Employees)	(3415)	(0)	(0)	(83)	(0)	(0)	(0)	(0)	(0)	(3498)
Lead Smelting:										
Observations	53.30%	15.38%	6.59%	2.20%	9.89%	7.14%	3.85%	1.65%	0.00%	100.00%
(Employees)	(521)	(150)	(64)	(21)	(97)	(70)	(38)	(16)	(0)	(977)
Total Employees	4259	193	100	113	197	107	108	46	65	5189

Some observations were used in more than one industry to represent similar operations. Numbers may not add due to rounding.
Source: Office of Regulatory Analysis, 1988.

Occupational Exposure

In addition to employees exposed in cadmium producing and using industries, a number of workers across a broad cross section of U.S. industries are occupationally exposed to cadmium. OSHA has identified twelve general occupations outside the above industries which use cadmium on a regular basis. These include: Chemical mixers and millers; electroplaters; furnace operators and molders; kiln or kettle operators; heat treaters; equipment cleaners; metal machining operators; painters; maintenance painters; repair and utility workers; hand welders, brazers, and solderers; and machine welders, brazers, and solderers. Workers in these occupations may be exposed to cadmium in a variety of the following ways.

Chemical mixers: exposed while mixing cadmium-based plastic stabilizers, cadmium-based pigments, cadmium in the metallic coating of materials, cadmium compounds used in the production of fungicides, and other compounds containing cadmium. Chemical mixers add dry cadmium (and other) compounds to a chemical or mechanical mixing operation. Exposures to cadmium are generally in the form of dust. Workers in this occupation include production testers and weighers, and mixing operatives who attend machines which crush, grind, blend, and mix a variety of substances including cadmium.

Electroplaters: exposed while measuring and adding dry cadmium-bearing powder to the plating tank. Included in this group are electrolytic plating and coating machine setters, operators, and tenders who work on plating or coating machines.

Furnace Operators and Molders: exposed to cadmium fumes given off by molten metal during molding, casting, and forging operations. Includes forging machine operators, metal molders, coremakers, casting machine operators, melting and refining furnace operators, and metal pourers and casters.

Kiln or Kettle Operators: exposed to cadmium compounds during chemical conversions, molding operations, or when glazes, paints, or other coatings are heated. Includes oven operators, annealing, roasting, and converting furnace operators, dryer operators, metal molding, coremaking, casting machine operators, and kiln operators.

Heat Treaters: exposed to cadmium fumes when heating metals coated with or containing cadmium. Exposure occurs while tending machines such as furnaces, baths, flame-hardening machines, and electronic induction machines.

Equipment Cleaners: exposed to cadmium when cleaning equipment contaminated with either cadmium metal or its compounds, including baghouses, electrostatic precipitators, process equipment, and the process area.

Metal Machine Operators: exposed to dust containing cadmium generated while grinding or forming metal bearing cadmium. Includes machinists, grinders, filers, sharpeners, grinding machine operators, and other machine operators.

Painters: exposed to cadmium when using cadmium-based pigments and cadmium metal in paint and metal sprays. Includes workers employed in detail design, decoration, coating machine operation, the operation of nonelectrolytic plating and coating machines, and other areas.

Maintenance Painters: exposed to cadmium-based pigments while spray painting during construction or maintenance projects.

Repair and Utility Workers: exposed to cadmium fumes generated by painting, welding, soldering, and brazing operations for repair and maintenance. Includes mechanics, millwrights, automotive body repairers, general utility maintenance repairers, bus and truck mechanics, and others.

Hand Welders, Brazers, and Solderers: exposed to fumes released from cadmium-bearing base metals, brazing rods, or solders. Includes structural metal workers, metal pattern workers, metal fabricators, and others.

Machine Welders, Brazers, and Solderers: exposed to cadmium fumes released from cadmium-bearing base metals, brazing rods, or solders. Includes welding machine operators in all areas, glaziers, assemblers, fabricators, and others.

There are approximately 506,900 workers in these occupations. OSHA estimates that these workers may be exposed to cadmium on a regular basis. Table VIII-B presents a profile of workers included in these cross-industry occupations and their exposures.

TABLE VIII-B.—EMPLOYMENT AND FREQUENCY DISTRIBUTION OF CADMIUM EXPOSURE OBSERVATIONS IN AFFECTED CROSS-INDUSTRY OCCUPATIONS

Occupation	Percent distribution of exposure observations and distribution of employees exposed to cadmium at exposure levels (μg/m)—										
	0-5	6-9	10-14	15-19	20-29	30-49	50-99	100-249	250-499	500+	Total
Chemical Mixer:											
Observations	51.09%	10.92%	6.99%	2.18%	5.68%	7.42%	6.55%	5.68%	2.62%	0.87%	100.00%
(Employees)	(10,386)	(2,219)	(1,420)	(443)	(1,154)	(1,509)	(1,331)	(1,154)	(532)	(177)	(20,329)
Electroplater:											
Observations	85.71%	0.00%	3.57%	0.00%	10.71%	0.00%	0.00%	0.00%	0.00%	0.00%	100.00%
(Employees)	5,286	(0)	(220)	(0)	(661)	(0)	(0)	(0)	(0)	(0)	(6,168)
Furnace Operator; Molder:											
Observations	91.39%	1.91%	0.96%	0.48%	0.96%	0.96%	2.39%	0.00%	0.00%	0.96%	100.00%
(Employees)	(18,530)	(388)	(194)	(97)	(194)	(194)	(485)	(0)	(0)	(194)	(20,277)
Kiln or Kettle Operator:											
Observations	86.67%	6.67%	6.67%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	100.00%
(Employees)	(887)	(68)	(68)	(0)	(0)	(0)	(0)	(0)	(0)	(0)	(1,024)
Heat Treater:											
Observations	50.00%	0.00%	16.67%	0.00%	0.00%	16.67%	0.00%	16.67%	0.00%	0.00%	10.00%
(Employees)	(260)	(0)	(87)	(0)	(0)	(87)	(0)	(87)	(0)	(0)	(519)
Equipment Cleaner:											
Observations	83.33%	0.00%	4.17%	8.33%	0.00%	4.17%	0.00%	0.00%	0.00%	0.00%	100.00%
(Employees)	(111)	(0)	(6)	(11)	(0)	(6)	(0)	(0)	(0)	(0)	(133)
Metal Machine Operator:											
Observations	63.11%	4.10%	7.38%	1.64%	6.56%	6.56%	4.92%	2.46%	3.28%	0.00%	100.00%
(Employees)	(40,060)	(2,601)	(4,682)	(1,041)	(4,162)	(4,162)	(3,122)	(1,561)	(2,081)	(0)	(63,472)
Painter:											
Observations	76.80%	160%	2.40%	0.80%	0.80%	3.20%	4.00%	1.60%	4.80%	4.00%	100.00%
(Employees)	(8,656)	(180)	(271)	(90)	(90)	(361)	(451)	(180)	(541)	(451)	(11,271)

TABLE VIII-B.—EMPLOYMENT AND FREQUENCY DISTRIBUTION OF CADMIUM EXPOSURE OBSERVATIONS IN AFFECTED CROSS-INDUSTRY OCCUPATIONS—Continued

Occupation	Percent distribution of exposure observations and distribution of employees exposed to cadmium at exposure levels (μg/m)—										
	0–5	6–9	10–14	15–19	20–29	30–49	50–99	100–249	250–499	500+	Total
Construction, Maintenance Painter:											
Observations	100.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	100.00%
(Employees)	(742)	(0)	(0)	(0)	(0)	(0)	(0)	(0)	(0)	(0)	(742)
Repair and Utility Worker:											
Observations	57.14%	10.00%	4.29%	1.43%	10.00%	5.71%	2.86%	7.14%	1.43%	0.00%	100.00%
(Employees)	(88,746)	(15,531)	(6,656)	(2,219)	(15,531)	(8,875)	(4,437)	(11,093)	(2,219)	(0)	(155,306)
Welder, Brazer Solderer-Hand:											
Observations	88.03%	2.56%	128%	0.85%	0.43%	1.71%	3.42%	1.71%	0.00%	0.00%	100.00%
(Employees)	(23,798)	(693)	(347)	(231)	(116)	(462)	(924)	(462)	(0)	(0)	(27,033)
Welder, Brazer Solderer-Machine:											
Observations	78.00%	3.14%	3.14%	1.96%	3.03%	3.49%	3.61%	2.10%	0.81%	0.70%	100.00%
(Employees)	(156,512)	(6,307)	(6,307)	(3,971)	(6,074)	(7,008)	(7,242)	(4,205)	(1,635)	(1,402)	(200,662)
Total: Employees	353,976	27,988	20,257	8,103	27,980	22,662	17,992	18,741	7,008	2,224	506,936

Numbers may not add due to rounding. Office of Regulatory Analysis, 1988.

C. Benefits Analysis

The benefits of a revised federal standard for occupational exposure to cadmium would be reductions in the incidence of cancer (primarily prostate and lung cancers) and kidney disorders. In estimating these benefits, OSHA used the exposure profile presented above in conjunction with quantitative risk assessments generated for each of the two types of illness.

The exposure estimates are based primarily on data contained in NIOSH's Health Hazard Evaluation (HHE) reports and OSHA's Integrated Management Information System (IMIS). Neither of these data sets was designed or intended to be used for extrapolation to all workplace exposures. The HHEs are generated as a result of written requests from plant managers or workers to inspect and monitor the workplace, and the IMIS data are based on OSHA monitoring of workplaces as a result of enforcement activities. Since neither is based on a random sample, their use may result in biased estimates of overall worker exposure and, consequently, occupational risk. OSHA requests comments regarding technical problems (including the direction and magnitude of biases) associated with estimating costs and benefits based on these data sets, as well as suggested methodologies that might be used to resolve these problems.

The estimates of reduced incidence of cancer and kidney dysfunction are based on median cadmium exposures. OSHA chose this measure because it is a fairly robust measure of central tendency, particularly in the absence of information to suggest that another measure would be more appropriate (e.g., when the underlying distribution is unknown). Occupational exposures often are assumed to be distributed lognormally, and on this basis the geometric mean is often considered to be the preferred measure of central tendency. However, the exposure data do not support the use of the geometric mean. (A standard statistical test for goodness of fit (the chi-square test) revealed that the hypothesis of a lognormal distribution for occupational cadmium exposure could be rejected at the 95% confidence level.) This could be, at least in part, a result of the sampling problems described above. Nevertheless, the exposure data do not justify a preference for any one underlying distribution assumption over another. Therefore, OSHA believes that using the median will help to reduce the effects of any errors that may be introduced into the analysis stemming from an inappropriate measure of central tendency as a result of an incorrect underlying distribution assumption.

OSHA requests comment on the desirability of using the median as opposed to the geometric mean (or some other measure of central tendency) under these circumstances. Furthermore, OSHA requests comments concerning the direction and magnitude of biases that may be introduced into the benefit estimates by using median exposures, especially under sampling conditions that are not random.

Cancer Risk

Evidence of the carcinogenicity of cadmium derives from both epidemiological studies and animal experimentation.

Several studies of workers exposed to cadmium indicate that the incidence rates of prostatic and lung cancers are elevated relative to the general population. OSHA's risk assessment of occupational exposure to cadmium was derived from the Takenaka rat bioassay data using the multistage model.

To estimate the expected number of cancer cases avoided due to the proposed revision of the standard, OSHA identified those workers now exposed to cadmium and calculated their cancer risk. Benefits which could be attributed to enforcement of the existing standard were not included (i.e., baseline exposures above 200 $\mu g/m^3$ were capped at 200 $\mu g/m^3$). OSHA estimates that by reducing the TWA PEL to 1 $\mu g/m^3$, the number of excess cancer deaths caused by overexposure to cadmium will be reduced by 638 deaths over a 45 year period, or 14 deaths per year. OSHA estimates that by reducing the TWA PEL to 5 $\mu g/m^3$, the number of excess cancer deaths caused by overexposure to cadmium will be reduced by 172 deaths over a 45 year period, or 4 deaths per year.

Kidney Dysfunction

There is also evidence that exposure to cadmium causes dysfunctions in the kidney.

OSHA's risk estimate of renal disorder is based upon two models, one developed by Falck and one developed by Ellis. Both models estimate the likelihood of developing kidney disease based on dose. Based on these models, OSHA has developed a range of the number of kidney disorder cases which may be prevented as a result of the proposed reductions in the PEL. OSHA examined each worker's job category to determine median exposure levels. It was assumed that workers do not rotate among positions and that the exposure

level remained constant. The benefits of each proposed standard with respect to this illness were then calculated by comparing the number of expected cases of kidney disorder before and after implementation of OSHA's proposed PELs. Using this methodology, OSHA estimates that for a TWA PEL of 1 $\mu g/m^3$ between 573 (Falck) and 8501 (Ellis) cases of occupationally related kidney disorders will be avoided over a working lifetime of 45 years (or 13 to 189 cases per year). OSHA also estimates that for a TWA PEL of 5 $\mu g/m^3$, between 476 (Falck) and 1852 (Ellis) cases of occupationally related kidney disorders will be avoided over a working lifetime of 45 years (or 11 to 41 cases per year).

OSHA believes that proper medical examinations can identify urine abnormalities in workers exposed to cadmium, often prior to the onset of more serious symptoms. The workers' exposures can then be reduced. However, OSHA has not attempted to quantify any benefits that may occur from the proposed medical surveillance program.

This proposal contains several provisions intended to reduce potential adverse effects of exposures to cadmium. Some of the provisions are interrelated. Careful analysis may suggest ways in which resources could be reallocated among the provisions to achieve a greater degree of worker protection for the same total cost. OSHA requests comments on the benefits to be expected from each of the separate provisions as they relate to each other and in light of the available health evidence. For example, how would evidence of a threshold effect with regard to kidney dysfunction affect the benefits of medical surveillance to detect kidney dysfunction at levels of exposure below the threshold?

D. Technological Feasibility

Under section 6(b)(5) of the OSH Act, the Agency is to set standards that "to the extent feasible" best protect workers from significant risks of material impairment of health (29 U.S.C. 655(b)(3)). OSHA does not believe that it can satisfy this obligation by using a lowest-common-denominator approach to protecting workers, i.e. by protecting all workers only to the extent that the most severe feasibility constraint on protecting any worker would allow. On the contrary, OSHA believes that if a minority of workers cannot be as effectively protected as the majority, that fact is not an adequate reason to forego protecting the majority to the extent feasible.

OSHA has developed this understanding of technological feasibility as a matter of policy from recent court decisions. The meaning of feasibility is most thoroughly analyzed in *USWA* v. *Marshall* 647 F. 2d at 1189. That analysis is adopted and further developed in a very recent decision concerning the asbestos standard, *Bldg. and Construction Trades Dept., AFL-CIO* vs. *Brock*, 838 F. 2d at 1258.

Under the OSH Act, which has been interpreted by the courts to be "technology forcing," OSHA is "not bound to the technological status quo." OSHA "at the very least, can impose a standard which only the most technologically advanced plants in an industry have been able to achieve—even if only in some of their operations some of the time. * * * OSHA can also force industry to develop and diffuse new technology. * * * So long as [OSHA] presents substantial evidence that companies acting vigorously and in good faith can develop the technology, OSHA can require industry to meet PELs never attained anywhere." *USWA* v. *Marshall*, 647 F. 2d at 1264–65.

In proving technological feasibility, OSHA is not required by the courts to provide "anything like certainty. * * * OSHA's duty is to show that modern technology has at least conceived some industrial strategies or devices which are likely to be capable of meeting the PEL and which the industries are generally capable of adopting." *Id.*, at 1266.

With such broad authority, OSHA must bear "the initial burden of proving the general feasibility of the standard for the industry as a whole at the rulemaking stage * * *. This proof creates a presumption of general feasibility, which shifts "to the employer in later proceedings the task of overcoming OSHA's initial finding." "[S]ince the presumption of feasibility remains rebuttable, in pre-enforcement review the court would not expect OSHA to prove the standard *certainly* feasible for *all* firms at *all* times in *all* jobs" (emphasis in original). Rather, OSHA "would have to justify the presumption, and the attendant shift in burden, with reasonable technological * * * evidence and analysis." *Id.*, at 1270.

Decribing this preliminary test of general feasibility that an OSHA standard must pass in a pre-enforcement review, the court sums up OSHA's burden of proof as follows:

> First, within the limits of the best available evidence, and subject to the court's search for substantial evidence, OSHA must prove a reasonable possibility that the typical firm will be able to develop and install engineering and work practice controls that can meet the PEL in most of its operations. OSHA can do so by pointing to technology that is either already in use or has been conceived and is reasonably capable of experimental refinement and distribution within the standard's deadlines. The effect of such proof is to establish a presumption that industry can meet the PEL without relying on respirators, a presumption which firms will have to overcome to obtain relief in any secondary inquiry into feasibility * * *. Insufficient proof of technological feasibility for a few isolated operations within an industry, or even OSHA's concession that respirators will be necessary in a few such operations, will not undermine this general presumption in favor of feasibility. Rather, in such operations, firms will remain responsible for installing engineering and work practice controls to the extent feasible, and for using them to reduce [exposures] as far as these controls can do so. In any proceeding to obtain relief from an impractical standard for such operations, however, the insufficient proof or conceded lack of proof will *reduce* the strength of the presumption a firm will have to overcome in justifying its use of respirators (emphasis in original) * * *. Such a standard of review for feasibility, of course, in no way ensures that all companies at all times and in all jobs can meet OSHA's demands * * *. *Id.* at 1272.

1. *Proposed TWA PEL of 1$\mu g/m^3$*

OSHA has preliminarily determined that the proposed standard with a TWA PEL of 1$\mu g/m^3$ is technologically feasible. OSHA recognizes that some industries may not be able to achieve the TWA PEL with engineering controls and work practices alone.

The methods which can be used to reduce employee exposure to cadmium include conventional technologies such as local exhaust ventilation systems, mechanized material transfer systems, improved housekeeping practices such as vacuuming and sealing fugitive emissions, and the use of respiratory protection if engineering controls are infeasible. Such technologies are commonly known, readily available, and currently used to some degree in the affected industries and occupations. OSHA's assessment of technological feasibility is based on information collected by the JACA Corporation and PEI Associates, consultants to OSHA, on current exposure levels resulting from existing controls, on the availability of controls needed to reduce exposures from current levels, and on other evidence present in the docket.

Local exhaust ventilation and process enclosure are the two principal types of engineering controls used to reduce employee exposure to cadmium dust and fume. Local exhaust ventilation systems consist of hoods, duct work, and collectors (baghouses). Hoods are recommended for various processes

within the affected industries and occupations, including the furnace operation and solution operation in the cadmium refining industry; the solution operation, wet solids operation, calcine operation, and dry solids operation in the pigment production industry; the solution operation and dry solids operation in the cadmium stabilizer production industry; and the impregnation operation, the coating operation, and plate preparation operation in the nickel-cadmium battery production industry. An enclosed screw conveyor is recommended for use in the cement transfer operation in the cadmium refining industry. In the twelve cross-industry occupations, a hood and exhaust system is recommended for workers in the chemical mixing, electroplating, and metal machine operator occupations.

Improving housekeeping practices, such as increasing the frequency of vacuuming, is recommended for all industries except electroplating, where exposures are already low.

In addition to the above engineering controls, worker exposures to cadmium may be reduced through implementing a fugitive emissions detection program, and through improved cleaning of equipment prior to maintenance operations.

OSHA anticipates that some respirator use will be necessary to meet the proposed TWA PEL of 1 $\mu g/m^3$ for certain processes in cadmium producing and using industries and in certain occupations. Tables VIII–C and VIII–D present estimated respirator use under a TWA PEL of 1 $\mu g/m^3$. In many of the plants in the affected industries respirators are already being used in some job categories. Most of the workers in the affected occupations already have exposures below a TWA PEL of 1 $\mu g/m^3$, but some of the remaining employees may not be able to be protected with engineering controls and would be required to use respirators. OSHA estimates that these employees would include approximately 26,000 metal machine operators that may need to be protected by respirators full time, and 155,000 repair and utility workers that may need to wear a respirator an average of 2 hours per week for intermittent exposures.

TABLE VIII–C.—RESPIRATOR USE TO COMPLY WITH A PEL OF 1 μG/M AFTER ENGINEERING CONTROLS ARE IMPLEMENTED; INDUSTRIES

Industry	Employment	Number of employees requiring respirators	Portion of shift (percent) respirators required	Number of employees, full-time equivalent respirator use
Cadmium refining and zinc smelting				
Solution operator	12	12	25	3
Cement operator	12	12	50	6
Furnace operator	37	37	100	37
Materials handler	37	0	0	0
Process supervisor	6	6	25	2
Maintenance technician	43	43	40	17
Total	147	110		65
Percent wearing respirators		75		44
Plastic stabilizer				
Dry process				
Solution operator	16	16	100	16
Dry solids operator	53	53	100	53
Process supervisor	4	4	25	1
Maintenance technician	16	16	40	6
Total	89	89		76
Percent wearing respirators		100		86
Plastic stabilizer				
Wet process				
Solution operator	88	88	100	88
Maintenance technician	22	22	40	9
Total	110	110		97
Percent wearing respirators		100		88
Pigment production				
Solution operator	18	18	100	18
Wet solids operator	22	22	100	22
Calcine operator	9	9	100	9
Dry solids operator	22	0	0	0
Process supervisor	4	4	25	1
Maintenance technician	4	4	50	2
Total	79	57		52
Percent wearing respirators		72		66
Electroplating				
Dry solids operator	2333	0	0	0
Maintenance technician	1165	0	0	0
Total	3498	0		0
Percent wearing respirators		0		0
Nickel-cadmium battery manufacture				
Materials handler	18	0	0	0
Impregnation operator	44	44	100	44
Coating operator	13	13	100	13
Plate preparation operator	70	70	100	70
Assembler	118	118	100	118
Process supervisor	4	4	50	2

TABLE VIII-C.—RESPIRATOR USE TO COMPLY WITH A PEL OF 1 μG/M AFTER ENGINEERING CONTROLS ARE IMPLEMENTED: INDUSTRIES—Continued

Industry	Employment	Number of employees requiring respirators.	Portion of shift (percent) respirators required	Number of employees, full-time equivalent respirator use
Maintenance technician	22	22	65	14
Total	289	271		261
Percent wearing respirators		94		90
Lead smelter				
Furnace operator	217	217	100	217
Material handler	82	82	100	82
Maintenance technician	320	320	75	240
Process supervisor	98	98	50	49
Sinter machine operator	95	95	100	95
Mixing room operator	13	13	100	13
Refinery operator	152	152	100	152
Total	977	977		848
Percent wearing respirators		100		87
Totals	5198	1614		1399
Percent wearing respirators		31		27

Source: Office of Regulatory Analysis, 1988.

TABLE VIII-D.—RESPIRATOR USE TO COMPLY WITH A PEL 1μg/m AFTER ENGINEERING CONTROLS ARE IMPLEMENTED: OCCUPATIONS

Occupation	Employment	Number of employees requiring respirators	Portion of shift (%) respirators required (percent)	Number of employees full-time equivalent respirator use
Chemical mixer	20,329	5,091	100	5,091
Electroplater	6,168	3,084	25	771
Furnace operator molders	20,277	0	0	0
Kiln or kettle operator	1,024	0	0	0
Heat treater	519	519	100	519
Equipment cleaner	133	133	25	33
Metal machine operator	63,472	25,685	100	25,685
Painter-prod'n & constr.	11,271	0	0	0
Maintenance painter	742	0	0	0
Repair, utility worker[1]	155,306	155,306	5	7,765
Welder, brazer, and solderer—hand	27,033	0	0	0
Welder, brazer, and solderer—machine	200,662	0	0	0
Total	506,936	189,818		39,865
Percent wearing respirators		37		8

[1] Workers in this category are assumed to need respirators an average of one shift out of 5 and 25% of the shift.
Source: Office of Regulatory Analysis, 1988.

2. Proposed TWA PEL of 5 $\mu g/m^3$

OSHA has preliminarily determined that the proposed standard with a TWA PEL of 5 $\mu g/m^3$ is technologically feasible. OSHA recognizes that some industries may not be able to achieve the TWA PEL with engineering controls and work practices alone.

The methods which can be used to reduce employee exposure to cadmium include conventional technologies such as local exhaust ventilation systems, mechanized material transfer systems, improved housekeeping practices such as vacuuming and sealing fugitive emissions, and the use of respiratory protection if engineering controls are infeasible. Such technologies are commonly known, readily available, and currently used to some degree in the affected industries and occupations. OSHA's assessment of technological feasibility is based on information collected by the JACA Corporation and PEI Associates, consultants to OSHA, on current exposure levels resulting from existing controls, on the availability of controls needed to reduce exposures from current levels, and on other evidence present in the docket.

Local exhaust ventilation and process enclosure are the two principal types of engineering controls used to reduce employee exposure to cadmium dust and fume. Local exhaust ventilation systems consist of hoods, duct work, and collectors (baghouses). Hoods are recommended for various processes within the affected industries and occupations, including the furnace operation and solution operation in the cadmium refining industry; the solution operation, wet solids operation, and calcine operation in the pigment production industry; the solution operation and dry solids operation in the cadmium stabilizer production industry; and the impregnation operation, the coating operation, and plate preparation operation in the nickel-cadmium battery production industry. An enclosed screw conveyor is recommended for use in the cement transfer operation in the cadmium refining industry. In the twelve cross-industry occupations, a hood and exhaust system is recommended for workers in the chemical mixing occupation.

Improving housekeeping practices, such as increasing the frequency of vacuuming, is recommended for all

industries except electroplating, where exposures are already low.

In addition to the above engineering controls, worker exposures to cadmium may be reduced through implementing a fugitive emissions detection program, and through improved cleaning of equipment prior to maintenance operations.

OSHA anticipates that some respirator use will be necessary to meet the proposed TWA PEL of 5 $\mu g/m^3$ for certain processes in cadmium producing and using industries and in certain occupations. Tables VIII-E and VIII-F present estimated respirator use under a TWA PEL of 5 $\mu g/m^3$. In many of the plants in the affected industries respirators are already being in some job categories. Most of the workers in the affected occupations already have exposures below a TWA PEL of 5 $\mu g/m^3$, but some of the remaining employees may not be able to be protected with engineering controls and would be required to use respirators. OSHA estimates that these employees would include approximately 5,000 chemical mixers and 500 heat treaters that may need to be protected by respirators full time.

TABLE VIII-E.—RESPIRATOR USE TO COMPLY WITH A PEL OF 5 μG/M AFTER ENGINEERING CONTROLS ARE IMPLEMENTED: INDUSTRIES

Industry	Employment	Number of employees wearing respirators	Portion of shift respirators required (percent)	Number of employees, full-time equivalent respirator use
Cadmium refining and zinc smelting				
Solution operator	12	0	0	0
Cement operator	12	12	50	6
Furnace operator	37	37	100	37
Materials handler	37	0	0	0
Process supervisor	6	0	0	0
Maintenance technician	43	43	40	17
Total	147	92		60
Percent wearing respirators		63%		41%
Plastic stabilizer dry process				
Solution operator	16	0	0	0
Dry solids operator	53	0	0	0
Process supervisor	4	0	0	0
Maintenance technician	16	0	0	0
Total	89	0		0
Percent wearing respirators		0%		0%
Plastic stabilizer wet process				
Solution operator	88	0	0	0
Maintenance technician	22	0	0	0
Total	110	0		0
Percent wearing respirators		0%		0%
Pigment production				
Solution operator	18	0	0	0
Wet solids operator	22	0	0	0
Calcine operator	9	9	100	9
Dry solids operator	22	0	0	0
Process supervisor	4	0	0	0
Maintenance technician	4	0	0	0
Total	79	9		9
Percent wearing respirators		11%		11%
Electroplating				
Dry solids operator	2333	0	0	0
Maintenance technician	1165	0	0	0
Total	3498	0		0
Percent wearing respirators		0%		0%
Nickel-cadmium battery manufacture				
Materials handler	18	0	0	0
Impregnation operator	44	0	0	0
Coating operator	13	0	0	0
Plate preparation operator	70	70	100	70
Assembler	118	118	100	118
Process supervisor	4	0	0	0
Maintenance technician	22	0	0	0
Total	289	188		188
Percent wearing respirators		65%		65%
Lead smelter				
Furnace operator	217	217	100	217
Materials handler	82	82	100	82
Maintenance technician	320	320	75	240
Process supervisor	98	0	0	0
Sinter machine operator	95	95	100	95
Mixing room operator	13	13	100	13
Refinery operator	152	0	0	0

TABLE VIII-E.—RESPIRATOR USE TO COMPLY WITH A PEL OF 5 μG/M AFTER ENGINEERING CONTROLS ARE IMPLEMENTED: INDUSTRIES—Continued

Industry	Employment	Number of employees wearing respirators	Portion of shift respirators required (percent)	Number of employees, full-time equivalent respirator use
Total	977	727		647
Percent wearing respirators		74%		66%
Totals	5189	1016		904
Percent wearing respirators		20%		17%

Source: Office of Regulatory Analysis, 1988.

TABLE VIII-F.—RESPIRATOR USE TO COMPLY WITH A PEL OF 5sG/M AFTER ENGINEERING CONTROLS ARE IMPLEMENTED: OCCUPATIONS

Occupation	Employment	Number of employees wearing respirators	Portion of shift (%) respirators required (percent)	Number of employees, full-time equivalent respirator use
Chemical mixer	20,329	5,091	100	5,091
Electroplater	6,168	0	0	0
Furnace operator, molders	20,277	0	0	0
Kiln or kettle operator	1,024	0	0	0
Heat treater	519	519	100	519
Equipment cleaner	133	0	0	0
Metal machine operator	63,472	0	0	0
Painter-prod'n & constr.	11,271	0	0	0
Maintenance painter	742	0	0	0
Repair, utility worker	155,306	0	0	0
Welder, brazer, and				
Solderer—hand	27,033	0	0	0
Welder, brazer, and				
Solderer—machine	200,662	0	0	0
Total	506,936	5,610		5,610
Percent wearing respirators		1%		1%

Source: Office of Regulatory Analysis, 1988.

E. Cost of Compliance

This section presents OSHA's estimates of the compliance costs that may be incurred by employers in the seven industry sectors and twelve cross-industry occupations affected by the proposed cadmium standard.

A baseline of current industry practice was determined for each cadmium producing and using industry from information on current production methods and available engineering controls obtained during the PEI and JACA information gathering efforts, and from submissions to the record. Costs of engineering controls recommended to achieve the proposed TWA PEL were estimated on the assumption that new controls would be added to those already in place or would be adopted where none are currently used. Separate cost calculations were done for the twelve cross-industry occupations identified as having cadmium exposures.

In addition to the costs for engineering controls and respirators, costs were estimated for the following regulatory provisions.

Exposure Monitoring

This provision of the proposed standard would require employers to determine whether any employee may be exposed to airborne concentrations of cadmium. Measurements would be made by monitoring the breathing zone of one representative employee over an eight hour period for each job classification and for each shift. Exposure monitoring would have to be done initially to determine current exposure levels. If initial monitoring indicated that exposures were less than the action level, no further monitoring would be required. Exposure monitoring would have to be done every three months if exposures were greater than the TWA PEL, or every six months when exposures are less than the TWA PEL but greater than or equal to the action level. Exposure monitoring for the 15 minute excursion limit would have to be done in conjunction with, and with the same frequency as, exposure monitoring for the TWA PEL. Recordkeeping would be required with exposure monitoring.

OSHA believes that at least two methods and types of monitoring devices, charcoal tubes and passive dosimeters, are currently available to take these measurements.

Protective Clothing and Equipment

This provision would require the employer to provide protective clothing and equipment when the employee is exposed to cadmium above the TWA PEL or the excursion limit. Protective clothing and equipment which may be required by the standard include gloves, head coverings, foot coverings, coveralls, face shields, and vented goggles. Many industries already provide personal protective clothing and equipment to workers exposed to high levels of cadmium.

Regulated Areas

This would require employers to establish a separate regulated area where air exposure levels exceed the TWA PEL or the excursion limit. This provision includes appropriate signs, limited access to the area, the provision of respirators to those entering the area, and the prohibition of activities including smoking, eating, drinking, chewing gum or tabacco, and the application of cosmetics.

Hygiene Facilities and Practices

This provision would require the employer to provide change rooms, showers, and separate lunch rooms for employees working in areas where airborne exposure to cadmium exceeds the TWA PEL. Based upon JACA site visits, it is assumed that showering and lunchroom facilities are already provided as part of the baseline in the major cadmium-using and -producing industries. A substantial part of the cost of this provision is attributable to wages for employee time to shower at the end of each workshift.

Medical Surveillance

This proposed provision would require preplacement, annual, and termination medical examinations for employees who will be or have been exposed to airborne concentrations of cadmium at levels at or above the action level. These examinations would require, but would not be limited to, a medical and work history, a complete medical examination, pulmonary function tests, blood analysis, and urinalysis. OSHA believes that all of these examinations can be performed at any clinic, doctor's office, or hospital, and are currently provided by many employers in the seven specific cadmium-using and -producing industries. Employees in the cross-industry occupations also are currently provided with all the required provisions of the medical exam except the urine and blood analysis.

Medical removal would be required if a physician determines in a written opinion that the employee should be removed from exposures at or above the action level or that the employee cannot wear a respirator. The employer is required to maintain records of employee medical exams.

Employer Obligations Under the Hazard Communication Standard

29 CFR 1910.1200 requires employers to train employees in handling hazardous chemicals present in the workplace. The Hazard Communication Standard also requires the use of labels and Material Safety Data Sheets to communicate hazards to employees. OSHA expects that by complying with the Hazard Communication Standard, employees working in areas which may be contaminated with cadmium will be made aware of the existing health hazards of cadmium exposure. However, some additional training and information requirements incremental to what is required by the Hazard Communication Standard are included in this standard.

Summary of Costs

Tables VIII–G and VIII–H summarize industry and occupation compliance cost estimates of the proposed cadmium standard with a 1 μg^3 TWA PEL and .5 $\mu g/m^3$ action level. Tables VIII–I and VIII–J summarize industry and occupation compliance cost estimates of the proposed cadmium standard with a 5 $\mu g/m^3$ TWA PEL and 2.5 $\mu g/m^3$ action level. The engineering costs associated with reaching the TWA PEL include ventilation control systems and enclosed material transfer systems. Non-engineering costs needed to achieve the proposed TWA PEL are related to work practices, including an emissions detection program, additional cleaning and decontamination time prior to maintenance activities, and improved housekeeping. Additional costs would be incurred to cover personal protective equipment including respirators, respirator programs, and respirator fit testing. Costs associated with the other provisions of the standard include those for medical surveillance (except medical removal), exposure monitoring, information and training, hygiene facilities, and regulated areas. In the instance that a worker must be medically removed and there is no alternative job for him/her, the unit cost for removal would include an administrative cost, wage retention for six months, training costs for a new hire to replace the removed employee, and the necessary medical examinations. (Since the extent of potential application of this provision is unknown, a total cost for the provision is not estimated in the summary of costs).

TABLE VIII–G.—SUMMARY OF ESTIMATED TOTAL ANNUAL COSTS ASSOCIATED WITH A 1 μG/M STANDARD FOR CADMIUM: INDUSTRIES

Proposed provisions	Cadmium refining	Dry process stabilizer production	Wet process stabilizer production	Pigment production	Nickel-cadmium battery production	Lead smelting	Electro plating	Total
Ventilation systems	$228,000	$111,500	$63,800	$230,600	$194,000	$0	$0	$827,900
Material transfer systems [1]	0	0	0	0	0	0	0	0
Total annual engineering costs	228,000	111,500	63,800	230,600	194,000	0	0	827,900
Fugitive emissions program	0	33,700	0	0	0	0	0	33,700
Decontamination program	1,000	0	0	0	0	0	0	1,000
Respirators	0	26,700	32,800	8,500	75,900	0	0	143,900
Respirator fit testing	0	900	1,100	0	2,200	0	0	4,200
Exposure monitoring	11,800	13,500	8,700	10,900	13,900	20,700	194,700	274,200
Medical surveillance	1,800	1,400	1,700	900	4,200	15,300	0	25,300
Hygiene practices	0	48,200	59,000	30,500	117,600	0	0	255,300
Information and training	700	2,200	2,600	1,800	2,500	9,300	0	19,100
Regulated areas	200	100	50	200	300	150	0	1,000
Housekeeping	40,700	74,900	32,300	32,300	6,400	19,400	0	206,000
Recordkeeping	500	400	500	400	800	2,400	0	5,000
Total annual nonengineering costs	56,700	202,000	138,750	85,500	223,800	67,250	194,700	968,700
Total annual cost of compliance: Industries	284,700	313,500	202,550	316,100	417,800	67,250	194,700	1,796,000

[1] Enclosed and automated system for use in cadmium refining industry only; estimated annual operating and maintenance costs and capital costs are more than offset by labor savings.

Source: Office of Regulatory Analysis, 1988, based on JACA Corporation, 1988.

TABLE VIII-H.—SUMMARY OF ESTIMATED TOTAL ANNUAL COSTS ASSOCIATED WITH A 1 μG/M STANDARD FOR CADMIUM: OCCUPATIONS

Proposed provisions:	Chemical mixer	Electro-plater	Furnace Operator: holder	Kiln or Kettle operator	Heat treater	Equip-ment cleaner	Metal machine operator	Painter	Mainte-nance painter	Repair and utility worker	Welder; brazer solder-er—hand	Welder, brazer solderer—machine	Total annual cost: Cross-industry occupations
Ventilation systems	$13,626,300	$2,757,800	$0	$0	$0	$0	$33,790,300	$0	$0	$0	$0	$0	$50,174,400
Material transfer systems [1]	0	0	0	0	0	0	0	0	0	0	0	0	0
Total annual engineering costs	$13,626,300	$2,757,800	$0	$0	$0	$0	$33,790,300	$0	$0	$0	$0	$0	$50,174,400
Fugitive emissions program	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0
Decontamination program	0	0	0	0	0	0	0	0	0	0	0	0	0
Respirators	2,717,100	1,031,600	0	0	287,100	44,500	13,708,000	0	0	10,390,400	0	0	28,178,700
Respirator fit testing	37,800	22,900	0	0	3,900	1,000	190,500	0	0	1,151,600	0	0	1,407,700
Exposure monitoring	506,400	205,700	308,800	15,600	28,000	7,200	1,959,800	171,600	11,300	8,371,000	411,700	3,055,600	15,052,700
Medical surveillance	594,700	292,000	0	0	51,200	15,900	2,534,900	0	0	15,000,400	0	0	18,489,100
Hygiene practices	3,000,500	1,817,600	0	0	305,900	78,400	15,138,200	0	0	18,306,800	0	0	38,647,400
Information and training	111,700	67,700	0	0	11,400	2,900	563,500	0	0	3,407,100	0	0	4,164,300
Regulated areas	4,600	2,800	0	0	500	0	23,000	0	0	7,000	0	0	37,900
Housekeeping	0	0	0	0	0	0	0	0	0	0	0	0	0
Recordkeeping	28,200	17,100	0	0	2,900	800	142,100	0	0	859,100	0	0	1,050,200
Total annual nonengin-eering costs	$7,001,000	$3,457,400	$308,800	$15,600	$690,900	$150,700	$34,260,000	$171,600	$11,300	$57,493,400	$411,700	$3,055,600	$107,028,000
Total annual cost of compliance: Occupations	$20,627,300	$6,215,200	$308,800	$15,600	$690,900	$150,700	$68,050,300	$171,600	$11,300	$57,493,400	$411,700	$3,055,600	$157,202,400

[1] Enclosed and automated system for use in cadmium refining industry only; estimated annual operating and maintenance costs and capital costs are more than offset by labor savings.
Source: Office of Regulatory Analysis, 1988, based on JACA Corporation, 1988.

TABLE VIII-I.—SUMMARY OF ESTIMATED TOTAL ANNUAL COSTS ASSOCIATED WITH A 5 μG/M STANDARD FOR CADMIUM: INDUSTRIES

Proposed provisions:	Cadium refining	Dry process stabilizer production	Wet process stabilizer production	Pigment production	Nickel-cadmium battery production	Electroplating	Lead smelting	Total
Ventilation systems	$228,100	$111,500	$63,800	$194,000	$194,000	$0	$0	$791,400
Material transfer systems [1]	0	0	0	0	0	0	0	0
Total annual engineering costs	$228,100	$111,500	$63,800	$194,000	$194,000	$0	$0	$791,400
Fugitive emissions program	$0	$33,700	$0	$0	$0	$0	$0	$33,700
Decontamination program	1,000	0	0	0	0	0	0	1,000
Respirators	0	0	0	1,400	52,700	0	0	54,100
Respirator fit testing	0	0	0	0	1,500	0	0	1,500
Exposure monitoring	5,800	6,800	4,600	6,600	9,100	194,700	16,500	244,100
Medical surveillance	1,800	1,400	1,700	900	4,200	0	15,300	25,300
Hygiene practices	0	0	0	4,900	82,000	0	0	86,900
Information and training	700	2,200	2,600	1,700	2,500	0	9,300	19,000
Regulated areas	150	0	0	100	200	0	100	550
Housekeeping	40,700	74,900	32,300	32,300	6,400	0	19,400	206,000
Recordkeeping	500	400	500	400	800	0	2,400	5,000
Total annual nonengin-eering costs	$50,650	$119,400	$41,700	$48,300	$159,400	$194,700	$63,000	$677,150
Total annual cost of com-pliance: Industries	$278,750	$230,900	$105,500	$242,300	$353,400	$194,700	$63,000	$1,468,550

[1] Enclosed and automated system for use in cadmium refining industry only; estimated annual operating and maintenance costs and capital costs are more than offset by labor savings.

Source: Office of Regulatory Analysis, 1988, based on JACA Corporation, 1988.

TABLE VIII-J.—SUMMARY OF ESTIMATED TOTAL ANNUAL COSTS ASSOCIATED WITH A 5 μg/m STANDARD FOR CADMIUM: OCCUPATIONS

Proposed provisions:	Chemical mixer	Electro-plater	Furnace operator; molder	Kiln or kettle operator	Heat treater	Equip-ment cleaner	Metal machine operator	Painter	Mainte-nance painter	Repair and utility worker	Welder, brazer, solderer—hand	Welder, brazer solderer—machine	Total annual cost: Cross-industry occupations
Ventilation systems	$13,626,300	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$13,626,300
Material transfer systems [1]	0	0	0	0	0	0	0	0	0	0	0	0	0
Total annual engineering costs	$13,626,300	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$13,626,300

TABLE VIII–J.—SUMMARY OF ESTIMATED TOTAL ANNUAL COSTS ASSOCIATED WITH A 5 μg/m STANDARD FOR CADMIUM: OCCUPATIONS—Continued

Proposed provisions:	Chemical mixer	Electro-plater	Furnace operator; molder	Kiln or kettle operator	Heat treater	Equip-ment cleaner	Metal machine operator	Painter	Mainte-nance painter	Repair and utility worker	Welder, brazer, solderer—hand	Welder, brazer solderer—machine	Total annual cost: Cross-industry occupations
Fugitive emissions program	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0
Decontamination program	0	0	0	0	0	0	0	0	0	0	0	0	0
Respirators	2,717,100	0	0	0	277,000	0	0	0	0	0	0	0	2,994,100
Respirator fit testing	37,800	0	0	0	3,800	0	0	0	0	0	0	0	41,600
Exposure monitoring	506,400	93,900	308,800	15,600	28,000	4,600	1,463,200	171,600	11,300	5,368,000	411,700	3,055,600	11,438,700
Medical surveillance	594,700	292,000	0	0	51,200	15,900	2,534,900	0	0	15,000,400	0	0	18,489,100
Hygiene practices	3,000,500	0	0	0	305,900	0	0	0	0	0	0	0	3,306,400
Information and training	111,700	67,700	0	0	11,400	2,900	563,500	0	0	3,407,100	0	0	4,164,300
Regulated areas	4,600	0	0	0	500	0	0	0	0	0	0	0	5,100
Housekeeping	0	0	0	0	0	0	0	0	0	0	0	0	0
Recordkeeping	28,200	17,100	0	0	2,900	800	142,100	0	0	859,100	0	0	1,050,200
Total annual nonengineering costs	$7,001,100	$470,700	$308,800	$15,600	$680,700	$24,200	$4,703,700	$171,600	$11,300	$24,634,600	$411,700	$3,055,600	$41,489,500
Total annual cost of compliance: Occupations	$20,627,300	$470,700	$308,800	$15,600	$680,700	$24,200	$4,703,700	$171,600	$11,300	$24,634,600	$411,700	$3,055,600	$55,115,800

[1] Enclosed and automated system for use in cadmium refining industry only; estimated annual operating and maintenance costs and capital costs are more than offset by labor savings.
Source: Office of Regulatory Analysis, 1988, based on JACA Corporation, 1988.

Annualized costs of capital include a 10% compound interest charge and the capital repayment over the useful life of the equipment. Variations in the useful life of each type of equipment were taken into account.

For a 1 $\mu g/m^3$ TWA PEL, total annual cost for the industry sectors identified is $1.8 million, affecting approximately 5200 employees. The total annual cost for the 506,900 employees in the twelve cross-industry occupations is $157.2 million. The combined annual cost for all affected industries and occupations with a TWA PEL of 1 ug/m^3 is $159.0 million.

For a 5 ug/m^3 TWA PEL, total annual cost for the industry sectors identified is $1.5 million, affecting approximately 5200 employees. The total annual cost for the 506,900 employees in the twelve cross-industry occupations is $55.1 million. The combined annual cost for all affected industries and occupations with a TWA PEL of 5 ug/m^3 is $56.6 million.

F. Economic Feasibility Analysis

The criteria for determining economic feasibility are provided in the "lead decision." There the court said:

A standard is not infeasible simply because it is financially burdensome (citation omitted) or even because it threatens the survival of some companies within an industry * * *. A standard is feasible if it does not threaten "massive dislocation * * *. No matter how initially frightening the projected * * * costs of compliance appear, a court must examine those cuts in relation to the financial health and profitability of the industry and the likely effect of such costs on unit consumer prices * * *. [T]he practical question is whether the standard threatens the competitive stability of an industry (citation omitted) or whether * * * the standard might wreck such stability or lead to undue concentration. * * * [To demonstrate economic feasibility], OSHA must construct a reasonable likelihood that these costs will not threaten the existence or competitive structure of an industry, even if it does portend disaster for some marginal firms. *USWA* v. *Marshall*, 847 F.2d 1189.

Thus, the fact that compliance costs could be burdensome to an industry or that some firms could go out of business because of these costs does not mean a standard is economically infeasible. The issue is not whether the standard creates economic hardship for specific firms in an industry or even for the industry as a whole. For a standard to be economically infeasible the courts require more. A standard is economically infeasible when the costs imposed are so overwhelming that they create "massive dislocation" of the industry, threaten the "existence" or "competitive stability" of the industry or lead to "undue concentration" within the industry. *USWA* v. *Marshall*, 647 F.2d 1189.

OSHA believes that it must base its determination of economic feasibility upon substantial evidence that an industry sector is not threatened with massive dislocation, undue concentration, or competitive instability attributable to the cost of complying with the standard at the time compliance deadlines arrive. In order to make this judgment, projected capital or annual costs of compliance must be examined in relation to the financial health and profitability of an industry sector and the likely effect of such costs on prices. A standard is economically feasible if costs can be passed on in price increases and/or absorbed by firms, and any necessary absorption of costs will not threaten the competitive stability of the industry.

OSHA has preliminarily determined that it is economically feasible for the affected industries and occupations to comply with the provisions of the proposed cadmium standard at a TWA PEL of 1 $\mu g/m^3$ and at a TWA PEL of 5 $\mu g/m^3$. OSHA expects that one small and one medium size dry process stabilizer plant and a small pigment production plant may experience a significant adverse economic impact as a result of this standard. Tables VIII–K and VIII–L present estimates of the cost per plant among cadmium-producing and -using industries and the cost per employee among cross-industry occupations to meet TWA PELs for cadmium of 1 $\mu g/m^3$ and 5 $\mu g/m^3$, respectively.

Tables VIII–M and VIII–N summarize the potential economic impact of the proposed cadmium standards on the industry sectors considered in this analysis for TWA PELs of 1 $\mu g/m^3$ and 5 $\mu g/m^3$, respectively. Compliance costs are presented as a percent of profits and as a percent of revenues for each affected industry by plant size. This represents the two extreme cases that may result: compliance costs absorbed completely from profits or compliance costs completely passed through to consumers. The actual outcome will generally be a combination of these two cases.

TABLE VIII-K.—INDUSTRY COSTS PER PLANT AND OCCUPATION COSTS PER EMPLOYEE WITH A 1 μg/m Standard

Cadmium using/producing plants	Per plant annual cost
Cadmium refining	
Small	$56,200
Large	57,700
Stabilizers: dry process	
Small	76,200
Medium	76,200
Large	80,700
Stabilizers: wet process	
Small	38,900
Large	43,100
Pigment production	
Small	60,800
Medium	62,300
Large	64,750
Nickel cadmium batteries	
Small	55,600
Medium	71,100
Large	78,200
Lead smelting	
Small	21,900
Large	24,000
Electroplating	
Small	200

Cross-industry occupation	Annual per employee cost
Chemical mixer	$1,015
Electroplater	1,009
Furnace operator	15
Kiln or kettle operator	15
Heat treater	1,334
Equipment cleaner	1,135
Metal machine operator	1,073
Painter	15
Maintenance painter	15
Repair and utility worker	373
Welder, brazer, solderer—hand	15
Welder, brazer, solderer—machine	15

Source: Office of Regulatory Analysis, 1988, based on JACA Corporation, 1988.

TABLE VIII-L.—INDUSTRY COST PER PLANT AND OCCUPATION COSTS PER EMPLOYEE WITH A 5 μG/M STANDARD

Cadmium using/producing plants	Per plant annual cost
Cadmium refining	
Small	$55,700
Large	56,000
Stabilizers: dry process	
Small	57,700
Medium	57,700
Large	58,000
Stabilizers: wet process	
Small	21,100
Large	21,400
Pigment production	
Small	48,100
Medium	48,500
Large	48,800

TABLE VIII-L.—INDUSTRY COST PER PLANT AND OCCUPATION COSTS PER EMPLOYEE WITH A 5 μG/M STANDARD—Continued

Cadmium using/producing plants	Per plant annual cost
Nickel cadmium batteries	
Small	49,000
Medium	60,000
Large	65,000
Lead smelting	
Small	20,500
Large	22,600
Electroplating	
Small	200

Cross-industry occupation	Annual per employee cost
Chemical mixer	$1,015
Electroplater	78
Furnace operator	15
Kiln or kettle operator	15
Heat treater	1,314
Equipment cleaner	184
Metal machine operator	75
Painter	15
Maintenance painter	15
Repair and utility worker	161
Welder, brazer, solderer—hand	15
Welder, brazer, solderer—machine	15

Source: Office of Regulatory Analysis, 1988, based on JACA Corporation, 1988.

TABLE VIII-M.—ESTIMATED ECONOMIC IMPACTS OF A 1 μG/M³ CADMIUM STANDARD

Industry and firm size (# of firms)	Compliance costs as a percent of before tax profit (percent)	Increased prices required to fully offset compliance costs of average firm (percent)	Average total annual per plant compliance costs (1987 dollars)
Cadmium refining			
Small (2)	NM	0.18	56,200
Large (3)	NM	0.18	57,700
Stabilizers: dry			
Small (1)	583.45	52.51	76,200
Medium (1)	228.30	20.55	76,200
Large (2)	37.08	3.34	80,700
Stabilizers: wet			
Small (3)	61.94	5.57	38,900
Large (2)	27.47	2.47	43,100
Pigment Production			
Small (1)	202.51	10.13	60,800
Medium (1)	69.25	3.46	62,300
Large (3)	28.66	1.43	64,750
Batteries			
Small (1)	46.36	1.85	55,600
Medium (4)	5.93	0.24	71,100
Large (1)	2.54	0.10	78,200
Lead smelting			
Small (2)	NM	0.07	21,900
Large (1)	NM	0.03	24,000
Electroplating			
Large (1166)	7.59	0.33	200

NM: Not meaningful.
Source: Office of Regulatory Analysis, 1988, based on JACA Corporation, 1988.

TABLE VIII-N.—ESTIMATED ECONOMIC IMPACTS OF A 5 μG/M³ CADMIUM STANDARD

Industry and firm size (# of firms)	Compliance costs as a percent of before tax profit (percent)	Increased prices required to fully offset compliance costs of average firm (percent)	Average total annual per plant compliance costs (1987 dollars)
Cadmium refining			
Small (2)	NM	0.17	55,700
Large (3)	NM	0.18	56,000
Stabilizers: dry			
Small (1)	441.77	39.76	57,700
Medium (1)	172.86	15.56	57,700
Large (2)	26.64	2.40	58,000
Stabilizers: wet			
Small (3)	33.61	3.03	21,100
Large (2)	13.64	1.23	21,400
Pigment production			
Small (1)	160.33	8.02	48,100
Medium (1)	53.89	2.69	48,500
Large (3)	21.69	1.08	48,800
Batteries			
Small (1)	40.83	1.63	49,000
Medium (4)	5.00	0.20	60,000
Large (1)	2.11	0.08	65,000
Lead smelting			
Small (2)	NM	0.07	20,500
Large (1)	NM	0.03	22,600
Electroplating (1166)	7.59	0.33	200

NM: Not meaningful.
Source: Office of Regulatory Analysis, 1988, based on JACA Corporation, 1988.

At a TWA PEL of 1 μg/m³, if none of the compliance costs were passed through to consumers, a typical firm in the affected industries would have a decrease in before-tax profit of 3 to 70 percent. The one small and one medium-sized firm in the dry process stabilizer manufacturing industry and the one small plant in pigment production would lose all before tax net profits.

At a TWA PEL of 1 μg/m³, if all of the compliance costs were passed through to consumers, prices in most of the affected industries would increase between 0.1 percent and 5 percent. In the dry process stabilizer industry, however, prices would have to increase 53 percent for the one small plant in this industry, and about 21 percent for the one medium-size plant. The small pigment producing plant would need a price increase of 10 percent to fully offset estimated compliance costs.

At a TWA PEL of 5 μg/m³, compliance costs range from 2 percent to 54 percent of profits for most of the affected plants. Alternatively, price increases of between 0.1 percent and 3 percent would fully offset compliance costs. For the one small and one medium sized plant in the dry process stabilizer production industry and for the one small plant in the pigment production industry, price increases of 8 percent to 40 percent would be needed, which may cause an adverse economic impact for these firms.

OSHA believes that at TWA PELs of 5 μg/m³ and 1 μg/m³, these changes are affordable in most of the industry sectors based on the relatively small size of the costs in relation to both profits and sales. Since cadmium is a co-product of zinc and lead smelting, it is unlikely that cadmium refiners will not be able to absorb the necessary costs. Other regulated facilities, with the exception of electroplaters, are all operating units or subsidiaries of large, well-capitalized manufacturing companies. These plants should be able to finance capital and up-front compliance costs. Estimated costs in the electroplating industry are relatively minor due to already low exposures and should be absorbed through negligible price increases.

OSHA anticipates that the plants identified above, i.e. the small and medium sized dry process plastic stabilizer plants and the small pigment production plant, may not be able to recoup compliance costs. These three plants employ a total of about 50 employees. The effect on industry output is expected to be negligible due to the low capacity utilization rates currently present in these industries.

In the twelve cross-industry occupations, no significant adverse impact is expected, since per-employee costs are relatively low and spread among many industries.

G. Regulatory Flexibility Analysis

Pursuant to the Regulatory Flexibility Act of 1980 (Pub. L. 96-353, 94 Stat. 1164 [5 U.S.C. 601]), OSHA has given special consideration to the economic impacts of the proposed standard on small entities. During the process of developing a revised standard for occupational exposure to cadmium, OSHA carefully considered size factors such as number of employees, total assets, and gross revenues to assure that the proposed standard would not have a disproportional impact on small firms. OSHA anticipates the possibility of an adverse economic impact on the one small and one medium sized firm in the dry process plastic stabilizer industry and on the one small pigment production plant. Firms in the other industries should be able to absorb and/or pass through compliance costs through a combination of reduced profits or increased prices. In the cross-industry occupations, costs are generally on a per-employee basis and thus should not have a disproportional effect on smaller firms. OSHA concludes that, with the exception of the three plants identified above, the proposed cadmium standard with a TWA PEL of 5 μg/m³ or 1 μg/m³ will not have a significant adverse

impact on a substantial number of small businesses.

IX. Environmental Impact Assessment

The environmental impact of lowering the permissible exposure limit for cadmium to 5 $\mu g/m^3$ or 1 $\mu g/m^3$ is expected to be insignificant. The proposed TWA PELS will cause a net decrease in atmospheric cadmium emissions because of the effectiveness of the controls in reducing ambient air cadmium levels. The control device selected to remove cadmium emissions captured from the air by exhaust hoods is a baghouse; a baghouse typically achieves control efficiencies above 99 percent.

There will be a negligible amount of additional solid waste generated as a result of the proposed standard. Most of the substances collected by the emission controls are recycled and reprocessed.

X. Summary and Explanation of the Proposed Standard

OSHA believes that, based on currently available information, the proposed requirements set forth in this notice are necessary and appropriate to provide adequate protection to employees exposed to cadmium. Numerous reference works, journal articles, and other data obtained by OSHA have been taken into consideration in the development of this proposed standard.

The language of the standard and the order of the various provisions are consistent with drafting in other recent OSHA health standards, such as the formaldehyde and benzene standards. OSHA believes that a similar style should be followed from standard to standard to facilitate uniformity of interpretation of similar provisions. Section 6(b)(5) of the Act states that health standards shall also be based on "experience gained under this and other health and safety laws."

Scope and Application: Paragraph (a)

The proposed standard applies to all occupational exposures to all forms of cadmium. This proposal includes the construction, agriculture, maritime and general industries where workers are subject to cadmium exposures that can adversely affect their health and that therefore need to be lowered.

The standard covers all industries, where cadmium exposure is routine, such as nickel-cadmium battery manufacturing, lead and zinc smelting and refining, cadmium refining, electroplating, plastic stabilizers and multi-colored pigments production. It also covers occupations common to many industries that may involve substantial exposure to cadmium during the handling or heating of cadmium compounds. These occupations include: Chemical mixers; furnace, kiln, or kettle operators; molders; heat treaters; equipment cleaners; metal machine operators; painters; electroplaters (in captive operations); utility workers; and welders, brazers, and solderers.

Under the Construction Safety Act (40 U.S.C. 333), 29 CFR 1911.10 and 29 CFR 1912.3, OSHA was required to establish an Advisory Committee on Construction Safety and Health (ACCSH) and to consult with that committee in the formulation of regulatory proposals which would apply to employment in construction. The Agency has determined that approximately 66,000 construction workers have potential cadmium exposure levels addressed by the proposed standard. Based on the record developed to date, the Agency preliminarily determined that it is appropriate to propose the same regulatory language for the construction industry as for general industry. Therefore, OSHA presented the proposed cadmium standard, along with pertinent explanatory materials, to the ACCSH, and on June 14, 1989, OSHA formally consulted with the ACCSH. At that time the Advisory Committee recommended that OSHA include the construction industry within the scope of the proposed rule. The ACCSH also established a work group to develop comments on the cadmium proposal and to consider what, if any, other regulatory provisions were reasonably necessary to protect construction workers from cadmium exposure. At its meeting on September 13, 1989, the committee provided further advice. It recommended that OSHA develop a separate cadmium standard for the construction industry with certain provisions tailored to the particular conditions in that industry.

OSHA has discussed these matters with the Construction Advisory Committee. OSHA anticipates that the Advisory Committee will be submitting pre-hearing comments to this rulemaking concerning the special conditions in the construction industry and a draft of any modifications to the proposed rule that may be appropriate and necessary to respond to these conditions. OSHA expects to place the final cadmium standard applicable to the construction industry in 29 CFR part 1926.

OSHA recognizes that the Committee's comments and other record evidence may lead the Agency in this rulemaking to promulgate a standard for the construction industry that is different in some respects from the standard for the general industry (29 CFR 1910.1027). OSHA requests the public and interested parties to provide information and comments on how, if at all, the proposed cadmium standard should be modified in its application to the construction industry.

The proposal also covers the agriculture industry. Although information on exposures to cadmium in agriculture is limited, OSHA has reason to believe that some cadmium exposures may occur in the agriculture industry, although rarely. OSHA's proposal covers all occupational exposures to cadmium because there may be serious health consequences for any person who is exposed to cadmium.

Definitions: Paragraph (b)

"Action level" is defined in this standard as an airborne concentration of cadmium (level) at or above which medical surveillance, air monitoring, and the provision of a respirator to any employee who requests one are required. Other requirements of the standard are not triggered until exposures reach the TWA PEL. Where exposures are determined to be below the action level, no further compliance activities are required of the employer, except for training of employees.

The action level concept has been used previously to trigger various provisions in other published final OSHA health standards, e.g., Asbestos (51 FR 22612, June 20, 1986), Benzene (52 FR 34460, September 11, 1987); Formaldehyde (52 FR 4668, December 4, 1987); Acrylonitrile (43 FR 45809, October 3, 1978); and Ethylene Oxide preamble (48 FR 17284, April 21, 1983). Use of an action level has been found to stimulate employers to lower exposure levels to avoid increasing costs of compliance with provisions triggered by the action level. As exposures are lowered the risk of illness among workers decreases.

In other standards and in this proposal, action levels have traditionally been set at one-half the PEL. The focus of OSHA's concern is the provision of immediate medical surveillance and follow-up care to workers exposed above the action level who in the past have been exposed to much higher cadmium levels than would be allowed under this standard. Evidence in the record indicates that for some of these workers, irreversible and progressive adverse health effects may rapidly develop unless medical intervention and reduction or cessation of exposures occur immediately (HRG/ICWU petition for an Emergency Temporary Standard, 1986, Ex. 0-1; ACGIH, Ex. 8-664; Lauwerys for

Cadmium Council, Ex. 12–07; Mason, Ex. 8–669, and Friberg, Ex. 8–668). For example, workers with 5 years of exposure to cadmium at levels of 100 $\mu g/m^3$ or workers with 10+ years of exposure to cadmium at levels of 50 $\mu g/m^3$ may be at such risk. Up to 10,000 workers are currently exposed at or above 100 $\mu g/m^3$ of cadmium, at least on a part-time basis. If they have not yet suffered damage from that exposure, in particular to their kidneys, they may be close enough to the threshold that any additional exposure would lead to material impairment of health.

As indicated in the Medical Surveillance section below, the purpose of the initial and periodic medical examinations is to establish the current health status of the employee. Of particular interest are past exposures to cadmium that may have damaged the workers' health. The medical surveillance program outlined below includes screening methods for early detection of illness and is targeted to the organ systems that are most sensitive to cadmium toxicity, namely the lungs and kidneys. This will facilitate the identification or diagnosis and treatment of chronic effects of cadmium toxicity at an early stage. OSHA is seriously considering alternate ways to address the issue of identifying workers in need of immediate medical care, one of which is an action level of one-fifth the TWA PEL of 5 $\mu g/m^3$. The more workers covered by medical surveillance, the greater the likelihood of identifying all who need immediate medical care. OSHA estimates that 60,000 to 100,000 more workers would have access to medical surveillance if the action level were set at 1 $\mu g/m^3$ for a TWA PEL of $\mu g/m^3$ than if it were set at half that TWA PEL.

Another alternative OSHA is considering for identifying workers in need of immediate medical care is the provision of special medical surveillance for workers with past high exposures.

OSHA has preliminarily determined that the action levels for the alternative TWA PELs are technologically feasible in the industries and occupations. Air monitoring at the proposed alternative action levels is feasible and achievable.

According to OSHA's risk assessment, there is continuing significant risk at a TWA PEL of 5 $\mu g/m^3$. Under the recent Asbestos decision (*Building and Construction Trades Department, AFL–CIO* vs. *Brock*, 838 F.2d 1258), where there is continuing significant risk, OSHA should use its legal authority to impose additional requirements on employers to further reduce risk when those requirements will result in a greater than de minimis incremental benefit to workers' health. OSHA's preliminary conculsion is that the action level will result in a very real and necessary further reduction in risk over that provided by the TWA PEL alone for these workers.

OSHA requests comment on whether the action level can be used to increase the likelihood of identifiying workers in need of immediate medical care by increasing the number of workers covered by medical surveillance and on which action level is appropriate. OSHA also requests comments on the use of other methods for identifying workers in need of immediate medical care, such as targeting screening efforts to those workers with higher past cumulative cadmium exposures. This issue is addressed in OSHA's question under 30, above. OSHA further requests comments on which, if any, special medical surveillance provisions would be necessary for workers under the traditional action level of one-half the TWA PEL.

In the Significance of Risk section, OSHA has outlined the Agency's concerns about inherent uncertainty in any quantitative risk assessment and has requested comments on OSHA's risk assessment and the appropriate level for the final TWA PEL. If during the public hearings evidence indicates that significant risk remains at a TWA PEL of 5 $\mu g/m^3$ and workers with past high exposures are in need of immediate medical care, OSHA would consider setting a different action level at one-fifth the TWA PEL of 5 $\mu g/m^3$ or additional medical provisions for veteran workers.

"Emergency" is defined as any occurence such as, but not limited to, equipment failure, rupture of containers, or failure of control equipment that results in an unexpected release of a significant amount of cadmium.

The concept of "Emergency exposure" is defined as that exposure to airborne cadmium which would occur if the employee were not using respiratory protective equipment. The definition is consistent with OSHA's previous use of the term in other standards.

The definitions of "Assistant Secretary", "authorized person", "Director", "high-efficiency particulate absolute [HEPA] air filter", and "regulated area" are consistent with OSHA's previous use of these terms found in other health standards. The employers' obligations with respect to HEPA filters and regulated areas are discussed later.

Permissible Exposure Limits: Paragraph (c)

OSHA proposes two alternative permissible exposure limits for all cadmium compounds, calculated as an 8-hour time-weighted average exposure (TWA PEL): give and one micrograms of cadmium per cubic meter of air. OSHA has proposed two PELs based upon its concern that respirator usage will be extensive at the lower proposed PEL and based upon its risk assessment described in Section VI of this preamble. Employers are required to assure that no employee is exposed to an airborne concentration of cadmium in excess of permissible exposure limits (PELs).

OSHA's risk assessment indicates significant risk of cancer and kidney dysfunction at the current PELs. A significant reduction in risks of cancer and kidney dysfunction will be achieved at either of the proposed TWA PELs. Reducing the proposed TWA PEL to 1 (5) $\mu g/m^3$ would substantially reduce the estimated total cancer risk to 2.1 (10.6) excess deaths per thousand workers. This reduced risk, nonetheless, remains significant.

Based upon the recent Asbestos decision, [*Building and Construction Trades Department, AFL–CIO* v. *Brock*, 838 F.2d 1258 (D.C. cir. 1988)], when there is significant risk remaining OSHA must take additional actions to the extent feasible to further reduce that risk. If OSHA were to rely exclusively on the TWA PEL to eliminate significant risk, then the TWA PEL would need to be set at a level less than 1 $\mu g/m^3$. This is because if 1 cancer death per 1,000 employees attributable to occupational exposure is taken to constitute a significant risk, then a significant risk would remain at both exposure levels. OSHA anticipates that the ancillary provisions in the proposed standard will further reduce risk.

OSHA is also setting an excursion limit (EL) of 5 (25) ug/m^3 averaged over a sampling period of fifteen minutes to further reduce cumulative exposures to employees. OSHA believes the EL is necessary particularly because the Agency's risk assessment indicates substantial risk at the proposed 8-hour TWA PELs.

Section 6(b)(5) of the OSH Act requires the Agency to adopt health standards that most adequately assure protection against significant risks of material health impairment, to the extent feasible. In proposing the adoption of an 8-hour TWA PEL for cadmium no lower than 1 (5) $\mu g/m^3$ based upon feasibility considerations, OSHA believes that significant cancer

risk would persist below those levels. The Agency believes that additional protection against such continuing significant risk would be provided by supplementing the TWA PEL with additional feasible control measures. OSHA believes, for example, that a limitation on short-term exposures of 5 (25) $\mu g/m^3$ over a 15-minute period will provide such protection and would be feasible in the affected industries and occupations. Compliance with the 15-minute excursion limit (EL) is expected to result in an incremental reduction in the total dose of cadmium that an employee would receive if protected by the 8-hour TWA PEL alone.

Control of short-term exposures in the workplace is generally recommended by the American Conference of Governmental Industrial Hygienists (ACGIH) as good industrial hygiene practice. The ACGIH asserts that even where there is not enough toxicological data to warrant a short-term exposure limit (STEL) based on acute effects, excursions above the TWA PEL should be controlled (Ex. No. 8–687, "Threshold Limit Values for Chemical Substances in the Work Environment," ACGIH). The ACGIH recommends that " * * * in a well controlled process exposure excursions should be held within some reasonable limits" (Ex. No. 8–687). Specifically, based on consideration of exposure variability generally observed in actual industrial processes, the ACGIH recommends that excursions and "short-term exposures should * * under no circumstances * * * exceed five times the TLV [TWA]" (Ex. No. 8–687).

Support for the proposed adoption of an excursion limit for cadmium is also found in OSHA's recent experience with its rulemaking for ethylene oxide (53 FR 11414). The issues addressed during that rulemaking with respect to the necessity of adopting an excursion limit to reduce significant risk are relevant to the cadmium rulemaking. In response to questions on the short-term limit set forth in the proposed rule for EtO (48 FR 17284, April 21, 1983), numerous comments and data were received by OSHA. However, the final EtO rule published on June 22, 1984, which lowered the permissible 8-hour TWA from 50 ppm to 1 ppm (49 FR 25734), reserved decision on the question of whether the standard should contain a short-term limit. In the June 22, 1984 final rule, OSHA stated that upon its review of comments submitted by the Office of Management and Budget (OMB) pursuant to Executive Order 12291, OSHA determined that certain issues relating to the short term limit were important and merited further consideration.

OSHA's decision not to issue a short-term limit for EtO centered on three findings. First, the available health data did not demonstrate the risks from EtO exposure to be dose rate-dependent. In other words, the studies did not indicate that the risk from exposure to a given dose of EtO are greater when that dose is distributed at high concentrations over a short period of exposure than when that dose is distributed at a lower concentration over a longer period of time. Second, since the effects of EtO are assumed to be *dose* dependent rather than dose-rate-dependent, OSHA concluded that reduction of the total dose was the critical factor in dealing with the significant risks of EtO exposure. Therefore, the Agency believed that the 1 ppm TWA PEL was sufficient to minimize significant risk, within the bounds of feasibility. Third, in terms of industrial hygiene and methods of controlling EtO, it was felt that compliance with the TWA PEL would in itself necessitate some control of short-term exposures, particularly for employees whose exposure consists primarily of short-term bursts.

A petition for review of OSHA's decision not to adopt a short-term limit for EtO subsequently was filed by the Public Citizen Health Research Group, pursuant to section 6 (f) of the OSH Act (29 U.S.C. 655(f)).

On July 25, 1986, the U.S. Court of Appeals for the District of Columbia Circuit issued a decision on the ethylene oxide standard (*Public Citizen Health Research Group* v. *Tyson*, 796 F.2d 1479) in response to the petition from Public Citizen. In that decision, the Court upheld OSHA's permissible exposure limit for 1 ppm as an 8-hour time-weighted average, finding that OSHA had "complied with the relevant legal standards in promulgating the 1 ppm PEL" (796 F.2d at 1503). In addition, the Court upheld OSHA's determination that the evidence in the rulemaking record did not establish the existence of a dose-rate relationship for the health effects of EtO. However, the Court rejected OSHA's argument that the lack of such an established dose-rate effect rendered it unnecessary for the Agency to promulgate a short-term limit for EtO. The Court noted:

> The agency recognized that EtO exposures at 1 ppm still allowed a significant health risk * * * If in fact a STEL would further reduce a significant health risk and is feasible to implement, then the OSH Act *compels* the agency to adopt it (barring alternative avenues to the same result) (796 F.2d at 1505).

Therefore, the Court said, in order for OSHA to avoid issuing a short term limit for EtO, the Agency must find either that a short-term limit would not reduce the significant risk remaining at the TWA PEL of 1 ppm, or that a short-term limit would not be feasible. If the Agency cannot make either of these two findings, the court continued, then a short-term limit must be issued. The court remanded the EtO standard to the Agency for further proceedings, specifically directing OSHA to "either adopt a short-term limit or explain why empirical or expert evidence on exposure patterns makes a short-term limit irrelevant to controlling long-term exposures." (*Public Citizen* v. *Tyson*, 796 F.2d at 1507).

Pursuant to the court decision, OSHA proposed the adoption of a 5 ppm 15-minute excursion limit for EtO on the basis of record evidence that compliance with the excursion limit would reduce the total dose for certain employees and, therefore, would reduce the significant risk from exposure to EtO that would exist without the excursion limit. Thus, an excursion limit for EtO was adopted in accordance with the Tyson decision.

OSHA believes that adoption of an excursion limit would reduce the total exposure dose for certain cadmium-exposed workers as it did for certain EtO workers, thus reducing the significant risk they would otherwise face at the TWA PEL alone. The Agency further believes that adoption of a cadmium excursion limit is justified under the Tyson decision. Thus, OSHA proposes the adoption of an excursion limit for cadmium to supplement the proposed TWA PEL and thereby reducing to the extent feasible the total dose and significant risk that cadmium workers are expected to continue to experience. The proposed excursion limit of 5 (25) $\mu g/m^3$ is in accordance with the ACGIH recommendation that such limits should not exceed five times the TWA PEL.

Exposure Monitoring: Paragraph (d)

The proposed standard imposes monitoring requirements pursuant to Section 6(b)(7) of the OSH Act (29 U.S.C. 655) which mandates that any standard promulgated under section 6(b) shall, where appropriate, "provide for monitoring or measuring of employee exposure at such locations and intervals, and in such manner as may be necessary for the protection of employees."

The purposes served by requiring periodic air sampling for employee exposures to cadmium include:

determination of the extent of exposure at the worksite; prevention of employee overexposure; identification of the sources of exposure to cadmium; collection of exposure data so that the employer can select the proper control methods to be used; and evaluation of the effectiveness of the selected methods. Monitoring enables employers to meet the legal obligation of the standard to ensure that their employees not be exposed to cadmium in excess of the prescribed levels and to notify employees of their exposure levels, as required by section 8(c)(3) of the Act. In addition, collection of exposure monitoring data enables the examining physician to be informed of the existence and extent of potential sources of occupational diseases.

Exposure monitoring is important to determine the level of cadmium to which employees are exposed. Exposure levels also act as triggers for implementing certain provisions of this standard to protect employees. Medical surveillance and exposure monitoring provisions of the standard are triggered, for example, by exposure at or above the action level or above the EL. All the remaining provisions of the standard are triggered by exposure above the TWA PEL.

The exposure monitoring provisions require the employer to determine the exposure for each employee exposed to cadmium. Samples must be taken within the employee's breathing zone (personal samples) and must represent the employee's exposure to airborne concentrations of cadmium over an eight-hour period for the TWA PEL, without regard to the use of respirators. In addition, for employees exposed at or above the action level and for certain other employees, the employer must also monitor employees who are expected to have the highest exposure levels over a fifteen minute period to determine whether they are exposed above the EL.

Air sampling for all employees may be required for initial monitoring in certain occupations. In many cases, the employer may monitor selected employees to determine "representative employee exposures." Representative exposure sampling is permitted when there are a number of employees performing essentially the same job under the same conditions. Under such circumstances it may be sufficient to monitor a fraction of such employees in order to obtain data that are "representative" of all employees. Representative personal sampling for employees engaged in similar work with cadmium exposure of similar length, duration and level can be achieved by measuring that member of the exposed group reasonably expected to have the highest exposure. This result is then attributed to the remaining employees of the group. At the very least, full-shift sampling must be conducted for each job function in each job classification, in each work area, and for each shift. These samples must consist of at least one sample representative of the entire shift or consecutive representative samples taken over the length of the shift.

Initial monitoring of workplace exposures is required of all employers who have a place of employment covered by this standard. The initial monitoring must be conducted within 60 days of the effective date of the final standard. However, to eliminate unneeded monitoring, if an employer has comparable and adequate workplace monitoring data gathered within 180 days prior to the effective date of the standard, those data will satisfy the requirements of this standard for initial monitoring.

The results of initial and periodic monitoring determine subsequent monitoring frequency. If the monitoring results show employee exposures are below the action level, (confirmed by another monitoring taken at least seven days later), then no further monitoring is required. OSHA estimates that well over half the estimated 512,000 workers exposed to cadmium are currently below 1 $\mu g/m^3$.

However, if changes occur, such as changes in the production process, raw materials, control equipment, personnel, work practices, or finished products, and these changes may lead to higher exposure, then the employer must resume monitoring. If the initial or periodic monitoring results show employee exposures at or above the action level, but at or below the TWA PEL, then the employer must repeat monitoring for these individuals at least every six months. If initial or periodic monitoring indicates exposures to be above the TWA PEL, then the employer must monitor every three months. OSHA believes these monitoring schedules, which are similar to those required by other OSHA standards such as arsenic, lead, and ethylene oxide, are necessary and sufficient to provide useful information for evaluating employees' exposures. The recent benzene and formaldehyde standards require semi-annual exposure monitoring. This proposed standard would require more frequent monitoring to promptly identify exposures above the proposed TWA PEL.

Monitoring for the EL is generally to be carried out simultaneously with, and according to, the required monitoring schedules discussed above. However, if an employee is exposed in excess of the EL, that employee shall be monitored at least once every six months even if the employee's 8-hour TWA exposure is below the action level. If the initial or periodic monitoring reveals the employee to be above the TWA PEL or at or above the action level, but on two consecutive measurements taken at least seven days apart the employee's exposure is not above the EL, no further monitoring for the EL is required.

Periodic monitoring provides the employer with assurance that employees are not experiencing higher exposures that may require the use of additional controls. In addition, periodic monitoring reminds employees and employers of the continued need to protect against the hazards associated with exposure to cadmium.

OSHA recognizes that monitoring can be a time-consuming, expensive endeavor and therefore offers employers the incentive of discontinuing monitoring for employees whose sampling results indicate exposures are below the action level. It is hoped that this will encourage employers to control their employees' exposures to cadmium below the action level, thus maximizing the protection of employees' health.

Employees are further protected because additional monitoring is required when there is a change in process, production, control equipment, personnel, work practices or other conditions which may result in new or additional exposures to cadmium.

The employer is required to use monitoring and analytical methods that have an accuracy, at a confidence level of 95 percent, of not less than plus or minus 25 percent for airborne concentrations of cadmium. Many laboratories presently have methods to detect cadmium at these and lower levels with at least the required degree of accuracy. Methods of measurement for each of the proposed TWA PELs are described in Appendix E.

The proposed standard further requires that employers notify each of their employees individually of the results of personal monitoring samples. Notification is to be given in writing. In addition, employers must post monitoring results in an appropriate location accessible to all affected employees. A written notice ensures that each employee is notified. Posting the results ensures that other employees, their designated representatives, supervisors, and employers are also aware of the results. The employer is obligated to provide written notice and

post results within 15 working days after receipt of the results. Whenever the TWA PEL and/or the EL is exceeded, the written notification must contain a statement that the TWA PEL and/or the EL has been exceeded and a description of the corrective action(s) being taken by the employer to reduce workplace exposures to or below the TWA PEL and/or the EL. The requirement to inform employees is in accordance with section 8(c)(3) of the Act and is necessary to assure employees that the employer is making efforts to furnish them with a safe and healthful work environment.

The employer is also required to allow employees or their designated representatives an opportunity to observe the employee exposure monitoring. This provision is required by section 8(c)(3) of the Act [29 U.S.C. 657(c)(3)]. It is provided for in paragraph (o) of the proposal and is discussed in more detail below.

Regulated Areas: Paragraph (e)

The proposed standard contains requirements that regulated areas be established wherever airborne exposures are above the TWA PEL and/or EL. Access to these areas is to be regulated and limited to authorized persons. Regulated areas are to be demarcated in any manner that adequately alerts employees of the boundaries of these areas. To increase the performance-orientation of this standard, no detailed requirements are specified on how the regulated areas should be demarcated.

Regulated areas are to be established not only when the TWA PEL is exceeded by also when the EL is exceeded. For example, whenever the TWA PEL or EL is exceeded during a maintenance operation, a regulated area shall be established for the length of time required to perform that operation.

The purpose of a regulated area is to ensure that employers make employees aware of the presence of cadmium at levels above the permissible exposure limits in the workplace, thereby minimizing the number of employees exposed. This may be achieved by posting warning signs. Since the use of respiratory protective equipment is required in regulated areas, the demarcation of such an area should effectively warn employees not to enter these areas unless they are authorized to do so and only if they are using the proper personal protective equipment. In this way, employees who work in another area of the worksite will not be unnecessarily exposed to cadmium if they are required by their job to work in a regulated area part the workday. Due to the serious nature of the adverse health effects of cadmium, no one should be in a regulated area without proper personal protection. This provision will reduce an employees' overall cadmium exposure thereby reducing that employees' risk of illness. OSHA considers this to be necessary in view of the remaining significant risk of cancer at either proposed TWA PEL.

The establishment of regulated areas is an effective means of limiting excess cadmium exposure to as few employees as possible. This is consistent with good industrial hygiene practice whenever exposure to a toxic substance can cause serious health effects. This requirement has additional benefits to employers in that, by limiting access to these areas to authorized persons, the employer's obligation to implement provisions of this standard for exposure above the TWA PEL or EL is limited to as few employees as possible.

Access to the regulated area is restricted to "authorized persons". For the purposes of this standard, these are persons required by their job duties to be present in the area, as authorized by the employer.

Regulated areas are to be established in all work areas, including maintenance operations, where either the TWA PEL or EL is exceeded. In OSHA's view, the existence of a hazard is the basis for determining the need for such protective measures, and not the type of operation or work being performed. Areas where exposures are temporarily over either the TWA PEL or the EL while maintenance is being performed, for example, need to be demarcated to warn employees who are not essential to the performance of that maintenance to keep out of the areas. Demarcation is also necessary to warn employees required to be in the regulated area that respirators must be worn to avoid excessive exposures via inhalation and that good personal hygiene practices must be observed to avoid exposures to cadmium via ingestion. Good personal hygiene practices include refraining from smoking or eating in regulated areas and washing hands and face after leaving the area. Readily observable temporary sign(s) posted at the boundary of the area that are consistent with signs required by the Hazard Communication Standard will be sufficient to remind employees that respirators and good personal hygiene practices are needed and that unprotected people should not enter the area.

Methods of Compliance: Paragraph (f)

The proposed standard requires employers to institute engineering and work practice controls as the primary means to reduce and maintain employee exposures to cadmium at levels at or below the TWA PEL or the EL. Engineering controls involved the installation of equipment, such as forced air ventilation, or the modification of a process, such as enclosing it. Work practice controls involve the manner in which a task is performed, such as how the worker positions himself/herself relative to the source of exposure and/or the engineering controls. The proposal also requires employers to implement engineering and work practice controls even if they establish that feasible engineering and work practice controls are inadequate to lower exposures to or below the TWA PEL or the EL. Engineering and work practice controls, in such circumstances, must be used to reduce employee exposures to the extent possible, and supplemental protection is to be provided through the use of respirators selected in accordance with paragraph (g).

Primary reliance on engineering controls and work practices is consistent with good industrial hygiene practice and with the Agency's traditional adherence to a hierarchy of preferred controls. However, regarding this traditional hierarchy of controls, OSHA published an Advance Notice of Proposed Rulemaking (ANPR) on February 22, 1983 (48 FR 7473) to solicit comments on methods of compliance issues. Among these issues was OSHA's preference for the use of engineering controls over respirators for control of employees' exposures to air contaminants. Many employers have felt the need for increased flexibility in the use of respiratory protection. Based on the comments received in response to the ANPR, OSHA published a Federal Register notice on June 5, 1989, (54 FR 23991) proposing to incorporate additional flexibility in its methods of compliance requirements. OSHA proposed to do this by setting forth more explicitly those circumstances under which respiratory protection generally may be used due to some infeasibility of engineering control. The five sets of circumstances that have been identified by OSHA from data in the record where engineering controls may generally be infeasible include:

1. During the time necessary to install engineering controls;
2. Where feasible engineering controls result in only a negligible reduction in exposure;
3. During emergencies, life saving, recovery operations, repair, shutdowns, and field situations where

there is a lack of utilities for implementing engineering controls;
4. Operations requiring added protection where there is a failure of normal controls; and
5. Entries into unknown atmospheres.

In addition to requesting comment on the appropriateness of allowing the use of respirators during the activities discussed above, OSHA requested comment on other alternatives in that proposed rule as well. Among other alternatives, comment was requested on the appropriateness of permitting the use of respirators: (1) For work situations in which the hazardous exposure is of very brief duration, and, (2) where the costs of the respirator program would be less than those of engineering controls and yet equal protection would be provided. A provision-by-provision discussion of these revisions is included in the proposed methods of compliance rule and will not be included here.

OSHA's traditional hierarchy specifies that engineering controls and work practices are to be used in preference to respirators. Engineering controls are reliable, provide consistent levels of protection to a large number of workers, can be monitored continually and inexpensively, allow for predictable performance levels, and remove toxic substances from the workplace. Once removed, the toxic substance no longer poses a threat to the employee. The effectiveness of engineering controls does not depend to any marked degree on human behavior, and the operation of equipment is not as vulnerable to human error as is personal protective equipment. For these reasons, engineering controls are preferred by OSHA.

Respirators may be relied upon by employers only under certain circumstances: in emergencies; when engineering or work practice controls have not yet been implemented; when the implementation of all such feasible controls fails to reduce exposures to or below the TWA PEL or EL; and during certain brief or intermittent operations.

Engineering controls can be grouped into 3 main categories: (1) Substitution, (2) containment and isolation, and (3) ventilation, both general and localized. Quite often a combination of these controls can be applied to an industrial hygiene control problem to achieve satisfactory air quality. It may not be and usually is not necessary or appropriate to apply all these measures to any specific potential hazard.

Substitution can be the appropriate solution to an industrial hygiene problem. One of the best ways to prevent workers from being exposed to a toxic substance is to stop using it entirely. Although substitution is not always possible, one should always consider whether non-toxic or a less toxic material could be substituted for a more toxic one. Another kind of substitution which may provide effective control of an air contaminant is exchanging one type of process equipment for another, or in some cases, exchanging one process for another process itself.

For example, a process change from batch operation to continuous operation will usually reduce exposure. This is true primarily because the frequency and duration of workers' potential contact with process materials is reduced in continuous operations. Similarily, automation of a process can further reduce the potential hazard.

In addition to substitution, containment (enclosure) and isolation should also be considered. Containment consists of installation of an enclosure (physical barrier) to contain the source of a hazard, thereby separating it from most workers. Workers can be isolated without such containment by appropriately placing the employees in a clean room or cab, or by placing the employees at a greater distance from the source of cadmium exposure.

Frequently containment and isolation maximize the benefits of additional engineering concepts such as local exhaust ventilation. For example, the charging of mixers is the most significant operation in many processes that use formulated ingredients. When one of the ingredients in the formulation is of relatively high toxicity, it is worthwhile to contain the mixing operation. That is, it is worthwhile to install a mixing room, thereby confining the airborne contaminants potentially generated by the operation to a small area. By ensuring containment, the application of ventilation principles to control the contaminant at the source (i.e., the mixer) is more effective.

Ventilation, general or local, is by far the most important engineering control available to the industrial hygienist. Its principal application is to maintain airborne concentrations of contaminants at acceptable levels in the workplace.

A local exhaust system is used to carry off an air contaminant by capturing the contaminant at or near its source before it spreads throughout the workplace. General ventilation, on the other hand, allows the contaminant to spread throughout the workroom but dilutes it by circulating large quantities of air into and out from the workroom. A local exhaust system is generally preferred to ventilation-by-dilution (general ventilation) because it provides a cleaner and healthier work environment. Also, a local exhaust system, with a relatively small volume of air, uses a smaller fan and dust collector.

Work practices, as distinguished from engineering controls, involve the way a task is performed. The Agency has also found that modified work practices can aid in achieving compliance with the TWA PEL and the EL. Some fundamental and easily implemented work practices are: (1) Following the proper procedures to minimize exposures in operating production and control equipment; and (2) not eating, drinking, smoking or storing foods in regulated areas.

Good housekeeping plays a key role in the control of occupational health hazards. Dust on overhead ledges and on floors should be removed before it is made airborne by traffic, vibration, and random air currents. Allowing accumulations of hazardous substances in a work area increases the risk that the worker's exposure will rise above the TWA PEL and/or action level. For example, immediate cleanup of any spills of toxic material is a very important work practice control measure. In addition, a regular cleanup schedule using HEPA filtered vacuum cleaners is an effective method of removing cadmium dust from the work area.

Periodic inspection and maintenance of process equipment and control equipment such as ventilation systems is an important work practice control. In plants where total containment is used as an engineering control, the failure of process equipment or of a ventilation system can result in hazardous exposures. Frequently, equipment which is near failure or in disrepair will not perform normally. Regular inspections can detect abnormal conditions so that maintenance can then be performed. If equipment is routinely inspected, replaced, or repaired before failure is likely, there is less chance that hazardous exposures will occur.

In addition to the above work practice controls, workers must know the proper operating procedures for engineering controls. If a worker inappropriately performs an operation away from an exhaust hood, the control measure will be of no use. Such action may also contaminate the work area. Workers can be alerted to safe operating procedures through booklets, instructional signs, labels, discussions at safety meetings, and through other educational means.

Good supervision provides further support for ensuring that proper work practices are carried out by the workers. By persuading a worker to position the exhaust hood properly or to improve work practices, such as weighing toxic materials or handling contaminated scoops or shovels, a supervisor can do much to minimize unnecessary exposure to air contaminants.

Employees' exposures also can be controlled by scheduling operations with the highest exposures at a time when the fewest employees are present. For example, clean-up operations in which toxic substances are involved might be performed at night or at times when the usual production staff is not be present. Such methods of controlling worker exposures to contaminants are known as administrative controls. However, one form of administrative control, worker rotation, is prohibited by OSHA as a method of compliance. Worker rotation reduces the extent of exposure to individual employees but increases the number of employees exposed. Since cadmium appears to be a carcinogen and causes kidney dysfunction at low exposure levels, OSHA is compelled to prohibit these practices, which would place more employees at risk.

OSHA has traditionally relied less on respirators in the hierarchy of controls because there are so many problems associated with their use. Often work is strenuous, and the increased breathing resistance of the respirator reduces its acceptability to employees. Severe safety problems are presented by respirators which may limit an employee's vision and ability to communicate. In some difficult and dangerous jobs, effective vision or communication is vital to a safe, efficient operation. Voice transmission through a respirator can be difficult, annoying, and fatiguing. In any event, movement of the jaw in speaking can cause leakage, thereby reducing the efficiency of the respirator and decreasing the employee's protection. Also skin irritation can result from wearing a respirator in hot, humid conditions. Such irritation can cause considerable distress to workers and may disrupt work schedules. To be used effectively, respirators must be individually selected; fitted and periodically refitted; conscientiously and properly worn; regularly maintained; and replaced as necessary. In many workplaces, these preconditions for effective respirator use are difficult, if not impossible, to achieve. For this reason and others, OSHA has concluded that reliance upon respirators should be minimized.

Workers exposed above the TWA PEL or EL are required to wear respirators during the performance of their normal job functions in order to lower their exposures to or below the TWA PEL or EL. Medical examinations evaluating an employee's physical fitness to wear a respirator are required to be provided within 30 days after assignment to an area where respirators are required for any employee who has not already had such an examination within the preceeding 12 months.

Industry claims in comments to the proposed respirator standard that respirators provide reliable employee protection when used in a good respirator program (Docket H-049, Respiratory Protection Revision). However, because respirator efficiency ultimately relies on the individual employee's good work practices, respirator programs place the burden of protection on the employee. Engineering controls may have certain disadvantages, such as their cost and availability, but these disadvantages are insufficient to justify the use of respirators alone. OSHA requests any information or data indicating that respirators offer equal or better protection than engineering controls. The role of other factors in providing this protection, such as the presence of hygiene facilities, should be evaluated with respirator usage as well.

Experience in industry shows that most healthy workers do not have physiological problems wearing properly chosen and fitted respirators. Common health problems such as claustrophobia (an intolerance of feeling enclosed and the subjective feeling of breathing difficulty), chronic rhinitis, nasal allergies, (where it is necessary to remove the respirator frequently to deal with nasal discharge) and chronic sinusitis may cause difficulty in breathing while wearing a respirator for employees affected by these health conditions.

Other difficulties associated with respirator use involve employees with respiratory system and cardiac diseases. Respiratory diseases include chronic obstructive pulmonary disease, emphysema, asthma, and moderate to severe pneumoconiosis, many of which can result from cadmium exposures. Cardiac or cardiorespiratory diseases that may affect respirator wear include coronary thrombosis, any type of congestive heart disease, other ischemic heart diseases, and hypertension.

The wearing of a negative pressure respirator increases the resistance to inspiration. The problem is reduced with powered air-purifying respirators and with positive pressure atmosphere-supplying respirators. Exhalation resistance with modern negative pressure respirators does not significantly increase expiratory effort.

The amount of difficulty associated with respirator use will clearly depend both on the degree of cardiorespiratory inadequacy and on the amount of physical effort required by the work. Some people who may have difficulty wearing a negative pressure respirator should be able to manage well with a positive pressure type respirator. The decision about the fitness of the individual to wear a respirator is a judgment that can only be made by a qualified physician who must take into account the state of the individual's health as well as the physical requirements of the job. OSHA requires medical examinations that target these health problems for workers required to wear respirators in accordance with paragraph (g).

In some circumstances (*e.g.*, certain maintenance and repair operations, emergencies, or during periods when equipment is being installed), OSHA recognizes that respirators may be essential to guarantee worker health and safety, and provision is made in paragraph (g) for their use as primary controls in these instances where engineering and work practice controls cannot be used to achieve either the TWA PEL or the EL. In other circumstances where work practices and engineering controls alone cannot reduce exposure levels to the TWA PEL and the EL, respirators also may be used for supplemental protection. In these situations, the burden of proof of infeasibility is appropriately placed on the employer.

In addition, paragraph (f)(2) requires an employer who has employees exposed over either the TWA PEL or the EL to establish and implement a written compliance program, which describes the methodology to be used to reduce employee exposure within his workplace to or below the TWA PEL and/or the EL. The plan must provide for this to be accomplished through engineering and work practice controls to the extent feasible and required by the standard. These written plans must include a schedule for implementation; must be furnished upon request for examination and copying to representatives of the Assistant Secretary, representatives of the Director of NIOSH, and affected employees or their representatives; and must be updated at least once a year

until employee exposures have been reduced to or below the TWA PEL and/or EL. Once employee exposures have been reduced to or below the TWA PEL or EL by engineering and/or work practice controls, the compliance plan need not be updated, unless, of course, exposures again rise above the TWA PEL or EL. In such cases, a new compliance plan would have to be drafted.

OSHA believes that the provision that requires the employer to give preference to engineering controls and work practices over the use of respirators will lead to a protective and cost-effective approach. This matter is being raised in the Agency's proceeding on methods of compliance (see Proposed rule, Methods of Compliance, June 5, 1989, 54 FR 23991). If evidence relevant to cadmium is submitted in the methods of compliance rulemaking, and the evidence is persuasive that some modification in required methods of compliance is indicated, OSHA will consider making appropriate changes to the cadmium standard.

Respiratory Protection: Paragraph (g)

Respirators are necessary as supplementary protection to reduce employee exposure when engineering and work practice controls cannot achieve the necessary reduction to or below he TWA PEL and the EL. Respirators may also be necessary at other times: while such controls are being implemented, during emergency situations, and for brief or intermittent exposures that cannot be controlled through engineering and work practice controls, (e.g. certain maintenance operations). Finally, a respirator must be provided by the employer for all authorized employees in regulated areas.

Due to the remaining significant risk at the action level, OSHA is also proposing to require employers to provide respirators to employees exposed above the action level who request one. This is in keeping with the precedent set forth in the Occupational Noise Exposure standard (29 CFR 1910.95) regarding the use of personal protective equipment. In that standard, under the hearing conversation program, paragraph (i), employers have to provide personal protective equipment to all employees exposed at or above the action level at no cost to employees.

The proposed standard requires that whenever respirators are required to reduce employee exposures, the employer must provide respirators appropriate to the exposure level and at no cost to the employee. Employers must also ensure that respirators are used properly when required. Because of the risk of serious adverse health effects from cadmium exposures, respirator usage is allowed in the above mentioned circumstances in order to reduce an employee's cumulative dose of cadmium.

The proposal contains specific requirements for the use, selection, maintenance, and fitting of respirators. Table 1 lists the type of respirator to be used at each airborne concentration of cadmium in the workplace. The respirator selection table is consistent with OSHA's proposed revisions to the respirator standard (29 CFR 1910.134). While the employer must select the appropriate respirator from the table on the basis of the airborne concentration of cadmium, the employer may always select a respirator providing greater protection, (i.e., one prescribed for higher concentrations of cadmium than present in his workplace).

The standard requires that employers permit employees to leave regulated areas to readjust the respirator facepiece for proper fit, to change the filters, or to replace the respirator. It also required employers to permit employees to have the regulated area to wash their faces to avoid potential skin irritation associated with respirator use.

OSHA is requiring quantitative fit testing of all tight-fitting air-purifying respirators (either positive or negative-pressure) when used at exposures exceeding 10 times the TWA PEL, (10 $\mu g/m^3$ for a TWA PEL of 1 $\mu g/m^3$, or 50 $\mu g/m^3$ for a TWA PEL 5 $\mu g/m^3$), because proper fit is essential to the performance of these respirators. Whenever quantitative fit testing is used to assess the fit of a negative pressure respirator, a fit factor of 10 times the protection factor for that class of respirators shall be achieved. When quantitative fit testing is used to assess the fit of a positive pressure respirator, the employer shall test a negative pressure respirator made by the same manufacturer, which is the same model and size, to determine whether the facepiece to face seal is adequate. The seal is adequate if the fit factor is 10 times the protection factor for the relevant class of negative pressure respirators. If the fit is not correct, cadmium contaminated workplace air may enter the facepiece through gaps and leaks in the facepiece seal, instead of passing through the filter material.

Obtaining a proper respirator fit may require the fit testing of a variety of different mask sizes from several manufacturers to select the facepiece with the best fit (i.e., least leakage around the faceseal) for each employee. A property fitted facepiece helps to reduce inhalation leakage to a minimum.

To tailor the testing to the circumstances of the employer's establishment, OSHA permits the employer to choose either quantitative or qualitative fit testing if cadmium exposures are less than 10 times the TWA PEL. Mandatory protocols for the type of testing the employer chooses are set forth in Appendix C.

Quantitative fit testing is a procedure whereby the level of penetration of a test agent of known concentration is measured inside the facepiece of the respirator. Quantitative respirator fit testing is generally recognized as the better method for determining how well a respirator fits a particular individual. It provides a quantitative assessment of the extent of the fit (i.e. the fit factor). It allows the employer to continue testing until the optimum or best fitting respirator is identified and selected for the employee. However, quantitative fit testing requires the use of moderately sophisticated testing equipment and is more expensive to perform than qualitative fit testing. This may reduce its availability in some worksites. Also, testing services may not be available in all parts of the country to provide quantitative fit testing services for small employers.

Qualitative fit testing does not provide a numeric measure of the tightness of the fit but simply determines whether a respirator fits or not. Qualitative fit testing is a technique whereby a person wearing a respirator is tested to see whether a test agent with a detectable odor or taste threshold can be detected inside the respirator. If the odor or taste cannot be detected, the respirator is said to fit. Qualitative fit testing is more subjective than quantitative testing because it depends on the individual's ability to detect the test agent.

OSHA believes that while quantitative fit testing may have some advantages, qualitative testing conducted in accordance with the protocols described in Appendix C can adequately accomplish the intent of the standard at lower exposure levels, to ensure that each employee is assigned and wears the respirator that provides a proper fit with the least possible leakage. Comments are requested on all aspects of fit testing.

It is important that all employees who wear respirators be medically screened as part of a regular medical examination to determine employee fitness for respirator usage. Respirator usage may present a burden to the employee's cardiopulmonary system. This burden may result in symptoms such as

shortness of breath, chest pain, dizziness or fatigue. These symptoms may be exacerbated by pre-existing lung disease such as chronic bronchitis, emphysema, asthma or pneumoconiosis. Paragraph (l)(7), therefore, requires that medical examinations be made available to workers with a job that requires the use of a respirator. The medical examination is required within 30 days of assignment to a job requiring a respirator unless the employee has received a complete medical examination within the preceeding 12 months. This is to ensure that individuals are not required to use a respirator without a timely medical examination evaluating their ability to wear one. The medical examination is made available to determine whether any health conditions exist which would affect the employee's ability to wear a respirator. If an examining physician determines, based on the employee's most recent exam, that an employee will be unable to function normally while wearing a respirator, then the employee shall be afforded the opportunity to transfer as set forth in paragraph (l)(12).

OSHA has not exempted occasional users of respirators from the medical evaluation requirement. OSHA believes that users need to be evaluated for their fitness to wear respirators as well. In addition, applying such an exemption might create administrative problems (Docket Number H-049, Respiratory Protection Revision).

The standard allows workers with cadmium exposures above the action level to request a respirator and requires employers to provide the respirator at no cost to the employee. Due to the serious nature of the adverse health effects of cadmium exposure, workers who are required to be trained under the provisions of the Hazard Communication Standard and are made aware of such health problems may choose to use respirators to further reduce their risks of disease. Medical examinations of the employee's fitness to wear a respirator will not be required prior to issuance of a respirator for workers who elect to wear respirators. However, examinations for respirator usage are required as part of a routine medical surveillance, at least once a year, for workers exposed to cadmium at or above the action level who voluntarily wear a respirator.

Since OSHA's risk assessment indicates a significant remaining cancer risk at a TWA PEL of 1 $\mu g/m^3$, or higher, OSHA has required all air purifying respirators to be equipped with a HEPA filter, regardless of the exposure level. OSHA believes that HEPA filters provide an extra margin of safety at all levels of exposure. OSHA requests comments on whether this provision is appropriate for exposures at lower levels.

The employee must be properly trained to wear the respirator, to know why the respirator is needed, and to understand the limitations of the respirator. An understanding of the hazard involved is necessary to enable employees to take steps for their own protection. The respiratory protection program implemented by the employer must conform to that set forth in 29 CFR 1910.134 which contains the basic requirements for proper selection, use, cleaning and maintenance of respirators.

Emergency Situations: Paragraph (h)

The proposed standard would require employers to prepare a written plan of action to be followed in the event of occurrences that would result in massive releases of airborne cadmium. Examples of such emergencies are ruptures of containers and control or operating equipment failures. Emergency plans are necessary to direct employees to act in ways that maximize their personal protection and minimize the hazards in the event of an emergency. Employees not engaged in correcting the emergency situations must be prohibited from the area and normal operations halted until the emergency is abated.

Protective Work Clothing and Equipment: Paragraph (i)

The standard requires that the employer provide protective clothing to employees who are exposed to cadmium at levels above the TWA PEL and to employees exposed at any level when skin or eye irritation occurs. For workers exposed above the EL, respirators are required, which must be maintained according to equipment handling provisions of this paragraph. Examples of such personal protective clothing are, but are not limited to, coveralls, shoe covers, head coverings, and goggles. Clean protective clothing and equipment shall be provided at least weekly to each affected employee.

The standard further requires that the employer be responsible for cleaning, laundering and disposing of the required protective clothing and equipment, to eliminate any potential exposure that might result were the clothing and equipment to be laundered or cleaned by the employee at home. Furthermore, the standard provides that the employer shall assure that workers change out of all protective clothing and equipment at the end of each work shift and that the clothing and equipment that is to be laundered, cleaned, or disposed of be placed in a closed container. The standard also requires that protective clothing be maintained and replaced as needed in order to ensure effectiveness.

Protective clothing and foot coverings are required to prevent contamination of the employee's street clothing and shoes. This will prevent cadmium exposure beyond the workplace. Wearing contaminated clothing outside the work area would lengthen the duration of exposure. In addition, cadmium could accumulate in employee's cars and homes exposing other individuals to the hazard.

The proposal provides that the employer shall ensure that all protective clothing is removed at the end of each work shift only in change rooms. Removal of cadmium from protective clothing by blowing, shaking, or any other means which disperses cadmium in the air is prohibited. Furthermore, the standard emphasizes the need to assure that contaminated clothing be stored in closed containers prior to laundering or disposal so that contamination of the change room is minimized and that employees who later handle the clothing are protected. The latter group are further protected by the requirement that they be informed of the potentially harmful effects of cadmium exposure and that warning labels be placed on the containers. Since these containers are to be located in the change room, it is appropriate to limit workers' removal of contaminated clothing to that area.

The proposed standard obligates the employer to provide personal protective clothing at no cost to the employee. Since the employer is responsible for reducing exposures below the permissible exposure limit, the obligation to provide personal protective equipment properly rests on the employer. The employer also is in the best position to provide the correct type of clothing and keep it in repair.

Hygiene Facilities and Practices: Paragraph (j)

The proposed standard requires employers to provide hygiene facilities and to assure employee compliance with basic hygiene practices that minimize additional sources of exposure to cadmium which may accumulate on a worker's clothes or body. The employer must provide adequate shower and washing facilities, clean rooms for changing clothes, and positive-pressure filtered-air lunchrooms for employees who are exposed above the TWA PEL. In addition, employers must assure that employees use the facilities as required by the standard as well as observe prohibitions on the use of cosmetics,

tobacco and chewing products, and food and beverages in regulated areas. OSHA expects that strict compliance with these provisions will virtually eliminate several sources of cadmium exposure that substantially contribute to overall exposure levels.

Several of these facilities and practices are presently required under current OSHA standards for General Environmental Controls in subpart J of 29 CFR part 1910. For example, § 1910.141(e) states that if a standard requires the employees to wear protective clothing, then the employer must provide change rooms with separate storage facilities for street and work clothing, and section 1910.141(g) requires the employer to prohibit the consumption of food and beverages in areas where there is exposure to toxic substances. The hygiene provisions of this paragraph are to augment the requirement of 29 CFR 1910.141 with additional requirements that are specifically applicable to cadmium exposure and to consolidate all related provisions under one standard.

OSHA believes it is essential for employees to have separate locker and storage facilities for street and work clothing to prevent cross-examination of their street clothes. This provision will minimize employee exposure to cadmium after the work shift ends, because it reduces the period in which they may be exposed to cadmium-contaminated work clothes.

Showering also reduces the worker's period of exposure to cadmium by removing cadmium which may accumulate on the skin and hair. Requiring employees to change out of work clothes and to shower before leaving the plant and to leave work clothing at the workplace significantly reduces the movement of cadmium from the workplace. These steps ensure that the duration of cadmium exposure does not extend beyond the workshift and provide added protection to employees and their families.

The proposed standard also requires employers to provide employees working in regulated areas with readily accessible positive-pressure filtered-air lunchrooms. Employers must also assure that employees wash their hands and face prior to eating or smoking and that employees not enter the lunchroom wearing protective clothing unless it is cleaned beforehand. Employers are given discretion to choose any method for removing surface cadmium that does not disperse the dust into the air.

To minimize the possibility of food contamination and to reduce the likelihood of additional exposure to loose cadmium dust through inhalation or ingestion, OSHA feels it is imperative that employees have a clean place to eat, free from cadmium exposure. Positive-pressure filtered-air lunchrooms will reduce employee exposure by limiting contamination from cadmium.

Housekeeping: Paragraph (k)

The proposed standard imposes the general housekeeping requirement to maintain all surfaces as free as is practicable of accumulations of cadmium. The standard bans the use of compressed air for cleaning and allows dry cleaning, that is shoveling, dry or wet sweeping, and brushing only if the employer shows that vacuuming or other methods that are usually as efficient as vacuuming are not effective under the current circumstances. It also requires that vacuuming be done with cleaners equipped with HEPA filters to prevent the dispersal of cadmium into the workplace. In addition, items contaminated with cadmium and consigned for disposal are to be collected and disposed of in sealed impermeable bags or other closed impermeable containers. These are exceptionally important provisions because they minimize additional sources of exposure that engineering controls generally are not designed to control.

Medical Surveillance: Paragraph (l)

Paragraph (l)(1)(i), the proposal requires each employer to institute a medical surveillance program for all employees who are or will be exposed at or above the action level or above the EL. Providing medical surveillance for employees exposed at or above the action level is consistent with other health standards that incorporate an action level and is considered by OSHA to be necessary and appropriate for monitoring the adequacy of the exposure limit to protect individual employees.

The proposal requires that the medical surveillance program provide each covered employee with an opportunity for a medical examination. Paragraph (l)(1)(ii) provides that all examinations and procedures be performed by or under the supervision of a qualified physician and be provided without cost to the employee. Clearly, a qualified physician is the appropriate person to be supervising and evaluating a medical examination. However, certain parts of the required examination do not necessarily require the physician's expertise and may be conducted by another person under the supervision of the physician.

This standard provides that all examinations and procedures shall be performed at a reasonable time and place. It is necessary that exams be convenient and be provided during the workday without loss of pay to the employee to assure that they are taken. The employer is required to establish and maintain an accurate record for each employee subject to medical surveillance.

The purpose of the initial medical examination is to: (1) Establish the current health status of the employee and to determine whether employment in areas with cadmium exposure is appropriate; (2) establish essential baseline data against which to measure any change which might be attributable to cadmium exposure; and (3) determine whether the individual can safely wear a respirator. OSHA believes that the preplacement examination assessing each worker's state of health prior to the beginning of exposure to cadmium is essential to determine whether an employee's health changes over the period of employment and to determine pre-existing conditions that could influence initial job placement.

The preplacement examination is to include a medical and work history oriented toward cadmium exposure, a complete physical examination of all systems, with emphasis on the respiratory system, cardiovascular system, hematopoietic system, musculoskeletal system, and genitourinary system; a chest X-ray (posterior-anterior 14 x 17 inches or reasonably standard size); pulmonary function tests including forced vital capacity (FVC) and forced expiratory volume at 1 second (FEV_1), conducted according to the American Thoracic Society Criteria (Ex. 8–663); blood analysis, including cadmium levels, blood urea nitrogen, complete blood count, and serum creatinine; urinalysis, including cadmium and creatinine levels, specific gravity, albumin, glucose and total and low molecular weight proteins; and any additional tests deemed appropriate by the examining physician.

This information, in conjunction with a complete physical examination of all systems, will assist the physician in the determination of the employee's health status, possible past exposures to cadmium or other substances that may have damaged organs or systems susceptible to cadmium toxicity, and suitability for employment in an area where exposure to cadmium will occur. Special emphasis is placed on the portions of the history and physical examination which evaluate organ systems known to be particularly susceptible to cadmium toxicity. Emphasis is placed on the respiratory

system because of the increased risk of lung cancer and fibrotic changes with cadmium exposure and because of the necessity to evaluate an employee's fitness for respirator usage. The cardiovascular system is emphasized because of the increased level of hypertension which has been related to cadmium exposure. An evaluation of the hematopoietic system will disclose anemia associated with cadmium exposure. Emphasis on the musculoskeletal system is included because osteomalacia, a condition caused by loss of calcium from the bone(s) through damaged kidneys, has been related to cadmium toxicity.

Tests used to provide further information on the respiratory system include a 14 x 17 inch or reasonably standard sized posterior-anterior chest x-ray and pulmonary function tests (including forced vital capacity (FVC) and forced expiratory volume at 1 second (FEV_1), conducted according to the American Thoracic Society criteria). The complete blood count is important in evaluating the hematopoietic system. Evaluation of the status of the genitourinary system is accomplished using results of the blood urea nitrogen, serum creatinine, urinalysis, and prostate palpation. Urinary cadmium is sometimes an indicator of levels of cadmium exposure, whereas low molecular weight protein levels, such as Beta-2 (β_2) microglobulin, are determined to assess the extent of cadmium accumulation and damage in the kidney. Urinary creatinine is determined to assist in the standardization of the urine cadmium level. The other urinalysis components (specific gravity, albumin, glucose, total protein, and microscopic examination of centrifuged sediment) are indicators of the status of renal function. The serum cadmium level is determined as an indication of cadmium exposure. Liver enzymes may be useful in the evaluation of function of the liver, an organ where cadmium can concentrate. OSHA requests comments on all aspects of this medical surveillance protocol. Specifically, should liver function tests be performed? Is cadmium in urine a meaningful indicator of increased risk of adverse health effects among workers occupationally exposed to cadmium? What standardized urine collection procedures are needed to assure results that are meaningful? Should end-of-shift, spot, or 24-hour samples be collected? Should the pH of urine be determined for β_2 microglobulin tests? Should all cadmium-in-urine results be standardized to micrograms of cadmium (μg Cd) per gram creatinine?

Also included in the initial or preplacement examination are any additional tests deemed appropriate by the examining physician. This provision authorizes the physician to include further tests which could assist the physician in determining the employee's suitability for work in an area in which cadmium exposure will occur or in determining whether a worker can safely wear a respirator.

OSHA proposes periodic medical examinations to be administered yearly. The purposes of the annual examination are: (1) The detection of excessive exposure to cadmium before the occurrence of significant biological effects; (2) the early detection of biological effects of cadmium; (3) the detection of non-occupationally-related diseases that might require reduction of cadmium exposure; (4) the prevention, if possible, of an employee exceeding a critical level of cadmium in the kidney, which could result in permanent kidney damage, or at least the minimization of further kidney damage; (5) fitness for respirator usage; and (6) monitor general health status and recent illnesses. Since the effects of cadmium are long term and cumulative, periodic examinations are required at one year intervals. More frequent reviews of specific biological tests may be necessary or may be required by OSHA if evidence indicates such tests are needed, particularly for those individuals whose past exposure was high for those with longer-term exposure. OSHA seeks comment on whether this provision should be tailored to differences in an employee's overall exposure, relating to factors like intermittency, frequency, duration, and level.

The employer shall undertake a reassessment of an employee's occupational exposure and work practices if examinations or biological monitoring results reveal the occurrence of any of the physical conditions set forth in paragraph (l)(4), of this standard. The employer then shall promptly take appropriate steps to correct the problem and to reduce the employee's exposures. In an effort to detect early an irreversible disease associated with cadmium exposure, the employer shall undertake such reassessment as soon as individual concentrations of cadmium in urine (CdU) and/or cadmium in blood (CdB) exceed 5 μg Cd/g creatinine or 5 μg Cd/liter of whole blood.

This requirement is in accordance with the recommendation made by a WHO study group in 1980 (Ex. 8-674), which is stricter than the ACGIH recommendation. The ACGIH (Ex. 8-667) recommends biological exposure indices (BEIs) of 10 μg/g creatinine in urine and 10 μg/liter blood. The ACGIH CdU BEI equates to an estimated critical concentration of cadmium in the renal cortex of 180-220 μg/g kidney wet weight. At that concentration, ACGIH projects that 10 percent of the population will develop renal dysfunction. Although the ACGIH correctly states that this would protect the majority of workers, OSHA believes that such a high level of risk is unacceptable, particularly in view of the fact that cadmium-induced renal dysfunction is irreversible.

Thus, prevention is imperative. Prevention can be more effectively accomplished by requiring employers to reassess occupational exposure and work practices of affected employees when Cd-U and/or Cd-B exceed 5 μg Cd/g urinary creatinine or 5 μg Cd/liter of whole blood and to take timely corrective actions. Such actions include reevaluation of hygiene facilities, respirator programs, maintenance of engineering control equipment, work practices, personal hygiene, and, if necessary, medical removal. After appropriate steps are taken to improve the work environment, hygiene, and work practices of the individual, the biological monitoring should then be repeated no more than three months after the initial monitoring to determine whether those steps had the desired effect of of reducing the employee's cadmium exposure. If not, further steps should be taken. OSHA seeks comment on all aspects of this provision.

A reassessment of an employee's occupational cadmium exposure is also required if there are repeated diagnoses of respiratory illness or infection. OSHA requires that such reassessment also take place if the FVC or FEV_1 is less than 80 percent of the expected value or the ratio of FEV_1/FVC times 100 is less than 75 percent of the expected value (Ex. No. 8-663). Further, OSHA requires such reassessment where there is persistent proteinuria or other abnormal laboratory results or clinical findings consistent with cadmium toxicity. OSHA requests comments on all aspects of this requirement. OSHA is particularly interested in whether or not pulmonary function tests should be evaluated in terms of changes from baseline test results.

OSHA also proposes periodic medical re-evaluation of workers required to wear respirators. The re-evaluation is necessary because an illness or a new medication may affect the employee's cardiovascular system. The impact on an employee's continuing ability to wear

a respirator then must be assessed. The re-evaluation will enable the physician to determine whether the individual can safely continue to wear the same type of respirator, should be re-fitted with another type, or should be removed from any area where respirator use is required.

Additional medical examinations are required for workers who have high levels of total and low weight proteins in the urine or who have signs of respiratory system abnormalities identified through the medical examination and for whom another medical reason for such findings has not been found. In addition, examinations are required for those who experience difficulty breathing during use of or fit testing for respirators. These additional tests are required to ensure that the appropriate evaluations are performed to diagnose and assist in the treatment of any present disease and prevent further disease.

Because of the potentially serious nature of the diseases identified by these tests and because it is known that a portion of workers will be unable to wear respirators, provisions are included in paragraph (l)(4) of this standard for additional tests to further evaluate an employee's health, to confirm the test results and to determine if medical removal is necessary. Some employers currently remove workers from cadmium exposure based on results of biological monitoring (Ex. 14-6). Because of employee reluctance to voluntarily participate in a program of medical surveillance if there is a possibility of loss of pay or other benefits, provisions are included in this standard to reduce the likelihood of such loss by providing medical removal protection.

Biological tests for both CdB and CdU levels may provide useful information about an employee's health. However, there are no generally agreed upon CdB and CdU levels that would apply to both new employees and veteran employees to indicate the presence or absence of disease. Workers who have high levels of cadmium in the blood (CdB) or urine (CdU) have probably had past exposures to cadmium that were high and/or long-term. These employees be at an increased risk of experiencing toxic effects of cadmium on the body. For new workers, cadmium concentrations in blood may be a useful indicator of exposure during recent months. The level of cadmium in urine is most likely correlated with cadmium body burden in workers without renal damage. In workers with long term, low-level exposure, an elevated urinary cadmium excretion may indicate that the cadmium concentration in the renal cortex is near the critical concentration above which permanent kidney damage occurs.

OSHA requests comments on the usefulness of measurements of cadmium in biological fluids as a screening method for the presence of disease in new and veteran employees.

The employer is also required to make any additional tests available if recommended by the examining physician. The employer is further required to make a medical examination available at the termination of employment to any employee who has been eligible for a medical examination in the past and to provide a medical examination as soon as possible to all employees who have been exposed to cadmium in an emergency, as stipulated in paragraphs (l)(5) and (l)(6), respectively, of this standard.

OSHA has not included a multiple physician review mechanism in this proposal. However, OSHA believes that such a provision might be necessary and appropriate. Multiple physician review is made available in the lead standard [29 CFR 1910.1025(j)(3)(iii)]. OSHA requests comments on whether such a mechanism is necessary and appropriate in this standard.

A medical examination at the termination of employment is required for all workers who have, in the past, been eligible for an annual medical examination under this proposed standard. This requirement is in keeping with other standards (Asbestos, Coke Oven Emissions, Arsenic, Acrylonitrile, and Ethylene Oxide). The need for this requirement in this standard is due, in part, to the way cadmium is transported, distributed, and stored in the body. After absorption, cadmium is transported via the blood stream to other body parts, where it is bound to proteins and stored. Low excretion rates lead to a very efficient retention of cadmium in the body. The bioloical half-life of cadmium is 20-37 years or more.

There is growing evidence that even after cessation of exposure to cadmium in the workplace, cadmium stored in one body compartment can be transported to the kidney. In this way, cadmium proteinuria may develop years after exposure in the workplace has ceased, provided that the exposure was substantial. There is, therefore, no tendency for proteinuria to decrease after removal from external cadmium exposure. Instead there can be an increase, which is substantial for some workers, and kidney damage can progress to a more severe stage of disease (Ex. 8-668). Consequently, it is important that the employee's health status regarding cadmium accumulation in the body be assessed at the employee's termination of employment.

Failure to find evidence of impairment at termination should not be viewed as a "clean bill of health." Physicians should use the opportunity of that examination to advise the employee of his/her cadmium body burden and prognosis, and to make recommendations for medical management and follow-up. For the worker, this information allows him/her to determine the courses of action necessary to sustain health. OSHA seeks comment on the appropriateness of requiring a chest x-ray in termination of employment exams.

Complete medical records at termination of employment are useful to physicians to determine the status of an employee's health and to assist in identifying health effects. Good medical records, including an examination at termination of employment, also would be useful in enumerating illnesses and deaths attributable to cadmium, for evaluating compliance programs, and for assessing the accuracy of the Agency's risk estimates. Such records are useful to assess the adequacy of the standard in preventing diseases. Provisions for collection of such information, including medical examinations at the end of employment, have been including in other standards mentioned previously.

OSHA further requests comment on the usefulness of requiring the reporting of abnormal biological monitoring test results on OSHA's Form 200, for reporting occupational illnesses to the Bureau of Labor Statistics. For example, should OSHA require that: CdB levels of 10 μg/l whole blood, or greater; CdU levels of 10 μg/gr creatinine, or greater; excess urinary proteins; cases of metal fume fever; and, abnormal pulmonary function test results be reported on that form?

The employer is required, in paragraph (l)(9), to provide the physician with the following information: A copy of this standard and its appendices; a description of the affected employee's former and current duties as they relate to the employee's cadmium exposure level; the employee's former and current exposure level or anticipated exposure level; a description of any personal protective and respiratory equipment used or to be used; and information or medical records from the employee's previous medical examinations that were provided or made available by the employer to the affected employee.

Making this information available to the physician will aid in the evaluation of the employee's health in relation to assigned duties and fitness to wear personal protective equipment, when required.

The employer is required to obtain a written opinion from the examining physician containing the results of the medical examination as they relate to occupational exposures; the physician's opinion as to whether the employee has any detected medical conditions which would place the employee at increased risk of material health impairment from exposure to cadmium; any recommended restrictions upon the employee's exposure to cadmium or upon the use of protective clothing or equipment such as respirators; and a statement that the employee has been informed by the physician of the results of the medical examination and of any medical conditions which require further evaluation or treatment. This written opinion must not reveal specific findings or diagnoses unrelated to occupational exposures. The employer must provide a copy of the opinion to the affected employee.

The purpose in requiring the examining physician to supply the employer with a written opinion is to provide the employer with a medical basis to aid in the determination of initial placement of employees and to assess the employee's ability to use protective clothing and equipment. The requirement that a physician's opinion be in written form will ensure that employers have had the benefit of this information. The employer shall provide a copy of the physician's written opinion to the affected employee within 30 days of its receipt. The requirement that an employee be provided with a copy of the physician's written opinion will ensure that the employee is informed of the results of the medical examination. The requirement that the physician sign the opinion is to ensure that the information that is given to the employer has been seen and read by the physician.

The purpose in requiring that specific findings or diagnoses unrelated to occupational exposures not be included in the written opinion is to encourage employees to take the medical examination by removing any concern that the employer will obtain adverse information about their physical condition that has no relation to occupational exposures. This provision has been included in prior standards.

Communication of Hazards to Employees: Paragraph (m)

In this proposed cadmium standard, OSHA includes a paragraph entitled: "Communication of Cadmium Hazards to Employees". This paragraph incorporates some requirements from OSHA's Hazard Communication Standard (HCS) and addresses the issue of transmitting information to employees about the hazards of cadmium through the use of: (1) signs, (2) labels, (3) material safety data sheets, and (4) information and training. Previous OSHA health standards generally included separate paragraphs on employee information and training and on signs and labels. This standard incorporates both of those areas, along with provisions on material safety data sheets, into paragraph (m), to be consistent with the (HCS).

OSHA's HCS [(29 C 1910.1200) for general industry and (29 CFR 1926.59) for the construction industry] requires all chemical manufacturers and importers to assess the hazards of the chemicals they produce or import and requires all employers to provide information concerning the hazards of such chemicals to their employees. The transmittal of hazard information to employees is to be accomplished by means of comprehensive hazard communication programs, which are to include container labeling and other forms of warning, material safety data sheets and employee training.

Since the HCS "is intended to address comprehensively the issue of evaluating the potential hazard of chemicals and communicating information concerning hazards and appropriate protective measures to employees" (52 FR 31877), OSHA proposes paragraph (m) entitled "Communication of Cadmium Hazards to Employees" to avoid repetition of those requirements now comprehensively laid out in §§ 1910.1200 and 1926.59. In paragraph (m), OSHA also proposes additional particular requirements that are needed to protect employees specifically exposed to cadmium. Paragraph (m) of this proposed cadmium standard has been designed to be substantively as consistent as possible with the HCS requirements for employers. While avoiding a duplicative administrative burden on employers attempting to comply with the requirements of several different applicable OSHA health standards, the proposed requirements nevertheless provide the necessary protection for employees through provisions for signs and labels, material safety data sheets, and employee information and training.

The proposed standard requires that regulated areas be posted with signs stating: "Danger, Cadmium, Cancer Hazard, Can Cause Lung and Kidney Damage, Authorized Personnel Only, Respirators and Protective Clothing Required in this Area". The proposed standard intends that the posting of these signs will serve as a warning to employees who may otherwise not know they are entering a regulated area. Such warning signs are required to be posted at all regulated areas, that is, whenever the permissible exposure limit is exceeded. For some work sites, regulated areas are permanent, because exposures there cannot be reduced below the TWA PEL or EL by the use of engineering controls. In those situations, the signs are needed to warn employees not to enter the area unless they are authorized, wearing respirators, and there is a need for entering the area.

Regulated areas may also exist on a temporary basis such as during maintenance and/or emergency situations. The use of warning signs in these types of situations is also important since a maintenance or emergency situation is by nature a new or unexpected exposure to employees who are regularly scheduled to work at these sites.

These signs are intended to supplement the training which employees are to receive under the other provisions of this paragraph, since even trained employees need to be reminded of the locations of regulated areas and of the precautions necessary to be taken before entering these dangerous areas.

The proposed standard requires that the signs must comply with paragraph (f) of the HCS and specifies the wording of the warning signs for regulated areas in order to ensure that the proper warning is given to employees. OSHA believes that the use of the word "Danger" is appropriate, based on the evidence of the toxicity and carcinogenicity of cadmium. "Danger" is used to attract the attention of workers in order to alert them to the fact they are in an area where either of the permissible exposure limits is exceeded and to emphasize the importance of the message that follows. The use of the word "Danger" is also consistent with other recent OSHA health standards dealing with carcinogens. The proposed standard also requires that the legend, "Respirators and Protective Clothing Required in this Area", be included on the warning sign. While OSHA recognizes that some employees entering the regulated areas may not be exposed above either the TWA PEL or the EL as averaged over a 15-minute period, it is still possible that many employees are assigned to work in these areas may remain in these locations for long enough periods of time so that they would be needlessly overexposed to

cadmium without the use of respirators and protective clothing. To ensure that these employees are adequately protected, it is necessary that the sign alert them to the need to wear respirators and protective clothing.

The proposal also requires that warning labels be affixed to all shipping and storage containers containing cadmium or cadmium compounds or such cadmium-contaminated items as clothing and equipment. The labels must be in compliance with paragraph (f) of the HCS and must state: "Danger, Contains Cadmium, Cancer Hazard, Avoid Creating Dust, Can Cause Lung and Kidney Damage". It is proposed that required labels would remain affixed to containers leaving the workplace. The purpose of this requirement is to ensure that all affected employees, not only those of a particular employer, are apprised to the hazardous nature of cadmium exposure where exposure could exceed the action level.

In addition to being consistent with the requirements of the HCS, these requirements carry out the mandate of section 6(b)(7) of the Act which requires OSHA health standards to prescribe the use of labels or other appropriate forms of warning to apprise employees of the hazards to which they are exposed.

In this proposed cadmium standard, OSHA also would require that the employer to obtain or develop and to distribute and provide access to a material safety data sheet (MSDS) for cadmium in accordance with the requirements of 29 CFR 1910.1200(g) and 29 CFR 1926.59. OSHA feels that a properly completed MSDS, if readily available to employees, can serve as an excellent, concise source of information regarding the hazards associated with cadmium. OSHA's primary intent in this section of the proposed standard, as stated in its recently promulgated HCS, is to ensure that employees will receive as much information as they need concerning the hazards posed by chemicals in their workplaces. The MSDS ensures that this information will be available to them in a usable, readily accessible and concise form. The MSDS also serves as the central source of information to employees and to downstream employers who must be provided with an MSDS if cadmium or a product containing cadmium is produced and shipped to them. Lastly, the MSDS serves as the basic source of information on the hazards of cadmium essential to the training provisions of this proposed standard.

Producers and importers of toxic substances have primary responsibility, under the HCS to develop or prepare the MSDS. The manufacturer or importer is most likely to have the best access to information about the product and is therefore responsible for disseminating this information to downstream users of the material. For employers whose employees' exposure to cadmium is from products received from outside sources, the information necessary for a complete MSDS or the MSDS itself is to be obtained from the manufacturer and made available to affected employees. The requirements for the information that is to be contained on the MSDS are explained in detail at 29 CFR 1910.1200(g) and 29 CFR 1926.59.

Paragraph (m)(4) of this proposed cadmium standard requires employers to provide all employees who are exposed to cadmium with information and training on cadmium at the time of initial assignment and at least annually thereafter. A record shall be maintained of the contents of such programs. The training program is to be in accordance with the requirements of the HCS paragraphs (h) (1) and (2), and to include the specific information required to be provided by that section and those items stipulated in paragraph (m)(4)(iii) of this standard. Employees are to be provided with an explanation of the contents of Appendices A (Substance Safety Data Sheet, Cadmium) and B (Substance Technical Guidelines, Cadmium) of the final cadmium standard. Employees also are to be informed where a copy of the final cadmium standard is accessible to them and to receive an explanation of the purpose and a description of the medical surveillance program required under paragraph (l) of this proposed standard.

OSHA has determined during other rulemakings that an information and training program, as incorporated in this proposed standard in an overall "Communication of Cadmium Hazards to Employees" paragraph, is essential to inform employees of the hazards to which they are exposed and to provide employees with the necessary understanding of the degree to which they themselves can minimize the health hazard potential. As part of an overall communication program for employees, training serves to explain and reinforce the information presented to employees on labels and material safety data sheets. These written forms of information and warning will be successful and relevant only when employees understand the information presented and are aware of the actions to be taken to avoid or minimize exposures thereby reducing the possibility of experiencing adverse health effects. Training is essential to an effective overall hazard communication program. Active employee participation in training sessions can result in the effective communication of hazard information to employees which can further result in workers taking conscientious protective actions at their job duties, thereby decreasing the possibility of occupationally-related illnesses and injuries.

OSHA proposes the training provisions of this standard to be in performance-oriented rather than specified and detailed language. The proposed standard, in requiring training to be in accordance with the requirements of 29 CFR 1910.1200 and 29 CFR 1926.59, lists the categories of information to be transmitted to employees and not the specific ways that this is to be accomplished. The use of such performance-oriented requirements will encourage employers to tailor their training needs to their specific workplaces, thereby resulting in the most effective training program suitable for each specific workplace.

OSHA believes that the employer is in the best position to determine how the training he or she is providing is being received and absorbed by the employees. OSHA has therefore laid out the objectives to be met and the intent of its training to ensure that employees are made aware of the hazards in their workplace and how they can help to protect themselves. The specifics of how this is to be accomplished are left up to the employer.

Recordkeeping: Paragraph (n)

The proposed cadmium standard requires employers to maintain exposure monitoring records and medical surveillance records. These requirements are proposed in accordance with section 8(c) of the Act which requires employers to keep and make available such records as the Secretary may prescribe as necessary or appropriate for the enforcement of the Act or for developing information regarding occupational injuries and illnesses, and with the regulation governing access to employee exposure and medical records (29 CFR 1910.20).

The proposal requires that records be kept of environmental monitoring results that identify the monitored employee and to accurately reflect the employee's exposure. Specifically, records must include the following information: (a) The date of monitoring, duration, and results of each of the samples taken; (b) a description of the job classification of the employee being monitored; (c) a description of the sampling and analytical methods used and evidence of their accuracy; (d) the type of respiratory protective devices, if any,

worn by the employee; (e) the names, social security numbers, and job classification of the employees monitored and of all other employees whose exposure the monitoring is intended to represent; and (f) a notation of any other conditions that might have been affected by the monitoring results.

The proposal requires that exposure measurement records be maintained for each measurement taken. The record may represent the exposure of more than one employee if representative sampling, as described in paragraph (d), is conducted.

A provision for the use of objective data in place of initial monitoring is included in this standard. Objective data are information demonstrating that a particular product or material containing cadmium cannot release dust or fumes in concentrations at or above the action level or above the EL even under the worst-case release conditions. Employers can use data from an industry-wide survey, when such a survey includes similar workplace conditions, operations, and job classifications as those of the employer, in order to estimate maximum cadmium exposure levels that could occur. Additionally, employers can use laboratory product test results to demonstrate that airborne concentrations must be below the action level.

In addition to records on employee exposure measurements, the employer is required to establish and maintain an accurate medical surveillance record for each employee subject to medical surveillance as required by paragraph (l) of this proposed standard. OSHA believes that medical records, like exposure monitoring records, are necessary and appropriate both to the enforcement of the standard and to the development of information regarding the causes and prevention of occupational illnesses. Furthermore, medical records are necessary for the proper evaluation of the employee's health.

The proposed standard requires that exposure records be kept for at least 30 years and that medical records be kept for duration of employment plus thirty years. It is necessary to keep these records for extended periods because of the long latency period commonly associated with carcinogenesis. Cancer often cannot be detected until 20 or more years after first exposure. The extended record retention period is therefore needed because diagnosis of disease in employees is assisted by, and in some cases can only be made by, having present and past exposure data as well as the results of present and past medical examinations. The employer shall maintain records of employee training for one year beyond the last date of employment of any trained employee. OSHA seeks comment on whether or not individual training records should be maintained for each employee.

The proposal specifies that access to exposure and medical records by employees, designated representatives, and OSHA shall be in accordance with 29 CFR 1910.20, OSHA's "Access to Employee Exposure and Medical Records" standard. That standard applies to records required by specific standards, such as this proposed cadmium standard, as well as records which are voluntarily created by an employer. Employees and their designated representatives are, in general, allowed unrestricted access to all relevant exposure monitoring records. Access to one's own medical records is also provided for employees (i.e., an employee may have access only to his or her own medical records) and, if the employee has given specific written consent, for the employee's designated representatives. OSHA retains access to both kinds of records, but its access to personally identifiable records is subject to agency rules of practice and procedure which have been published at 29 CFR 1913.10 (see 45 FR 35384).

The transfer of employee exposure monitoring and medical records is to be in accordance with the provisions of paragraph (h) of 29 CFR 1910.20. If an employer ceases to do business and there is no successor employer, the employer is to notify NIOSH and transmit the records to the Director of NIOSH for retention, if requested.

OSHA seeks comment on these specific recordkeeping provisions. Requirements for recordkeeping under the Paperwork Reduction Act are discussed under section XI—Clearance of Information Collection Requirements.

Observation of Monitoring: Paragraph (o)

This proposed cadmium standard contains provisions for the observation of exposure monitoring. This provision is in accordance with section 8(c) of the OSH Act which requires that employers provide employees and their representatives with the opportunity to observe monitoring of employee exposures to toxic substances or harmful physical agents. Observation procedures are set forth which require the observer, whether it be an employee or a designated representative, to be provided with the personal protective clothing and equipment that is required to be worn by the employees who are working in the area. The employer is required to ensure the use of such clothing and equipment or respirators, and is responsible for requiring that the observer complies with all other applicable safety and health procedures.

Dates: Paragraph (p)

It is proposed that the standard become effective 60 days after the date of publication in the **Federal Register**. OSHA proposes that the requirements for exposure monitoring and employee information and training be completed 90 days after the effective date of the final rule (150 days after publication in the **Federal Register**). The requirements for respiratory protection and medical surveillance are proposed to be completed 90 days after the effective date except for use of powered air-purifying respirators provided under section XIV paragraph (g)(2)(ii) which are to be provided within one (1) year from publication in the **Federal Register**. In addition, establishment of regulated areas is proposed to be completed not later than 90 days after the effective date. Written compliance programs required by paragraph (f)(2) of this standard as a result of monitoring shall be completed and available for inspection and copying no later than one (1) year after the effective date of the standard. Implementation dates for the completion of the engineering and work practice requirements are proposed to be no later than 2 years after the effective date of the final rule. This is to allow affected employers sufficient time to design (where necessary), obtain, and install the necessary control equipment. Planning and construction of hygiene and lunchroom facilities is proposed to be completed as set forth in section XIV paragraph (p)(2)(vi). OSHA solicits comments on the adequacy of these proposed start-up dates.

Appendices: Paragraph (q)

The proposed standard contains 5 appendices which are designed to assist employers and employees in implementing the provisions of this standard. Appendix C is incorporated as part of this standard and imposes additional mandatory obligations on employers covered by this standard. Appendices A, B, D, and E are nonmandatory and are included primarily to provide information and guidance. In addition, these appendices are not intended to detract from any obligation that the proposed standard imposes.

The Appendices that are included in the standard are:

Appendix A—Substance Safety Data Sheet, Cadmium
Appendix B—Substance Technical Guidelines, Cadmium
Appendix C—Qualitative and Quantitative Fit Testing Procedures
Appendix D—Medical and Occupational History with Reference to Cadmium Exposure (suggested format)
Appendix E—Sampling and Analysis

XI. 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 for collection of information requirements contained in proposed rules to OMB not later than the date of publication of the proposal in the **Federal Register**. It also requires agencies to include a statement in the notice of proposed rulemaking indicating that such information requirements have been submitted for review to OMB 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.
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> * * * * *
>
> 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.
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> * * * * *
>
> 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 sections of the proposed cadmium standard which may create recordkeeping requirements are paragraphs (d) exposure monitoring, (f)(2) compliance program, (l) medical surveillance, (m) communication of cadmium hazards, and (n) recordkeeping, among others.

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 rule on occupational exposure to cadmium to OMB for review under section 3504(h) of that Act.

Public reporting burden for this collection of information is estimated to vary from 5 minutes (recordkeeping) to 8 hours (compliance program) per response, with an average of 0.38 hours per response for a TWA PEL of 1 $\mu g/m^3$ or an average of 0.32 hours per response for a TWA PEL of 5 $\mu g/m^3$, including time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. Send comments regarding the burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden, to the Office of Information Management, Department of Labor, Room N-1301, 200 Constitution Avenue, NW., Washington, DC, 20210; and to the Office of Information and Regulatory Affairs, Office of Management and Budget, Washington, DC, 20503.

XII. Public Participation—Notice of Hearing

Pursuant to section 6(b)(3) of the Act, an opportunity to submit oral testimony concerning the issues raised by the proposed standard will be provided at an informal public hearing scheduled to begin at 9:30 a.m. at places and on dates as follows:

Washington, DC: June 5, 1990.
The Auditorium, Frances Perkins Department of Labor Building, 200 Constitution Ave., NW, Washington, DC, 20210.
Denver, Colorado: July 17, 1990.
Cripple Creek/Silver Heels Room, Holiday Inn, 1450 Glen Arm Place, Denver, Colorado, 80202.

Notice of Intention to Appear

All persons desiring to participate at the hearings must file in quadruplicate a Notice of Intention to Appear, postmarked on or before April 4, 1990, addressed to Mr. Tom Hall, OSHA Division of Consumer Affairs, Docket No. H-057a, Room N-3647, U.S. Department of Labor, Third Street and Constitution Ave., NW., Washington, DC 20210; telephone 202-523-6024. The Notice of Intention to Appear also may be transmitted by facsimile to (202) 523-5046 or (for FTS) to 8-523-5046, provided the original and 4 copies of the notice are sent to the above address thereafter.

Notices of intention to appear, which will be available for inspection and copying at the OSHA Docket Office (Room N-2625), telephone 202-523-7894, 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 requested for the presentation;

(4) The specific issues that will be addressed;

(5) A statement of the position that will be taken with respect to each issue addressed;

(6) Whether the party intends to submit documentary evidence, and if so, a brief summary of that evidence; and

(7) At which hearing or hearings the party wishes to testify.

Filing of Testimony and Evidence Before Hearings

Any party requesting more than 10 minutes for a presentation at the hearing or submitting documentary evidence must provide in quadruplicate the complete text of the testimony including any documentary evidence to be presented at the hearing to the OSHA Division of Consumer Affairs. This material must be postmarked by April 27, 1990, and will be available for inspection and copying at the OSHA Docket Office. Each such submission will be reviewed in light of the amount of time requested in the Notice of Intention to Appear. In those 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.

Any party who has not substantially complied with this requirement may be limited to a 10 minute presentation. Any party who has not filed a notice of intention to appear may be allowed to testify, as time permits, at the discretion of the Administrative Law Judge.

OSHA emphasizes that the hearing is open to the public, and that interested persons are welcome to attend. However, only persons who have filed proper Notices of Intention to Appear at the hearing will be entitled to ask questions and otherwise participate fully in the proceeding.

Conduct and Nature of Hearings

The hearings will commence at 9:30

a.m. on June 5, 1990. At that time, any procedural matters relating to the proceeding will be resolved.

The informal nature of the rulemaking hearings to be held is established in the legislative history of section 6 of the Act and is reflected by the OSHA hearing regulations (see 29 CFR 1911.15 (a)). Although the presiding officer is an Administrative Law Judge and questioning by interested persons is allowed on crucial issues, it is clear that the proceeding shall remain informal and legislative in type. The intent, in essence, is to provide an opportunity for effective oral presentation by interested persons which can be carried out expeditiously and in the absence of rigid procedures which might unduly impede or protract the rulemaking process.

The hearing will be conducted in accordance with 29 CFR part 1911. The hearings will be presided over by an Administrative Law Judge who will have all the powers necessary or appropriate to conduct a full and fair informal hearing as provided in 29 CFR part 1911, including the powers:

1. To regulate the course of the proceedings;
2. To dispose of procedural requests, objections, and comparable matters;
3. To confine the presentations to matters pertinent to issues raised;
4. To regulate the conduct of those present at the hearing by appropriate means;
5. In the Judge's discretion, to question and permit the questioning of any witness and to limit the time for questioning;
6. In the Judge's discretion, to keep the record open for a reasonable, stated time to receive written information and additional data, views and arguments from any person who has participated in the oral proceedings.

Written Comments

Interested persons are invited to submit written comments on the issues raised in this hearing notice. Written comments must be postmarked by April 27, 1990, and submitted in quadruplicate to the Docket Office, Docket number H-057a, Room N-2625, U.S. Department of Labor, 200 Constitution Ave. NW., Washington, DC 20210. The telephone number of the Docket Office is (202) 523-7894, and its hours of operation are 8:15 a.m. to 4:45 p.m. Monday through Friday except Federal holidays. Comments limited to 10 pages or less in length may also be transmitted by facsimile to (202) 523-5046 or (for FTS) to 8-523-5046 provided the original and 4 copies of the comment are sent to the Docket Officer thereafter. Written submissions must clearly identify the issues raised in this notice which are addressed and the position taken on each issue.

All materials submitted will be available for inspection and copying at this address. All timely submissions will be part of the record of the proceeding.

Certification of Record and Final Determination After Hearing

Following the close of the hearings or of any posthearing comment period, the presiding Administrative Law Judge will certify the record to the Assistant Secretary of Labor for Occupational Safety and Health. The Administrative Law Judge does not make or recommend any decisions as to the content of a final standard. The proposed standard will be reviewed in light of all oral and written submissions received as part of the record, and a standard will be issued based on the entire record of the proceeding, including the written comments and data received from the public.

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

Cadmium, Occupational safety and health, Chemicals, Cancer, Health, Risk-assessment.

XIII. Authority and Signature

This document was prepared under the direction of Gerard F. Scannell, Assistant Secretary of Labor for Occupational Safety and Health, U.S. Department of Labor, 200 Constitution Ave. NW., Washington, DC 20210.

It is issued under section 6(b) of the Occupational Safety and Health Act of 1970 (29 U.S.C. 655), Secretary of Labor's Order No. 9-83 (48 FR 35736) and 29 CFR part 1911.

Signed at Washington, DC, this 25th day of January 1990.

Gerard F. Scannell,

Assistant Secretary of Labor.

XIV. The Proposed Standard

General Industry

PART 1910—[AMENDED]

Part 1910 of title 29 of the Code of Federal Regulations is hereby proposed to be amended as follows:

Subpart B—[Amended]

1. The authority citation for subpart B of 29 CFR part 1910 continues to read as follows:

Authority: Secs. 4, 6 and 8 of the Occupational Safety and Health Act, 29 U.S.C. 653, 655, 657; Walsh-Healey Act, 41 U.S.C. 35 et seq; Service Contract Act of 1965, 41 U.S.C. 351 et seq; sec. 107, Contract Work Hours and Safety Standards Acts (Construction Safety Act), 40 U.S.C. 333; sec. 41, Longshoremen's and Harbor Workers' Compensation Act, 33 U.S.C. 941; National Foundation of Arts and Humanities Act, 20 U.S.C. 951 et seq.; Secretary of Labor's Order No. 12-71 (36 FR 8754); 8-76 (41 FR 25059), or 9-83 (48 FR 35736), as applicable; and 29 CFR part 1911.

Sections 1910.16 and 1910.19 also issued under 29 CFR part 1911.

2. A new paragraph (k) is proposed to be added to § 1910.19 to read as follows:

§ 1910.19 Special provisions for air contaminants.

* * * * *

(k) *Cadmium.* Section 1910.1027 shall apply to the exposure of every employee to cadmium in every employment and place of employment covered by §§ 1910.12, 1910.13, 1910.14, 1910.15, 1910.16, 1926, and 1928 in lieu of any different standard on exposures to cadmium which would otherwise be applicable by virtue of those sections.

Subpart Z—[Amended]

3. The authority citation for subpart 2 of part 1910 continues 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 part 1911.

Section 1910.1000 Tables 2-1, 2-2, 2-3 also issued under 5 U.S.C. 553.

Section 1910.1000 not issued under 29 CFR Part 1911, except for "Arsenic" and "Cotton Dust" listings in Table 1-1.

Section 1910.1001 also issued under Sec. 107 of Contract Work Hours and Safety Standards Act, 40 U.S.C. 333.

Section 1910.1002 not issued under 29 U.S.C. 655 or 29 CFR part 1911; also issued under 5 U.S.C. 553.

Sections 1910.1003 through 1910.1018 also issued under 29 U.S.C. 653.

Section 1910.1025 also issued under 29 U.S.C. 653 and 5 U.S.C. 553.

Section 1910.1028 also issued under 29 U.S.C. 653.

Section 1910.1043 also issued under 5 U.S.C. 551 et seq.

Sections 1910.1045 and 1910.1047 also issued under 29 U.S.C. 653.

Section 1910.1048 also issued under 29 U.S.C. 653.

Sections 1910.1200, 1910.1499 and 1910.1500 also issued under 5 U.S.C. 553.

§ 1910.1000 [Amended]

4. The entries "Cadmium fume (Z37.5–1970) * * * 0.1mg/m³ * * * 0.3 mg/m³" and "Cadmium dust (Z37.5–1970) * * * 0.2 mg/m³ * * * 0.6 mg/m³" are proposed to be deleted from Table 2–2 of § 1910.1000.

5. A new § 1910.1027 and Appendices A,B,C,D, and E to the section are proposed to be added to subpart Z to read as follows:

§ 1910.1027 Cadmium.

(a) *Scope and application.* This standard applies to all occupational exposures to cadmium in all industries covered by the Occupational Safety and Health Act, including construction, agriculture and maritime.

(b) Definitions

Action level is defined as an airborne concentration of cadmium of 2.5 micrograms per cubic meter of air (2.5 $\mu g/m^3$), calculated as an 8-hour time-weighted average permissible exposure limit (TWA PEL), if the TWA PEL is set at 5 $\mu g/m^3$, or alternatively of 0.5 $\mu g/m^3$.

Assistant Secretary means the Assistant Secretary of Labor for Occupational Safety and Health, U.S. Department of Labor, or designee.

Authorized person means any person authorized by the employer and required by work duties to be present in regulated areas.

Director means the Director of the National Institute for Occupational Safety and Health (NIOSH), U.S. Department of Health and Human Services, or designee.

Emergency means any occurrence such as, but not limited to, equipment failure, rupture of containers, or failure of control equipment that results in an unexpected and significant release of cadmium.

Employee exposure means the exposure to airborne cadmium that would occur if the employee were not using respiratory protective equipment.

High-efficiency particulate absolute [HEPA] air filter means a filter capable of trapping and retaining at least 99.97 percent of 0.3 micrometer-diameter, mono-disperse particles.

Regulated area means an area demarcated by the employer where airborne concentrations of cadmium exceed, or can reasonably be expected to exceed a permissible exposure limit expressed either as an 8-hour time-weighted average (TWA PEL) or as an excursion limit (EL).

(c) *Permissible exposure limits.—(1) Eight-Hour, Time-Weighted Average Permissible Exposure Limit (TWA PEL).* The employer shall assure that no employee is exposed to an airborne concentration of cadmium in excess of [five micrograms per cubic meter of air (5 $\mu g/m^3$), calculated as an eight-hour time-weighted average exposure (TWA PEL) or one microgram per cubic meter (1 $\mu g/m^3$, TWA PEL)].

(2) *Excursion Limit (EL).* The employer shall assure that no employee is exposed to an airborne concentration of cadmium in excess of [twenty-five micrograms per cubic meter of air (25 $\mu g/m^3$), as averaged over a sampling period of fifteen (15) minutes for a TWA PEL of 5 $\mu g/m^3$ or five $\mu g/m^3$ (5 $\mu g/m^3$), as averaged over a 15 minute sampling period for a TWA PEL of 1 $\mu g/m^3$].

(d) *Exposure monitoring.*—(1) *General.* (i) Each employer who has a workplace or work operation covered by this standard shall perform monitoring in accordance with paragraph (d)(2) of this section to determine accurately the airborne concentration of cadmium to which employees may be exposed.

(ii) Determinations of employee exposure shall be made from breathing zone air samples that are representative of the 8-hour TWA exposure of each employee and/or of a 15 minute period exposure of each employee at operations where there is reason to believe exposures are above the EL.

(iii) Representative 8-hour TWA exposures shall be determined for each employee on the basis of one or more personal breathing zone air samples representing full shift exposure from each shift for each job classification in each work area. Where several employees perform the same job function in the same job category on the same shift in the same work area in which the length, duration, and level of cadmium exposures are similar, an employer may sample a fraction of the employees instead of all employees in order to meet this requirement. In representative sampling, the employees sampled shall be those expected to have the highest cadmium exposures.

(2) *Initial monitoring.* (i) Except as provided for in paragraphs (d)(2)(ii) and (d)(2)(iii) of this section, each employer who has a workplace or work operation covered by this standard shall perform initial monitoring of employees who, without regard for respirator usage, are, or may reasonably be expected to be, exposed to airborne concentrations at or above the action level or above the EL.

(ii) Where the employer has monitored after [insert date 180 days prior to publication in the **Federal Register**], under conditions closely resembling those currently prevailing and where that monitoring satisfies all other requirements of this standard, the employer may rely on such earlier monitoring results to satisfy the requirements of paragraph (d)(2)(i) of this section.

(iii) Where the employer has objective data, as defined in paragraph (n)(2) of this section, demonstrating that cadmium exposures will not exceed airborne concentrations at or above the action level or above the EL under the expected conditions of processing, use, or handling, the employer may rely upon such data instead of implementing initial monitoring.

(3) *Monitoring frequency (periodic monitoring).* (i) If the initial or periodic monitoring reveals employee exposures to be above the TWA PEL, the employer shall monitor at least every three months for each employee exposed above the TWA PEL. The employer shall continue these quarterly measurements until at least two consecutive measurements, taken at least seven days apart, are at or below the permissible exposure limit.

(ii) If the initial or periodic monitoring reveals employee exposures to be at or above the action level but at or below the TWA PEL, the employer shall repeat such monitoring for employees so exposed at least every six months. The employer shall continue these semi-annual measurements until at least two consecutive measurements, taken at least seven days apart, are below the action level.

(iii) If either the initial or the periodic monitoring indicates that employee exposures are below the action level and that result is confirmed by the results of another monitoring taken at least seven days later, the employer may discontinue the monitoring for those employees whose exposures are represented by such monitoring.

(iv) Except as stated below in this paragraph, exposure monitoring to determine whether the EL has been exceeded shall be carried out at the same time as, and according to the schedules for monitoring under paragraphs (d)(3)(i)–(d)(3)(iii) of this section. Thus, if the initial or periodic monitoring carried out under these paragraphs reveals employee exposures to be below the action level, then, with

the exception stated below, no further monitoring for the EL is required. If such initial or periodic monitoring reveals employee exposures to be at or above the action level, the employer shall monitor those employees who are expected to have the highest exposure levels to determine whether they are exposed above the EL. Specifically, if the initial or periodic monitoring carried out under paragraphs (d)(3)(i)–(d)(3)(iii) of this section reveals employee exposures to be at or above the action level but at or below the TWA PEL, the employer shall monitor the appropriate employees at least every six months to determine whether any are exposed in excess of the EL. Or, if such initial or periodic monitoring reveals employee exposures to be above the TWA PEL, the employer shall monitor the appropriate employees at least every three months to determine whether they are exposed in excess of the EL. If the initial or periodic monitoring reveals employee exposure to be above the TWA PEL or at or above the action level, but two consecutive measurements taken at least seven days apart reveal the employee exposure is at or below the EL, no further monitoring for the EL is required. However, even if the initial or periodic monitoring carried out under paragraphs (d)(3)(i)–(d)(3)(iii) of this section reveals employee exposures to be below the action level, if it is determined that an employee is or may reasonably be expected to be exposed above the EL, that employee shall be monitored at least every six months for his/her excursion exposure until two consecutive measurements, taken at least seven days apart, are at or below the EL.

(4) *Additional monitoring.* The employer also shall institute the exposure monitoring required under paragraphs (d)(2)(i) and (d)(3) of this section whenever there has been a change in production, process, raw material, control equipment, personnel or work practices that may result in new or additional exposures at or above the action level or above the EL, or whenever the employer has any other reason to suspect that a change may result in new or additional exposures at or above the action level or above the EL.

(5) *Employee notification of monitoring results.* (i) Within 15 working days after the receipt of the results of any monitoring performed under this standard, the employer shall notify each affected employee individually in writing of the results. In addition, within the same period the employer shall post the results of the exposure monitoring in an appropriate location that is accessible to all affected employees.

(ii) Wherever monitoring results indicate that employee exposure exceeds the TWA PEL or the EL, the employer shall include in the written notice a statement that the TWA PEL and/or EL has been exceeded and a description of the corrective action being taken by the employer to reduce employee exposure to or below the TWA PEL and/or EL.

(6) *Accuracy of measurement.* The employer shall use a method of monitoring and analysis that has an accuracy (to a confidence level of 95%) of not less than plus or minus 25 percent (± 25%) for airborne concentrations of cadmium at or above the level being investigated.

(e) *Regulated Areas*—(1) *Establishment.* The employer shall establish regulated areas wherever airborne concentrations of cadmium are, or can reasonably be expected to be, in excess of the permissible exposure limits (TWA PEL or EL) prescribed in paragraph (c) of this section.

(2) *Demarcation.* Regulated areas shall be demarcated from the rest of the workplace in any manner that adequately establishes and alerts employees of the boundaries of the regulated area.

(3) *Access.* Access to regulated areas shall be limited to authorized persons or to persons authorized by the OSH Act or regulations issued pursuant thereto.

(4) *Provision of respirators.* Each person entering a regulated area shall be supplied with and required to use a respirator, selected in accordance with paragraph (g)(2) of this section.

(5) *Prohibited activities.* The employer shall assure that employees do not eat, drink, smoke, chew tobacco or gum, or apply cosmetics in regulated areas.

(f) *Methods of Compliance*—(1) *Engineering and work practice controls.* (i) When any employee is exposed to cadmium above either the TWA PEL or EL prescribed in paragraph (c) of this section, the employer shall implement engineering and work practice controls to the extent feasible to reduce and maintain employee exposure at or below the TWA PEL and/or EL.

(ii) Wherever engineering and work practice controls are not sufficient to reduce employee exposure to or below the TWA PEL and/or EL prescribed in paragraph (c) of this section, the employer nonetheless shall implement such controls to reduce exposures to the lowest levels achievable and shall supplement such controls with respiratory protection that complies with the requirements of paragraph (g) of this section.

(iii) The employer shall not use employee rotation as a means of compliance with the TWA PEL and/or EL.

(2) *Compliance program.* (i) Where the TWA PEL and/or EL is exceeded, the employer shall establish and implement a written compliance program to reduce employee exposure to or below the TWA PEL and/or EL by means of engineering and work practice controls, as required by paragraph (f)(1) of this section. To the extent that engineering and work practice controls cannot reduce exposures to or below the TWA PEL and/or EL, the employer shall include in the written compliance program the use of appropriate respiratory protection to achieve compliance with the TWA PEL and/or EL. The compliance program shall include a written plan for emergency situations, as specified in paragraph (h) of this section.

(ii) The written compliance programs shall be reviewed and updated at least annually, or more often if necessary, to reflect significant changes in the employer's compliance status.

(iii) Written compliance programs shall be submitted upon request for examination and copying to the Assistant Secretary, the Director, affected employees, and authorized employee representatives.

(g) *Respiratory Protection*—(1) *General.* Where respirators are required by this standard, the employer shall provide them at no cost to the employee and, except for situations covered by paragraph (g)(1)(vi) of this section, shall assure that they are used in compliance with this standard. Respirators shall be used in the following circumstances:

(i) When exposures exceed the TWA PEL and/or the EL, during the time period necessary to install or implement feasible engineering and work practice controls;

(ii) When exposures exceed the TWA PEL and/or the EL and when engineering and work practice controls are not feasible, in maintenance and repair activities and during brief or intermittent operations;

(iii) In regulated areas as prescribed in paragraph (e) of this section;

(iv) In work situations where the employer has implemented all feasible engineering and work practice controls and such controls are not sufficient to reduce exposures to or below the permissible exposure limits;

(v) In emergencies; and

(vi) Whenever an employee who is exposed above the action level requests a respirator.

(2) *Respirator selection.* (i) Where respirators are required under this standard, the employer shall select and provide the appropriate respirator as specified in Table 1. The employer shall select respirators from among those jointly approved as acceptable protection against cadmium dust, fume, and mist by the Mine Safety and Health Administration (MSHA) and by the National Institute for Occupational Safety and Health (NIOSH) under the provisions of 30 CFR part 11.

(ii) The employer shall provide a powered, air-purifying respirator (PAPR) in lieu of any negative pressure respirator specified in Table 1 whenever:

(A) An employee chooses to use this type of respirator; and

(B) This respirator will provide adequate protection to the employee.

TABLE 1.—RESPIRATORY PROTECTION FOR CADMIUM

Airborne concentration or condition of use [a]	Respirator Type [b]
10 × or less	A half mask, air-purifying respirator equipped with a HEPA [c] filter [d].
25 × or less	A powered air-purifying respirator with a loose-fitting hood or helmet equipped with a HEPA filter, or A supplied-air respirator with a loose-fitting hood or helmet facepiece operated in the continuous flow mode.
50 × or less	A full facepiece air-purifying respirator equipped with a HEPA filter, or A powered air-purifying respirator with a tight-fitting half mask equipped with a HEPA filter, or A supplied-air respirator with a tight-fitting half mask operated in the continuous flow mode.
250 × or less	A powered air-purifying respirator with a tight-fitting full facepiece equipped with a HEPA filter, or A supplied-air respirator with a tight-fitting full facepiece operated in the continuous flow mode.
1000 × or less	A supplied-air respirator with half mask or full facepiece operated in the pressure demand or other positive pressure mode.
Greater than 1000 × or unknown concentrations	A self-contained breathing apparatus with a full facepiece operated in the pressure demand or other positive pressure mode, or A supplied-air respirator with a full facepiece operated in the pressure demand or other positive pressure mode and equipped with an auxiliary escape type self contained breathing apparatus operated in the pressure demand mode.
Fire fighting	A self-contained breathing apparatus with full facepiece operated in the pressure demand or other positive pressure mode.

[a] Concentrations expressed as multiple of the 8-hour TWA PEL.

[b] Respirators assigned for higher environmental concentrations may be used at lower exposure levels. Quantitative fit testing is required for all tight-fitting air purifying respirators where airborne concentration of cadmium exceeds 10 times the TWA PEL (10 $\mu g/m^3$ or 50 $\mu g/m^3$). Full facepiece is required when eye irritation is experienced.

[c] HEPA means High Efficiency Particulate Absolute.

[d] Qualitative or quantitative fit testing is required.

Source: *Respiratory Decision Logic,* NIOSH, 1987.

(3) *Respirator program.* (i) Where respiratory protection is required, the employer shall institute a respirator protection program in accordance with 29 CFR 1910.134.

(ii) The employer shall permit each employee who is required to use an air purifying respirator to leave the regulated area to change the filter elements or replace the respirator whenever an increase in breathing resistance is detected and shall maintain an adequate supply of filter elements for this purpose.

(iii) Employees who are required to wear respirators shall be permitted to leave the regulated area to wash their faces and respirator facepieces whenever necessary to prevent skin irritation associated with respirator use.

(iv) If an employee exhibits difficulty in breathing while wearing a respirator during a fit test or during use, the employer shall make available to the employee a medical examination in accordance with paragraph (l) of this section to determine if the employee can wear a respirator while performing the required duties.

(v) No employee shall be assigned tasks requiring the use of respirators if, based upon his or her most recent examination, an examining physician determines that the employee will be unable to function normally while wearing a respirator. As prescribed in paragraphs (l)(11) and (l)(12) of this section, such employee shall be given the opportunity to transfer to a position where no respirator use is required. That position shall be with the same employer, in the same geographical area, and with the same seniority status and rate of pay the employee had just prior to such transfer, if such a position is available.

(4) *Respirator fit testing.* (i) The employer shall assure that the respirator issued to the employee exhibits the least possible facepiece leakage and that the respirator is fitted properly.

(ii) For each employee wearing a tight-fitting, air purifying respirator (either negative or positive pressure) who is exposed to airborne concentrations of cadmium that do not exceed 10 times the TWA PEL (10 $\mu g/m^3$ or 50 $\mu g/m^3$), the employer shall perform either quantitative or qualitative fit testing at the time of initial fitting and at least annually thereafter. If quantitative fit testing is used for a negative pressure respirator, a fit factor that is at least 10 times the protection factor for that class of respirators (Table 1) shall be achieved at testing.

(iii) For each employee wearing a tight-fitting air purifying respirator (either negative or positive pressure) who is exposed to airborne concentrations of cadmium that exceed 10 times the TWA PEL (10 $\mu g/m^3$ or 50 $\mu g/m^3$), the employer shall perform quantitative fit testing at the time of initial fitting and at least annually thereafter. For negative-pressure respirators, a fit factor that is at least 10 times the protection factor for that class of respirators (Table 1) shall be achieved during quantitative fit testing.

(iv) Fit testing shall be conducted in accordance with Appendix C of this section.

(h) *Emergency situations.* The employer shall develop a written plan for dealing with emergency situations involving substantial releases of airborne cadmium. The plan shall include provisions for the use of appropriate respirators and personal protective equipment. In addition, employees not essential to correcting the emergency situation shall be restricted from the area and normal operations halted in that area until the emergency is abated.

(i) *Protective work clothing and equipment—(1) Provision and use.* If an employee is exposed to cadmium above the TWA PEL, measured without regard to respirator usage, or if the possibility of skin or eye irritation exists from cadmium exposures at any exposure levels, the employer shall provide at no cost to the employee, and assure that the employee uses, appropriate protective work clothing and equipment such as, but not limited to:

(i) Coveralls or similar full-body work clothing;

(ii) Gloves, head coverings, and foot coverings; and

(iii) Face shields, vented goggles, or other appropriate protective equipment which complies with 29 CFR 1910.133.

(2) *Provision for EL.* If an employee is exposed above the EL, the employer shall provide at no cost to the employee, and assure that the employee uses, appropriate respiratory equipment.

(3) *Removal and storage.* (i) The employer shall assure that employees remove all protective clothing and equipment contaminated with cadmium at the completion of the work shift and do so only in change rooms provided in accordance with paragraph (j)(1) of this section.

(ii) The employer shall assure that no employee takes cadmium contaminated protective clothing or equipment out of the workplace except for those employees authorized to do so, for the purposes of laundering, cleaning, maintenance, or disposal at an appropriate location or facility.

(iii) The employer shall assure that contaminated protective clothing and contaminated equipment when removed for laundering, cleaning, maintenance, or disposal, is placed and stored in closed containers. These storage facilities shall be designed to prevent dispersion of cadmium dust.

(iv) The employer shall assure that containers of contaminated protective clothing and equipment that are to be taken out of the change rooms or the workplace for laundering, cleaning, maintenance or disposal, shall bear labels in accordance with paragraph (m)(2) of this section.

(4) *Cleaning, replacement, and disposal.* (i) The employer shall provide clean protective clothing and equipment at least weekly to each affected employee. The employer shall clean, launder, repair, and replace protective clothing and equipment required by this paragraph to maintain their effectiveness and shall be responsible for the disposal of such clothing and equipment.

(ii) The employer shall prohibit the removal of cadmium from protective clothing and equipment by blowing, shaking, or any other means that disperses cadmium into the air.

(iii) The employer shall assure that any laundering of contaminated clothing or cleaning of contaminated equipment in the workplace is done in a manner so as to prevent the release of airborne cadmium in excess of the permissible exposure limits prescribed in paragraph (c) of this section.

(iv) The employer shall inform any person who launders or cleans protective clothing or equipment contaminated with cadmium of the potentially harmful effects of exposure to cadmium.

(v) Any employer who gives contaminated clothing to another person for laundering or contaminated equipment to another person for cleaning shall inform such person that the clothing and equipment should be laundered or cleaned in a manner to effectively prevent the release of airborne cadmium in excess of the permissible exposure limits.

(vi) Contaminated clothing and equipment shall be transported in sealed impermeable bags, or other closed, impermeable containers, and labeled in accordance with paragraph (m)(2) of this section.

(j) *Hygiene facilities and practices.*—(1) *Change rooms.* (i) The employer shall provide clean change rooms for employees who work in areas where their airborne exposure to cadmium, without regard to the use of respirators, is above the TWA PEL.

(ii) The employer shall assure that change rooms are equipped with separate storage facilities for street clothes and for protective clothing and equipment that are designed to prevent dispersion of cadmium and prevent contamination of the employee's street clothes.

(2) *Showers and handwashing facilities.* (i) The employer shall assure that employees who work in areas where their airborne exposure, without regard to the use of respirators, is above the TWA PEL have the opportunity to shower during the end of the work shift.

(ii) The employer shall provide handwashing and shower facilities that comply with § 1910.141 (d)(1), (d)(2) and (d)(3) for workers exposed without regard to the use of respirators, above the TWA PEL.

(iii) The employer shall provide handwashing facilities for workers exposed without regard to the use of respirator above the EL.

(iv) The employer shall assure that employees who work in regulated areas where their airborne cadmium exposure is above the TWA PEL and/or EL, without regard to the use of respirators, wash their hands and faces prior to eating, drinking, smoking, chewing tobacco or gum, or applying cosmetics.

(3) *Lunchrooms.* (i) The employer shall provide lunchroom facilities for employees who work in areas where their airborne exposure, without regard to the use of respirators, is above the TWA PEL.

(ii) The employer shall assure that lunchroom facilities have a positive-pressure, tempered, filtered air supply, and are readily accessible to employees.

(iii) The employer shall assure that employees do not enter lunchroom facilities with protective work clothing or equipment unless surface cadmium has been removed from the clothing or equipment by vacuuming or some other method that removes dust without causing cadmium to become airborne.

(k) *Housekeeping.*—(1) All surfaces shall be maintained as free as practicable of accumulations of cadmium.

(2) All spills and sudden releases of material containing cadmium shall be cleaned up as soon as possible.

(3) Surfaces contaminated with cadmium may not be cleaned by the use of compressed air.

(4) HEPA-filtered vacuuming equipment shall be used for vacuuming. The equipment shall be used and emptied in a manner that minimizes the reentry of cadmium into the workplace.

(5) Shoveling, dry or wet sweeping, and brushing may be used only where vacuuming or other methods that minimize the likelihood of cadmium becoming airborne have been tried and found not to be effective.

(6) Waste, scrap, debris, bags, containers, equipment, and clothing contaminated with cadmium and consigned for disposal shall be collected and disposed of in sealed impermeable bags or other closed, impermeable containers. These bags and containers shall be labeled in accordance with paragraph (m)(2)(i) of this section.

(l) *Medical surveillance.*—(1) *General.* (i) The employer shall institute a medical surveillance program for all employees who are or will be exposed to airborne concentrations of cadmium at or above the action level or above the EL.

(ii) The employer shall assure that all medical examinations and procedures are performed by or under the supervision of a qualified physician and are provided without cost to the employee and at a reasonable time and place.

(2) *Initial examinations.* (i) The employer shall provide an initial or

preplacement examination to all employees who are or will be exposed to airborne concentrations of cadmium at or above the action level or above the EL. The examination shall be provided to the employees within 30 days after initial assignment to an area with exposures at or above the action level or no later than 90 days after the standard goes into effect, whichever date comes last.

(ii) The initial or preplacement medical examination shall include at a minimum:

(A) A detailed medical and work history, with emphasis on: past exposure to cadmium, smoking history, and any history of renal, cardiovascular, respiratory, hematopoietic, musculoskeletal and/or neurological system dysfunction;

(B) A complete physical examination of all systems with emphasis on: the respiratory, cardiovascular, hematopoietic, musculoskeletal and genitourinary systems;

(C) A 14" x 17" or reasonably standard sized posterior-anterior chest X-ray;

(D) Pulmonary function tests to include forced vital capacity (FVC) and forced expiratory volume at 1 second (FEV_1);

(E) Blood analysis including cadmium levels, blood urea nitrogen, complete blood count, serum creatinine, and liver enzymes;

(F) Urinalysis including cadmium and creatinine levels, specific gravity, albumin, glucose, a microscopic examination of centrifuged sediment, and a determination of total protein and low molecular weight proteins;

(G) Prostate palpation in males over 40 years of age; and

(H) Any additional tests deemed appropriate by the examining physician.

(3) *Periodic examinations.* (i) The employer shall provide periodic medical examinations at least annually to each employee exposed to airborne concentrations of cadmium at or above the action level or above the EL.

(ii) The content of the periodic medical examinations shall conform to the requirements in paragraph (l)(2)(ii) of this section except that the frequency of chest X-rays shall be determined by the examining physician.

(4) *Additional examinations and referrals.* (i) The employer shall reassess an employee's occupational exposures and work practices and shall promptly take appropriate steps to reduce an employee's exposures whenever a physician determines in a written opinion that:

(A) the concentration of cadmium in the employee's urine (CdU) exceeds 5 μg per gram creatinine;

(B) the concentration of cadmium in the employee's blood (CdB) exceeds 5 μg per liter whole blood;

(C) relative to the initial or preplacement exam and after adjusting for the age and smoking habits of the person examined, FVC or FEV_1 is $<80\%$ of predicted values, or the ratio FEV_1/FVC times 100 is $<75\%$ of predicted values;

(D) there are repeated diagnoses of respiratory tract disease;

(E) there are repeated diagnoses of upper or lower respiratory infections; or

(F) persistent proteinuria or other abnormal laboratory or clinical findings consistent with cadmium toxicity develop. (See Appendix A of this section).

The appropriate steps to reduce an employee's exposures include review of hygiene facilities, reevaluation of respiratory programs, reevaluation of the maintenance of engineering control equipment, reassessment of an employee's work practices and personal hygiene, and, if necessary, medical removal in accordance with paragraph (l)(11) of this section.

(ii) Whenever, in accordance with paragraph (l)(4)(i) of this section, the employer has reassessed an employee's occupational exposure and work practices due to the employee's CdU level having exceeded 5 $\mu g/g$ creatinine or the employee's CdB level having exceeded 5 μg/liter of whole blood, and the employer has taken appropriate corrective measures to reduce the employee's exposure to cadmium, the employer shall provide to the employee the relevant blood, urine or other tests within three months after the corrective action has been taken. If the results of any of these tests continue to be outside the limits specified in paragraph (l)(4)(i) of this section, the employer shall provide the relevant test on a quarterly basis until the results are within the stated limits.

(iii) Where the results of tests for total or low molecular weight proteins in urine are abnormal, the examining physician shall evaluate in more detail the toxic effects of cadmium on the renal system.

(iv) Where the results of the examination of the respiratory system indicate that the FVC or the FEV_1 is less than 80% of predicted values, or the ratio of FEV_1/FVC times 100 is less than 75% of the predicted values, or the employee experiences difficulty breathing during the use of or fit testing for respirators, restriction from respirator use shall be considered and the physician will further evaluate the employee's ability to wear a respirator.

(5) *Examination at termination of employment.* (i) At the time of termination of employment, the employer shall provide a medical examination to any employee who, at any time previously, has been eligible for an annual medical examination under paragraph (l)(3) of this section. However, if the last examination was less than six months prior to the date of termination, no further examination is required unless otherwise specified.

(ii) The medical examination at termination of employment shall be in accordance with the requirements of the periodic examinations stipulated in paragraph (l)(3) of this section.

(6) *Examinations for employees exposed in an emergency.* (i) In addition to the medical surveillance required in paragraphs (l)(1)–(l)(5) of this section, the employer shall provide medical examinations as soon as possible to all employees who may have been acutely exposed to cadmium because of an emergency.

(ii) The examination shall include a work history, medical history, and a physical examination with emphasis on the respiratory system and other organ systems considered appropriate by the examining physician. (See Appendix A at II(B)(1) for symptoms of acute overexposure.)

(7) *Examination for respirator usage.* To determine an employee's fitness for respirator use, the employer shall provide the medical examination specified in paragraph (l)(2) and (l)(3) of this section to any employee assigned to a job that requires the use of a respirator. The medical examination shall be provided within 30 days of assignment to such job or no later than 90 days after the standard goes into effect, whichever is later, to any employee without a previous periodic medical evaluation within the preceeding 12 months. The previous medical evaluation must satisfy the requirements of paragraph (l)(3) of this section.

(8) *Recent examinations.* A medical examination is not required to be provided in accordance with paragraphs (l)(2) and (l)(3) of this section if adequate records show that the employee has been examined in accordance with the requirements of these paragraphs within the past one year period. However, in that case such records shall be maintained as part of the employee's medical record and the next specified medical examination shall be made available to the employee

within one year of the previous examination.

(9) *Information provided to the physician.* The employer shall provide the following information to the examining physician:

(i) A copy of this standard and appendices.

(ii) A description of the affected employee's former, current and anticipated future duties as they relate to the employee's cadmium exposure.

(iii) The employee's former and current occupational exposure levels or, for employees not yet occupationally exposed to cadmium, the employee's anticipated exposure levels.

(iv) A description of any personal protective and respiratory equipment used or to be used.

(v) Information from previous medical examinations that were provided or made available by the employer to the affected employee.

(10) *Physician's written opinion.* (i) The employer shall obtain a written, signed opinion from each examining physician in reference to each medical examination performed for each employee. This written opinion shall contain the results of the medical examination as they relate to occupational exposures to cadmium and shall include:

(A) The physician's opinion as to whether the employee has any detected medical condition(s) that would place the employee at increased risk of material impairment to health from further exposure to cadmium, including any indications of potential cadmium toxicity as identified in paragraph (l)(4)(i)(A)–(l)(4)(i)(F) of this section;

(B) The results of any testing or related evaluation concerning cadmium exposure carried out as part of the examination;

(C) Any recommended removal from, or limitation on the activities or duties of the employee or upon the employee's use of personal protective equipment such as clothing or respirators; and

(D) A statement that the employee has been informed by the physician of the results of the medical examination and of any medical conditions resulting from cadmium exposure that require further evaluation or treatment.

(ii) The employer shall instruct the physician not to reveal orally or in the written opinion given to the employer specific findings or diagnoses unrelated to occupational exposures.

(iii) The employer shall provide a copy of the physician's written opinion to the affected employee within 30 days of its receipt.

(11) *Medical removal plan.* (i) The employer shall remove an employee from work where exposure to cadmium is at or above the action level on each occasion that a physician determines in a written opinion that the employee should be removed from such exposure due to abnormal levels of urinary proteins or illnesses, abnormal test results, or other signs or symptoms of cadmium-related dysfunction.

(ii) For any employee removed under paragraph (l)(11)(i) of this section, the employer shall provide follow-up medical examinations every three months until a decision is made by the examining physician that the employee may be returned to his/her normal job, or a decision is made that the employee must be permanently removed from cadmium exposures above the action level.

(iii) The employer shall remove an employee from work having an exposure to cadmium above the TWA PEL or EL whenever a physician determines in a written opinion that the employee cannot wear a respirator.

(iv) For any employee removed under paragraph (l)(11)(iii) of this section the employer shall provide a follow-up medical examination within three months to determine if the removal must be permanent.

(12) *Medical removal protection benefits.* (i) Whenever an employee is removed under paragraphs (l)(11)(i) or (l)(11)(iii) of the section, the employer shall transfer the employee to a comparable job that meets the exposure limits imposed in those paragraphs on the employee's exposure to cadmium as soon as one becomes available.

(ii) The employer shall provide full medical removal protection benefits for a maximum of 6 months each time an employee is removed under paragraphs (l)(11)(i) or (l)(11)(iii) of this section.

(iii) The requirement in paragraph (l)(12)(ii) of this section that the employer provide full medical removal protection benefits means that the employer shall maintain the total normal earnings, seniority and other employment rights and benefits of an employee as though the employee had not been removed from the employee's normal exposure to cadmium.

(13) *Recordkeeping.* The employer shall establish and maintain medical records as specified in paragraph (n)(3) of this section.

(m) *Communication of cadmium hazards to employees*—(1) *Warning signs.* (i) Warning signs shall be provided and displayed in regulated areas. In addition, warning signs shall be posted at all approaches to regulated areas so that an employee may read the signs and take necessary protective steps before entering the area.

(ii) The warning signs required by paragraph (m)(1)(i) of this section shall comply with requirements of the Hazard Communication Standard 29 CFR 1910.1200(f) (general industry) and 29 CFR 1926.59(f) and bear the following information:

DANGER
CADMIUM
CANCER HAZARD
CAN CAUSE LUNG AND KIDNEY DAMAGE
AUTHORIZED PERSONNEL ONLY
RESPIRATORS REQUIRED IN THIS AREA

(2) *Warning labels.* (i) Shipping and storage containers containing cadmium, cadmium compounds, or cadmium contaminated clothing, equipment, waste, scrap, or debris shall bear appropriate warning labels, as specified in paragraph (m)(2)(ii) of this section.

(ii) The labels shall comply with the requirements of the Hazard Communication Standard 29 CFR 1910.1200(f) (general industry) and 29 CFR 1926.59 (construction industry), and shall include the following information:

DANGER
CONTAINS CADMIUM
CANCER HAZARD
AVOID CREATING DUST
CAN CAUSE LUNG AND KIDNEY DAMAGE

(3) *Material safety data sheets.* Employers who are manufacturers or importers of cadmium or cadmium compounds shall comply with the requirements regarding development and distribution of material safety data sheets as specified in 29 CFR 1910.1200(g) of OSHA's Hazard Communication Standard. All employers with employees potentially exposed to cadmium compounds shall maintain material safety data sheets and provide their employees with access to them, in accordance with the requirements of 29 CFR 1910.1200(g) and 29 CFR 1926.59(g).

(4) *Employee information and training.* Employers shall provide employees with information and training in accordance with the requirements of the Hazard Communication Standard, 29 CFR 1910.1200(h) (general industry), and 29 CFR 1926.59(h) (construction industry). In addition:

(i) The employer shall institute a training program for all employees who are potentially exposed to cadmium, assure employee participation in the program, and maintain a record of the contents of such program.

(ii) Training shall be provided prior to or at the time of initial assignment to a job involving potential exposure to cadmium and at least annually thereafter.

(iii) The employer shall make the training program understandable to the

employee. The employer shall assure that each employee is informed of the following:

(A) The health hazards associated with cadmium exposure with special attention to the information in Appendix A of this section;

(B) The quantity, location, manner of use, release, and storage of cadmium and the specific nature of operations that could result in exposure to cadmium, especially exposures above the TWA PEL or EL;

(C) The engineering controls and work practices associated with the employee's job assignment;

(D) The measures employees can take to protect themselves from exposure to cadmium, including modification of their habits such as smoking and personal hygiene, and specific procedures the employer has implemented to protect employees from exposure to cadmium such as appropriate work practices, emergency procedures, and personal protective equipment;

(E) The details of the hazard communication program developed by the employer, including an explanation of the signs, labeling system and material safety data sheets, and how employees can obtain and use the appropriate hazard information;

(F) The purpose, proper selection, fitting, proper use, and limitations of respirators and protective clothing;

(G) The purpose and a description of the medical surveillance program required by paragraph (l) of this section;

(H) The contents of this standard and its appendices, and

(I) The right of any employee exposed to cadmium at or above the action level or above the EL to obtain:

(*1*) Medical examinations as required by paragraph (l) of this section at no cost to the employee;

(*2*) The employee's medical records required to be maintained by paragraph (n)(3) of this section; and

(*3*) All air monitoring results representing the employee's exposure to cadmium and required to be kept by paragraph (n)(1) of this section.

(iv) Access to information and training materials.

(A) The employer shall make a copy of this standard and its appendices readily available without cost to all affected employees and shall provide a copy if requested.

(B) The employer shall provide to the Assistant Secretary or the Director, upon request, all materials relating to the employee information and the training program.

(n) *Recordkeeping*— (1) *Exposure monitoring.* (i) The employer shall establish and keep an accurate record of all air monitoring prescribed in paragraph (d) of this section.

(ii) This record shall include at least the following information:

(A) The monitoring date, duration, and results of each sample taken;

(B) The name, social security number, and job classification of the employee monitored and of all other employees whose exposures the monitoring is intended to represent;

(C) A description of the sampling and analytical methods used and evidence of their accuracy;

(D) The type of respiratory protective devices worn by the monitored employee, if any;

(E) Any other conditions that might have affected the employee monitoring results.

(iii) The employer shall maintain this record for at least thirty (30) years in accordance with § 1910.20.

(2) *Objective data for exempted operations.* (i) For purposes of this standard, objective data are information demonstrating that a particular product or material containing cadmium or a specific process, operation, or activity involving cadmium cannot release dust or fumes in concentrations at or above the action level or above the EL even under the worst-case release conditions. Objective data can be obtained from an industry-wide study or from laboratory product test results from manufacturers of cadmium-containing products or materials. The data the employer uses from an industry-wide survey must be obtained under workplace conditions closely resembling the processes, types of material, control methods, work practices and environmental conditions in the employer's current operations.

(ii) The employer shall maintain a record for at least 30 years of the objective data relied upon.

(3) *Medical surveillance.* (i) The employer shall establish and maintain an accurate record for each employee subject to medical surveillance by paragraph (l)(1)(i) of this section.

(ii) The record shall include at least the following information:

(A) The name, social security number, and description of the duties of the employee;

(B) A copy of the physician's written opinions;

(C) A copy of the medical history, and the results of any physical examination and all test results which are required to be provided by this standard (x-rays, pulmonary function tests, etc.) or which have been obtained to further evaluate any condition occurring as a result of cadmium exposure;

(D) Any employee's medical complaints that might be related to exposure to cadmium; and

(E) A copy of the information provided to the physician as required by paragraph (l)(9) of this section.

(iii) The employer shall assure that this record is maintained for the duration of employment plus thirty (30) years, in accordance with 29 CFR 1910.20.

(4) *Training.* The employer shall maintain the records of an employee's training for one (1) year beyond the last date of employment of that trained employee.

(5) *Availability.* (i) The employer, upon written request, shall make all records required to be maintained by this standard available to the Assistant Secretary and the Director for examination and copying.

(ii) The employer, upon request, shall make all records required to be maintained by paragraphs (n)(1) and (n)(2) of this section available for examination and copying to affected employees, former employees, designated representatives, the Director, and the Assistant Secretary, in accordance with 29 CFR 1910.20(a)-(e) and (g)-(i).

(iii) The employer, upon request, shall make employee medical records required to be kept by paragraph (n)(3) of this section available for examination and copying to the subject employee and to anyone having the specific written consent of the subject employee, and to the Director and the Assistant Secretary in accordance with 29 CFR 1910.20.

(6) *Transfer of records.* (i) Whenever an employer ceases to do business and there is no successor employer to receive and retain records for the prescribed period or the employer intends to dispose of any records required to be preserved for at least 30 years, the employer shall comply with the requirements concerning transfer of records set forth in 29 CFR 1910.20(h).

(o) *Observation of monitoring.*— (1) *Employee observation.* The employer shall provide affected employees or their designated representatives an opportunity to observe any monitoring of employee exposure to cadmium conducted in accordance with paragraph (d) of this section.

(2) *Observation procedures.* When observation of the monitoring requires entry into an area where the use of protective clothing or equipment is required, the employer shall provide the observer with, and the observer shall be required to use such clothing and equipment and shall comply with all

other applicable safety and health procedures.

(p) *Dates.*— (1) *Effective date.* This standard shall become effective (insert date 60 days from publication of the final rule in the **Federal Register**).

(2) *Start-up dates.* All obligations of this standard commence on the effective date except as follows:

(i) *Exposure monitoring.* Initial monitoring required by paragraph (d)(2) of this section shall be completed as soon as possible and in any event not later than 60 days after the effective date of this standard.

(ii) *Regulated areas.* Regulated areas required to be established by paragraph (e) of this section shall be set up as soon as possible after the results of exposure monitoring are known and in any event not later than 90 days after the effective date of this standard.

(iii) *Respiratory protection.* Respiratory protection required by paragraph (g) of this section shall be provided as soon as possible and in any event not later than 90 days after the effective date of this standard.

(iv) *Compliance program.* Written compliance programs required by paragraph (f)(2) of this section shall be completed and available for inspection and copying as soon as possible but no later than 1 year after the effective date of this standard.

(v) *Methods of compliance.* The engineering controls required by paragraph (f)(1) of this section shall be implemented as soon as possible but no later than 2 years after the effective date of this standard. Work practices shall be implemented as soon as possible. Work practice controls that are directly related to the engineering controls to be implemented in accordance with the compliance plan shall be implemented as soon as possible after such engineering controls are implemented.

(vi) *Hygiene and lunchroom facilities.* Construction plans for change rooms, showers, handwash facilities, and lunchroom facilities shall be completed no later than 120 days after the effective date of this standard and these facilities shall be constructed and in use no later than 1 year after the effective date of this standard. However, if as part of the compliance plan required by paragraph (f)(2) of this section it is predicted by an independent engineering firm that engineering controls and work practices will reduce exposures to or below the permissible exposure limit by 2 years after the effective date of this standard, then hygiene and lunchroom facilities need not be constructed. However, if after the engineering controls are completed engineering and work practice controls have not in fact succeeded in reducing exposure to or below the TWA PEL, then hygiene and lunchroom facilities shall be constructed and in use no later than two years and 8 months after the effective date of this standard.

(vii) *Employee information and training.* Employee information and training required by paragraph (m)(4) of this section shall be provided as soon as possible and in any event not later than 90 days after the effective date of this standard.

(viii) *Medical surveillance.* Medical examinations required by paragraph (l) of this section shall be provided as soon as possible and in any event not later than 90 days after the effective date of this standard.

(q) *Appendices.* (1) Appendix C of this section is part of this standard and the contents of this Appendix is mandatory.

(2) Appendices A, B, D, and E to this standard are purely informational and are not intended to create any additional obligations not otherwise imposed or to detract from any existing obligations.

Appendix A to § 1910.1027—Substance Safety Data Sheet; Cadmium

I. Substance Identification

A. Substance: Cadmium.

B. 8-Hour, Time-weighted-average, Permissible Exposure Limit (TWA PEL):

1. TWA PEL: One (five) micrograms of cadmium per cubic meter of air [1 (5) $\mu g/m^3$], time-weighted average (TWA) for an 8-hour workday.

2. Excursion Limit (EL): Five (twenty-five) micrograms per cubic meter of air [1 (25) $\mu g/m^3$] as a 15 minute Excursion Limit (EL) for a TWA PEL of 1(5) $\mu g/m^3$, respectively.

C. Appearance: Cadmium metal—soft, blue-white, malleable, lustrous metal or grayish-white powder.

II. Health Hazard Data

A. Routes of Exposure.

Cadmium can cause local skin or eye irritation. Cadmium can affect your health if you inhale it or if you swallow it.

B. Effects of overexposure.

1. Short-term (acute) exposure: Cadmium is much more dangerous by inhalation than by ingestion. The concentration of Cadmium that is immediately dangerous to life or health (IDLH) is 40 milligrams per cubic meter of air (40,000 micrograms per cubic meter of air). Severe exposure may occur before symptoms. Early symptoms may include mild irritation of the upper respiratory tract, a sensation of constriction of the throat, a metallic taste and/or a cough. A period of 1–10 hours may precede the onset of rapidly progressing shortness of breath, chest pain, and flu-like symptoms with weakness, fever, headache, chills, sweating and muscular pain. Acute pulmonary edema usually develops within 24 hours and reaches a maximum by 3 days. If death from asphyxia does not occur, symptoms may resolve within a week.

2. Long-term (chronic) exposure. Repeated or long-term exposure to cadmium, even at relatively low concentrations, may result in kidney damage and an increased risk of cancer of the lung and of the prostate.

III. Protective Clothing and Equipment

A. Respirators: You may be required to wear a respirator for non-routine activities, in emergencies, while your employer is in the process of reducing cadmium exposures through engineering controls, and where engineering controls are not feasible. If respirators are worn in the future, they must have a joint Mine Safety and Health Administration (MSHA) and National Institute for Occupational Safety and Health (NIOSH) label of approval.

Cadmium does not have a detectable odor except at levels well above the permissible exposure limits. If you can smell cadmium while wearing a respirator, proceed immediately to fresh air. If you experience difficulty breathing while wearing a respirator, tell your employer.

B. Protective clothing: You may be required to wear impermeable clothing, gloves, a face shield, or other appropriate protective clothing to prevent skin contact with cadmium. Where protective clothing is required, your employer must provide clean garments to you as necessary to assure that the clothing protects you adequately. Replace or repair protective clothing that has become torn or otherwise damaged.

C. Eye protection: You may be required to wear splash-proof or dust resistant goggles to prevent eye contact with cadmium.

IV. Emergency First Aid Procedures

A. Eye exposure: Direct contact may cause redness or pain. Wash eyes immediately with large amounts of water, lifting the upper and lower eyelids. Get medical attention immediately.

B. Skin exposure: Direct contact may result in irritation. Remove contaminated clothing and shoes immediately. Wash affected area with soap or mild detergent and large amounts of water. Get medical attention immediately.

C. Ingestion: Ingestion may result in vomiting, abdominal pain, nausea, diarrhea, headache and sore throat. Treatment for symptoms must be administered by medical personnel. Under no circumstances should the employer allow any person whom he retains, employs, supervises or controls to engage in therapeutic chelation. Such treatment is likely to translocate cadmium from pulmonary tissue to renal tissue. Get medical attention immediately.

D. Inhalation: If large amounts of cadmium are inhaled, the exposed person must be moved to fresh air at once. If breathing has stopped, perform cardiopulmonary resuscitation. Administer oxygen if available. Keep the affected person warm and at rest. Get medical attention immediately.

E. Rescue: Move the affected person from the hazardous exposure. If the exposed person has been overcome, attempt rescue only after notifying at least one other person of the emergency and putting into effect established emergency procedures. Do not become a casualty yourself. Understand your

emergency rescue procedures and know the location of the emergency equipment before the need arises.

V. Medical Requirements

If you are exposed to cadmium at or above the action level, your employer is required to provide a medical examination, laboratory tests and a medical history within 150 days of publication of the standard in the Federal Register and annually thereafter. These tests shall be provided without cost to you. In addition, if you are accidentally exposed to cadmium under conditions known or suspected to constitute toxic exposure to cadmium, your employer is required to make special tests available to you.

VI. Observation of Monitoring

Your employer is required to perform measurements that are representative of your exposure to cadmium and you or your designated representative are entitled to observe the monitoring procedure. You are entitled to observe the steps taken in the measurement procedure, and to record the result obtained. When the monitoring procedure is taking place in an area where respirators or personal protective clothing and equipment are required to be worn, you or your representative must also be provided with, and must wear the protective clothing and equipment.

VII. Access to Records

You or your representative are entitled to see the records of measurements of your exposure to cadmium. Your medical examination records can be furnished to your physician or designated representative upon request by you to your employer.

Appendix B to § 1910.1027—Substance Technical Guidelines for Cadmium

I. Cadmium Metal

A. Physical and Chemical Data

1. Substance Identification
Chemical name: Cadmium
Formula: Cd
Molecular Weight: 112.4
Chemical Abstracts Service (CAS) Registry No.: 7740-43-9
Other Identifiers: RETCS EU9800000; EPA D006; DOT 2570 53
Synonyms: Colloidal Cadmium: Kadmium (German): CI 77180
2. Physical data
Boiling point: (760 mm Hg): 765 degrees C
Melting point: 321 degrees C
Specific Gravity: (H_2O=@ 20 °C): 8.64
Solubility: Insoluble in water; soluble in dilute nitricacid and in sulfuric acid.
Appearance: Soft, blue-white, malleable, lustrous metal or grayish-white powder.

B. Fire, Explosion and Reactivity Data

1. Fire
Fire and Explosion Hazards: The finely divided metal is pyrophoric, that is the dust is a severe fire hazard and moderate explosion hazard when exposed to heat or flame. Burning material reacts violently with extinguishing agents such as water, foam, carbon dioxide, and halons.
Flash point: Flammable (dust)
Extinguishing media: Dry sand, dry dolomite, dry graphite, or sodimum chloride.
2. Reactivity
Conditions contributing to instability: Stable when kept in sealed containers under normal temperatures and pressure, but dust may ignite upon contact and with air. Metal tarnishes in moist air.
Incompatibilities: Ammonium nitrate, fused: reacts violently or explosively with cadmium dust below 20 °C. Hydrozoic acid: violent explosion occurs after 30 minutes. Acids: reacts violently, forms hydrogen gas. Oxidizing agents or metals: strong reaction with cadmium dust. Nitryl fluoride at slightly elevated temperature: glowing or white incandescence occurs. Selenium: react exothermically. Ammonia: corrosive reaction. Sulfur dioxide: corrosive reaction. Fire extinguishing agents (water, foam, carbon dioxide, and halons): reacts violently. Tellurium: incandescent reaction in hydrogen atmosphere.
Hazardous decomposition products: The heated metal rapidly forms highly toxic, brownish fumes of oxides of cadmium.

C. Spill, Leak and Disposal Procedures

1. *Steps to be taken if the material is released or spilled.* Do not touch spilled material. Stop leak if you can do it without risk. Do not get water inside container. For large spills, dike spill for later disposal. Keep unnecessary people away. Isolate hazard area and deny entry. The Superfund Amendments and Reauthorization Act of 1986 Section 304 requires that a release equal to or greater than the reportable quantity for this substance (1 pound) must be immediately reported to the local emergency planning committee, the state emergency response commission, and the National Response Center (800) 424-8802; in Washington, DC metropolitan area (202) 426-2675.

II. Cadmium Oxide

A. Physical and Chemical Data

1. Substance identification
Chemical name: Cadmium Oxide
Formula: CdO
Molecular Weight: 128.4
CAS No.: 1306-19-0
Other Identifiers: RTECS EV1929500
Synonyms: Kadmu tlenek (Polish)
2. Physical data
Boiling point (760 mm Hg): 950 degrees C decomposes
Melting point: 1500°C
Specific Gravity: (H^2O=1 @ 20° C): 7.0
Solubility: Insoluble in water; soluble in acids and alkalines
Appearance: Red or brown crystals

B. Fire, Explosion and Reactivity Data

1. Fire
Fire and Explosion Hazards: Negligible fire hazard when exposed to heat or flame.
Flash point: Nonflammable
Extinguishing media: Dry chemical, carbon dioxide, water spray or foam.
2. Reactivity
Conditions contributing to instability: Stable under normal termperatures and pressures.
Incompatibilities: Magnesium may reduce CdO_2 explosively on heating.
Hazardous decomposition products: Toxic fumes of cadmium.

C. Spill, Leak and Disposal Procedures

1. *Steps to be taken if the material is released or spilled.* Do not touch spilled material. Stop leak if you can do it without risk. For small spills, take up with sand or other absorbent material and place into containers for later disposal. For small dry spills, use a clean shovel to place material into clean, dry container and then cover. Move containers from spill area. For larger spills, dike far ahead of spill for later disposal. Keep unnecessary people away. Isolate hazard area and deny entry. The Superfund Amendments and Reauthorization Act of 1986 Section 304 requires that a release equal to or greater than the reportable quantity for this substance (1 pound) must be immediately reported to the local emergency planning committee, the state emergency response commission, and the National Response Center (800) 424-8802; in Washington, DC metropolitan area (202) 426-2675.

III. Cadmium Sulfide

A. Physical and Chemical Data

1. Substance Identification
Chemical name: Cadmium sulfide
Formula: CdS
Molecular weight: 144.5
CAS No. 1306-23-6
Other Identifiers: RTECS EV3150000
Synonyms: Aurora yellow; Cadmium Golden 366; Cadmium Lemon Yellow 527; Cadmium Orange; Cadmium Primrose 819; Cadmium Sulphide; Cadmium Yellow; Cadmium Yellow 000; Cadmium Yellow Conc. Deep; Cadmium Yellow Conc. Golden; Cadmium Yellow Conc. Lemon; Cadmium Yellow Conc. Primrose; Cadmium Yellow Oz. Dark; Cadmium Yellow Primrose 47-1400; Cadmium Yellow 10G Con.; Cadmium Yellow 892; Cadmopur Golden Yellow N; Cadmopur Yellow: Capsebon; C.I. 77199; C.I. Pigment Orange 20; CI Pigment Yellow 37; Ferro Lemon Yellow; Ferro Orange Yellow; Ferro Yellow; Greenockite; NCI-C02711.
2. Physical date
Boiling point (760 mm. Hg): subline in N_2 at 980°C
Melting point: 1750 degrees C (100 atm)
Specific Gravity (H_20=1@ 20°c): 4.82
Solubility: Slightly soluble in water; soluble in acid.
Appearance: Light yellow or yellow-orange crystals.

B. Fire, Explosion and Reactivity Data

1. Fire
Fire and Explosion Hazards: Neglible fire hazard when exposed to heat or flame.
Flash point: Nonflamable.
Extinguishing media: Dry chemical, carbon dioxide, water spray or foam.
2. Reactivity
Conditions contributing to instability: Generally non-reactive under normal conditions. Reacts with acids to form toxic hydrogen sulfide gas.
Incompatibilities: Reacts vigorously with iodine monochloride.
Hazardous decomposition products: Toxic fumes of cadmium and sulfur oxides.

C. Spill Leak and Disposal Procedures

1. Steps to be taken if the material is released or spilled. Do not touch spilled material. Stop leak if you can do it without risk. For small, dry spills, with a clean shovel place material into clean, dry container and cover. Move containers from spill area. For larger spills, dike far ahead of spill for later disposal. Keep unnecessary people away. Isolate hazard and deny entry.

IV. Cadmium Chloride

A. Physical and Chemical Data

1. Substance Identification

Chemical name: Cadmium chloride

Formula: $CdC1_2$

Molecular weight: 183.3

CAS No. 10108–64–2

Other Indentifiers: RTECS EY0175000

Synonyms: Caddy; Cadmium dichloride; NA 2570 (DOT); UI–CAD; dichlorocadmium

2. Physical data

Boiling point (760 mm Hg): 960 degrees C

Melting point: 568 degrees C

Specific Gravity (H_20=1@ 20°c): 4.05

Solubility: Soluble in water (140 g/100 cc); soluble in acetone.

Appearance: small, white crystals.

B. Fire, Explosion and Reactivity Data

1. Fire

Fire and Explosion Hazards: Negligible fire and negligible explosion hazard in dust form when exposed to heat or flame.

Flash point: Nonflamable.

Estinguishing media: Dry chemical, carbon dioxide, water spray or foam.

2. Reactivity

Conditions contributing to instability: Generally stable under normal temperatures and pressures.

Incompatibilities: Bromine triflouride rapidly attacks cadmium chloride. A mixture of potassium and cadmium chloride may produce a strong explosion on impact.

Hazardous decomposition products: Termal decomposition may release toxic fumes of hydrogen chloride, chloride, chlorine or oxides of cadmium.

C. Spill Leak and Disposal Procedures

1. Steps to be taken if the materials is released or spilled. Do not touch spilled material. Stop leak if you can do it without risk. For small, dry spills, with a clean shovel place material into clean, dry container and cover. Move containers from spill area. For larger spills, dike far ahead of spill for later disposal. Keep unnecessary people away. Isolate hazard and deny entry. The Superfund Amendments and Reauthorization Act of 1986 Section 304 requires that a release equal to or greater than the reportable quantity for this substance (100 pounds) must be immediately reported to the local emergency planning committee, the state emergency response commission, and the National Response Center (800) 424–8802; in Washington, DC Metropolitan area (202) 426–2675.

Appendix C to § 1910.1027—Qualitative and Quantitative Fit Testing Procedures

I. Fit Test Protocols

A. General:

The employer shall include the following provisions in the fit test procedures. These provisions apply to both qualitative fit testing (QLFT) and quantitative fit testing (QNFT). All testing is to be conducted annually.

1. The test subject shall be allowed to pick the most comfortable respirator from a selection including respirators of various sizes from different manufacturers. The selection shall include at least three sizes of elastomeric facepieces of the type of respirator that is to be tested, i.e., three sizes of half mask; or three sizes of full facepiece. Respirators of each size must be provided from at least two manufacturers.

2. Prior to the selection process, the test subject shall be shown how to put on a respirator, how it should be positioned on the face, how to set strap tension and how to determine a comfortable fit. A mirror shall be available to assist the subject in evaluating the fit and positioning the respirator. This instruction may not constitute the subject's formal training on respirator use; it is only a review.

3. The test subject shall be informed that he/she is being asked to select the respirator which provides the most comfortable fit. Each respirator represents a different size and shape, and if fitted, maintained and used properly, will provide substantial protection.

4. The test subject shall be instructed to hold each facepiece up to the face and eliminate those which obviously do not give a comfortable fit.

5. The more comfortable facepieces are noted; the most comfortable mask is donned and worn at least five minutes to assess comfort. Assistance in assessing comfort can be given by discussing the points in item 6 below. If the test subject is not familiar with using a particular respirator, the test subject shall be directed to don the mask several times and to adjust the straps each time to become adept at setting proper tension on the straps.

6. Assessment of comfort shall include reviewing the following points with the test subject and allowing the test subject adequate time to determine the comfort of the respirator:

(a) Position of the mask on the nose;

(b) Room for eye protection;

(c) Room to talk; and

(d) Position of mask on face and cheeks.

7. The following criteria shall be used to help determine the adequacy of the respirator fit:

(a) Chin properly placed;

(b) Adequate strap tension, not overly tightened;

(c) Fit across nose bridge;

(d) Respirator of proper size to span distance from nose to chin;

(e) Tendency of respirator to slip; and

(f) Self-observation in mirror to evaluate fit and respirator position.

8. The test subject shall conduct the negative and positive pressure fit checks as described below or in ANSI Z88.2–1980. Before conducting the negative or positive pressure test, the subject shall be told to seat the mask on the face by moving the head from side-to-side and up and down slowly while taking in a few slow deep breaths. Another facepiece shall be selected and retested if the test subject fails the fit check tests.

(a) *Positive pressure test.* Close off the exhalation valve and exhale gently onto the facepiece. The face fit is considered satisfactory if a slight positive pressure can be built up inside the facepiece without any evidence of outward leakage of air at the seal. For most respirators this method of leak testing requires the wearer to first remove the exhalation valve cover before closing off the exhalation valve and then carefully replacing it after the test.

(b) *Negative pressure test.* Close off the inlet opening of the canister or cartridge(s) by covering with the palm of the hand(s) or by replacing the filter seal(s). Inhale gently so that the facepiece collapses slightly, and hold the breath for ten seconds. If the facepiece remains in its slightly collapsed condition and no inward leakage of air is detected, the tightness of the respirator is considered satisfactory.

9. The test shall not be conducted if there is any hair growth between the skin and the facepiece sealing surface, such as stubble beard growth, beard, or long sideburns which cross the respirator sealing surface. Any type of appeal which interferes with a satisfactory fit shall be altered or removed.

10. If a test subject exhibits difficulty in breathing during the tests, she or he shall be referred to a physician trained in respiratory disease or pulmonary medicine to determine, in accordance with paragraph (l)(2) and (3) of this standard, whether the test subject can wear a respirator while performing her or his duties.

11. The test subject shall be given the opportunity to wear the successfully fitted respirator for a period of two weeks. If at any time during this period the respirator becomes uncomfortable, the test subject shall be given the opportunity to select a different facepiece and to be retested.

12. The employer shall maintain a record of the fit test administered to an employee. The record shall contain at least the following information:

(a) Name of employee;

(b) Type of respirator;

(c) Brand, size of respirator;

(d) Date of test; and

(e) Where QNFT is used, the fit factor and strip chart recording or other recording of the results of the test. The record shall be maintained until the next fit test is administered.

13. Exercise regimen. Prior to the commencement of the fit test, the test subject shall be given a description of the fit test and the test subject's responsibilities during the test procedure. The description of the process shall include a description of the test exercises that the subject will be performing. The respirator to be tested shall be worn for at least 5 minutes before the start of the fit test.

14. Test Exercises. The test subject shall perform exercises, in the test environment, in the manner described below:

(a) Normal breathing. In a normal standing position, without talking, the subject shall breathe normally.

(b) Deep breathing. In a normal standing position, without talking, the subject shall breathe slowly and deeply, taking care so as to not hyperventilate.

(c) Turning head side to side. Standing in place, the subject shall slowly turn his/her head from side to side between the extreme positions on each side. The head shall be held at each extreme momentarily so the subject can inhale at each side.

(d) Moving head up and down. Standing in place, the subject shall slowly move his/her head up and down. The subject shall be instructed to inhale in the up position (i.e., when looking toward the ceiling).

(e) Talking. The subject shall talk out loud slowly and loud enough so as to be heard clearly by the test conductor. The subject can read from a prepared text such as the Rainbow Passage, count backward from 100, or recite a memorized poem or song.

(f) Grimace. The test subject shall grimace by smiling or frowning.

(g) Bending over. The test subject shall bend at the waist as if he/she were to touch his/her toes. Jogging in place shall be substituted for this exercise in those test environments such as shroud type QNFT units which prohibit bending at the waist.

(h) Normal breathing. Same as exercise 1.

Each test exercise shall be performed for one minute except for the grimace exercise which shall be performed for 15 seconds. The test subject shall be questioned by the test conductor regarding the comfort of the respirator upon completion of the protocol. If it has become uncomfortable, another model of respirator shall be tried.

B. Qualitative Fit Test (QLFT) Protocols

1. General

(a) The employer shall assign specific individuals who shall assume full responsibility for implementing the respirator qualitative fit test program.

(b) The employer shall assure that persons administering QLFTs are able to prepare test solutions, calibrate equipment and perform tests properly, recognize invalid tests, and assure that test equipment is in proper working order.

(c) The employer shall assure that QLFT equipment is kept clean and well maintained so as to operate within the parameters for which it was designed.

2. Isoamyl Acetate Protocol

(a) Odor threshold screening. The odor threshold screening test, performed without wearing a respirator, is intended to determine if the individual tested can detect the odor of isoamyl acetate.

(1) Three 1-liter glass jars with metal lids are required.

(2) Odor free water (e.g., distilled or spring water) at approximately 25 degrees C shall be used for the solutions.

(3) The isoamyl acetate (IAA) (also known as isopentyl acetate) stock solution is prepared by adding 1 cc of pure IAA to 800 cc of odor free water in a 1 liter jar and shaking for 30 seconds. A new solution shall be prepared at least weekly.

(4) The screening test shall be conducted in a room separate from the room used for actual fit testing. The two rooms shall be well ventilated and shall not be connected to the same recirculating ventilation system.

(5) The odor test solution is prepared in a second jar by placing 0.4 cc of the stock solution into 500 cc of odor free water using a clean dropper or pipette. The solution shall be shaken for 30 seconds and allowed to stand for two to three minutes so that the IAA concentration above the liquid may reach equilibrium. This solution shall be used for only one day.

(6) A test blank shall be prepared in a third jar by adding 500 cc of odor free water.

(7) The odor test and test blank jars shall be labeled 1 and 2 for jar identification. Labels shall be placed on the lids so they can be periodically peeled, dried off and switched to maintain the integrity of the test.

(8) The following instruction shall be typed on a card and placed on the table in front of the two test jars (i.e., 1 and 2): "The purpose of this test is to determine if you can smell banana oil at a low concentration. The two bottles in front of you contain water. One of these bottles also contains a small amount of banana oil. Be sure the covers are on tight, then shake each bottle for two seconds. Unscrew the lid of each bottle, one at a time, and sniff at the mouth of the bottle. Indicate to the test conductor which bottle contains banana oil."

(9) The mixtures used in the IAA odor detection test shall be prepared in an area separate from where the test is performed, in order to prevent olfactory fatigue in the subject.

(10) If the test subject is unable to correctly identify the jar containing the odor test solution, the IAA qualitative fit test shall not be performed.

(11) If the test subject correctly identifies the jar containing the odor test solution, the test subject may proceed to respirator selection and fit testing.

(b) Isoamyl acetate fit test.

(1) The fit test chamber shall be similar to a clear 55-gallon drum liner suspended inverted over a 2-foot diameter frame so that the top of the chamber is about 6 inches above the test subject's head. The inside top center of the chamber shall have a small hook attached.

(2) Each respirator used for the fitting and fit testing shall be equipped with organic vapor cartridges or offer protection against organic vapors. The cartridges or masks shall be changed at least weekly.

(3) After selecting, donning, and properly adjusting a respirator, the test subject shall wear it to the fit testing room. This room shall be separate from the room used for odor threshold screening and respirator selection, and shall be well ventilated, as by an exhaust fan or lab hood, to prevent general room contamination.

(4) A copy of the test exercises and any prepared text from which the subject is to read shall be taped to the inside of the test chamber.

(5) Upon entering the test chamber, the test subject shall be given a 6-inch by 5-inch piece of paper towel, or other porous, absorbent, single-ply material, folded in half and wetted with 0.75 cc of pure IAA. The test subject shall hang the wet towel on the hook at the top of the chamber.

(6) Allow two minutes for the IAA test concentration to stabilize before starting the fit test exercises. This would be an appropriate time to talk with the test subject: to explain the fit test, the importance of his/her cooperation, and the purpose for the head exercises; and to demonstrate some of the exercises.

(7) If at any time during the test, the subject detects the banana like odor of IAA, the respirator fit is inadequate. The subject shall quickly exit from the test chamber and leave the test area to avoid olfactory fatigue.

(8) If the respirator fit was inadequate, the subject shall return to the selection room and remove the respirator, repeat the odor sensitivity test, select and put on another respirator, return to the test chamber and again begin the procedure described in paragraph (I)(B)(2)(b) (1) through (7) of this appendix. The process continues until a respirator that fits well has been found. Should the odor sensitivity test be failed, the subject shall wait about 5 minutes before retesting. Odor sensitivity will usually have returned by this time.

(9) When a respirator is found that passes the test, its efficiency shall be demonstrated for the subject by having the subject break the face seal and take a breath before exiting the chamber.

(10) When the test subject leaves the chamber, the subject shall remove the saturated towel and return it to the person conducting the test. To keep the test area from becoming contaminated, the used towels shall be kept in a self sealing bag so there is no significant IAA concentration build-up in the test chamber during subsequent tests.

3. Saccharin Solution Aerosol Protocol

The saccharin solution aerosol QLFT protocol is the only currently available, validated test protocol for use with particulate disposable dust respirators not equipped with high-efficiency filters. The entire screening and testing procedure shall be explained to the test subject prior to the conduct of the screening test.

(a) Taste threshold screening. The saccharin taste threshold screening, performed without wearing a respirator, is intended to determine whether the individual being tested can detect the taste of saccharin.

(1) Threshold screening as well as fit testing subjects shall wear an enclosure about the head and shoulders that is Approximately 12 inches in diameter by 14 inches tall with at least the front portion clear and that allows free movements of the head when a respirator is worn. An enclosure substantially similar to the 3M hood assembly, parts # FT 14 and # FT 15 combined, is adequate.

(2) The test enclosure shall have a ¾-inch hole in front of the test subject's nose and mouth area to accommodate the nebulizer nozzle.

(3) The test subject shall don the test enclosure. Throughout the threshold

screening test, the test subject shall breathe through his/her wide open mouth with tongue extended.

(4) Using a DeVilbiss Model 40 Inhalation Medication Nebulizer the test conductor shall spray the threshold check solution into the enclosure. This nebulizer shall be clearly marked to distinguish it from the fit test solution nebulizer.

(5) The threshold check solution consists of 0.83 grams of sodium saccharin USP in 1 cc of warm water. It can be prepared by putting 1 cc of the fit test solution (see (b)(5) below) in 100 cc of distilled water.

(6) To produce the aerosol, the nebulizer bulb is firmly squeezed so that it collapses completely, then released and allowed to fully expand.

(7) Ten squeezes are repeated rapidly and then the test subject is asked whether the saccharin can be tasted.

(8) If the first response is negative, ten more squeezes are repeated rapidly and the test subject is again asked whether the saccharin is tasted.

(9) If the second response is negative, ten more squeezes are repeated rapidly and the test subject is again asked whether the saccharin is tasted.

(10) The test conductor will take note of the number of squeezes required to solicit a taste response.

(11) If the saccharin is not tasted after 30 squeezes (step 10), the test subject may not perform the saccharin fit test.

(12) If a taste response is elicited, the test subject shall be asked to take note of the taste for reference in the fit test.

(13) Correct use of the nebulizer means that approximately 1 cc of liquid is used at a time in the nebulizer body.

(14) The nebulizer shall be thoroughly rinsed in water, shaken dry, and refilled at least each morning and afternoon or at least every four hours.

(b) Saccharin solution aerosol fit test procedure

(1) The test subject may not eat, drink (except plain water), or chew gum for 15 minutes before the test.

(2) The fit test uses the same enclosure described in (a) above.

(3) The test subject shall don the enclosure while wearing the respirator selected in section (a) above. The respirator shall be properly adjusted and equipped with a particulate filter(s).

(4) A second DeVilbiss Model 40 Inhalation Medication Nebulizer is used to spray the fit test solution into the enclosure. This nebulizer shall be clearly marked to distinguish it from the screening test solution nebulizer.

(5) The fit test solution is prepared by adding 83 grams of sodium saccharin to 100 cc of warm water.

(6) As before, the test subject shall breathe through the open mouth with tongue extended.

(7) The nebulizer is inserted into the hole in the front of the enclosure and the fit test solution is sprayed into the enclosure using the same number of squeezes required to elicit a taste response in the screening test.

(8) After generating the aerosol the test subject shall be instructed to perform the exercises in section I.A. 14 above.

(9) Every 30 seconds the aerosol concentration shall be replenished using one half the number of squeezes as initially.

(10) The test subject shall indicate to the test conductor if at any time during the fit test the taste of saccharin is detected.

(11) If the taste of saccharin is detected, the fit is deemed unsatisfactory and a different respirator shall be tried.

4. Irritant Fume Protocol

(a) The respirator to be tested shall be equipped with high-efficiency particulate air (HEPA) filters.

(b) The test subject shall be allowed to smell a weak concentration of the irritant smoke before the respirator is donned to become familiar with its characteristic odor.

(c) Break both ends of a ventilation smoke tube containing stannic oxychloride, such as the MSA part No. 5645, or equivalent. Attach one end of the smoke tube to a low flow air pump set to deliver 200 milliliters per minute.

(d) Advise the test subject that the smoke can be irritating to the eyes and instruct the subject to keep his/her eyes closed while the test is performed.

(e) The test conductor shall direct the stream of irritant smoke from the smoke tube towards the face seal area of the test subject. He/She shall begin at least 12 inches from the facepiece and gradually move to within one inch, moving around the whole perimeter of the mask.

(f) The exercises identified in section I. A. 14 above shall be performed by the test subject while the respirator seal is being challenged by the smoke.

(g) Each test subject passing the smoke test without evidence of a response shall be given a sensitivity check of the smoke from the same tube once the respirator has been removed to determine whether he/she reacts to the smoke. Failure to evoke a response shall void the fit test.

(h) The fit test shall be performed in a location with exhaust ventilation sufficient to prevent general contamination of the testing area by the test agent.

C. Quantitative Fit Test (QNFT) Protocol

1. *General.* (a) The employer shall assign specific individuals who shall assume full responsibility for implementing the respirator quantitative fit test program.

(b) The employer shall ensure that persons administering QNFT are able to calibrate equipment and perform tests properly, recognize invalid tests, calculate fit factors properly and assure that test equipment is in proper working order.

(c) The employer shall assure that QNFT equipment is kept clean and well maintained so as to operate at the parameters for which it was designed.

2. *Definitions.* (a) Quantitative fit test. The test is performed in a test chamber. The normal air-purifying element of the respirator is replaced by a high-efficiency particulate air (HEPA) filter in the case of particulate QNFT aerosols or a sorbent offering contaminant penetration protection equivalent to high-efficiency filters where the QNFT test agent is a gas or vapor.

(b) Challenge agent means the aerosol, gas or vapor introduced into a test chamber so that its concentration inside and outside the respirator may be measured.

(c) Test subject means the person wearing the respirator for quantitative fit testing.

(d) Normal standing position means standing erect and straight with arms down along the sides and looking straight ahead.

(e) Maximum peak penetration method means the method of determining test agent penetration in the respirator as determined by strip chart recordings of the test. The highest peak penetration for a given exercise is taken to be representative of average penetration into the respirator for that exercise.

(f) Average peak penetration method means the method of determining test agent penetration into the respirator utilizing a strip chart recorder, integrator, or computer. The agent penetration is determined by an average of the peak heights on the graph or by computer integration, for each exercise except the grimace exercise. Integrators or computers which calculate the actual test agent penetration into the respirator for each exercise will also be considered to meet the requirements of the average peak penetration method.

(g) "Fit Factor" means the ration of challenge agent concentration outside with respect to the inside of a respirator inlet covering (facepiece or enclosure).

3. *Apparatus.* (a) Instrumentation. Aerosol genetration, dilution, and measurement systems using corn oil or sodium chloride as test aerosols shall be used for quantitative fit testing.

(b) Test chamber. The test chamber shall be large enough to permit all test subjects to perform freely all required exercises without disturbing the challenge agent concentration or the measurement apparatus. The test chamber shall be equipped and constructed so that the challenge agent is effectively isolated from the ambient air, yet uniform in concentration throughout the chamber.

(c) When testing air-purifying respirators, the normal filter or cartridge element shall be replaced with a high-efficiency particulate filter supplied by the same manufacturer.

(d) The sampling instrument shall be selected so that a strip chart record may be made of the test showing the rise and fall of the challenge agent concentration with each inspiration and expiration at fit factors of at least 2,000. Integrators or computers which integrate the amount of test agent penetration leakage into the respirator for each exercise may be used provided a record of the readings is made.

(e) The combination of substitute air-purifying elements, challenge agent and challenge agent concentration in the test chamber shall be such that the test subject is not exposed in excess of an established exposure limit for the challenge agent at any time during the testing process.

(f) The sampling port on the test specimen respirator shall be placed and constructed so that no leakage occurs around the port (e.g. where the respirator is probed), a free air flow is allowed into the sampling line at all times and so that there is no interference with the fit or performance of the respirator.

(g) The test chamber and test set up shall permit the person administering the test to

observe the test subject inside the chamber during the test.

(h) The equipment generating the challenge atmosphere shall maintain the concentration of challenge agent inside the test chamber constant to within a 10 percent variation for the duration of the test.

(i) The time lag (interval between an event and the recording of the event on the strip chart or computer or integrator) shall be kept to a minimum. There shall be a clear association between the occurrence of an event inside the test chamber and its being recorded.

(j) The sampling line tubing for the test chamber atmosphere and for the respirator sampling port shall be of equal diameter and of the same material. The length of the two lines shall be equal.

(k) The exhaust flow from the test chamber shall pass through a high-efficiency filter before release.

(l) When sodium chloride aerosol is used, the relative humidity inside the test chamber shall not exceed 50 percent.

(m) The limitations of instrument detection shall be taken into account when determining the fit factor.

(n) Test respirators shall be maintained in proper working order and inspected for deficiencies such as cracks, missing valves and gaskets, etc.

4. *Procedural Requirements.* (a) When performing the initial positive or negative pressure test the sampling line shall be crimped closed in order to avoid air pressure leakage during either of these tests.

(b) An abbreviated screening isoamyl acetate test or irritant fume test may be utilized in order to quickly identify poor fitting respirators which passed the positive and/or negative pressure test and thus reduce the amount of QNFT time. When performing a screening isoamyl acetate test, combination high-efficiency organic vapor cartridges/canisters shall be used.

(c) A reasonably stable challenge agent concentration shall be measured in the test chamber prior to testing. For canopy or shower curtain type of test units the determination of the challenge agent stability may be established after the test subject has entered the test environment.

(d) Immediately after the subject enters the test chamber, the challenge agent concentration inside the respirator shall be measured to ensure that the peak penetration does not exceed 5 percent for a half mask or 1 percent for a full facepiece respirator.

(e) A stable challenge concentration shall be obtained prior to the actual start of testing.

(f) Respirator restraining straps shall not be overtightened for testing. The straps shall be adjusted by the wearer without assistance from other persons to give a reasonable comfortable fit typical of normal use.

(g) The test shall be terminated whenever any single peak penetration exceeds 5 percent for half masks and 1 percent for full facepiece respirators. The test subject shall be refitted and retested. If two of the three required tests are terminated, the fit shall be deemed inadequate.

(h) In order to successfully complete a QNFT, three successful fit tests are required. The results of each of the three independent fit tests must exceed the minimum fit factor needed for the class of respirator (e.g. half mask respirator, full facepiece respirator).

(i) Calculation of fit factors.

(1) The fit factor shall be determined for the quantitative fit test by taking the ratio of the average chamber concentration to the concentration inside the respirator.

(2) The average test chamber concentration is the arithmetic average of the test chamber concentration at the beginning and of the end of the test.

(3) The concentration of the challenge agent inside the respirator shall be determined by one of the following methods:

(i) Average peak concentration

(ii) Maximum peak concentration

(iii) Integration by calculation of the area under the individual peak for each exercise. This includes computerized integration.

(j) Interpretation of test results. The fit factor established by the quantitative fit testing shall be the lowest of the three fit factor values calculated from the three required fit tests.

(k) The test subject shall not be permitted to wear a half mask, or full facepiece respirator unless a minimum fit factor equivalent to at least 10 times the hazardous exposure level is obtained.

(l) Filters used for quantitative fit testing shall be replaced at least weekly, or whenever increased breathing resistance is encountered, or when the test agent has altered the integrity of the filter media. Organic vapor cartridges/canisters shall be replaced daily (when used) or sooner if there is any indication of breakthrough by a test agent.

Appendix D to § 1910.1027—Occupational Health History Interview With Reference to Cadmium Exposure

Directions

(To be read by employee and signed prior to the interview)

Please answer the questions you will be asked as completely and carefully as you can. These questions are asked of everyone who works with cadmium. You will also be asked to give blood and urine samples, and have a chest x-ray and lung tests. The doctor will give your employer a written opinion on whether you are physically capable of doing your job. The results of the tests will go to both the doctor and your employer. Legally, the doctor cannot share personal information you may tell him/her with your employer.

If you are just being hired the results of this interview and examination will be used to:

(1) establish your health status and see if working with cadmium might be expected to cause unusual problems,

(2) determine your health status today and see if there are changes over time,

(3) see if you can wear a respirator safely.

If you are not a new hire:

OSHA says that everyone who works with cadmium can have an examination supervised by a doctor every year. The reasons for this are:

(a) if there are changes in your health, either because of cadmium or some other reason, to find them early,

(b) to prevent kidney damage.

Please sign below.

I have read these directions and understand them:

_____

Employee signature and date

Thank you for answering these questions.

(Suggested Format)

BILLING CODE 4510-26-M

Name _____ Age _____ Social Security II _____

Company _____ Job _____ Type of exam: [ ] Preplacement [ ] Periodic [ ] Termination [ ] Other

Blood Pressure _____
Pulse Rate _____

1. How long have you worked at the job listed above? [ ] not yet hired [ ] number of months [ ] number of years

2. Please complete the following information, beginning with your most recent job:

Previous jobs	Employer and Type of business	Number years employed	Exposures

3. Have you ever been told by a doctor that you had bronchitis? [ ] yes [ ] no

How long ago? [ ][ ] number of months
[ ][ ] number of years

4. Have you ever been told by a doctor that you had emphysema? [ ] yes [ ] no

How long ago? [ ][ ] number of months
[ ][ ] number of years

5. Have you ever been told by a doctor that you had other lung problems? [ ] yes [ ] no

If yes, please describe type of lung problems and when you had these problems _____

6. In the past year, have you had a cough? [ ] yes [ ] no

If yes, did you cough up sputum? [ ] yes [ ] no

If yes, How long did the cough with sputum production last? [ ] less than 3 months [ ] 3 months or longer

For how many years have you had episodes of cough with sputum production lasting this long? [ ] less than one [ ] 1 [ ] 2 [ ] longer than 2

7. Have you ever smoked cigarettes? [ ] yes [ ] no

8. Do you now smoke cigarettes? [ ] yes [ ] no

9. If you smoke or have smoked cigarettes, for how many years have you smoked, or did you smoke? [ ] less than 1 year [ ][ ] number of years

What is or was the greatest number of packs per day that you have smoked? [ ][ ] number of packs

If you quit smoking cigarettes, how many years ago did you quit? [ ] less than 1 year [ ][ ] number of years

How many packs a day do you now smoke? [ ][ ] number of packs per day

10. Have you ever been told by a doctor that you had a kidney or urinary tract disease or disorder [ ] yes [ ] no

11. Have you ever had any of these disorders?

Kidney stones [ ] yes [ ] no

Protein in urine [ ] yes [ ] no

Blood in urine [ ] yes [ ] no

Difficulty urinating [ ] yes [ ] no

Other kidney / Urinary disorders [ ] yes [ ] no

Please describe problems, age, treatment, and followup for any kidney or urinary problems you have had: _____

12. Have you ever been told by a doctor or other health care provider who took your blood pressure that your blood pressure was high? [ ] yes [ ] no

13. Have you ever been advised to take any blood pressure medication? [ ] yes [ ] no

14. Are you presently taking any blood pressure medication? [ ] yes [ ] no

15. Are you presently taking any other medication? [ ] yes [ ] no

16. Please list any blood pressure or other medications and describe how long you have been taking each one:

Medicine	How long taken

17. Have you ever been told by a doctor that you have diabetes? (sugar in your bloodor urine) [ ] yes [ ] no

If yes,

Do you presently see a doctor about your diabetes? [ ] yes [ ] no

How do you control your blood sugar? [ ] diet alone [ ] diet plus oral medicine [ ] diet plus insulin (injection)

18. Have you ever had any broken bone(s)? [ ] yes [ ] no

If yes,

Have you ever been told by a doctor that the bone(s) took a long time to heal? [ ] yes [ ] no

Please describe bone(s) broken, your age, and how break(s) happened:

Bone	Age	How happened

19. Have you ever been told by a doctor that you had:
    a) anemia [ ] yes [ ] no
    b) a low blood count? [ ] yes [ ] no

20. Do you presently feel that you tire or run out of energy sooner than normal or sooner than most other people your age? [ ] yes [ ] no

    If yes, for how long have you felt that you tire easily? [ ] less than 1 year [ ][ ] number of years

21. Have you given blood within the last year? [ ] yes [ ] no

    If yes,

    How many times? [ ][ ] number of times

    How long ago was the last time you gave blood? [ ] less than 1 month [ ][ ] number of months

22. Have you recently had any injuries with heavy bleeding? [ ] yes [ ] no

    If yes, How long ago? [ ] less than 1 month [ ][ ] number of months

    Describe _____

23. Have you recently had any surgery? [ ] yes [ ] no

    If yes, please describe _____

24. Have you seen any blood lately in your stool or after a bowel movement? [ ] yes [ ] no

25. Have you ever had a test for blood in your stool? [ ] yes [ ] no

    If yes, did the test show any blood in the stool? [ ] yes [ ] no

    What further evaluation and treatment were done? _____

26. In addition, the following questions address your ability to wear a respirator. Additional information for the physician can be found in The Respiratory Protective Devices Manual.

Have you ever been told by a doctor that you have asthma? [ ] yes [ ] no

Are you presently taking shots for asthma? [ ] yes
Are you presently taking pills for asthma? [ ] no

Have you ever had a heart attack? [ ] yes [ ] no

If yes, how long ago? [ ][ ] No. of months [ ][ ] No. of years

Have you ever had pains in your chest? [ ] yes [ ] no

If yes, when did it usually happen?
While resting [ ]
While working [ ]
While exercising [ ]
Activity didn't matter [ ]

Have you ever had a thyroid problem? [ ] yes [ ] no

Have you ever had a seizure or fits? [ ] yes [ ] no

Have you ever had a stroke (cerebrovascular accident)? [ ] yes [ ] no

Have you ever had a ruptured eardrum or a serious hearing problem? [ ] yes [ ] no

Do you now have a claustrophobia, by that is meant fear of crowded or closed in spaces or any psychological problems that would make it hard for you to wear a respirator? [ ] yes [ ] no

27. Please complete only one of the following two sections

FOR WOMEN ONLY

Do you have menstrual periods? [ ] yes [ ] no

If yes, do you have normal bleeding? [ ] yes [ ] no

FOR MEN ONLY

Have you ever been told by a doctor that you had any problems with your prostate gland? [ ] yes [ ] no

If yes, please describe type of problem(s) and what was done to evaluate and treat the problem(s) _____

FOR WOMEN ONLY

Are your periods regular [ ] yes [ ] no

Do you have children [ ] yes [ ] no

How many? [ ][ ]

Are the children healthy? [ ] yes [ ] no

Do any of the children have birth defects? [ ] yes [ ] no

If so, how many children? [ ][ ]

FOR WOMEN and MEN

Have you had any problem with fertility (want children but cannot)? [ ] yes [ ] no

BILLING CODE 4510-26-C

Appendix E to § 1910.1027

This Appendix is divided into two parts. The first part, Appendix-E1, is written for a time-weighted average (8-hour) permissible exposure limit (TWA PEL) of 5 $\mu g/m^3$, an action level of 2.5 $\mu g/m^3$ (and is applicable for an action level of 1 $\mu g/m^3$), and an excursion limit (EL) of 25 $\mu g/m^3$. The second part, Appendix-E2, is written for a TWA PEL of 1 $\mu g/m^3$, an action level of 0.5 $\mu g/m^3$, and an EL of 5 $\mu g/m^3$.

Appendix–E1

Method Number: ID–189 (Proposed).
Matrix: Air.
Target Concentration: 5 $\mu g/m^3$ (TWA).
Collection Procedure: A known volume of air is drawn through a 37-mm diameter filter cassette containing a 0.8-μm mixed cellulose ester membrane filter (MCEF).
Recommended Air Volume: 200 L to 960 L.
Recommended Sampling Rate: 2.0 L/min.
Analytical Procedure: Air filter samples are wet-ashed with nitric acid. After digestion, a small amount of hydrochloric acid is added. The samples are then diluted to volume with deionized water and analyzed by atomic absorption spectroscopy with an oxidizing air/acetylene flame.
Qualitative Detection Limit: 0.25 $\mu g/m^3$ for a 200 L air sample.
Quantitative Detection Limit: 1 $\mu g/m^3$ for a 200 L air sample.
Precision: (CV_1=0.010.
Method Classification: Validated.

ID–189

Cadmium in Workplace Atmosphere (Flame AAS)

1. Introduction

1.1. Scope

This method describes the collection of airborne elemental cadmium and cadmium compounds on 0.8-um mixed cellulose ester membrane filters and their subsequent analysis by flame atomic absorption spectroscopy. It is applicable for TWA measurements at the target level of 5 $\mu g/m^3$ and should be used concurrently with Method ID–189GF (7.6) which is applicable for Action Level TWA and EL measurements. It is also applicable for the collection and analysis of cadmium wipe and bulk material samples. The analytical method does not differentiate between cadmium fume and cadmium dust samples. It also does not differentiate between elemental cadmium and its compounds.

1.2. Principle

Airborne elemental cadmium and cadmium compounds are collected on a 0.8-um mixed cellulose ester membrane filter (MCEF). The air filter samples are wet-ashed with concentrated nitric acid to destroy the organic matrix and dissolve the cadmium analytes. A small amount of concentrated hydrochloric acid is added to help dissolve other metals which may be present. The samples are diluted with deionized water and then aspirated into the oxidizing air/acetylene flame of an atomic absorption spectrophotometer for analysis of elemental cadmium.

1.3. History

Previously, two OSHA sampling and analytical procedures for cadmium were used concurrently (7.1., 7.2.). Both of these procedures also required 0.8-um mixed cellulose ester membrane filters for the collection of air samples. These cadmium air filter samples were analyzed by either flame atomic absorption spectroscopy (AAS) (7.1.) or inductively coupled plasma/atomic emission spectroscopy (ICP) (7.2.). The new flame AAS method for the analysis of cadmium is similar to the old procedure given in the General Metals Method ID–121 (7.1.) with some modifications.

1.4. Properties (7.3.)

Elemental cadmium is a silver-white, blue-tinged, lustrous metal which is easily cut with a knife. It is slowly oxidized by moist air to form cadmium oxide. It is insoluble in water, but reacts readily with dilute nitric acid. Some of the physical properties and other descriptive information of elemental cadmium are given below:

CAS No.	7440–43–9
Atomic Number	48
Atomic Symbol	Cd
Atomic Weight	112.41
Melting Point	321°C
Boiling Point	765°C
Density	8.65 g/mL (25 °C)

The properties of specific cadmium compounds are described in reference 7.3.

2. Detection Limit (7.4.)

2.1. The qualitative detection limit for the analytical procedure is 0.05 μg cadmium for a 10 mL solution volume. This corresponds to 0.25 $\mu g/m^3$ for a 200 L air volume.

2.2. The quantitative detection limit for the analytical procedure is 0.2 ug cadmium for a 10 mL solution volume. This corresponds to 1 ug/m^3 for a 200 L air volume.

3. Precision and Accuracy

The average recovery of seventeen spiked MCEF samples containing cadmium in the range of 0.5 to 2.0 times the TWA target concentration of 5 $\mu g/m^3$ (assuming a 400 L air volume) was 104.0% with a pooled coefficient of variation (CV_1) of 0.010 (7.4.).

4. Interferences

There are no known interferences in either sampling or analysis (7.5.).

5. Sampling

5.1. Apparatus

5.1.1. Filter cassette unit for air sampling: A 37–mm diameter mixed cellulose ester membrane filter (Millipore type AA or equivalent with a pore size of 0.8 um) contained in a 37–mm polystyrene two- or three-piece cassette filter holder. The filter is supported with a cellulose backup pad. The cassette is sealed with a shrinkable gel band.

5.1.2. A calibrated personal sampling pump whose flow is determined to an accuracy of ±5% at the recommended flow rate with the filter cassette unit in line.

5.2. Procedure

5.2.1. Sample with the air filter cassettes for elemental cadmium and its compounds in accordance with current instructions in OSHA directives to the industrial hygienist.

5.2.2. Collect air samples at a flow rate of 2.0 L/min. A full-shift (at least seven hours) sample is recommended with a maximum air volume of 960 L, if the filter does not become overloaded. The minimum suggested air volume is 200 L.

5.2.3. Replace the end plugs into the filter cassettes immediately after air sampling.

5.2.4. Securely wrap each sample filter cassette end-to-end with an OSHA Form 21 sample seal.

5.2.5. Submit at least one blank sample with each set of air samples. The blank sample should be handled the same as the other samples except that no air is drawn through it.

5.2.6. Ship the samples to the laboratory for analysis as soon as possible in a suitable container designed to prevent damage in transit.

6. Analytical Procedure

6.1. Apparatus

6.1.1. Atomic absorption spectrophotometer (Perkin Elmer Model 5000 or its equivalent) equipped with a nebulizer and a four inch (one slot) burner head for use with an air/acetylene flame.

6.1.2. Oxidant: compressed air which has been filtered to remove water, oil and other foreign substances.

6.1.3. Fuel: standard commercially available tanks of acetylene dissolved in acetone; tanks should be equipped with flash arresters. CAUTION: Do not use grades of acetylene available from certain suppliers that contain solvents other than acetone which may damage the PVC tubing used in some instruments.

6.1.4. Pressure-reducing valves: two gauge, two-stage pressure regulators to maintain fuel and oxidant pressures somewhat higher than the controlled operating pressures of the instrument.

6.1.5. Cadmium hollow cathode lamp.

6.1.6. Hot plate, capable of reaching 150 °C.

6.1.7. 125 mL Phillips beakers.

6.1.8. Bottles, 500-mL, narrow-mouth, polyethylene or glass with leakproof caps: use for storage of standards.

6.1.9. Volumetric flasks, volumetric pipets, beakers and other associated general laboratory glassware.

6.1.10. Forceps.

6.2. Reagents

All reagents should be ACS analytical reagent grade or better.

6.2.1. Deionized water with a resistivity of at least 200k ohms.

6.2.2. Concentrated nitric acid, HNO_3.

6.2.3. Concentrated hydrochloric acid, HC1.

6.2.4. Diluting solution (4% HNO_3, 0.4% HC1): Add 40 mL HNO_3 and 4 mL HC1 carefully to approximately 500 mL deionized water and then dilute to 1000 mL.

6.2.5. 1000 $\mu g/mL$ cadmium standard stock solution: Use a commercially available certified 1000 $\mu g/mL$ cadmium standard or, alternatively, dissolve 1.0000 g of cadmium metal in a minimum volume of 1:1 HC1 and dilute to 1 L with 4% HNO_3.

6.3 Safety Precautions

6.3.1. Wear safety glasses and gloves at all times.

6.3.2. Handle acid solutions with care. Handle all cadmium samples and solutions with extra care. Avoid their direct contact with work area surfaces, eyes, skin and

clothes. Flush acid solutions which contact the skin or eyes with copious amounts of water.

6.3.3. Perform all acid digestions and acid dilutions in a fume hood.

6.3.4. Exercise care when using laboratory glassware. Do not use chipped pipets, volumetric flashs, beakers or any glassware with sharp edges exposed in order to avoid the possibility of cuts or abrasions.

6.3.5. Never pipet by mouth.

6.3.6. Refer to the instrument instruction manual and reference 7.1. for proper and safe operation of the atomic absorption spectrophotometer and associated equipment.

6.3.7. Since metallic elements and other toxic substances are vaporized during flame operation, it is imperative that an exhaust system be used. Always ensure that the exhaust system is operating properly during instrument use.

6.4. Glassware Preparation

6.4.1. Clean the Phillips beakers by refluxing with 1:1 nitric acid on a hot plate in a fume hood. Thoroughly rinse with deionized water and then invert the beakers to allow them to drain dry.

6.4.2. Rinse volumetric flasks and all other glassware with 10% nitric acid and deionized water prior to use.

6.5. Standard Preparation

6.5.1. Prepare 5, 10 and 100 μg/mL cadmium working standard stock solutions by making appropriate serial dilutions of the 1000 μg/mL cadmium standard stock solution with the diluting solution described in Section 6.2.4.

6.5.2. Prepare cadmium standards to be analyzed in the range of 0.02 to 2.0 μg/mL by making appropriate serial dilutions of the working standards with the same diluting solution. A suggested method of preparation of these standards is given in Table I. Store these standard solutions in the 500-mL, narrow-mouth polyethylene or glass bottles with leakproof caps.

6.6. Sample Preparation

6.6.1. Carefully transfer each sample filter with forceps from its filter cassette unit to a clean, separate 125-mL Phillips beaker along with any loose dust found in the cassette. Label each Phillips beaker with the appropriate sample number.

6.6.2. Add 5 mL of concentrated nitric acid to each Phillips beaker containing an air filter sample. Place the Phillips beakers on a hot plate in a fume hood and heat the samples until approximately 1 mL remains. The sample solution in each Phillips beaker should become clear. If it does not, wet-ash the sample with another portion of concentrated nitric acid.

6.6.3. After completing the HNO_3 digestion and cooling the samples, add 40 μL of concentrated HCl to each air sample solution. Swirl and then gently warm the contents of each Phillips beaker.

6.6.4. Quantitatively transfer each cooled air sample solution from its Phillips beaker to a clean 40-mL volumetric flask. Dilute each flask to volume with deionized water and then mix well.

6.7. Analysis

Initially analyze all of the air samples for their cadmium content by flame atomic absorption spectroscopy (AAS) according to the instructions given below. If the concentration of cadmium in a sample is less than 0.04 μg/mL, proceed with the graphite furnace AAS analysis of the sample as described in reference 7.6.

The mentioned instrument settings are for the specific instrument models used in the OSHA laboratory. These settings may vary when using other systems.

6.7.1. Set up the atomic absorption spectrophotometer for the air/acetylene flame analysis of cadmium according to the manufacturer's operational instructions. For the source lamp, use the cadmium hollow cathode lamp operated at the manufacturer's recommended current rating for continuous operation.

6.7.2. Make the following initial instrument settings on the Perkin Elmer Model 5000 spectrophotometer:

Slit=High
Slitwidth=0.7 nm
Wavelength=228.8 nm
Mode=AA/ABS
Signal=Cont
Integration Time=0.5 Sec
Range=UV

6.7.3. Optimize the energy reading of the spectrophotometer at 228.8 nm by adjusting the lamp position according to the manufacturer's instructions.

6.7.4. Light the air/acetylene flame and autozero the instrument while aspirating a deionized water blank.

6.7.5. Optimize conditions such as burner head alignment and fuel and oxidant flow rates to give a maximum absorbance reading of the aspirated 2 μg/mL standard. The 2 μg/mL cadmium standard should give an absorbance reading of about 0.350 abs. units.

6.7.6 Reset the MODE parameter from ABS to CONC and the INTEGRATION TIME to at least 3.0 SEC.

6.7.7. To increase instrument response, scale expand the absorbance reading of the aspirated 2 μg/mL standard approximately four times.

6.7.8. Autozero the instrument once again while aspirating a deionized water blank and then reset the SIGNAL parameter from CONT to HOLD. Turn on the printer, if one is available, and date and label the printer paper.

6.7.9. Aspirate the standards and samples directly into the flame and record their absorbance readings. Aspirate the deionized water blank into the flame immediately after every standard or sample to correct for and monitor any baseline drift. Record also this baseline absorbance reading of the deionized water blank. Label each standard and sample reading and its accompanying baseline reading.

6.7.10. It is recommended that the entire series of standards be analyzed at the beginning and end of the analysis of a set of samples to ensure that the standard readings are reproducible. Also, analyze a standard after every five or six samples to monitor the performance of the spectrophotometer.

6.7.11. Bracket the samples with standards during the analysis. Repeat the analysis of approximately 10% of the samples for a check of precision.

6.7.12. Record the final instrument settings on the printer paper output at the end of the analysis.

6.8. Calculations

6.8.1. Correct for baseline drift by subtracting each baseline absorbance reading from its corresponding standard or sample absorbance reading.

6.8.2. Use a least squares regression program to plot a calibration curve of absorbance reading versus the concentration (μg/mL) of cadmium in each standard.

6.8.3. Determine the concentration (μg/mL) of cadmium, C, corresponding to the absorbance reading in each analyzed sample from the resulting calibration curve.

6.8.4. Calculate the total amount (μg) of cadmium, W, in each sample from the solution volume (mL):

W = (C)(sample vol, mL)(DF)

Where: DF = Dilution Factor (use only if applicable)

6.8.5. Make a blank correction for each air sample by subtracting the total amount of cadmium in the corresponding blank sample from the total amount of cadmium in the sample.

6.8.6. Calculate the concentration of cadmium in an air sample in units of mg/m^3 or $\mu g/m^3$ by using one of the following equations:

$mg/m^3 = W_{bc}/(\text{Air vol sampled, L})$

or

$\mu g/m^3 = (W_{bc})(1000\ ng/\mu g)/(\text{Air vol sampled, L})$

Where: W_{bc} = blank corrected total amount (μg) of cadmium in the sample.

7. References:

7.1. *OSHA Analytical Methods Manual;* U.S. Department of Labor, Occupational Safety and Health Administration, OSHA Analytical Laboratory, Salt Lake City, UT; Am. Conf. of Governmental Ind. Hyg. (ACGIH): Cincinnati, OH, 1985; Method ID-121.

7.2. *OSHA Analytical Methods Manual;* U.S. Department of Labor, Occupational Safety and Health Administration, OSHA Analytical Laboratory, Salt Lake City, UT; Am. Conf. of Governmental Ind. Hyg. (ACGIH): Cincinnati, OH, 1985; Method ID-125.

7.3. Windholz, M., Ed.; *The Merck Index,* 10th ed.; Merck & Co.: Rahway, NJ, 1983.

7.4. *Backup Data Report for Cadmium (Flame AAS), Method ID-189,* Inorganic Division, OSHA Analytical Laboratory, Salt Lake City, UT, 1988.

7.5. *Analytical Methods for Atomic Absorption Spectrophotometry,* The Perkin-Elmer Corporation: Norwalk, CT, 1973.

7.6. *Cadmium in Workplace Atmospheres (GF-AAS), Method ID-189GF,* Inorganic Division, OSHA Analytical Laboratory, Salt Lake City, UT, 1988.

TABLE I.—CD STANDARD PREPARATION

Standard (μg/mL)	mL stock	Stock solution (μg/mL)	Final vol (mL)
0.02	10	1	500
0.05	5	5	500
0.10	5	10	500
0.20	10	10	500
0.50	25	10	500

TABLE I.—CD STANDARD PREPARATION—Continued

Standard (μg/mL)	mL stock	Stock solution (μg/mL)	Final vol (mL)
1.00	5	100	500
2.00	10	100	500

Cadmium in Workplace Atmosphere (GF-AAS)

Method Number: ID-189GF (Proposed).
Matrix: 2.5 ug/m³ (Action Level TWA), 25 ug/m³ (EL).
Collection Procedure: A known volume of air is drawn through a 37-mm diameter filter cassette containing a 0.8-um mixed cellulose ester membrane filter (MCEF).
Recommended Air Volumes: 200 L to 960 L for Action Level TWA, 30 L for EL.
Recommended Sampling Rate: 2.0 L/min.
Analytical Procedure: Air filter samples are wet-ashed with nitric acid. After digestion, a small amount of hydrochloric acid is added. The samples are then diluted to volume with deionized water and analyzed by flameless atomic absorption spectroscopy using a heated graphite furnace atomizer.
Qualitative Detection Limit: 0.07 ug/m³ for a 30 L air sample.
Quantitative Detection Limit: 0.33 ug/m³ for a 30 L air sample.
Precision: (CV_1) = 0.074.
Method Classification: Validated.

ID-189GF

Cadmium in Workplace Atmospheres (GF-AAS)

1. Introduction

1.1. Scope

This method describes the collection of airborne elemental cadmium and cadmium compounds on 0.8-um mixed cellulose ester membrane filters and their subsequent analysis by flameless atomic absorption spectroscopy using a heated graphite furnace atomizer. It is applicable for Action Level TWA and EL measurements and should be used concurrently with Method ID-189 (7.8.) which is applicable for TWA PEL measurements at the target level of 5 ug/m³. The analytical method does not differentiate between cadmium fume and cadmium dust samples. It also does not differentiate between elemental cadmium and its compounds.

1.2. Principle

Airborne elemental cadmium and cadmium compounds are collected on a 0.8-um mixed cellulose ester membrane filter (MCEF). The air filter samples are wet-ashed with concentrated nitric acid to destroy the organic matrix and dissolve the cadmium analytes. Before the samples are diluted with deionized water, a small amount of concentrated hydrochloric acid is added to help dissolve other metals which may be present. Aliquots of each sample and a matrix modifier are injected into the graphite tube of an atomic absorption spectrophotometer/graphite furnace assembly for analysis of elemental cadmium. The matrix modifier is added to stabilize the cadmium metal and eliminate sodium chloride as an interference during the high temperature charring step of the analysis (7.1., 7.2.).

1.3. History

Previously, two OSHA sampling and analytical procedures for cadmium were used concurrently (7.3., 7.4.). Both of these procedures also required 0.8-um mixed cellulose ester membrane filters for the collection of air samples. These cadmium air filter samples were analyzed by either flame atomic absorption spectroscopy (AAS) (7.3.) or inductively coupled plasma/atomic emission spectroscopy (ICP) (7.4.). A new flame AAS method (7.8.) for the analysis of cadmium is similar to the old procedure given in the General Metals Method ID-121 (7.3.) with some modifications. None of these analytical methods are sensitive enough for measuring workplace exposure to airborne cadmium at the lower Action Level TWA and STEL concentration levels.

1.4. Properties (7.5.)

Elemental cadmium is a silver-white, blue-tinged, lustrous metal which is easily cut with a knife. It is slowly oxidized by moist air to form cadmium oxide. It is insoluble in water, but reacts readily with dilute nitric acid. Some of the physical properties and other descriptive information of elemental cadmium are given below:

CAS No.	7440-43-9
Atomic number	48
Atomic symbol	Cd
Atomic weight	112.41
Melting point	321 °C
Boiling point	765 °C
Density	8.65 g/mL (25 °C)

The properties of specific cadmium compounds are described in reference 7.5.

2. Detection Limit (7.6.)

2.1. The qualitative detection limit for the analytical procedure is 2 ng cadmium for a 10 mL solution volume. This corresponds to 0.07 μg/m³ for a 30 L air volume.

2.2. The quantitative detection limit for the analytical procedure is 10 ng cadmium for a 10 mL solution volume. This corresponds to 0.33 μg/m³ for a 30 L air volume.

3. Precision and Accuracy (7.6.)

The average recovery of eighteen spiked MCEF samples containing cadmium in the range of 0.5 to 2.0 times the Action Level TWA target concentration of 1 μg/m³ (assuming a 200 L air volume) was 99.4% with a pooled coefficient of variation (CV_1) of 0.074. The average recovery of six spiked MCEF samples containing cadmium at 0.1 times the Action Level TWA target concentration was 97.0% with a coefficient of variation (CV_1) of 0.068.

4. Interferences

There are no known spectral line interferences (7.7.). Background absorption is minimized by using a deuterium arc or Zeeman background corrector and the addition of a matrix modifier.

5. Sampling

5.1. Apparatus

5.1.1. Filter cassette unit for air sampling: A 37-mm diameter mixed cellulose ester membrane filter (Millipore type AA or equivalent with a pore size of 0.8-um) contained in a 37-mm polystyrene two- or three-piece cassette filter holder. The filter is supported with a cellulose backup pad. The cassette is sealed with a shrinkable gel band.

5.1.2. A calibrated personal sampling pump whose flow is determined to an accuracy of ± 5% at the recommended flow rate with the filter cassette unit in line.

5.2. Procedure

5.2.1. Sample with the air filter cassettes for elemental cadmium and its compounds in accordance with current instructions in OSHA directives to the industrial hygienist.

5.2.2. Collect air samples at a flow rate of 2.0 L/min. A full-shift (at least seven hours) sample is recommended for Action Level TWA measurements with a maximum air volume of 960 L, if the filter does not become overloaded. A 15 min sample is recommended for STEL level measurements with a minimum suggested air volume of 30 L.

5.2.3. Replace the end plugs into the filter cassettes immediately after sampling.

5.2.4. Securely wrap each sample filter cassette end-to-end with an OSHA Form 21 sample seal.

5.2.5. Submit at least one blank sample with each set of air samples. The blank sample should be handled the same as the other samples except that no air is drawn through it.

5.2.6. Ship the samples to the laboratory for analysis as soon as possible in a suitable container designed to prevent damage in transit.

6. Analytical Procedure

6.1. Apparatus

6.1.1. Atomic absorption spectrophotometer (Perkin Elmer Model 5000 or its equivalent) equipped with a deuterium arc background corrector.

6.1.2. Graphite furnace (Perkin Elmer Model HGA-500 or its equivalent).

6.1.3. Auto sampler (Perkin Elmer Model AS-40 or its equivalent) or autopipets for accurately injecting 10- and 20-μL sample aliquots into the graphite furnace tube.

6.1.4. Digital printer (Perkin Elmer Model PRS-10 or its equivalent).

6.1.5. Strip chart recorder (Perkin Elmer Model 56 or its equivalent).

6.1.6. Inert purge gas for graphite furnace: compressed gas cylinder of purified argon.

6.1.7. Two gauge, two-stage pressure regulator for the argon gas cylinder.

6.1.8. Cadmium hollow cathode lamp or electrodeless discharge lamp and power supply.

6.1.9. Graphite tubes, pyrolytically coated.

6.1.10. Hot plate, capable of reaching 150 °C.

6.1.11. 125 mL Phillips beakers.

6.1.12. Bottles, narrow-mouth, polyethylene or glass with leakproof caps: used for storage of standards and matrix modifier.

6.1.13. Volumetric flasks, volumetric pipets, beakers and other associated general laboratory glassware.

6.1.14. 2.0-2.5 mL polyethylene sample cups for use with the auto sampler.

6.1.15. Forceps.

6.2. Reagents

All reagents should be ACS analytical reagent grade or better.

6.2.1. Deionized water with a resistivity of at least 200k ohms.

6.2.2. Concentrated nitric acid, HNO_3.

6.2.3. Concentrated hydrochloric acid, HC1.

6.2.4. Ammonium phosphate, monobasic, $NH_4H_2PO_4$.

6.2.5. Magnesium nitrate, $Mg(NO_3)_2$.

6.2.6. Diluting solution (4% HNO_3, 0.4% HC1): Add 40 mL HNO_3 and 4 mL HC1 carefully to approximately 500 mL deionized water and then dilute to 1000 mL with deionized water.

6.2.7. 1000 ug/mL cadmium standard stock solution: Use a commercially available certified 1000 ug/mL cadmium standard or, alternatively, dissolve 1.0000 g of cadmium metal in a minimum volume of 1:1 HC1 and dilute to 1 L with 4% HNO_3.

6.2.8. Matrix modifier: Dissolve 1.0 g $NH_4H_2PO_4$ and 0.15 g $Mg(NO_3)_2$ in approximately 200 mL deionized water. Add 1 mL HNO_3 and then dilute to 500 mL with deionized water.

6.3. Safety Precautions

6.3.1. Wear safety glasses and gloves at all times.

6.3.2. Handle acid solutions with care. Handle all cadmium samples and solutions with extra care. Avoid their direct contact with work area surfaces, eyes, skin and clothes. Flush acid solutions which contact the skin or eyes with copious amounts of water.

6.3.3. Perform all acid digestions and acid dilutions in a fume hood.

6.3.4. Exercise care when using laboratory glassware. Do not use chipped pipets, volumetric flasks, beakers or any glassware with sharp edges exposed in order to avoid the possibility of cuts or abrasions.

6.3.5. Never pipet by mouth.

6.3.6. Refer to the instrument instruction manuals for proper and safe operation of the atomic absorption spectrophotometer, graphite furnace and associated equipment.

6.3.7. Since metallic elements and other toxic substances are vaporized during graphite furnace operation, it is imperative that an exhaust vent be used. Always ensure that the exhaust system is operating properly during instrument use.

6.4. Glassware Preparation.

6.4.1. Clean the Phillips beakers by refluxing with 1:1 nitric acid on a hot plate in a fume hood. Thoroughly rinse with deionized water and then invert the beakers to allow them to drain dry.

6.4.2. Rinse volumetric flasks and all other glassware with 10% nitric acid and deionized water prior to use.

6.5. Standard Preparation

6.5.1. Prepare 10, 100 and 1000 ng/mL cadmium working standard stock solutions by making appropriate ten-fold serial dilutions of the 1000 μg/mL cadmium standard stock solution with the diluting solution described in Section 6.2.6.

6.5.2. Prepare cadmium standards to be analyzed in the range of 1.0 to 40 ng/mL by making appropriate serial dilutions of the working standards with the same diluting solution. A suggested method of preparation of these standards is given in Table I. Store these standard solutions in the narrow-mouth polyethylene or glass bottles with leakproof caps. Prepare fresh daily.

6.6. Sample Preparation

6.6.1. Carefully transfer each sample filter with forceps from its filter cassette unit to a clean, separate 125-mL Phillips beaker along with any loose dust found in the cassette. Label each Phillips beaker with the appropriate sample number.

6.6.2. Add 5 mL of concentrated nitric acid to each Phillips beaker containing an air filter sample. Place the Phillips beakers on a hot plate in a fume hood and heat the samples until approximately 1 mL remains. The sample solution in each Phillips beaker should become clear. If it does not, wet-ash the sample with another portion of concentrated nitric acid.

6.6.3. After completing the HNO_3 digestion and cooling the samples, add 40 uL of concentrated HCl to each air sample solution. Swirl and then gently warm the contents of each Phillips beaker.

6.6.4. Quantitatively transfer each cooled air sample solution from its Phillips beaker to a clean 10-mL volumetric flask. Dilute each flask to volume with deionized water and then mix well.

6.7. Analysis

Initially analyze all of the air samples for their cadmium content by flame atomic absorption spectroscopy (AAS) according to OSHA method ID-189 (7.8.). If the concentration of cadmium in a sample is less than 0.04 μg/mL (40 ng/mL), proceed with the graphite furnace AAS analysis of the sample as described below.

The mentioned instrument settings are for the specific instrument models used in the OSHA laboratory. These settings may vary when using other systems.

6.7.1. Set up the atomic absorption spectrophotometer with the deuterium arc background corrector, graphite furnace and auto sampler for flameless atomic absorption analysis of cadmium according to the manufacturer's operational instructions. Use of pyrolytically coated graphite tubes is recommended. For the source lamp, use a hallow cathode or electrodeless discharge cadmium lamp operated at the manufacturer's recommended rating for continuous operation.

6.7.2. Make the following initial instrument settings on the Perkin Elmer Model 5000 spectrophotometer:

Slit = Low
Slitwidth = 0.2nm
Wavelength = 228.8 nm
Mode = AA-BG/ABS
Signal = Cont
Integration Time = 0.5 Sec
Range = UV

6.7.3. The settings for the graphite furnace parameters may vary according to the condition of the graphite tube and other factors. The recommended initial settings for the Perkin Elmer Model HGA-500 graphite furnace are given in Table II.

6.7.4. Turn on the strip chart recorder by setting the power switch to the SERVO position. Set the CHART SPEED to 20 mm/minute and the CHART RANGE to 10 mV.

6.7.5. Optimize the energy reading of the spectrophotometer at 228.8 nm by adjusting the lamp position according to the manufacturer's instructions. Minimize the ABSORBANCE reading by aligning the graphite furnace assembly in the light beam of the spectrophotometer according to the manufacturer's instructions.

6.7.6. Autozero the spectrophotometer and run the instrument until the baseline stops drifting.

6.7.7. Autozero the spectrophotometer once again and use the ZERO control on the chart recorder to set the pen to approximately 1 cm above the zero line on the paper.

6.7.8. Reset the MODE parameter on the spectrophotometer to PEAK HT/REC ABS or PEAK AREA/REC ABS and the INTEGRATION TIME to 6.0 SEC. Turn on the digital printer and date and label the printer paper.

6.7.9. Inject a 20 μL aliquot of the standard, sample or reagent blank solution to be analyzed into the graphite tube followed by a 10 uL overlay of the matrix modifier. Analyze the aliquot and record and label the peak height or peak area absorbance reading measured on the printer paper. Label the peak on the chart paper.

6.7.10. It is recommended that a high standard be analyzed two or three times to check for reproducibility and sensitivity before starting the analysis of a set of samples.

6.7.11. It is also recommended that the entire series of standards be analyzed at the beginning and end of the analysis of a set of samples to ensure that the standard readings are reproducible. Also, analyze a standard after every five or six samples to monitor the performance of the system.

6.7.12. Bracket the sample readings with standards during the analysis. If the absorbance reading of a sample is above the absorbance reading of the highest standard, dilute the sample with the diluting solution, reanalyze and use the appropriate dilution factor in the calculations.

6.7.13. Repeat the analysis of approximately 10% of the samples for a check of precision.

6.7.14. Record the final instrument settings on the chart paper output at the end of the analysis. Date and label the chart paper.

6.8. Calculations

6.8.1. Use a least squares regression program to plot a calibration curve of peak height or peak area absorbance reading versus the concentration (ng/mL) of cadmium in each standard.

6.8.2. Determine the concentration (ng/mL) of cadmium, C, corresponding to the peak height or peak area absorbance reading in each analyzed sample from the resulting calibration curve.

6.8.3. Calculate the total amount (ng) of cadmium, W, in each sample from the sample solution volume (mL):

W = (C)(sample vol, mL)(DF)

Where: DF = Dilution Factor (use only if applicable)

6.8.4. Make a blank correction for each air sample by subtracting the total amount of cadmium in the corresponding blank sample from the total amount of cadmium in the sample.

6.8.5. Calculate the concentration of cadmium in an air sample in unts of $\mu g/m^3$ or mg/m^3 by using one of the following equations:

$\mu g/m^3 = W_{bc}/(\text{Air vol sampled, L})$

or

$mg/m^3 = W_{bc}/[(\text{Air vol sampled, L})(1000\ ng/\mu g)]$

Where: W_{bc} = blank corrected total amount (ng) of cadmium in the sample.

7. References:

7.1. Slavin, W. *Graphite Furnace AAS—A Source Book;* Perkin-Elmer Corp., Spectroscopy Div.: Ridgefield, CT, 1984; p. 18 and pp. 83–90.

7.2. Grosser, Z., Ed.; *Techniques in Graphite Furnace Atomic Absorption Spectrophotometry;* Perkin-Elmer Corp., Spectroscopy Div.; Ridgefield, CT, 1985; p. 106.

7.3. *OSHA Analytical Methods Manual;* U.S. Department of Labor, Occupational Safety and Health Administration, OSHA Analytical Laboratory, Salt Lake City, UT; Am. Conf. of Governmental Ind. Hyg. (ACGIH): Cincinnati, OH, 1985; Method ID–121.

7.4. *OSHA Analytical Methods Manual;* U.S. Department of Labor, Occupational Safety and Health Administration, OSHA Analytical Laboratory, Salt Lake City, UT; Am. Conf. of Governmental Ind. Hyg. (ACGIH): Cincinnati, OH, 1985; Method ID–125.

7.5. Windholz, M., Ed.; *The Merck Index,* 10th ed.; Merck & Co.: Rahway, NJ 1983.

7.6. *Backup Data Report for Cadmium (GF–AAS), Method ID–189GF,* Inorganic Division, OSHA Analytical Laboratory, Salt Lake City, UT, 1988.

7.7. *Analytical Methods for Atomic Absorption Spectrophotometry,* The Perkin-Elmer Corporation: Norwalk, CT, 1973.

7.8. *Cadmium in Workplace Atmospheres (Flame AAS), Method ID–189,* Inorganic Division, OSHA Analytical Laboratory, Salt Lake City, UT, 1988.

TABLE I.—CD STANDARD PREPARATION

Standard (ng/mL)	mL stock	Stock solution (ng/mL)	Final vol (mL)
1.0	10	10	100
2.0	20	10	100
5.0	5	100	100
10.0	10	100	100
20.0	20	100	100
30.0	30	100	100
40.0	4	1000	100

TABLE II.—GRAPHITE FURNACE PARAMETERS

[Perkin Elmer Model HGA-500]

Step (sec)	Ramp time (sec)	Hold time (sec)	Temp (°C)	Argon flow (mL/min)	Record	Read (sec)	BOC (sec)
1	15	20	100	300			
2	15	45	500	300			
3	0	8	20	300			1
4	0	8	2100	250	−10	0	
5	0	20	20	300			
6	0	8	2500	300			

BOC = Background Offset Correction.

Appendix–E2

Method Number: ID–189GF (Proposed)

Applicability: This method is applicable for TWA PEL, Action Level TWA and EL measurements and should be used concurrently with Method ID–189 (7.8)

Matrix: Air

OSHA Standards: 1.0 $\mu g/m^3$ (TWA)
0.5 $\mu g/m^3$ (Action Level TWA)
5.0 $\mu g/m^3$ (EL)

Collection Procedure: A known volume of air is drawn through a 37-mm diameter filter cassette containing a 0.8-um mixed cellulose ester membrane filter (MCEF)

Recommended Air Volumes: 200 L to 960 L for TWA and Action Level TWA 30 L for EL

Recommended Sampling Rate: 2.0 L/min

Analytical Procedure: Air filter samples are wet-ashed with nitric acid. After digestion, a small amount of hydrochloric acid is added. The samples are then diluted to volume with deionized water and analyzed by flameless atomic absorption spectroscopy using a heated graphite furnace atomizer

Validation Range: 0.1 $\mu g/m^3$ to 2.0 $\mu g/m^3$ for a 200 L air volume

Quantitative Detection Limit: 0.33 $\mu g/m^3$ for a 30 L air sample

Precision: $(CV_1) = 0.074$

Method Classification: Validated ID–189GF

Cadmium in Workplace Atmospheres (GF–AAS)

1. Introduction

1.1. Scope

This method describes the collection of airborne elemental cadmium and cadmium compounds on 0.8-um mixed cellulose ester membrane filters and their subsequent analysis by flameless atomic absorption spectroscopy using a heated graphite furnace atomizer. It is applicable for TWA PEL, Action Level TWA and EL measurements and should be used concurrently with Method ID–189 (Reference 7.8) which is applicable for TWA measurements at twice the target level of 1 $\mu g/m^3$ or greater (assuming a 200 L air volume). The analytical method does not differentiate between cadmium fume and cadmium dust. It also does not differentiate between elemental cadmium and its compounds.

1.2. Principle

Airborne elemental cadmium and cadmium compounds are collected on a 0.8-um mixed cellulose ester membrane filter (MCEF). The air filter samples are wet-ashed with concentrated nitric acid to destroy the organic matrix and dissolve the cadmium analytes. Before the samples are diluted with deionized water, a small amount of concentrated hydrochloric acid is added to help dissolve other metals which may be present. Aliquots of each sample and a matrix modifier are injected into the graphite tube of an atomic absorption spectrophotometer/graphite furnace assembly for analysis of elemental cadmium. The matrix modifier is added to stabilize the cadmium metal and eliminate sodium chloride as an interference during the high temperature charring step of the analysis (7.1., 7.2.).

1.3. History

Previously, two OSHA sampling and analytical procedures for cadmium were used concurrently (7.3., 7.4.). Both of these procedures also required 0.8-um mixed cellulose ester membrane filters for the collection of air samples. These cadmium air filter samples were analyzed by either flame atomic absorption spectroscopy (AAS) (7.3.) or inductively coupled plasma/atomic

emission spectroscopy (ICP) (7.4.). A new flame AAS method (7.8.) for the analysis of cadmium is similar to the old procedure given in the General Metals Method ID–121 (7.3.) with some modifications. None of these analytical methods are sensitive enough for measuring workplace exposure to airborne cadmium at the lower TWA PEL, Action level TWA and EL concentration levels. Elemental cadmium is a silver-white, blue-tinged, lustrous metal which is easily cut with a knife. It is slowly oxidized by moist air to form cadmium oxide. It is insoluble in water, but reacts readily with dilute nitric acid. Some of the physical properties and other descriptive information of elemental cadmium are given below:

CAS No. 7440–43–9
Atomic Number 48
Atomic Symbol Cd
Atomic Weight 112.41
Melting Point 321 °C
Boiling Point 765 °C
Density 8.65 g/mL (25 °C)

The properties of specific cadmium compounds are described in reference 7.5.

2. Detection Limit (7.6.)

2.1. The qualitative detection limit for the analytical procedure is 2 ng cadmium for a 10 mL solution volume. This corresponds to 0.07 $\mu g/m^3$ for a 30 L air volume.

2.2 The quantitative detection limit for the analytical procedure is 10 ng cadmium for a 10 mL solution volume. This corresponds to 0.33 $\mu g/m^3$ for a 30 L air volume.

3. Precision and Accuracy (7.6.)

The average recovery of eighteen spiked MCEF samples containing cadmium in the range of 0.5 to 2.0 times the TWA target concentration of 1 $\mu g/m^3$ (assuming a 200 L air volume) was 99.4% with a pooled coefficient of variation (CV_1) of 0.074. The average recovery of six spiked MCEF samples containing cadmium at 0.1 times the TWA target concentration was 97.0% with a coefficient of variation (CV_1) of 0.068.

4. Interferences

There are no known spectral line interferences (7.7.). Background absorption is minimized by using a deuterium arc or Zeeman background corrector and the addition of a matrix modifier.

5. Sampling

5.1. Apparatus

5.1.1. Filter cassette unit for air sampling: A 37-mm diameter mixed cellulose ester membrane filter (Millipore type AA or equivalent with a pore size of 0.8-um) contained in a 37-mm polystyrene two- or three-piece cassette filter holder. The filter is supported with a cellulose backup pad. The cassette is sealed with a shrinkable gel band.

5.1.2. A calibrated personal sampling pump whose flow is determined to an accuracy of ±5% at the recommend flow rate with the filter cassette unit in line.

5.2. Procedure

5.2.1. Sample with the air filter cassettes for elemental cadmium and its compounds in accordance with current instructions in OSHA directives to the industrial hygienist.

5.2.2. Collect air samples at a flow rate of 2.0 L/min. A full-shift (at least seven hours) sample is recommended for TWA and Action Level TWA measurements with a maximum air volume of 960 L, if the filter does not become overloaded. A 15 min sample is recommended for EL level measurements with a minimum suggested air volume of 30 L.

5.2.3. Replace the end plugs into the filter cassettes immediately after sampling.

5.2.4. Securely wrap each sample filter cassette end-to-end with an OSHA Form 21 sample seal.

5.2.5. Submit at least one blank sample with each set of air samples. The blank sample should be handled the same as the other samples except that no air is drawn through it.

5.2.6. Ship the samples to the laboratory for analysis in a suitable container designed to prevent damage in transit.

6. Analytical Procedure

6.1. Apparatus

6.1.1. Atomic absorption spectrophotometer (Perkin Elmer Model 5000 or its equivalent) equipped with a deuterium arc background corrector.

6.1.2. Graphite furnace (Perkin Elmer Model HGA–500 or its equivalent).

6.1.3. Auto sampler (Perkin Elmer Model AS–40 or its equivalent) or autopipets for accurately injecting 10- and 20-uL sample aliquots into the graphite furnace tube.

6.1.4. Digital printer (Perkin Elmer Model PRS–10 or its equivalent).

6.1.5. Strip chart recorder (Perkin Elmer Model 56 or its equivalent).

6.1.6. Inert purge gas for graphite furnace: compressed gas cylinder of purified argon.

6.1.7. Two gauge, two-stage pressure regulator for the argon gas cylinder.

6.1.8. Cadmium hollow cathode lamp or electrodeless discharge lamp and power supply.

6.1.9. Graphite tubes, pyrolytically coated.

6.1.10. Hot plate, capable of reaching 150 °C.

6.1.11. 125 mL Phillips beakers.

6.1.12. Bottles, narrow-mouth, polyethylene or glass with leakproof caps: used for storage of standards and matrix modifier.

6.1.13. Volumetric flasks, volumetric pipets, beakers and other associated general laboratory glassware.

6.1.14. 2.0–2.5 mL polyethylene same cups for use with the auto sampler.

6.1.15. Forceps.

6.2 Reagents

All reagents should be ACS analytical reagent grade or better.

6.2.1. Deionized water with a resistivity of at least 200k ohms.

6.2.2. Concentrated nitric acid, HNO_3.

6.2.3. Concentrated hydrochloric acid, HCl.

6.2.4. Ammonium phosphate, monobasic, $NH_4H_2PO_4$.

6.2.5. Magnesium nitrate, $Mg(NO_3)_2$.

6.2.6. Diluting solution (4 percent HNO_3, 0.4 percent HCl): Add 40 mL HNO_3 and 4 mL HCl carefuly to approximately 500 mL deionized water and then dilute to 1000 μmL with deionized water.

6.2.7. 1000 $\mu g/mL$ cadmium standard stock solution: Use a commercially available certified 1000 $\mu g/mL$ cadmium standard or, alternatively, dissolve 1.0000 g of cadmium metal in a minimum volume of 1:1 HCl and dilute to 1 L with 4 percent HNO_3.

6.2.8. Matrix modifier: Dissolve 1.0 g $NH_4H_2PO_4$ and 0.15 g $Mg(NO_3)_2$ in approximately 200 mL deionized water. Add 1 mL HNO_3 and then dilute to 500 ml with deionized water.

6.3. Safety Precautions

6.3.1. Wear safety glasses and gloves at all times.

6.3.2. Handle acid solutions with care. Handle all cadmium samples and solutions with extra care. Avoid their direct contact with work area surfaces, eyes, skin and clothes. Flush acid solutions which contact the skin or eyes with copious amounts of water.

6.3.3. Perform all acid digestions and acid dilutions in a fume hood.

6.3.4. Exercise care when using laboratory glassware. Do not use chipped pipets, volumetric flasks, beakers or any glassware with sharp edges exposed in order to avoid the possibility of cuts or abrasions.

6.3.5. Never pipet by mouth.

6.3.6. Refer to the instrument instruction manuals for proper and safe operation of the atomic absorption spectrophotometer, graphite furnace and associated equipment.

6.3.7. Since metallic elements and other toxic substances are vaporized during graphite furnace operation, it is imperative that an exhaust vent be used. Always ensure that the exhaust system is operating properly during instrument use.

6.4. Glassware Preparation

6.4.1. Clean the Phillips beakers by refluxing with 1:1 nitric acid on a hot plate in a fume hood. Thoroughly rinse with deionized water and then invert the beakers to allow them to drain dry.

6.4.2. Rinse volumetric flasks and all other glassware with 10-percent nitric acid and deionized water prior to use.

6.5. Standard Preparation

6.5.1. Prepare 10, 100 and 1000 ng/mL cadmium working standard stock solutions by making appropriate ten-fold serial dilutions of the 1000 $\mu g/mL$ cadmium standard stock solution with the diluting solution described in Section 6.2.6.

6.5.2. Prepare cadmium standards to be analyzed in the range of 1.0 to 40 ng/mL by making appropriate serial dilutions of the working standards with the same diluting solution. A suggested method of preparation of these standards is given in Table I. Store these standard solutions in the narrow-mouth polyethylene or glass bottles with leakproof caps. Prepare fresh daily.

6.6. Sample Preparation

6.6.1. Carefully transfer each sample filter with forceps from its filter cassette unit to a clean, separate 125-mL et-ash the sample with another portion of concentrated nitric acid.

6.6.3. After completing the HNO_3 digestion and cooling the samples, add 40 μL of concentrated HCl to each air sample solution. Swirl and then gently warm the contents of each Phillips beaker.

6.6.4. Quantitatively transfer each cooled air sample solution from its Phillips beaker to a clean 10-mL volumetric flask. Dilute each flask to volume with deionized water and then mix well.

6.7. Analysis

Initially analyze all of the air samples for their cadmium content by flame atomic absorption spectroscopy (AAS) according to OSHA Method ID-189 (7.8.). If the concentration of cadmium in a sample is less than 0.04 μg/mL (40 ng/mL), proceed with the graphite furnace AAS analysis of the sample as described below. The mentioned instrument settings are for the specific instrument models used in the Salt Lake City Analytical Laboratory (SLCAL). These settings may vary when using other systems.

6.7.1. Set up the atomic absorption spectrophotometer with the deuterium arc background corrector, graphite furnace and auto sampler for flameless atomic absorption analysis of cadmium according to the manufacturer's operational instructions. Use of pyrolytically coated graphite tubes is recommended. For the source lamp, use a hollow cathode or electrodeless discharge cadmium lamp operated at the manufacturer's recommended rating for continuous operation.

6.7.2. Make the following initial instrument settings on the Perkin Elmer Model 5000 spectrophotometer:

Slit=Low
Slitwidth=0.2nm
Wavelength=228.8 nm
Mode=AA-BG/ABS
Signal=Cont
Integration Time=0.5 SEC
Range=UV

6.7.3. The settings for the graphite furnace parameters may vary according to the condition of the graphite tube and other factors. The recommended initial settings for the Perkin Elmer Model HGA-500 graphite furnace are given in Table II.

6.7.4. Turn on the strip chart recorder by setting the power switch to the SERVO position. Set the CHART SPEED to 20 mm/minute and the CHART RANGE to 10 mV.

6.7.5. Optimize the energy reading of the spectrophotometer at 228.8 nm by adjusting the lamp position according to the manufacturer's instructions. Minimize the ABSORBANCE reading by aligning the graphite furnace assembly in the light beam of the spectrophotometer according to the manufacturer's instructions.

6.7.6. Autozero the spectrophotometer and run the instrument until the baseline stops drifting.

6.7.7. Autozero the spectrophotometer once again and use the ZERO control on the chart recorder to set the pen to approximately 1 cm above the zero line on the paper.

6.7.8. Reset the MODE parameter on the spectrophotometer to PEAK HT/REC ABS or PEAK AREA/REC ABS and the INTEGRATION TIME to 6.0 SEC. Turn on the digital printer and date and label the printer paper.

6.7.9. Inject a 20 μL aliquot of the standard, sample or reagent blank solution to be analyzed into the graphite tube followed by a 10 μL overlay of the matrix modifier. Analyze the aliquot and record and label the peak height or peak area absorbance reading measured on the printer paper. Label the peak on the chart paper.

6.7.10. It is recommended that a high standard be analyzed two or three times to check for reproducibility and sensitivity before starting the analysis of a set of samples.

6.7.11. It is also recommended that the entire series of standards be analyzed at the beginning and end of the analysis of a set of samples to ensure that the standard readings are reproducible. Also, analyze a standard after every five or six samples to monitor the performance of the system.

6.7.12. Bracket the sample readings with standards during the analysis. If the absorbance reading of a sample is above the absorbance reading of the highest standard, dilute the sample with the diluting solution, reanalyze and use the appropriate dilution factor in the calculations.

6.7.13. Repeat the analysis of approximately 10 percent of the samples for a check of precision.

6.7.14. Record the final instrument settings on the chart paper output at the end of the analysis. Date and label the chart paper.

6.8. Calculations

6.8.1. Use a least squares regression program to plot a calibration curve of peak height or peak area absorbance reading versus the concentration (ng/mL) of cadmium in each standard.

6.8.2. Determine the concentration (ng/mL) of cadmium, C, corresponding to the peak height or peak area absorbance reading in each analyzed sample from the resulting calibration curve.

6.8.3. Calculate the total amount (ng) of cadmium, W, in each sample from the sample solution volume (mL):

W=(C) (sample vol, mL)(DF)

Where: DF=Dilution Factor (use only if applicable)

6.8.4. Make a blank correction for each air sample by subtracting the total amount of cadmium in the corresponding blank sample from the total amount of cadmium in the sample.

6.8.5. Calculate the concentration of cadmium in an air sample in units of $\mu g/m^3$ or mg/m^3 by using one of the following equations:

$\mu g/m^3 = W_{bc}/(\text{Air vol sampled, L})$, or

$mg/m^3 = W_{bc}/[(\text{Air vol sampled, L})(1000\ ng/\mu g)]$

Where: W_{bc}=blank corrected total amount (ng) of cadmium in the sample.

7. References:

7.1. Slavin, W. *Graphite Furnace AAS—A Source Book;* Perkin-Elmer Corp., Spectroscopy Div.: Ridgefield, CT, 1984; p. 1 and pp. 83–90.

7.2. Grosser, Z., Ed.; *Techniques in Graphite Furnace Atomic Absorption Spectrophotometry;* Perkin-Elmer Corp., Spectroscopy Div.: Ridgefield, CT, 1985; p. 106.

7.3. *OSHA Analytical Methods Manual;* U.S. Department of Labor, Occupational Safety and Health Administration, OSHA Analytical Laboratory, Salt Lake City, UT; Am. Conf. of Governmental Ind. Hyg. (ACGIH): Cincinnati, OH, 1985; Method ID-121.

7.4. *OSHA Analytical Methods Manual;* U.S. Department of Labor, Occupational Safety and Health Administration, OSHA Analytical Laboratory, Salt Lake City, UT; Am. Conf. of Governmental Ind. Hyg. (ACGIH): Cincinnati, OH, 1985; Method ID-125.

7.5. Windholz, M., Ed.; *The Merck Index,* 10th ed.; Merck & Co.: Rahway, NJ, 1983.

7.6. *Backup Data Report for Cadmium (GF-AAS), Method ID-189GF,* Inorganic Division, OSHA Analytical Laboratory, Salt Lake City, UT, 1988.

7.7. *Analytical Methods for Atomic Absorption Spectrophotometry,* The Perkin-Elmer Corporation: Norwalk, CT, 1973.

7.8. *Cadmium in Workplace Atmospheres (Flame AAS), Method ID-189,* Inorganic Division, OSHA Analytical Laboratory, Salt Lake City, UT, 1988.

TABLE I.—CD STANDARD PREPARATION

Standard (ng/ml)	mL Stock	Stock solution (ng/mL)	Final vol (mL)
1.0	10	10	100
2.0	20	10	100
5.0	5	100	100
10.0	10	100	100
20.0	20	100	100
30.0	30	100	100
40.0	4	1000	100

TABLE II.—GRAPHITE FURNACE PARAMETERS

[Perkin Elmer Model HGA-500]

Step	Ramp time (sec)	Hold time (sec)	Temp (°C)	Argon flow (ml/min)	Record (sec)	Read (sec)	BOC (sec)
1	15	20	100	300	.....	.....	.....
2	15	45	500	300	.....	.....	.....
3	0	8	20	300	.....	.....	1
4	0	8	2100	250	−10	0	.....
5	0	20	20	300	.....	.....	.....
6	0	8	2500	300	.....	.....	.....

BOC=Background Offset Correction

Cadmium in Workplace Atmospheres (Flame AAS)

Method Number: ID-189.

Applicability: This method is applicable for TWA measurements at twice the target level of 1.0 $\mu g/m^3$ or greater (assuming a 200 L air volume) and should be used concurrently with Method ID-189F (7.6.) which is applicable for TWA PEL, Action Level TWA and EL measurements.

Matrix: Air.

OSHA Standards:

1 $\mu g/m^3$ (TWA PEL)
0.5 $\mu g/m^3$ (Action Level TWA)
5 $\mu g/m^3$ (EL)

Collection Procedure: A known volume of air is drawn through a 37-mm diameter filter cassette containing a 0.8-um mixed cellulose ester membrane filter (MCEF).

Recommended Air Volume: 200 L to 960 L.

Recommended Sampling Rate: 2.0 L/min.

Analytical Procedure: Air filter samples are wet-ashed with nitric acid. After digestion, a small amount of hydrochloric acid is added. The samples are then diluted to volume with dionized water and analyzed by atomic absorption spectroscopy with an oxidizing air/acetylene flame.

Validation Range: 5.0 $\mu g/m^3$ to 20.0 $\mu g/m^3$ for a 200 L air volume.

Quantitative Detection Limit: 1 $\mu g/m^3$ for a 200 L air sample.

Precision: (CV_1)=0.010.

Method Classification: Validated.

Cadmium in Workplace Atmospheres (Flame AAS)

1. Introduction

1.1. Scope. This method describes the collection of airborne elemental cadmium and cadmium compounds on 0.8-um mixed cellulose ester membrane filters and their subsequent analysis by flame atomic absorption spectroscopy. It is applicable for TWA measurements at twice the target level of 1 $\mu g/m^3$ or greater and should be used concurrently with Method ID-189GF (7.6.) which is applicable for TWA PEL, Action Level TWA and EL measurements. It is also applicable for the collection and analysis of cadmium wipe and bulk material samples. The analytical method does not differentiate between cadmium fume and cadmium dust. It also does not differentiate between elemental cadmium and its compounds.

1.2. Principle. Airborne elemental cadmium and cadmium compounds are collected on a 0.8-um mixed cellulose ester membrane filter (MCEF). The air filter samples are wet-ashed with concentrated nitric acid to destroy the organic matrix and dissolve the cadmium analytes. A small amount of concentrated hydrochloric acid is added to help dissolve other metals which may be present. The samples are diluted with deionized water and then aspirated into the oxidizing air/acetylene flame of an atomic absorption spectrophotometer for analysis of elemental cadmium.

1.3. History: Previously, two OSHA sampling and analytical procedures for cadmium were used concurrently (7.1., 7.2.). Both of these procedures also rquired 0.8-um mixed cellulose ester membrane filters for the collection of air samples. These cadmium air filter samples were analyzed by either flame atomic absorption spectroscopy (AAS) (7.1.) or inductively coupled plasma/atomic emission spectroscopy (ICP (7.2.). The new flame AAS method for the analysis of cadmium is similar to the old procedure given in the General Metals Method ID-121 (7.1.) with some modifications.

1.4. Properties (7.3.): Elemental cadmium is a silver-white, blue-tinged, lustrous metal which is easily cut with a knife. It is slowly oxidized by moist air to form cadmium oxide. It is insoluble in water, but reacts readily with dilute nitric acid. Some of the physical properties and other descriptive information of elemental cadmium are given below:

CAS No.: 7440-43-9
Atomic Number: 48
Atomic Symbol: Cd
Atomic Weight: 112.41
Melting Point: 321 °C
Boiling Point: 765 °C
Density: 8.65 g/mL (25 °C)

The properties of specific cadmium compounds are described in reference 7.3.

2. Detection Limit (7.4.)

2.1. The qualitative detection limit for the analytical procedure is 0.05 μg cadmium for a 10 mL solution volume. This corresponds to 0.25 $\mu g/m^3$ for a 200 L air volume.

2.2. The quantitative detection limit for the analytical procedure is 0.2 μg cadmium for a 10 mL solution volume. This corresponds to 1 $\mu g/m^3$ for a 200 L air volume.

3. Precision and Accuracy

The average recovery of seventeen spiked MCEF samples containing cadmium in the range of 5 to 20 times the TWA target concentration of 1 $\mu g/m^3$ (assuming a 200 L air volume) was 104.0 percent with a pooled coefficient of variation (CV_1) of 0.010 (7.4.).

4. Interferences

There are no known interferences in either sampling or analysis (7.5.).

5. Sampling

5.1. Apparatus

5.1.1. Filter cassette unit for air sampling: A 37-mm diameter mixed cellulose ester membrane filter (Millipore type AA or equivalent with a pore size of 0.8 μm) contained in a 37-mm polystyrene two- or three-piece cassette filter holder. The filter is supported with a cellulose backup pad. The cassette is sealed with a shrinkable gel band.

5.1.2. A calibrated personal sampling pump whose flow is determined to an accuracy of ±5 percent at the recommended flow rate with the filter cassette unit in line.

5.2. Procedure

5.2.1. Sample with the air filter cassettes for elemental cadmium and its compounds in accordance with current instructions in OSHA directives to the industrial hygienist.

5.2.2. Collect air samples at a flow rate of 2.0 L/min. A full-shift (at least seven hours) sample is recommended with a maximum air volume of 960 L, if the filter does not become overloaded. The minimum suggested air volume is 200 L.

5.2.3. Replace the end plugs into the filter cassettes immediately after air sampling.

5.2.4. Securely wrap each sample filter cassette end-to-end with an OSHA Form 21 sample seal.

5.2.5. Submit at least one blank sample with each set of air samples. The blank sample should be handled the same as the other samples except that no air is drawn through it.

5.2.6. Ship the samples to the laboratory for analysis in a suitable container designed to prevent damage in transit.

6. Analytical Procedure

6.1. Apparatus

6.1.1. Atomic absorption spectrophotometer (Perkin Elmer Model 5000 or its equivalent) equipped with a nebulizer and a four inch (one slot) burner head for use with an air-acetylene flame.

6.1.2. Oxidant: compressed air which has been filtered to remove water, oil and other foreign substances.

6.1.3. Fuel: standard commercially available tanks of acetylene dissolved in acetone; tanks should be equipped with flash arresters. CAUTION: Do not use grades of acetylene available from certain suppliers that contain solvents other than acetone which may damage the PVC tubing used in some instruments.

6.1.4. Pressure-reducing valves: two gauge, two-stage pressure regulators to maintain fuel and oxidant pressures somewhat higher than the controlled operating pressures of the instrument.

6.1.5. Cadmium hollow cathode lamp.

6.1.6. Hot plate, capable of reaching 150 °C.

6.1.7. 125 mL Phillips beakers.

6.1.8. Bottles, 500-mL, narrow-mouth, polyethylene or glass with leakproof caps: used for storage of standards.

6.1.9. Volumetric flasks, volumetric pipets, beakers and other associated general laboratory glassware.

6.1.10. Forceps.

6.2. Reagents

All reagents should be ACS analytical reagent grade or better.

6.2.1. Deionized water with a resistivity of at least 200k ohms.

6.2.2. Concentrated nitric acid, HNO_3.

6.2.3. Concentrated hydrochloric acid, HC1.

6.2.4. Diluting solution (4 percent HNO_3, 0.4 percent HC1): Add 40 mL HNO_3 and 4 mL HC1 carefully to approximately 500 mL deionized water and then dilute to 1000 mL with deionized water.

6.2.5. 1000 $\mu g/mL$ cadmium standard stock solution: Use a commercially available certified 1000 $\mu g/mL$ cadmium standard or, alternatively, dissolve 1.0000 g of cadmium metal in a minimum volume of 1:1 HC1 and dilute to 1 L with 4 percent HNO_3.

6.3. Safety Precautions

6.3.1. Wear safety glasses and gloves at all times.

Federal Register

Tuesday
February 6, 1990

Part III

Department of Labor

Office of the Secretary

Women's Bureau; Announcement of Expected Timetable for Issuing SGAs and RFPs in Fiscal Year 1990 and for Making Grant and Contract Awards; Notice

DEPARTMENT OF LABOR

Office of the Secretary

Women's Bureau; Announcement of Expected Timetable for Issuing SGAs and RFPs in Fiscal Year 1990 and for Making Grant and Contract Awards

AGENCY: Office of the Secretary, Women's Bureau, Labor.

ACTION: Notice.

SUMMARY: The Women's Bureau, National Office (Washington, DC), announces an expected timetable for issuing Solicitations for Grant Applications (SGAs) and Requests for Proposals (RFPs) to implement its Fiscal Year (FY) 1990 procurement program. Information is also provided on the expected schedule for making grant and contract awards. This notice is intended to assist prospective Offerors in scheduling the preparation of their proposals and to provide an indication of the approximate start date (*i.e.*, the date of grant or contract award) for the projects to which the SGAs and RFPs refer. It is anticipated that approximately $245,000 will be awarded through these solicitations in FY 1990.

FOR FURTHER INFORMATION CONTACT: Ms. Dora E. Carrington, Chief, Office of Administrative Management, U.S. Department of Labor, Office of the Secretary, Women's Bureau, 200 Constitution Avenue NW., Room S-3305, Washington, DC 20210; telephone number: (202) 523-6606.

SUPPLEMENTARY INFORMATION:

I. Background

The Women's Bureau, National Office, research, demonstration program and technical assistance plan for FY 1990, published in the January 16, 1990, issue of the **Federal Register**, set out the subject areas within which the National Office procurement program would be developed in FY 1990 and provided a general description of the specific projects that may be the subject of SGAs and RFPs issued in FY 1990. This notice provides information on the expected timetable for issuing those solicitations and for making grant and contract awards.

Plans for an annual competition for grant applications sponsored by the Women's Bureau, National Office, were also announced in the January 16, 1990, issue of the **Federal Register**. The expected timetable for issuing the SGA for the annual grant competition will be announced in a **Federal Register** notice planned to be issued in the coming weeks. That notice will provide detailed information on applying for assistance through the annual grant competition.

II. Expected Timetable for Issuing Solicitations and Making Grant and Contract Awards

The timetable for issuing SGAs and RFPs for all projects included in the research, demonstration program and technical assistance plan published in the January 16, 1990, issue of the **Federal Register** is as follows:

Late February 1990	Availability of SGAs and RFPs announced in the *Commerce Business Daily* and/or the **Federal Register**.
Late March 1990	SGAs and RFPs mailed to individuals and organizations who have requested copies of the solicitations in writing.
Late April to early May 1990.	Offerors' proposals due in the Office of Procurement Services, U.S. Department of Labor, Washington, DC
Early June 1990	Evaluation of proposals completed.
Late June 1990	Negotiations conducted for final offers.
Mid-July 1990	Notice of action taken on proposals issued to Offerors.
Late August 1990	Awards made and start of project work authorized.

The above timetable is approximate and is subject to change. Individuals and organizations in responding to the solicitations referred to in this notice should obtain copies of the relevant SGA(s) or RFP(s) when issued. Those SGAs and RFPs will indicate the final timetable for the steps in the procurement process that result in the award of a grant or contract.

Signed at Washington, DC, this 30th day of January 1990.

Debra R. Bowland,

Acting Director, Women's Bureau.

[FR Doc. 90-2534 Filed 2-5-90; 8:45 am]

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federal register

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Part IV

Department of the Interior

Fish and Wildlife Service

50 CFR Part 17

Endangered and Threatened Wildlife and Plants; Determination of Threatened Status for Lesquerella Congesta (Dudley Bluffs Bladderpod) and Physaria Obcordata (Dudley Bluffs Twinpod and the Plant Calyptronoma rivalis; Rules

DEPARTMENT OF THE INTERIOR

Fish and Wildlife Service

50 CFR Part 17

RIN 1018-AB38

Endangered and Threatened Wildlife and Plants; Final Rule To Determine Lesquerella Congesta (Dudley Bluffs Bladderpod) and Physaria Obcordata (Dudley Bluffs Twinpod) To Be Threatened Species

AGENCY: Fish and Wildlife Service, Interior.

ACTION: Final rule.

SUMMARY: The U.S. Fish and Wildlife Service determines two plants, *Lesquerella congesta* (Dudley Bluffs bladderpod) and *Physaria obcordata* (Dudley Bluffs twinpod) from Rio Blanco County, Colorado, to be threatened species under the authority of the Endangered Species Act of 1973, as amended. Both members of the mustard family, these species have been found only in or on the outer edge of the Piceance Basin in Colorado. Both species grow on oil shale outcrops. These species are known from five major populations each, two of which occur together. Most sites are on public land administered by the Bureau of Land Management, with the remainder located on private land or Colorado Division of Wildlife land. Within the Piceance Basin, the two plants occur in the multimineral oil shale zone, an area containing rich deposits of oil shale and sodium minerals (nahcolite and dawsonite). If project designs for development of these deposits do not include plans for conservation of these two mustards, both species could be significantly impacted. The determination that *Lesquerella congesta* and *Physaria obcordata* are threatened species will provide them protection under the authority of the Endangered Species Act of 1973, as amended.

EFFECTIVE DATE: March 8, 1990.

ADDRESSES: The complete file for this rule is available for inspection, by appointment, during normal business hours at the Colorado State Supervisor's Office, Fish and Wildlife Enhancement, 730 Simms Street, Room 290, Golden, Colorado 80401, and at the Western Colorado Fish and Wildlife Enhancement Office, 529 25½ Road, Suite B-113, Grand Junction, Colorado 81505.

FOR FURTHER INFORMATION CONTACT: Mr. John Anderson, botanist, Fish and Wildlife Enhancement, at the Grand Junction address above, (303) 243-2778 or FTS 322-0351.

SUPPLEMENTARY INFORMATION:

Background

Two new species of wild mustards, *Lesquerella congesta* (Dudley Bluffs bladderpod) and *Physaria obcordata* (Dudley Bluffs twinpod), were discovered in 1982 during a floristic inventory of the Piceance Basin conducted by the Colorado Natural Heritage Inventory for the Bureau of Land Management (Bureau)(Colorado Natural Areas Program 1987). An earlier collection of *L. congesta*, unrecognized as such, was made in 1959. They were subsequently described by Dr. Reed Rollins, an expert on the mustard family, who visited the Piceance Basin and observed them at Dudley Bluffs in 1983 (Rollins 1983, Rollins 1984). With the exception of the recently described *Penstemon debilis* (O'Kane and Anderson 1987), these two herbaceous perennials are the rarest of several oil shale plant species in the Piceance Basin.

L. congesta is an extremely small cushion plant only 1-3 centimeters (0.4-1.2 inches) in diameter with fruiting stems up to 1.5 centimeters (0.6 inches) tall. The cushion growth habit is an adaptation to erosive badland soils, which has evolved independently in several unrelated taxa in this area. *L. congesta* has small, linear, entire, silvery leaves 8-13 millimeters (0.3-0.5 inches) long, bright yellow flowers, and rounded, pubescent fruits 2.5-3.5 millimeters (0.10-0.14 inches) wide.

P. obcordata is 12-18 centimeters (4.8-7.2 inches) tall with oblanceolate, entire leaves 1.0-1.5 centimeters (0.4-0.6 inches) wide and 4.0-8.0 centimeters (1.6-3.8 inches) long, with a silvery sheen due to a dense covering of overlapping, dish-shaped trichomes. It has yellow flowers, 7-9 millimeters (0.3-0.4 inches) long, and slightly inflated, heart-shaped (obcordate) fruits.

These two rare mustards grow on barren white outcrops exposed along drainages through erosion from downcutting of streams in the Piceance Basin. Each species, however, has a slightly different microenvironment. While the twinpod grows on steep sideslopes, the bladderpod grows above it on level surfaces at the points of ridges; the bladderpod also occurs by itself where narrow outcrops of level white shale are exposed. Because more sideslope habitat is available (for instance, there is no ridgepoint habitat at Calamity Ridge), the bladderpod is the rarer of the two species.

The strata exposed in the Piceance Basin are derived from the Eocene Green River and Uinta Formations (Cashion and Donnell 1974). The rich, oil-shale-bearing Green River Formation formed as a lacustrine deposit in Lake Uinta, forming fine-textured shale. Later, Lake Uinta filled with sand and silt deposits, which formed the coarser-grained overlying Uinta Formation. Thus, the surface of the Piceance Basin is filled with the Uinta Formation above and the thick shale beds of the Parachute Creek member of the Green River Formation below. The shale rims of the Piceance Basin, such as Calamity Ridge, are formed from upturned strata of the Green River Formation.

At the interface of the two formations, in the middle of the Piceance Basin, the lakebed Green River Formation shale intertongues with the deltaic and fluvial sandstones and siltstones of the Uinta Formation. For instance, at Dudley Bluffs, the type locality of the two species, the ridge and hillside supporting the bladderpod and twinpod is formed by strata of Unit 5 of the Uinta Formation on the top and Unit 4 at the base, with the Thirteen Mile Creek Tongue of the Green River Formation on the midslope where the twinpods grow. The bladderpod only occurs at or near the end of the ridge where erosion has removed the overlying Unit 5 from the point as the ridge recedes. Along Yellow Creek, the Dudley Bluffs bladderpod and twinpod grow primarily on other narrow tongues of white shale within the Uinta Formation, whereas at Calamity Ridge the twinpod grows on outcrops of the Parachute Creek Member of the Green River Formation. Elevational ranges for these species are 1,860-2,010 meters (6,140-6,644 feet) for *L. congesta* and 1,806-2,255 meters (5,960-7,440 feet) for *P. obcordata*. The surrounding hills and mesas support pinyon-juniper woodlands.

In 1986, the Colorado Natural Areas Program followed up on the 1982 inventory by conducting field work on *P. obcordata* to determine its rarity and range (Colorado Natural Areas Program 1987). Sites of *L. congesta* were delineated at the same time. During this survey, populations of both species were found for the first time along Yellow Creek, the next drainage west of Piceance Creek and about 5 miles away. The largest known populations of both species, approximately 10,000 individuals each, were discovered growing together at the junction of Piceance Creek and Ryan Gulch, 2 miles north of Dudley Bluffs. Between the 1982 inventory and the 1986 survey, all major drainages in the Piceance Basin were surveyed. Both species were found only along Piceance and Yellow Creeks, and

the twinpod at Calamity Ridge. During the 1988 field season, the author visited all the wild mustard sites and more precisely delineated their geological habitat.

L. congesta has five populations on approximately 50 total acres over a range of 10 miles. *P. obcordata*, which occurs on outcrops further upstream on Piceance Creek and downstream on Yellow Creek, has a range of 15 miles, plus the two populations on Calamity Ridge, for a total of five major populations on approximately 250 acres. However, the Dudley Bluffs and Ryan Gulch sites, which are only 2 miles apart, contain most members of the species.

The Dudley Bluffs bladderpod and twinpod occur mostly on land administered by the Bureau, with the exception of portions of the Dudley Bluffs site on private land (containing twinpod) and a portion of the Yellow Creek sites on Colorado Division of Wildlife land (containing bladderpod). The Bureau has designated the Federal portions of the Dudley Bluffs site and one of the Calamity Ridge sites as Areas of Critical Environmental Concern (Bureau of Land Management 1987a).

L. congesta and *P. obcordata* grow on tongues of white Green River shale within the overlying Uinta Formation, which is considered overburden to the thick underlying oil shale deposits. Except for the Calamity Ridge sites, all the occurrences are within the multimineral oil shale area. Beneath the overburden of the surface Uinta Formation, this area at the center of the Piceance Basin contains thick, rich sections of oil shale in the mahogany zone and the sodium minerals nahcolite (sodium bicarbonate) and dawsonite (a potential source of alumina) in the underlying saline zone. *L. congesta* and *P. obcordata* are vulnerable to impacts resulting from future development and extraction of these oil shale minerals and associated activities.

Federal action involving these species began on September 27, 1985, when the Fish and Wildlife Service (Service) published a notice of review in the **Federal Register** (45 FR 39526) covering plants being considered for classification as endangered or threatened. *L. congesta* and *P. obcordata* were included in this notice as Category 2 species. Unfortunately, *L. congesta* was erroneously listed as *L. condensata*, a common species. Category 2 comprises taxa for which information now in possession of the Service indicates that proposing to list them as endangered or threatened species is possibly appropriate, but for which substantial data on biological vulnerability and threats are not currently known or on file. The present proposal is based on more current biological data from the Colorado Natural Areas Program (1987).

Section 4(b)(3)(B) of the Endangered Species Act (Act) (16 U.S.C. 1531 *et seq.*), as amended in 1982, requires the Secretary of the Interior to make findings on certain petitions within 1 year of their receipt. All taxa contained in the 1985 notice, including *L. congesta* and *P. obcordata*, were treated as being petitioned on October 11, 1985. In October 1986, October 1987, and October 1988, the Service made the 12-month finding that the petition to list *L. congesta* and *P. obcordata* was warranted, but precluded by other listing actions of higher priority. The Service published a proposed rule to list *L. congesta* and *P. obcordata* as threatened species on January 24, 1989 (54 FR 3499), constituting the next 12-month finding that would have been required on or before October 7, 1989.

Summary of Comments and Recommendations

In the January 24, 1989, proposed rule (54 FR 3499) and associated notifications, all interested parties were requested to submit factual reports or information that might contribute to the development of a final rule. Appropriate State and Federal agencies, county governments, scientific organizations, and other interested parties were contacted and requested to comment. A public hearing was requested by the Rio Blanco County Board of Commissioners (County) and by the Associated Governments of Northwest Colorado on March 9, 1989. On March 28, 1989, the Service published a notice (54 FR 12663) extending the initial comment period to April 26, 1989, to accommodate the requested public hearing which was held on April 13, 1989, in Meeker, Colorado. Newspaper notices announcing the public hearing and the extension of the comment period were published in the Meeker Herald on April 6 and 13, 1989, and in the Rocky Mountain News on April 6 and 7, 1989. At the hearing, a Service botanist read a prepared statement and showed slides of the plants and their habitat. Individuals in the audience were then given the opportunity to present their oral comments. Following the comments there was a question and answer period. Two dozen people attended the public hearing and six presented oral comments. Nine written comments also were received in response to the proposed rule. The State Natural Areas Programs both commented at the public hearing and sent in a separate written comment. Thus, there were 14 comments overall.

Six comments in support were received, including the State, conservation groups, a botany professor, and other interested individuals: six comments in opposition were received from a local (county) government, oil shale and nahcolite companies, and a consulting geologist; and two comments were neutral. Written and oral comments presented at the public hearing and received during the public comment period are covered in the following summary. Comments of similar content are grouped into a number of general issues. These issues and the Service's response to each are discussed below.

Issue 1: Oil shale and nahcolite companies questioned the observed rarity of the species. In their view, there was a possibility that the plants might be more common than currently known and, therefore, not qualify for threatened or endangered status. Their rationale was as follows:

First, there are large areas of oil shale outcrops outside the Piceance Basin in Colorado, Utah, and Wyoming that may contain the two wild mustards.

Second, the adequacy of knowledge of their range and, hence, rarity was questioned based upon an inadequate knowledge of their geologic habitat; therefore, they could occur elsewhere in other habitats.

Third, the adequacy of inventory for these species was questioned based on the amount of time spent and the large areas of the Piceance Basin to be covered.

Response: Based on the extensive evidence gathered to date, it is unlikely that these particular species of wild mustards will be found outside the Piceance Basin.

First, evolution in these genera is characterized by local endemism. Rugged topography and varied geologic substrates led to population isolation which, in turn, resulted in the evolution of localized species with restricted distribution, rather than several ecotypes of one common species. For example, other new species of twinpod have been described recently in Wyoming. Herbarium records for these genera in Utah and Wyoming were checked at regional herbaria and no specimens and, hence, no new locations were discovered.

Second, since the proposed rule was developed, additional field work was conducted to more precisely characterize their geological habitat. This new data has been incorporated into the final rule. The two wild

mustards were found to have very specific, but slightly different, microhabitats within or adjacent to the Piceance Basin, as explained in the "Background" section. Most populations are contained within the center of the Piceance Basin where the Green River and Uinta Formations intertongue. The Calamity Ridge twinpod population, though not technically within the Basin, lies on the outer rim of the Piceance Basin.

Third, inventories for rare plants are stratified based on their specific potential habitat, i.e., areas considered likely to be potential habitat are thoroughly searched. This approach maximizes the probability of discovering new populations. Therefore, an inventory of the entire Piceance Basin was not necessary, only that portion characterized as potential habitat. Once the initial 1982 inventory was completed and results analyzed, those species determined to be the rarest, such as these wild mustards, were then made the specific subject of an inventory that was the basis for the 1987 status report. After both inventories, these wild mustards were still found to be rare species. Given the degree of search effort already expended, were new populations to be found in the future, it is unlikely that they would significantly alter overall population estimates or the conclusion that these are rare species capable of becoming extinct in the foreseeable future if protective measures are not undertaken.

Issue 2: The oil shale companies stated that there are no current threats to these species because there is no current oil shale mining occurring in the Piceance Basin.

Response: The proposed rule to list these species as threatened recognized planned oil shale development as being large scale, but not imminent. Because this development could potentially endanger these plants which were not protected under State or Federal law, the plants fit the definition of threatened species under the Act, i.e., species likely to become endangered within the foreseeable future throughout all or a significant portion of their range. It should be noted that new Federal subsidies for oil shale development have been proposed by Congressional committees for fiscal year 1990.

Issue 3: The oil shale companies stated that designating Areas of Environmental Concern for the plants on Bureau of Land Management Land while further inventories are being conducted would provide adequate protection.

Response: Although most of the wild mustard sites are located on Bureau land, the designation of these areas as Areas of Critical Environmental Concern would still allow for multiple use without the degree of protection afforded a species designated as threatened under the Act. Management of these multiple uses, particularly those that might conflict with the protection of these rare plants, would require more vigilant management by the Bureau. For example, in the Yanks Gulch Area of Critical Environmental Concern containing the twinpod, significant impacts from livestock trampling were observed in 1988 by the author on the hillside where the twinpod occurs. Listing the species as threatened under the Act would provide greater protection through its requirement for section 7 interagency consultation, section 9 prohibitions against take, and recovery actions.

Issue 4: The oil shale companies stated that, since the plants are locally common (as stated in the proposed rule), the populations are healthy and there are no threats to them.

Response: Many rare plant species are characterized by locally abundant populations restricted to small areas of specialized habitat. The threat to plants with this pattern of rarity is the vulnerability of their small acreage, which could easily be impacted significantly by surface disturbance from many different causes.

Issue 5: One oil shale company expressed a concern about future recovery actions possibly affecting their operations (tract "C–A" on Bureau land).

Response: The Service has no plans at this time for recovery actions on the "C–A" oil shale tract. No populations occur on this tract, thus recovery activities will be carried out elsewhere.

Issue 6: The County stated that there was inadequate data in the status report on population ecology on which to base a listing.

Response: The standardized New York Botanical Garden format (Henifin et al. 1981) which was used for the status report differentiates between minimally necessary information and other additional data. Under that model, population ecology is considered additional, but not necessary, data. Adequate data has been collected on all necessary categories and the Service believes this data supports listing as threatened. One of the results of species listing tends to be collection of additional data, such as population ecology, in order to better understand the species and the limiting factors causing its rarity.

Issue 7: The County stated that scientific collecting of the plants in small populations could have more of an effect than development activities.

Response: As stated in the proposed rule, the Service does not know of any over-collection for scientific purposes. Fortunately, most populations are locally abundant and over-collecting has not yet posed a threat. Listing of the species will initiate the permit process that regulates the degree of collecting.

Issue 8: The County stated that they did not agree with a statement in the status report that livestock grazing could be a threat. This belief was echoed by another attendee at the public hearing.

Response: The status report refers to the threat of grazing as a possibility, not a fact, and the proposed rule does not even refer to grazing as a threat. On the other hand, as mentioned earlier, significant impacts from livestock trampling were observed to occur on the hillside where the twinpod occurs in the Yanks Gulch Area of Critical Environmental Concern.

Issue 9: The County raised the point that surface disturbance may actually favor *P. obcordata* by reducing competition from other plants.

Response: P. obcordata has been observed to colonize small disturbed areas, such as road cuts, below communities where it is already found. However, were large-scale surface mining of oil shale to occur, widespread habitat destruction would occur, and natural recolonization of very large disturbed areas would be unlikely without a nearby seed source.

Issue 10: One attendee at the public hearing offered to show the Service other *Lesquerella* sites.

Response: The Service contacted this commenter after the hearing. The commenter stated he would be visiting the area where he thought he saw the species, and would bring specimens back if he found any. As of this writing, the Service has not received further word on this subject from the commenter.

Issue 11: Two attendees at the public hearing wished to know whether it would be possible to transplant or revegetate these species to minimize the probability of conflict with development activities.

Response: As yet, no research has been conducted with these species to determine whether transplantation or revegetation could be used as techniques to minimize conflict. Were development contemplated in the Piceance Basin in the near future, several years of lead time would be

required to evaluate the efficacy of these techniques, *e.g.*, evaluating survivorship within transplanted or revegetated areas. It has been noted, however, that other species of *Physaria* are relatively easy to propagate from seed.

Summary of Factors Affecting the Species

After a thorough review and consideration of all information available, the Service has determined that *Lesquerella congesta* and *Physaria obcordata* should be classified as threatened species. Procedures found at section 4(a)(1) of the Endangered Species Act and regulations (50 CFR part 424) promulgated to implement the listing provisions of the Act were followed. A species may be determined to be an endangered or threatened species due to one or more of the five factors described in section 4(a)(1). These factors and their application to *Lesquerella congesta* Rollins (Dudley Bluffs bladderpod) and *Physaria obcordata* Rollins (Dudley Bluffs twinpod) are as follows:

A. *The present or threatened destruction, modification, or curtailment of its habitat or range.* Portions of the multimineral oil shale area, including Dudley Bluffs, Ryan Gulch, and Yellow Creek, overlay oil shale deposits that are potentially recoverable by open-pit mining (Bureau of Land Management 1984). The rest of the area is suitable for underground mining of oil shale. A pilot project for a nahcolite solution mine has been constructed on Bar D Mesa between Piceance Creek, Yellow Creek, Ryan Gulch, and a 125,000 tons per year commercial mine, including evaporation ponds and a pipeline, has been proposed which would cover 254 acres (Bureau of Land Management 1986, Bureau of Land Management 1987b). Currently, the Bureau is reserving the multimineral area from commercial leasing until improved multimineral recovery technology is developed. However, leases for noncommercial research tracts not exceeding 2,000 acres will still be considered. Because of the massive scale of potential development in the limited area in which *L. congesta* and *P. obcordata* occur, a significant portion of the habitat of these two wild mustards would be destroyed and/or modified and their range possibly curtailed if development occurs. Up to 100 and 72 percent of the acreages on which *L. congesta* and *P. obcordata* occur, respectively, could be developed. There is already a designated linear utility corridor for pipelines, transmission lines, and roads along Ryan Gulch (Bureau of Land Management 1987a), and potential corridors exist along Dudley Gulch, Piceance Creek, and Yellow Creeks (Bureau of Land Management 1984). One of the Calamity Ridge sites has been bisected by a road (Colorado Natural Areas Program 1987).

B. *Overutilization for commercial, recreational, scientific, or educational purposes.* No such detrimental uses of these plants are known.

C. *Disease or predation.* No threats are known from disease or predation.

D. *The inadequacy of existing regulatory mechanisms.* There were no Federal or State laws protecting *L. congesta* and *P. obcordata* on Federal, State, or private lands prior to this listing. The Bureau's designation of one area each at Dudley Bluffs and Calamity Ridge as Areas of Critical Environmental Concern has provided and continues to provide for priority management of *L. congesta* and *P. obcordata* at these sites. However, these areas only protect about 20 percent of these species' limited habitat (about 50 acres for *L. congesta* and 250 acres for *P. obcordata*). The Act would provide additional protection and encourage active management through the "Available Conservation Measures" discussed below.

E. *Other natural or man-made factors affecting its continued existence.* These species' pattern of rarity, being locally abundant on small areas of specialized habitat, makes them particularly vulnerable to surface disturbances despite their high densities.

The Service has carefully assessed the best scientific and commercial information available regarding the past, present, and future threats faced by these species in determining to make this rule final. Based on this evaluation, the preferred action is to list *Lesquerella congesta* and *Physaria obcordata* as threatened. These species are restricted endemics with threats from potential oil shale development which could cause the two species to become endangered within the foreseeable future throughout all or a significant portion of their range; thus, they are threatened species as defined by the Act. Were large-scale oil shale development in the Piceance Basin imminent, these species would have been considered for endangered status. The Bureau has designated two areas containing these species as Areas of Critical Environmental Concern, which will provide for priority management (although impacts may still occur as noted above in "Comments" section), but neither species was protected by any State or Federal legislation prior to this listing. For reasons given below, it is not considered prudent to propose designation of critical habitat.

Critical Habitat

Section 4(a)(3) of the Act, as amended, requires that, to the maximum extent prudent and determinable, the Secretary designate any habitat of a species which is considered to be critical habitat at the time the species is determined to be endangered or threatened. The Service finds that designation of critical habitat is not presently prudent for *L. congesta* and *P. obcordata*. The designation of critical habitat is not considered prudent when such designation would not be of net benefit to the species. No benefit to these species can be identified from critical habitat designation that would outweigh the potential threat of vandalism or collection, which might increase if detailed habitat maps were published. The major populations of these species are accessible by major roads and their high densities on small acreages make them vulnerable to vandalism or collection.

Few, if any, additional benefits would be provided to these species by the critical habitat designation that would not already be provided by listing these species as threatened, particularly as the majority of the populations are located on lands under Federal jurisdiction. Any Federal action that would impact these plants' habitat would affect the plants as rooted organisms and, consequently, would be addressed through consultation under section 7 consultation. Moreover, section 9(a)(2)(B) of the Act, as implemented by 50 CFR 17.61 and 17.71, makes it unlawful to remove and reduce to possession any listed species of plant from areas under Federal jurisdiction. The Bureau is aware of the occurrences on their land and of its obligation under section 7 of the Act. Additional protection was extended by the 1988 amendments to the Act, which prohibited the malicious damage or destruction of listed plants on Federal lands, and the removal, cutting, digging up, or damaging or destroying of these plants on areas not under Federal jurisdiction in knowing violation of any State law or regulation, including State criminal trespass law. All involved parties and landowners have been or will be notified of the location and importance of protecting these species' habitat, and such protection will be addressed through the recovery process.

Available Conservation Measures

Conservation measures provided to species listed as endangered or threatened under the Endangered

Species Act include recognition, recovery actions, requirements for Federal protection, and prohibitions against certain practices. Recognition through listing encourages and results in conservation actions by Federal, State, and private agencies, groups, and individuals. The Endangered Species Act provides for possible land acquisition and cooperation with the States and requires that recovery actions be carried out for all listed species. The protection required of Federal agencies and the prohibitions against certain activities involving listed plants are discussed, in part, below.

Section 7(a) of the Act, as amended, requires Federal agencies to evaluate their actions with respect to any species that is proposed or listed as endangered or threatened and with respect to its critical habitat if any is being designated. Regulations implementing this interagency cooperation provision of the Act are codified at 50 CFR part 402. Section 7(a)(2) requires Federal agencies to ensure that activities they authorize, fund, or carry out are not likely to jeopardize the continued existence of such a species or to destroy or adversely modify its critical habitat. If a Federal action may affect a listed species or its critical habitat, the responsible Federal agency must enter into formal consultation with the Service.

L. congesta and *P. obcordata* occur largely on Federal land administered by the Bureau. The Bureau's involvement could include section 7 consultation on multimineral development and land exchanges with energy companies to bring the privately owned sites into Federal ownership and protection. On both Federal and private land, the Service expects that listing would elevate the awareness of these plants' status and foster efforts aimed toward their conservation.

The Act and its implementing regulations found at 50 CFR 17.71 and 17.72 set forth a series of general trade prohibitions and exceptions that apply to all threatened plants. All trade prohibitions of section 9(a)(2) of the Act, implemented by 50 CFR 17.71, would apply. These prohibitions, in part, would make it illegal for any person subject to the jurisdiction of the United States to import or export, transport in interstate or foreign commerce in the course of commercial activity, sell or offer for sale this species in interstate or foreign commerce, or to remove and reduce to possession the species from areas under Federal jurisdiction. Seeds from cultivated specimens of threatened plant species are exempt from these prohibitions provided that a statement of "cultivated origin" appears on their containers. In addition, for endangered plants, the 1988 amendments (Pub. L. 100-478) to the Act prohibit the malicious damage or destruction on Federal lands and the removal, cutting, digging up, or damaging or destroying of listed plants in knowing violation of any State law or regulation, including State criminal trespass law. Certain exceptions apply to agents of the Service and State conservation agencies. The Act and 50 CFR 17.72 also provide for the issuance of permits to carry out otherwise prohibited activities involving threatened species under certain circumstances. With regard to *L. congesta and P. obcordata*, it is anticipated that few, if any, trade permits would ever be sought or issued since these species are not common in cultivation or in the wild. Requests for copies of the regulations on plants and inquiries regarding them may be addressed to the Office of Management Authority, U.S. Fish and Wildlife Service, P.O. Box 3507, Arlington, Virginia 22203 (703/358-2104).

National Environmental Policy Act

The Fish and Wildlife Service has determined that an Environmental Assessment, as defined under the authority of the National Environmental Policy Act of 1969, need not be prepared in connection with regulations adopted pursuant to section 4(a) of the Endangered Species Act of 1973, as amended. A notice outlining the Service's reasons for this determination was published in the **Federal Register** on October 25, 1983 (48 FR 49244).

References Cited

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Cashion, W.B., and J.R. Donnell. 1974. Revision of nomenclature of the upper part of the Green River Formation, Piceance Creek Basin, Colorado, and Eastern Uinta Basin, Utah. U.S. Geological Survey Bulletin 1394-G. 9 pp.

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O'Kane, S.L., and J.L. Anderson. 1987. *Penstemon debilis* (Scrophulariaceae): a new species from Colorado endemic to oil shale. Brittonia 39:412-416.

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Author

The primary author of this final rule is John L. Anderson, botanist, U.S. Fish and Wildlife Service, Grand Junction, Colorado (303/243-2778, FTS 322-0351; see **ADDRESSES** above.)

List of Subjects in 50 CFR Part 17

Endangered and threatened species, Fish, Marine mammals, Plants (agriculture).

Regulation Promulgation

PART 17—[AMENDED]

Accordingly, part 17, subchapter B of chapter I, title 50 of the Code of Federal Regulations, is amended as set forth below:

1. The authority citation for part 17 continues to read as follows:

Authority: 16 U.S.C. 1361-1407; 16 U.S.C. 1531-1543; 16 U.S.C. 4201-4245; Pub. L. 99-625, 100 Stat. 3500; unless otherwise noted.

2. Amend § 17.12(h) by adding the following, in alphabetical order under the family Brassicaceae, to the List of Endangered and Threatened Plants:

§ 17.12 Endangered and threatened plants.

* * * * *

(h) * * *

Species		Historic range	Status	When listed	Critical habitat	Special rules
Scientific name	Common name					
Brassicaceae—Mustard family						
*	*	*	*	*	*	
Lesquerella congesta	Dudley Bluffs bladderpod	U.S.A. (CO)	T	373	NA	NA
*	*	*	*	*	*	
Physaria obcordata	Dudley Bluffs twinpod	U.S.A. (CO)	T	373	NA	NA
*	*	*	*	*	*	

Dated: January 24, 1990.

Jay L. Gerst,

Acting Director, Fish and Wildlife Service.

[FR Doc. 90-2642 Filed 2-5-90; 845am]

BILLING CODE 4310-55-M

50 CFR Part 17

RIN 1018-AB31

Endangered and Threatened Wildlife and Plants; Determination of Threatened Status for the Plant Calyptronoma rivalis

AGENCY: Fish and Wildlife Service, Interior.

ACTION: Final rule.

SUMMARY: The Service determines *Calyptronoma rivalis* (palma de manaca) to be a threatened species pursuant to the Endangered Species Act of 1973 (Act), as amended. *Calyptronoma rivalis* is a palm tree that is endemic to the island of Puerto Rico. The three remaining natural populations are restricted to the subtropical moist and subtropical wet limestone forests of the northwest part of the island. The species is threatened by erosion due to flash flooding, and by agricultural expansion, and rural development. Flash flooding has increased due to extensive deforestation in surrounding areas. This final rule for *Calyptronoma rivalis* will implement the Federal protection and recovery provisions afforded by the Act.

EFFECTIVE DATE: March 8, 1990.

ADDRESSES: The complete file for this rule is available for inspection, by appointment, during normal business hours at the Caribbean Field Office, U.S. Fish and Wildlife Service, P.O. Box 491, Boqueron, Puerto Rico 00622 and at the Service's Southeast Regional Office, Suite 1282, 75 Spring Street SW., Atlanta, Georgia 30303.

FOR FURTHER INFORMATION CONTACT: Ms. Susan Silander at the Caribbean Field Office address (809/851-7297) or Mr. Tom Turnipseed at the Atlanta Regional Office address (404/331-3583 or FTS 242-3583).

SUPPLEMENTARY INFORMATION:

Background

Calyptronoma rivalis was first collected in 1901 by L.M. Underwood and R.F. Grigg in San Sebastian of western Puerto Rico. In 1923, N. Britton and P. Wilson referred to this species as *Calyptrogyne occidentalis*; however, L.H. Bailey, in his 1938 monograph on the group, provided sufficient evidence to place the species in a separate genus *Calyptronoma*. Authorities on the palm family accept this opinion and view this palm as an endemic species. Until recently, the species was known only from the type locality, where 44 individuals occur. Additional populations were discovered along the Camuy River in 1981 and later along the Guajataca River, both in northwestern Puerto Rico (Vivaldi and Woodbury 1981, Natural Heritage Program 1989). About 220 individuals are presently known from these populations. In addition, seeds have been collected from mature specimens and a small number of seedlings cultivated from these have been introduced into the Puerto Rico Department of Natural Resource's Rio Abajo Commonwealth Forest and the nearbly Camp Guajataca of the Boy Scouts.

Calyptronoma rivalis is an arobrescent palm that may reach 30 to 40 feet (8 to 10 meters) in height and 6 to 10 inches (13 to 25 cm) in diameter. The spineless, pinnate leaves may reach up to 12 feet (3 meters) and have petioles and sheaths up to 2 feet long (.8 meter). The inflorescence is a drooping panicle about 3 feet (1 meter) long. The flowers are in triads of two males and one female and are borne on sunken pits. Fruits are only .24 inch (6 millimeters) in diameter and are subglobose and reddish when ripe. All fruits mature at approximately the same time and fall with the persistent flower parts still attached to the base.

Only three natural populations and two small, introduced populations are known: San Sebastián, Camuy River, Guajataca River, Camp Guajataca and the Rio Abajo Commonwealth Forest. All occur in the semievergreen seasonal forests of the karst region of northwestern Puerto Rico at elevations of 100 to 150 meters. All three natural populations are found in level or nearly level areas along stream banks. Deforestation in the surrounding areas has increased the threat of flash flooding and therefore the establishment of seedlings may be difficult. The construction of a road in the Camuy area resulted in the destruction of a large portion of that population.

Calyptronoma rivalis was recommended for Federal listing by the Smithsonian Institution (Ayensu and DeFilipps 1978). The species was included among the plants being considered as candidates for proposal to list as endangered or threatened species by the Fish and Wildlife Service in notices of review published in the **Federal Register** (45 FR 82480) dated December 15, 1980; the November 28, 1983, update (48 FR 53640) of the 1980 notice; and the September 27, 1985, revised notice (50 FR 39526). The species was designated category 1 (species for which the Service has substantial information supporting the appropriateness of proposing to list them as endangered or threatened) in each of these three notices.

In a notice published in the **Federal Register** on February 15, 1983 (48 FR 6752), the Service reported the earlier acceptance of the new taxa in the Smithsonian's 1978 book as under petition within the context of section 4(b)(3)(A) of the Act, as amended in 1982. The Service made subsequent petition findings in 1984 through 1988 that listing *Calyptronoma rivalis* was warranted but precluded by other pending listing actions of a higher priority, and that additional data on vulnerability and threats were still being gathered. The Service proposed listing *Calyptronoma rivalis* on February 7, 1989 (54 FR 5983), which constituted the final required petition finding in accordance with section 4(b)(3)(B)(ii) of the Act.

Summary of Comments and Recommendations

In the February 7, 1989, proposed rule and associated notifications, all interested parties were requested to submit factual reports of information that might contribute to the development