NIOSH Comments to DOL

POST-HEARING COMMENTS OF THE NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH ON THE OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION'S PROPOSED RULE ON OCCUPATIONAL EXPOSURE TO CADMIUM

29 CFR Part 1910
Docket No. H-057a

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Centers for Disease Control
National Institute for Occupational Safety and Health

9/18/90

The following items are post-hearing comments by the National Institute for Occupational Safety and Health (NIOSH) in response to requests for information that were made at the Occupational Safety and Health Administration (OSHA) cadmium hearing on July 17, 1990.

1. **NIOSH Risk Assessment on Cadmium**

   The NIOSH risk assessment on cadmium was sent to OSHA on September 5, 1990.

2. **A representative of the American Iron and Steel Institute (AISI) asked whether NIOSH had evaluated the technological feasibility for controlling exposures to cadmium in the steel industry by specific processes or operations.**

   Based on comments to the OSHA docket (H-057a) by the AISI, several steel-making operations are listed as potentially problematic with respect to low-level cadmium exposure. They are:

   1. Blast furnace operations
   2. Sinter plant operations
   3. Basic oxygen, electric, and open-hearth furnace operations
   4. Leaded steel production
   5. Maintenance, cleanup and repair operations
   6. Lead and zinc coating operations
   7. Teeming and pouring operations

   NIOSH has not performed evaluations of technological feasibility of controlling cadmium in these steel-making operations. However, the overall principles of control technology that were outlined in the NIOSH testimony [NIOSH 1990a] would apply to these operations as well.

   Because cadmium is a nonessential contaminant of steel, one control option would be to segregate feedstocks containing cadmium (e.g., recycled scrap) as much as possible. Other innovative control approaches also may be candidates for research, such as the use of scavenging materials to bind the cadmium. Although we are not aware of any specific scavenging materials, the underlying issue is that there may be innovative control approaches that would be more effective (and perhaps less expensive) than using ventilation alone.

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16. Abstract (Limit: 200 words) This testimony contains comments from NIOSH regarding requests for information at an OSHA hearing on cadmium (7440439). The following steel making operations were listed as potentially problematic with respect to low level cadmium exposure: blast furnace operations; sinter facility operations; basic oxygen, electric, and open hearth furnace operations; leaded steel production; maintenance, cleanup and repair operations; lead and zinc coating operations; teeming and pouring operations. The estimate of total workers potentially exposed to cadmium compounds is given as 297,684 for 15,501 facilities. NIOSH recommends that workers with occupational exposure to cadmium not smoke in the workplace. Population studies are reviewed which deal with trace metals in occupationally and nonoccupationally exposed individuals; the exposure and accumulation of cadmium in populations from Japan, the United States and Sweden; normal levels of cadmium in diet, urine, blood, and tissues of inhabitants of the United States; and urinary cadmium and beta-2-microglobulin levels of individuals. Other topics briefly noted include air monitoring, medical monitoring and laboratory measurements of blood and urine cadmium concentrations.

17. Document Analysis a. Descriptors

b. Identifiers/Open-Ended Terms NIOSH-Publication, NIOSH-Author, NIOSH-Testimony, Millar-J-D, Cadmium-compounds, Cadmium-poisoning, Metalworking-industry, Biological-monitoring, Urinalysis, Blood-analysis, Risk-analysis, Steelworkers

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3. **A representative of the AISI asked if the NIOSH National Occupational Exposure Survey (NOES) estimates of number of workers exposed to cadmium included the steel industry. Specifically, does the NIOSH NOES estimate of the number of workers exposed to cadmium include the steel industry and blast furnace operations?**

   Yes. The NOES profile by 2-digit SIC code (SIC 33) that was submitted to the OSHA docket [NIOSH 1990b] included SIC 3312 (Blast Furnaces and Steel Mills). However, from the statistical sample of 23 facilities surveyed by NIOSH, workers in only one plant in SIC 3312 had potential exposure to cadmium and cadmium compounds.

4. **OSHA requested that NIOSH update its original NOES estimate to the docket of the number of workers exposed to cadmium.**

   The original estimate was by 2-digit SIC code. Attachment 1 shows the updated estimate by 3-digit SIC code. The estimate of total workers potentially exposed to cadmium compounds, which is 297,684 for 15,501 plants, is different from the original submission to OSHA in May 1990 [NIOSH 1990b], because the NOES file has been updated.

5. **A representative of the AISI asked if NIOSH had evidence of the inappropriate use of negative pressure respirators, in particular, related to cadmium exposure.**

   Exhibit 1 of the NIOSH comments to the OSHA docket on methods of compliance, and enclosed for OSHA's use now (as Attachment 2), contained many examples of inappropriate use of negative pressure respirators as documented from the NIOSH health hazard evaluation program. The NIOSH Guide to Industrial Respiratory Protection, submitted with the NIOSH comments in May 1990 also listed respirator usage problems as identified on respirator field studies and respirator users notices. In its response to the OSHA docket on methods of compliance, NIOSH used this documentation to support its preference for engineering technology to control exposures.

6. **OSHA requested NIOSH comments on exposure to cadmium and smoking**

   NIOSH recommends that workers with occupational exposure to cadmium not smoke in the workplace. Additionally, workers with exposure to cadmium should be encouraged to stop smoking. This is consistent with previous NIOSH policy regarding smoking by workers who are at increased risk of lung cancer due to exposure to a known or potential occupational carcinogen [NIOSH 1979; 1987].

   Some of the reasons for the recommendation against smoking in cadmium-contaminated work areas are:

a. Cigarette smoke contains cadmium -- reported levels range from 1.0 to 6.5 μg per cigarette [Mussalo-Rauhamaa et al. 1986], with the more usual range reported being between 1.0 and 2.0 μg per cigarette [Friberg et al. 1974; Mussalo-Rauhamaa et al. 1986]. When a cigarette burns, up to 70 percent of the cadmium may enter the airborne particulate phase [Friberg et al. 1974; Mussalo-Rauhamaa et al. 1986; Lewis et al. 1972] to be inhaled by the smoker as mainstream smoke, or by other people as sidestream smoke.

b. Tobacco products may become contaminated by cadmium while being carried in work clothes' pockets, and while being handled in the workplace [Piscator et al. 1976]. Handwashing at work may not eliminate cadmium residue from the hands [Roels et al. 1982]. This can lead to increased cadmium ingestion through mouth contact, and also increased cadmium inhalation as the contaminated paper is burned.

c. Smoking may increase the respiratory tidal volume [Tobin et al. 1982] and therefore increase the amount of ambient cadmium that is deposited in the lungs. Alveolar deposition of 5 μm particles increases by 40%. This increase is associated with a one-liter increase in inhalational volume [Pavia et al. 1977].

d. Cigarettes cause lung cancer [DHHS 1989] -- smoking will further increase the risk that cadmium-exposed workers will develop lung cancer. Whether the combined effects of these insults are greater than additive is unknown.

e. Smoking causes emphysema [DHHS 1989] and cadmium has been associated with emphysema [Elinder 1986]. Whether the combined effects of these insults are greater than additive is unknown.

ANSWERS TO SPECIFIC QUESTIONS ON SMOKING POSED IN OSHA'S MEMO DATED JULY 2, 1990

1) Is smoking likely to increase the smokers' body burden of cadmium?

Yes. Smokers with and without occupational exposure to cadmium have been noted to have increased tissue levels of cadmium [Adamsson et al. 1979; Kjellstrom 1979; Ostergaard 1977].

2) Is smoking likely to contribute to cadmium-induced disease?

Yes. Any source of cadmium may contribute to cadmium-induced disease, and smoking does increase the body burden of cadmium. However, it must be noted that smoking alone, without another source of cadmium exposure, is unlikely to produce a body burden of cadmium high enough to induce renal disease. Friberg et al. [1974] estimated that a smoker would need to smoke greater than six packs of cigarettes per day for 50 years to reach cadmium levels associated with renal disease.

3) If the answer to question 1) or 2) is affirmative, should OSHA:

a) Require the examining physician to inquire about the patient's smoking history and status?

Yes. Identification of workers who smoke will allow interventions to be focused on those persons at increased risk. Interventions from the physician or health care staff should include:

-- encouragement to stop smoking

-- education concerning the hazards of smoking, especially in cadmium-contaminated areas.

b) Require the examining physician to advise the patient that smoking is a source of cadmium exposure that is cumulative with whatever occupational exposure the patient absorbs, and that the patient therefore should stop smoking?

Yes. The examining physician should advise the patient that smoking is a source of exposure to cadmium and to stop smoking.

c) Require the employer to include some discussion of the special problems of smoking in the training program for cadmium exposed workers?

Yes. In regard to smoking, the following should be incorporated into hazard communication information:

-- smoking causes an increase in the body cadmium burden. This alone may not be enough to cause cadmium-induced disease, but any cadmium absorbed through smoking is additive to the amount absorbed through other means (i.e., from work exposures)

-- smoking in a cadmium-contaminated area will increase the absorption of cadmium above that associated with smoking alone

-- both smoking and cadmium have been causally associated with lung cancer and emphysema.

d) Require the employer to include in the training program for cadmium exposed workers information concerning available smoking cessation programs and to distribute self-help smoking cessation program material?

Yes.

e) **Ban smoking in all work areas where occupational exposure to cadmium exists?**

Yes. Smoking should be banned in all cadmium production areas as well as break areas that are on site. Smoking materials should not be carried into work areas; they should be stored with street clothes, and only allowed after showering and changing out of work clothing.

7. **OSHA requested information on the distribution of population levels of urine β-2-microglobulin and urine cadmium, with a breakdown by smoker/nonsmoker categories if possible.**

There are four distinct United States data sets that summarize the population distribution of urine cadmium [Johnson et al. 1975; Kjellstrom 1979; Kowal et al. 1979; Kowal et al. 1981]. Two of these data sets include some information on smoking status [Kjellstrom 1979; Kowal et al. 1979]. Only one data set covers the United States population distribution of urine β-2-microglobulin [Kowal et al. 1979; Kowal et al. 1981]. Again, smoking information was collected but was not fully reported.

Although the data analyses reported in the literature have some validity, great caution must be exercised when extrapolating the conclusions to other populations [Johnson et al. 1975; Kjellstrom 1979; Kowal et al. 1979; Kowal et al. 1981].

In evaluating the published literature, NIOSH considered the following:

- how was the study population chosen;
- was the study group truly representative of the overall group from which it was drawn in regards to age group, sex, smoking habits, and occupation;
- were persons occupationally exposed included or excluded from the study;
- was the presence or absence of any other significant source of cadmium contamination discussed, such as environmental contamination;
- how do the authors define the population "normal" values.

The references for these analyses are listed and discussed below. (Two additional papers, discussing Swedish [Elinder et al. 1978] and Japanese [Kjellstrom et al. 1977] populations exclusively, are also submitted with these comments).

As stated on page 19 of the NIOSH testimony [NIOSH 1990a], background levels of cadmium in urine for the general population without occupational exposure to cadmium are usually below 2 μg/gm creatinine. The upper limit

of normal for urine β-2-microglobulin will vary between approximately 0.2 and 0.3 mg/gm creatinine when determined by the method described on page 18 of the NIOSH testimony [NIOSH 1990a].

POPULATION STUDIES

Trace metals in occupationally and nonoccupationally exposed individuals. Johnson DE, Tillery JB, Prevost RJ [1975]. Environ Health Perspect 10:151-158.

216 persons were studied to determine the effect of automobile exhaust emissions. None of the occupations reported were considered by NIOSH to have occupational cadmium exposure [NIOSH 1983]. Policemen on foot had the highest cadmium urine level mean (mean = 1.4 µg Cd/ℓ, standard deviation of 1.05).

Exposure and accumulation of cadmium in populations from Japan, the United States, and Sweden. Kjellstrom T [1979]. Environ Health Perspect 28:169-197.

Urine cadmium levels were studied in Dallas white males, of whom 63 were older than 20 years of age. The population was drawn from hospital volunteer staff and service club members without known occupational exposure to cadmium. Smokers and nonsmokers were included in the study, but the groups were not analyzed separately.

The distributions of urine cadmium levels from each age range fit a log-normal distribution best. However, note that there were only 7 individuals in the 30-39 age range. Age-specific means and standard deviations are tabulated. No reported mean exceeds 0.8 µg Cd/ℓ urine.

Normal levels of cadmium in diet, urine, blood, and tissues of inhabitants of the United States. Kowal NE, Johnson DE, Kraemer DF, Pahren HR [1979]. J Toxicol Environ Health 5:995-1014.

Urine cadmium levels were studied in two distinct populations, in Chicago and Dallas. The Dallas population was the same white male cohort described above [Kjellstrom 1979]. The Chicago population, in 1974, encompassed 120 persons older than 20 years of age of both sexes, mostly white. Two years later, 102 persons older than 20 years of age (most of whom were in the 1974 study) were again studied in Chicago. The methods used to choose the study populations were not described; no information regarding occupational exposures is available.

The mean urine cadmium levels, along with range, geometric mean and geometric standard deviation, are tabulated by age group. The highest mean reported is 1.115 µg Cd/ℓ urine. The Chicago data did not show a significant increase ($p < 0.05$) of cadmium with an increase of age; the Dallas data did.

Aggregate smoking data is also reported, distinguishing between never, former, and current smokers. The highest reported mean was for current smokers of all ages in Chicago (1974). This value of urine cadmium level was 1.038 μg Cd/ℓ urine.

Urinary cadmium and β-2-microglobulin levels of persons aged 20-74 from a subsample of the national HANES II survey, 1976-1980. Kowal NE, Kraemer DF [1981]. In: Proceedings of the Third International Cadmium Conference, Miami, FL. New York, NY: Cadmium Council, pp. 119-122.

The subsample described in this paper is a nonrandom grab sample of the overall National Health and Nutrition Examination Survey II (NHANES II). NHANES II sampled from all states of the U.S. There was deliberate oversampling of persons residing in poverty areas, and persons less than 6 years old or greater than 59 years old. The NHANES II design requires age group weighting prior to statistical analysis [per telephone conversation with Robert Murphy, National Center for Health Statistics, August 16, 1990].

One thousand spare urine specimens of persons aged 20-74 were collected from the last 13 sites that were visited by NHANES II. These were forwarded to EPA for separate laboratory analysis of cadmium and β-2-microglobulin. No weighting for age was done for these subsample measurements even though the age group 60-69 is overrepresented.

The authors state that it "is evident that low values of urinary cadmium and β-2-microglobulin predominate in the population, but that a small fraction of the population may have quite high levels." The data, summed over both sexes and all ages, is presented as frequency distributions. The authors calculate that 10%-21% of the variability of the cadmium values is explained by age differences, but this was not considered when generating the frequency distributions. Caution must be used in extrapolating these data to other populations.

Information on occupation was collected through NHANES, but was not presented in this paper.

Urinary cadmium and beta 2-microglobulin: normal values and concentration adjustment. Kowal NE, Zirkes M [1983]. J Toxicol Environ Health 11:607-624.

This paper presents further analyses of the same 1000-person subsample of the NHANES II cohort as described above [Kowal and Kraemer 1981]. The authors found that 94.5% of the subsample had urine cadmium levels less than 2.6 μg/g creatinine. In addition, 94.6% of the subsample had urine β-2-microglobulin levels below 320 μg/g creatinine.

This subsample overrepresents persons over 59 years of age. Therefore, caution must be used in extrapolating these data to the general U.S. population.

8. **OSHA requested discussion of why NIOSH chose not to advocate the use of blood cadmium monitoring.**

The level of cadmium in blood reflects both the total body burden as well as more recent cadmium exposure [Hassler et al. 1983]. Because blood cadmium is not a specific indicator of the total body burden, it is limited as a predictor of long-term renal health effects. Urine cadmium usually provides a better indication of total body burden.

As a predictor of short-term health effects, blood cadmium is also limited. Blood cadmium levels reflect the average exposure to cadmium over the previous several months [Kjellstrom 1979; Lauwerys et al. 1979]. Sudden exposure changes, such as removal from the workplace for one month, may not be reflected in blood cadmium measurements [Lauwerys et al. 1979]. Reports describing the effect of intermittent, high exposures on blood cadmium levels are absent from the published literature.

Serial measurements of blood cadmium on a monthly basis [Kjellstrom 1979] can detect trends in a worker's exposure. However, monitoring blood is more invasive (and perhaps less acceptable to workers) than monitoring urine, and, as discussed above, the interpretation of blood cadmium levels is more complex than of urine cadmium levels.

9. **OSHA requested a description of information being collected by the National Health and Nutrition Examination Study (NHANES) (i.e., urine β-2-microglobulin and urine retinol binding protein, urine and blood cadmium levels).**

NHANES III data collection includes urine cadmium levels of persons aged 6 and greater. The survey does not include measurement of blood cadmium, urine β-2-microglobulin or urine retinol binding protein.

The NHANES III design encompasses two three-year data collection cycles. Data collection for the first cycle is scheduled to be completed in September 1991. The results, weighted to account for deliberate oversampling of smaller population components, will be published six to twelve months later. Information concerning NHANES III was obtained from Geraldine McQuillan, Ph.D., epidemiologist in charge of the laboratory component (301/436-7080), at the National Center for Health Statistics (NCHS).

During NHANES II (1978-1979), in collaboration with EPA, 1000 samples of randomly voided urine were analyzed for cadmium and β-2-microglobulin. Kowal and Zirkes reported on this data in 1979. A further discussion of their paper is under the population distribution discussion. The NCHS contact for this information is Robert Murphy (301/436-7080).

10. **OSHA requested clarification of the NIOSH recommendations for scheduled air monitoring; whether quarterly monitoring for cadmium could be reduced.**

It is good industrial hygiene practice to conduct air monitoring on a quarterly basis; NIOSH does not recommend a reduction in this schedule. Exposure levels will vary because of day-to-day fluctuations in the manufacturing process, and because of inherent sampling and analytical variability. Even when other substances are being monitored on a regular schedule (i.e., for flyash) and those substances are low, it cannot be assumed that cadmium is also low. If a surrogate is used, the burden of proof would be on industry to prove that monitoring the level of a surrogate substance indeed correlates consistently with the actual level of cadmium.

11. **OSHA requested clarification of the NIOSH recommendation against a time-limited cadmium exposure trigger for medical monitoring.**

NIOSH advises against a time-limited trigger because cadmium is cumulative in the body, with a long half-life, and is also potentially carcinogenic. NIOSH recommends that medical surveillance should be triggered by occupational exposure to any quantifiable level of cadmium.

12. **OSHA requested information on the standardization of laboratory measurements of blood and urine cadmium.**

Attachment 3 is a draft response prepared by the Center for Environmental Health and Injury Control of the Centers for Disease Control. Before this response on laboratory standardization is finalized, NIOSH would like to receive comments on this material from OSHA and, if possible, the public.

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