# Retrospective Mortality Study of Cadmium Workers- An Update

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ILZRO - International Lead Zinc Research Organization

Abstract

Human and animal data implicate cadmium as a possible lung carcinogen. A retrospective cohort mortality study, conducted by NIOSH, found a statistically significant excess of deaths from lung cancer (Obs=20, Exp=11.38, SMR=176) among 602 U.S. cadmium production workers. The SMR for lung cancer increased with increasing cumulative exposure to cadmium, equalling 53, 152, and 280, in the low, medium, and high exposure categories. Tobacco lifetime-use questionnaires were available from the employer on 50% of study participants. Fewer of the cadmium workers were current cigarette smokers in 1965 (near the mid-point of the study) than among the white male U.S. population (49% v.s. 73%). Adjusting the overall SMR for the deficit of smoking would raise the relative risk estimate in this cohort from 176 to approximately 263. Careful attention to potential confounders is essential in occupational studies. A nested case-control study will further assess the role of cadmium exposure, tobacco smoking and arsenic to the lung cancer excess in this cohort.

(SLIDE 1) Today I will discuss the results of a retrospective cohort mortality study of a group of cadmium production workers. Most of you are familiar with the study. As you know, the most striking finding is an excess of deaths from lung cancer, compared to the number of deaths expected from U.S. rates. The increased mortality from lung cancer has been cited by both the EPA, in a risk assessment document proposing that cadmium be classified a human carcinogen for the lung, and by NIOSH, in a current intelligence bulletin on cadmium.

I will review the background, methods and results of the study briefly. I will also describe data that have recently become available to NIOSH on exposures other than cadmium at the plant. In particular, we have received additional information about the tobacco smoking habits of the study members, and about arsenic exposure at the plant than that which was available at the time of publication. Both agents are lung carcinogens that could be important confounders, if inadequately controlled for.

BACKGROUND

Let me begin by reviewing the background of the study. The recent study extended an earlier mortality study of workers at the same plant. (SLIDE 2) Lemen and other NIOSH researchers previously studied causes of death through 1973 of 292 cadmium workers who worked at least 2 years at a small plant that recovers cadmium from "bag house dust". The major

exposures of these workers since 1926 were to dusts of cadmium oxide and cadmium sulfide and to cadmium fume. Relative to U.S. white males, the cadmium workers experienced an over two-fold excess of deaths from respiratory cancer (SMR=235). The study also found an over threefold excess of deaths from prostatic cancer (SMR=347).

The results stimulated interest in the role of cadmium as a potential carcinogen. Concern about the carcinogenicity of cadmium initially focussed on the prostate, because cadmium accumulates in this organ, and because two previous occupational reports had noted excess deaths from prostatic cancer at a single small British battery plant. The excess of deaths from lung cancer was discounted because no information was available at that time on cigarette smoking and arsenic exposure.

In 1980, we decided to extend followup of workers at this plant. Although small, the population had several unusual attributes making further study useful. (Slide 3- Advantages of the study population) 1) First was the opportunity for prolonged followup. Over 82% of the workers hired before 1970 had achieved 20 or more years of followup.

2) Second, compared to other cohorts of cadmium workers, exposures were high. For example, urine cadmium levels, available on nearly all workers since 1960, show that <u>over 80%</u> of sampled workers had a median level of 20 ug/l or greater. By contrast, <u>less than 3%</u> of workers in the British

cadmium registry had similar exposures. High past exposures at the U.S. plant offset the relatively small sample size.

3-4) Two other advantages were the availability of extensive historical exposure data, and the presence of some information on arsenic exposure and cigarette smoking which could be used to adjust for the potential confounding effects of these factors. I will return to these later.

(Slide- Methods) This slide reviews the objectives and methods that we used in extending study of the cohort. As listed, the study extended vital status followup for an additional five years (through 1978); included short-term employees (i.e. those who had worked six months to 2 years between 1940 and 1969) to provide an internal comparison; analysed mortality by both length of employment, and by cumulative exposure to cadmium; and assessed the effect of cigarette smoking and arsenic exposure.

(Slide-Inhalation exposures in "high" exposure areas) To compute cumulative exposure to cadmium, we combined work histories with the detailed industrial hygiene air measurements seen here. These were collected by the company since the mid-1940's, and compiled by Dr. Tom Smith, an industrial hygienist formerly with the company. These inhalation estimates have been adjusted by Dr. Smith to reflect actual exposures, taking into account the mode of sampling, and respirator usage.

(Slide - Vital status by length of employment) The extended cohort included 602 white males, 179 of whom were deceased. Eighty-seven additional deaths had occurred since the previous NIOSH study. Vital status was determined for 98% of the population; the 2% who were lost to followup.

RESULTS

(Slide-all cause mortality) I will now summarize the results. All-cause mortality was slightly below that of the U.S. male population. As you know, an SMR of 100 is equivalent to the experience of the U.S. population. The 5% deficit in deaths due to all causes was due to a large, statistically significant deficit of deaths from cardiovascular disease. The SMR for circulatory diseases equalled 65, with an upper 95% confidence limit of 85.

I will focus further discussion upon malignant causes of death, particularly lung cancer.

(Slide-Mortality from cancers) This slide shows the number of observed and expected deaths from all malignancies, and from respiratory and genitourinary cancer. Deaths due to respiratory cancer were 65% above the number expected. The excess of deaths from respiratory cancer is statistically significant, despite the inclusion of 257 "short term workers" in this analysis.

Deaths due to genitourinary cancer, a category that includes prostatic cancer, were 35% above expected, but the excess was not statistically significant.

(Slide-Deaths due to genitourinary cancer) I will deal only briefly with prostatic cancer. This slide shows that three of the six deaths from genitourinary cancer were due to cancer of the prostate (point to the top three). No new deaths had occurred during the additional follow-up period. Furthermore, one of the cases included in the original study, a plant guard, was excluded from our study because he did not work six months in a production area. There remained three deaths from prostatic cancer, which was no longer significantly elevated above the 1.41 expected.

I will focus the rest of this discussion on respiratory cancer. All of the 20 deaths due to respiratory cancer were due to cancer of the lung, trachea and bronchus.

(Slide-Lung cancer before and after 1926) To minimize the possible contribution of arsenic in explaining these lung cancers, we stratified the cohort into workers employed before, and those employed after January 1, 1926. The plant functioned as an arsenic smelter prior to 1926. An over seven-fold increase (SMR=714) in lung cancer mortality was observed among persons hired prior to 1926. The excess was smaller among workers hired after that date, and was statistically significant only for workers employed two or more years (SMR=229).

We next looked for a dose-response relationship between lung cancer mortality and cadmium exposure. Again we excluded workers hired before 1926 to minimize the contribution of arsenic exposure.

(Slide-lung cancer by length of employment) First we examined lung cancer mortality by length of employment. No deaths from lung cancer occurred among the short term workers. Lung cancer mortality was twice that expected from U.S. rates among workers employed for two or more years. However, the SRR did not increase with more prolonged employment. (Note- the measure of effect here is the directly standardized rate ratio, or SRR. The null value is 1 rather than 100, as with SMR's).

To explain this curiously flat dose-response relationship, we hypothesized that length of employment might be a crude measure of exposure. Much of the cadmium exposure might be incurred during the early years of employment in entry level jobs that had higher exposures.

(Slide-lung cancer by cumulative exposure) Cumulative exposure, the product of time in a job and intensity of the exposure is a more precise measure of individual exposure. When lung cancer mortality is analysed by cumulative exposure to cadmium, a strongly positive dose-response trend is seen. This trend is apparent with both the directly standardized SRR's and the SMR's. The regression slope for this trend is significantly greater than zero.

Note- Boundaries for the strata used in this analysis were selected based on current occupational exposure limits. The lowest stratum represents what a worker would accumulate during 40 years of exposure at up to 40 $ug/m^3$, within the current NIOSH recommended TWA. The middle stratum is between the NIOSH recommended and the OSHA legal standard of 200 $ug/m^3$, and the highest stratum is equivalent to 40 years at above the OSHA legal limit.

(Slide- Colorado rates- lung cancer by cumulative exposure) Since our study results were published, we have also examined lung cancer mortality of the cadmium workers compared to death rates of the state of Colorado. Comparison with Colorado rates, since 1950, show a similar, if more pronounced, trend. The SMR for lung cancer again increases with cumulative exposure to cadmium; a nearly four-fold increase is evident in the highest exposure group.

(Dark slide) Because these findings focussed our concern upon lung cancer, we examined whether cadmium exposure, cigarette smoking or arsenic exposure best explained the results. Several techniques are available to differentiate between multiple exposures in occupational studies. The preferred method is a nested case-control study. NIOSH is presently conducting such a study, but the results will not be ready for some time.

An indirect method to examine the contribution of smoking is to assess whether causes of death known to be associated with smoking are also increased. For example, if lung cancer mortality is increased due to

smoking, then other smoking-related causes of death should also be increased. The statistically significant deficit of deaths from cardiovascular disease argues against excess smoking among the cadmium workers. Coronary heart disease death rates are usually 1.5 to 2-fold higher among current smokers than among non-smokers. Here the SMR for circulatory diseases is 65, 35% lower than that of the U.S. population. The deficit of cardiovascular deaths is greater than that usually seen in occupational popualtions. It exceeds the usual "healthy worker effect" and makes heavy smoking very unlikely.

A more direct method to assess the effect of smoking is to obtain individual tobacco smoking histories and to compare the smoking habits of the workers to those of the U.S. comparison population. We did not have these histories at the time of the published report, but we have obtained them subsequently. The data were collected by the company from medical records and from a questionnaire survey mailed to surviving workers or their next of kin in 1982. Lifetime smoking histories were available on 298 (50%) of the cohort.

Using the tobacco questionnaires, we computed cigarette smoking habits as of July 1, 1965. The year 1965 is the earliest year that data are available from the Health Interview Survey of the National Center for Health Statistics regarding the cigarette smoking habits of the U.S. white male population. U.S. white males served as the comparison group in the

mortality study. 1965 was closer to the midpoint of the study than were subsequent national smoking surveys.

(Slide- Comparison of cadmium workers with U.S. population) This slide shows the cigarette smoking habits of the cadmium workers in 1965 compared with participants in the 1965 Health Interview Survey. A larger percentage of the cadmium workers were nonsmokers (not currently smoking cigarettes in 1965) than white males in the U.S. general population (51% v.s. 27%). Similarly, a smaller percentage of the cadmium workers were "heavy" smokers than in the general population (10% v.s. 20%).

These findings are at odds with those of critics of our study. Based on the same data, Drs. Lamm and White have claimed in testimony to the EPA and in several scientific conferences that the cadmium workers were unusually heavy smokers. One explanation for the discrepancy is that these authors have failed to consider the smoking habits of the comparison population, and have not actually compared smoking prevalence in the cadmium workers with that of US males.

(Slide- Axelson adjustment) A technique for estimating the probable effect of cigarette smoking in an occupational study has been developed by Dr. Olaf Axelson. This technique estimates the change in the SMR likely to result from disparities in cigarette smoking. The information required to compute this includes the cigarette smoking habits of the exposed workers, comparable information for the comparison group, and the relative risk for

lung cancer associated with each level of smoking. This slide shows the format of the Axelson adjustment. Because the cadmium workers smoke less, they would be expected to have 31% fewer deaths from lung cancer than U.S. males. Instead, the cadmium workers had 76% more deaths (SMR for lung cancer = 1/6). If we adjust the SMR in the cadmium cohort to reflect the lower levels of tobacco smoking, we find an overall SMR for lung cancer of 263 (compared to an unadjusted value of 176). Disregarding uncertainties about the absolute value of the SMR in this population, the important finding is that relatively lower tobacco smoking causes the study to under-estimate the effect of cadmium, not to over-estimate it.

A second factor which could explain the excess of lung cancer deaths is uncontrolled exposure to arsenic. Even excluding workers hired before 1926 does not exclude all arsenic exposure. Workers who unload, roast, and calcine feedstock were exposed to arsenic contaminating feedstock even after 1926.

Adjustments for arsenic in the published version of our paper were based upon urine arsenic levels and airborn arsenic concentrations since 1940. Urine arsenic levels measured since 1960 indicate that actual arsenic exposures have been approximately at background, averaging only 46 ug/l. Using the urine arsenic data, measures of airborne arsenic, and the OSHA risk assessment model, we estimated that residual arsenic exposure should result in no more than 0.77 lung cancers.

Critics of our study claim that arsenic concentration in feedstock was actually much higher from 1926-1940 than in subsequent years, and that we underestimated exposure to arsenic in the early years, before 1940. They base this criticism upon historical records of arsenic in feedstock entering the plant. (Slide- Arsenic concentration in feedstock, White) Dr. Lowell White, a former employee of the company, compiled this figure and submitted it in testimony to the EPA. It shows estimates of the concentration of arsenic in feedstock entering the plant by year. Dr Steve Lamm refered to the figure in a presentation at the 1985, AIHA conference. To quote, "Plant history indicates three industrial eras with respect to arsenic at this work site--prior to 1926 when the arsenic plant on site was active, 1926-1940 when the feedstock contained about 5% arsenic, and after 1940 when the feedstock arsenic dropped to about 1%."

We have obtained the records of arsenic in feedstock from the company and have analysed the data through 1958. (Slide- Arsenic Concentrations in Feedstock, 1924-58) We calculated the actual geometric mean of arsenic concentration in feedstock and found the actual value to be lower than the ASARCO estimates indicate. Arsenic concentrations in feedstock prior to 1926 were high, approximately 60%. There was a precipitous drop in 1925, and arsenic concentrations in feedstock thereafter have been below 5% with the exception of the years 1930 and 1931.

(Slide- Total arsenic in feedstock per year) The total number of pounds of arsenic processed per year show a similar pattern. Total pounds decreased

dramatically between 1925 and 1926, and remained relatively constant thereafter. Arsenic has been present in feedstock after 1925, but the pattern of arsenic intake does not justify excluding or separating out workers hired before 1940.

As stated, we are continuing to assess the separate and joint contributions of cadmium, arsenic, and smoking to the mortality experience of these workers. A nested case-referent study is in progress but will not be completed for some time.

To conclude, this population of cadmium workers offered a rare opportunity among occupational cohorts. The workers have had long-term, heavy exposure to cadmium. High quality industrial hygiene data exist since the 1940's. In addition, some data are available on cigarette smoking, as well as some data allowing us to infer past exposures to arsenic. The company is to be commended on collecting and preserving these records.

1) Thun MJ, Schnorr TM, Smith AB, Halperin WE, Lemen RA. Mortality among a cohort of U.S. cadmium production workers- an update. JNCI, 74;2:325-333, 1985.

2) Takenaka S, Oldiges M, Konig H, Hochrainer D, Oberdorster G. Carcinogenicity of inhaled cadmium chloride aerosols in W rats. JNCI 1983; 70:367-373.

3) U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, Current Intelligence Bulletin 42, Cadmium (Cd), September 27, 1984, DHHS (NIOSH) Publication No. 84-116.

4) U.S. Environmental Protection Agency, Office of Research and Development, Office of Health and Environmental Assessment. Updated mutagenicity and carcinogenicity assessment of cadmium. 1985, EPA-600/8-83-025A

5) Lamm SH. Methodological problems underlying the cadmium controversy. Paper presented at the 1985 American Industrial Hygiene Conference, Las Vegas, Nevada, May, 1985.

6) Lamm SH. Separating out sources of lung cancer risk at cadmium plant. Paper presented at

7) White LD. Testimony submitted to the metals subcommittee, Environmental Health Committee,, Science Advisory Board, Environmental Protection Agency, November 6, 1984.

8) White LD, Varner MO, Hine CH, Crookston F, Watson RB. An epidemiological study of cadmium-exposed workers. Paper presented at the 1985 American Industrial Hygiene Conference, Las Vegas, Nevada, May, 1985.

# Mortality Among A Cohort of U.S. Cadmium Production Workers- An Update

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RESULTS OF MORTALITY STUDY BY LEMEN ET. AL.,1976

EXCESS CANCER MORTALITY AMONG 292 CADMIUM PRODUCTION WORKERS

EMPLOYED FOR TWO OR MORE YEARS

| | DEATHS | SMR | 90%CI |
|---|---|---|---|
| RESPIRATORY CANCER | 12 | 235 | 136-381 |
| PROSTATIC CANCER | 4 | 347 | 119-796 |

## ADVANTAGES OF THE STUDY POPULATION

1) Opportunity for Prolonged Followup

   (82.5% of workers have 20+ years of followup)

2) Cohort Highly Exposed

3) Extensive Exposure Information Available

4) Some Data Available To Control For Potential Confounders

## METHODS

1) FOLLOWUP EXTENDED 5 YEARS, FROM 1974-78

2) SHORT TERM WORKERS INCLUDED TO PROVIDE INTERNAL COMPARISON

3) MORTALITY ANALYZED BY TWO EXPOSURE MEASURES

   (A) LENGTH OF EMPLOYMENT

   (B) CUMULATIVE EXPOSURE

4) EFFECT OF ARSENIC AND CIGARETTE SMOKING ASSESSED

ESTIMATES OF INHALATION EXPOSURES ($MG/M^3$)

IN HIGH EXPOSURE DEPARTMENTS OVER TIME*

| TIME PERIOD | SAMPLING | ROASTER | MIXING | CALCINE | FOUNDRY | RETORT |
|---|---|---|---|---|---|---|
| PRE-1950 | 1.0 | 1.0 | 1.5 | 1.5 | 0.8 | 1.5 |
| 1950-1954 | 0.6 | 0.6 | 0.4 | 1.5 | 0.1 | 0.2 |
| 1955-1959 | 0.6 | 0.6 | 0.4 | 1.5 | 0.1 | 0.2 |
| 1960-1964 | 0.6 | 0.6 | 0.4 | 0.4 | 0.1 | 0.2 |
| 1965-1976 | 0.6 | 0.6 | 0.4 | 0.15 | 0.04 | 0.2 |

* FROM SMITH ET AL.

VITAL STATUS OF WHITE MALE CADMIUM PRODUCTION WORKERS

BY DURATION OF EMPLOYMENT

| | EMPLOYED 6-23 MONTHS | | EMPLOYED 2+ YEARS | | TOTAL | |
|---|---|---|---|---|---|---|
| | # | (%) | # | (%) | # | (%) |
| ALIVE | 189 | (74) | 222 | (64) | 411 | (69) |
| DEAD | 60 | (23) | 119 | (35) | 179 | (29) |
| LOST TO FOLLOW UP | 8 | ( 3) | 4 | (1) | 12 | ( 2) |
| TOTAL | 257 | | 345 | | 602 | |

Overall Mortality Among White Male Cadmium Workers
With Six or More Months of Production Work, 1940-69

| | Observed | Expected | SMR | 95% Confidence Interval |
|---|---|---|---|---|
| All Causes of Death | 179 | 188.87 | 95 | 81 - 110 |

MORTALITY FROM SELECTED CAUSES OF DEATH AMONG WHITE MALES WITH SIX OR MORE MONTHS OF CADMIUM PRODUCTION WORK, 1940-69.

| CAUSE OF DEATH | ICD 7TH REVISION | OBSERVED | EXPECTED | SMR | 95% CONFIDENCE INTERVAL |
|---|---|---|---|---|---|
| ALL CANCER | 140 - 199 | 41 | 36.46 | 112 | 81 - 153 |
| RESPIRATORY CANCER | 160 - 164 | 20 | 12.15 | 165 | 101 - 254 |
| GENITO-URINARY CANCER | 177 - 182 | 6 | 4.45 | 135 | 49 - 293 |

MORTALITY FROM LUNG CANCER (ICD 162-163) BY DATE OF HIRE,
WHITE MALE CADMIUM PRODUCTION WORKERS

| | OBSERVED | EXPECTED | SMR | 95% CONFIDENCE INTERVAL |
|---|---|---|---|---|
| HIRED PRIOR TO 1/1/26 | 4 | 0.56 | 714 | 195-1829 |
| HIRED ON OR AFTER 1/1/26 | | | | |
| OVERALL COHORT | 16 | 10.87 | 147 | 84-239 |
| ≥2 YEARS EMPLOYMENT | 16 | 7.00 | 229 | 131-371 |

## Deaths Due to Genitourinary Cancer Among Cadmium Production Workers

| Case | 7th ICD Revision | Death Certificate Diagnosis | Age at Death | Date of Death | Latency (Yrs.) | Duration (Yrs.) |
|---|---|---|---|---|---|---|
| 1 | 177* | Carcinoma of prostate | 64 | 4/51 | 38 | 32 |
| 2 | 177 | Metastatic carcinoma of prostate | 70 | 2/72 | 32 | 4 |
| 3 | 177 | Probable carcinoma of prostate | 79 | 12/60 | 31 | 18 |
| 4 | 180 | Renal cell carcinoma | 64 | 11/76 | 43 | 9 |
| 5 | 181 | Ca of bladder | 49 | 10/61 | 15 | 2 |
| 6 | 181 | Metastatic, transitional cell type, ca of bladder | 63 | 10/77 | 22 | 1 |

* - Date of first employment was prior to 1/1/26.

Lung Cancer (ICD 162-163) Mortality By Duration of Employment, White Males Hired on or After 1/1/26

| DURATION OF EMPLOYMENT | No. of Deaths | Mortality Rate* | SRR** |
|---|---|---|---|
| 6-23 Months | 0 | 0 | - |
| 2-9 Years | 9 | 15.73 | 2.2 |
| 10-19 Years | 3 | 14.28 | 2.0 |
| 20+ Years | 4 | 16.28 | 2.2 |
| US White Males | - | 7.27 | 1 |

* Rate x 10,000 person years, directly standardized for age and calendar time to the person-years distribution of the overall cadmium cohort.

** Standardized rate ratio (SRR)= directly standardized mortality rate of subgroup/summary rate for US white males.

## COMPARISON WITH US RATES

### LUNG CANCER (ICD 162-163) MORTALITY BY CUMULATIVE EXPOSURE TO CADMIUM: WHITE MALES HIRED ON OR AFTER 1/1/26

| CUMULATIVE EXPOSURE (MG-DAYS/$M^3$) | FORTY YEAR T.W.A. EQUIVALENT+ | PERSON-YEARS AT RISK | DEATHS | SMR* | SRR** |
|---|---|---|---|---|---|
| $\leq 584$ | $\leq 40$ UG/$M^3$ | 7005 | 2 | 53 | .48 |
| 585-2920++ | 41-200 UG/$M^3$ | 5825 | 7 | 152 | 1.55 |
| $\geq 2921$ | $\geq 200$ UG/$M^3$ | 2214 | 7 | 280 | 3.45 |
| | US WHITE MALES | - | - | 100 | 1.00 |

## COMPARISON WITH COLORADO RATES

## LUNG CANCER (ICD 162-163) MORTALITY BY CUMULATIVE EXPOSURE TO CADMIUM: WHITE MALES HIRED ON OR AFTER 1/1/26

| CUMULATIVE EXPOSURE (MG-DAYS/$M^3$) | FORTY YEAR T.W.A. EQUIVALENT+ | PERSON-YEARS AT RISK | DEATHS | SMR | SRR |
|---|---|---|---|---|---|
| $\leq 584$ | $\leq 40$ UG/$M^3$ | 7005 | 2 | 76 | |
| 585-2920++ | 41-200 UG/$M^3$ | 5825 | 7 | 212 | |
| $\geq 2921$ | $\geq 200$ UG/$M^3$ | 2214 | 7 | 387 | |
| | US WHITE MALES | - | - | 100 | 1.00 |

CIGARETTE SMOKING HABITS, 1965
CADMIUM WORKERS V.S. U.S. POPULATION.

| | NONSMOKERS | MODERATE SMOKERS (1-24/DAY) | HEAVY SMOKERS (25+/DAY) |
|---|---|---|---|
| CADMIUM WORKERS | 50.7% | 39.2% | ~~00.0%~~ 10.1 |
| U.S. 1965 | 27.1% | 53.0% | 20.0% |

TECHNIQUE USED TO ADJUST FOR CIGARETTE SMOKING.

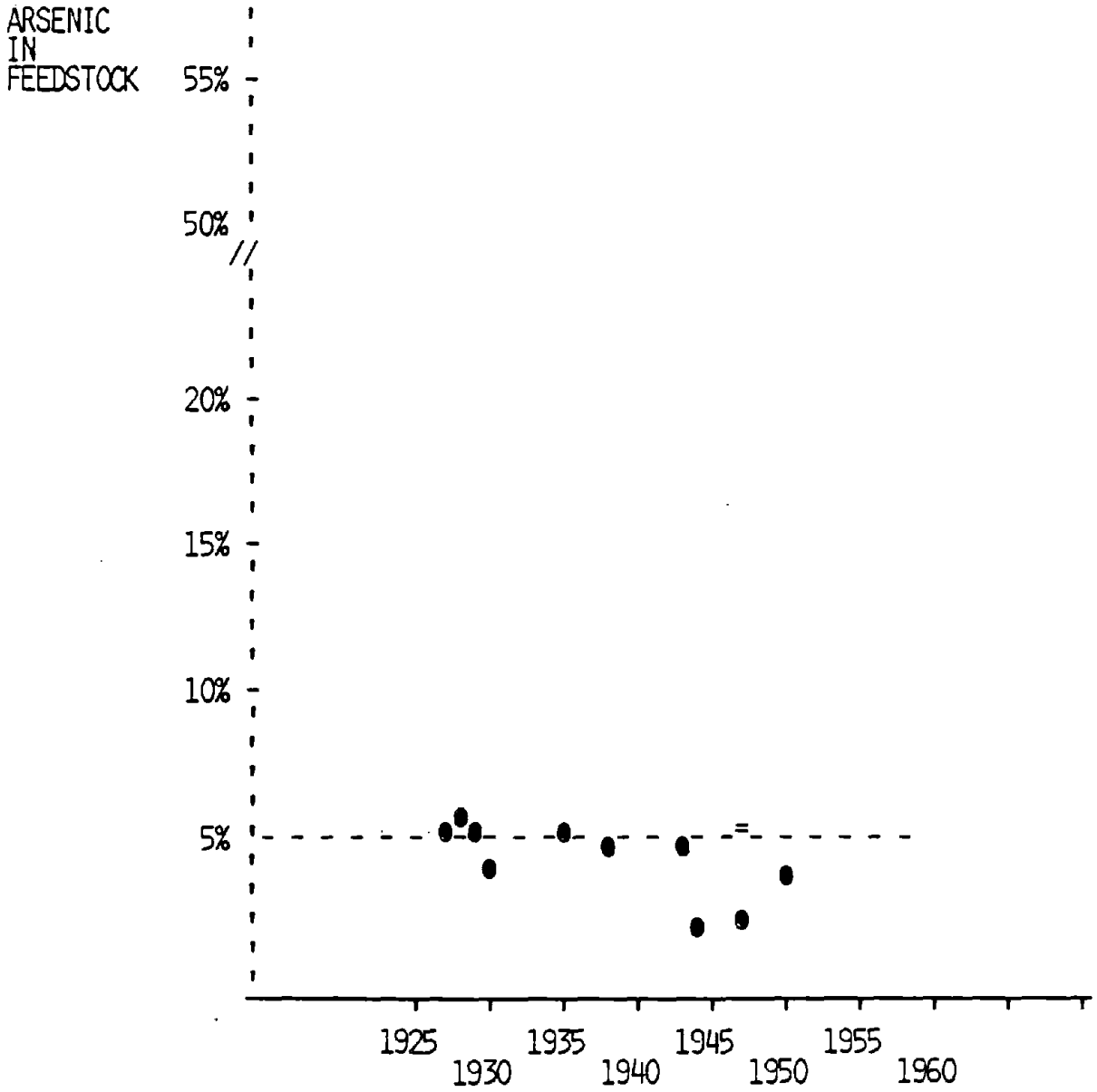
PERCENT OF POPULATION, 1965*

| | NONSMOKERS (1x) | MODERATE+ SMOKERS (10x) | HEAVY++ SMOKERS (20x) | RATE RATIO OF OVERALL POPULATION RELATIVE TO NONSMOKERS | RATE RATIO RELATIVE TO U.S. |
|---|---|---|---|---|---|
| POPULATION | | | | | |
| EXPOSED | 50.7% | 39.2% | ~~00.0%~~ 10.1 | 6.45 | 0.67 |
| U.S. | 27.1% | 53.0% | 20% | 9.571 | 1.00 |



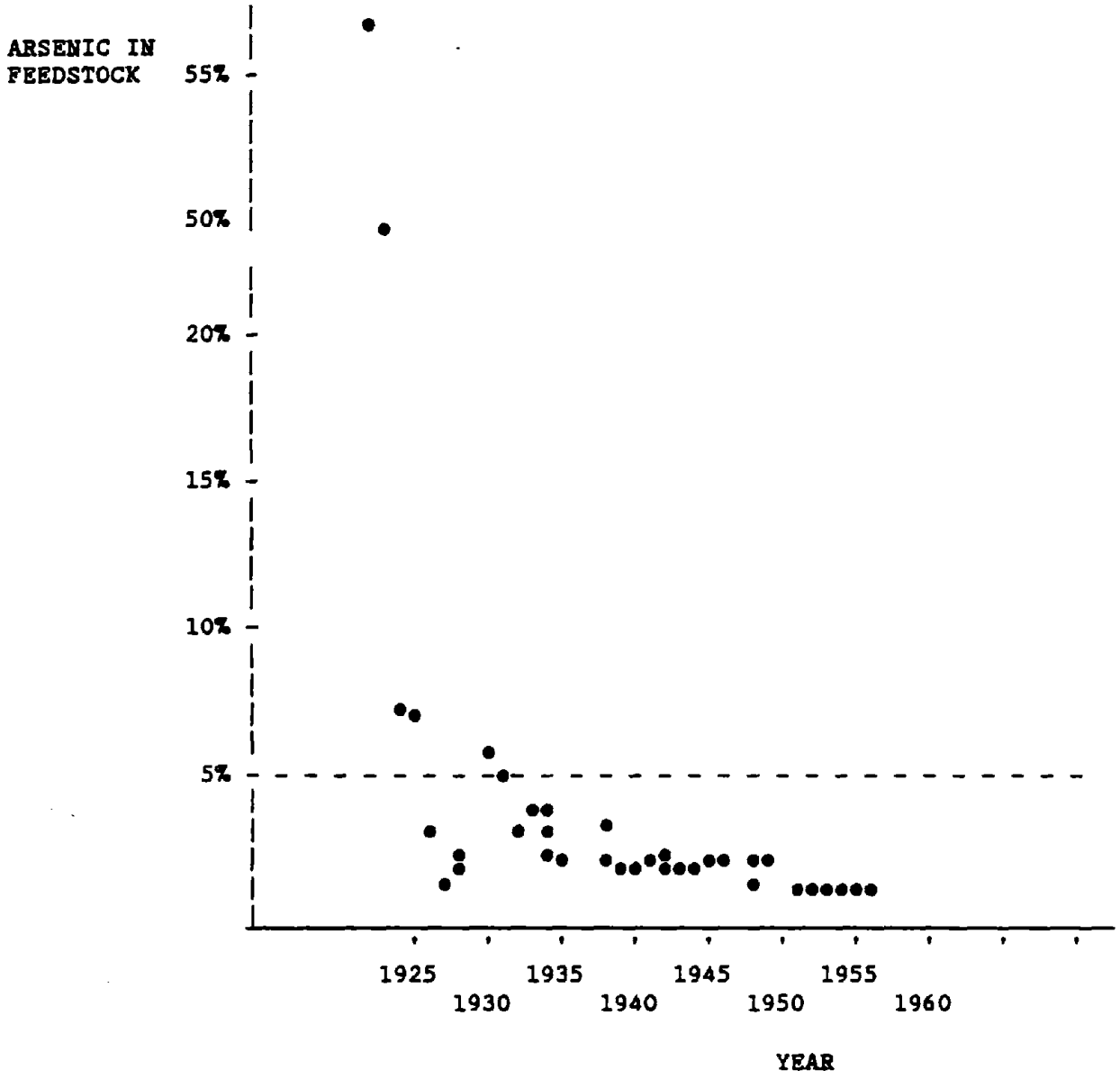
* USABLE INFORMATION AVAILABLE ON 273 PERSONS
(1) AXELSON O, 1978.
(2) BLAIR A, SPIRITUS, 1981.
(+) 1-24 CIGARETTES/DAY
(++) 25+ CIGARETTES/DAY

AVERAGE PERCENTAGE OF ARSENIC IN RECEIPTED FEED MATERIAL, GLOBE PLANT, 1928-52 AS REPORTED BY INDUSTRY SCIENTISTS*

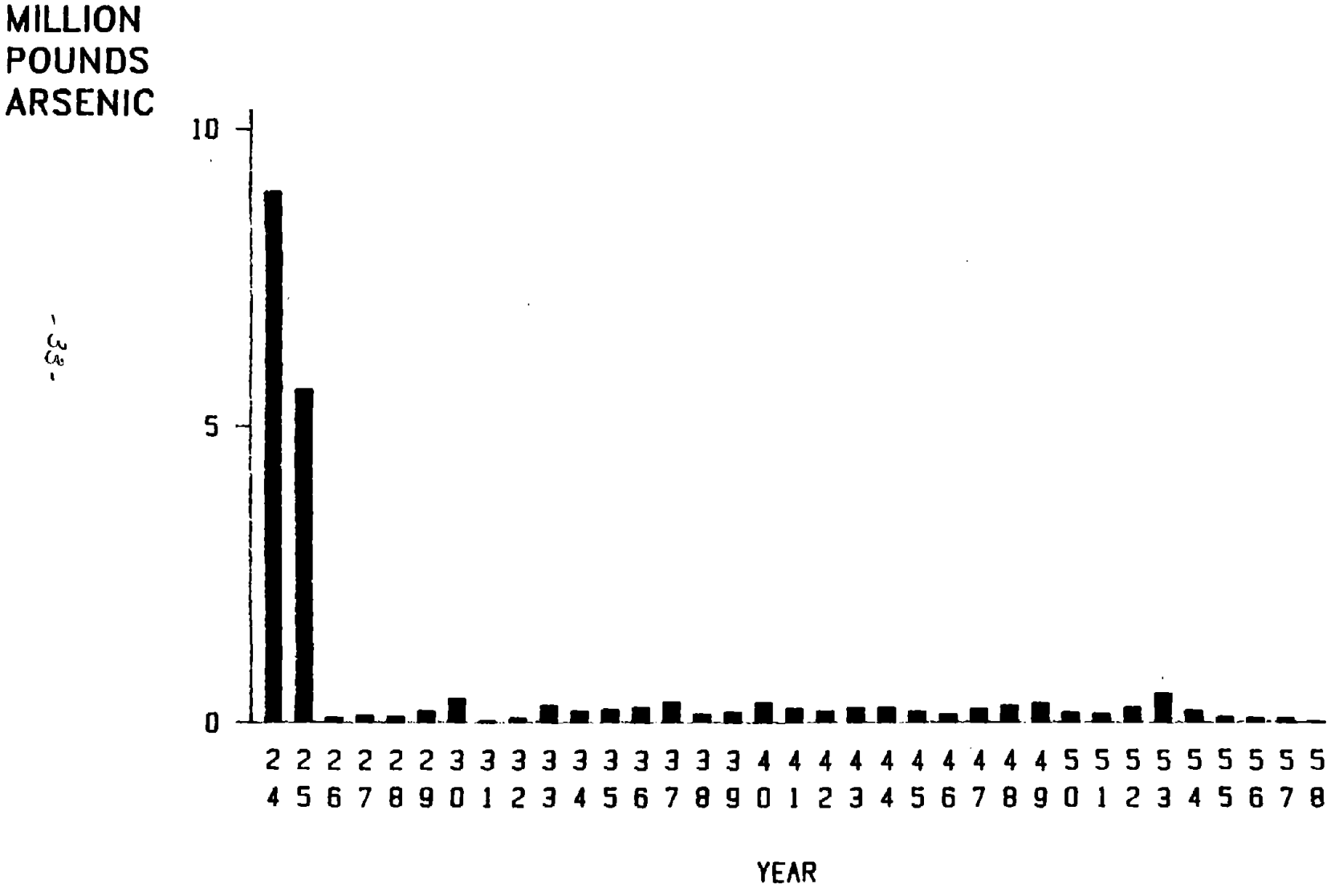



* ORIGINAL SOURCE DOES NOT SPECIFY WHETHER AVERAGE SIGNIFIES ARITHMETIC OR GEOMETRIC MEAN, NOR MEANING OF INTERVALS.

PERCENTAGE ARSENIC IN RECEIPTED FEED MATERIAL, GLOBE PLANT
COMPUTED AS GEOMETRIC MEAN, NIOSH 1985



## TOTAL POUNDS OF ARSENIC PROCESSED PER YEAR
## CADMIUM PRODUCTION PLANT, 1924–1958



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15. Supplementary Notes

16. Abstract (Limit: 200 words) A retrospective cohort mortality study was conducted among a group of workers exposed to cadmium (7440439), to determine specific causes of death among these workers. This report reviewed the findings of this study and presented additional findings concerning exposures to other hazards at this facility. The study examined the causes of death for 292 cadmium workers who worked at least 2 years at a small facility that recovered cadmium from bag house dust. Exposures included dusts of cadmium-oxide (1306190) and cadmium-sulfide (1306236) and to fumes of cadmium itself. Over a two fold excess of deaths from respiratory cancer was noted among these workers when compared with the general population and over a three fold excess of deaths from prostatic cancer among the men. A follow up of this study cohort revealed that all cause mortality was slightly below that of the United States male population. The Standardized Mortality Ratio for circulatory disease was 65. Deaths due to respiratory cancer were 65% above the number expected. All of the 20 deaths from respiratory cancer were due to cancer of the lung, trachea and bronchus. Deaths due to genitourinary cancer were 35% above expected levels. The effects of arsenic (7440382) exposure in workers hired prior to 1926 were considered. An over seven fold increase in lung cancer mortality was observed among persons hired prior to 1926.

17. Document Analysis a. Descriptors

b. Identifiers/Open-Ended Terms NIOSH-Author, Cadmium-poisoning, Cadmium-compounds, Mortality-data, Cancer-rates, Occupational-exposure, Lung-cancer, Risk-factors, Epidemiology, Cadmium-dust

c. COSATI Field/Group

| 18. Availability Statement | 19. Security Class (This Report) | 21. No. of Pages 33 |
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| | 22. Security Class (This Page) | 22. Price |