

Mortality among workers in a die-casting and electroplating plant

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SILVERSTEIN M, MIRER F, KOTELCHUCK D, SILVERSTEIN B, BENNETT M. Mortality among workers in a die-casting and electroplating plant. *Scand j work environ health* 7 (1981): suppl 4, 156—165. A proportional mortality and case-referent analysis of 238 deaths among hourly employees in an automobile hardware manufacturing plant was conducted. The major operations of the plant were zinc die casting and electroplating. Chemical exposure included die-casting emissions and mists from chrome and nickel plating. The chief proportional mortality finding was a significant excess of lung cancer among both white men and women. A case-referent analysis indicated a possible association between lung cancer and work in certain departments. The findings support the hypothesis of a work-related carcinogenic risk. Follow-up recommendations have been made.

Key terms: chromates, chromic acid, lung cancer, nickel, polycyclic aromatic hydrocarbons.

In March 1980 one of us (MB) reported an apparent excess of lung cancer deaths (unpublished results) after reviewing 225 death certificates of local union members and retirees who had died from 1 January 1974 to 31 December 1978. The study group had worked in a midwestern plant which started operations in the early 1950s and manufactured hardware and trim components for use primarily in the automobile industry.

During the 1950s and 1960s, the major operations of the plant were zinc alloy die casting; buffing, polishing and metal-cleaning of zinc and steel parts; and electroplating with copper, nickel and chrome. Since 1971 other operations, including urethane plastics fabrication, have been sub-

stituted for much of the plating and die casting, which have continued at a reduced volume. The specific chemical exposures at this plant and others of its type have never been precisely characterized nor adequately studied for carcinogenic effect.

This study extends the preliminary investigation by exploring the association of lung cancer risk with work histories. Proportional mortality and case-referent findings are reported.

Materials and methods

Study cohort

The study cohort consisted of hourly employees and retirees with at least 10 a of credited pension service in the plant who died between 1 January 1974 and 31 December 1978. The data used for analyses were 238 death certificates and employer personnel records. Four persons for whom death certificates could not be obtained were excluded from the analysis.

Fourteen deceased employees with less than 10 a of credited service were known

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to us but excluded from the analysis. These 14 were part of a larger group of workers who left employment before accruing 10 a of credited service and subsequently died. The total number and characteristics of this group were unknown, and it could not be assumed that those 14 who were identified were a representative sample.

Ascertainment of the study cohort was concluded to be reasonably complete because several different sets of pension, insurance, and benefits records have been cross-checked. These types of records have historically been maintained quite accurately. Deceased hourly employees who have transferred to salaried positions or other plants of this company prior to death were not identified, but the number is unlikely to be substantial.

The distribution of deaths by age, race, and sex is found in table 1.

Proportional mortality methods

Death certificates were coded for underlying cause of death by a trained nosologist according to the *Eighth Revision of the International Classification of Disease Adapted* (ICDA). The proportions of deaths from specified causes for groups defined by sex, race, age, and year of death were determined. They were compared with US national mortality statistics. Calculations using comparable state and county figures produced similar results, and they are not presented in this report. Results were reported as the ratio

of observed to expected deaths, the proportional mortality ratio (PMR) (17) in various categories. Mantel-Haenszel chi-square determinations were used for the statistical testing (14).

Total years of actual plant service (excluding layoffs, sick leave, etc) and latency (approximated as the time between hiring and death) were determined for each deceased worker. There was a narrow range of latency because most of the workers in the study were hired between 1953 and 1955 and all died within a 5-a period.

Case-referent analysis

A case-referent analysis was made to test the association of lung cancer risk with work experience. All 28 white male and 10 white female lung cancer deaths were matched to study population referents of the same sex and race who died of non-malignant cardiovascular disease (ICDA 390-458) (table 2). Two referents were identified whose age at death was within 2 a of each white male case. One referent was identified whose age at death was within 3 a of each white female lung cancer case. The cases and referents were compared for length of employment in individual departments. Odds ratios were used to approximate the relative risk of lung cancer for workers employed in departments where plating and die casting was done (13). Statistical significance was determined by chi-square and Fisher exact tests. There was no significant correlation between matching factors and the areas

Table 1. Cohort characteristics — Age at death.

Age (a)	White males		White females		Nonwhite males		Nonwhite females	
	N	%	N	%	N	%	N	%
35—39	3	1.7	0	0	2	12.5	0	0
40—44	4	2.3	2	4.1	0	0	0	0
45—49	13	7.6	10	20.4	2	12.5	0	0
50—55	30	17.4	8	16.3	3	18.8	1	100
55—59	24	14.0	12	24.5	3	18.8	0	0
60—64	31	18.0	10	20.4	4	25.0	0	0
65—69	27	15.7	3	6.1	1	6.3	0	0
70—74	28	16.3	4	8.2	1	6.3	0	0
75—79	11	6.4	0	0	0	0	0	0
≥80	1	0.6	0	0	0	0	0	0
Total	172	100	49	100	16	100	1	100

Table 2. Characteristics of the lung cancer cases and referents.

	Cases (mean $\pm$ SD)	Referents (mean $\pm$ SD)
White males		
Number	28	56
Age at death (a)	61.9 $\pm$ 7.6	61.9 $\pm$ 7.4
Total actual years in plant	16.3 $\pm$ 3.5	15.8 $\pm$ 3.9
Years in plant, 10 or more before death	11.1 $\pm$ 2.0	10.3 $\pm$ 2.8
White females		
Number	10	10
Age at death (a)	55.0 $\pm$ 7.0	54.3 $\pm$ 6.7
Total actual years in plant	15.8 $\pm$ 3.0	14.3 $\pm$ 3.7
Years in plant, 10 or more before death	8.7 $\pm$ 2.5	9.0 $\pm$ 2.7

under study; consequently it was not necessary to analyze the data by matched pairs or triplets.

The work history of the cases and referents was abstracted from company personnel records. Total years of actual service (excluding periods of layoff, sick leave, etc) in each department were tabulated for each individual. In addition the years of experience in each department contributing 10 a or more prior to death were determined.

Production process and chemical exposures

Quantitative chemical exposure profiles for either individual departments or workers were not determined. Qualitative assessments were made on the basis of plant inspections by Union industrial hygienists, discussions with high-seniority hourly employees, and a review of historical documents describing plant operations.

The major products of the plant during the 1950s and 1960s were small zinc die castings plated with chrome, nickel, and copper for use in automobiles. The production of castings started with zinc metal which was alloyed with 4 % aluminum and less than 1 % magnesium in the floor furnaces and transferred to individual die-casting machines. Molten metal was maintained at 760–860°F (404–460°C)

and injected under pressure into steel dies with surfaces sprayed with a lubricant.

Next the cooled castings were trimmed, buffed, and polished prior to electroplating involving successive applications of copper, nickel, and chrome.

A remelting of scrap metal took place in large reverberatory furnaces in a partially enclosed room. Before 1970 up to 15 % of this scrap was at least partially plated. This remelting was termed "burning off chrome."

Two major classes of chemical exposures with potential health consequences were identified. One was the die-casting emissions consisting of metal fume and organic combustion products from the contact of hot metal with die lubricants and hydraulic oils. The composition of these organic materials has not been determined. The second was mists associated with nickel- and chrome-plating solutions and a variety of plating additives.

Very little industrial hygiene data are available. Several airborne chromic acid levels in 1978 were less than one-half the existing permissible exposure limit (PEL) of 100 $\mu\text{g}/\text{m}^3$ of the Occupational Safety and Health Administration. One breathing zone sample result from 1959 was available. It was five times greater than the current PEL. Past reports of chrome ulcers and perforated nasal septa are consistent with this higher level. Worker exposures were reported to have been progressively reduced through increased ventilation, machine enclosures, and mechanization over the period of employment of the deceased workers in the study.

Most workers in the study cohort were exposed to a mixture of chemicals because the plant design was open with few barriers between or within departments and because many of the major departments contained a mix of plating and die-casting operations. In addition many workers changed jobs and departments frequently.

Results

Proportional mortality

PMRs were calculated for 54 categories of cause of death. They are summarized for

major causes of death and specific cancer sites among white men and women in tables 3 and 4. Only 17 deaths were recorded for nonwhite men and women. There were two cancer deaths in these groups, and no statistically significant excess or deficit for any cause of death.

All cancers. The PMR for all cancers for all the sex/race groups combined was 1.27 ($p < 0.05$). For the white men it was 1.35 ($p < 0.05$). For the white women the PMR of 1.27 was not statistically significant.

Lung cancer. The PMR for lung cancer was 2.09 for all the sex/race groups com-

bined, 1.91 for white men, and 3.70 for white women ($p < 0.001$ for all groups.)

Other cancers. There were two cases of adrenal gland cancer among the white men, although only one was coded as the underlying cause of death and treated as a cancer death for statistical purposes. One 49-year-old white man was found, on autopsy, to have an angiosarcoma of the heart and major vessels. The man's cause of death had been identified as cardiovascular disease on the death certificate and was so treated in the analysis. Several other specific types of cancer were present in excess, although the numbers were small and did not reach statistical significance.

Table 3. Proportional mortality ratios — All causes.

Cause of death ^a	White men (Observed/expected)	White women (Observed/expected)
All malignant neoplasms (140—209)	53/39.36 = 1.35 *	23/18.13 = 1.27
Ischemic heart disease (410—413)	62/66.47 = 0.93	10/10.48 = 0.95
Stroke (430—438)	9/10.18 = 0.88	4/3.49 = 1.15
All respiratory diseases (460—519)	14/10.55 = 1.33	2/2.19 = 0.92
All pneumonia (480—486)	5/3.20 = 1.56	0/0.78 = 0.00
Emphysema (492)	2/2.85 = 0.70	1/0.40 = 2.51
All digestive diseases (520—577)	10/9.03 = 1.11	1/3.10 = 0.32
Cirrhosis of liver (571)	9/5.60 = 1.61	1/1.88 = 0.43
Accidents and suicide (E 800 — E 999)	10/13.07 = 0.77	3/3.38 = 0.89
All other	14/23.34 = 0.60	6/8.23 = 0.73

^a According to the International Classification of Disease, eighth revision.

* $p < 0.05$.

Table 4. Proportional mortality ratios — Specific cancer sites.

Site ^a	White men (Observed/expected)	White women (Observed/expected)
All malignant neoplasms (140—209)	53/39.39 = 1.35 *	23/18.13 = 1.27
Stomach (151)	4/1.58 = 2.54	0/0.42 = 0.00
Large Intestine (153)	1/3.45 = 0.29	2/1.64 = 1.22
Liver (155, 156)	2/0.56 = 3.55	0/0.27 = 0.00
Pancreas (157)	3/2.16 = 1.39	1/0.71 = 1.40
Respiratory system (160—163)	30/15.37 = 1.95 ***	10/2.80 = 3.57 ***
Lung (162, 163)	28/14.68 = 1.91 ***	10/2.70 = 3.70 ***
Larynx (161)	2/0.61 = 3.30	0/0.07 = 0.00
Lymphosarcoma, reticulosarcoma (200)	2/0.70 = 2.85	0/0.30 = 0.00
Leukemia (204—207)	1/1.33 = 0.75	1/0.48 = 2.08
Skin (172, 173)	1/0.71 = 1.42	1/0.25 = 3.94
Kidney (189)	1/1.04 = 0.96	0/0.28 = 0.00
Buccal cavity, pharynx (140—149)	1/1.29 = 0.77	0/0.30 = 0.00
Prostate (185)	3/2.14 = 1.40	—
Uterus, corpus (181, 182)	—	1/0.50 = 1.98
Breast (174)	—	5/4.66 = 1.07
Other and unknown	4/5.64 = 0.71	2/4.93 = 0.41

^a According to the International Classification of Disease, eighth revision.

* $p < 0.05$, *** $p < 0.001$.

Table 5. Proportional mortality ratio results for white males by years of service — Lung cancer.

Actual length of service in plant (a)	Total number of deaths	Observed number of lung cancer deaths	Expected number of lung cancer deaths	Observed/expected deaths
< 15	75	10	6.06	1.65
≥ 15	97	18	8.62	2.09***
Total	172	28	14.68	1.91***

*** $p < 0.001$.

Table 6. Proportional mortality ratio results for white males by years of latency — Lung cancer.

Latency (a)	Total number of deaths	Observed number of lung cancer deaths	Expected number of lung cancer deaths	Observed/expected deaths
< 22.5	108	16	8.98	1.78 *
≥ 22.5	64	12	5.70	2.11 **
Total	172	28	14.68	1.91 ***

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Noncancer deaths. There were no significant excesses or deficits for any noncancer causes of death. Among the white men the total respiratory disease deaths were in slight excess, but the number of emphysema deaths was not greater than expected.

Lung cancer and years of service. White men who worked in the plant fewer than 15 a before death had a lung cancer PMR of 1.65 (table 5). Those with more than 15 a of actual service had a lung cancer PMR of 2.09 ($p < 0.001$). The lung cancer PMR for white males did not vary substantially with age at death (PMR = 1.87 for those whose age at death was less than 60; PMR = 1.94 for those whose age at death was 60 or greater).

Lung cancer and latency. The deceased white males were divided into two groups, those with less and those with more than 22.5 a³ between hiring and death (table 6). The PMR increased from 1.78 ($p < 0.05$) to 2.11 ($p < 0.01$) as latency increased.

Case-referent analysis

Results presented with respect to the cases and referents only consider the time worked 10 a or more prior to death.

³ The approximate midpoint of the latencies for lung cancer.

(Additional calculations were made using the employees entire work experience. The pattern of findings was essentially unchanged.) The results were as follows.

1. The percentages of actual years worked in various departments by the cases and referents are indicated in table 7.

White male cases spent 11.8 % of their employment in department 5 in comparison to the corresponding 4.3 % for the referents. (1.33 ± 0.44 a compared to 0.46 ± 0.15 a, $p < 0.05$, t-test). This was the only department for which there was a more than twofold difference.

The pattern for white women was different. The fraction of time spent in departments 3 and 7 by the cases was four times greater than that of the referents; however these differences were not statistically significant.

2. Odds ratios were calculated separately depending on whether employees had ever worked in a department, had worked there at least 1 a, or had worked there at least 5 a (table 8).

For the white men there were three departments (departments 5, 8, 38) for which the odds ratio increased with duration of work in that department. This trend was greatest for department 5. The odds ratio of 9.17 for those who worked ≥ 5 a in department 5 was statistically

significant ($p = 0.04$, Fisher's exact test). The cases and referents who worked in department 5 did not differ significantly with respect to either age at death or years of actual plant service.

For white women there were two departments (departments 3, 7) for which the odds ratio increased with increasing duration of work. These findings were based

on a small number of observations and were not statistically significant.

Discussion

1. The proportional mortality analysis demonstrated a statistically significant excess of total cancer deaths ($PMR = 1.27$) in

Table 7. Percentages of actual years worked by cases and referents in various departments (10 a or more before death).

Department	White men		White women	
	Cases	Referents	Cases	Referents
1	2.1	2.4	9.2	27.3
2	1.5	1.0	8.1	11.2
3	5.2	7.6	23.9	5.3
4	5.1	6.8	8.8	7.2
5	11.8	4.3	16.6	9.9
6	3.5	4.1	11.0	10.1
7	6.3	6.4	14.2	2.9
8	6.2	4.1	6.6	7.0
9	1.0	2.3	0.7	16.9
10	2.3	1.5	0.9	2.2
11, 20 ^a	17.0	27.4	0	0
34, 69 ^a	8.2	9.4	0	0
38, 60 ^a	19.2	13.0	0	0
All others	10.6	9.7	0	0
Total	100	100	100	100

^a These departments were assigned different numbers at different times. Department 11 was equivalent to department 20, etc.

Table 8. Odds ratios for work of varying duration by department — Lung cancer cases vs referents.

Department	White men			White women	
	Ever vs never worked	More vs less than 1 a	More vs less than 5 a	Ever vs never worked	More vs less than 1 a
1	0.68	0.65	1.0	1.00	0.43
2	0.77	2.07	1.0	0.38	0.43
3	1.0	0.55	0.65	4.0	6.0
4	0.65	0.26	0.65	6.0	2.67
5	1.36	2.18	9.17 ^a	5.44	1.56
6	1.62	1.17	1.0	9.0	0.38
7	0.84	1.16	0.65	1.56	2.25
8	0.68	1.42	4.23	—	—
9	0.40	0.31	1.0	0.43	0.0
10	1.10	2.12	1.0	1.0	0.0
20, 11 ^b	0.63	0.68	0.46	—	—
38, 60 ^b	1.25	1.25	1.52	—	—

^a Only a small number of observations were involved. The chi-square was 5.1 ($p < 0.05$) uncorrected and 3.22 ($0.05 < p < 0.10$) with the Yates continuity correction. With the Fisher exact test $p = 0.04$.

^b Department 11 was equivalent to 20 and department 38 was equivalent to 60 because of changes in department numbers.

this cohort of 238 deceased hourly employees from a large die-casting and plating plant. The excess was almost entirely accounted for by lung cancer deaths. The lung cancer increase was substantial in magnitude for both white men (PMR = 1.91, $p < 0.001$) and white women (PMR = 3.70, $p < 0.001$).

2. The evidence does not support the "healthy worker effect" as an explanation for the cancer increases. Cancer PMRs may sometimes be high among groups who have low cardiovascular disease death rates and normal cancer death rates. The combination of a low death rate from other causes and a normal death rate from cancer would mathematically result in an increased proportion of cancer deaths (15, 16). Such an apparent increase in cancer deaths would be expected to be distributed among of all sites. However, in this investigation, the elevated cancer proportion was essentially limited to lung cancer, other types being at, or close to, expected levels.

The ratio of deaths from one cancer type to all cancer deaths, or the proportional cancer mortality ratio (PCMR), is a quantitative demonstration of this observation (3). For lung cancer among white men in this cohort the PCMR was 1.42 ($p < 0.05$). For white women it was 2.92 ($p < 0.001$) (table 9.)

3. The evidence does not support increased cigarette smoking as the explanation for the cancer increases. Such an explanation would require that employees in this plant, as a group, have smoked sufficiently more than the general population to account for the cancer increase. But were this the case, an increase in other

smoking-related illnesses would also be expected. Workers in this cohort, however, did not have excess deaths from emphysema, coronary heart disease, or cancer of the bladder, although there was a small but not significant excess of deaths from respiratory diseases.

4. There is additional positive evidence to support the hypothesis that the cancer excess was work-related. In particular the lung cancer excess increased with the workers' years of actual service in the plant and with increasing latency periods.

The case-referent analysis revealed an association between lung cancer and work in certain departments where there was mixed exposure to emissions from die-casting and plating operations. Most of these findings were based on small numbers and only the association of lung cancer among white men with work for ≥ 5 a in department 5 was statistically significant. Prior to 1971 department 5 was one of the major die-casting and plating areas of the plant.

Odds ratios increased with duration of work in four other departments (departments 8 and 38 among white men; departments 3 and 7 among white women); however these associations were weak. In department 38 (Skilled Trades-Maintenance) workers tended to have mobility throughout the plant and most were not permanently assigned to a specific location. This situation was in contrast with that of department 20 (Skilled Trades-Tool-Room), where the workers had less plant-wide mobility than maintenance workers. There was no apparent relation between tool-room work and lung cancer. In the 1950s and 1960s, department 7 was an area

Table 9. Proportional cancer mortality for lung cancer deaths.

Group	Total number of deaths (All cancers)	Observed number of deaths (Lung cancer)	Expected number of deaths (Lung cancer)	Observed/ expected deaths
White males	53	28	19.77	1.42*
White females	23	10	3.43	2.92***
Black males	2	1	0.72	1.38
Black females	0	0	0.00	—
Total	78	39	23.92	1.63***

* $p < 0.05$, *** $p < 0.001$.

of substantial plating and die-casting activity. There was also die-casting and plating activity, though to a smaller degree, in department 3.

The odds ratios were, in general, lowest in departments of low chemical exposure (departments 1, 4, 20 among white men).

While the case-referent findings suggested certain relationships, they lacked complete internal consistency. Department 6, for example, was probably the most similar to department 5 in the nature of chemical exposures in the 1950s and 1960s; yet there was no increasing trend of relative risk among white men. Work in department 4 was judged to have low chemical exposure; yet the odds ratios for white women were elevated.

5. The epidemiologic findings must be interpreted in light of knowledge of the toxicology of chemical exposures thought to have occurred.

The apparent chemical exposure of greatest extent in this plant was smoke from die-casting operations which may contain polycyclic aromatic hydrocarbons (PAH). PAH exposure has not been adequately studied in die-casting and heat-treating operations of the type found in the study plant. However, mixtures of polycyclic aromatic hydrocarbons, including known carcinogens such as benzo(a)pyrene, commonly arise from the pyrolysis of organic matter and are common industrial air contaminants. Their presence has been demonstrated in foundry and forging operations (7, 8, 10, 25) and is a possibility where hot metal comes in contact with lubricating oils, coolants, hydraulic fluids, or organic resins and binders.

The chemical exposures in this plant with the best-documented health consequences are compounds of chrome and nickel (9, 24). Mortality studies of workers exposed to nickel oxide, nickel sulfide, and carbonyl in refineries have found excesses of lung and nasal cancers (5, 11, 18). There are a few case reports of respiratory cancer among workers exposed to nickel in nonrefining operations such as plating and grinding, but no epidemiologic data are available. We are unaware of any animal studies of the inhalation of the soluble nickel compounds used in the plating operations (21).

Studies of chrome platers have produced contradictory results. A retrospective study of chrome platers in 54 British plants found an excess of cancers of the lung, pleura, gastrointestinal tract, and other sites in a comparison with matched referents (23). Recently Blair reported significant proportional excesses of cancer of the esophagus and liver among deceased metal platers assumed to be exposed to chromium, nickel, and degreasing solvents (1). A third study found no cancer excess in 48 deaths among electroplaters exposed to chromic acid (3).

Three other types of occupational exposure to chromium compounds have received attention. Mortality studies have found increased rates of respiratory cancer among workers in the chromite ore industry (6, 22). Exposures were to a mixture of substances including both soluble and insoluble chromates resulting from the roasting of chromite ore. Second, investigators have found excesses of respiratory cancer among workers in chrome pigment manufacturing (4, 12) and attributed the findings to insoluble chromate salt pigments synthesized from soluble hexavalent (VI) chromium compounds. Third, spray painting with chromate pigment products has been twice studied by the proportional mortality method. Increased respiratory cancer was found among zinc chromate spray painters (3). A second study of spray painters probably exposed to chromate pigments in the automobile industry was inconclusive in that proportional increases in respiratory and other cancer deaths were present for the work force as a whole with no apparent distinction between spray painters and others (2). Exposure histories were incomplete.

Recent literature reviews, including criteria documents (19, 20) of the National Institute for Occupational Safety and Health have concluded that insoluble chromium (VI) compounds are carcinogenic, while soluble compounds, including chromic acid, are not. This distinction does not appear to be supported by the available evidence and remains an open toxicologic issue. The present study lends additional weight to the possibility that chromic acid by itself, or in synergism with other chemicals, is an occupational cancer hazard.

6. In summary, this study demonstrates increased proportional lung cancer mortality among workers in a plating and die-casting plant. While the study provides evidence for a work-related etiology, the inability to produce a convincing identification of a chemical cause and the inherent limitations of the PMR methodology indicate the need for continuing investigations in this industry. Prompt action is indicated in light of the continuing widespread exposures of the types present in the study plant:

A. A standardized mortality ratio study among workers in the study plant is indicated to determine whether the increased proportions of cancer deaths reflect an increased mortality rate.

B. Expanding the proportional mortality investigation to similar manufacturing plants would be the most rapid way to provide additional information on chemical causation and to determine whether the findings in this study are unique to this single plant or shared by others of its type. Case-referent analysis considering additional factors such as smoking history and job classification, as well as departmental assignment, is indicated as more mortality data is accumulated. Such a study would be the most informative in workplaces where plating and die casting were separate and where workers did not have such mixed exposures. In the study plant the small excesses of stomach, liver, laryngeal cancer, and lymphosarcoma and the occurrence of several rare cancers requires further evaluation, including a review of clinical records. The possibility that the liver cancer and cirrhosis deaths are both related to toxic chemical exposures should be considered.

C. A voluntary medical surveillance program for the early detection of lung cancer among workers at the study plant has been initiated.

D. An industrial hygiene review of operations in this plant is in progress. Smoke from die cast operations should be chemically analyzed for potential cancer-causing agents such as benzo-a-pyrene or other polynuclear aromatic hydrocarbons. The

chemical composition of die lubricants and plating additives used in the past, and currently in use, should be reviewed. The epidemiologic findings warrant the control of all air contaminants associated with the historical exposures in the plant to the lowest level attainable by state of the art of ventilation.

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