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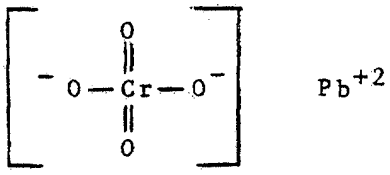
Common Name: Lead Chromate

Chemical Name: Chromic acid [H_2CrO_4], lead(2+) salt (1:1)

Synonyms: Chrome Yellow Light*
Chrome Yellow Medium*
Chrome Primrose Yellow*
C.I. Pigment Yellow 34*

CAS Registry Nos.: 7758-97-6
8049-64-7
15804-54-3
11119-70-3
1344-37-2

Chemical Structure:



Physical Properties: (34,58)

Description:	Yellow powder
Molecular Weight:	323.18
Boiling Point:	Decomposes
Melting Point:	844°C
Density:	6.12 g/mL @ 15°C
Vapor Pressure:	----
Solubility:	Practically insoluble in water (580 ug/L @ 25°C); insoluble in acetic acid; soluble in solutions of fixed alkali hydroxides, dilute nitric acid.
Conversion Factors:	----

* Pb 59.8 - 61.3%; Cr 10.2 - 14%

This literature search contains 18 pages of text and 85 references.

This search was prepared by Marsha A. Lee. See the last page of this document for its updating history.

N 27683

Exposure Standards:

TLV® = 0.05 mg/m^3 (as Cr)

OSHA PEL = 0.1 mg/m^3 (as CrO_3) (Ceiling)
CFR 29:1910.1000

OSHA PEL = 0.05 mg/m^3 (as Pb)
CFR 29:1910.1025

AEL = 0.05 mg/m^3 (as Cr; the 60 ug/100 grams of blood AEL for inorganic lead also applies)

Blood lead levels are the best indicator of lead exposure. At blood lead levels of 60 ug/100 mL, action should be taken to determine the source of exposure. However, individuals may present increased blood levels without clinical effects. Normal background levels of blood lead are 20-40 ug/100 mL. Up to 60 ug/100 mL there are biochemical changes in the blood apparently without physiologic consequence. Blood levels exceeding 60-80 ug/100 mL indicates overexposure to lead but not necessarily with intoxication symptoms. Clinical signs of overexposure may not appear until the lead blood level reaches 100 ug/100 mL (17b).

The level of lead in the workplace that is acceptable for women of childbearing potential to protect the fetus is 5 ug/m^3 for 24-hour exposures. However, the best method to determine exposure is to use the 30 ug/100 grams of blood lead level (17a).

DOT Classification:

CFR 49:172.101 Wastewater treatment sludge from the treatment of Chrome yellow pigment is listed as a hazardous material.

EPA RCRA Status:

CFR 40:261.32 Wastewater treatment sludge from the treatment of Chrome yellow pigment is listed as a hazardous waste.

EPA Drinking Water Standard

Maximum lead and chromium concentrations allowed in public drinking water are 0.05 mg/L (8).

FDA Status:

No information available.

TSCA Inventory:

Yes

TOXICITY

Summary:

The acute oral and inhalation toxicities of lead chromate are low; the ALD for the rat is greater than 5000 mg/kg. The skin is highly sensitive to some hexavalent chromium compounds. Even though lead chromate is on the list of suspected carcinogens, the status of its carcinogenicity is not conclusive since there is a study that finds lead chromate to be not carcinogenic. Lead chromate is a mutagen.

A. Acute

1. Oral

   - o ALD (rat) = > 5000 mg/kg (16a,b).
   - o ALD (rat) = > 11,000 mg/kg (16c).
   - o Groups of rats were fed a diet containing 0.2% lead as lead chromate for two days. One diet contained lead chromate of 500-1000 u particle size. The second diet was comprised of lead chromate of < 50 u particle size. The smaller-sized particles were associated with an increased tissue lead content which was more marked for kidney than for blood (1).

2. Skin

   - o Hexavalent chromium compounds are the most sensitizing because of their solubility and capacity to penetrate into skin and then into the cell. Five ug Cr/g of chromate in cement can sensitize and elicit dermatitis (21).

3. Eyes

   No information available.

4. Inhalation

   - o No deaths occurred in rats exposed to concentrations of 2.6, 4.9, or 34 mg/L for six hours; 44 mg/L for three hours; or 66 mg/L for one hour. The fur and body of the rats were stained yellow. There was an inhibition of normal weight gain. Pathology revealed the compound in the lungs of the rats in amounts roughly corresponding to the exposures. There was also a small amount of edema in the lungs of all animals. The inhalation of 34 mg/L for six hours produced a chronic pneumonitis in 2/2 rats.

Rats receiving 4.9 mg/L or more showed evidence of gastritis which was presumably due to swallowed material (16a).

- ^{51}Cr-labelled lead chromate was studied in the rat using a single intratracheal dose. The insoluble lead chromate was poorly absorbed from the lungs. The authors also found that the more insoluble a chromate was in water, the higher its elimination via the feces. Absorbed chromium was retained in the spleen and the bone marrow. On the basis of the minor resorption of lead chromate, it was concluded that chromium levels in blood and urine were not indicative of exposure to insoluble chromates via the inhalatory route (2).

- A group of rats received, by intratracheal instillation, a lead chromate paint particulate suspension equivalent to 1 mg lead/kg body weight. At post-mortem, five weeks after exposure, the vast majority of the dosed lead in the paint matrix remained in the lung. The blood delta-aminolevulinic acid dehydratase (ALA-D) level was not significantly different from control animals (18).

5. Injection Studies

Intraperitoneal

- LD50 (guinea pig) = 400 mg/kg (50).

B. Extended Studies

1. Oral

- Groups of rats were fed diets containing 0.1% of a lead chromate-containing paint. The paints tested contained 0.42%, 1.95%, and 12.43% lead chromate. The experiment lasted 13 weeks. The rats fed the 12.43% lead chromate paint had an increase in blood lead but no other symptoms. None of the other animals were affected (51).

- Ninety-day feeding studies were conducted using young adult albino rats to determine the toxicologic effects and tissue residues resulting from ingesting lead-containing pigments: light chrome yellow, medium chrome yellow, and primrose chrome yellow. Groups of rats were fed each pigment at dietary levels of either 2000, 5000, or 20,000 ppm.

Exposure to lead was reflected in increased concentrations of lead in blood and tissues as well as increased urinary excretion of aminolevulinic acid (ALA). Reduced body weights and food intake, and lowered hemoglobin and hematocrit values were observed in groups fed the chemicals at $\geq$ 5000 ppm. Kidney weights were increased in the females at 500 ppm and in males and females at 20,000 ppm of light chrome yellow. Similar changes were seen in males and females at 20,000 ppm medium chrome yellow and at both 5000 and 20,000 ppm primrose chrome yellow. Renal lesions, involving tubular epithelial cells, were produced in all groups fed primrose chrome yellow. These changes were dose-related in terms of incidence and severity. Kidney lesions were seen at feeding levels of $\geq$ 5000 ppm with either light chrome yellow or medium chrome yellow (35).

- Ninety-day feeding studies were conducted in six-month old beagle dogs to determine the toxicologic effects and tissue residues resulting from ingesting lead-containing pigments: light chrome yellow, medium chrome yellow, and primrose chrome yellow. Groups of four dogs of each sex were fed each pigment at dietary levels of either 2000, 5000, or 20,000 ppm. Measurements of lead exposure, including blood and tissue concentrations, and urinary aminolevulinic acid (ALA) excretion, revealed dose-related increases in these parameters. Early signs of lethargy, anorexia, dehydration, and emaciation, followed by hyperirritability, disorientation, motor ataxia, and convulsions preceding death were seen in animals fed $\geq$ 5000 ppm light chrome yellow, primrose chrome yellow, and 20,000 ppm medium chrome yellow. Decreased hemoglobin concentration, hematocrit, and altered erythrocyte morphology were produced by all chemicals tested, although the level of exposure needed to produce the changes varied considerably. Pathologic changes of the kidney, focal alterations to the epithelial cells of the proximal tubules, including necrosis, sloughing of cells, increased mitotic figures, and enlarged cells were produced by all pigments (5,35).

- Calves were fed ten or 100 ppm $PbCrO_4$ in their diet for 100 days. No effects were seen on feed consumption, weight gain or nitrogen excretion. Lead accumulated in the liver and kidneys only in calves fed 100 ppm. No ultrastructural changes in the kidney, liver or cerebral cortex, hepatic intracellular inclusions, nerve degeneration, astrocyte swelling, or other clinical signs of lead poisoning were observed (14).

2. Inhalation

- A group of 72 rats was exposed to a mixture of pigment grade lead chromate, barium chromate, and zinc chromate. The rats inhaled this mixture four days a week, from four to five hours a day for up to two years. Between five and ten grams of the mixture were delivered into the air of the chamber each day. Histological examination often revealed peribronchial focal adenomatosis as well as squamous cell metaplasia of the bronchial epithelium lining bronchiectases. None of the lungs contained a primary cancer. Inflammatory changes were also observed (32).

3. Subcutaneous Injection

- A single injection of 30 mg of lead chromate in a group of 40 rats gave rise to 26/40 injection-site sarcomas within 117-150 weeks. No local sarcomas occurred in vehicle treated controls (45-47).

- Rats were administered 30 mg of chrome yellow dissolved in saline. The pigment was determined to be highly carcinogenic (48).

4. Intratracheal Instillation

- No pulmonary adenomas occurred in 13 guinea pigs given six intratracheal instillations at three-month intervals. Negative results were also obtained in rabbits given from three to five injections (61).

5. Intramuscular Implant

- A group of 50 rats was administered nine monthly injections of 8 mg of lead chromate in trioctanoin. Animals were killed when moribund of when a growth reached a size of 2 x 2 cm. Sixty-four percent of the animals developed malignant tumors at the injection site. Three renal carcinomas were also found. No such tumors appeared in a similar group of 22 controls injection with the vehicle (24).

- A single injection-site tumor occurred in a group of 35 rats administered a 25 mg intramuscular implantation. In another group of 35 rats administered a 25 mg intrapleural injection, three injection-site tumors were seen (32,33).

- A group of 25 mice was administered four monthly injections of 3 mg of lead chromate in trioctanoin. Two lymphomas were seen within 16 months and three lung adenocarcinomas within 24 months among 17 animals necropsied. Similar incidences occurred in trioctanoin-injected and untreated control groups (24).

- Seven different lead chromate pigments were examined in a two-year study using an intrabronchial pellet implantation system whereby pellets loaded with test material were surgically implanted into the lower left bronchus of rats. There were four squamous carcinomas out of 400 animals, and therefore, the results are not statistically significant (42).

C. Carcinogenic Potential

- Lead chromate produced sarcomas at the site of its subcutaneous (45-47), intramuscular (24,32,33), and intrapleural (32,33) administration. It also produced renal carcinomas following its intramuscular administration. No lung tumors were seen in a chronic inhalation study (32) or an intratracheal instillation study (61).

- Industrial exposure to chromate pigments has been associated with an increased incidence of lung tumors (6,9,10,22,38,39).

- The Syrian hamster embryo-SA7 viral enhancement assay tested lead chromate's capacity to interact directly or indirectly with cellular DNA. Clear evidence of transformation potential in this system was apparent and is indicated by a chemically produced, dose-related, enhanced transformation effect at two or more consecutive concentrations (31).

- Lead chromate is listed as a suspect occupational carcinogen and is on the NIOSH Suspected Carcinogen list for 1978 (56).

- Seven hundred rats in groups of 100 for each of seven different lead chromate pigments (lead chromate, primrose chrome yellow, molybdate chrome orange, light chrome yellow, medium chrome yellow, LD chrome yellow supra, and silica-encapsulated medium chrome yellow) had a stainless steel wire mesh pellet coated with pigment surgically implanted into the left bronchiolus. The surviving animals were necropsied at the end of the study which was two years after the implant. Rats that died during

the study were autopsied shortly thereafter. Histopathology of the lung sections were made, accounting for all stages and types of lung epithelial inflammation. There were no bronchial carcinomas in the control group which had pellets coated with cholesterol. Two other positive control groups had pellets coated with known carcinogens calcium chromate and 20-methylcholanthrene. Four out of the 700 rats developed squamous carcinomas of the bronchus which was the cause of death. The author feels this is not statistically significant to classify the lead chromate pigments as carcinogens; at most they could be classified as weak carcinogens. The author also concludes that when the concentration of pigment is low or masked (as in encapsulation) it may not be carcinogenic at all (40,41).

D. Mutagenic Potential

- The potential mutagenicity of the carcinogen lead chromate was tested by the following battery of microbial tests: the Escherichia coli $PolA^+/PolA^-$ survival test; the Salmonella/microsome His^+ reversion assay; the Escherichia coli Trp^+ reversion test as a plate assay; the Escherichia coli Gal^+ forward mutation test; and the Saccharomyces cerevisiae assay for mitotic recombination. Lead chromate is mutagenic in Salmonella and in Saccharomyces and is thus identified as a microbial mutagen by this battery. Metabolic activation by rat liver homogenate (S9) is not required for the mutagenic activity of lead chromate. The most statistically significant, positive result is found with a supplementary assay, the Escherichia coli fluctuation test. To determine whether the lead ion and/or the chromate ion were responsible for the mutagenicity observed, lead chloride and chromium trioxide (chromic acid) were also tested. In Eshcerichia coli fluctuation tests, the ranges of maximal mutagenicity for chromium trioxide and lead chromate overlap at the concentration 10^{-5}M, whereas lead chloride shows no mutagenicity and little lethality at concentrations up to 10^{-3}M. Thus, it appears that the chromate ion is responsible for the mutagenicity of lead chromate (52,53).

- Chrome yellow was not mutagenic when tested in Salmonella typhimurium strains TA98 and TA100 in the presence of an activation system (49).

- Lead chromate was mutagenic for his^- strains of Salmonella typhimurium by inducing both frameshifts and base-pair substitutions. However, addition of

either microsomal fractions from rat liver or of human erythrocyte lysates resulted in complete loss of mutagenicity. As confirmed by chemical analysis, reversal of mutagenicity could be ascribed to reduction of the metal to the inactive trivalent form (57).

- Molybdenum orange and chromium orange were studied in the Salmonella/microsome test. Both pigments were positive to Salmonella typhimurium strain TA100 (spot test) but negative to strains TA1535, TA1537, TA1538, and TA98 (13).

- Low-solubility chromates such as lead chromate (which is classified here as a potent carcinogen) were studied for their solubility and clastogenic effect in a cultured Chinese hamster cell line (Don) and compared to potassium chromate which is a highly soluble chromate and a weak carcinogen. Lead chromate is slightly soluble in water, Tyrode's solution, protein or amino acid solution, fetal calf serum, and culture medium. There was a good correlation between the frequencies of chromosomal aberrations in the cells treated with the chromate and Cr concentrations dissolved from the chromate in culture medium. Lead chromate exhibited remarkable frequency of chromosomal aberration in the cells. The carcinogenicity of low-solubility chromates may be related to their low solubility in body fluid (37).

- Lead chromate was not mutagenic in the Chinese hamster ovary bioassay. Both zinc chromate and potassium dichromate were mutagenic in this test. The negative response of lead chromate is thought to be related to its low solubility in the culture medium (54).

- Lead chromate enhanced transformation of Syrian hamster embryo cells by a simian adenovirus. All of the known carcinogens increased the transformation (3).

- Lead chromate was mutagenic in Escherichia coli using a modified incubation procedure (36).

- Lead chromate caused dose-related increases in chromosome aberration and sister-chromatid exchange in human lymphocytes. No increase in DNA damage was observed in CHO cells, possibly due to the relative insensitivity of the CHO cells and the limited solubility of lead chromate is tissue culture medium. The mutagenicity of lead chromate in human

lymphocytes appears to be entirely due to the chromate ion since chromosome aberrations were induced by potassium chromate but not lead chloride (15).

- The influence if nitrilotriacetic acid (NTA) on the mutagenic and clastogenic activity of several water-insoluble or poorly soluble chromium compounds was determined in the Salmonella/microsome assay (plate test on strain TA100) and the sister chromatid exchange (SCE) test in mammalian cell cultures (CHO line). NTA does not itself induce gene mutations not does it increase the frequency of SCE. Lead chromate and chromium orange were clearly mutagenic in the Salmonella/microsome test only in the presence of NTA or NaOH. The mutagenicity of lead chromates is correlated with the amounts of Cr(VI) solubilized by NTA or alkali, as detected by the colorimetric reaction with diphenylcarbazide and atomic absorption spectrophotometry. In the SCE assay, the insoluble lead chromates are directly clastogenic owing to prolonged treatment conditions and cellular endocytosis. The chromosome-damaging activity of lead chromate is significantly increased by NTA but not by NaOH (43,63).

- Three independent laboratories tested blind studies on lead chromate using a common cell pool and the same lot of fetal calf serum in the Syrian hamster embryo clonal transformation assay system. They found that lead chromate induced morphological transformation without exogenous metabolic activation (62).

- Cytogenetical effects of lead chromate were evaluated in vivo. When 0.5, 1, 2, and 4 g/kg of lead chromate suspensions in 0.5% gum arabic were administered i.p. to male mice twice, the frequencies of micronucleated polychromatic erythrocytes in the femoral bone marrow were significantly different from that in the vehicle control. As the dose increased, the ratio of the polychromatic to normochromatic erythrocytes decreased in a dose-response fashion. The authors conclude that lead chromate in vivo gives rise to chromosomal damage as well as disturbance in hematopoiesis (64).

E. Developmental and Reproductive Toxicity

No information available.

F. Metabolism

- The biological half life of lead in body blood and soft tissues is reported as about 20 days. That for chrome is about 80 days (17c).

- An evaluation of exposure of two groups totaling 185 men to amounts of lead in the form of lead chromate, never less than 4.0 mg of $PbCrO_4$ per exposure day for 18 months, and to over 18 mg $PbCrO_4$ per exposure day for from 12 to 15 months showed no clinical or laboratory symptoms of lead absorption. From the above group, 26 men were selected on the basis of continuous exposure to the highest concentrations of lead chromate. This group had exposure days of over 18.3 mg lead chromate per day for 13 months, and to 6.2 mg lead chromate per day for 27 additional months. The men on the job cooperated by wearing the test collecting devices. At the end of 40 months, the group Basophilic Aggregation was 0.7 and the lead-in-urine average was .039 mg/L. Again, no clinical or laboratory results deviating from the normal were found (28,29).

G. Biochemical Studies

No information available.

H. Human Exposure

- Exposure to hexavalent chromium compounds may cause skin ulcers, ulcers of the nasal mucosa, and perforations of the nasal septum. Most of these injuries have been associated with soluble chromates (55).

- Fibrous lead chromate (50 ug/mL, 24 hours) was more cytotoxic than equal doses of nonfibrous lead chromate to human bronchial epithelial cells. However, no obvious cytotoxic responses were observed in rat alveolar macrophages exposed to the lead chromate particles (60).

I. Epidemiology

- This recent overview of the chromate industry reviews several studies of workers in pigment-producing factories and other chrome-related industries (e.g., spray painters of chrome pigment). The final analysis reveals that workers in "the chromate-producing industry are subject to an increased risk of cancer of the respiratory tract" (40).

- Workers occupationally exposed to lead chromate suspensions were examined to investigate the relationships between chromium exposure and otorhinolaryngological systems. The most representative symptoms of chromium exposure were nasal congestion, ulcerations, and repeated sore throats. Atmospheric concentrations less than 50 ug/m^3 were significantly related to ulceration and perforation of the septum. There was good correlation between the specific pathology of chromium and blood and urinary chromium levels greater than the norms (27).

- A study of lead chromate workers included three lead chromate plants in operation since the mid 1920's, 1941, and 1949, respectively. Employees of two plants were found to have had only lead chromate exposures, while workers in the third plant (analyzed separately) also had zinc chromate exposure. The industrial hygiene study of the three plants showed that nearly one-half of the samples reached or exceeded OSHA standards for lead and chromium. (The standards in effect at the time of this study were: Pb 0.2 mg/m^3; Chromium, soluble chromic, or chromous salts 0.5 mg/m^3 as Cr; and for chromic acid or chromates 0.1 mg/m^3 as CrO_3). The levels of Pb and Cr found in this study were assumed to be lower than levels in the past because of some process changes and modifications of engineering controls and work practices; however, this difference could not be quantitated because of changes in analytical methods. The three-plant cohort analysis included a total of 548 men among whom 53 deaths had occurred prior to December 31, 1974. Standardized mortality ratios (SMRs) were determined by comparing the observed deaths due to major causes with the number of expected deaths based on the 1960 male population of the state in which each plant was located. For all employees in the two plants with lead chromate exposure, a three-fold excess of respiratory cancer was observed. Workers employed prior to 1960 were analyzed separately. For all persons in this group, regardless of length of employment, the SMR for respiratory cancer was 173.5; for persons who were hired prior to 1960 and who had worked there for at least ten years, the SMR was 236.4. Five deaths due to stomach cancer were observed which was seven times the expected number. The small number of deaths from major causes made tests for statistical significance unjustifiable. Nevertheless, there appeared to be an excess of respiratory cancer in the workers studied, the excess generally occurring in older long-term workers (6,19).

- Lung cancer mortality of workers making zinc and lead chromate pigments was studied. Factories A and B produced both lead and zinc chromate pigments and virtually all the workers studied had exposures to both. The manufacture of zinc chromate ceased at factory A in 1964, and at factory B in 1976; factory C manufactured lead chromate pigments only. The study covers 396 men starting work at factory A from 1932 to 1967, 136 men at factory B from 1948 to 1967, and 114 men at factory C from 1946 to 1967. Ninety-four short-term workers at factory A with from three- to eleven-months employment during 1932-1945 were also included. Exposure levels were not specified but were expressed as high, medium, or low depending on the amount of dust in the work area. Twenty lung-cancer deaths occurred among the 252 workers at factory A who were initially employed sometime between 1932 and 1954 and who had been employed for more than one year. Of 175 workers in the factory who were said to have had high or medium exposure, 18 died from lung cancer, as compared with 8.17 expected lung-cancer deaths. There was no excess in the low exposure group. In factory B, seven lung-cancer deaths occurred among 16 workers with all durations of employment and with high and medium exposure who were first employed sometime between 1948-1967 as compared with 1.43 expected lung-cancer deaths. No excess of lung cancer was observed among 114 workers at factory C who were exposed only to lead chromate and first employed between 1946-1967 (9,10).

- A study was made of a small Norwegian company which produced chromate pigments since 1948. The employees were exposed to lead chromate, zinc chromate, sodium dichromate, zinc oxide, and other components of the pigment manufacturing process. Twenty-four workers were chosen for study who had more than three years of employment up to 1972. Using the cancer registry of Norway, three workers were identified as having bronchial carcinoma between 1951 and 1972 and one having a gastro-intestinal cancer. On the basis of 0.079 expected lung cancers, the relative risk in these workers was 38. While past exposures to hexavalent chromium are unknown, present exposures ranged from 0.01 to 1.35 mg/m^3 (38).

- The risk of cancer of the respiratory tract after exposure to chromium was previously noted and preventive measures taken. To asses the possibility of a cancer risk in workers exposed to lead chromate pigment dust, a study including seven factories in

four countries was designed. Unfortunately, most of the employees had also been exposed to zinc chromate which has been implicated in other studies as being associated with cancer. Due to difficulties in follow-up of causes of death in France and Italy, only five factories - those in The Netherlands and in West Germany - were included for evaluation. The final number of 1921 employees contributed 28,512 person-years of exposure. A moderately but consistently increased risk of lung cancer and other respiratory tract cancers was apparent for the workers for four of the five factories. In no instance did the observed deaths range below the expected numbers, even when classifying the workers by duration of employment. The results are consistent with the hypothesis that exposure to chromate pigments may cause increase incidences of cancer of the respiratory tract (22).

- Eight lung cancer deaths were reported in three plants that manufactured lead and zinc chromate pigments. The deaths occurred between 1932 and 1940. The workers had been exposed to high dust levels. Ulcerations of the nasal septum occurred with perforations in three cases, unstated in three other cases, and negative in two others. There were chrome ulcers on other parts of the body (25).

- Two bronchogenic carcinoma deaths following seven and 22 years of service in a plant producing chromate pigments was reported. The working conditions were described as high dust concentrations. Both cases had perforations of the nasal septum. Tissue analysis at autopsy showed elevated chromium content of lung and other tissues (39).

- The effect of exposure to lead chromate dust was studied in 26 workers in two pigment factories. Seven workers were exposed to less than 0.1 mg/m^3 (as Pb), five workers to 0.1-0.2 mg/m^3, and 14 workers to greater than 0.2 mg/m^3. The hemoglobin level was clearly subnormal in the last group. No other correlation existed between symptoms and the degree of exposure (30).

- A study was made to determine whether there is an increased mortality, especially with respect to cancer of the lung, among spray painters exposed to chromate-containing paints. The study was conducted among a cohort from ten automobile assembly plants. A total of 4215 deaths were included in the analysis of this report. Among decedents with some spray

painting experience, there were no statistically significant excesses in lung cancer mortality (4).

- A number of studies have been done in the chromate-producing industry. The chemical process employed in this industry involves extraction of chromium from the ferrous ore known as chromite ore. The ore is dried, crushed, mixed with limestone and the carbonate of either sodium or potassium, and heated in a rotary furnace to convert the insoluble oxide to soluble monochromate. The monochromate is extracted from the furnace roast by repeated washing with water and treated with sulfuric acid to form dichromate, with sodium sulfate as a by-product. A large excess of lung cancer was found among these workers. In none of these studies has the existence of lead chromate been mentioned. However, the existence of these studies is indicated because of the similarities of chromium exposure in this industry and that in the chromate pigment industry. For specific information on these studies, see Reference 34.

- A mortality study was conducted on 574 male workers from three chromate-producing facilities. The cohort included workers who were exposed to lead chromate for at least six months. Two of the facilities also had exposures to other chromates. Causes of death were determined from company records or death certificates. Risk of death from lung cancer and other causes was described as a Standardized Mortality Ratio (SMR). Exposures to lead and chromium were estimated from company records and results of industrial hygiene surveys. These surveys done at the time of the study showed that the time weighted average concentrations exceeded OSHA limits in nearly 50% of the collected samples. Company records and prior surveys indicated even higher concentrations. No attempt was made to correlate individual exposures with the mortality survey. The 246 males exposed to only lead chromate were observed for 4768 person years. Twenty-one deaths occurred with 33.1 expected, giving an SMR of 63.5. The slight excess of lung cancer (three observed and 2.3 expected) was not statistically significant (7).

- Lung cancer mortality among 1152 men working at three English chromate pigment factories was studied from the 1930s or 1940s until 1981. Workers at one of the factories were exposed only to lead chromate; workers in the other two factories were exposed to other chromates as well. In the lead chromate-only factory, workers experienced normal mortality

(observed/exposed deaths = 7/6.45). The results provide no indication that lead chromate induces lung cancer in man, even under conditions conducive to lead poisoning (11).

- Long-term mortality was studied in a group of 57 chromate pigment workers who suffered clinical lead poisoning, mostly between 1930 and 1945. One death was attributed to lead poisoning, and there were significant excesses of deaths from nephritis (observed/expected deaths = 3/0.24) and cerebrovascular disease (9/2.20). There were also non-significant excesses for respiratory diseases (7/3.59), and accidents and violence (3/1.13). The deaths from nephritis followed long spells of service exceeding ten years. Poisoning appeared to have more adverse long-term effects on older workers: 15 men aged $\geq$ 40 at the time of poisoning experienced generally high mortality, and 30 years later or by the end of 1981, only two survived instead of the expected seven. The risk of cerebrovascular disease appeared to be unrelated to duration of exposure and affected even men employed for under one year. Excluding the 57 lead-poisoned men, other workers at the factories showed no excess mortality from cerebrovascular disease (12).

- A high incidence of lung cancer mortality was determined for workers engaged in the manufacture of chromate pigments. An environmental survey of the work area revealed that seven employees were exposed to from 7.5 to 98.2 ug Cr^{+6}/m^3 air and three employees were exposed to from 160 to 300 ug inorganic Pb/m^3 air, both exceeding the current recommended levels. No personal samples exceeded current OSHA standards for either Cr^{+6} or inorganic Pb (20).

- This study was designed to try to quantify the mortality from cancer and other diseases among workers in European factories producing chromate pigments. The exposures were to both zinc and lead chromates and, therefore, could not distinguish those people exposed to only lead chromate. This report deals with cancer of the respiratory tract, particularly lung cancer. The total workforce of each factory was followed up, more successfully when they were employed before 1960. Observed deaths from five factories were compared with expected deaths calculated on the basis of mortality figures for the factory's region. Further analysis concerned data of relevant cohorts (only people observed a minimum of ten years and exposure

beginning before 1965), certainty of complete records for the entire workforce to assure a complete cohort instead of prevailing healthy survivors, and exclusion of foreign nationals. The overall mortality did not deviate from the expected rates. Lung cancer rates were always in excess of expected numbers. The pattern of duration of exposure indicates that the lung cancer risk does not show a clear dose-response effect with time of employment. Because of the mixed nature of exposure, conclusions must be limited with the effect that the results obtained are consistent with the hypothesis that working in a chrome-processing plant environment is associated with an increased incidence of lung cancer and with a higher probability of dying from lung cancer compared with the general public (23).

- A study of mortality was carried out in a factory manufacturing lead and zinc chromates between 1958 and 1977. The number of workers exposed who had worked for more than six months during that period was 251. Out of 50 people who died, only 30 causes of death were known. Eleven cases of broncho-pulmonary cancers was higher than the 2.38 to be expected. Account was also taken of the influence of smoking and certain working conditions (26).

- A cohort of 1946 men employed between 1940 and 1979 in a zinc and lead chromate pigment factory was analyzed for cancer mortality because of reports of increased lung cancer incidences. The expected number of cases were determined using cause-age-time-sex-race specific mortality rates obtained from the U.S. Vital Statistical Data. Employees working less than one month were excluded. Thirty-six lung cancer deaths were observed; 23 were expected. There were seven deaths from pancreatic cancer; four were expected, and nine deaths from stomach cancer whereas five were expected. When workers were grouped according to intensity of chemical exposure, increased mortality was found for those employed for ten years or employed for over two years with intensive chemical exposure (44,59).

J. Aquatic/Environmental Studies

No information available.

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