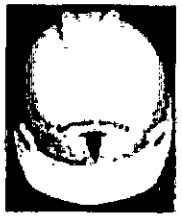

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Chromium Toxicity

Chromium enters the air, water, and soil mostly in the chromium (III) and chromium (VI) forms. In air, chromium compounds are present mostly as fine dust particles which eventually settle over land and water. Chromium can strongly attach to soil and only a small amount can dissolve in water and move deeper in the soil to underground water. Fish do not accumulate much chromium in their bodies from water.

Chromium (III)

Chromium (III) is an essential nutrient that helps the body use sugar, protein, and fat. Cr (III) is a very stable oxidation state for chromium. In this state, the chrome is labile and kinetically very slow to react or form complexes. It is not a strong oxidiser and the human's natural body acidity is enough for the chrome to keep to this Cr (III) state. (reference)

Chromium (VI)

Breathing high levels of chromium (VI) can cause irritation to the nose, such as runny nose, nosebleeds, and ulcers and holes in the nasal septum. Ingesting large amounts of chromium (VI) can cause stomach upsets and ulcers, convulsions, kidney and liver damage, and even death.

Skin contact with certain chromium (VI) compounds can cause skin ulcers. Some people are extremely sensitive to chromium (VI) or chromium (III). Allergic reactions consisting of severe redness and swelling of the skin have been noted.

Cr (VI) is not a very stable state when compared to Cr(III). The Cr (VI) is a very strong oxidizing agent (therefore very fast in reacting, unlike Cr (III) and likely to form complexes).

The main reason why Cr (VI) is so toxic is that one of the reduction products of Cr (VI) is Cr (V). Chrome (V) is a known carcinogen and will lodge in any tissue to form cancerous growths. There are reports that chromium (V) is also a factor leading to premature senility in parts of Russia.

In the body, the acidity and action of enzymes on Cr (VI) will promote the formation in small quantities of Cr (V). However, as the size of this is normally too large to be adopted by a tissue, the Cr (V) will pass out. The only place where the Cr (V) is likely to lodge is in some of the fine capillaries in either the kidneys, intestines or lungs.

During the passage out, Cr (VI) will continue to oxidize anything it can, leaving deposits of the relatively safe Cr (III) and completely unsafe Cr (V) behind.

How likely is chromium to cause cancer?

Several studies have shown that chromium (VI) compounds can increase the risk of lung cancer. Animal studies have also shown an increased risk of cancer. The World Health Organization (WHO) has determined that chromium (VI) is a human carcinogen. The Department of Health and Human Services (DHHS) has determined that certain chromium (VI) compounds are known to cause cancer in humans. The EPA has determined that chromium (VI) in air is a human carcinogen.

ATSDR Agency for Toxic Substances & Disease Registry

Environmental Health and Medicine Education

Chromium Toxicity
What Are the Physiologic Effects of Chromium Exposure?

Course: WB 1466
CE Original Date: December 18, 2008
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Learning Objectives

Upon completion of this section, you will be able to

describe physiologic effects, other than cancer, associated with chromium exposure and
describe the carcinogenic effects associated with Cr(VI) exposure.

Introduction

Major factors governing the toxicity of chromium compounds are oxidation state and solubility. Cr(VI) compounds, which are powerful oxidizing agents and thus tend to be irritating and corrosive, appear to be much more toxic systemically than Cr(III) compounds, given similar amounts and solubilities. Although mechanisms of biological interaction are uncertain, this variation in toxicity may be related to the ease with which Cr(VI) can pass through cell membranes and its subsequent intracellular reduction to reactive intermediates.

Mechanism of Chromium Toxicity

Since Cr(III) is poorly absorbed by any route, the toxicity of chromium is mainly attributable to the Cr(VI) form. It can be absorbed by the lung and gastrointestinal tract, and even to a certain extent by intact skin.

The reduction of Cr(VI) is considered to serve as a detoxification process when it occurs at a distance from the target site for toxic or genotoxic effect while reduction of Cr(VI) may serve to activate chromium toxicity if it takes place in or near the cell nucleus of

target organs [Dayan and Paine 2001]. If Cr(VI) is reduced to Cr(III) extracellularly, this form of the metal is not readily transported into cells and so toxicity is not observed. The balance that exists between extracellular Cr(VI) and intracellular Cr(III) is what ultimately dictates the amounts and rates at which Cr(VI) can enter cells and impart its toxic effects [Cohen, Kargacin *et al.* 1993].

Cr(VI) enters many types of cells and under physiological conditions can be reduced by hydrogen peroxide (H_2O_2), glutathione (GSH) reductase, ascorbic acid, and GSH to produce reactive intermediates, including Cr(V), Cr(IV), thiylradicals, hydroxyl radicals, and ultimately, Cr(III). Any of these species could attack DNA, proteins, and membrane lipids, thereby disrupting cellular integrity and functions [De Mattia, Bravi *et al.* 2004].

Respiratory Effects

Occupational exposures often include mixed exposure to both Cr(III) and Cr(VI) [EPA 1998].

Human occupational experience clearly indicates that, when inhaled, chromium compounds are respiratory tract irritants, resulting in airway irritation, airway obstruction, and lung, nasal, or sinus cancer. Dose, exposure duration, and the specific compound involved can determine chromium's adverse health effects.

Pulmonary irritant effects following inhalation of chromium dust can include

asthma,
chronic bronchitis,
chronic irritation,
chronic pharyngitis,
chronic rhinitis,
congestion and hyperemia,
polyps of the upper respiratory tract,
tracheobronchitis, and
ulceration of the nasal mucosa with possible septal perforation [Lindberg and Hedenstierna 1983; Dayan and Paine 2001].

Radiographic analysis from several reports revealed enlargement of the hilar region and lymph nodes [PHS 1953; Sluis-Cremer and du Toit 1968]. Consistent associations have been found between employment in the chromium industries and significant risk for respiratory cancer (see Carcinogenic Effects).

A delayed anaphylactoid reaction was reported in a male

worker occupationally exposed to chromium vapors from Cr(VI) trioxide baths and chromium fumes from stainless steel welding. A subsequent inhalation challenge with sodium chromate resulted in a reaction including late-onset urticaria, angioedema, and bronchospasm accompanied by tripling of plasma histamine levels [Moller, Brooks *et al.* 1986].

Many cases of nasal mucosa injury (inflamed mucosa, ulcerated septum, and perforated septum) have been reported in workers exposed to Cr(VI) in chrome-plating plants and tanneries [ATSDR 2000]. A 1983 study of 43 chrome-plating plants in Sweden, where workers were exposed almost exclusively to Cr(VI) acid, revealed that all workers with nasal mucosa ulceration or perforation were periodically exposed to at least 20 micrograms per cubic meter ($\mu g/m^3$) when working near the plating baths (The newest U.S. permissible exposure level in the workplace for chromates and chromic acid is 5 $\mu g/m^3$ as a ceiling). The period of exposure for workers experiencing nasal mucosal ulceration varied from 5 months to 10 years [Lindberg and Hedenstierna 1983]. A recent epidemiological study of U.S. workers found that the median time from date first employed to date of first diagnosis of nasal ulceration was less than a month; the median Cr(VI) concentration was similar to concentrations reported in the Swedish study [Gibb, Lees *et al.* 2000].

Occupational exposure to Cr(III) has also been associated with respiratory effects. One man developed coughing, wheezing, and decreased forced volume after an inhalation exposure to a sample of Cr(III) sulfate [Novey, Habib *et al.* 1983]. In an industrial hygiene survey of 60 ferrochromium workers exposed to Cr(III) and Cr(VI) (0.02-0.19 mg total chromium/m^3) conducted in 1975, appreciably higher incidences of subjective symptoms of coughing, wheezing, and dyspnea were reported compared with controls. However, due to the tobacco smoking that cannot be excluded as a confounding factor, the increase in subjective respiratory symptoms and decreased pulmonary function parameters cannot be unequivocally be attributed to chromium exposure [Langard S 1980].

The respiratory system in animals is also a primary target for inhalation exposure to chromium. Histological examination of the lung tissue revealed alterations representing mild nonspecific irritation after exposure to 0.9 or 25 mg Cr(III) trichloride for 30 min [Henderson, Rebar *et al.* 1979].

An extensive epidemiological survey was conducted of

housewives who lived in an area of Tokyo, Japan, in which contamination from chromium slag at a construction site was discovered in 1973. The exposed population reported a higher incidence of subjective complaints of nasal irritation than the control population in the early years of the study, buy in later years the difference between the two groups became progressively less [ATSDR 2000].

Skin Effects

Dermal exposure to chromium has been demonstrated to produce irritant and allergic contact dermatitis [Polak 1983; Bruynzeel, Hennipman *et al.* 1988]. Primary irritant dermatitis is related to the direct cytotoxic properties of chromium, while allergic contact dermatitis is an inflammatory response mediated by the immune system. Allergic contact dermatitis is a cell-mediated immune response that occurs in a two-step process. In the first step (induction), chromium is absorbed into the skin and triggers the next step - an immune response (sensitization). Sensitized individuals will exhibit an allergic dermatitis response when exposed to chromium above a threshold level [Polak 1983]. Localized erythematous or vesicular lesions at points of contact or generalized eczematous dermatitis should suggest sensitization [Lewis 2004].

Chromium allergic dermatitis is characterized by symptoms of

dryness,
erythema,
fissuring,
papules,
scaling,
small vesicles, and
swelling [MacKie 1981; Adams 1990].

Solubility and pH appear to be the primary determinants of the capacity of individual chromium compounds to elicit an allergic response [Polak, Turk *et al.* 1973; Fregert and Fregert 1981]. The low solubility Cr(III) compounds are much less efficient contact allergens than Cr(VI) [Spruit, van Neer *et al.* 1966].

Penetration of the skin will cause painless erosive ulceration ("chrome holes") with delayed healing. These commonly occur on the fingers, knuckles, and forearms. The characteristic chrome sore begins as a papule, forming an ulcer with raised hard edges. Ulcers can penetrate deep into soft tissue or become the site of secondary infection, but are not known to lead to

malignancy [Deng, Fleeger *et al.* 1990; Geller 2001; Lewis 2004; Meditext 2005].

In addition, occupational exposure Cr(VI) compounds has been associated with effects on the skin, nasal septum, and eardrum [Gibb, Lees *et al.* 2000].

Chromium is one of the most common skin sensitizers and often causes skin sensitizing effect in the general public. A possible source of chromium exposure is waste dumps for chromate-producing plants causing local air or water pollution.

Carcinogenic Effects

Occupational exposure to Cr(VI) compounds in a number of industries has been associated with increased risk of respiratory system cancers [ATSDR 2000].

Baetjer was one of the first to review the literature presented prior to 1950 on the occurrence of cancer in chromate-exposed workers [Baetjer 1950].

The first epidemiological study of chromate production workers in the United States that demonstrated an association with lung cancer was conducted with 1,445 workers in seven plants engaged in the extraction of chromates from ore from 1930 to 1947. The percentage death due to cancer of the respiratory system was 21.8%; the percentage expected was 1.4% [Machle and Gregorius 1948].

In another key epidemiological study involving workers at a chromate production plant who had worked at the plant for more than 1 year from 1931 to 1949, the percentage of deaths due to lung cancer was 18.2%; the percentage expected was 1.2%. For the 332 workers first employed from 1931 to 1937, the percentage of deaths due to lung cancer was close to 60% of all cancer deaths, with a latency period of approximately 30 years [Mancuso 1951; Mancuso 1975].

Studies of workers in the chromium pigment, chrome-plating, and ferrochromium industries showed a statistically significant association between worker exposure to Cr(VI) and lung cancer [Langard and Norseth 1975; Sheffet, Thind *et al.* 1982; Frentzel-Beyme 1983; Langard and Vigander 1983; Davies 1984; ATSDR 2000].

In addition to lung cancer, a number of epidemiological studies of workers in chromate industries also showed significantly increased risk for nasal and sinus cancers

[ATSDR 2000].

On the basis of these and other studies, the U.S. Environmental Protection Agency (EPA) and the International Agency for Research on Cancer (IARC) have classified inhaled Cr(VI) as a known human carcinogen [IARC 1990; EPA 1998]. The World Health Organization (WHO) has determined that Cr(VI) is a human carcinogen. The Department of Health and Human Services (DHHS) has determined that Cr(VI) compounds are known to cause cancer in humans [ATSDR 2000].

Lung cancer risk in relation to airborne levels of Cr(VI) was analyzed for chromium chemical production workers and a dose-response relationship was observed in that long-term workers had a higher lung cancer risk than short-term workers [Hayes, Lilienfeld *et al.* 1979]. An analysis of lung cancer risk suggests a potential excess risk of death from lung cancer among U.S. workers exposed to the previous permissible exposure limit (PEL) for Cr(VI) of 52 $\mu g/m^3$ [Braver, Infante *et al.* 1985]. More recent studies also disclosed excess risk of lung cancer death resulting from occupational exposure to Cr(VI) compounds [Gibb, Lees *et al.* 2000; Park, Bena *et al.* 2004].

Stratified analysis of lung cancer mortality showed a trend of increasing mortality with higher cumulative exposure levels. The analyses stratified by duration of employment and time since first exposure indicate a consistency of results among those employed the longest and with the longest elapsed time since first exposure. The latter suggests a latency period of approximately 20-35 years, which is compatible with other research [Luippold, Mundt *et al.* 2003].

Carcinogenicity appears to be associated with the inhalation of the less soluble/insoluble Cr(VI) compounds. The toxicology of Cr(VI) does not reside with the elemental form. It varies greatly among a wide variety of very different Cr(VI) compounds [Katz and Salem 1993].

Epidemiological evidence strongly points to Cr(VI) as the agent in carcinogenesis. Solubility and other characteristics of chromium, such as size, crystal modification, surface charge, and the ability to be phagocytized, compounds might be important in determining cancer risk [Norseth 1981; Langard 1983; Gad 1989].

In addition to the occupational studies, a retrospective

environmental epidemiological study was conducted in residents of a county in Sweden where two ferrochromium alloy industries are located. No indication was found that residence near these industries is associated with an increased risk of lung cancer [Axelsson, Rylander *et al.* 1980].

A number of chronic inhalation studies provide evidence that Cr(VI) is carcinogenic in animals [ATSDR 2000].

No evidence exists to indicate that Cr(III) can cause cancer in animals or humans [IARC 1990; EPA 1998].

Mechanism of Cr(VI)-Induced Carcinogenicity

The mechanism(s) of Cr(VI)-induced carcinogenicity is not completely understood. The toxicity of chromium within the cell may result from damage to cellular components during the hexavalent to trivalent chromium reduction process, by generation of free radicals, including DNA damage [ATSDR 2000]. Recent studies indicate a biological relevance of non-oxidative mechanisms in Cr(VI) carcinogenesis [Zhitkovich, Song *et al.* 2001].

Renal Effects

Renal effects after inhalation or oral exposure to Cr(VI) compounds have been reported.

Although glomerular injury has been noted in chromium workers, the predominant renal injury is tubular, with low doses acting specifically on the proximal convoluted tubules. Injury to the brush border membrane is a feature of chromate nephropathy [Kirschbaum, Sprinkel *et al.* 1981]. Severe poisoning can lead to acute tubular necrosis and acute renal failure [Sharma, Singhal *et al.* 1978]. Low-dose chronic Cr(VI) exposure typically results only in transient renal effects. Elevated urinary β2-microglobulin levels (an indicator of renal tubular damage) have been found in chrome platers, and higher levels have generally been observed in younger persons exposed to higher Cr(VI) concentrations [Lindberg and Vesterberg 1983]. Sensitive immunochemical techniques for the measurement of specific proteins in the urine have been used for the early detection of kidney damage, a possible threshold having been indicated at exposure levels yielding 15 µg/g creatinine in urine [Franchini and Mutti 1988].

Occupational exposure to Cr(III) does not appear to be associated with renal effects (ATSDR 2000]. No renal

impairment based on urinary albumin, retinol binding protein, and renal tubular antigens was found in 236 workers employed in the ferrochromium production industry [Foa, Riboldi *et al.* 1988].

Hepatic Effects

Cr(VI) has been reported to cause severe liver effects in four of five workers exposed to chromium trioxide in the chrome plating industry. The reported liver effects include derangement of the liver cells, necrosis, lymphocytic and histocytic infiltration, and increases in Kupffer cells [Pascale, Waldstein *et al.* 1952].

Cases of hepatic effects after oral exposure to Cr(VI) compounds have also been reported. Elevated liver enzyme levels were reported flowing ingestion of 150 mL solution containing 22.5 g potassium dichrome. [Kolaciski, Kostrzewski *et al.* 1999] Hepatomegaly [Michie, Hayhurst *et al.* 1991; Meert, Ellis *et al.* 1994] and hepatic failure [Loubieres, de Lassence *et al.* 1999; Stift, Friedl *et al.* 2000] have also been noted in the cases of acute poisoning.

Exposure to Cr(III) has not been found to cause any liver effects in workers employed in two factories that produced Cr(III) oxide or Cr(III) sulfate [Korallus, Ehrlicher *et al.* 1974b].

Gastrointestinal Effects

In a study of 97 workers from a chrome plant exposed to a mixture of insoluble chromite ore containing Cr(III) and soluble Cr(VI) as sodium chromate and dichromate, gastrointestinal radiography revealed that 10 of the workers had ulcer formation, and of these, six had hypertrophic gastritis. Nearly all of the workers breathed through the mouth while at work and swallowed the chromate dust, thereby directly exposing the gastrointestinal mucosa [Mancuso 1951]. Most of the previous studies reporting gastrointestinal effects, however, did not compare the workers with appropriate controls.

Cases of gastrointestinal effects after oral exposure to Cr(VI) compounds have also been reported. In one study, a 14-year-old boy who died after ingesting 7.5 mg Cr(VI)/kg as potassium dichromate experienced abdominal pain and vomiting before death. Autopsy revealed gastrointestinal ulceration [Kaufman, DiNicola *et al.* 1970]. In another study, a 44-year-old man died of gastrointestinal hemorrhage after ingesting 4.1 mg Cr(VI)/kg as chromic acid solution [Saryan and Reedy

1988].

Cardiovascular Effects

Case reports of humans who died after ingesting Cr(VI) compounds have described cardiovascular effects as part of the sequelae leading to death.

A 22-month-old boy who ingested an unknown amount of sodium dichromate died of cardiopulmonary arrest. Autopsy revealed early hypoxic changes in the myocardium [Ellis, Brouhard *et al.* 1982]. A 35-year-old woman developed cardiovascular collapse and shock within a few hours following ingestion of 50 mL chromic acid [Loubieres, de Lassence *et al.* 1999]. A woman ingested 400 ml of leather tanning solution containing 48 grams of basic chromium sulphate ($CrOHSO_4$). The patient died of cardiogenic shock, complicated by pancreatitis and gut mucosal necrosis and hemorrhage [van Heerden, Jenkins *et al.* 1994]. A 33-year-old male developed hypotension, ventricular arrhythmias, severe respiratory distress, and metabolic acidosis after ingesting an unknown amount of a liquid wood preservative containing chromium trioxide, arsenic pentoxide, and copper oxide [Hay, Derazon *et al.* 2000].

Hematological Effects

Cases of hematological effects have been reported in humans after the ingestion of lethal or sublethal doses of Cr(VI) compounds. In a case of an 18-year-old woman who ingested a few grams of potassium dichromate, decreased hemoglobin content and hematocrit, and increased total white blood cell counts, reticulocyte counts, and plasma hemoglobin were found 4 days after ingestion. These effects were indicative of intravascular hemolysis [Sharma, Singhal *et al.* 1978].

Laboratory analysis of a 35-year-old woman, who died 12 hours after ingesting 50 ml of pure chromic acid [25 g Cr(VI)], revealed anemia (hemoglobin 56 g/L, hematocrit 17 percent) and thrombocytopenia [Loubieres, de Lassence *et al.* 1999].

Reproductive and Developmental Effects

One study showed wives of stainless steel welders were at higher risk of spontaneous abortions [Bonde, Olsen *et al.* 1992]. The more recent study [Hjollund, Bonde *et al.* 1995], however, did not corroborate those findings. No data were located regarding chromium in adverse human developmental effects.

Several animal studies provide evidence that Cr(VI), after oral exposure, is a developmental toxicant in rats and mice [ATSDR 2000]. Adverse developmental effects in animals include greater incidence of post-implantation loss, decreased fetal body weight, reduced ossification, and decreased number of live fetuses.

Genotoxic and Mutagenic Effects

The mechanism of chromium-induced genotoxicity is not fully understood.

In one experiment, Cr(VI) plus glutathione induced DNA damage in vitro, whereas Cr(III) with or without glutathione did not. Chromium seems to exert its genetic effects by binding directly to DNA. It can produce stable DNA-chromium complexes, DNA strand breaks, DNA-DNA cross links, and DNA-protein cross links. The active species for DNA binding seems to be the trivalent form [De Flora, Bagnasco *et al.* 1990; Cohen, Kargacin *et al.* 1993; Meditext 2005].

A recent clinical study reported strong DNA oxidative damage from the urinary samples of the patient who ingested 2 to 3 grams of potassium dichromate in a suicide attempt [Hantson, Van Caenegem *et al.* 2005]. Another study showed an involvement of the oxidative damage pathway in the mechanism of toxicity of chromium in occupationally exposed individuals [Goulart, Batoreu *et al.* 2005].

Cr(VI) compounds are clearly mutagenic in the majority of experimental situations [De Flora, Bagnasco *et al.* 1990; Cohen, Kargacin *et al.* 1993]. It has caused chromosome aberrations in mammalian cells and has been associated with increased frequencies of chromosome aberrations in lymphocytes from chromate production workers. Increases in sister chromatid exchanges were seen in lymphocytes from workers exposed to chromium, cobalt, and nickel dusts [WHO 1990; Meditext 2005].

Other Effects

In a chrome plating plant where poor exhaust resulted in excessively high concentration of chromium trioxide fumes, workers experienced symptoms of dizziness, headache, and weakness when working over the chromate tanks [Lieberman 1941].

Erosion and discoloration of the teeth may occur with Cr(VI) compounds exposure. In addition, papillomas of

the oral cavity and larynx have been reported in workers exposed to high air concentration of Cr(VI) [Hathaway, Proctor *et al.* 1996].

Severe corneal injury may result from ocular contact with solid or concentrated solutions of chromic acid and other Cr(VI) compound [Grant 1993].

Key Points

When inhaled, chromium compounds are respiratory tract irritants and can cause pulmonary sensitization.
Chronic inhalation of Cr(VI) compounds increases the risk of lung, nasal, and sinus cancer.
Severe dermatitis and usually painless skin ulcers can result from contact with Cr(VI) compounds.
Chromium compounds can be sensitizers as well as irritants.
DHHS, EPA, WHO, and IARC have all recognized Cr(VI) as a human carcinogen.
Occupational exposure to Cr(VI) compounds in a number of industries has been associated with increased risk of respiratory system cancers.
Latency for Cr(VI)-induced lung cancer can be greater than 20 years.
Some studies indicated that reversible renal tubular damage can occur after low-dose, chronic Cr(VI) exposure.
Occupational exposure to Cr(III) does not appear to be associated with renal effects.
Cr(VI) compounds can cause mild to severe liver abnormalities.
Some Cr(VI) compounds, such as potassium dichromate and chromium trioxide, are caustic and irritating to gastrointestinal mucosal tissue.
Ingestion of a lethal dose of chromate can result in cardiovascular collapse.
Oral exposure to Cr(VI) compounds may result in hematological toxicity.
Potential reproductive effects of chromium in humans have not been adequately investigated.
Data indicate that Cr(VI) compounds are teratogenic in animals.
Cr(VI) compounds induced DNA damage, gene mutation, sister chromatid exchange, chromosomal aberrations in a number of targets, including animal cells *in vivo* and animal and human cells in vitro.

Progress Check

7. Which of the following is a major target of inhalation exposure to chromium compounds?

A. Gastrointestinal tract.
B. Respiratory tract.
C. Cardiovascular system.
D. Central nervous system.

Answer:

Clear

To review relevant content, see Introduction, Respiratory Effects in this section.

8. Which of the following health effects from exposure to chromium is often reportedly seen in the general public?

A. Its carcinogenicity.
B. Its irritant effect.
C. Its skin sensitizing effect.
D. Its skin sensitizing effect.

Answer:

Clear

To review relevant content, see Skin Effects in this section.

9. Which of following statements is NOT correct?

A. Latency for Cr(VI)-induced lung cancer can be greater than 20 years.
B. No cancers, other than lung cancer, are associated with occupational chromium exposure.
C. No evidence exists to indicate that Cr(III) can cause cancer in animals or humans.
D. Epidemiological evidence strongly points to Cr(VI) as the agent in carcinogenesis. Solubility and other characteristics of chromium compounds and Cr(VI) dust particles may be important in

determining cancer risk.

Answer:

Clear

To review relevant content, see Carcinogenic Effects in this section.

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