

General description

Sources

Chromium (Cr) is a grey, hard metal most commonly found in the trivalent state in nature. Hexavalent (chromium(VI)) compounds are also found in small quantities. Chromite (FeOCr_2O_3) is the only ore containing a significant amount of chromium. The ore has not been found in the pure form; its highest grade contains about 55% chromic oxide (1).

In Europe, ferrochromium is produced mainly in Finland, France, Italy, Norway, Sweden and former Yugoslavia. Potassium chromate is produced mainly in Germany, Italy, Switzerland and the United Kingdom. Sodium chromate and dichromate are now among the most important chromium products, and are used chiefly for manufacturing chromic acid, chromium pigments, in leather tanning and for corrosion control (2,3).

Chromium levels in soil vary according to area and the degree of contamination from anthropogenic chromium sources. Tests on soils have shown chromium concentrations ranging from 1 to 1000 mg/kg, with an average concentration ranging from 14 to about 70 mg/kg (4). Chromium(VI) in soil can be rapidly reduced to chromium(III) by organic matter. As chromium is almost ubiquitous in nature, chromium in the air may originate from wind erosion of shales, clay and many other kinds of soil. In countries where chromite is mined, production processes may constitute a major source of airborne chromium. In Europe, endpoint production of chromium compounds is probably the most important source of chromium in air.

Occurrence in air

Information on concentrations of total and speciated chromium in the atmosphere is limited. Measurements carried out above the North Atlantic, north of latitude 30° north, several thousands of kilometres from major land masses, showed concentrations of chromium of $0.07\text{--}1.1\text{ ng/m}^3$ (5). The concentrations above the South Pole were slightly lower.

The following chromium concentrations have also been reported: 0.7 ng/m^3 in the Shetland Islands and Norway, 0.6 ng/m^3 in northwest Canada, $1\text{--}140\text{ ng/m}^3$ in continental Europe, $20\text{--}70\text{ ng/m}^3$ in Japan, and $45\text{--}67\text{ ng/m}^3$ in Hawaii (6). Monitoring of the ambient air during the period 1977–1980 in many urban and rural areas of the United States of America showed chromium concentrations to range from 5.2 ng/m^3 (24-hour background level) to 156.8 ng/m^3 (urban annual average); the maximum concentration determined in the United States in any one measurement was about 684 ng/m^3 (24-hour average) (2). Ranges of chromium levels in Member States of the European Union were given in a recent survey as follows: remote areas $0\text{--}3\text{ ng/m}^3$; urban areas: $4\text{--}70\text{ ng/m}^3$, and industrial areas $5\text{--}200\text{ ng/m}^3$ (7). Mass median diameters of chromium-containing particulates in ambient air have been reported to be in the range $1.5\text{--}1.9\text{ }\mu\text{m}$ (4).

Chromium levels in the industrial environment have only been reported in a few studies. Many of the studies referred to by IARC (2) were performed before 1960. Considering the

great difficulties associated with the analysis of the various chromium compounds (8), the results obtained during the last 10–15 years may be considerably more reliable than those in the early studies.

From the point of view of toxicity and carcinogenicity, chromium(VI) compounds are of much greater significance for workers and the general population than are trivalent and other valence states of chromium compounds (9). Therefore, chromium(VI) and chromium(III) have to be considered separately. This is, however, difficult to do when only total chromium is measured.

Analytical methods

Atomic absorption spectrometry is the method usually applied for analytical determination of chromium in urine and blood (range 10–1000 µg/litres) as well as from air samples (8,10). However, this method does not permit speciation of chromium. The calorimetric method traditionally employed, using the violet complex of 1,5-diphenylcarbazide, is still used for analysing chromium in urine. Specific analytical determination of the different chromium species is possible using various chromatography methods (11,12). Neutron absorption analysis has been used for more than 20 years (8), but nowadays particle-induced X-ray emission (PIXE) analysis has become a method of choice for detection of low levels of chromium in tissues (13).

Routes of exposure

Air

The bronchial tree is the primary target organ for carcinogenic effects of chromium(VI). Inhalation of chromium-containing aerosols is therefore a major concern with respect to exposure to chromium compounds. The retention of chromium compounds from inhalation, based on a 24-hour respiratory volume of 20 m³ in urban areas with an average chromium concentration of 50 ng/m³, is about 3–400 ng. Individual uptake may vary depending on concomitant exposure to other relevant factors, e.g. tobacco smoking, and on the distribution of the particle sizes in the inhaled aerosol. Chromium has been determined as a component of cigarette tobacco produced in the United States, its concentration varying from 0.24 to 6.3 mg/kg (2), but no clear information is available on the fraction that appears in mainstream tobacco smoke.

Drinking-water

The concentration of chromium in water varies according to the type of the surrounding industrial sources and the nature of the underlying soils. An analysis of 3834 tap-water samples in representative cities of the United States showed a chromium concentration ranging from 0.4 to 8 µg/litre (4).

Food

The daily chromium intake from food is difficult to assess because studies have used methods that are not easily comparable. The chromium intake from typical North American diets was found to be 60–90 µg/day (4) and may be generally in the range 50–200 µg/day.

The chromium content of British commercial alcoholic beverages was reported to be slightly higher than that of wines produced in the United States, namely 0.45 mg/litre for wine, 0.30 mg/litre for beer, and 0.135 mg/litre for spirits (4).

Table 1. Levels of daily chromium intake by humans from different routes of exposure

Route of exposure	Daily intake	Absorption
Foodstuff	<200 µg	<10 µg
Drinking water	0.8–16 µg	<1 µg
Ambient air	<1000 ng	<5 ng

Toxicokinetics

Only limited experimental data are available on the biokinetics of chromium (14). It can be assumed that the rate of uptake in the airways is to a great extent governed by the size distribution of the inhaled particles (2) as well as by the water solubility of the compounds (14). Chromium is generally present in the trivalent or hexavalent oxidation states, but may also occur in biological materials in the quadrivalent or pentavalent state.

It has been suggested that chromium concentrations in human lungs increase with age (15). Absorption by inhalation exposure appears to occur rapidly for water-soluble chromium(VI) compounds (14), although it is difficult to quantify the extent of uptake. A preliminary estimate of pulmonary absorption, following deposition of chromium(III) chloride in the lungs by instillation, indicates that approximately 5% is absorbed within a few hours (4). Data from studies on the level of chromium in lung tissue are available (16), but such results are not yet useful for retrospective assessment of *a priori* risk of lung cancer, mainly because the time-course of level reduction is unclear, so that latency time for development of cancer cannot be accounted for. A number of studies on the fate of chromium(VI) and chromium(III) following intratracheal administration have provided some information on pulmonary retention and absorption of chromium compounds (17–18). Chromium(III) is retained to a greater extent in the lungs than is chromium(VI) (17). Chromates with low water solubility are mainly cleared to the gastrointestinal tract, whereas more soluble chromates are absorbed into the blood (18).

Following oral exposure, absorption of chromium in the gastrointestinal tract is low, at an estimated 5% or less (4). Studies on the uptake of chromium(VI) compounds in the gastrointestinal tract indicate that the rate of uptake is to a great extent governed by the water solubility of the compounds (1,2,14). Results from *in vitro* studies indicate that gastrointestinal juices are capable of reducing chromium(VI) to chromium(III); however, data from *in vivo* studies are insufficient to demonstrate whether this reduction process has the capacity to eliminate any differences in absorption between ingested chromium(VI) and chromium(III) compounds (4). Pulmonary cells have been shown *in vitro* to have some capacity to reduce hexavalent chromium; however, this capacity is low compared to that of liver cells (19).

Tests on female experimental animals showed that absorbed chromium(III) and chromium(VI) can be transported to a limited extent to the fetus *in utero*. However, available data do not allow quantitative estimates of fetal exposure. In humans, the chromium concentration in the tissues of newborn babies has been found to be higher than that found later in life. There is a significant decline in concentration in children until about 10 years of age. Subsequently, there is a slight increase in the lung tissue concentration, but a slight

decrease in that in other tissues. Chromium transported by the blood is distributed to other organs, the most significant retention being found in the spleen, liver and bone marrow (20–23).

In both animals and humans, elimination of absorbed chromium from the body is biphasic, with a rapid phase, representing clearance from the blood, and a slower phase, representing clearance from tissues. The primary route of elimination is urinary excretion, which accounts for a little over 50%; fecal excretion accounts for only 5%. The remaining chromium is deposited into deep body compartments, such as bone and soft tissue. Elimination from these tissues proceeds very slowly; the estimated half-time for whole-body chromium elimination is 22 days for chromium(VI) and 92 days for chromium(III) following intravenous administration (4).

Biomarkers

A number of investigations have been performed to study the presumed association between chromium in the work atmosphere of welders welding manual metal arc (MMA) on chromium-nickel steel and the levels of the element in biological fluids (24–26). Interaction has been demonstrated with smoking: Sjøgren et al. (27) and Kalliomäki et al. (28) found higher levels of chromium in the urine of smokers than for nonsmokers who weld MMA on stainless steel (SS). Stridsklev et al. (29) also found a significantly higher levels of chromium in the biological fluids of smoking than among nonsmoking MMA/SS welders.

Health effects

It is generally accepted that chromium is an essential element for humans (23). Chromium deficiency has been described in both humans and animals, but a clear quantitative definition of the daily requirement of chromium in human nutrition has not been arrived at (23). WHO (30) estimates that the daily minimum population mean intake likely to meet normal requirements for chromium might be approximately 33 µg/person.

Effects on experimental animals and *in vitro* test systems

Mutagenic and carcinogenic effects

Animal experiments have not been very successful in identifying the compounds that may result in increased risk of bronchiogenic cancer, which is well documented in chromate workers (31). Hueper (32) reported three injection-site tumours in 31 rats following intrapleural injection of chromite ore roast. Baetjer et al. (33) were unable to induce bronchiogenic cancer in mice and rats by inhalation of chromium compounds. They reported four cases of lymphosarcoma, but such effects have not been observed by other authors. Sintered calcium chromate has been found to induce injection-site sarcomas in mice (34). Following intratracheal injection of calcium chromate and strontium chromate in 218 rats, Hueper & Payne (35) found three fibrosarcomas after two years of observation, the individual chromate dose being 10–12.5 mg. Nettesheim et al. (36) induced 14 adenocarcinomas and adenomas in the bronchial tree among 131 mice during inhalation experiments using calcium chromate dust (13 mg/m³ for 25 hours per week for lifetime). Levy & Venitt (37) administered intrabronchial implantations of chromates in rats and found that only chromates with low solubility caused a significant increase in bronchial carcinomas.

Evidence has been presented demonstrating the mutagenic capacity of a number of hexavalent chromium compounds *in vitro* and *in vivo*. An extensive review of the relevant studies is available (38).

Effects on humans

Toxicological effects

Chrome ulcers, corrosive reactions on the nasal septum, acute irritative dermatitis and allergic eczematous dermatitis have been recorded among subjects exposed to chromium(VI) compounds (1,4). Ulcerations or perforations of the nasal septum were reported in two-thirds of subjects following inhalation exposure to chromium(VI) (as chromic acid) resulting from exposure at peak concentrations of more than 20 µg/m³ (39). The data available in published reports do not suggest any dose–response relationship between local exposure to chromium(VI) compounds and the development of septal ulceration of the nose. Mancuso & Hueper (40) described a spotty, moderately severe but not nodular pneumoconiosis in chromate workers. Sluis-Cremer & du Toit (41) reported fine nodular pneumoconiosis in a few chromite workers in South Africa, but these results were not confirmed by other authors.

Systemic effects of chromium compounds in the kidneys, liver, skin and airways of animals and humans have been reviewed (42–44). As the current levels of exposure to chromium compounds in general are quite low in workplaces in the western world, the likelihood of major toxic effects occurring in the skin, liver, kidneys and blood-forming organs is only small. Such effects may, however, still occur among workers in developing countries.

The appearance of ulcerations and subsequent perforations of the nasal septum following exposure to chromium(VI) compounds is considered to be one of the markers of toxic effects resulting from exposure (45). Deposition of particulates containing chromium(VI) may explain the occurrence of septal ulceration at low levels of exposure (46).

Necrosis of the kidneys has been reported, starting with tubular necrosis and undamaged glomeruli, as well as diffuse necrosis of the liver and subsequent loss of architecture (47). A number of reports on human ingestion of chromium(VI) compounds, resulting in major gastrointestinal bleeding from ulcerations of the intestinal mucosa, with cardiovascular shock as a possible result, appeared 80–90 years ago, sometimes resulting in kidney or liver damage when the patient survived (48).

In humans, systemic effects have been reported to occur in the airways, cardiovascular system, kidneys and liver (48,49). In the kidneys, high doses of chromates (10–20 mg/kg) induce necroses of the proximal and distal tubule (47,49,50). According to experiments in which mice were injected with chromium(VI), this toxic effect may be inhibited by adding to the injection agents that supply glutathione, whereby this was seen to prevent chromium(VI) from harming the mouse kidney (51).

Mutagenic and carcinogenic effects

There is a large body of documentation on excess risk of cancer in respiratory organs among workers exposed to chromates, as reviewed by IARC (2). Cancer was documented in a chromate-exposed worker 100 years ago. The early literature, as well as most of the epidemiological data from the late 1940s onwards has been reviewed (2,3,49,50). Only some of these data are discussed below.

Alwens & Jonas (52) reported 20 cases of lung cancer in workers producing dichromates in Frankfurt am Main, Germany, the mean time from first exposure to the development of cancer being about 30 years. Machle & Gregorius (53) observed 156 deaths in their study group of approximately 1445 workers recruited from seven major chromate-producing plants in the United States. There were 46 deaths from cancer; 32 were due to lung cancer, giving a 16-fold excess compared with expected figures.

In a study on dichromate production workers in England, Bidstrup & Case (54) found 12 lung cancer cases versus 3.3 expected. When this population, plus workers employed at a later date, was followed up by Alderson et al. (55), the excess lung cancer risk had dropped to 1.8. Two cases of nasal sinus cancer were also observed in this study. In a study of two groups of lung cancer hospital patients in the United States, Baetjer (56) found a 28.6-fold excess risk in chromate production workers. Further documentation on lung cancer risk in chromate production workers was presented by Mancuso & Hueper (40), Taylor (57), Ohsaki et al. (58), and Hayes et al. (59).

Using data collected in 1949 for exposure estimation, Mancuso (60) presented a study on chromate production workers. Weighted average exposure to soluble, insoluble and total chromium, combined with length of exposure, were applied to dose/time subdivisions. He found that compounds of chromium in both the trivalent and hexavalent states carry a carcinogenic potential. No studies have confirmed his results (2,3).

Some studies have reported on the content of chromium in lung tissue in lung cancer patients with previous chromate exposure (61–63). However, none of the authors was successful in elucidating exposure–response relationships on the basis of such data.

Excess risk of developing cancer in the gastrointestinal tract was indicated in a Russian study of ferrochromium workers (64), but not in two Scandinavian studies (65,66). In one of these studies (66), the relative risk of lung cancer was between 2.3 and 8.5, depending on the choice of reference population. Chromium(VI) was found in the working atmosphere in both of the plants concerned and might have been the prime cause of the excess lung cancer risk found in these studies. In a subsequent follow-up from 1978 through 1985 of the Norwegian cohort, expanded to include about 30% more workers ($n = 1235$), excess lung cancer was present in the ferrochromium subgroup (ratio of observed to expected cases = 10/6.5; absolute risk = 9.5×10^{-4}) (67).

Lung cancer in chromium-pigment workers was first reported by Gross & Kölsch (68), who found eight lung cancer cases among workers in three small chromium pigment-producing enterprises. In a cohort study of zinc chromate pigment workers, a 30-fold excess for lung cancer was found based on six cases in a subgroup of 24 highly exposed workers (69). Excess lung cancer risk among chromium pigment manufacturers was confirmed in other studies (70,71), but with much lower relative risks. Hayes et al. (72) observed a cohort recruited from 1940 through 1982 comprising 1879 male workers employed for one month or more between January 1940 and December 1969. Air concentrations of chromium had been measured in recent years, indicating levels of about 0.5 mg/m^3 for exposed jobs and more than 2 mg/m^3 for highly exposed jobs. Among 41 observed lung cancer deaths (expected = 35.3), 24 had occurred among workers in jobs with exposure to chromium pigment. Lung cancer deaths in the whole cohort corresponded to an absolute risk of 8.1×10^{-4} , versus 7.0×10^{-4} expected. For workers with cumulative work periods of 1–9 and 10+ years, the

standardized mortality ratios (SMRs) were 176 and 174 respectively (test for trend, $P = 0.04$). Among those with 10+ years of employment, the SMR for those in whom it was 30 years since first employment was 321 based on six cases. A slight excess of lung cancer risk in chromium pigment spray painters was demonstrated in one study (73), but no excess was found in another study (74). There is strong evidence that zinc chromate is a more potent carcinogen than lead chromate (4,70).

In the chromium-plating industry exposure to chromium trioxide may constitute a lung cancer hazard. Royle (75) found 17 lung cancer cases among 1238 platers compared to 10 in a matched reference population. He also observed excess cases of gastrointestinal cancer. Similar results were observed among hard-chromium platers (76) with three lung cancer cases versus 0.7 expected. In a Japanese cohort of 265 chromium platers, one lung cancer death occurred, versus 1.1 expected (77,78), but the follow-up was very short. Four out of 11 lung cancer cases under 55 years admitted to major Tokyo hospitals (79) had mainly been exposed to sodium chromate and another four mainly to potassium chromate and chromate trioxide.

Fumes from stainless steel (SS) welding contain chromium(VI). In Europe, SS welding constitutes the work situation in which the largest number of workers are currently significantly exposed to chromium(VI) (2,80). Hence, it is conceivable that SS welders may be at excess risk of cancer. Only a few studies have considered SS welders separately from other welders (2,81–85). In general, a slight excess of lung cancer, with relative risks ranging from 1.0 to 4.0, was observed in these studies, but the excess has not been much higher than for mild steel (MS) welders who in general are unexposed to chromium(VI). In one of these studies, Becker et al. (83) followed a group of 1224 SS welders. Those who had used the MMA method 20–29 years earlier were the only group with excess deaths by lung cancer; four observed deaths versus 2.1 expected. In a subsequent follow-up (84) which included 1694 turners as reference, the authors differentiated between definite smokers and nonsmokers (ex-smokers excluded), revealing a higher SMR (179) among the smoking SS welders than among the smoking turners (134). In a multinational study performed by IARC, comprising 11 092 MS and SS welders from nine European countries, one of the analyses gave an SMR for lung cancer of 157 (ratio of observed to expected cases = 13/8.3) among the SS welders (85). However, a recent study (86) revealed an excess of six cases versus 1.6 expected among a group comprising 255 highly exposed mild steel welders. On the basis of these data, it is still not clear whether exposure to chromium(VI) in welding fumes is the prime cause of the excess lung cancer frequently observed in welders (2,86,87). Hence, it is not yet clear whether chromium(VI) in SS welding fumes constitutes a cancer hazard among these welders.

A number of chromium(VI) compounds have been demonstrated to be mutagenic or clastogenic, and to result in the transformation of cells in a variety of test systems. Elevated prevalences of both chromosomal aberrations and sister chromatid exchange in lymphocytes are found in workers exposed to chromates. Chromium(III) seems to be inactive in *in vitro* systems unless the conditions of exposure allow purified DNA to be directly exposed. Under such conditions chromium(III) may cause modifications in the physicochemical properties as well as decreased fidelity of replication. The present evidence on genetic effects resulting from exposure to chromium compounds seems sufficient to conclude that chromium(VI) compounds are mutagenic in humans. A slight excess of chromosomal aberrations has also been found in SS welders (2,88,89).

Evaluation of human health risks

Exposure

Chromium is ubiquitous in nature. Available data, generally expressed as total chromium, show a concentration range of 5–200 ng/m³. There are only few valid data on the valence state and on the bioavailability of chromium in the ambient air.

Health risk evaluation

Chromium(III) is recognized as a trace element that is essential to both humans and animals. Chromium(VI) compounds are toxic and carcinogenic, but the various compounds have a wide range of potencies. As the bronchial tree is the major target organ for carcinogenic effects of chromium(VI) compounds, and cancer primarily occurs following inhalation exposure, uptake in the respiratory organs is of great significance in respect of the subsequent risk of cancer in humans. IARC has stated that for chromium and certain chromium compounds there is sufficient evidence of carcinogenicity in humans (group 1) (2).

A large number of epidemiological studies have been carried out on the association between human exposure to chromates and the occurrence of cancer, particularly lung cancer, but only a few of these include measurements of exposure (52,59,60,66,69,90,91). Measurements were made mainly at the time that the epidemiological studies were performed, whereas the carcinogenic effect is caused by exposure dating back 15–30 years. Hence, there is a great need for studies that include historical data on exposure.

Four sets of data for chromate production workers can be used for the quantitative risk assessment of chromium(VI) lifetime exposure (59,66,69,72,90,91). The average relative risk model is used in the following to estimate the incremental unit risk.

Using the study performed by Hayes et al. on chromium production workers (59), several cohorts were investigated for cumulative exposure to chromium(VI) in terms of µg/m³-years by Braver et al. (91) (cumulative exposure = usual exposure level in µg/m³ × average duration of exposure). Average lifetime exposures for two cohorts can be calculated from the cumulative exposures of 670 and 3647 µg/m³-years, as 2 µg/m³ and 11.4 µg/m³, respectively ($X = \mu\text{g}/\text{m}^3 \times 8/24 \times 240/365 \times (\text{No. of years})/70$).

The relative risk for these two cohorts, calculated from observed cases and expected lung cancers, was 1.75 and 3.04. On the basis of the vital statistics data, the background lifetime probability of death due to lung cancer (P_0) is assumed to be 0.04. The risks (unit risk, UR) associated with a lifetime exposure to 1 µg/m³ can therefore be calculated to be 1.5×10^{-2} and 7.2×10^{-3} , respectively ($\text{UR} = P_0(R - 1)/X$). The arithmetic mean of these two risk estimates is 1.1×10^{-2} .

A risk assessment can also be made on the basis of the study carried out by Langård et al. on ferrochromium plant workers in Norway (66,67). The chromium concentration to which the workers were exposed is not known, but measurements taken in 1975 showed a geometric mean value of about 530 µg/m³. Assuming that the content of chromium(VI) in the sample was 19% and previous concentrations were at least as high as in 1975, the ambient concentration would have been about 100 µg/m³. On the assumption that occupational exposure lasted for about 22 years, the average lifetime exposure can be determined as 6.9 µg/m³ ($X = 100 \mu\text{g}/\text{m}^3 \times 8/24 \times 240/365 \times 22/70$).

When workers in the same plant who were not exposed to chromium were used as a control population, the relative risk (RR) of lung cancer in chromium-exposed workers was calculated to be 8.5. Therefore, the lifetime unit risk is 4.3×10^{-2} .

Since earlier exposures must have been much higher than the values measured in 1975, the calculated unit risk of 4.3×10^{-2} can only be considered as an upper-bound estimate. The highest relative incidence rate ever demonstrated in chromate workers in Norway is about 38, at an exposure level for chromium(VI) of about 0.5 mg/m^3 (69,90). This relative rate is based on the incidence of bronchial cancer of 0.079 in the total Norwegian male population, irrespective of smoking status. If the average exposure duration is about 7 years, the average lifetime daily exposure is calculated to be $11 \text{ } \mu\text{g/m}^3$ ($X = 500 \text{ } \mu\text{g/m}^3 \times 8/24 \times 240/365 \times 7/70$). The incremental unit risk was calculated to be 1.3×10^{-1} . This very high lifetime risk may be due to the relatively small working population.

Differences in the epidemiological studies cited may suggest that the different hexavalent chromium compounds have varying degrees of carcinogenic potency.

The estimated lifetime risks based on various epidemiological data sets, in the range of 1.3×10^{-1} to 1.1×10^{-2} , are relatively consistent. As a best estimate, the geometric mean of the risk estimates of 4×10^{-2} may be taken as the incremental unit risk resulting from a lifetime exposure to chromium(VI) at a concentration of $1 \text{ } \mu\text{g/m}^3$.

Using some other studies and different risk assessment models, the United States Environmental Protection Agency (EPA) estimated the lifetime cancer risk due to exposure to chromium(VI) to be 1.2×10^{-2} . This estimate placed chromium(VI) in the first quartile of the 53 compounds evaluated by the EPA Carcinogen Assessment Group for relative carcinogenic potency (4).

Guidelines

Information on the speciation of chromium in ambient air is essential since, when inhaled, only hexavalent chromium is carcinogenic in humans. The available data are derived from studies among chromium(VI)-exposed workers. When assuming a linear dose-response relationship between exposure to chromium(VI) compounds and lung cancer, no safe level of chromium(VI) can be recommended. At an air concentration of chromium(VI) of $1 \text{ } \mu\text{g/m}^3$, the lifetime risk is estimated to be 4×10^{-2} .

It should be noted that chromium concentration in air is often expressed as total chromium and not chromium(VI). The concentrations of chromium(VI) associated with an excess lifetime risk of 1:10 000, 1:100 000 and 1:1 000 000 are 2.5 ng/m^3 , 0.25 ng/m^3 and 0.025 ng/m^3 , respectively.

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