MiningWatch Canada
Mines Alerte
Suite 508, 250 City Centre Avenue, Ottawa, Ontario, Canada K1R 6K7
tel. (613) 569-3439 — fax: (613) 569-5138 — info@miningwatch.ca — www.miningwatch.ca

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Potential Toxic Effects of Chromium, Chromite Mining and Ferrochrome Production: A Literature Review

MiningWatch Canada

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This document is part of a series produced by MiningWatch Canada about the risks of chromium exposure. Additional fact sheets summarizing risks to the environment, chromite workers and nearby populations are also available online at: www.miningwatch.ca/chromium

Executive Summary

Chromium (Cr) is an element that can exist in six valence states, 0, II, III, IV, V and VI, which represent the number of bonds an atom is capable of making. Trivalent (Cr-III) and hexavalent (Cr-VI) are the most common chromium species found environmentally. Trivalent is the most stable form and its compounds are often insoluble in water. Hexavalent chromium is the second most stable form, and the most toxic. Many of its compounds are soluble. Chromium-VI has the ability to easily pass into the cells of an organism, where it exerts toxicity through its reduction to Cr-V, IV and III. Most Cr-VI in the environment is created by human activities.

Chromium-III is found in the mineral chromite. The main use for chromite ore mined today is the production of an iron-chromium alloy called ferrochrome (FeCr), which is used to make stainless steel.

Extensive chromite deposits have been identified in northern Ontario 500 km north-east of Thunder Bay in the area dubbed the Ring of Fire. They are the largest deposits to be found in North America, and possibly in the world. Plans by Cliffs Natural Resources for an open pit and underground chromite mine and ore processing facility in the Ring of Fire and a ferrochrome production facility (includes a chromite smelter) to be located somewhere in Ontario are currently undergoing economic feasibility studies and environmental assessments. Other companies also have plans for mining chromite and other metals in the same area.

Soil, sediment, water and air can all become contaminated with chromium through industrial activities. Dust from industry operations such as mining and smelting settles out of the air, polluting soils and surface water. Most soluble chromium eventually settles onto sediment. Contamination of soil, surface and groundwater can also occur through release of industrial wastewater and leaching of soluble Cr-VI compounds from wastes such as mine tailings, waste rock, dust and slag piles.

Chromium-VI reduction to Cr-III is favoured under normal atmospheric conditions and in low oxygen soils, water or sediments coupled with the presence of favourable pH values, organic matter, reductants or microorganisms. Chromium-III oxidation to Cr-VI occurs under favourable pH and moisture conditions with the presence of manganese oxides, alkali oxides or calcium compounds, during bush fires, water treatment, waste incineration and chromite ore dry grinding and smelting. Chromium-VI can have long residence times in surface water, groundwater and can persist in soil for years, even with favourable reducing conditions.

Chromite mining and ferrochrome production

Chromite ore is mined, crushed and processed to produce chromite concentrate. To create ferrochrome, chromite concentrate is combined with a reductant (coke, coal, charcoal or quartzite) in a high temperature submerged arc furnace or direct current arc furnace. See Appendix 1 for resources about ferrochrome production processes.

Chromite ore mining and concentration produces overburden and waste rock, dust, tailings and tailings water. Ferrochrome production creates air pollution, dust, slag (waste produced during ferrochrome separation from other ore elements) and process water. These waste materials have the potential to be contaminated with chromium and other heavy metals and chemicals of concern.

By exposing chromite ore to aboveground conditions that promote weathering and oxidation, Cr-III may be converted to mobile Cr-VI. Dry milling (grinding) of chromite ore is now known to convert Cr-III to Cr-VI and efforts are required to avoid producing and spreading hexavalent chromium during mining activity. Studies suggest that milling ores while wet or in a non-oxygenated atmosphere (i.e. nitrogen) will decrease Cr-VI formation. Preliminary observations have shown that the duration of milling also affects the amount of Cr-VI formed.

Ferrochrome production emits air pollutants such as nitrogen oxides, carbon oxides and sulfur oxides (NO_x, CO_x, SO_x) and particulate dusts that contain heavy metals such as chromium, zinc, lead, nickel and cadmium. During the high temperature smelting of chromite ore, some Cr-III is converted to toxic Cr-VI, contaminating the dust. Prior to smelting, steps employed in some processes, such as milling and agglomeration (i.e. sintering) may also produce Cr-VI. Due to the leaching potential of these contaminants, ferrochrome arc furnace dust is categorized as toxic waste in Canada (waste K091) and must be treated before disposal in order to prevent leaching toxins into the environment. Health risks via inhalation are also a concern.

Smelting also produces Cr-VI contaminated slag, with estimated total chromium contents of 2 to 12%. The discovery that dry pulverizing of slag and ore converts Cr-III to Cr-VI may be very important during steps such as ferrochrome recovery from slag and end-uses of crushed slag. When metal recovery from slag is part of the production process, contaminated water is also produced and may require treatment. Some ferrochrome facilities sell crushed slag for industrial end-uses, a potential recognized by Cliffs. There are environmental concerns about contaminant leaching that must be addressed prior to slag sale and use.

According to the Cliffs chromite project proposal, 6,000-12,000 tonnes of ore and 65,000 tonnes of waste rock will be produced per day. The operation's predicted tailings output would require an area of 250 hectares to contain it. An estimated 2,100 tonnes of slag would be produced per day by the ferrochrome plant, all of which would be cooled with water that will likely require treatment. The concern over water pollution is heightened by the proposed FeCr plant location near Lake Wanapitei, a drinking water source for the City of Sudbury.

The Canadian Metal Mining Effluent Regulations do not include a limit for the amount of chromium allowable in mine effluent to be discharged to the environment. The effluent is

required to pass an acute lethality test for rainbow trout, which means that it cannot kill more than 50% of the fish tested within 96 hours.

Canadian and Ontario criteria require that no more than 5000 ug/L of chromium be present in waste leachate. This limit is the same as the American limit, but significantly more than the Italian limit of 2000 ug/L. In addition, many other countries have set limits for Cr-VI specifically, which range from 20 to 1500 ug/L (Appendix 2, Table A-6).

Aquatic Ecosystems

In aquatic ecosystems chromium is known to bioaccumulate in algae, aquatic plants, invertebrates and fish. Uptake, accumulation and effects are influenced by species, organism size, sex and developmental stage, presence of other contaminants, water temperature, pH, alkalinity and salinity. Researchers acknowledge there is still little information on chromium uptake and effects in freshwater species.

Studies have observed toxic effects of hexavalent chromium at relatively low concentrations (parts per billion an pars per million). These include reduced growth and photosynthesis in algae and aquatic plants; and lethal toxicity, behaviour changes and decreased growth, reproduction and survival in invertebrates. Fish exposed to hexavalent chromium have shown changes in physical and bio-chemcical conditions, increased hatching time, DNA damage and reduced survival.

Chromium-III in water seems to be more toxic to fish than chromium-VI. It decreases reproductive success, can cause death at relatively low doses and deposits on the gills. Chromium-VI does not deposit on gills but enters the fish and exerts toxic effects on internal organs such as the liver and kidney.

The vast majority of studies on aquatic life are conducted in labs, not observed on wild populations in the field, and no studies were located where contamination was the result of chromite and ferrochrome industry. The one field study located that examined wild fish found no difference between the chromium concentration in unexposed fish compared to bluegill *(Lepomis macrochirus)*and largemouth bass *(Micropterus salmoides)* exposed to chromium (100 – 300 ugCr-VI/L).

The Canadian Water Quality Guideline for the Protection of Freshwater Aquatic Life is currently set at 1 ugCr-VI/L, based on toxicity to invertebrate *Ceriodaphnia dubia* and an interim guideline for Cr-III is set at 8.9 ugCr-III/L, based on toxicity to rainbow trout *(Oncorhynchus mykiss)*.

The Canadian Interim Sediment Quality Guideline for bioavailable total chromium is 37.3 mg/kg of dry sediment, while the Probable Effects Level for total chromium (for aquatic organisms) is 90 mg/kg. Cr-VI can be present in the top layer of sediment when the overlying water contains oxygen, but no guideline has been established for Cr-VI in sediment. The Ontario Contaminated

Site Condition Standard for total chromium in sediment is set at the upper limit of typical sediment concentration in the province, 26 mg/kg (Appendix 2, Table A-3).

Chromium is not considered likely to biomagnify in the aquatic food chain. While Cr-VI taken up into an organism is easily transported into cells where it exerts toxicity through its reduction to Cr-III, it is hypothesized that this reduction renders the chromium less dangerous to predators consuming the organism, since Cr-III is not as easily taken into cells and can be expelled from the body. A number of studies have found chromium concentration decreases in organisms higher up a food chain. However, some evidence exists for significant effects of chromium contamination in the aquatic food chain. Chromium bioaccumulation has been observed in the food (invertebrates) and bodies of lesser scaups, a population of migratory birds experiencing dramatic population decline. Little data exist regarding chromium threshold effect levels and effects on reproductive capacity of birds in field situations and scientists acknowledge that more research is needed to understand potential risks of excess chromium to wildlife.

Terrestrial Ecosystems

Information on the doses of chromium in water or food that cause health problems in mammals come mostly from toxicology tests done in the lab on mice and rats. Effects observed on animals in experimental doses through food, water or injection include: cancers, reproductive harm, behavioral changes, reduced growth and survival. While many laboratory studies have analyzed chromium toxicity to animals, very few field studies have been conducted about the effects of environmental chromium pollution on wildlife and we did not find any information directly relevant to potential impacts of chromite mining and ferrochrome production on terrestrial wildlife.

Hexavalent chromium can negatively impact soil ecology in a variety of ways such as decreasing soil micro-organism presence, diversity and function. Canada's soil chromium guidelines for the protection of environmental and human health are generally lower than soil standards in other countries. The upper limits of typical Ontario chromium concentrations in uncontaminated soils are slightly higher than the Canadian guidelines, thus the Ontario Site Condition Soil Standards have set higher limits than the Canadian guidelines (Appendix 2, Table A-4).

Terrestrial plants can accumulate Cr-III and VI from soil, sediment, water and atmospheric deposition on leaves. Plant exposure to excess Cr-III or VI can negatively affect plant health and survival. It is still unclear whether Cr-III or Cr-VI is more toxic to plants.

Chromium toxicity to plants has been observed at exposure to levels as low as 160 ugCr-VI/L and 104 ug Cr-III/L when grown in soilless solution and 1.8 mg Cr-VI/kg and 21 mg Cr/kg when grown in soil. Canada has set Quality Criteria for agricultural irrigation water at 4.9 ug/L for Cr-III (interim) and 8 ug/L for Cr-VI (Appendix 2, Table A-1). Based on plant contact with soil, a Canadian Provisional Guideline for the protection of environmental health has been calculated at 0.4 mgCr-VI/kg for agricultural soils, and 1.4 mgCr-VI/kg for industrial and commercial soils (Appendix 2, Table A-4).

Sensitivity to and effects of chromium vary between species, making toxicity predictions difficult without extensive plant studies. Toxic effects on plants from chromium exposure include:

- reduced growth;
- decreased chlorophyll production causing yellow leaves;
- narrow leaves;
- small root systems;
- damage to root membranes and ability to take up water;
- alteration of uptake and translocation of essential elements (i.e. nitrogen, potassium, calcium etc.);
- decreased or complete inhibition of seed germination;
- delayed growth;
- decreased seed yield;
- wilting;
- death

The amount an fate of chromium taken up by plants varies among species due to differences in absorption, transportation and storage of metals. Although most plants studied store the majority of chromium in their roots, translocation to all other parts of a plant does occur Many studies have found low plant ability to transport chromium from root to other plant parts. However, greater chromium concentrations in leaves than in roots has also been observed and several species with the ability to accumulate over 1000 mgCr/kg in their above ground parts have been identified and are classified as hyperaccumulators. The original form of chromium taken up by the plant may also influence its storage location.

It has been suggested that a "soil-plant-barrier" protects the terrestrial food chain from excess chromium because of: a) the insolubility of most chromium in soils; b) > 90% of taken up chromium stored in plant roots; c) plant toxicity occurring below levels thought to affect plant consumers. This may not apply in the following cases:

- where industrial contamination provides bioavailable Cr-VI;
- in soils where Cr-III is readily oxidized to Cr-VI;
- with plant species capable of accumulating levels higher than those thought to affect consumers;
- where a greater proportion of Cr is stored in aboveground plant parts; and
- where plant roots are sources of food for wildlife or humans

There is no documented evidence for biomagnification of chromium from soil to plant to animal. However, researchers acknowledge there is a lack of study regarding the risk of chromium in the food chain.

Human Health Effects

Human exposure pathways to chromium are inhalation, ingestion and skin contact. Chromium-III and VI are known to accumulate in animal and human tissues. Excretion from the body is very slow, with elevated chromium concentrations observed in human tissues even decades after exposure ceased.

Observed toxic effects of chromium compounds to humans or laboratory animals include developmental issues, damage to skin, respiratory, reproductive and digestive systems and cancer. Chromium-VI is much more toxic than Cr-III because of its greater ability to enter cells and its strong oxidation potential. Once inside cells, Cr-VI is reduced and produces free radicals, Cr-V, Cr-IV and eventually Cr-III, which are believed to be responsible for toxic and carcinogenic effects. Toxicity varies among chromium compounds and most non-lab studies do not track exposure to a specific compound, but assess risk based on exposure to total Cr (all valence states present), Cr-III or Cr-VI compounds. The effects of low level chronic exposures and the interactions between co-contaminants (ex. from industrial work, chromium from other sources such as cigarettes) are not fully understood.

As described above, Cr-VI has a greater ability to cross cell membranes than does Cr-III. Some scientists believe that the human body's saliva, digestive juices and red blood cells provide protection from chromium toxicity by reducing ingested Cr-VI to Cr-III prior to cell absorption. Chromium-VI toxicity could then occur only when the amount of chromium ingested is above a threshold where ability of the body to reduce Cr is overwhelmed. Based on this theory, arguments have been made that Cr-VI between 1000 and 10 000 ug/L is safe for human consumption.

Other researchers do not believe that chromium reduction is a reliable safeguard against toxicity and have shown that not all Cr-VI is reduced by the body before absorption into cells. Significant accumulation of chromium due to both Cr-III and Cr-VI ingestion has been seen in animal and human tissues. It is known that reducing capabilities vary among individuals and among animal species, adding uncertainty to the conclusions of animals studies extrapolated to humans, and the protection of sensitive individuals. The effectiveness of the body's reducing processes, the possible threshold levels for various exposure pathways and the effects of various chromium valence states in cells are not yet fully understood. Groups such as Health Canada, the American Occupational Safety and Health Administration and the European Union's Scientific Committee on Occupational Exposure Limits have all concluded that current information is not enough to support the existence of a threshold below which Cr-VI is non-toxic (MOE 2011a).

Chronic low-level skin exposure to Cr-III or Cr-VI can cause permanent sensitisation that leads to a skin condition called allergic contact dermatitis (ACD). This occurs with chronic exposures between 4-25 ppm Cr-VI and has been observed with occupational and environmental exposure to chromium. A review of many studies identified 10 ppm Cr-VI as the threshold at which no more than 10% of exposed individuals developed skin sensitisation. The same threshold was 500 ppm for Cr-III. Estimates of the population fraction to become sensitised range from 0.08% in general populations to 40% in occupationally exposed populations. Cr-VI compounds can also cause inflammation, eczema and, at levels between 20-25 ppm, open sores (ulcers). A no-observed-effect-level (NOEL) has not been identified for Cr-VI causing ACD or ulcers.

The chromium present in many materials used every day, such as food, detergents, leather and paints, is capable of eliciting the allergic response. Thus, chromium ACD is a serious and unresolvable issue for many individuals. Reports of skin lesions and eczema lasting years are not uncommon, leading to significant amount of work time lost and changes of occupation. Changes

in occupation did not necessarily improve skin conditions and often resulted in negative social and economic impacts. As there is no specific treatment for chromium induced skin issues, prevention of sensitisation is recommended as the best solution.

Some evidence shows that ingestion of Cr-VI and Cr-III can produce ACD in sensitised individuals, although more research is needed. Studies are also needed to determine if chromium inhalation can cause ACD in sensitised individuals.

Both Cr-III and VI are respiratory and mucous irritants. Chromium-VI is linked to occupational asthma, irritation of the nose, throat and lungs, ulceration and perforation (holes) in the septum (structure between the nostrils). Septal ulcers and perforations have been observed with exposure to as little as 0.09 ugCr-VI/m^3 and reversible impairment of lung function with exposures as low as 2 ugCr-VI/m^3.

While the Canadian Council of Ministers of the Environment (CCME) has no Air Quality Guidelines for the protection of human health and the environment for chromium, Ontario's Ministry of Labour has set 8-hr average workplace air standards for Cr-0, III (500 ug/m^3), soluble Cr-VI (50 ug/m^3) and insoluble Cr-VI (10 ug/m^3). These Cr-VI limits are higher than the levels set by the American National Institute for Occupational Safety and Health (Cr-VI - 1ug/m^3 over 10 hours) and the Occupational Safety and Health Administration (soluble Cr-VI as CrO_3 – 5 ug/m^3 over 8 hours) (Appendix 2, Table A-5).

In June 2011, the Government of Ontario set Ambient Air Quality Criteria (AAQC) and Air Standards for Cr-VI compounds and other Cr compounds (0, II and III) (Appendix 2, Table A-5). AAQC are used for environmental assessments, while Air Standards provide enforceable regulations with the goal of protecting communities from industrial air pollution. The standards are under Ontario Regulation 419/05: Air Pollution - Local Air Quality, under the Environmental Protection Act. They will become effective July 1, 2016, after a five-year phase-in period for industry. Companies may also apply for less stringent site-specific or sector-based technical standards (MOE 2011b).

Chromium-III is not considered a developmental toxin. Some evidence for the reproductive toxicity of Cr-III has been observed, but other studies have reported conflicting results and more research is required.

It is known that chromium is transferred from mother to young through the placenta and mother's milk. Developmental toxicity effects of Cr-VI have been observed in animals. Increased birth and developmental defects in children have been informally noted in areas of poorly regulated chromite mining, chrome, leather and tannery production, but no scientific studies have been located which investigate this further. Damage to male and female reproductive systems in animals and humans exposed to Cr-VI has been observed. A number of other studies have shown no effects on human or animal reproductive systems. Regulatory bodies cite the need for more research before a defensible characterisation of human reproductive and developmental risks can be done.

Ingestion of large amounts of hexavalent chromium compounds is known to cause nausea, vomiting, stomach and intestine damage, anaemia, kidney and liver damage and failure, coma

and death. Health effects observed in a human population chronically exposed to approximately 20 000 ugCr-VI/L in drinking water contaminated by a ferrochrome plant included mouth sores, diarrhoea, stomach pains, indigestion, vomiting, and higher levels of white blood cells than the reference population. Some evidence for inhalation of Cr-VI in dust leading to gastrointestinal, kidney and liver damage has been found, but findings to date are not conclusive.

The International Agency for Research on Cancer (IARC) states that metallic chromium and trivalent chromium are not classifiable regarding human carcinogenicity due to insufficient evidence. In the US Environmental Protection Agency's (EPA) Integrated Risk Information System, no carcinogenicity has been reported for any types of exposure to Cr-III compounds. However, modelling has predicted that regardless of the lower capacity of Cr-III to be absorbed into cells, Cr-III exposure has the potential to accumulate in human tissues at a level that has caused genotoxic or mutagenic effects in cells and live animals. Thus, more research is called for.

Hexavalent chromium is classified as a known human carcinogen by Health Canada, the US EPA (via inhalation) and the IARC. Chromium-VI exposures via inhalation at occupational levels are known to cause lung and sinonasal cancers. Increased risk of bronchial cancer and stomach cancer due to occupational chromium exposure has also been observed in epidemiological studies.

Chromium-VI risks related to non-respiratory type cancers and exposure pathways other than inhalation are controversial. Studies have found links between Cr-VI ingestion and non-respiratory cancers including: skin, lung, lip/oral cavity/pharynx, breast, liver, kidney, bladder, gastrointestinal tract, urinary tract, testes, prostate, brain, stomach, bone, lymphoma and leukemia, but some of these links have been called into question by other studies that have not found these links. Notable developments regarding oral exposure to Cr-VI in recent years show a shift of regulatory bodies such as the US EPA and the California EPA towards considering oral exposure to Cr-VI a likely human carcinogen.

Chromite Mining Exposure Effects

No recent studies on the health of chromite miners have been located. Two studies found evidence in chromite miners of pneumoconiosis, a lung disease caused by dust inhalation. As these studies were conducted in the 1950s and 60s, they may not be relevant to today's industrial hygiene conditions.

The presence of Cr-VI in chromite mining processes is generally thought to be low. Chromium-VI identified in breathable dust at mechanized and semi-mechanized open pit chromite mines in India had maximum values below occupational health and safety guidelines. Chromium-VI has also been measured in unprocessed chromite ore and crushed lumpy ore, but this may be due to the analytical method as sample grinding could generate Cr-VI. It is not clear whether the crushing and sorting techniques employed in concentrating chromium at a mine site have the same potential to create Cr-VI as does the grinding of the ore in the lab, but the results from mine dusts seem to indicate this is the case.

Ferrochrome Production Exposure Effects

Ferrochrome workers experience relatively high exposure to Cr-VI. Studies have linked ferrochrome worker exposure to Cr-VI in dust is to asthma, nasal skin damage and septal perforation. Exposure to dust has been linked to generalised obstructive lung disease and decreased lung function. Use of breathing filters can help avoid these issues.

While it is known that chronic exposure to high levels of Cr-VI in the air causes lung cancer, according to the International Agency for Research on Cancer, epidemiological studies regarding the lung cancer risks for ferrochrome workers are inconclusive. Risks for other cancers are also debated, as described previously.

Environmental Exposure Effects

Studies have observed health effects in general populations exposed to industrial chromium contamination. These include: skin irritations, signs of kidney disease, gastrointestinal problems and increased cancer mortality. These studies are challenged by other studies which observed no such effects and challenges inherent in most epidemiological studies of environmental contaminants, including confounding factors and uncertainty regarding important components such as actual doses of chromium, statistical power, lifestyle and other variables affecting cancer incidence, comparable reference populations and cancer latency time.

Guidelines and Regulations

Environmental and occupational exposure to Cr-VI is still considered a “major human health issue”. Due to the complex chemical and toxic behaviours of chromium, regulators must contend with a large degree of uncertainty.

The need for greater understanding of chromium toxicity continues to spur research and in turn, new regulations. For example, in 2006 the US Occupational Safety and Health Administration (OSHA) substantially reduced its permissible exposure limit for Cr-VI in air due to recent research. The Ontario Ministry of Environment updated their original (1996) Site Standards for soil and groundwater in 2009, which substantially lowered the allowable amounts of total chromium in soil, Cr-VI in subsurface soil, total chromium in non-potable groundwater and Cr-VI in potable groundwater. Standards were also created for soil and groundwater within 30 m of a surface water body.

Canada and California’s maximum contaminant levels for total chromium in drinking water (50 ug/L) are half that of the US EPA standards (100 ug/L). Canada and the USA do not currently have separate drinking water standards for Cr-VI, but the American EPA is in the process of evaluating Cr-VI oral exposure (ingestion) for carcinogenicity. California has recently reduced the public health goal for Cr-VI in drinking water in order to protect of more sensitive subgroups (fetuses, newborns, and people with low stomach acidity).

The US EPA has estimated continuous daily exposures to chromium considered safe for humans over a lifetime (known as reference values) for non-carcinogenic risks and has rated their level of confidence in these values based. The low and medium levels of confidence the EPA has in their reference values indictes the need for further research.

Conclusion

There is a long history of research regarding chromium toxicity. This research has provided the understanding that chromium is linked to a wide variety of toxic effects on animal and human health. Recent research shows the new and more stringent regulations for Cr-VI, to which some governments are responding. For all the efforts to understand the nature of chromium toxicity, many questions are still unanswered.

Some of these questions include:

1. Is there a threshold below which the body can detoxify ingested Cr-VI?
2. Does Cr-VI ingestion cause cancer in humans and if so, what cancers and at what doses?
3. Does Cr-VI inhalation cause any cancers other than lung and sinonasal and if so at what doses?
4. Does Cr-VI inhalation cause gastrointestinal, kidney and liver damage and if so, at what doses?
5. Do any Cr-III compounds cause cancer and if so, which and at what doses?
6. What are the safe exposure levels to Cr-VI that do not cause allergic contact dermatitis and skin ulcers?
7. What are the reproductive and developmental effects of Cr-VI and Cr-III on humans and at what doses?
8. Can chromium toxicity predictions for plants and animals be made which take into account the complex influences of different soil types, water characteristics, plant and animal species?
9. Which is more toxic to plants, Cr-III or Cr-VI?
10. What forms of Cr are present in plants after uptake and what risks might these pose to plant consumers?
11. Since chromium exposure can change the nutrient uptake of plants, what are the potential health effects of this for plant consumers?
12. Have standard tests been underestimating Cr-III toxicity to algae?
13. Does chromium biomagnify up aquatic and terrestrial food chains?

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Chromium and Chromite

Chromium (Cr) is an element that is often occurs naturally in igneous rocks as the mineral chromite ($FeCr_2O_4$). Chromium has many uses due to important properties such as colour, hardness and resistance to corrosion (Barceloux 1999). Today, the majority of mined chromite ore is processed into an iron-chromium alloy called ferrochrome (FeCr), which is required to make stainless steel (Government of Ontario 2011).

Where is chromite found?

Ninety-five percent of identified global chromium resources are located in Southern Africa and Kazakhstan, which produced 39%and 15% of global chromite ore respectively in 2010. India is also an important chromium supplier today, providing 17% of the 2010 world supply (USGS 2011). In recent years, chromite deposits have been identified in Northern Ontario, Canada. These deposits are located 500 km north-east of Thunder Bay in the area dubbed by the mining industry as the Ring of Fire. They are the largest deposits to be found in North America, and possibly in the world (Government of Ontario 2011). Plans by Cliffs Natural Resources for an open pit/underground chromite mine and ore processing facility in the Ring of Fire and a ferrochrome production facility to be located somewhere in Ontario are currently undergoing economic feasibility studies and environmental assessments (Cliffs 2011). Though Cliffs is the main proponent for chromite mining other companies have potential for mining chromite and other metals in the Ring of Fire.

Chromium chemistry

The element chromium can exist in six valence states, 0, II, III, IV, V and VI, which represent the number of bonds an atom is capable of making. Metallic chromium (Cr-0) does not occur naturally in the environment and Cr-II is unstable and converted quickly to Cr-III. Chromium-IV and V are also unstable and occur briefly as intermediates of conversions between Cr-III and Cr-VI. Trivalent (III) and hexavalent (VI) are the environmentally important chromium species (Zayed and Terry 2003).

Chromium is most commonly found in nature as Cr-III, which is the most stable species. The source of chromium for mining - the mineral chromite is made up of trivalent chromium. Most Cr-III compounds are insoluble in water and Cr-III is considered to be an essential trace element for human diets, although ingestion of large amounts can cause toxic effects (Zayed and Terry 2003).

The second most common and stable form of chromium in the environment is Cr-VI. It is also the most toxic, with toxicity ranging 100-1000 times higher than that of Cr-III (Godgul and Sahu 1995). Many Cr-VI compounds are highly soluble in water. Chromium-VI occurs naturally in the rare mineral crocoite, but most Cr-VI in the environment is created by human activities (Zayed and Terry 2003).

Chromium in the environment

Soil, sediment, water and air can all become contaminated with chromium. Dust from industry operations such as mining and smelting settles out of the air, polluting soils and surface water. Most chromium eventually settles into sediments whether initially soluble or not (Barceloux 1999). Contamination of soil, surface water and groundwater can also occur through release of industrial wastewater and leaching of soluble Cr-VI compounds from wastes such as mine tailings, waste rock, dust and slag piles (Tiwary *et al.* 2005; Wang *et al.* 2011).

The valence state of chromium in the environment is affected by dynamic processes that are influenced by a number of complex physical, biological and chemical factors. Depending on environmental conditions, chromium can be reduced from VI to III or oxidised from III to VI. Both types of reactions may occur simultaneously in the same system.

Atmospheric Cr-VI is likely to undergo reduction to Cr-III due to conditions inherent in the atmosphere. The estimated amount of time for half of Cr-VI released to the atmosphere to be converted to Cr-III ranges between 16 hours and 4.8 days (Kimborough *et al.* 1999). Depending on the size of particles it is associated with, most chromium will settle out of the air within 10 days of emission (Barceloux 1999).

Soil type and mineral content influence chromium valence states in soils (Bagdon and Hazen 1991). In low oxygen soils, water or sediments, Cr-VI can be reduced to Cr-III if favourable pH values, organic matter levels, reductants or microorganisms are present. Chromium-III can be oxidised to Cr-VI under favourable pH and moisture conditions in the presence of manganese oxides, alkali oxides or calcium compounds (Cooper 2002; Zayed and Terry 2003; Dube *et al.* 2003; Ma and Garbers-Craig 2006b), during bush fires (Panichev *et al.* 2007), water treatment chlorination (Health Canada 1986; Lai and McNeill 2006), waste incineration (Prokisch *et al.* 1997) and chromite ore dry grinding (Beukes and Guest 2001) and smelting (Erdem *et al.* 2005). Chromium-VI is the dominant form in shallow oxygenated waters (Barceloux 1999) and oxygen-rich soils (Chandra and Kulshreshtha 2004). Since many Cr-VI compounds are soluble, they easily leach deeper into the soil and groundwater, where it is unlikely they will be reduced to Cr-III due to a lack of organic matter which is necessary for the reduction to occur (Prokisch *et al.* 1997). Cr-VI can have long residence times in surface water, groundwater (Chandra and Kulshreshtha 2004) and can persist in soil for years, even with favourable reducing conditions (Sharma *et al.* 2003; Leita *et al.* 2009).

Chromite mining and ferrochrome production

Chromite ore is mined, crushed and processed to produce chromite concentrate. To create ferrochrome, chromite concentrate is combined with a reductant (coke, coal, charcoal or quartzite) in a high temperature submerged arc furnace or direct current arc furnace (ICDA 2011). See Appendix 1 for additional resources about ferrochrome production.

Mining and processing wastes and pollution

Chromite ore mining and concentrating produces dust, overburden, waste rock, tailings and tailings water. Ferrochrome production creates air pollution, dust, slag (waste produced during ferrochrome separation from ore) and process water. These waste materials have the potential to be contaminated with chromium and a number of other heavy metals and chemicals of concern.

According to the Cliffs chromite project proposal, 6000-12 000 tonnes of ore and 65 000 tonnes of waste rock will be produced each day at the mine site. The operation's predicted tailings output would require an area of 250 hectares to contain it. An estimated 2100 tonnes of slag would be produced per day by the ferrochrome plant, all of which would be cooled with water which would likely require treatment (Cliffs 2011). Concern over potential water pollution is heightened by the proximity of the proposed FeCr plant to Lake Wanapitei, a drinking water source for the City of Sudbury (May 2011). Pollution resulting from chromite mining and ferrochrome production is discussed in detail below.

The Canadian Metal Mining Effluent Regulations do not include a limit for the amount of chromium allowable in mine effluent to be discharged to the environment. The effluent is required to pass an acute lethality test for rainbow trout, which means that it cannot kill more than 50% of the fish tested within 96 hours. (Environment Canada 2010b).

Canadian and Ontarian criteria require that no more than 5000 ug/L of chromium be present in waste leachate or it is considered toxic waste. This limit is the same as the American limit, but significantly more than the Italian limit of 2000 ug/L. In addition, many other countries have set limits for Cr-VI specifically, which range from 20 to 1500 ug/L (Appendix 2, Table A-6).

Chromite mining pollution

Mining breaks up and brings rock to the surface, altering the stability of the rock components and giving rise to concerns about contaminants leaching into water and soils. By exposing chromite ore to aboveground conditions that promote weathering and oxidation, Cr-III may be converted to mobile and highly toxic Cr-VI (Godgul and Sahu 1995). In addition, it is now known that dry milling (grinding) of chromite ore converts some Cr-III to Cr-VI, a process previously thought to occur only at higher temperatures. As much as 168.1mg Cr-VI/kg ore has been measured after 10 minutes of milling. Efforts are therefore required to avoid producing and spreading hexavalent chromium during mining activities.

Studies suggest that milling ores while wet or in a non-oxygenated atmosphere (i.e. nitrogen) will decrease Cr-VI formation. Glastonbury's ore milling tests under nitrogen produced approximately 15 mgCr-VI/kg ore, several orders of magnitude less than regular milling tests, although more than Canadian soil guidelines for industrial and residential areas (Appendix 2 , Table A-4). Preliminary observations have shown that the duration of milling also affects the amount of Cr-VI formed. (Beukes and Guest 2001; Glastonbury *et al.* 2010).

Chromium-VI is not the only potential contaminant in chromite mining waste. In addition to chromium, the following metals and chemicals of concern are capable of leaching out of waste at neutral or alkaline pH: arsenic, antimony, cadmium, cobalt, manganese, mercury, molybdenum, nickel, selenium, sulphate and zinc. Unlike the standardised tests for predicting acid drainage risks, tests for predicting neutral drainage are not standardised or well researched (Stantec 2004). To date details of the chromite ore composition have not been released by Cliffs.

The Erin Brokovich Story

The film Erin Brockovich highlighted the now well-known example of long-term Cr-VI contamination of groundwater is Hinkley, California. From 1954 to 1966, the cooling tanks of the Pacific Gas and Electric Company's (PG&E) natural gas compression station used Cr-VI as a rust inhibitor. The resulting Cr-VI contaminated wastewater was stored in unlined ponds, which allowed the Cr-VI to percolate down approximately 80 feet into the groundwater, which was used as drinking water by residents (CRWQCB 2011).

Cases of cancer, birth defects and other health issues lead residents from Hinkley and other affected towns to file lawsuits against the company. PG&E paid $333 million in 1996 and $295 million in 2006 to settle most of these lawsuits (Kahn 2010; Pierson and So 2006).

As of December 2011, the concentration of Cr-VI in the groundwater is over 3.1 ug/L and the contamination continues to spread(Kahn 2010; CRWQCB 2011).

Around the open pit chromite mine in Kemi, Finland the average heavy metal values in pine bark were higher than background values: chromium (12.4 times higher), titanium (7.7 times), iron (5.3 times), nickel (3.0 times) and zinc (1.9 times) (Poykio *et al.* 2005). No studies were found that investigated how these pollution levels might affect the health of the local environment.

In India, unconfined dumping of chromite mine overburden, ores and waste rock has led to severe environmental degradation. Chromium contamination in the order of 250-3000 ugCr/L, in surface water was caused by drainage from a chromite mine and its overburden piles (Dhal *et al.* 2010). Another Indian study of open pit chromite mine recorded high hexavalent chromium levels in groundwater (30 ugCr-VI/L to 800 ugCr-VI/L) and surface water (30 ugCr-VI/L to 140 ugCr-VI/L) (Tiwary *et al.* 2005). When compared with the Canadian and American Cr-VI Water Quality Guidelines for Protection of Aquatic Life (1-16 ugCr-VI/L) and the Dutch regulation for *total* Cr in groundwater (30 ugCr/L) (Appendix 2, Table A-1) these levels are of concern.

High concentrations of chromium have been found in Indian mine effluents 20 ugCr-VI/L to 120 ugCr-VI/L and in mine seepage 50 ugCr-VI/L to 1220 ugCr-VI/L. In a 24 hour leaching test, soil from the mine property had a total of 2900 ugCr-VI/L (Tiwary *et al.* 2005), which is above the waste leachate limits of many countries (20-1500 ugCr-VI/L) (Appendix 2, Table A-6).

Ferrochrome production pollution

Ferrochrome production emits air pollution including nitrogen oxides, carbon oxides and sulfur oxides (NO_x, CO_x and SO_x). In addition, some compounds from the ore are emitted as gasses during smelting and then return to solids in the smelter dust. The reductant used in smelting may emit gas phases of arsenic, cadmium, chlorine, lead and sulphur, depending on whether coke, coal or charcoal is used (Ma and Garbers-Craig 2006a; 2006b).

Particulate dusts created during ferrochrome production have environmental and occupational health implications. For every tonne of ferrochrome created, up to 18-25kg of dust may be created. Dust collected during production must be treated before disposal in order to prevent the leaching of toxins such as chromium, zinc, lead, nickel and cadmium (Ma and Garbers-Craig 2006b, Bulut *et al.* 2009). Due to the leaching potential of these and other contaminants, ferrochrome arc furnace dust is categorized as toxic waste in Canada (waste K091) (Environment Canada 2010a).

> "The existence and treatment of wastes from stainless steel and ferrochrome production remain a challenge and an issue of concern" (Ma and Garbers-Craig 2006a).

The high temperature smelting of chromite ore converts some Cr-III into Cr-VI (Erdem *et al.* 2005). Chromium-VI cannot form in the highly reducing environment of the furnace but it is assumed to form at the top of the furnace or in the off-gas ducts where oxygen is available. Additional steps in the FeCr process may be employed depending on whether the ore is lumpy or fine. If an ore produces fines, the needed milling and agglomeration (i.e. sintering) steps prior to smelting can also produce Cr-VI (Beukes *et al.* 2010). The first tests on the "Big Daddy" chromite deposit just south of Cliff's Black Thor deposit yielded close to 100% lumpy ore (KWG 2011). The Cliffs project proposal does not identify the proportion of lumpy ore in the Black Thor deposit.

Leaching tests on FeCr dusts found that pH conditions favoured the stability of mobile Cr-VI compounds (Ma and Garbers-Craig 2006a). Up to 40% of the chromium in FeCr dust has been measured to be the

leachable and bioavailable Cr-VI (Cox and Linton 1985). Studies of industrially produced chromium contaminated dust show that Cr-VI is preferentially present in smaller particle sizes (< 10um), which makes them more available to humans via inhalation and subsequent ingestion (US EPA 1990).

The conversion of Cr-III to toxic Cr-VI during high temperature chromite smelting also results in contaminated slag (waste produced during separation of ferrochrome from ore) (Erdem *et al.* 2005). Total chromium content in slag has been estimated to range between 2 and 12% (Riekkola-Vanhanen 1999). In 2001, researchers were surprised to discover that dry pulverizing of slag and ore converts Cr-III to Cr-VI. This may be very important during chromite processing, ferrochrome recovery from slag and end-uses of crushed slag (Beukes and Guest 2001; Coetzer *et al.* 1997; Lind *et al.* 2001). When metal recovery from slag is part of the production process, contaminated water is also produced and may require treatment (Erdem *et al.* 2005). Some ferrochrome facilities sell crushed slag for construction end-uses though there are environmental concerns about contaminant leaching that must be addressed prior to slag use (Lind *et al.* 2001). Cliffs has made note of potential uses for its slag by industry (Cliffs 2011).

Studies on chromium leaching from FeCr slag have shown varied results. Two studies conducting standardised leaching tests (toxicity characteristic leach procedure TCLP) on crushed and ground slag did not exceed the US EPA regulation of 5000 ugCr/L (Gericke 1998; Erdem *et al.* 2005). These tests do not, however, analyse for leaching activity over time or leaching rates. It should be noted that the US regulation specifies the allowable amount of total Cr, not Cr-VI, and is higher than amounts allowed in a number of other countries such as Italy and Spain (2000 ug/L and 4000 ug/L, respectively) (Ma and Garbers-Craig 2006b). In the 2005 study, sequential washing of ferrochrome slag resulted in Cr-VI leaching from crushed slag Cr-VI (610 ug/L) and ground slag (3800 ug/L) (Erdem *et al.* 2005). These measurements are much higher than regulations specifying allowable Cr-VI in waste leachate, ranging from 50 ug/L to 1500 ug/L (Appendix 2, Table A-6) (Ma and Garbers-Craig 2006b).

A study of arc furnace dust from a ferrochrome smelter stack in Zimbabwe found that chromium was the most abundant heavy metal present. Air emissions from the stack polluted soils, resulting in 700 mgCr/kg at 700m from the furnace. One of the six furnace stacks emitted an average of 54.6 tonnes of Cr per year (Pumure *et al.* 2002).

A Finnish study analysing pine needles as bioindicators of heavy metal deposition found that needles sampled near a ferrochrome and steel plant had amounts of iron, molybdenum, strontium, and titanium 4.4 to 10.6 times higher than needles sampled near a pulp and paper mill. Chromium was 150 times greater near the ferrochrome and steelworks plant, with a maximum of 437 mg/kg (Poykio and Peramaki 2003). No studies investigating the effects of this pollution on plants or animals were found.

A maximum Cr-VI concentration of 7070 mg/kg dust was measured in emissions from the final stage of ferrochrome production in South Africa and exceeded the American Conference of Governmental Industrial Hygienists Documentation 8 hour exposure limit for *total* Cr by over 1000 times. Samples taken in the rainy season showed contribution of Cr-VI to surrounding soils, as seen by a gradient increasing towards the plant, with a maximum of 12.7 mg/kg soil. Chromium-VI concentration in grass was observed to be up to 4.2 mg/kg within 1km of the ferrochrome plant (Mandiwana *et al.* 2007).

Pollution from a ferrochromium plant in the JinZhou Province of China resulted in contamination of drinking water with up to 20 000 ugCr-VI/L and caused health problems to exposed citizens (Table 6) (Zhang and Li 1987; US EPA 1998).

In the Hunan Province of China, a ferroalloy plant deposited slag directly on the ground and discharged wastewater into the sewage system that runs through agricultural land. This resulted in long-term soil and vegetable contamination. In the soil, total chromium content ranged from 90 to 6200 mg/kg and

hexavalent chromium ranged from 0.1 to 252 mg/kg. All vegetables sampled contained more than 4 times the permissible amount of chromium (2.1 - 18.8 mg/kg dry weight). A health risk assessment based on estimated site-specific exposure to local vegetables and soil concluded there were elevated cancer risks as high as $6.6x10^{-6}$ for agricultural exposure and $2.2x10^{-5}$ for residential exposure to soil. The USEPA defines unacceptable cancer risk as greater than one in a million ($1x10^{-6}$) (Wang *et al.* 2011).

Environmental effects and toxicity

It is well established that microorganisms, plants and animals are capable of accumulating chromium from their environments. On the other hand, there is an inadequate amount of research regarding the form of chromium taken up (III or VI), the subsequent effects of accumulation and the doses at which effects occur. Most studies have been done in laboratories, which limits our ability to understand actual responses in the field.

Aquatic life

Chromium in aquatic ecosystems is known to bioaccumulate in algae, aquatic plants, invertebrates and fish (Marchese *et al.* 2008; Dwivedi *et al.* 2010). Metal bioaccumulation varies greatly among aquatic species, as seen by a wide range of chromium bioconcentration factors observed for fish and invertebrates (Marchese *et al.* 2008; Kimborough *et al.* 1999; Weegman and Weegman 2007). Uptake and effects are influenced by the species, organism size, sex and developmental stage, presence of other contaminants, water temperature, pH, alkalinity and salinity (Eisler 1986). Researchers acknowledge there is still little information on chromium uptake and effects in freshwater species (Marchese *et al.* 2008).

Duckweed (*Lemna spp.*) is the most chromium sensitive aquatic macrophyte found to date and exhibited reduced growth from exposure to 10 ugCr-VI/L (Eisler 2007). Some algae have exhibited severe signs of toxicity from as little as 1-10 ug Cr-VI/L (Health Canada 1994). Cr-III has also been observed to affect algae (320 ug/L), and the standard test procedure may be underestimating its toxicity (Pawlisz 1997; Vignati *et al*. 2010).

Laboratory tests have shown toxic effects of chronic Cr-III exposure at concentrations as low as 5 and 6 ug/L. The effects observed included decreased success in fertilization and reduced growth of rainbow trout (*Oncorhynchus mykiss*). At concentrations as low as 44 ug/L, aquatic invertebrate *Daphnia magna* showed decreased fecundity (Billard and Roubaud 1985; MOE 2011a; Eisler 2007).

Evidence suggests that Cr-III in water is more toxic to fish than Cr-VI as it damages gills, and can cause reproductive damage and even death at relatively low doses (Health Canada 1994). Chromium-VI does not deposit on gills but enters the fish and exerts toxic effects on internal organs such as the liver and kidney. When placed in non-contaminated environments, fish that have accumulated chromium in their bodies can excrete some of it, but studies have observed continued elevated levels of chromium in tissues such as liver, kidney and muscle as long as 90 days after exposure (Eisler 1986).

Observed effects of Cr -VI in fish from laboratory studies include:

- increased hatching time for salmon (10 ug/L) (Pawlisz 1997);
- reduced growth of rainbow trout and Chinook salmon fingerlings (16-21 ug/L) (Eisler 1986);
- DNA damage (24-54 ug/L) (Farag *et al.* 2006);
- altered lipid metabolism and digestion (120 ug/L) (Farag *et al.* 2006);
- microscopic lesions, gross abnormalities, reduced weights (120 ug/L) (Farag *et al.* 2006);
- reduced survival (120-266 ug/L) (Farag *et al.* 2006);

- decreased disease resistance (500 ug/L) (Sugatt 1980);
- altered enzyme activities and metabolism (2600 ug/L) (Sastry and Sunita 1983);
- oxidative stress of liver and kidneys (4250, 8570 ug/L) (Velma and Tchounwou 2011)

Laboratory studies have also observed Cr-VI effects on freshwater invertebrates including:

- decreased fecundity and survival (10 ug/L) (Trabalka and Gehrs 1977);
- over 50% immobilized (presumed death) (53 ug/L) (Hickey 1989);
- behavioral changes indicating stressful/pre-lethal conditions (100 ug/L) (Catalan 1982);
- reduced growth (1100-3000 ug/L) (Honig *et al.* 1980)

The literature has important limitations for understanding the potential impacts of chromite mining and ferrochrome processing on natural aquatic ecosystems as nearly all the studies located were conducted in labs and did not include observations of wild populations in the field. Furthermore, of the available studies, none were directly related to chromium released from chromite mining or ferrochrome processing. The only field study located that examined wild fish found no difference between the chromium concentration in muscles of control fish compared to those in bluegill *(Lepomis macrochirus)* and largemouth bass *(Micropterus salmoides)* exposed to 100 – 300 ugCr-VI/L in White Oak Lake water contaminated by electrical plant cooling tower waters. The authors hypothesized that the chromium was in a form that was not bioavailable to the fish, or that the fish were capable of regulating accumulation (Elwood *et al.* 1980).

The Canadian Water Quality Guideline for the Protection of Freshwater Aquatic Life is currently set at 1 ugCr-VI/L, based on toxicity to the invertebrate *Ceriodaphnia dubia* and an interim guideline for Cr-III is set at 8.9 ugCr-III/L, based on toxicity to rainbow trout (Appendix 2,Table A-1). There is currently no chromium tissue residue guidelines for the protection of wildlife that consume aquatic prey species (CCME 1999).

The Canadian Interim Sediment Quality Guideline for bioavailable total chromium is 37.3 mg/kg of dry sediment, while the Probable Effects Level for total chromium(for aquatic organisms) is 90 mg/kg. Cr-VI can be present in the top layer of sediment when the overlying water contains oxygen, but no guideline has been established for Cr-VI in sediment. The Ontario Contaminated Site Condition Standard for total chromium in sediment is set at the upper limit of typical sediment concentrations in the province, 26 mg/kg (Appendix 2, Table A-3).

Aquatic food chain risks

Chromium-VI that is taken up into an organism is easily transported into cells where it exerts toxicity through its reduction to Cr-III. However, it is hypothesized that this reduction renders the chromium less dangerous to predators consuming the organism, since Cr-III is not as easily taken into cells and can be expelled by the predator. (Arillo and Melodia 1991). A number of studies have found chromium concentration decreases in organisms higher up a food chain (Kimborough *et al.* 1999). Chromium is therefore not considered likely to biomagnify in the aquatic food chain (Eisler 2007; MOE 2011a).

A case which contradicts the suggestions above has been documented with potential population-level effects of chromium in the food chain of an aquatic bird – the lesser scaup. Bioaccumulation of chromium was identified in the migratory lesser scaup population in the course of searching for the cause of their dramatic population decline. The elevated chromium levels detected in the scaups' livers were lower than any levels known to cause effects in lab studies, but little data exist regarding chromium threshold effect levels and effects on reproductive capacity of birds in field situations (Custer *et al*. 2003). A later study found chromium accumulation in invertebrates eaten by scaup (>0.1 ugCr/g in fingernail clams and amphipods) and hypothesised this may be influencing the declining bird population (Weegman and

Weegman 2007). Chromium pollution in the studied areas was thought to be from fertilisers and pesticides, which may represent very different pollution burdens than those from chromite mining and ferrochrome processing. More research is needed to understand potential risks of excess chromium to aquatic life.

Terrestrial life

Information on the doses of chromium in water or food that cause health problems in mammals come mostly from toxicology lab tests done on mice and rats. Effects observed on animals in experimental doses through food, water or injection include: cancers, reproductive harm, behavioral changes, reduced growth and reduced survival (Nriagu and Kabir 1995). Laboratory studies conducted on mammals have determined *Lowest Observed Adverse Effects Levels* (LOAELs) for Cr-VI in drinking water of: 100 000 ug/L for mice, 70 000 ug/L for rats and 62 700 ug/L for dogs. LOAELs of Cr-III in drinking water of rats be 28 000 ug/L) and mice (5 000 ug/L) and are notably lower than those for Cr-VI (Pawlisz 1997).

Canada has set Interim Quality Criteria of 50 ugCr-VI/L and 50 ugCr-III/L for water provided to livestock (Appendix 2, Table A-1).

Very few field studies have been conducted about the effects of environmental chromium pollution on wildlife and we did not find any information directly relevant to potential impacts of chromite mining and ferrochrome production on terrestrial wildlife.

Soil organisms are important in creating soil structure and cycling organic matter and elements in the environment. Relatively small amounts of Cr-VI (1-10 mg/kg) and Cr-III (25-100 mg/kg) can negatively impact the number, type, health and activity of soil organisms and so impact the health of the soil ecosystem (MOE 2011a; Viti *et al.* 2006). Tests conducted under and near a steel alloy plant slag heap in China found that chromium was related to decreased microorganism numbers and enzyme activity (Huang 2008). Ten thousand to fifteen thousand ug/L of Cr-VI in water applied to soil was lethal to two species of earthworms (Eisler 1986). Total chromium levels of 671 to 1400 mg/kg killed 50% of a tested earthworm population (*Eisenia fetida*) and 1000 mgCr/kg significantly reduced earthworm (*Eisenia andrei)* growth and cocoon production (CCME 1999).

Canada's chromium in soil guidelines for the protection of environmental and human health are generally lower than soil standards in other countries .On Canadian agricultural, residential and park soils total Cr should be below 64 mg/kg and Cr-VI below 0.4 mg/kg. For industrial and commercial lands, the limits are 87 mgCr/kg and 1.4 mgCr-VI/kg.

The upper limits of typical Ontario chromium concentrations in uncontaminated soils are slightly higher than the Canadian guidelines, with 0.66 mg/kg Cr-VI and 67 to 70 mg/kg total chromium. The Ontario Site Condition Soil Standards have set higher limits than the Canadian guidelines. Limits for soils of all property uses are 160 mgCr/kg and 8 to 10 mgCr-VI/kg. Lower limits for soil within 30m of a water body are set at 67 to 70 mgCr/kg and 0.66 mgCr-VI/kg (Appendix 2,Table A-4).

Plants can accumulate Cr-III and VI from soil, sediment, water and atmospheric deposition on leaves. Plant exposure to excess Cr-III or VI can negatively affect plant health and survival. The complex influence of soil chemistry and differences between plant species make it impossible to generalise. Questions also remain regarding the potential for toxic forms of chromium to be present in plants and contribute toxic effects through the diets of wildlife and humans.

Increases in plant growth and yield have been observed when small amounts of Cr-III are available to plants, but whether plants require Cr-III as an essential element is still not clear (Kimborough *et al.* 1999; Zayed and Terry 2003; Sharma *et al.* 2003). There is some debate about whether Cr-III or Cr-VI is more toxic to plants. Most studies have found Cr-VI effects occur at lower concentrations and with greater severity, but at least one study has observed more severe effects induced by Cr-III (Gardea-Torresdey *et al.* 2005).

Chromium toxicity to plants has been observed at exposure to levels as low as 160 ugCr-VI/L (Adema and Henzen 1989) and 104 ugCr-III/L (Pawlisz 1997) when grown in soil-less solution and 1.8 mg/kg (added as Cr-VI) when grown in soil (Adema and Henzen 1989). Canada has Quality Criteria for agricultural irrigation water, set at 4.9 ug/L for Cr-III (interim) and 8 ug/L for Cr-VI (Appendix 2, Table A-1). Based on plant contact with soil, a Canadian Provisional Guideline for the protection of environmental health has been calculated at 0.4 mgCr-VI/kg for agricultural soils, and 1.4 mgCr-VI/kg for industrial and commercial soils (Appendix 2, Table A-4).

Toxic effects to plants exposed to excess chromium include: reduced growth, decreased chlorophyll production causing yellow leaves, narrow leaves, small root systems, decreased or complete inhibition of seed germination, delayed growth, decreased seed yield, wilting and death (Dube *et al.* 2003; Zayed and Terry 2003). Excess chromium damages root membranes and a plant's ability to take up water. It also alters uptake and translocation of essential elements such as nitrogen, iron, potassium, magnesium, manganese, phosphorous, calcium, sulphur, copper and zinc (Zayed and Terry 2003; Dube *et al.* 2003; Gardea-Terresdey *et al.* 2005). Not only is this detrimental to plant health, but a change in plant nutrient balances may also affect wildlife and human health. Sensitivity and effects vary between species, making toxicity predictions difficult without extensive plant studies.

The amount of chromium taken up by plants varies among species due to differences in absorption, transportation and storage of metals (Zayed *et al.* 1998; Chandra and Kulshreshtha 2004). Although most plants studied store the majority of chromium in their roots, translocation to all other parts of a plant does occur in proportions that vary across plant species. Many studies have found low plant ability to transport chromium from roots to other plant parts. However, greater chromium concentrations in leaves than in roots have also been observed (Dube *et al.* 2003) and several species with the ability to accumulate over 1000 mgCr/kg in their above ground parts have been identified and are classified as hyperaccumulators. For example, a study by Peterson in 1975 found levels in leaves as high as 48,000 mgCr/kg (Zayed and Terry 2003) and a recent study documented the ability of rice to effectively translocate chromium from root to shoot at 100 days growth (Mohanty *et al.* 2011). These hyperaccumulators can be aquatic or terrestrial plants and include swamp grass (*Leersia hexandra*), ragweed (*Ambrosia artemisiifolia*), water ferns (*Salvinia molesta*), duckweed (*Spirodela polyrhiza*) and mustard greens (*Brassica juncea*) (Srivastav *et al.* 1994; Liu *et al.* 2011). The original form of chromium taken up by the plant may also influence its storage location, as seen in a study that observed Cr-VI exposure resulted in more translocation from stem to leaf than Cr-III exposure (Gardea-Torresdey *et al.* 2005).

There is evidence that plants can convert Cr-VI to Cr-III in their roots (Zayed *et al.* 1998; Zayed and Terry 2003; Howe *et al.* 2003) though there are differences in observed rates and extent of reduction to Cr-III. Some studies support the concept that plants can quickly convert all Cr-VI to Cr-III (Lytle *et al.* 1998; Zayed *et al.* 1998; Wang *et al.* 2011) while other studies have found evidence of Cr-VI and its intermediates, Cr-V and Cr-IV, persisting in plants and algae (Micera and Dessi 1988; Liu *et al.* 1995; Mishra 1995; Aldrich *et al.* 2003; Howe *et al.* 2003). As Cr-VI intermediates are implicated in chromium's toxic effects (Stearns *et al.* 1995b), their presence in plants could "produce dangerous effects to ecological cycles" (Micera and Dessi 1988).

Terrestrial food chain risks

Some researchers suggest that there is a "soil-plant-barrier" that protects the terrestrial food chain from excess chromium due to: a) the insolubility of most chromium in soils; b) >90% of chromium that is taken up is stored in plant roots; c) plant toxicity occurring below levels thought to affect plant consumers (Zayed and Terry 2003). This may not , however, apply in the following cases:

- Where industrial contamination provides bioavailable Cr-VI;
- In soils where Cr-III is readily oxidized to Cr-VI;
- With plant species capable of accumulating levels at or above those thought to affect consumers;
- With plants species that store a greater proportion of Cr in aboveground parts; and
- Where plant roots are sources of food for wildlife or humans.

There is no documented evidence for biomagnification of chromium from soil to plant to animal (ATSDR 2008a), though researchers also acknowledge there is a lack of study regarding the risk of chromium in the food chain (Lind *et al.* 2001; ATSDR 2008a; Peralta-Videa 2009).

Human health effects

The human exposure pathways to chromium are inhalation, ingestion (swallowing) and dermal (skin) contact. Chromium exposure in humans and laboratory animals is known to cause cancer, interfere with physical development and har the skin, respiratory, reproductive and digestive systems.

> Environmental and occupational exposure to Cr-VI is still considered a "major human health issue" today (Nickens *et al.* 2010).

Chromium-VI is much more toxic than Cr-III because of its greater ability to enter cells and its strong oxidation potential (Katz and Salem 1993). Once inside cells, Cr-VI reduction produces free radicals, Cr-V, Cr-IV and eventually Cr-III, which are believed to be responsible for toxic and carcinogenic effects (Stearns *et al.* 1995b; Li *et al.* 2011). Toxicity varies among chromium compounds and most non-lab studies do not track exposure to a specific compound, but assess risk based on exposure to total Cr (Cr present in all valence states), Cr-III or Cr-VI compounds. Inhalation of Cr-VI is considered more dangerous than ingestion. (OEHHA 2011). The effects of low level chronic exposures and the interactions between co-contaminants (ex. from industrial work or chromium from other sources such as cigarettes) are not well understood.

As described above, Cr-VI has a greater ability to cross cell membranes than does Cr-III. Some scientists believe that the human body's saliva, digestive juices and red blood cells provide protection from chromium toxicity by reducing ingested Cr-VI to Cr-III before cell absorption. Chromium-VI toxicity could then occur only when the amount of chromium ingested is above a threshold where the ability of the body to reduce chromium is overwhelmed (De Flora *et al.* 1997). Based on this theory, arguments have been made that Cr-VI between 1000 and 10 000 ug/L is safe for human consumption (De Flora 2000; Paustenbach *et al.* 2003).

Other researchers do not believe that natural chromium reduction in the body is a reliable safeguard against toxicity. Comparison of chromium uptake found that orange juice is better at reducing Cr-VI than human stomach acid (Costa 1997). Higher levels of chromium observed in human and animal tissues after

Cr-VI ingestion compared to Cr-III ingestion prove that not all Cr-VI is reduced by the body before absorption (Kerger *et al.* 1996; Costa 1997; Davidson *et al.* 2004). Significant accumulation of chromium due to Cr -III ingestion has been seen in animal tissues including liver, lungs, spleen and heart (Stearns *et al.* 1995a). Chromium accumulation due to Cr-VI ingestion has also been seen in human and animal tissues including blood, bone, testis, liver, spleen and kidneys (Kerger *et al.* 1996; Finley *et al.* 1997; Sutherland *et al.* 2000; NTP 2007; US EPA 2010). It is known that reducing capabilities vary among individuals and among animal species, adding uncertainty to the use of animals studies extrapolated to humans, and the protection of sensitive individuals. Groups such as Health Canada, the American Occupational Safety and Health Administration and the European Union's Scientific Committee on Occupational Exposure Limits have all concluded that current information is not enough to support the existence of a threshold below which Cr-VI is non-toxic (MOE 2011a). The effectiveness of the body's reducing processes, the possible threshold levels for various exposure pathways and the effects of various chromium species in cells at higher than background levels are not yet fully understood.

Excretion of accumulated chromium-III and VI from human and animal tissues can be very slow. Autopsies performed on chromate workers who died of lung cancer measured chromium accumulation in tissues approximately 20 years after chromium exposure had ended (Ishikawa *et al.* 1994). Workers exposed to Cr-VI in Japan had high levels of chromium in respiratory organs, spleen, liver, kidney and heart tissues, up to 30 years post-exposure (Teraoka 1981); and tannery workers exposed to Cr-III had long-term elevated chromium levels in their bloodstream (Aitio *et al.* 1984; Randall and Gibson 1987).

Dermal exposure

Direct skin contact with Cr-VI compounds can cause inflammation, eczema and open sores (ulcers) (Barceloux 1999). Chromium-VI exposure levels between 20-25 ppm are known to cause skin ulcers and the no-observed-effect-level (NOEL) for ulcers is not known (Shelnutt *et al.* 2007). Some Cr-III and VI compounds can be absorbed and accumulate in the body through the skin. Large-scale dermal exposure to chromic acid can cause severe burns and toxic effects similar to those of ingestion (Barceloux 1999).

Chronic low-level exposure to Cr-III or VI causes some people to become permanently sensitised to chromium and develop a skin condition called allergic contact dermatitis (ACD). This occurs with chronic exposures between 4-25 ppm Cr-VI (Shelnutt *et al.* 2007). A review of many studies identified 10 ppm Cr-VI as the threshold at which no more than 10% of exposed individuals developed skin sensitisation. The same threshold was 500 ppm for Cr-III (Bagdon and Hazen 1991). A no-observed-effect-level (NOEL) has not been identified for Cr-VI causing ACD. Estimates of the fraction of a population to become sensitised range from 0.08% in general populations to 40% in occupationally exposed populations (Shelnutt *et al.* 2007).

Chromium is present in many materials used every day, such as food, detergents, leather and paints, and is capable of provoking the allergic response. Since it is so

Housewives in Japan and ACD

Non-occupational exposures to a Cr-VI contaminated environment can also induce ACD, as seen in a study of housewives exposed to chromium contaminated slag from a chemical plant that was used as urban fill in Tokyo, Japan. A 1987 study by the Tokyo Metropolitan Government Bureau of Sanitation (TMGBS) found significant increases in eczema and ACD in summer months compared to a non-exposed control group. Contact with contaminants is likely greater in the summer and several studies have linked exposure to sunlight with greater severity of chromium induced skin problems (Bagdon and Hazen 1991).

difficult to avoid everyday exposure to small amounts of chromium, ACD is a serious and unresolvable issue for many individuals (Shelnutt *et al*. 2007). Reports of skin lesions and eczema lasting years are not uncommon, leading to a significant amount of work time lost (Bagdon and Hazen 1991) and changes of occupation. Changes in occupation did not necessarily improve skin conditions and often resulted in negative social and economic impacts. As there is no specific treatment for chromium induced skin issues, prevention of sensitisation is recommended as the best solution (Breit and Turk 1976).

Some evidence shows that ingestion of Cr-VI and Cr-III can produce ACD in sensitised individuals, although more research is needed. Studies are also needed to determine if chromium inhalation can cause ACD in sensitised individuals (Shelnutt *et al.* 2007).

Inhalation and the respiratory system

Both Cr-III and VI are respiratory and mucous irritants while Cr-VI is linked to occupational asthma (Assem and Zhu 2007). Chromium-VI dusts alos irritate the nose, throat and lungs, and can cause respiratory inflammation, nosebleeds, ulceration and perforation (holes) in the septum (structure between the nostrils) (Barceloux 1999). Septal ulcers and perforations have been observed with exposure to as little as 0.09 ugCr-VI/m^3 (MOE 2011a) and reversible impairment of lung function with exposures as low as 2 ugCr-VI/m^3 (Lindberg and Hedenstierna 1983).

No evidence of Cr-III inhalation induced asthma has been documented, and Cr-III compounds are not considered respiratory sensitizers. However, animal studies show that chronic inhalation of Cr-III can induce inflammation in the respiratory tract (Santonen *et al.* 2009). Toxicity information for acute inhalation exposure to Cr-III compounds is not available (Assem and Zhu 2007).

While the Canadian Council of Ministers of the Environment (CCME) has no Air Quality Guidelines for the protection of human health and the environment for chromium (Appendix 2, Table A-5), Ontario's Ministry of Labour has set 8-hr average workplace air standards for Cr (0, III) (500 ug/m^3), soluble Cr-VI (50 ug/m^3) and insoluble Cr-VI (10 ug/m^3). These Cr-VI limits are higher than the levels set by the American National Institute for Occupational Safety and Health (Cr-VI - 1ug/m^3 over 10 hours) and the Occupational Safety and Health Administration (soluble Cr-VI as CrO_3 – 5 ug/m^3 over 8 hours) (Appendix 2, Table A-5)

In June 2011, the Government of Ontario set Ambient Air Quality Criteria (AAQC) and Air Standards for Cr-VI compounds and other Cr compounds (0, II and III) (Table 1). AAQC are used for environmental assessments, while Air Standards provide enforceable regulations with the goal of protecting communities from industrial air pollution. The standards are under Ontario Regulation 419/05: Air Pollution - Local Air Quality, under the Environmental Protection Act. They will become effective July 1, 2016, after a five-year phase-in period for industry. Companies may also apply for less stringent site-specific or sector-based technical standards (MOE 2011b).

Table 1 Environmental standards and guidelines for chromium in air.
PM_{10} is particulate matter <10um in diameter. TSP is total suspended particulate size fraction. MOE is Ministry of the Environment.

Limit (ug/m^3)	Cr	Source	Details
none	-	Canada[1]	Air Quality Guidelines for the protection of human health and environment
1.5	II, III	Ontario MOE[2]	24-hr average Ambient Air Quality Criterion set in 1982
1.5	0, II, III	Ontario MOE[2]	0.5-hr average Standard based on Cr-III respiratory effects, effective 2016
0.5	0, II, III	Ontario MOE[2]	24-hour average Air Standard and Ambient Air Quality Criterion based on Cr-III respiratory effects, effective 2016
0.00035	VI, PM_{10}	Ontario MOE[2]	24-hr average Ambient Air Quality Criterion, based on Cr-VI carcinogenicity, effective 2016
0.00007	VI, PM_{10}	Ontario MOE[2]	annual Ambient Air Quality Criterion, based on Cr-VI carcinogenicity, effective 2016
0.002	VI, TSP	Ontario MOE[2]	0.5-hr average Air Standard for Cr(VI), based on Cr-VI carcinogenicity, effective 2016
0.0007	VI, TSP	Ontario MOE[2]	24-hr Ambient Air Quality Criterion, based on Cr-VI carcinogenicity, effective 2016
0.00014	VI, TSP	Ontario MOE[2]	annual Air Standard and Ambient Air Quality Criterion, based on Cr-VI carcinogenicity, effective 2016

Effects on reproduction and development

Developmental toxicity of Cr-VI has been observed in animals, but to date there has not been not enough work done to characterise the potential for developmental effects on humans (Barceloux 1999; ATSDR 2008a; Banu et al. 2008). Chromium can be transferred from mother to young through the placenta and mother's milk (Barceloux 1999) and increased birth and developmental defects in children have been informally noted in areas of poorly regulated chromite mining, leather tanning (using Cr) and chrome production (Blacksmith Institute 2007). No scientific studies investigating the potential relationship between these effects and specific chromium exposures in these locations have been located.

Damage to male and female reproductive systems in animals and humans exposed to Cr-VI has been observed (Table 2 and Table 3). Several noteworthy studies documented abnormal menses, increases in post-birth hemorrage and birth complications in a group of women exposed to industrial chromium contamination (Makarov and Shmitova 1978; Shmitova 1978; Shmitova 1980). However, the Agency for Toxic Substances and Disease Registry (ATSDR) reviewed Shmitova's 1978 and 1980 studies and judged they were of too poor quality to support any conclusions about chromium effects on human reproduction (ATSDR 2008a). A number of other studies have shown no effects on human or animal reproductive systems (Barceloux 1999; ATSDR 2008a).

Chromium-III is not considered a developmental toxin (Santonen 2009), though some evidence for the reproductive toxicity of Cr-III has been observed. Other studies have reported conflicting results (MOE 2011a) and more research is required.

[1] Canadian Council of Ministers of the Environment. 1999. http://ceqg-rcqe.ccme.ca/
[2] Ontario Ministry of the Environment. 2011.
http://www.downloads.ene.gov.on.ca/envision/env_reg/er/documents/2011/010-6353.pdf

Table 2 Some observed reproductive and developmental effects of chromium in animal studies.

Study Description	Observed Effects
O'Heany 1986: animals exposed to Cr	malformations including cleft palates, skeletal deformations
Murthy *et al.* 1996: adult female rats exposed to Cr-VI in drinking water	toxic to ovaries: significantly decreased number of follicles (250, 500, 750 mg/L), significantly decreased # ova and ovarian tissue damage (500, 750 mg/L), significantly increased estrus cycle and infertility (750 mg/L)
Junaid *et al.* 1996: mice exposed to Cr-VI in drinking water	toxic to fetuses and embryos (250, 500, 750 mg/L); significant increase of gross and skeletal abnormalities (250, 500 mg/L)
Elbetieha and Al-Hamood 1997: male and female mice exposed to Cr-III and Cr-VI in drinking water, ranging from 2000 - 5000 mg/L	(III): decreased male fertility; (VI): decreased implantations and viable fetuses; (III) and (VI): increased fetal death, changes in reproductive organ weights
Kanojia *et al.* 1998: female rats exposed to Cr-VI in drinking water	decreased fertility, toxic to fetuses, changes to mating (estrous) cycle, significantly increased gross and skeletal abnormalities (5.57, 10.18, 13.56 mg/rat/day)
Aruldhas *et al.* 2004; Subramanian et al. 2006: male monkeys exposed to Cr-VI in drinking water	toxic to reproductive organs and sperm (100, 200, 400 mg/L)
Aruldhas *et al.* 2005: male monkeys exposed to Cr-VI in drinking water	toxic to sperm, changed endocrine organ weight, testis enzymes levels (100, 200, 400 mg/L)
Banu *et al.* 2008: young female rats exposed to Cr-VI in mother's milk (mothers drinking water with 200 mgK_2Cr2O_7/L)	Toxic to ovaries, altered hormone production, delayed puberty, increased resorption of embryos
Oliveira *et al.* 2010: male mice injected with Cr-VI as K_2CrO_4	toxic to sperm (5, 10 mg/kg)

Table 3 Some observed reproductive effects of chromium in humans.

Study Description	Observed Effect
Makarov and Shmitova 1978: women occupationally exposed to chromium	abnormal menstruation
Shmitova 1978; Shmitova 1980: women working in chromium compound plant	postnatal hemorrhage and birth complications
Li *et al.* 2001: men working in chrome plating plant	Significantly decreased sperm count and motility
Danadevi *et al.* 2003: men exposed to chromium and nickel in welding fumes	toxic to testes, decreased sperm concentrations with increased Cr blood levels, increased semen abnormalities
Kumar *et al.* 2005: men working in chromium sulfate manufacturing	Significantly increased sperm abnormalities

Effects on other human health systems

Ingestion of large amounts of hexavalent chromium, for example occurring as accidental chemical ingestion, is known to cause nausea, vomiting, stomach and intestinal damage, anemia, kidney and liver damage and failure, coma and even death (Barceloux 1999; ATSDR 2008a). Health effects observed in a human population chronically exposed to approximately 20 000 ugCr-VI/L in drinking water contaminated by a ferrochrome plant included mouth sores, diarrhea, stomach pains, indigestion, vomiting, and higher levels of white blood cells than the reference population (Zhang and Li 1987; US EPA 1998). Exposure to contaminated slag used as fill in a city caused symptoms characteristic of early kidney disease (TMGBS 1987). Some evidence implicating occupational Cr exposure to heart disease and inhalation of Cr-VI in dust leading to gastrointestinal, kidney and liver damage has been found, but findings to date are not conclusive (Gibb *et al*. 2000, Wedeen and Qian 1991; Barceloux 1999; Assem and Zhu 2007).

Carcinogenicity

The International Agency for Research on Cancer (IARC) states that metallic chromium (0) and trivalent chromium are not classifiable as human carcinogens due to insufficient evidence (IARC 1990). The US Environmental Protection Agency's Integrated Risk Information System, states that no carcinogenicity has been reported for any types of exposure to Cr-III compounds (US EPA 1998).

The carcinogenicity of Cr-III ingestion has not been entirely ruled out. Modeling has predicted that regardless of the lower capacity of Cr-III to be absorbed into cells, Cr-III exposure has the potential to accumulate in human tissues at a level that has caused genotoxic or mutagenic effects in cells and live animals (Stearns *et al.* 1995a). It cannot be determined without further research what level of occupational or environmental exposure to trivalent chromium compounds may lead to elevated cancer risks.

Hexavalent chromium is classified as a known human carcinogen by Health Canada, the US EPA (via inhalation) and the IARC (1994; 1998; 1990). The evidence conclusively shows that chronic inhalation of Cr-VI causes lung and sinonasal cancers at occupational levels experienced by workers in chromate (a Cr-VI compound) chemical production (De Flora 2000). Increased risks of bronchial cancer have also been observed in a number of epidemiological studies investigating occupational chromium exposure of workers in chrome plating, chromate chemical production and chromate pigment production (Katz and Salem 1993). Gold miners working in Ontario in the mid 1900s suffered increased stomach cancers that were linked to exposure to chromium found in the ore (Kusiak *et al.* 1993).

Some researchers have declared that oral exposure to Cr-VI is proven to be non-carcinogenic through many epidemiological and animal studies (De Flora 2000), but it is acknowledged by most that it is not known if oral exposure to chromium causes cancer (Costa 1997; Davidson *et al.* 2004; Costa and Klein 2006; Paustenbach *et al.* 2003).

The amount of evidence linking oral exposure to Cr-VI and cancer is increasing. For example, animal oral exposure to Cr-VI has been observed to damage DNA (ATSDR 2008a), increase incidence of stomach tumours (Borneff *et al*. 1968) and produce oral and intestinal tumours (NTP 2008; Stout *et al.* 2009). The Agency for Toxic Substances and Disease Registry maintains that there is evidence of increased risk of stomach cancers in both animals and people consuming Cr-VI polluted water (ATSDR 2008a). In 2004, a study observed a strong link between ingested Cr-VI and increased skin cancer in mice exposed to UV radiation (Davidson *et al.* 2004).

Few human epidemiological studies have been conducted for Cr-VI oral exposure and those that have been conducted are limited by factors such as study design and uncertainties regarding actual exposure levels, co-contaminants etc. A number of these studies are included in Table 6. Consensus and regulatory decisions about Cr-VI ingestion have been complicated by controversy surrounding an influential study of chronic Cr-VI ingestion in a human population (Zhang and Li 1987). See Appendix 3 for more information about the controversy.

Overall, both laboratory animal studies and human epidemiological studies have presented some evidence that chromium ingestion may be linked to a range of cancers including those of: skin, lung, lip/oral cavity/pharynx, breast, liver, kidney, bladder, gastrointestinal tract, urinary tract, testes, prostate, brain, stomach, bone, lymphoma and leukemia (Costa 1997; Davidson *et al.* 2004; Linos *et al.* 2011). These are contradicted by other studies investigating chromium ingestion and non-respiratory cancer mortality that have found no such effects (De Flora 2000; Kerger *et al.* 2009).

Notable developments in recent years regarding regulation of oral exposure to Cr-VI show a turning towards recognizing oral exposure to Cr-VI a likely human carcinogen (OEHHA 2009; Stern 2010; US EPA 2010). While Canada and the USA do not currently have separate drinking water standards for Cr-VI, the EPA is in the process of evaluating Cr-VI oral exposure carcinogenicity. A 2010 draft report of the US EPA's toxicological assessment for Cr-VI in drinking water classifies it as "likely to be carcinogenic to humans." However the final EPA determination has not been released and external peer reviewer comments varied in either support of the classification, or argument for a lesser classification (US EPA 2010; Byczkowski *et al.* 2011).

In 2009 the California EPA released a draft report on Cr-VI stating that "The findings of available human, animal, genotoxic, and toxicokinetic studies all indicate that hexavalent chromium is a possible human carcinogen by the oral route" (OEHHA). The state has set a very low (non-enforceable) Public Health Goal (PHG) of 0.02 ug/L specifically for Cr-VI in drinking water (EWG 2010). The PHG will play a role in determining California's enforceable maximum contaminant level for Cr-VI, which has yet to be set (EWG 2010).

Chromite mining exposure risks

No recent studies on the health of chromite miners have been located. One study in 1957 found evidence of pneumoconiosis, a lung disease due to dust inhalation, in miners who had worked only in chromite mines (Walters 1957). A second study examined ten chromite miners with radiological evidence of pneumoconiosis. Five had mined solely in chromite mines. This study suggested that the lung damage was benign and did not cause fibrosis (Sluis-Cremer and Du Toit 1968). As mentioned above, increased stomach cancers in Ontario gold miners were attributed to chromium exposure. It should be noted that industrial hygiene conditions have improved since these studies.

The presence of Cr-VI in chromite mining processes is generally thought to be low. A recent study analyzing air-borne breathable dust levels and their chromium content at mechanized and semi-mechanized open pit chromite mines in India identified Cr-VI in the dust. The maximum values of hexavalent chromium (0.2242 and 0.4186 ug/m^3) in the dust were below occupational health and safety guidelines (Appendix 2, Table A-5) (Panigrahi *et al.* 2006).

In 2007, another study measured Cr-VI in unprocessed chromite ore (0.38 to 0.44 µg/g) and crushed lumpy ore (0.62 to 0.76 µg/g) (Mandiwana *et al.*). This could have implications for the potential exposure of mine workers. The findings of this study have been questioned by other researchers suggesting the

results were affected by the grinding of the ore samples, which facilitated the oxidation of Cr-III to Cr-VI. (Glastonbury *et al.* 2010). It is not clear whether the crushing and sorting techniques employed in ore processing and chromium concentration at a mine site have the same potential to create Cr-VI as Glastonbury *et al.* (2010) showed in laboratory grinding. The higher Cr-VI concentrations in ore that had been crushed prior to grinding in the lab by Mandiwana *et al.* (2007) and the Cr-VI measured in dust by Panigrahi *et. al.* (2006) (which was not ground for analysis) indicate that this may be case.

Ferrochrome production exposure risks

Since the 1930's, worker exposure (including ferrochrome workers) to Cr-VI in dust has been linked to occupational asthma (Joules 1932; Bergmann 1934; Langard 1980; IARC 1990). Nasal skin damage and septal perforation have also been noted in ferrochrome workers due to exposure to Cr-VI. These effects can be prevented through the use of breathing filters (Axelsson *et al.* 1980).

In addition to risks from exposure to chromium, ferrochrome workers may risk respiratory disease due to overexposure to silica and dust (Moulin *et al.* 1990; Scott and Grayson). Increased prevalence of generalized obstructive lung disease (GOLD) and decreased lung function was noted in workers of ferrochromium and ferrosilicon plants. It was concluded that these effects were due to dust exposure and not to chromium exposure (Langard 1980).

While it is known that chronic exposure to Cr-VI in the air causes lung cancer, the most striking increases of lung cancer incidence (20-40% greater than expected incidence) have been observed for workers in chromium industry operations other than ferrochrome production (Langard *et al.* 1980). This is not to diminish the health risks of ferrochrome production, which, does provide relatively high exposure to Cr-VI (Barceloux 1999). Estimated levels of exposure to chromium in air at a ferrochromium plant are shown in Table 4. According to the International Agency for Research on Cancer, epidemiological studies regarding the lung cancer risks for ferrochrome workers are inconclusive (IARC 1990). Risks for other cancers are also debated, as previously detailed.

Table 4 Estimated levels of chromium exposure in a ferrochrome plant in ug of Cr per cubic metre of air. From Axelsson *et al.* 1980.

Work site	Exposure (ug/m^3)	
	Cr-0 + Cr-III	*Cr-VI*
Arc-furnaces	2500	250
Transport, metal grinder, sampling	500-2500	10-50
Maintenance	2500	50

Table 5 lists a number of studies and their conclusions regarding ferrochrome workers and cancer. It should be noted that in studies where worker disease incidence and/or mortality are lower than the reference population, the "healthy worker effect" may be responsible. The healthy worker effect is a potential bias arising from the fact that workers must be relatively healthy to remain employed, while a general population reference group includes people in all health conditions (Li and Sung 1999).

Contaminated environment exposure risks

Industrial chromium pollution has led to a number of studies investigating health risks to exposed people in the general population, some of which have been summarised below (Table 6). While not unanimous, some evidence links environmental pollution by chromium industries to health effects such as skin irritations, kidney disease (TMGBS 1987), gastrointestinal problems (Zhang and Li 1987) and increased cancer mortality (Zhang and Li 1987; Ao and Wang 1988; Beaumont *et al.* 2008; Linos *et al.* 2011). As with most broad epidemiological studies, these studies are challenged by confounding factors and uncertainty such as actual doses of chromium, statistical power, lifestyle and other variables affecting cancer incidence, comparable reference populations and cancer latency time.

Table 5 Summary of epidemiological studies investigating ferrochrome workers and cancer.

Cancers investigated	Study Details	Observations	Risks
Pokrovskaya and Shabynina 1973: lung, esophagus, all-cancers combined	USSR ferrochromium workers from 1955-1969 exposed to Cr-III, VI and some to benzo[a]pyrene vs. municipal general population	significant increased relative risk for lung cancer in males of some age groups; significant increased relative risks for esophagus & all-cancer	increase
Axelsson *et al.* 1980: leukemia, stomach, small intestine/colon, rectum, trachea/bronchus/lung/pleura, prostate, all others	1932 male ferrochromium plant workers in Sweden employed ≥ 1 year from 1930-1975; compared to county or national cancer statistics	No significant differences between workers and general population for mortality of each cancer and all-cancers	no increase
Langard *et al.* 1980: lung and bronchial, stomach, prostate	Norwegian ferrochromium and ferrosilicon workers employed from 1928-1965, observed until 1977; compared to national and local population cancer rates	lower overall cancer mortality and all-cancer incidence; increased lung cancer incidence in workers vs. national rates and non-chromium exposed workers (both workers exposed to PAHs); increased prostate cancer incidence in ferrochrome workers vs. national rates	increase
Langard *et al.* 1990: lung, bronchial, kidney, prostate, stomach	Norwegian ferrochromium and ferrosilicon workers first employed before 1960, or before 1965; observed from 1953 to 1985	increased lung, prostate and kidney cancer incidence in ferrochromium workers	increase
Moulin *et al.* 1990: buccal cavity/pharynx/larynx, esophagus, stomach, intestine, rectum, pancreas, trachea/bronchus/lung, bladder, prostate, brain, sarcoma, lymphoma, leukemia	Study of ferrochrome and stainless steel workers in France with exposures to nickel, Cr-III, Cr-VI and for some, PAHs as well; compared to national average	lower overall mortality, significantly lower benign respiratory disease mortality vs. national mortality; non-significant excesses of lung, stomach, prostate and brain cancer vs. national data; significantly increased lung cancer deaths in ferrochromium workers vs. other workers, may be related to PAH exposure; lung cancer deaths occurred in workers who began work before 1964, possibly evidence of improved working conditions	increase
Moulin *et al.* 1992: lung	workers employed ≥3 yrs. between 1968-84 in ferroalloy & stainless steel plant (mostly ferrochrome)	no significant increase in lung cancer mortality in ferroalloy workers vs. national mortality	no increase
Gatto *et al.* 2010: gastrointestinal tract: oral, esophageal, stomach, rectum and colon	meta-analysis of 32 studies from 1950-2009 investigating workers exposed to Cr-VI, including ferrochromium workers	no statistically greater risk of gastrointestinal cancers in workers	no increase
Wang *et al.* 2011: Estimated cancer risk based on exposure	risk analysis for lifetime ferrochrome plant employee in China considering inhalation, skin absorption and contaminated soil ingestion	Calculated cancer risks of $3.3x10^{-6}$ to $1.7x10^{-3}$ are greater than acceptable risk ($1x10^{-6}$)	Increased risk

Table 6 Some epidemiological studies of human general populations exposed to chromium through environmental contamination by industry.

Study details	Contamination Levels	Observations
Axelsson and Rylander 1980: comparison of lung cancer mortality rates between communities in Sweden with ferrochrome plants and communities without, county and national rates	Max. atmospheric chromium 0.1 – 0.4 ug/m^3, 50-100x greater than unexposed areas	no relation between lung cancer and community exposure to ferrochrome exhaust
Tokyo Metropolitan Government Bureau of Sanitation 1987: study of housewives exposed to chromium contaminated slag from a chemical plant that was used as urban fill in Tokyo, Japan	not available	significant increases in eczema, allergic contact dermatitis in summer; increased headaches, heaviness of head, chronic fatigue, dizziness, diarrhea, constipation, blood and high protein in urine in exposed population
Zhang and Li 1987: cancer mortality study in population exposed to drinking water with Cr-VI from a ferrochrome plant near JinZhou City, China, 1960-1978	up to 20 000 ug/L drinking water	mouthsores, diarrhea, stomach pains, indigestion, vomiting, higher levels of white blood cells, increased stomach, lung and all-cancer mortality in exposed group
Ao and Wang 1988: study of agricultural village exposed to drinking water contaminated with Cr-VI from a ferrochrome plant near JinZhou, China, 1970s-1980s	up to 20 000 ug/L drinking water	increased cancer mortality rate in exposed group
Taioli *et al.* 1995: study of New Jersey residents at risk for exposure to Cr-VI contaminated slag & with elevated urinary Cr	Unknown; Slag from Cr-VI compound manufacture was dumped & used for fill & construction from 1905-1975	significantly elevated DNA-protein crosslinks in exposed group, a sign of DNA damage; No other signs of chromium toxicity identified
Beaumont *et al.* 2008: reanalysis of unpublished Dr Zhang's JinZhou City, China cancer mortality data	up to 20 000 ug/L drinking water	increased stomach and lung cancer mortality rates in exposed group
ATSDR 2008b: analysis of lung cancer occurrence in residents living near sites where chromate (Cr-VI) manufacturing slag was dumped in New Jersey	Variable, unknown: slag with Cr-VI used for fill & construction from 1905-1975	Higher number of lung cancer cases in groups closer to historical slag contamination
Kerger *et al.* 2009: reanalysis of unpublished Dr Zhang's JinZhou City, China cancer mortality data	up to 20 000 ug/L drinking water	lung, stomach and all-cancer mortality rates not significantly different between exposed and unexposed groups
Linos *et al.* 2011: Study of cancer mortality of individuals exposed to Cr-VI in drinking water for approx. 20 years in Greece	41 – 146 ug/L drinking water	Significant increase mortality from: liver, lung, female kidney/genital/urinary cancer; Increased mortality for lip/oral cavity/pharynx, stomach, female colon, female breast, prostate cancers, leukemia, and all-cancers compared to nearby reference population

Chromium Toxicity: Regulations and Guidelines

Environmental and occupational exposure to Cr-VI is considered a "major human health issue" (Nickens *et al.* 2010). Due to the complex chemical and toxic behaviours of chromium, regulators must contend with a large amount of uncertainty when setting chromium limits (Bartlett and Veslind 1998). Standards and guidelines from various countries for chromium in water, sediment, soil, air and industrial waste are outlined in Appendix 2 and include those previously referenced in this paper.

The need for greater understanding of chromium toxicity continues to spur research and in turn, new regulations. For example, in 2006 the US Occupational Safety and Health Administration (OSHA) revised its permissible exposure limit for Cr-VI in air from 52 ug/m^3 to 5 ug/m^3 due to recent research (Nickens *et al*. 2010). The Ontario Ministry of Environment updated their original (1996) Site Standards for soil and groundwater in 2009. While Ontario's allowable amounts of Cr-VI in non-potable groundwater and total Cr in subsurface soil were raised, the allowable amounts were lowered substantially for total chromium in soil (decreased from 1000-750 to 160 mg/kg), Cr-VI in subsurface soil (from 600-11000 to 40 mg/kg), total chromium in non-potable groundwater (from 2000 to 810 mg/kg) and Cr-VI in potable groundwater (from 50 to 25 mg/kg). Standards were also created for soil and groundwater within 30m of a surface water body (MOE 2004; MOE 2011c).

Canada and the USA do not currently have drinking water standards specifically for Cr-VI, but the EPA is in the process of evaluating Cr-VI oral exposure carcinogenicity. California has recently set a public health goal (PHG) of 0.02 ug/L for Cr-VI in drinking water. This is a decrease from the initially proposed PHG of 0.06, in order to include protection of more sensitive subgroups of the population (fetuses, newborns, people with low stomach acidity). The PHG will play a role in determining California's enforceable maximum contaminant level for Cr-VI, which has yet to be set (EWG 2010). Canada and California's maximum contaminant levels for total chromium in drinking water (50 ug/L) are half of the US EPA standards (100 ug/L).

Table 7 summarises the US EPA's human health reference values for chromium non-carcinogenic risks and the EPA's level of confidence in these values. Reference values are estimated continuous daily exposures considered safe for humans over a lifetime. Confidence in these values is based on the quality of the study upon which the value is based, and the number and quality of supporting studies. The low confidence the EPA has in their reference values exemplifies the need for further research.

Table 7 Oral Reference Dose (Rfd) and Inhalation Reference Concentration (RfC) for human exposure to hexavalent chromium for non-carcinogenic risks.
US EPA 1998.

	Reference value	Confidence
Rfd: chronic oral exposure to soluble salts of Cr-VI	3 ug/kg body mass per day	low
RfC: chronic inhalation of Cr-VI particulates	0.1 ug/m^3 of air per day	medium
RfC: chronic inhalation of chromic acid mists and dissolved Cr -VI aerosols	0.008 ug/$m^3$of air-day	low

Conclusion

There is a well established body of literature documenting the hazards of Cr-VI toxicity while the potential hazards of Cr-III remain less clear. This research has provided the understanding that chromium can be linked to a wide variety of toxic effects on animal and human health. Given this initiatives to establish chromite mining and ferrochrome processing in Canada need to be carefully reviewed for environmental and human health risks. Increased understanding of chromium toxicity has led some governments to establish more stringent regulations for Cr-VI.

For all the efforts to understand the nature of chromium toxicity, many questions are still unanswered. Some of these questions include:

1. Is there a threshold below which the body can detoxify ingested Cr-VI?
2. Does Cr-VI ingestion cause cancer in humans and if so, what cancers and at what doses?
3. Does Cr-VI inhalation cause any cancers other than lung and sinonasal and if so at what doses?
4. Does Cr-VI inhalation cause gastrointestinal, kidney and liver damage and if so, at what doses?
5. Do any Cr-III compounds cause cancer and if so, which and at what doses?
6. What are the safe exposure levels to CrVI that do not cause allergic contact dermatitis and skin ulcers?
7. What are the reproductive and developmental effects of Cr-VI and Cr-III on humans and at what doses?
8. Can chromium toxicity predictions for plants and animals be made which take into account the complex influences of different soil types, water characteristics, plant and animal species?
9. Which is more toxic to plants, Cr-III or Cr-VI?
10. What forms of Cr are present in plants after uptake and what risks might these pose to plant consumers?
11. Since chromium exposure can change the nutrient uptake of plants, what are the potential health effects of this for plant consumers?
12. Have standard tests been underestimating Cr-III toxicity to algae?
13. Does chromium biomagnify up aquatic and terrestrial food chains?

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Glossary and Acronyms[3]

ACD: allergic contact dermatitis; a skin condition

Acute exposure: exposure for a short time period, and usually at high concentrations

ATSDR: Agency for Toxic Substances and Disease Registry

Bioaccumulation: the total accumulation of a contaminant in a living organism due to contaminant uptake without equal removal

Bioavailable: the extent to which a contaminant is free for uptake by an organism

Bioconcentration: net accumulation in and on an organism of a contaminant from water only

Bioconcentration Factor (BCF): the ratio of concentrations of contaminant in the organism and dissolved in water (organism / water)

Biomagnification: an increase in concetration from one food chain level to the next due to accumulation of the contaminant from food (eg prey to predator increase)

Carcinogenic: capable of causing cancer

CCME: Canadian Council of Ministers of the Environment

Chronic Exposure: exposure over a long time period

Cr: the element chromium

EPA: Environmental Protection Agency

Epidemiology: the science concerned with the cause, incidence, prevalence and distribution of infections and non-infectious diseases in populations

FeCr: ferrochromium or ferrochrome; an alloy of iron and chromium

Genotoxic: capable of causing damage to genetic materials i.e. chromosomes or DNA

IARC: International Agency for Research on Cancer

Leaching: the dissolving of materials from a solid into a liquid

Mutagenic: capable of causing mutations

NIOSH: National Institute for Occupational Safety and Health

OSHA: Occupational Safety and Health Administration

Oxidation: A reaction in which the atoms in an element lose electrons and the valence of the element is correspondingly increased

[3] Definitions based on Newman MC and Unger MA. 2002. Fundamentals of Ecotoxicology. Lewis Publishers, Florida. 458p.

Reduction: A reaction in which the atoms in an element gain electrons and the valence of the element is correspondingly decreased

ng: nanogram, 1000 nanograms = 1 microgram = 0.001 milligram

ppb: parts per billion, ug/L or ug/kg

ppm: parts per million, mg/L or mg/kg

total chromium: chromium from all valence states measured together

ug: microgram, 1000 micrograms = 1 milligram

WHO: World Health Organization

Appendix 1– Additional Resources

Ferrochrome Production Processes
See the International Chromium Development Association's Ore Processing Description at:
http://www.icdacr.com/index.php?option=com_content&VIew=article&id=136&Itemid=341&lang=en

Ferrochrome Dust[4] [5]
Sources of ferrochrome dust are a) slag and metal expulsion from the furnace electrode holes, b) fine particle mobility and c) in-furnace vaporisation and subsequent precipitation in the off-gas duct. Coarse dust particles are collected by equipment called the cyclone separator and fine particles by the baghouse filter system. These dusts pose a threat to groundwater unless treated before disposal. Three types of treatment have generally been used for electric arc furnace dust:

1. Direct recycling processes wherein dust is returned to the furnace. This may increase the amount of energy required by the furnace.
2. Pyrometallurgical and hydrometallurgical recovery processes: Pyrometallurgical methods require high investment costs and recover chromium, nickel, iron, zinc, lead and cadmium from the dust. Hydrometallurgical methods recover zinc, but are also economically challenging and may not result in dust within toxicity limits.
3. Stabilization/solidification processes, which are considered effective and economical.

[4] Ma G and Garbers-Craig AM. 2006a. Cr(VI) containing electric furnace dusts and filter cake from a stainless steel waste treatment plant Part 1 - Characteristics and microstructure. Ironmaking and Steelmaking. 33(3), 229-237.
[5] Ma G and Garbers-Craig AM. 2006b. Cr(VI) containing electric furnace dusts and filter cake from a stainless steel waste treatment plant: Part 2 – Formation mechanisms and leachability. Ironmaking and Steelmaking. 33(3), 238-244.

Appendix 2 - Chromium Regulations and Guidelines

Total chromium refers to all forms of chromium present and does not differentiate between Cr-III and Cr-VI.

Table A- 1 Standards and guidelines for chromium in environmental and agricultural waters.

Limit (ug/L)	Cr	Source	Details
Freshwater			
8.9	III	Canada[6] & Ontario[7]	Interim Water Quality Guidelines for Protection of Aquatic Life
74	III	USA[8]	chronic exposure Water Quality Criteria for Protection of Aquatic Life
570	III	USA[8]	acute exposure Water Quality Criteria for Protection of Aquatic Life
1	VI	Canada6 & Ontario7	Water Quality Guideline for Protection of Aquatic Life
11	VI	USA[8]	chronic exposure Water Quality Criteria for Protection of Aquatic Life
16	VI	USA[8]	acute exposure Water Quality Criteria for Protection of Aquatic Life
50	III	Canada[6]	livestock water Interim Quality Criteria
50	VI	Canada[6]	livestock water Interim Quality Criteria
4.9	III	Canada[6]	irrigation water Interim Quality Criteria
8	VI	Canada[6]	irrigation Water Quality Criteria
1	total	Netherlands[9]	shallow groundwater Target Value
2.5	total	Netherlands[9]	deep groundwater Target Value
30	total	Netherlands[9]	groundwater Intervention Value (requires remediation)
810	total	Ontario[10]	non-potable groundwater standard
140	VI	Ontario[10]	non-potable groundwater standard
640	total	Ontario[10]	non-potable groundwater standard in shallow soil site or within 30m of water body
110	VI	Ontario[10]	non-potable groundwater standard in shallow soil site or within 30m of water body
Marine			
56	III	Canada[6]	Interim Water Quality Guidelines for Protection of Aquatic Life
1.5	VI	Canada[12]	Water Quality Guidelines for Protection of Aquatic Life
1100	VI	USA[8]	acute exposure Water Quality Criteria for Protection of Aquatic Life
50	VI	USA[8]	chronic exposure Water Quality Criteria for Protection of Aquatic Life

[6] Canadian Council of Ministers of the Environment. 1999. http://ceqg-rcqe.ccme.ca/ The Canadian Environmental Quality Guidelines are national, scientifically derived guidelines for parameter levels that should not result in risk to "biota, their functions, or any interactions that are integral to sustaining health of ecosystems." They do not integrate site-specific conditions, but are useful tools for broadly assessing risk.

[7] Ontario Ministry of the Environment. 1994. Water Quality Objectives http://www.ene.gov.on.ca/stdprodconsume/groups/lr/@ene/@resources/documents/resource/std01_079681.pdf

[8] US EPA http://water.epa.gov/scitech/swguidance/standards/current/index.cfm#D

[9] Dutch Intervention Values. 2009. http://www.esdat.com.au/Environmental%20Standards/Dutch/ENGELSE%20versie%20circulaire%20Bodemsanering%202009.pdf

[10] Ontario Ministry of the Environment. 2011. Generic Site Condition Standards for Contaminated Sites. http://www.ene.gov.on.ca/stdprodconsume/groups/lr/@ene/@resources/documents/resource/stdprod_086518.pdf

Table A- 2 Standards and guidelines for chromium (Cr) in drinking water.

Limit (ug/L)	Cr	Source	Details
50	total	World Health Organization[11]	Provisional Guideline
50	total	Canada[12]	Maximum Acceptable Concentration
50	total	Ontario[13]	Quality Standard
50	total	Ontario[10]	potable groundwater standard
25	VI	Ontario[10]	potable groundwater standard
50	total	California[14]	Maximum Contaminant Level
100	total	USA[15]	Maximum Contaminant Level
in progress	VI	USA[15]	Maximum Contaminant Level
0.02	VI	California[14]	Public Health Goal; Maximum Contaminant Level in progress

Table A- 3 Standards and guidelines for chromium (Cr) in sediment, mg/kg dry weight.

Limit (mg/kg dw)	Cr	Source	Details
Freshwater			
37.3	total	Canada[6]	Interim Quality Guideline
90	total	Canada[6]	Probable Effects Level
26	total	Ontario[10]	Site Condition Standard
Marine			
52.3	total	Canada[6]	Interim Quality Guideline
160	total	Canada[6]	Probable Effects Level

[11] World Health Organization. 2011. http://www.who.int/water_sanitation_health/publications/2011/9789241548151_ch12.pdf

[12] Guideline for Canadian Drinking Water Quality. 2010. http://www.hc-sc.gc.ca/ewh-semt/pubs/water-eau/2010-sum_guide-res_recom/index-eng.php#a3

[13] Ontario Ministry of Environment. 2011. http://www.e-laws.gov.on.ca/html/regs/english/elaws_regs_030169_e.htm

[14] California. 2011. http://www.cdph.ca.gov/certlic/drinkingwater/Documents/Chromium6/FAQs-chromium6-07-27-2011.pdf

[15] US EPA. http://water.epa.gov/drink/contaminants/basicinformation/chromium.cfm

Table A- 4 Standards and guidelines for chromium (Cr) in soil.

Limit (mg/kg)	Cr	Source	Details
64	total	Canada[6]	Guideline for protection of environmental and human health for agricultural, residential/parklands
0.4	VI	Canada[6]	Provisional Guideline for protection of environmental health for agricultural, residential/parklands
87	total	Canada[6]	Guideline for the protection of environmental and human health for industrial and commercial lands
1.4	VI	Canada[6]	Provisional Guideline for the protection of environmental health for industrial and commercial lands
160	total	Ontario[10]	All land uses, Soil Standard
10	VI	Ontario[10]	All land uses, medium to fine textured Soil Standard
8	VI	Ontario[10]	All land uses, coarse Soil Standard
18 000	total	Ontario[10]	All land uses, subsurface (>1.5m deep) Soil Standard
11 000	total	Ontario[10]	All land uses, subsurface (>1.5m deep) medium to fine textured Soil Standard
40	VI	Ontario[10]	All land uses, subsurface (>1.5m deep) coarse Soil Standard
70	total	Ontario[10]	Residential/Parkland/Institutional/Industrial/Commercial/Community property use Soil Standard within 30m of water body
67	total	Ontario[10]	Agricultural or Other property use Soil Standard within 30m of water body, potable groundwater condition
0.66	VI	Ontario[10]	All property use Soil Standard within 30m of water body, potable groundwater condition
390	total	USA[16]	Soil Screening Level for non-carcinogenic systemic toxicity (i.e. liver and kidney damage)
78,000	III	USA[16]	Soil Screening Level for non-carcinogenic systemic toxicity (i.e. liver and kidney damage)
390	VI	USA[16]	Soil Screening Level for non-carcinogenic systemic toxicity (i.e. liver and kidney damage)
270	VI	USA[16]	Soil Screening Level in residential soils; protective of lung cancer
75	total	Switzerland[17]	Maximum Allowable Value
100	total	Hungary[17]	Maximum Allowable Value
<150	total	China[18]	Environmental Quality Standard for Soils pH <6.5
100	total	Netherlands[17]	Target Value
75	total	Berlin[17]	Soil Reuse Value
2.5	VI	Berlin[17]	Soil Reuse Value
600	total	UK[17]	Maximum Allowable Value in gardens
25	VI	UK[17]	Maximum Allowable Value in gardens
1000	total	UK[17]	Maximum Allowable Value parks
25	VI	UK[17]	Maximum Allowable Value parks

[16] US EPA http://www.epa.gov/superfund/health/conmedia/soil/pdfs/appd_a.pdf
[17] Prokisch J. 1997. Journal of Chromatography A. 774, 363–371.
[18] Liu J. et al. 2011. Journal of Hazardous Materials. 188, 85-91.

Table A- 5 Environmental and occupational standards and guidelines for chromium in air.

PM_{10} is particulate matter <10um in diameter. TSP is total suspended particulate size fraction. MOL is Ministry of Labour. NIOSH is the National Institute for Occupational Safety and Health. OSHA is the Occupational Safety and Health Administration. *values are measured as CrO_3; all other values are for Cr alone, not Cr compounds.

Limits (ug/m^3)	Cr	Source	Details
Environmental			
none	-	Canada[6]	Air Quality Guidelines for the protection of human health and environment
1.5	II, III	Ontario[19]	24-hr average Ambient Air Quality Criterion set in 1982
1.5	0, II, III	Ontario[19]	0.5-hr average Standard based on Cr-III respiratory effects, effective 2016
0.5	0, II, III	Ontario[19]	24-hour average Air Standard and Ambient Air Quality Criterion based on Cr-III respiratory effects, effective 2016
0.00035	VI, PM_{10}	Ontario[19]	24-hr average Ambient Air Quality Criterion, based on Cr-VI carcinogenicity, effective 2016
0.00007	VI, PM_{10}	Ontario[19]	annual Ambient Air Quality Criterion, based on Cr-VI carcinogenicity, effective 2016
0.002	VI, TSP	Ontario[19]	0.5-hr average Air Standard for Cr(VI), based on Cr-VI carcinogenicity, effective 2016
0.0007	VI, TSP	Ontario[19]	24-hr Ambient Air Quality Criterion, based on Cr-VI carcinogenicity, effective 2016
0.00014	VI, TSP	Ontario[19]	annual Air Standard and Ambient Air Quality Criterion, based on Cr(VI) carcinogenicity, effective 2016
Occupational			
500	0, III	Ontario MOL[20]	8-hr average Occupational Exposure Limit
50	VI	Ontario MOL[20]	8-hr average Occupational Exposure Limit to chromate from chromite ore processing
50	VI, soluble	Ontario MOL[20]	8-hr average Occupational Exposure Limit
10	VI, insoluble	Ontario MOL[20]	8-hr average Occupational Exposure Limit
500	0, II, III	USA NIOSH[21]	8-hr average Recommended Exposure Limit
1	VI	USA NIOSH[21]	10-hr average Recommended Exposure Limit
1000	0, insoluble	USA OSHA[21]	8-hr average Permissible Exposure Limit
500	II, III	USA OSHA[21]	8-hr average Permissible Exposure Limit
*5	VI, soluble	USA OSHA[21]	8-hr average Permissible Exposure Limit

[19] Ontario Ministry of the Environment. 2011. http://www.downloads.ene.gov.on.ca/envision/env_reg/er/documents/2011/010-6353.pdf
[20] Ontario Ministry of Labour. 2010. http://www.labour.gov.on.ca/english/hs/pubs/oel_table.php
[21] Centers for Disease Control and Prevention. http://www.cdc.gov/niosh/npg/nengapdxc.html

Table A- 6 Standards and guidelines for chromium in effluent and waste leachate.

Limit (ug/L)	Cr	Source	Details
Effluent Discharges			
no regulation	-	Canada[22]	Metal Mine Effluent Regulations
10	total	Central Pollution Control Board, New Delhi, India [23]	Maximum Permissible Limit
Waste Leachate			
5000	total	Canada[24]	Hazardous Waste Transport Regulation
5000	total	Ontario[25]	Hazardous Waste Quality Criteria
5000	total	USA[26]	Maximum Acceptable Level
2000	total	Italy[26]	Maximum Acceptable Level
200	VI	Italy[26]	Maximum Acceptable Level
500	VI	Spain[26]	Maximum Acceptable Level
100	VI	Germany[26]	Maximum Acceptable Level, Class II
50	VI	Germany[26]	Maximum Acceptable Level, Class I
1500	VI	Japan[26]	Maximum Acceptable Level
20	VI	South Africa[26]	Estimated Environmental Concentration Limit
4700	III	South Africa[26]	Estimated Environmental Concentration Limit
4000	III	Spain[26]	Maximum Acceptable Level

[22] Department of Justice. MMER. http://laws-lois.justice.gc.ca/eng/regulations/SOR-2002-222/page-16.html#h-51
[23] Dwivedi S. et al. 2010. Journal of Hazardous Materials. 173, 95-101.
[24] Environment Canada. 2005. http://laws-lois.justice.gc.ca/eng/regulations/SOR-2005-149/page-19.html#h-34
[25] Ontario Environmental Protection Act. Schedule 4. http://www.e-laws.gov.on.ca/html/regs/english/elaws_regs_900347_e.htm#BK31 Accessed Feb 3, 2012.
[26] Ma G and Garbers-Craig AM. 2006. Ironmaking and Steelmaking. 33(3), 238-244.

Appendix 3 - Chromium Ingestion, Carcinogenicity and Controversy

In 1987 Zhang and Li published a paper documenting the cancer rates for villagers exposed to Cr-VI contamination in drinking water from a ferrochromium plant near JinZhou, China. Chromium levels were approximately 20 000 ug/L in the drinking water, which had turned yellow from the pollution. The 1987 study stated there was a higher per capita rate of cancers, including lung cancer and stomach cancer, in those exposed to chromium compared to a reference population.[27] [28] However in 1997, a second paper published under Zhang and Li (a different co-author with the surname Li) presented a reanalysis of the data and concluded "results do not indicate an association of cancer mortality with [chromium] exposure."[29] [30]

It later came to light that consultants of ChemRisk Inc., hired by the Pacific Gas and Electric (PG&E) utility company, were involved in this second paper. Around the same time, PG&E was embroiled in a lawsuit in California for contaminating drinking water with hexavalent chromium. The suit was eventually settled out of court, with PG&E paying 333 million in damages.[31]

An investigation begun by California's Agency's Office of Environmental Health Hazard Assessment uncovered a number of ethical and scientific issues with the 1997 study. It is alleged that ChemRisk wrote and submitted the 1997 paper for publication without Dr. Zhang's full approval and for the benefit of their industrial clients. A detailed article discussing the allegations can be found on the EWG website.[31] In 2006, the second Zhang and Li paper (1997) was retracted by the publishing journal, due to the authors' failure to disclose all financial and intellectual input.[32]

ChemRisk researchers maintain that misconduct allegations against them are false and that Dr. Zhang had full authority over the paper's contents.[33] By the time of the controversy, Dr. Zhang was deceased. Six months after the paper retraction, his co-author Dr. Li, contested the accuracy of the Wall Street Journal exposé articles and the journal retraction.[34]

Since then, several re-analyses of the data from these populations have put forth contradictory conclusions, either supporting or refuting chromium links to cancer.[35] [36] Both Zhang and Li papers noted that no dose-response relationship was observed in the population. This has been interpreted by some as evidence against a link between

[27] Zhang JD and Li XL. 1987. Chromium pollution of soil and water in JinZhou. Journal of Chinese Preventative Medicine. 21, 262-264. Cited in US EPA 1998.
[28] US EPA: United States Environmental Protection Agency. 1998. Integrated Risk Information System: Chromium (VI) (CASRN 18540-29-9) Available at: http://www.epa.gov/iris/subst/0144.htm Accessed on: December 18, 2011.
[29] Zhang JD. and Li SK. 1997. Cancer mortality in a Chinese population exposed to hexavalent Chromium in water. Journal of Occupational and Environmental Medicine. 39, 315-320. (retracted).
[30] Smith AH. 2009. Author's Response to On the Chromium Reanalysis. Letters to the Editor. Epidemiology. 20(4), 626. Available at: http://journals.lww.com/epidem/toc/2009/07000#-428312208 Accessed on: January 17, 2012.
[31] Environmental Working Group. December 2005. How PG&E's Scientists-For-Hire Reversed Findings of Cancer Study. Available at: http://www.ewg.org/erinbrockovichchromium6lawsuit/overview Accessed on: December 18, 2012.
[32] Brandt-Rauf P. 2006. Editorial Retraction. Journal of Occupational and Environmental Medicine. 48(7), 749. Available at: http://www.defendingscience.org/newsroom/upload/JOEM_Retraction.pdf
[33] Paustenbach D. 2009. On the Chromium Reanalysis. Letters to the Editor. Epidemiology. 20(4), 625-626. Available at: http://journals.lww.com/epidem/toc/2009/07000#-428312208 Accessed on: January 17, 2012.
[34] Phillips ML. December 22 2006. Chromium paper retracted unfairly, author says. The Scientist: Magazine of Life Sciences. Available at: http://classic.the-scientist.com/news/display/38457/
[35] Beaumont JJ, Sedman RM, Reynolds SD, Sherman CD, Li L-H, Howd RA, Sandy MS, Zeise L, Alexeef GV. 2008. Cancer Mortality in a Chinese population Eexposed to hexavalent chromium in drinking water. Epidemiology. 19(1), 12-23.
[36] Kerger BD, Butler WJ, Paustenbach DJ, Zhang JD and Li SK. 2009. Cancer mortality in Chinese populations surrounding an alloy plant with chromium smelting. Journal of Toxicology and Environmental Health, Part A. 72, 329–344.

chromium and the excess cancers, while other researchers have not interpreted the data this way, and both sides have levelled criticisms of analysis methods used by dissenting parties.[37] [38]

ChemRisk scientists involved in the 1997 paper include William Butler and Tony Ye, who went on to form their own company, Environmental Risk Analysis Inc., Dennis Paustenbach, founder and president of ChemRisk and Brent D Kerger.[31]

Proponents for the other side of the debate include California Environmental Protection Agency's Office of Environmental Health Hazard Assessment scientists Jay Beaumont and Richard Sedman, as well as Allan H. Smith of the School of Public Health, University of California at Berkeley.

[37] Kerger BD, Butler WJ, Ye T, Li S. 2009. Chromium (VI) Ingestion and Cancer. Letters to the Editor. Epidemiology. 20(4), 627. Available at: http://journals.lww.com/epidem/toc/2009/07000#-428312208 Accessed on: January 17, 2012.

[38] Beaumont JJ, Sedman RM, Sandy MS, Zeise L, Reynolds SD. 2009. Author's Response to Chromium (VI) Ingestion and Cancer. Letters to the Editor. Epidemiology. 20(4), 628. Available at: http://journals.lww.com/epidem/toc/2009/07000#-428312208 Accessed on: January 17, 2012.