



Canadian Environmental Protection Act

Priority Substances List
Assessment Report

Chromium and its Compounds



Government
of Canada

Gouvernement
du Canada

Environment
Canada

Environnement
Canada

Health
Canada

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Canada



PRIORITY SUBSTANCES LIST
ASSESSMENT REPORT

CHROMIUM AND ITS COMPOUNDS

Government of Canada
Environment Canada
Health Canada

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Notice to Readers

Pesticidal uses of chromium were not assessed in this report. The pesticide chromic acid is subject to the provisions of the *Pest Control Products Act*, and its regulatory status as a heavy-duty wood preservative is currently being re-evaluated (see Announcement A92-02: "Re-evaluation of Heavy Duty Wood Preservatives", Agriculture Canada, Food Production and Inspection Branch, July 2, 1992). As part of the pesticide re-evaluation process, Environment Canada and Health Canada will assess the potential effects on the environment and on human health resulting from these pesticidal uses of chromium.

Synopsis

Chromium is a naturally occurring metal that is present principally in the trivalent or hexavalent forms in small amounts throughout the Canadian environment. Chromium ore is not currently produced in Canada; however, approximately 74 000 tonnes (gross weight) of various chromium-containing materials were imported into Canada in 1991. Chromium is used in a wide variety of industrial applications in Canada including the production of stainless and heat-resistant steels, refractory products such as bricks and mortars, and in pigments, metal finishing, leather tanning, and wood preservatives. Both trivalent and hexavalent forms of chromium are released into the environment in Canada as a result of these industrial uses, as well as from the production and combustion of fossil fuels, and the smelting and refining of nonferrous base metals.

Typical (median or mean) concentrations of dissolved hexavalent chromium in Lake Ontario and Lake Brie, as well as in rivers and streams in parts of British Columbia, Alberta, Ontario, and Quebec are likely 5 to 15 times greater than the effects threshold estimated for the most sensitive aquatic species identified. Furthermore, average concentrations of chromium in soils contaminated with dissolved hexavalent chromium at several Canadian wood preservation facilities are 19 to 1700 times greater than levels of hexavalent chromium reported to harm some plants and microbial communities. Although trivalent chromium is likely the dominant chromium species in most sediments, soils, and biological tissues, there was insufficient information to determine whether organisms are adversely affected by exposure to trivalent chromium in Canada.

Chromium (in trivalent and hexavalent forms) occurs at low concentrations, primarily in particulate form in the atmosphere, and consequently it is unlikely to have a marked influence on the earth's solar radiation balance. Trivalent chromium is not oxidized by ozone and therefore cannot be linked to any ozone-depleting reactions in the atmosphere. As such, chromium is not expected to contribute significantly to global warming or depletion of stratospheric ozone.

Based on an estimation of the average total daily intake of chromium (total) from air, drinking water, food, and soil for various age groups of the general population, food is likely the most significant source of human exposure in Canada. Based on the weight of evidence of carcinogenicity in occupationally exposed populations, the group of hexavalent chromium compounds as a whole (since available data do not permit an assessment of individual compounds within the group) is classified as "Carcinogenic to Humans", i.e., as substances for which there is believed to be some chance of adverse health effects at any level of exposure. For such substances, estimated exposure is compared to quantitative estimates of carcinogenic potency to characterize risk and provide guidance for further action, i.e., analysis of options to reduce exposure. For hexavalent chromium, such a comparison suggests that the priority for analysis of options to reduce exposure would be moderate to high. In addition, hexavalent chromium may induce skin sensitivity in a small portion of the general population.

Trivalent chromium is considered to be an essential element in human nutrition. Estimated average daily intake of total chromium does not exceed the recommended

daily intake for trivalent chromium. Levels of trivalent chromium that are demonstrated to cause adverse effects in limited studies in experimental animals greatly exceed typical concentrations of total chromium in relevant environmental media in Canada.

Based on these considerations, it has been concluded that dissolved and soluble forms of hexavalent chromium are entering or may enter the environment in a quantity or concentration or under conditions that are having or may have a harmful effect on the environment. There are insufficient data to determine if trivalent forms of chromium are entering or may enter the environment in a quantity or concentration or under conditions that are having or may have a harmful effect on the environment. It has been concluded that neither hexavalent nor trivalent forms of chromium are entering or may enter the environment in a quantity or concentration or under conditions that constitute or that may constitute a danger to the environment on which human life depends. It has also been concluded that the group of hexavalent chromium compounds as a whole is entering the environment in a quantity or concentration or under conditions that may constitute a danger in Canada to human life or health, while the group of trivalent chromium compounds as a whole is not entering the environment in a quantity or concentration or under conditions that may constitute a danger in Canada to human life or health.

1.0 Introduction

The *Canadian Environmental Protection Act* (CEPA) requires the Minister of the Environment and the Minister of Health to prepare and publish a Priority Substances List that identifies substances, including chemicals, groups of chemicals, effluents, and wastes that may be harmful to the environment or constitute a danger to human health. The Act also requires both Ministers to assess these substances and determine whether they are "toxic" as defined under Section 11 of the Act which states:

"...a substance is toxic if it is entering or may enter the environment in a quantity or concentration or under conditions

- (a) having or that may have an immediate or long-term harmful effect on the environment;
- (b) constituting or that may constitute a danger to the environment on which human life depends; or
- (c) constituting or that may constitute a danger in Canada to human life or health."

Substances that are assessed as "toxic" as defined under Section 11 may be placed on the List of Toxic Substances (Schedule I of CEPA). Consideration can then be given to developing regulations, guidelines, or codes of practice to control any aspect of these substances' life cycle, from the research and development stage through manufacture, use, storage, transport, and ultimate disposal.

The assessment of whether chromium and its compounds are "toxic", as defined under Section 11 of CEPA, was based on the determination of whether they **enter** or are likely to enter the Canadian environment in a concentration or quantities or under conditions that could lead to **exposure** of humans or other biota at levels that could cause adverse **effects**.

Data relevant to the assessment of entry, environmental exposure, and environmental effects were identified from published reviews on chromium including those of the International Programme on Chemical Safety (IPCS, 1988), U.S. EPA (1984; 1988; 1990), the Electric Power Research Institute (EPRI, 1986; 1988), Bencko (1985), Jaworski (1985), Phillips (1988), and Nriagu and Nieboer (1988). Extensive reviews of background data prepared under contract by Dr. Peter Outridge, then of the University of Toronto (chromium in wildlife), Ms. Hayla Evans of Trent University (aquatic toxicology of chromium), Dr. L. Evans and Dr. M. Goss of the University of Guelph (chemistry of chromium in soils), and Dr. Pat Doyle, then under contract with the Department of Energy, Mines and Resources (distribution of chromium in Canadian soils) were also consulted in preparing this report. Finally, a number of scientific data bases were searched for information published between 1988 and 1992, including Science Citation Index, Medline, Medlars II, TOXLINE, ENVIROLINE, Aquatic Sciences and Fisheries Abstracts (ASFA), Chemical Evaluation Search and Retrieval

System (CESARS), CHEMICAL ABSTRACTS, CURRENT CONTENTS, BIOLOGICAL ABSTRACTS, POLLUTION ABSTRACTS, and Environmental Periodical Bibliography. Only information identified before the end of December 1992 is included in this report.

For assessment of data other than those considered to be critical to the assessment of whether chromium and its compounds are considered to be "toxic" to human health under the Act, evaluations of other agencies, such as the International Agency for Research on Cancer (IARC, 1990), the Agency for Toxic Substances and Disease Registry (ATSDR, 1991), the International Programme on Chemical Safety (IPCS, 1988), the Health and Safety Executive of the U.K. (Fairhurst and Minty, 1989), and the National Institute of Public Health and Environmental Protection of the Netherlands (Sloof *et al.*, 1990) have been consulted where appropriate. An extensive background review of available data on the effects of chromium on human health, completed under contract by BIBRA Toxicology International (1992), was also consulted. In addition, a review of available toxicological literature and a summary of data on levels of chromium in the environment were prepared by Hilcon Consultants and B.A.R. Environmental, respectively. Relevant data were also requested from the Mining Association of Canada, the Chemical Manufacturers' Association, Dominion Colour Corporation, Ciba-Geigy Canada Ltd., M & T Chemicals Ltd., Proctor and Gamble Inc., the Industrial Health Foundation (U.S.), as well as officials of several provincial and federal agencies.

To identify additional literature relevant to the assessment of the effects of chromium on human health that was not included in the reviews consulted, computer literature searches were conducted on various data bases. In July 1990, a search was conducted on Canadian Research Index (MICROLOG), Scientific and Technical Information (CISTIMON), Cooperative Documents Project (CODOC), UNIVRES, and Environment Canada Departmental Library Catalogue (ELIAS) to identify information on levels of chromium in the Canadian environment published from 1974 to 1989. A search was conducted on Medline in December 1990 to identify information on the toxicity of chromium published in 1989 and 1990. The data bases SQUAREF, ELIAS, CODOC, CISTIMON, MICROLOG, TOXLINE, and Embase were searched in March 1992 to identify relevant literature published from 1990 to 1992. Computer literature searches were also conducted biweekly on the MEDLINE and TOXLINE data bases to identify any references not included in the previous searches. Data identified after the period of peer review (i.e., April 1993) were not considered for inclusion.

Although review articles were consulted where appropriate, studies that form the basis for determining whether chromium is "toxic" under CEPA have been evaluated by

the following Environment Canada staff (effects on the environment) and Health Canada staff (effects on human health):

Environment Canada

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Health Canada

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M.E. Meek

Quantitative estimates of carcinogenic potency were provided by J. Shedden and S. Bartlett of Health Canada.

This report contains a Synopsis that will appear in the Canada Gazette. Section 2.0 is a summary of technical information critical to the assessment. The assessment of whether chromium is "toxic" as defined under CEPA is presented in Section 3.0. Supporting documentation that presents the technical information in greater detail is also available upon request.

As part of the review and approvals process established by Environment Canada for its contributions to assessment reports, environmental sections of this document were reviewed by Dr. C.D. Wren (Ecological Services for Planning, Guelph), Dr. M.G. Johnson (formerly of Fisheries and Oceans Canada), Prof. M.S. Simmons (Department of Environmental and Industrial Health, University of Michigan, Ann Arbor), and Prof. Raymond D. Harbison (Center for Environmental and Human Toxicology, University of Florida, Gainesville).

Comments on the adequacy of coverage of the literature relevant to human health in the supporting documentation were received from Dr. I. Harding-Barlow (a consulting toxicologist, United States), Dr. K. Mundt (Applied Epidemiology, Inc., United States), and Dr. M. Huvinen (chief medical officer, Outokumpu Tyoterveysspalvelut Oy, Finland). Sections of the supporting documentation relevant to human exposure were forwarded to officials of the Mining Association of Canada for identification of additional pertinent data. Following peer review of sections of the supporting documentation and Assessment Report relating to human health by Dr. H.J. Gibb (United States Environmental Protection Agency) and Dr. J. Siemiatycki (Institut Armand Frappier), these sections were approved by the Standards and Guidelines Rulings Committee of the Bureau of Chemical Hazards of Health Canada. The final Assessment Report was subsequently reviewed and approved by the Environment Canada/Health Canada CEPA Management Committee.

Copies of this Assessment Report and the unpublished supporting documentation are available upon request from:

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2.0 Summary of Information Critical to Assessment of "Toxic"

2.1 Identity, Properties, Production, and Uses

Chromium (Cr), with atomic number of 24 and relative atomic weight of 52.0, belongs to Group VIB in the Periodic Table. Elemental chromium is a grey, lustrous metal that is extremely resistant to ordinary corrosive agents. Although more than 40 chromium-containing minerals have been identified, and chromium is the seventh most abundant element in the earth as a whole, it is typically present only in trace amounts (i.e., < 1000 µg/g) in surface (i.e., crustal) rocks and soils. Nearly all the chromium in crustal rock occurs in the trivalent form with the most important ore mineral being chromite, FeCr₂O₄. Although chromium can exist in nine different oxidation states, from (-II) to (VI), only chromium (III) and (VI) are common in natural environments. In this report, unless otherwise specified, "chromium" refers to total chromium; similarly "trivalent" or "hexavalent" chromium refers to total chromium of each valency.

Ore-grade chromite [Cr(III)] has been identified at more than 250 locations in Canada, with the ore resources estimated to be about 20 x 10⁶ tonnes (t) (Energy, Mines and Resources, 1989). The principal deposits occur in Quebec, Ontario, British Columbia, Manitoba, and Newfoundland. The ores in the first three provinces are low grade (Cr₂O₃ content <25%); however, the Newfoundland deposits are of medium grade (Cr₂O₃ content of up to 53%) although they occur in isolated localities (Phillips, 1988). Domestic ores were mined in the past when there were limitations on imports; however, Canada now depends on foreign sources for all its chromium requirements.

Canada imported about 74 000 t (gross weight) of various chromium-containing materials in 1991, a decrease from about 94 800 t in 1989 (Statistics Canada, 1989; 1991). Materials imported in 1991 included chromium ferroalloys (55%), chromite ores/concentrates (28%), chromium-containing chemicals (7%), and about 10% as miscellaneous chromium-bearing substances, including waste scrap and powder.

The consumption of chromium ferroalloys was about 41 000 t in 1991 (Statistics Canada, 1991). Ferromanganese is an alloy used in the production of stainless and heat-resistant steels used in corrosive environments such as petrochemical processing, high-temperature environments such as turbines and furnaces, and in consumer goods such as cutlery and decorative trim (Phillips, 1988). Copper-chromium alloys are used in electrical applications that require high strength and good conductivity, while copper-nickel-chromium alloys are used in marine equipment requiring corrosion resistance (Nriagu, 1988). The automobile industry is a major user of chromium alloys in the form of stainless steel components, catalytic converters, chrome trims, and other control and decorative systems. Chromium-containing superalloys with high heat resistance are used in aircraft engines and other aerospace equipment (Nriagu, 1988).

About 24 000 t of chromite ores and concentrates were imported into Canada in 1991 (Statistics Canada, 1991). These materials are used to make refractory products

such as bricks, mortars, and ramming mixtures for domestic iron and steel, portland cement, glass, and in nonferrous metals industries (Nriagu, 1988; Phillips, 1988).

The principal chromium-containing chemicals imported for use by Canadian industries include chromium oxide, chromium chloride, and chromium sulphate (Statistics Canada, 1989; 1990; 1991). These compounds are used mainly in pigments, metal finishing, leather tanning (trivalent compounds), and wood preservatives (hexavalent compounds) (Nriagu, 1988). Smaller quantities are used in textiles, drilling muds, toners for copying machines, water treatment and cooling waters, magnetic tapes, and catalysts (U.S. EPA, 1984).

2.2 Entry into the Environment

Chromium occurs naturally in small amounts in all rocks and soils, as relatively inert Cr(III) solid phases, and is released into the aquatic environment in limited quantities by weathering and erosion of these materials. Chromium enters the atmosphere in Canada from such non-anthropogenic sources as windblown dusts, volcanic emissions, seasalt aerosols, dusts from wild fires, and vegetative debris. Atmospheric emissions of chromium from natural sources in Canada have not been quantified; however, since windblown dusts are the primary natural source of chromium in the earth's atmosphere (Nriagu, 1990), wind erosion of prairie soils may be a significant natural source of airborne chromium in central Canada. While most chromium released from natural sources is probably in the trivalent form, both Cr(III) and Cr(VI) can be present in anthropogenic wastes released into the environment.

It is estimated that about 84 t of chromium (including both tri- and hexavalent forms) are released into the Canadian atmosphere from anthropogenic sources each year. In addition, data from a partial inventory of discharges to water and land from industrial sources in Canada indicate that the annual loadings of chromium to these media exceed 27 and 5000 tonnes/year (t/yr), respectively.

Of the 84 t/yr of chromium released to the atmosphere by industries in Canada, approximately 51% is attributable to fossil fuel combustion, 29% to various industrial processes (including iron and steel production, and refractory and chemical processing), and 12% to transportation-related activities such as motor vehicle operation (Table 1). Minor sources include fugitive road dusts (from the wear of tires and brake linings), chromium-based automotive catalytic converters, and the release of chromated fine powders used as toners in copying machines (ATSDR, 1988).

The available information is insufficient to permit a complete inventory of chromium loadings to Canadian surface waters. However, since chromium is used in a wide variety of manufactured products, it is likely that chromium enters the aquatic environment in Canada from many industrial sources. Based on available data, at least 27 t of chromium are released annually in liquid discharges from Canadian base metal smelters and refineries, as well as from iron and steel plants, metal finishing plants, and petroleum refineries in Ontario. Data on emissions for 1988 indicate that nonferrous base metal smelters and refineries throughout Canada discharged liquid effluents

Table 1 Anthropogenic Emissions of Total Chromium to the Atmosphere in Canada*

Sources	Annual Emission (tonnes/year)	Percent of Total Emissions (%)
Industrial Process		29
Mining and milling of asbestos	1	
Nonferrous metal smelting	2**	
Iron and steel foundries	7	
Cement production	1	
Refractory and chemical processing	6	
Mirror and glass production	4	
Cooling towers	3	
Stationary Fuel Consumption		51
Power generation	24****	
Commercial fuel consumption	4	
Industrial consumption	9	
Residential fuel combustion	6	
Transportation		12
Motor vehicle (gasoline and diesel)	5	
Railroad	2	
Marine	1	
Off-road (gasoline and diesel)	2	
Solid Waste Incineration	2	2
Miscellaneous	5	6
Total	84	100

* revised from Jaques (1987)

* * from MacLatchy (1992)

*** from Cormier (1991)

containing approximately 2 t of chromium (MacLatchy, 1992). Results of a national survey of waste discharges from Canadian metal finishing plants indicate that approximately 16 t of chromium are released annually into Canadian surface waters from these sources (Jaimet, 1990).

Quantities of chromium released in industrial effluents in Ontario have also been estimated for the period 1987 to 1991 under the Municipal/Industrial Strategy for Abatement (MISA) program. Iron and steel plants in Ontario released about 7.7 t/yr of chromium into provincial waters (the St. Marys River, Lake Erie, and Lake Ontario) during approximately 12 months in 1989/1990 (MOE, 1991c). Treated effluents from these iron and steel mills typically contained 10 to 26 µg/L of chromium. Process effluents released from petroleum refineries in Ontario in 1988/1989 contained 6-month average concentrations of 87 and 126 µg/L of chromium, approximately 10% of which was Cr(VI) (MOE, 1989a; 1990). Based on these data, more than 1 tonne of chromium is discharged annually in liquid effluent from petroleum refineries into the St. Clair River and Lake Ontario.

Due to the large number of small industries that use chromium, significant quantities of chromium can also be discharged to surface waters from sewage treatment plants. The geometric mean concentrations of chromium in raw sewage entering 37 municipal water treatment plants in Ontario in 1987 ranged up to 820 µg/L; the average for all 37 plants was 51 µg/L (MOE, 1988). Similar levels of chromium have been reported in raw sewage in Regina, Edmonton, Calgary, Winnipeg, and Vancouver (Nielsen and Hrudey, 1983; Rao and Viraraghavan, 1992). Chromium was detected (detection limit = 10 µg/L) at concentrations of up to 140 µg/L in more than half of the samples of treated effluent from the 37 municipal water treatment plants monitored under the MISA program. Other potentially significant sources of chromium in the aquatic environment in Canada include discharges from leather tanning industries, urban stormwater runoff [e.g., 40 to 60 µg/L of chromium in Toronto in 1977 (Metro Toronto RAP Report, 1988)], effluent streams from pulp and paper mills [e.g., 6-month average concentrations of 30 to 45 µg/L of chromium for sulphate-type mills in Ontario in 1990 (MOE, 1991a;b)], and discharges from thermal generating stations [e.g., mean concentrations of 10 to 39 µg/L of chromium from plants in Ontario (Ontario Hydro, 1992)].

It is estimated that wastes containing more than 5000 t of chromium in various forms are dumped annually onto land in Canada. Jaimet (1990) estimated that in 1988, Canadian metal-finishing sludges contained 1600 t of chromium and that most of the chromated sludge was dumped in municipal landfills. Nonferrous base metal smelters and refineries disposed of about 2100 t of chromium in the form of slags (2000 t), sludges, and solid wastes on land in Canada in 1988 (MacLatchy, 1992). Coal burning power generating stations generated ashes containing about 838 t of chromium in 1988, which mostly end up on land (Cormier, 1991). The chromium contents of European household wastes typically fall in the range of 50 to 100 µg/g (Rousseaux *et al.*, 1989). Assuming similar concentrations in the 12×10^6 t of solid wastes generated each year by

Canadian households (UNEP, 1987), the annual disposal of chromium in this manner is estimated to be 600 to 1200 t. With a treatment removal efficiency of more than 80% (MOE, 1988), most of the chromium entering municipal wastewaters annually is removed as sludge, some of which is applied to Canadian agricultural lands (Webber and Shames, 1987).

Improper handling and storage of wood preservation chemicals that contain chromium can result in the transfer of large quantities of hexavalent chromium to local soils (Bamwoya *et al.*, 1991). Weathering of treated lumber exposed to acidic rainfall can also release chromium to soils (Warner and Solomon, 1990).

Only a limited amount of information was identified on concentrations of chromium in leachates at waste disposal sites in Canada. The concentrations of Cr(VI) in leachate from an industrial waste disposal site in Welland, Ontario, have exceeded 120 µg/L (MOE, 1991c). The concentrations of total chromium in refinery landfill leachates in Ontario averaged about 8.6 µg/L (MOE, 1991c), while levels in the leachates from the bottom ash from thermal generating stations in Ontario averaged 44 µg/L (Ontario Hydro, 1992).

2.3 Exposure-related Information

2.3.1 Fate

The fate of chromium discharged into the environment will vary depending on its chemical form (Murray *et al.*, 1983; Campbell and Yeats, 1984). Trivalent chromium tends to be associated with relatively inert solid phases. Chromium(III) can therefore accumulate and persist in sediments and soils, but its availability for uptake by biota may be limited. Results of recent studies indicate that labile forms of Cr(III) may be oxidized photochemically to Cr(VI) in aerobic surface waters. Most forms of Cr(VI) compounds are quite soluble and are not readily adsorbed onto particulate matter. Chromium(VI) can thus persist in bioavailable form in aerobic surface waters and soil pore waters, but it is reduced to the less mobile forms of Cr(III) in anaerobic conditions.

Chromium is released to the atmosphere primarily in particulate form. Although little is known about the chemical form of chromium in ambient air, Cr(III) oxides are expected near fossil fuel combustion and ore-processing plants, while Cr(VI) species should be present near chromate manufacturing and processing plants. While no information has been identified on the speciation of chromium in air in Canada, hexavalent chromium was determined to comprise 3 to 8% of the total chromium in four samples of ambient air in California (California Air Resources Board, 1985). Since airborne chromium is associated mostly with particulate phases (Davidson and Wu, 1989), it is removed from the atmosphere by both dry fallout and wet precipitation. The residence time of chromium in the atmosphere is estimated to be less than 14 days (Nriagu *et al.*, 1988).

Since Cr(III) forms highly insoluble oxides, hydroxides, and phosphates and is adsorbed by suspended particles, dissolved Cr(III) is removed rapidly from surface

waters by settling particulate matter (Cranston and Murray, 1978). However, Cr(III) can also form stable complexes with many dissolved or colloidal, organic, and inorganic ligands. This complexed Cr(III) is relatively unaffected by adsorption and precipitation reactions, and can thus remain in the water column (Masscheleyn *et al.*, 1992). Results of recent studies by Johnson *et al.* (1992) indicate that, in deep anoxic water, colloidal Cr(III) can be the dominant species of chromium. Although there are few oxidants capable of converting Cr(III) to Cr(VI), and the oxidation kinetics are normally very slow, it has recently been suggested that labile (including dissolved and colloidal) forms of Cr(III) can be converted to Cr(VI) relatively quickly by strong oxidants such as H₂O₂ that are produced photochemically in aerobic surface waters (Pettine and Millero, 1990; Pettine *et al.*, 1991; 1992; Nriagu *et al.*, 1993).

Due to its association with suspended particulate phases, a large proportion of the Cr(III) discharged to surface water is transferred to sediment. In aerobic sediments, some Cr(III) can be oxidized by manganese oxides and hydroxides present at the sediment-water interface (Saleh *et al.*, 1989; Bartlett and James, 1988), and it has been suggested that the resulting Cr(VI) can be released to the overlying waters especially by bioturbation processes (Gaillard *et al.*, 1986).

In contrast to Cr(III), Cr(VI) is not readily adsorbed to surfaces and, since most of its salts are soluble, much of the Cr(VI) released to aerobic surface waters is present in a soluble form as hydrochromate, chromate, and dichromate ionic species (Rai *et al.*, 1989). However, dissolved Cr(VI) can be converted to Cr(III) by a host of reducing agents such as S²⁻, Fe(II), fulvic acid, low molecular weight organic compounds, and proteins, and is thus removed from solution, especially in deeper anaerobic waters (Nriagu *et al.*, 1993). The effectiveness of these reducing agents varies with pH, redox conditions, and total concentrations of chromium (Nriagu *et al.*, 1993). A small amount of Cr(VI) can also be taken up by plankton and released as Cr(III) at lower depths where oxygen is depleted (Beaubien, 1993).

Although naturally occurring chromium is present as relatively inert forms of Cr(III) in most soils, with time the Cr(III) can be mobilized by acid leaching (podzolization) (Bartlett and James, 1988). Chromium(III) can be oxidized to Cr(IV) by the manganese oxides present in soils, but only a small percentage of the Cr(III) in soils is normally present in oxidizable forms (Bartlett and James, 1988). The oxidation of Cr(III) to Cr(VI) is facilitated by the presence of moisture and small amounts of organic matter, and can be enhanced in surface soils by elevated temperatures created in brush fires (Cary, 1982).

Chromium(IV) added to or formed in soils can be removed from solution by uptake into living organisms, leaching (resulting in transfer to groundwater), adsorption, or reduction to relatively immobile Cr(III) (Bartlett and James, 1988). In general, reduction is favoured in anaerobic (e.g., waterlogged or organic-rich) or acidic soils (Bartlett and James, 1988; Masscheleyn *et al.*, 1992). Adsorption is expected to be most effective in neutral to slightly acidic soils, especially those containing large amounts of iron oxides (Rai *et al.*, 1989). Adsorption of Cr(VI) can reduce or completely prevent its

reduction to Cr(III); increasing soil pH by liming or addition of phosphate fertilizers, would likely result in the remobilization of adsorbed Cr(VI) (Bartlett and James, 1988).

In general, Cr(VI) is absorbed into cells more readily than Cr(III) (Nieboer and Jusys, 1988) (see also Section 2.4). Chromium is readily accumulated by aquatic biota (NAS, 1974; Jackson, 1988; Havas and Hutchinson, 1987) and reported bioconcentration factors typically range from 100 to 1000 (CCREM, 1987). Chromium is not, however, biomagnified in either aquatic or terrestrial food chains (Mance, 1987; Outridge and Scheuhammer, 1993). In most organisms, Cr(VI) is reduced to Cr(III), the form that is commonly found in proteins, enzymes, and nucleotides (Nieboer and Jusys, 1988).

2.3.2 Concentrations*

Chromium has been detected frequently in samples of ambient air, surface water, drinking water, food, and sediments in both uncontaminated and contaminated areas. Levels of chromium in contaminated soils (excluding those at wood preservation plants) and biota in Canada are less well quantified. In most of the studies on the distribution of chromium in the Canadian environment, only total chromium was measured; little information was identified on the species of chromium present in environmental media. Based on a limited number of samples of ambient air in California, most of the chromium in this medium is likely to be in the trivalent form. Results of recent studies in Canada and elsewhere, however, indicate that Cr(VI) is the dominant form of dissolved chromium in surface waters. Levels of Cr(III) could nevertheless be elevated in some deep anoxic waters and in water receiving direct discharges of Cr(III) - containing wastes. Nearly all of the chromium in soils [excluding those contaminated with Cr(VI)] (Bartlett and James, 1988), sediments (excluding those immediately below the interface with overlying aerobic waters) (Nriagu *et al.*, 1993), and biological tissues (Anderson, 1981; Nieboer and Jusys, 1988) is likely present as Cr(III).

Mean airborne concentrations of chromium in 12 Canadian cities from 1987 to 1990 ranged from 0.003 to 0.009 $\mu\text{g}/\text{m}^3$ (Dann, 1991), while levels in nonurban areas were usually $<0.001 \mu\text{g}/\text{m}^3$ (Nriagu *et al.*, 1988; Barrie and Hoff, 1985). In the city of Hamilton (Ontario), where several iron and steel mills are located, the mean concentrations of chromium at several sites in 1990 ranged from 0.006 to 0.020 $\mu\text{g}/\text{m}^3$; the maximum value was 0.68 $\mu\text{g}/\text{m}^3$ (MOE, 1991c). Near industrial point sources in central Ontario, concentrations as high as 1.25 $\mu\text{g}/\text{m}^3$ have been recorded (Environment Canada, 1991).

Few data have been identified on levels of chromium in indoor air. It was not detected (detection limit = 0.08 $\mu\text{g}/\text{m}^3$) in the air in kitchens in five homes in Toronto

* Due in part to the uncertain accuracy of older data on concentrations of chromium in environmental media (Sturgeon and Berman, 1987), data reported in this section generally reflect results of the most recent studies available.

(Mann Testing Laboratories, 1992). Concentrations in indoor air (range 0.001 to 0.017 $\mu\text{g}/\text{m}^3$) in homes near a chromate waste site in New Jersey were "typically lower" than those in outdoor air; air in the homes of smokers contained nearly twice as much chromium as homes of non-smokers (0.0115 versus 0.006 $\mu\text{g}/\text{m}^3$), based on a small number of samples ($n = 3$ or 4) (Lioy *et al.*, 1992).

Average concentrations of total chromium [i.e., including Cr(III) and Cr(VI) in dissolved and particulate phases] in uncontaminated surface and marine waters are generally below 1.0 $\mu\text{g}/\text{L}$ (Mayer, 1988; Rossmann and Barres, 1988; Beaubien, 1993; Erickson and Fowler, 1987). However, much higher concentrations have been reported in contaminated surface waters in many parts of Canada. In Alberta, total Cr concentrations in a number of streams sampled in 1988 ranged from 1 to 55 $\mu\text{g}/\text{L}$ and averaged 5.1 $\mu\text{g}/\text{L}$ (Alberta Environment, 1989). Total chromium concentrations in samples collected in 1988 from 11 rivers across British Columbia averaged 6.9 $\mu\text{g}/\text{L}$ (range: 0.3 to 165 $\mu\text{g}/\text{L}$) (Environment Canada, 1989a). In a survey, conducted from 1986 to 1988, of the tributaries of the St. Lawrence River in the province of Quebec, the mean concentration of total chromium was 7.1 $\mu\text{g}/\text{L}$, with a range of 1.5 to 92 $\mu\text{g}/\text{L}$ (Quebec Ministry of the Environment, 1989a). Median concentrations of total chromium were estimated to be approximately 4 $\mu\text{g}/\text{L}$ in rivers and streams of central Ontario in 1987 (MOE, 1991d). High mean levels were reported in surface waters in the Metropolitan Toronto area (11 $\mu\text{g}/\text{L}$ in the Don River and 8 $\mu\text{g}/\text{L}$ in the Humber River), as well as in Oshawa (8 $\mu\text{g}/\text{L}$ in Oshawa Creek). Elevated mean concentrations of total chromium were also found in rivers in Ottawa (7 $\mu\text{g}/\text{L}$ in the Jock River), and in northwestern Ontario at Thunder Bay (10 to 38 $\mu\text{g}/\text{L}$ in the Kaministiquia River, 18 $\mu\text{g}/\text{L}$ in the Mission River, and 8 to 14 $\mu\text{g}/\text{L}$ in the McKellar River) (MOE, 1991d). Based on comparison of levels of chromium in filtered and unfiltered North American river water (Merritt, 1975; Gibbs, 1977; Allan, 1986; Campbell and Yeats, 1984; Kauss *et al.*, 1988), and the results of recent studies of the speciation of dissolved chromium in aerobic lake waters (Johnson *et al.*, 1992; Balistrieri *et al.*, 1992; Beaubien, 1993), about 10 to 60% of the total chromium content of Canadian rivers is present as dissolved Cr(VI).

Median concentrations of total (i.e., dissolved plus particulate-phase) chromium in surface water samples collected in the early 1980s in the Great Lakes were 0.09, 0.13, 0.39, and 0.82 $\mu\text{g}/\text{L}$ in Lakes Superior, Huron, Erie, and Ontario, respectively (Rossmann and Barres, 1988). Median concentrations in filtered (0.5 μm) samples were 0.08, 0.11, 0.27, and 0.77 $\mu\text{g}/\text{L}$ for Lakes Superior, Huron, Erie, and Ontario, respectively, implying that 70 to 90% of the chromium is in the soluble form (Rossmann and Barres, 1988). In a recent study, all of the dissolved chromium in Lake Erie and Lake Ontario was determined to be Cr(VI) (Beaubien, 1993).

Median concentrations of chromium in two surveys of drinking water supplies in 70 and 71 cities across Canada in 1976 and 1977, respectively, were ≤ 2.0 $\mu\text{g}/\text{L}$ (Méranger *et al.*, 1979; Méranger *et al.*, 1981). Similar concentrations have been reported in more recent municipal and provincial monitoring programs, with mean values ranging from 0.3 to 4.3 $\mu\text{g}/\text{L}$ (see supporting documentation).

Freshwater sediments in many parts of Canada have become contaminated with chromium as a result of loadings from industrial sources. The most severely affected sites in Ontario include the St. Marys River system with concentrations of 31 000 µg/g dry weight (d.w.) in Tannery Bay (St. Marys River RAP Report, 1992) and the Welland River downstream from a steel manufacturing plant, where the concentrations of chromium exceeded 5120 µg/g (d.w.) compared to 10 µg/g (d.w.) at the upstream control site (Dickman *et al.*, 1990). Concentrations ranged up to 1920 µg/g (d.w.) in Detroit River sediments (Lum and Gammon, 1985) and 564 µg/g (d.w.) in Hamilton Harbour sediments (Nriagu *et al.*, 1983). Elevated concentrations of two- to four-fold above local background levels have also been reported in sediments from Lake Simcoe (Johnson and Nicholls, 1988) in Ontario; Belledune and Dalhousie Harbours in New Brunswick (Samant *et al.*, 1990); Saguenay Fjord in Quebec (Pelletier and Canuel, 1988); the Detroit River (Fallon and Horvath, 1985; Hamdy and Post, 1985); Lake Ontario off the Niagara River (Mudroch *et al.*, 1988); the St. Lawrence River (Loring, 1979); and the Fraser River drainage basin in British Columbia (Schreier *et al.*, 1987).

The concentrations of chromium in soils from 173 sites in Canada ranged from 10 to 100 µg/g (d.w.) [mean value of 43 µg/g (d.w.)] (McKeague and Wolynetz, 1980). High levels, of over 1000 µg/g (d.w.), have been reported in soils associated with chromium-enriched serpentine bedrock in western Newfoundland (Roberts, 1980). Mean concentrations of chromium in agricultural soils of Ontario were reported to range from 10 to 22 µg/g (d.w.) (Frank *et al.*, 1976). Elevated concentrations [over 100 µg/g (d.w.), compared to 38 µg/g in uncontaminated soils nearby] were reported in soils adjacent to two scrap yards in Winnipeg (Manitoba Environment and Workplace Safety and Health, 1989). Levels of chromium in soils around the smelter at Belledune, New Brunswick were 40 to 120 µg/g (d.w.) and were greater than those away from the smelter (MacMillan, 1982), while the mean concentration of chromium in soil near a generating station fueled by coal containing significant amounts of chromium in Alberta was 25 µg/g (Van Voris *et al.*, 1985). Concentrations of chromium have also been greater in surface soil near several Canadian wood treatment facilities that use preservative formulations containing dissolved Cr(VI). Chromium levels in soil near a wood preserver's property in Neepawa, Manitoba averaged 243 µg/g (d.w.) compared to only 10 µg/g (d.w.) at an adjacent uncontaminated site (Manitoba Environment and Workplace Safety and Health, 1989). The average concentration around a wood preservation plant in Roblin, Manitoba was about 718 µg/g (d.w.) compared to 30 µg/g (d.w.) for the control site (Manitoba Environment and Workplace Safety and Health, 1989). Henning and Konasewich (1984) found in excess of 200 µg/g (d.w.) of chromium in several soils at a wood preservation plant in British Columbia, compared to background concentrations of 10 to 20 µg/g (d.w.). Average chromium concentrations in surface soil near two wood treatment facilities in Atlantic Canada were 1170 and 1760 µg/g (d.w.) [the maximum was 5280 µg/g (d.w.)]; levels were lower [generally <300 µg/g (d.w.)] at two other wood treatment operations in Atlantic Canada (Bamwoya *et al.*, 1991).

Data on concentrations of chromium in Canadian terrestrial biota are sparse. Based on limited information, the concentrations of chromium in plants in Canada are normally less than 4 µg/g (d.w.), even close to potential point sources such as roadways and coal-fired generating stations (Percy, 1983; Van Voris *et al.*, 1985; McIlveen, 1986; Frank *et al.*, 1987; Emerson, 1988). Concentrations of chromium in the liver, kidney, and feathers of adult herring gulls (*Larus argentatus*) collected in 1983 from the Great Lakes basin averaged 0.55, 0.12, and 0.39 µg/g wet weight (w.w.) while the respective values for preledged gulls were 0.09, 0.10, and 0.71 µg/g (w.w.) (Struger *et al.*, 1987). Mean concentrations of chromium in the livers of Leach's storm-petrels (*Oceanodroma leucorhoa*), Atlantic puffins (*Fratercula arctica*), double-crested cormorants (*Phalacrocorax auritus*), and herring gulls collected in 1988 from different parts of Atlantic Canada ranged from <1.0 to 4.4 µg/g (d.w.) (Elliott *et al.*, 1992). Levels in common tern populations in Hamilton Harbour and Long Island Sound averaged 9.4 to 9.8 µg/g (d.w.) of Cr in bone, compared to <2.0 µg/g (d.w.) in liver and 3 to 4 µg/g (d.w.) in kidney (Connors *et al.*, 1975).

With regard to aquatic organisms, concentrations of chromium in samples of attached filamentous green algae *Ulothrix zonata* collected in 1979 were reported to be 2 to 18 µg/g (d.w.) in Georgian Bay, 0.9 to 5.8 µg/g (d.w.) in the North Channel, 4.5 to 5.5 µg/g (d.w.) in Lake Huron, 1.4 to 8.4 µg/g (d.w.) in Lake Superior, and 6.8 to 8.8 µg/g (d.w.) in Lake Ontario (Jackson, 1988; Jackson *et al.*, 1990). For floating Cladophora, concentrations of chromium were 2 to 23 µg/g (d.w.) in Georgian Bay and 3.9 to 4.6 µg/g (d.w.) in Lake Huron, and for submerged samples from Georgian Bay, the reported concentrations were 7 to 29 µg/g (d.w.) (Jackson, 1988). Samples of net plankton obtained at five locations in Lake Huron in 1983 contained 2.7 µg/g (d.w.) of Cr; the freshwater shrimp, *Mysis relicta*, contained 3.4 µg/g (d.w.) of Cr and the amphipod, *Pontoporeia* sp., contained 2.9 to 13 µg/g (d.w.) of Cr (Environment Canada, 1991). Midge larvae and mayflies (*Hexagena*) collected in 1985 from Midland Bay and Penetang Bay (Lake Huron) had 1.2 to 1.7 µg/g (w.w.) of Cr, compared to 1.3 µg/g at the control area (Severn Sound RAP Report, 1988). Krantzberg (1985) reported higher concentrations of chromium in benthos larvae [5.7 µg/g (d.w.)] in lakes of the Muskoka-Haliburton area, Ontario, compared to the values for adult benthic species [1.0 µg/g (d.w.)] from the same water. Up to 28 µg/g (w.w.) were detected in Spermatophyta, 4.5 µg/g (w.w.) in Gastropoda, 20 µg/g (w.w.) in Cladophora, and 2.3 µg/g (w.w.) in Pelecypoda collected from 1979 to 1982 from the Cornwall-Massena section of the St. Lawrence River (Kauss *et al.*, 1988). Levels in predatory and bottom-feeding fish from 14 lakes in northern Ontario averaged 0.23 µg/g (w.w.) of Cr, the range being 0.19 to 0.27 µg/g (w.w.) (Johnson, 1987). Concentrations of chromium in whole fish samples collected in 1985 and 1986 from the Fraser River, British Columbia were in the range of 0.17 to 0.23 µg/g (d.w.) for Coho salmon, prickly sculpin, Northern squawfish, reidside shiner, threespine stickleback, and large-scale sucker (Schreier *et al.*, 1987).

Most foodstuffs contain chromium. In a recent survey of a wide range of Canadian foodstuffs, concentrations ranged from non-detectable (i.e., <0.004 µg/g) to 0.1 µg/g in wheat and bran cereals (Mann Testing Laboratories, 1992). Levels in food may be increased through the use of utensils containing chromium during processing and preparation. Although no data were identified on concentrations of chromium in human breast milk in Canada, based on review of the "better quality" studies in other countries, Kumpulainen (1992) reported that mean levels ranged from 0.25 to 0.39 µg/L.

A variety of household products, such as cleaning agents, contain chromium. It is also released from wood products treated with preservatives containing chromium. The mainstream smoke of Canadian cigarettes has been estimated to contain 0.147 µg per cigarette (Labstat Incorporated, 1992).

2.4 Toxicokinetics and Essentiality

In general, hexavalent chromium compounds penetrate biological membranes much more readily than trivalent compounds. Based on review of available literature, estimates of the proportion of trivalent chromium that is absorbed from the gastrointestinal tract range up to about 3%, while up to about 10% of ingested hexavalent chromium is estimated to be absorbed; organic complexes are even more readily absorbed. Much of ingested hexavalent chromium is reduced to trivalent chromium before absorption. Absorption of inhaled chromium is believed to be greater, with estimated proportions of up to 12% and 30% for trivalent and hexavalent compounds, respectively; some hexavalent chromium is reduced to the trivalent species by the epithelial lining of the lung. Absorption is also dependent on the physical/chemical properties of the specific compound. Absorbed chromium is widely distributed throughout the body, including to the fetus, via the bloodstream. After crossing cellular membranes, hexavalent chromium may be reduced to trivalent forms, via a number of hypothesized intermediates. Absorbed chromium is eliminated from the body largely in the urine, while most of the unabsorbed portion is excreted in the feces.

Trivalent chromium is considered to play an essential role in the metabolism of glucose in humans and animals, as it is a key component of the glucose tolerance factor. The National Academy of Sciences in the United States has recommended daily intakes of trivalent chromium for adults of between 50 and 200 µg/day, based on the absence of signs of chromium deficiency in the majority of the population consuming an average of 50 µg/day, and the lack of toxic signs in supplementation trials in humans receiving 150 µg/day in addition to dietary intake (NAS, 1989). Recommended daily intakes for other age groups are 10 to 40, 20 to 60, 20 to 80, and 30 to 120 µg/day for children aged 0 to 6 months, 6 months to 1 year, 1 to 3 years, and 3 to 6 years, respectively (NAS, 1980). However, Anderson *et al.* (1993) suggested that some of these values should be redefined, based on the observation that apparently healthy breast-fed infants of 17 women were receiving much less than the recommended daily amount specified by the National Academy of Sciences. In the Nutrition Recommendations of the Report of the Scientific Review Committee of Health and Welfare Canada, it has more recently

been concluded that "...there are insufficient data, however, to establish a minimum requirement for adults..." (Health and Welfare Canada, 1990).

2.5 Effects-related Information

2.5.1 *Experimental Animals and In Vitro*

The toxicity of chromium compounds depends principally on valency, as well as physical/chemical properties of the specific compounds. To the extent possible, data on the effects of trivalent and hexavalent chromium have been addressed separately. More detailed information on the effects of specific hexavalent and trivalent chromium compounds is included in the supporting documentation. In general, the acute toxicity of chromium compounds in experimental animals increases with water solubility; oral LD₅₀s for trivalent compounds have been reported to range from 140 to 522 mg of Cr(III)/kg body weight (b.w.), while reported LD₅₀ values for hexavalent compounds ranged from 13 to 795 mg of Cr(VI)/kg(b.w.). Skin sensitization has been induced in animals by both trivalent and hexavalent chromium.

Most short-term or subchronic studies in which animals have been administered chromium by ingestion have involved hexavalent chromium. The observed effects in these predominantly limited investigations were restricted largely to those on the liver and kidney (histological effects as well as alterations in biochemical parameters), and alterations in hematological parameters; the lowest effect levels were in the range of 50 to 100 mg of Cr(VI)/[kg(b.w.)·d] (Diaz-Mayans *et al.*, 1986; Coogan *et al.*, 1991; Kumar and Barthwal, 1991; Kumar and Rana, 1982; 1984; Kumar *et al.*, 1985; Laborda *et al.*, 1987; Kennedy *et al.*, 1976; Christofano *et al.*, 1976). In short-term or subchronic investigations, the effect of inhaled hexavalent chromium on the lung or immunological parameters have been examined. Observed effects included inflammatory changes in the respiratory tract and alterations in response and morphology of macrophages at 180 µg of Cr(VI)/m³ and above, although some effects on macrophage function and immunological response (not necessarily adverse) have been reported at 25 µg/m³ (Glaser *et al.*, 1985; Johansson *et al.*, 1986a;b; 1987; Popper *et al.*, 1992).

Less information is available on the short-term and subchronic toxicity of trivalent chromium. Decreased weights of the spleen and liver were reported in rats ingesting trivalent chromium in the diet at doses of approximately 72 g[kg(b.w.)·d] and above { 72 000 mg[kg(b.w.)·d] } for 90 days (Ivankovic and Preussmann, 1975). Morphological and functional alterations have been observed in the alveolar macrophages of rabbits exposed to 600 mg of Cr(III)/m³ for up to 21 weeks (Johansson *et al.*, 1986a;b).

The chronic toxicity and carcinogenic potential of ingested or inhaled chromium compounds have not been extensively investigated. Most of the studies identified are not considered adequate to evaluate the carcinogenicity of chromium, nor are any considered adequate to determine an effect level for non-neoplastic endpoints. No adverse effects or increase in the incidence of tumours at any site were reported in limited studies in which rats (two strains), mice (one or two strains), or dogs (three strains) were administered hexavalent chromium in the diet or drinking water for periods of up to four years

(MacKenzie *et al.*, 1958; Schroeder *et al.*, 1963; Gross and Heller, 1946; Borneff *et al.*, 1968; Anwar *et al.*, 1961). Alterations in hematological and biochemical parameters, which were not related to concentration, were noted in ddY mice administered drinking water containing 25 to 100 mg/L of hexavalent chromium for up to one year (Maruyama, 1982).

Available data on the carcinogenicity of inhaled hexavalent chromium from principally limited studies are equivocal; although small increases in the incidence of lung tumours were observed in several studies (Popper *et al.*, 1992; Glaser *et al.*, 1986; Adachi *et al.*, 1986; Adachi, 1987). Only borderline increases, the significance of which was not always reported, have been reported in larger investigations (Steffee and Baetjer, 1965; Baetjer *et al.*, 1959; Nettesheim *et al.*, 1971). Based on available data, hexavalent chromium has been, at most, weakly carcinogenic in experimental animals exposed via inhalation. Although many of these studies were limited by small group sizes, inadequate histopathological examination, and/or single exposure levels, effects on the respiratory system have been reported in rats, including accumulation of eosinophilic substances inside the alveolar lumen, focal thickened septa, interstitial fibrosis, and focal bronchiolo-alveolar hyperplasia (Glaser *et al.*, 1986), granulomata, giant cells, and abscesses (Steffee and Baetjer, 1965). Effects on the respiratory system observed in mice included perforated nasal septum, loss of cilia, proliferation of goblet cells or basal cells, squamous metaplasia or hyperplasia of the trachea, larynx, bronchus, or lungs, emphysema (Adachi *et al.*, 1986; Adachi, 1987), necrosis, and alveolar bronchiolization and proteinosis (Nettesheim *et al.*, 1971). In addition, respiratory effects have been reported in rabbits [perforated nasal septum (Steffee and Baetjer, 1965)] and guinea pigs [alveolar and interstitial inflammation and alveolar hyperplasia (Steffee and Baetjer, 1965)]. The lowest-observed-effect-level (LOEL) for such effects was reported to be 0.1 mg/m³ [as a 3:2 mixture of Cr(VI) and Cr(III)] (Glaser *et al.*, 1986).

No increase in the incidence of tumours at any site has been observed in the few limited earlier studies in which rats or mice were administered trivalent chromium in drinking water or the diet (MacKenzie *et al.*, 1958; Schroeder *et al.*, 1963; 1964; 1965; Ivankovic and Preussmann, 1975), although these studies were largely inadequate for assessment of potential carcinogenicity. Non-dose related changes in hematological and biochemical parameters have been reported in ddY mice administered trivalent chromium in the drinking water at concentrations of 25 to 100 mg/L for up to one year (Maruyama, 1982). Although slight increases in the incidence of tumours of the respiratory system or lymphosarcomas were observed in three studies in which rats and mice were exposed to a mixture of trivalent and hexavalent chromium by inhalation, these increases could not be clearly attributed to the trivalent species (Glaser *et al.*, 1986; Baetjer *et al.*, 1959).

Hexavalent chromium compounds have been consistently positive in several genotoxicity assays in nonmammalian systems and *in vitro* and *in vivo* mammalian systems, inducing DNA damage, gene mutation, sister chromatid exchange, chromosomal aberrations, aneuploidy, cell transformation, and dominant lethal mutations (IARC, 1990; De Flora *et al.*, 1990). While a variety of genetic effects have been

induced by trivalent chromium in subcellular or acellular Systems, in general, trivalent compounds have not been genotoxic in cultured animal or human cells (IARC, 1990; De Flora *et al.*, 1990). Intracellular reduction of hexavalent chromium to Cr(V), Cr(IV), and Cr(III) is believed to be required for genotoxicity.

It has been proposed (e.g., Jones, 1990; De Flora *et al.*, 1989) that there may be a threshold for the carcinogenicity of hexavalent chromium, based on the hypothesis that the administered dose must exceed the extracellular capacity to reduce (i.e., by gastric juices or in the mucosal lining) hexavalent chromium to the trivalent species (the form usually found associated with the DNA), which has a lesser ability to cross cellular membranes. Similarly, the ability of trivalent chromium that is produced intracellularly to cross the nuclear membrane is limited. However, as cellular uptake of trivalent chromium has been demonstrated (Alcedo and Wetterhahn, 1990), although to a lesser degree than the hexavalent species, and since the entry of hexavalent chromium into cells is rapid and extracellular reduction in the mucosal lining is incomplete (Witmer, 1991), currently available data are insufficient to support the hypothesis of a threshold for the interaction of chromium with genetic material.

Little information was identified on the reproductive or developmental toxicity of trivalent or hexavalent chromium compounds in experimental animals when administered by routes most relevant to exposure in the general environment (i.e., inhalation or ingestion). There is no consistent evidence of adverse effects on development at doses that were not maternally toxic; similarly, effects on reproduction have not been consistently reported in available studies. Histological changes in the ovaries of rats have been reported at 0.02 mg of Cr(VI)/m³ (Davydova, 1980), while effects on spermatogenesis in mice have been noted at 3.5 mg of Cr(VI)/[kg(b.w.)·d] and 3.4 mg of Cr(III)/[kg(b.w.)·d], with effects occurring more frequently in mice exposed to hexavalent chromium (Zahid *et al.*, 1990).

2.5.2 Humans

The carcinogenicity of chromium compounds has been investigated in a wide range of occupationally exposed populations. In most of the studies, quantitative data on levels of chromium to which the study populations were exposed were inadequate to determine a quantitative exposure-response relationship; in addition, smoking was not taken into account as a potential confounding factor. As well, in many studies, the workers were concomitantly exposed to other substances, such as nickel, polycyclic aromatic hydrocarbons (PAHs), or asbestos.

In studies in the chrome-plating industry, an association between exposure to hexavalent chromium and cancer of the respiratory system has been consistently observed (Sorahan *et al.*, 1987; Royle, 1975; Silverstein *et al.*, 1981; Franchini *et al.*, 1983), with some indication of an association with duration of exposure. Where smoking was taken into account (Royle, 1975), it did not explain the excess of cancers; in addition, it is unlikely that smoking would account for the large excesses observed (i.e., two- to three-fold). These workers were also exposed to several other chemicals,

including nickel, but where it was possible to take these exposures into account (Sorahan *et al.*, 1987), they did not explain the excess.

There was consistent evidence of excess respiratory cancer, and in one study, nasal cancer, associated with exposure to a variety of both trivalent and hexavalent chromium compounds in chromate production workers (Taylor, 1966; Enterline, 1974; Hayes *et al.*, 1979; Davies *et al.*, 1991; Satoh *et al.*, 1981a;b; Watanabe and Fukuchi, 1975; 1984; Ohsaki *et al.*, 1978; Korallus *et al.*, 1982; De Marco *et al.*, 1988; Mancuso, 1975). Although smoking was not taken into account in any of these investigations, it was unlikely to account for the large excesses observed (generally approximately two-fold, with increases of eight- and nine-fold also reported). In almost all cases where examined, the excess was associated with duration and degree of exposure. Decreases in mortality due to lung cancer were also associated with introduction of control measures to reduce exposure. In the study with the most extensive information on exposure, 332 chromate production workers employed for at least one year from 1931 to 1937 were classified by categories of estimated cumulative exposure to total chromium, soluble chromium (principally hexavalent), and insoluble chromium (principally trivalent), based on monitoring data obtained in 1949. Rates of death due to lung cancer as of 1974 increased with estimated cumulative exposure to total chromium (0 to 741.5 per 100 000 for cumulative exposure of <0.50 to 6.00 mg/m³-years), as well as with estimated cumulative exposure to soluble chromium (80.2 to 998.7 per 100 000 for <0.25 to 2.00 mg/m³-years) and insoluble chromium (0 to 649.6 per 100 000 for <0.25 to 4.00 mg/m³-years) (Mancuso, 1975). It should be noted, however, that the number of deaths within each subgroup was small (n = 0 to 16), and no information was presented on smoking habits or concomitant exposure of workers to other substances.

There was consistent evidence of an excess of lung cancer in the most sensitive studies in chromate pigment production workers exposed predominantly to hexavalent chromium (Davies, 1984; Hayes *et al.*, 1989; Langard and Vigander, 1983; Haguenoer *et al.*, 1981). Although smoking was not taken into account in any of these investigations, it is unlikely to explain the large excesses observed (generally a two- to four-fold increase, though a 44-fold increase based on incidence in a small study was also reported). Where examined in sufficiently sensitive studies, there was an association between lung cancer excess and both duration and degree of exposure. Although no increase in mortality due to lung cancer was observed in a recent study in Japanese chromate pigment production workers (Kano *et al.*, 1993), the numbers of observed and expected deaths were small and it is not possible to determine if the concentrations to which these workers were exposed were comparable to those reported in studies in which excesses in mortality due to this cause were observed.

Associations between exposure to hexavalent chromium and respiratory cancer have been observed in some, but not all, studies in workers using chromate pigments. In the two largest studies, mortality due to respiratory or lung cancer was significantly elevated, and increased with duration of exposure, although these workers were also exposed to other substances (Dalager *et al.*, 1980; Bertazzi *et al.*, 1981). No significant

increase in respiratory cancer was noted in a smaller proportionate mortality study and in the accompanying nested case-control study (Chiazze *et al.*, 1980).

The results of studies in stainless steel workers who are exposed to hexavalent chromium fumes, as well as nickel, have been inconclusive. Although there were increases in the incidence of or mortality due to respiratory cancer in two studies (Becker *et al.*, 1991; Simonato *et al.*, 1991), these increases were not clearly attributable to exposure to hexavalent chromium, as asbestos appeared to be an important confounding factor in the former study, and mortality was not related to cumulative exposure to chromium in the latter report.

Mortality due to cancer of the respiratory tract and bladder/urinary tract was increased in a group of Icelandic masons exposed to hexavalent chromium, among other metals (Rafnsson and Jóhannsdóttir, 1986). No evidence of excess mortality due to cancer was reported in two studies in chrome leather workers who are exposed to a variety of chromium compounds, including hexavalent compounds, in addition to several known carcinogenic substances (Pippard *et al.*, 1985; Stern *et al.*, 1987). In a large, multi-industry study in Montreal, there was a significant association between occupational exposure to chromium, and hexavalent chromium specifically, and cancer of the lung and kidney (Siemietycki, 1991).

There is little consistent convincing evidence of associations between exposure to trivalent chromium and cancer. Mortality due to stomach cancer was increased in a large cohort study in Ontario gold miners who were exposed to trivalent chromium, and was related to duration of exposure and an indirect measure of cumulative exposure to chromium, but not to cumulative exposure to other substances (i.e., arsenic or mineral fibres) (Kusiak *et al.*, 1993); however, an association between stomach cancer and exposure to trivalent chromium has not been confirmed in other analytical epidemiological studies. There was no clear association between exposure to chromium (primarily in the metallic and trivalent forms) and incidence of or mortality due to respiratory cancer in one small study (Langård *et al.*, 1990) and two larger studies (Moulin *et al.*, 1990; Axelsson *et al.*, 1980) in ferrochromium alloy workers, although the numbers of deaths due to this cause were small (i.e., 10 to 18). In the cohort studied by Moulin *et al.* (1990), the excess in lung cancer mortality was demonstrated to be related more to exposure to polycyclic aromatic hydrocarbons than to chromium in an accompanying case-control study. Increases in mortality due to cancer of the lung and esophagus were reported in the limited study of Russian ferrochromium workers, although these workers were also exposed to polycyclic aromatic hydrocarbons (Pokrovskaya and Shabynina, 1973). Mortality due to lung cancer was not elevated in workers at a nickel/chromium foundry (Cornell and Landis, 1984). However, the incidences of cancer of the oral cavity and pharynx and colon/rectum were elevated in workers exposed principally to metallic chromium in the manufacture of stainless steel objects (Svensson *et al.*, 1989). Mortality due to lung and stomach cancer was increased compared to national rates in a limited ecological study of residents of a village in China whose drinking water contained high concentrations of hexavalent chromium (Zhang and

Li, 1987). However, no analytical studies have been identified in which the carcinogenicity of ingested chromium in human populations was investigated.

A variety of non-neoplastic effects on the respiratory system has been reported in several epidemiological studies of populations occupationally exposed to chromium compounds. Increased mortality due to non-malignant respiratory disease has been reported in one large study in chromeplaters exposed to hexavalent chromium (Sorahan *et al.*, 1987). Mixed results have been obtained in studies in workers involved in the production of chromium compounds who were largely exposed to hexavalent chromium; increased mortality due to non-malignant respiratory disease was reported by Davies *et al.* (1991) only in workers exposed to relatively high concentrations prior to improvements in the plant, and in an earlier study by Taylor (1966), while no excess deaths due to these causes were noted by Hayes *et al.* (1979; 1989) and Satoh *et al.* (1981a). No increased mortality due to non-malignant respiratory disease was reported in studies in welders exposed to hexavalent chromium (Becker *et al.*, 1991; Simonato *et al.*, 1991), painters using chromate pigments (Dalager *et al.*, 1980; Chiazze *et al.*, 1980), chrome leather tanners exposed to chromium in the hexavalent form (Stern *et al.*, 1987), stone masons using cement containing hexavalent chromium (Rafnsson and Jóhannesdóttir, 1986), ferrochromium workers exposed to both hexavalent and trivalent chromium (Moulin *et al.*, 1990; Axelsson *et al.*, 1980), or workers manufacturing objects from stainless steel who were exposed to metallic chromium (Svensson *et al.*, 1989). Deaths due to non-neoplastic respiratory disease were increased in nickel-chromium alloy foundry workers (Cornell and Landis, 1984).

Non-fatal respiratory effects, including nasal ulceration and septum perforation, coughing, sneezing, nasal irritation and bleeding, phlegm production, hemoptysis, bronchial asthma and bronchitis, as well as decreased pulmonary function, have been reported in inherently limited cross-sectional studies in workers exposed to hexavalent or trivalent chromium at concentrations as low as 2 to 20 $\mu\text{g Cr}/\text{m}^3$ (as hexavalent chromium) (Lindberg and Hedenstierna, 1983). Data on exposure in these studies have been largely insufficient to firmly establish levels at which such effects occurred.

Although the effects of chromium on the kidney have been investigated in several studies in occupationally exposed populations, in general, the evidence for an association between exposure to hexavalent chromium and kidney dysfunction or damage has been inconsistent (Verschoor *et al.*, 1988; Lindberg and Vesterberg, 1983; Franchini *et al.*, 1978; Satoh *et al.*, 1981a;b; Sassi, 1956; U.S. PHS, 1953; Franchini and Mutti, 1988; Mutti *et al.*, 1985; Littorin *et al.*, 1984; Vyskocil *et al.*, 1992; Herber *et al.*, 1989). There is no evidence that exposure to metallic chromium (chromium 0) or trivalent chromium is associated with renal dysfunction, based on the results of a small number of studies (Triebig *et al.*, 1987; Foa *et al.*, 1988).

The results of the better conducted and documented studies in which circulating lymphocytes from chromium-exposed workers have been examined for chromosomal aberrations, micronuclei, sister chromatid exchanges, and changes in the number of chromosomes have generally been negative (Fairhurst and Minty, 1989). Although the

frequency of "complications" and toxicosis was reported to be higher in pregnant women exposed to chromium compounds in two limited studies in Russia (Shmitova, 1978; 1980), no effects on semen quality or levels of several hormones in the serum were observed in a cross-sectional study in welders in Denmark (Bonde and Ernst, 1992).

Skin ulceration and allergic contact dermatitis have been reported in numerous case reports and epidemiological studies in occupationally exposed populations. The prevalence of chromium skin sensitization in the general population in North America has been estimated to be 1.6%, based on a response rate of 5.2% to potassium dichromate in dermatitis patients and assuming that dermatitis patients may comprise up to 25% of the general population (Paustenbach *et al.*, 1992). Bagdon and Hazen (1991) have determined that the threshold concentration for skin hypersensitivity reactions (i.e., 10% or less positive response to patch testing) to hexavalent chromium was 0.001% (10 mg/kg or mg/L), based on review of studies involving 301 challenge tests in human subjects. Trivalent chromium compounds are generally less potent skin sensitizers; the threshold for evoking hypersensitivity (i.e., $\leq 10\%$ positive response) was estimated to be 0.05% (500 mg/kg or mg/L), based on data obtained in 28 subjects exposed to the sulphate and nitrate salts (Bagdon and Hazen, 1991).

2.5.3 Ecotoxicology

This section focuses on studies in particularly sensitive nonhuman species or population groups. Data on the effects of specific chromium compounds are presented in the supporting documentation. Based on limited data, mammals are generally considered to be more sensitive to Cr(VI) than to Cr(III) for most toxicological endpoints examined; however, this is not always true for aquatic biota (Holdway, 1988; Janus and Krajnc, 1990). In fact, the available information indicates that Cr(III) resembles Al(III) in its mode of toxicity and is more toxic to fish than Cr(VI) (Holdway, 1988; Janus and Krajnc, 1990). The high deposition of Cr(III) in fish gills leads to tissue damage including hyperplasia, clubbing of lamellae and necrosis, and the impairment of the ability to osmoregulate and respire (Moore, 1991). By contrast, Cr(VI) rapidly passes through the gills and affects such organs as the liver, kidney, and spleen.

Information identified on the toxicity of chromium to aquatic biota includes data from acute and chronic studies in algae, invertebrates, and fish; the data base for terrestrial organisms includes studies of effects on soil microbial processes and growth of vascular plants. No relevant toxicological data were identified for sediment-dwelling biota, or for wild mammals, while information on the effects of chromium in birds was limited.

Based on reviews by the United States Environmental Protection Agency (U.S. EPA, 1985) and the Canadian Council of Resource and Environment Ministers (CCREM, 1987), *Daphnia* spp. appear to be particularly sensitive to chromium. The average number of young produced per adult *Ceriodaphnia reticulata* was reduced by about 30% following 7 days of exposure to 0.5 $\mu\text{g/L}$ of Cr (VI), while the decrease was 20% for *D. magna* exposed to 1.5 $\mu\text{g/L}$ of Cr(VI) for 14 days (Elnabarawy *et al.*, 1986).

Exposure of *D. magna* to 2.5 µg/L of Cr(VI) for 7 days caused a 28% reduction in juvenile survival and a 22% reduction in the number of young produced (Trabalka and Gehrs, 1977; U.S. EPA, 1985). Elnabarawy *et al.* (1986) reported a 20% death rate for adult *D. pulex* exposed to 1.5 µg/L of Cr(VI) for 14 days. The lowest reported median lethal concentration (LC₅₀) of Cr(III) for *Daphnia magna* was 6 µg/L (Janus and Krajnc, 1990).

Some aquatic microflora are also very sensitive to chromium. Less than 50% of the flagellated green algae, *Euglena gracilis*, survived when exposed to 1.0 µg/L of Cr(VI) for one hour (Yongue *et al.*, 1979). Concentrations of Cr(VI) as low as 2.0 µg/L are acutely toxic to the blue-green algae, *Microcystis aeruginosa* (Bringmann, 1975; Bringmann and Kuhn, 1978). The growth of *Chlorella reinhardii* was strongly inhibited (by over 75% compared to control) at concentrations of 10 µg/L of Cr(VI) following an 8-day exposure (Zarafonetis and Hampton, 1974). The growth rate of the estuarine diatom, *Thalassiosira pseudonana*, was reduced by 50% when exposed to 2.0 µg/L of Cr(VI) in water with low levels of sulphate, while photosynthesis, as measured by fluorescence yield, was also reduced by 50% when exposed to 1.0 µg/L of Cr(VI) (Riedel, 1984). The 4-day EC₅₀ (concentration that suppressed growth in 50% of the test population) was 3 µg/L of Cr(VI) for *Stephanodiscus hantzschii*, a diatom common in hard, eutrophic waters (Janus and Krajnc, 1990).

Available information indicates that fish are more sensitive to Cr(III) than to Cr(VI) (Holdway, 1988; Janus and Krajnc, 1990). The mean 96-h LC₅₀ for Cr(III) has been reported to be about four-fold lower than that for Cr(VI) in salmonid fish, with their reproductive cycles being particularly sensitive to Cr(III) (Holdway, 1988). Exposure of the eggs and spermatozoa of rainbow trout (*Oncorhynchus mykiss*) to 5 µg/L of Cr(III) reduced the fertilization rate by 60 to 70% (Billard and Roubaud, 1985). Mortality of swim-up fry of the Atlantic salmon (*Salmo salar* L.) was about 10% at 10 µg/L of Cr(VI) and 70% at 100 µg/L during a 68-day exposure (Grande and Anderson, 1983). The threshold concentration of Cr(VI) for avoidance/preference reactions in rainbow trout (*Oncorhynchus mykiss*) was reported as 28 µg/L (Anestis and Neufeld, 1986).

The results of short-term tests indicate that a variety of effects are induced in soil microbes (including changes in species abundance, respiration, nitrogen transformation, and enzyme activities) when the added concentrations are 25 to 100 µg/g (d.w.) of Cr(III) and 1 to 10 µg/g (d.w.) of Cr(VI), respectively (Chang and Broadbent, 1981; 1982; Tabatabai, 1977; Ross *et al.*, 1981; Drucker *et al.*, 1979). In long-term tests, Williams (1988) reported that addition of 10 µg/g (d.w.) of Cr(III) affected the arylsulphatase activity, while soil respiration was affected at 150 µg/g (d.w.), and phosphatase activity at 280 µg/g (d.w.).

Although both Cr(III) and Cr(VI) are equally available to plants grown in nutrient solutions (NRCC, 1976), the results of most studies indicate that Cr(VI) is consistently more toxic than Cr(III). When added to sandy soils, 5 µg/g (d.w.) of Cr(VI) induced iron chlorosis in oats, retarded stem development in tobacco, and inhibited the uptake of

micronutrients by soybean (Hunter and Vergnano, 1953; Soane and Saunder, 1959; Turner and Rust, 1971). Levels of 200 µg/g (d.w.) of Cr(III) in soils resulted in a significant (23 to 36%) reduction in the yields of grass, lettuce, and radish (Sykes *et al.*, 1981). Williams (1988) and Janus and Krajnc (1990) reported that levels of Cr(III) of 150 µg/g (d.w.) or more in soil can inhibit the growth of sensitive plant species depending on the nature of the soil.

Controlled studies on the toxicity of chromium to wild mammals and birds are very limited. Studies on laboratory mammals are presented in Subsection 2.5.1. Studies on domestic species of birds are scarce. Turkey hens fed 10 µg/g (w.w.) of Cr(III) in their diet produced significantly fewer eggs than controls; however, egg fertility and hatchability were unaffected (Frobish, 1980). No adverse effects on survival and growth were observed in male domestic chickens exposed to 100 µg/g (w.w.) of Cr(VI) in the diet for 32 days (Romoser *et al.*, 1961). In the only identified study on wild birds, common terns (*Sterna hirundo*) in Rhode Island (where high levels of chromium have been detected in soil, air, and surface water), no effects on growth, reproductive success, or clutch size were observed, although concentrations of uric acid in the blood at the most contaminated site were significantly higher than at other sites (Custer *et al.*, 1986). The elevation in uric acid levels (suggestive of an alteration of kidney function) may have been due to chromium in the diet [up to 7.6 µg/g (d.w.) in the main prey species (killifish) which is higher than the level in prey in uncontaminated sites]. Eisler (1986) and Outridge and Scheuhammer (1993) reviewed an unpublished study by S.D. Haseltine in which juvenile American black ducks (*Anas rubripes*) fed 10 µg/g (d.w.) of Cr(III) in the diet had increased concentrations of uric acid and reduced growth and survival. In adults administered 10 and 50 µg/g in the diet, there were no effects on survival, reproduction, and blood chemistry. These data were not available for critical review.

3.0 Assessment of "Toxic" under CEPA

3.1 CEPA 11(a) Environment

Although there is currently no domestic production of chromium ore, about 74 000 t (gross weight) of various chromium-containing materials were imported into Canada in 1991 for use primarily in the production of stainless and heat-resistant steels, refractory products such as bricks and mortars, and in pigments, metal finishing, leather tanning, and wood preservatives. Releases of chromium in large quantities (84 t to the atmosphere, >27 t to water, and >5000 t to land) from various anthropogenic sources in Canada have increased concentrations of chromium reported in Canadian air, water, soils, and sediments.

Chromium(III) can be released naturally in limited quantities into water by weathering and erosion of solid phases of Cr(III) found in rock and soil. Both Cr(III) and Cr(VI) are also released to the environment in Canada in anthropogenic wastes. Since Cr(III) forms highly insoluble oxides, hydroxides, and phosphates and is strongly adsorbed by particulate matter, Cr(III) can accumulate and persist in sediment and soil but its bioavailability is normally limited. Chromium(VI), on the other hand, is soluble and can persist in a bioavailable form in aerobic surface water and oxic soil pore water.

Information on the speciation of chromium in the environment is limited. Recent studies in Canada and elsewhere suggest that Cr(VI) is the dominant form of dissolved chromium in surface waters, although Cr(III) levels could be elevated in deep anoxic waters and in water receiving direct discharges of Cr(III)-containing wastes. Nearly all of the chromium in soils [excluding those contaminated with Cr(VI)] (Bartlett and James, 1988), buried sediments (Nriagu *et al.*, 1993), and biological tissues (Anderson, 1981; Nieboer and Jusys, 1988) is most likely present as Cr(III). Although mammals are considered to be more sensitive to Cr(VI) than Cr(III), available information suggests that fish are more sensitive to Cr(III). Due to differences in the bioavailability and toxicity of hexavalent and trivalent forms of chromium, their potential to cause harmful effects to organisms in Canada was evaluated separately.

Hexavalent Chromium. The lowest identified LOEL (lowest observed effect level) for dissolved Cr(VI) to freshwater organisms is a 7-day LOEL of 0.5 µg/L, for impaired reproduction in the daphnid, *Ceriodaphnia reticulata*. This value is similar to effect levels reported in chronic tests with *Daphnia magna* [1.5 to 2.5 µg of Cr(VI)/L; reduced survival and reproduction] and *Daphnia pulex* [1.5 µg of Cr(VI)/L; reduced survival]. The LOEL for *C. reticulata* [0.5 µg of Cr(VI)/L] was divided by a factor of 10, to account for potential differences in laboratory and field conditions as well as differences in sensitivity among species, to obtain an estimated effects threshold of 0.05 µg of Cr(VI)/L. Median concentrations of chromium reported recently in filtered surface water from Lake Erie and Lake Ontario were 0.27 µg/L and 0.77 µg/L, respectively, most of which was likely present as dissolved Cr(VI). These levels are 5 to 15 times greater than the estimated effects threshold for Cr(VI). Furthermore, mean (or median) concentrations of 4 to 7 µg/L of total chromium have recently been reported in

unfiltered samples of water from rivers and streams in British Columbia, Alberta, Ontario, and Quebec. Limited data on average amounts of chromium in filtered as opposed to unfiltered samples of river water indicate that from 10 to 60% of the chromium detected in unfiltered samples is typically in dissolved form and, based on studies of chromium speciation in lakes, most of this is likely present as Cr(VI). If it is assumed that only 10% of the chromium detected in Canadian rivers and streams is present in bioavailable form as dissolved Cr(VI), hexavalent chromium levels would be 8 to 14 times greater than the estimated effects threshold of 0.05 µg of Cr(VI)/L in many surface waters in these provinces. Therefore, aquatic organisms could be adversely affected by exposure to dissolved Cr(VI) in surface waters in many parts of Canada.

Adverse effects, including chlorosis and reduced growth and yield, were observed in several varieties of agricultural plants exposed to 5 µg/g (d.w.) of added Cr(VI) in soil. Furthermore, added concentrations of 1 to 10 µg/g (d.w.) of Cr(VI) adversely affected soil microbial processes such as respiration. Average concentrations of chromium in soils contaminated with dissolved Cr(VI) at some wood preservation plants in Manitoba and Atlantic Canada ranged from 243 to 1760 µg/g (d.w.). If it is assumed that background concentrations were approximately 50 µg/g (d.w.) of chromium (this overestimates background values reported in Manitoba), average concentrations of added Cr(VI) at these contaminated sites ranged from approximately 190 to 1700 µg/g (d.w.). Concentrations of added Cr(VI) in soils at wood preservation plants in Canada are therefore 38 to 340 times higher than levels of added Cr(VI) reported to harm some plants, and 19 to 1700 times higher than concentrations that can harm soil microbial communities. Therefore, soil microbial communities and some terrestrial plants could be adversely affected by exposure to Cr(VI) in Canadian soils near wood preservation plants.

Therefore, based on available data, dissolved and soluble forms of hexavalent chromium are entering or may enter the environment in a quantity or concentration or under conditions that are having or may have a harmful effect on the environment.

Trivalent Chromium. The mean 96-h LC₅₀ for Cr(III) was about four-fold lower than that for Cr(VI) in salmonid fish (Holdway, 1988). Exposure of *Oncorhynchus mykiss* to 5.0 µg/L of Cr(III) suppressed fertilization potential by 60 to 70% (Billard and Roubaud, 1985). There were no reliable data identified, however, on concentrations of Cr(III) in freshwaters of Canada, or on total chromium in waters where concentrations of Cr(III) would be expected to be elevated [e.g., deep anoxic lake water, or near outfalls from Canadian industries using Cr(III)]. Consequently, it is not possible to determine whether fish, which are particularly sensitive to trivalent chromium, could be adversely affected by exposure to Cr(III) in Canadian waters.

Benthic organisms are exposed to chromium [most likely Cr(III)], at concentrations as much as several hundred times greater than normal background levels, in sediments from the Canadian Great Lakes and their connecting channels, as well as

from Lake Simcoe, the Welland, St. Lawrence and Fraser Rivers, and Belledune and Dalhousje Harbours in New Brunswick. However, no toxicological data were identified that would permit estimation of effect thresholds to determine the significance of such exposure for these biota.

Although soil microbial populations and some vascular plants were reported to be adversely affected when exposed to Cr(III) in soil, data on the amounts and forms of chromium in contaminated Canadian soils were insufficient to determine whether terrestrial organisms are adversely affected by exposure to Cr(III) in such Canadian environments. Furthermore, due to the lack of acceptable toxicological data, it is not possible to determine whether Canadian birds or mammals are adversely affected by exposure to Cr(III) in their diet (probably the primary source of trivalent chromium).

Therefore, based on available data, it is not possible to determine whether trivalent forms of chromium are entering or may enter the environment in a quantity or concentration or under conditions that are having or may have a harmful effect on the environment.

3.2 CEPA 11(b) Environment on Which Human Life Depends

Chromium occurs primarily in particulate form in the atmosphere. Based on the low concentrations of chromium in the atmosphere (typically about 0.003 to 0.010 $\mu\text{g}/\text{m}^3$), it is unlikely that chromium will have any marked influence on the earth's solar radiation balance. Trivalent chromium is not oxidized by ozone and therefore cannot be linked to any ozone-depleting reactions in the atmosphere. As such, chromium compounds are not expected to contribute significantly to global warming or depletion of stratospheric ozone.

Therefore, based on available data, neither the hexavalent nor trivalent forms of chromium are considered to be entering the environment in a quantity or concentration or under conditions that constitute a danger to the environment on which human life depends.

3.3 CEPA 11(c) Human Life or Health

Based on available toxicological and epidemiological data, the potential effects of chromium and its compounds on human health vary considerably with valency. Therefore, to the extent possible, the species of chromium compounds commonly present in the environment (i.e., hexavalent and trivalent chromium) have been assessed separately with respect to whether they are "toxic" under Paragraph 11(c) of CEPA.

3.3.1 Population Exposure*

Estimates of the average total daily intake of chromium (on a body weight basis) for the general population in Canada are presented in Table 2. Based on these estimates, ingestion in food (primarily in the trivalent form) likely represents the principal route of chromium intake for all age groups, followed by drinking water, soil (particularly in infants and young children), and air. Average total daily intake from environmental media in Canada is estimated to be <1.6, 0.3 to 0.7, <1.5, <0.9, <0.5, and <0.4 µg/[kg(b.w.)·d] in non-breast-fed infants, breast-fed infants, toddlers, young children, teenagers, and adults, respectively. Cigarette smoking may increase total daily intake by 0.04 to 0.05 µg/[kg(b.w.)·d].

Intake may be elevated in populations residing in the vicinity of anthropogenic sources, although there are insufficient data regarding exposure to chromium in such populations to estimate overall intake. Sufficient data were available to quantify increased exposure in these areas only through the ingestion of contaminated soil and dust. Based on the range of mean concentrations of chromium (25 to 1760 µg/g) in soil or dust near a coal-powered generating station in Alberta (Van Voris *et al.*, 1985), a base metal smelter in New Brunswick (MacMillan, 1982), six wood treatment plants in Manitoba (Manitoba Environment and Workplace Safety and Health, 1989) and Atlantic Canada (Bamwoya *et al.*, 1991), and a chromate waste site in New Jersey (Lioy *et al.*, 1992), estimated intake in such soil and dust ranges from 0.007 to 8.8 µg/[kg(b.w.)·d], with the greatest intake in young children.

3.3.2 Effects

Hexavalent Chromium.** Based on the available data, carcinogenicity is considered to be the most sensitive endpoint for assessment of "toxic" under Paragraph 11(c) of CEPA. The weight of evidence for carcinogenicity has therefore been considered, based on the criteria developed for this purpose for the "Determination of 'Toxic' under Paragraph 11(c) of the *Canadian Environmental Protection Act*" (Environmental Health Directorate, 1992). Furthermore, the available data base for assessment of non-neoplastic effects of hexavalent chromium in both humans and experimental animals is limited.

* Due to the lack of identified data on the speciation of chromium in various environmental media, it was not possible to estimate the exposure of the general population to trivalent or hexavalent chromium separately. Therefore, estimated values refer to total chromium.

** Since, in the majority of the critical epidemiological studies, the specific hexavalent chromium compounds to which the study populations were exposed are not indicated, this assessment applies to the group of hexavalent chromium compounds as a whole, rather than to individual hexavalent chromium compounds. It is recognized, however, that the toxicity of these compounds may vary with their physical/chemical properties.

Table 2 Estimated Average Daily Intake of Chromium by the General Population in Canada

Medium	Estimated Daily Intake { $\mu\text{g}/([\text{kg}(\text{b.w.})\cdot\text{d}]$ }				
	0 to 0.5yr ^a	0.5 to 4 yr ^b	5 to 11yr ^c	12 to 19yr ^d	20 to 70yr ^e
Water ^f	0.03 to 0.5	0.02 to 0.3	0.01 to 0.1	0.007 to 0.1	0.006 to 0.09
Food ^g	<0.9(NBF) 0.03 to 0.04 (BF)	<1.0	<0.7	<0.4	<0.3
Air ^h	0.0009 to 0.003	0.001 to 0.003	0.001 to 0.004	0.001 to 0.003	0.001 to 0.003
Soil/Dirt ⁱ	0.2	0.2	0.06	0.02	0.01
Total	<1.6 (NBF) 0.3 to 0.7 (BF)	<1.5	<0.9	<0.05	<0.4
Tobacco Smoking ^j	—	—	—	0.05	0.04

- ^a Assumed to weigh 7 kg, breath 2 m³ of air per day, drink 0.75 L of water per day, and ingest 35 mg of soil per day (Environmental Health Directorate, 1992).
- ^b Assumed to weigh 13 kg, breath 5 m³ of air per day, drink 0.8 L of water per day, and ingest 50 mg of soil per day (Environmental Health Directorate, 1992).
- ^c Assumed to weigh 27 kg, breath 12 m³ of air per day, drink 0.9 L of water per day, and ingest 35 mg of soil per day (Environmental Health Directorate, 1992).
- ^d Assumed to weigh 57 kg, breath 21 m³ of air per day, drink 1.3 L of water per day, and ingest 20 mg of soil per day (Environmental Health Directorate, 1992).
- ^e Assumed to weigh 70 kg, breath 23 m³ of air per day, drink 1.5 L of water per day, and ingest 20 mg of soil per day (Environmental Health Directorate, 1992).
- ^f Based on a range of mean concentrations of chromium in drinking water of 0.3 to 4.3 $\mu\text{g}/\text{L}$ reported in several relatively recent surveys across Canada (Alberta Environment, 1988; Regional Municipality of Ottawa-Carleton, 1988; City of Winnipeg, 1989; Environment Canada, 1989b; c; d; e; Quebec Ministry of the Environment, 1989b; Health and Welfare Canada 1989; Greater Vancouver Regional District, 1989; Municipality of Metropolitan Toronto, 1989; Nova Scotia Department of Health and Fitness, 1989; MOE, 1989b; Saskatchewan Environment and Public Safety, 1989; City of Montreal, 1989; Mann Testing Laboratories, 1992; Minéraux Noranda Inc., 1992). This range is consistent with results in earlier national surveys (Meranger *et al.*, 1979; Neri *et al.*, 1975).
- ^g Based on concentrations of chromium in Canadian foodstuffs reported by Mann Testing Laboratories (1992) and food consumption patterns (Health and Welfare Canada, 1977). Data were not available to permit determination of speciation of chromium in food. Estimates of intake in breast milk in infants were based on the concentrations of chromium in breast milk reported in the better conducted studies in the United States and Finland reviewed by Kumpulainen (1992) of 0.25 to 0.39 ng/mL or $\mu\text{g}/\text{L}$ and a daily consumption of 0.75 L/day of breast milk (NBF = non-breast fed; BF = breast fed).
- ^h Based on a range of mean airborne concentrations of chromium of 0.003 to 0.009 $\mu\text{g}/\text{m}^3$ in the most recent (1987 to 1990) survey of Canadian cities (Dann, 1991).
- ⁱ Based on the mean concentration chromium levels in various Canadian soil types of 43 mg/kg (McKeague and Wolynetz, 1980). Similar values have been reported in Canadian soils (NRCC, 1976) and in street and house dust in other countries (Fergusson and Kim, 1991).
- ^j Based on estimated chromium content of mainstream cigarette smoke of 0.147 μg per Canadian cigarette (Labstat Incorporated, 1992) and 20 cigarettes smoked per day.

The carcinogenicity of hexavalent chromium has been investigated in a wide range of occupationally exposed populations. In the chromeplating, chromate production, and chromate pigment industries, exposure to hexavalent chromium compounds has been consistently demonstrated in numerous studies to be associated with increased mortality due to cancer of the respiratory system (see Subsection 2.5.2). There was considerable evidence in many of these studies of a relationship between some measure of duration or degree of exposure and mortality due to respiratory cancer. In addition, the introduction of measures to control the exposure of chromate production workers resulted in a reduction in mortality due to respiratory cancer.

Although there are many principally early epidemiological studies in which the association between deaths due to cancer and exposure to chromium in occupationally exposed populations has been investigated, most are considered quite limited, i.e., there was a lack of sub-cohort and dose-response analyses and inadequate account of potential confounding factors. For example, smoking habits were not taken into account in most of these investigations. In those studies in which such information was considered, however, smoking habits did not explain the observed excess in respiratory cancer (Siemiatycki *et al.*, 1988; Blair *et al.*, 1985). In addition, the observed excesses (generally two- to three-fold) were likely too large to be attributable solely to smoking. Moreover, where exposures to other substances were taken into account, they generally did not explain the observed excesses. However, despite all the limitations of these studies, many of which would have contributed to the obfuscation of an association between exposure to chromium and the development of cancer, an excess of lung cancer (although not always statistically significant) was observed among chromium VI-exposed workers in a large number of studies.

The carcinogenicity of hexavalent chromium in experimental animals has not been extensively investigated. No increase in the incidence of any type of tumours was reported in the few available, principally limited, studies in which hexavalent chromium was administered orally to animal species. The results of the few studies in which rats have been exposed to hexavalent chromium compounds by inhalation have generally been inconclusive; at most, hexavalent chromium has been weakly carcinogenic in rats. In addition, hexavalent chromium compounds have been consistently genotoxic in a variety of short-term *in vitro* and *in vivo* assays, although the results of studies of clastogenicity in peripheral lymphocytes of humans are inconclusive.

On the basis of its documented carcinogenicity in human populations, hexavalent chromium has been included in Group I ("Carcinogenic to Humans") of the classification scheme developed for the determination of "toxic" under Paragraph 11(c) of CEPA (Environmental Health Directorate, 1992). For such substances, where data permit, the estimated total daily intake or concentrations in relevant environmental media are compared to quantitative estimates of carcinogenic potency to characterize risk and provide guidance for further action (i.e., analysis of options to reduce exposure). Carcinogenic potency is expressed as the dose or concentration that induces a 5% increase in the incidence of or mortality due to relevant tumours (TD_{0.05}) (Environmental Health Directorate, 1992).

The data considered most relevant to the quantification of the cancer potency associated with exposure to hexavalent chromium in the general environment are those obtained in epidemiological studies in exposed populations. The use of results of epidemiological studies to quantitatively estimate cancer potency obviates the need for interspecies extrapolation. In addition, there are few adequate studies in which the carcinogenicity of hexavalent chromium in animal species has been investigated.

Although there has been an association between exposure to hexavalent chromium and respiratory cancer in numerous epidemiological investigations in occupationally exposed populations, in the majority of these studies, insufficient information on exposure was presented to characterize a quantitative exposure-response relationship. The study in which the most information on exposure was provided is that by Mancuso (1975), in which the mortality due to lung cancer as of 1974 was investigated in a cohort of 332 men employed at a chromate production plant between 1931 and 1937.* Although the cohort in this study was small, workers were classified into several categories of cumulative exposure to total chromium, and soluble (principally hexavalent) or insoluble (principally trivalent) chromium. In addition, the period of follow-up was sufficiently long to account for the latency period of development of lung cancer. However, mortality by age group was only reported for total chromium; such information is required for comparison with the general population. Therefore, an estimate for the carcinogenic potency has been derived based on exposure to total chromium.

It should be noted, however, that estimates of cumulative exposure were based on analysis of 137 samples from nine departments conducted in 1949. Since production was reported to have increased "tremendously" since the earlier years of operation, it is possible that concentrations to which workers were exposed during the period of 1931 to 1937 were overestimated. Conversely, however, in an earlier report of airborne concentrations in this plant in 1949 (which provided the basis for the exposure characterization presented by Mancuso), it was noted that levels before 1949 were likely greater than those measured in that year due to improvements in equipment and processes (Bourne and Yee, 1950). In addition, no information was available on the smoking habits of the cohort, or other possible confounding factors, such as exposure to other compounds.

A detailed description of the mathematical derivation of the constant concentration that corresponded to a 5% increase in mortality due to lung cancer ($TD_{0.05}$), based on the data reported by Mancuso (1975), is presented in the supporting documentation. The age-specific death rate for lung cancer was assumed to be a time-weighted quadratic function of exposure to chromium, which is additive to the death rate for the general population assumed not to be exposed to chromium. The increase in probability of death due to constant lifetime exposure to chromium has been determined, based on the assumption that there are no competing causes of death and exposure is constant for a

* A comprehensive study of chromium workers has recently been conducted in Baltimore. This study involved a large number of workers for whom extensive data on exposure are available for several years (Gibb, 1993). However, the results of this study were not available at the time of completion of this assessment.

period equal to the median survival time of 75 years. The $TD_{0.05}$ for inhaled chromium (total) was estimated to be $4.6 \mu\text{g}/\text{m}^3$. Calculated exposure/potency indices (EPIs) for the range of mean concentrations of chromium reported in ambient air in several cities across Canada (0.003 to $0.009 \mu\text{g}/\text{m}^3$) range from 6.5×10^{-4} to 2.0×10^{-3} .*

It should be noted, however, that while the exposure/potency indices were based on comparison of data on levels of total chromium in ambient air in Canada to the concentrations of total chromium to which the chromate production workers were exposed, the proportion of total chromium that is hexavalent in the two types of environments likely differs substantially.

An indirect estimate of the carcinogenic potency of hexavalent chromium may be derived from the study by Mancuso (1975). In an earlier study at the same chromate production plant, it was reported that the proportion of trivalent to hexavalent chromium present in most areas of the plant was about 6:1 or less (Bourne and Yee, 1950), although the number of workers in each area of the plant was not specified. Thus, the concentrations of hexavalent chromium may be estimated to be one seventh ($1/7$) of the reported concentrations of total chromium. Based on this assumption, the $TD_{0.05}$ for hexavalent chromium has been estimated to be $0.66 \mu\text{g}/\text{m}^3$. Although no information has been identified on the relative proportion of various species of chromium in ambient air in Canada, hexavalent chromium was reported to comprise 3 to 8% of the total chromium in a limited number of samples of ambient air in California (California Air Resources Board, 1985). Assuming that the hexavalent species comprises a similar proportion of total chromium in ambient air in Canada (i.e., 3 to 8%), concentrations of hexavalent chromium would be 0.00009 to $0.00072 \mu\text{g}/\text{m}^3$. The resulting exposure/potency index for hexavalent chromium is estimated to range from 1.4×10^{-4} to 1.1×10^{-3} .** Based solely on considerations of potential health effects, therefore, the priority for further action (i.e., analysis of options to reduce exposure to hexavalent chromium) is, therefore, considered to be moderate to high. It should be recognized, however, that these exposure/potency indices are extremely imprecise, as they were derived from estimates of carcinogenic potency based on the study by Mancuso (1975), which involved small numbers of workers, limited early data on exposure, and no control for potential confounding factors, such as smoking or concomitant exposure to other compounds. More appropriate estimates of the exposure/potency index for hexavalent chromium may be derived if additional information on the speciation of chromium in the atmosphere in Canada and the results of the ongoing epidemiological study of chromium workers in Baltimore are obtained, as is suggested in Section 4.0.

* Calculated $TD_{0.05}$ s and EPIs refer to the group of chromium compounds as a whole, since available data do not permit an estimate of the potency of individual chromium compounds.

** Calculated $TD_{0.05}$ s and EPIs refer to the group of hexavalent chromium compounds as a whole, since available data do not permit estimation of the carcinogenic potency of individual hexavalent chromium compounds.

Since hexavalent chromium* is classified as "Carcinogenic to Humans" (i.e., a substance for which there is considered to be some probability of harm for the critical effect at any level of exposure), it is concluded that it is entering the environment in a quantity or concentration or under conditions that may constitute a danger in Canada to human life or health.

This approach is consistent with the objective that exposure to substances classified as carcinogenic to humans should be reduced wherever possible and obviates the need to establish an arbitrary "de minimis" level of risk for determination of "toxic" under the Act.

In addition to the documented carcinogenicity of hexavalent chromium in occupationally exposed human populations, a proportion of the general population is also highly sensitive to the dermatological effects of hexavalent chromium. The prevalence of sensitization to hexavalent chromium in the general population in North America is estimated to be 1.6% (Paustenbach *et al.*, 1992).

Trivalent Chromium. For trivalent chromium, carcinogenicity is considered to be the most sensitive endpoint for assessment of "toxic" under Paragraph 11(c) of CEPA. Only a few, principally limited, epidemiological studies of cancer mortality in workers exposed to chromium primarily in the trivalent form have been identified; an increase in mortality due to cancer has not been consistently observed in these studies. Available data are insufficient, therefore, to assess the carcinogenicity of trivalent chromium in human populations.

No increase in the incidence of tumours at any site has been observed in the few available earlier inadequate studies in which rats or mice were administered trivalent chromium in drinking water or the diet. In studies in which rats or mice were exposed to mixed chromium (trivalent and hexavalent) compounds by inhalation, there were no increases in the incidence of tumours that were ascribed to trivalent chromium. In studies reviewed by the International Agency for Research on Cancer (IARC, 1990) in which experimental animals were administered trivalent chromium compounds by routes other than ingestion or inhalation, there was no evidence of carcinogenicity, although many of these studies were considered to be limited. Trivalent chromium compounds have not been genotoxic in the majority of short-term *in vitro* and *in vivo* tests conducted to date, although there is some evidence that trivalent chromium is genotoxic, but has very limited ability to cross intact cellular membranes.

* This assessment of whether hexavalent chromium is "toxic" to human life or health is considered to apply to the group of hexavalent chromium compounds as a whole, since available data do not permit an assessment of the risks posed by individual hexavalent chromium compounds to human health

On the basis of the inadequate data base on carcinogenicity in studies in experimental animals and the limited data base in exposed human populations, trivalent chromium is classified in Group VI ("Unclassifiable with Respect to Carcinogenicity in Humans") of the classification scheme developed for the determination of "toxic" under Paragraph 11(c) of CEPA (Environmental Health Directorate, 1992).

Available data on the non-neoplastic effects of trivalent chromium in epidemiological and toxicological studies conducted to date are also limited. Effects on the respiratory system have been observed in some cross-sectional studies of workers exposed to trivalent chromium, although data on exposure were inadequate to establish effect levels. Although a segment of the general population may be sensitive to the dermatological effects of trivalent chromium, it has been estimated that skin hypersensitivity is likely to occur in no more than 10% of the population exposed to 500 mg/kg or mg/L of trivalent chromium in environmental media (Bagdon and Hazen, 1991).

Alterations in hematological and biochemical parameters have been reported in one study in which mice were administered trivalent chromium in the drinking water for one year (Maruyama, 1982); however, this study is inadequate as a basis for derivation of effect levels, since no histopathological examinations were conducted. Effects on pulmonary macrophages have been reported in rabbits exposed to 0.6 mg/m³ of trivalent chromium by inhalation for up to 21 weeks (Johansson *et al.*, 1986a; b; 1987). However, histopathological examination in this investigation was limited to the lungs.

Trivalent chromium is considered to be an essential trace element in human nutrition. The only identified recommended daily intake for adults is between 50 and 200 µg (NAS, 1989); recommended daily intakes for other age groups are 10 to 40, 20 to 60, 20 to 80, and 30 to 120 µg for 0 to 6 months, 6 months to 1 year, 1 to 3 years, and 3 to 6 years of age, respectively (NAS, 1980). The average daily intake of total chromium from food (the principal source of trivalent chromium) for various age groups of the general population in Canada is estimated to range from 0.03 to <0.9, <1.0, <0.7, <0.4, and <0.3 µg/[kg(b.w.)·d] for infants, toddlers, young children, teenagers, and adults (Table 2), respectively, or 0.2 to <6.3, <13, <19, <23, and <21 µg/day. Therefore, the daily intake of chromium by the various age groups of the general population does not exceed the only identified recommended intakes (NAS, 1980; 1989).

Since trivalent chromium* is considered to be an essential nutrient for which estimated average daily intake does not exceed the only identified recommended daily intake, and since levels demonstrated to cause effects in limited toxicological studies in experimental animals or clinical dermatological studies in humans greatly exceed those reported in the general environment in Canada, it has been concluded that trivalent chromium is not entering the environment in a quantity or concentration or under conditions that constitute a danger in Canada to human life or health.

3.4 Conclusions

Based on these considerations, it has been concluded that dissolved and soluble forms of hexavalent chromium are entering or may enter the environment in a quantity or concentration or under conditions that are having or may have a harmful effect on the environment, while there are insufficient data to determine if trivalent forms of chromium are entering or may enter the environment in a quantity or concentration or under conditions that are having or may have a harmful effect on the environment. It has been concluded that neither hexavalent nor trivalent forms of chromium are entering or may enter the environment in a quantity or concentration or under conditions that constitute or that may constitute a danger to the environment on which human life depends. It has also been concluded that the group of hexavalent chromium compounds as a whole is entering the environment in a quantity or concentration or under conditions that may constitute a danger in Canada to human life or health, while the group of trivalent chromium compounds as a whole is not entering the environment in a quantity or concentration or under conditions that may constitute a danger in Canada to human life or health.

* This assessment of whether trivalent chromium is "toxic" to human life or health is considered to apply to the group of trivalent chromium compounds as a whole, since available data do not permit an assessment of the risks posed by individual trivalent compounds to human health.

4.0 Recommendations for Research and Evaluation

Acquisition of data in the following areas would permit a more complete assessment of the risks to the health of the general population and to the environment associated with exposure to chromium.

- 1) Additional information on the forms and environmental fate of chromium [particularly Cr(III)] being released by pertinent industrial operations, such as leather tanning, metal finishing, cooling towers, steel, stone and clay production, primary and secondary smelters, and chemical and power plants.
- 2) Information on the concentrations, forms, and bioavailability of both trivalent and hexavalent chromium in Canadian air, water, soils, and sediments, particularly near industrial sources.
- 3) Data on the effects of chromium [especially Cr(III)] associated with sediment on benthic communities in Canada, and on the effects of chromium in amphibians and forest communities.
- 4) Additional data on concentrations of chromium in organisms consumed by wildlife, particularly in areas where sediments contain large amounts of chromium. Studies of effects on wildlife species in these areas are also desirable.
- 5) Studies on the potential carcinogenicity of chromium compounds in fish.
- 6) In order to better characterize the risk of cancer associated with exposure to chromium in the general environment, it would be desirable to recalculate the estimate of potency, based on the results of an ongoing epidemiological study of chromium workers in Baltimore, once the results are available.

On the basis of the available information, which indicates that Cr(III) is the dominant form of chromium in most environmental media, and because there were insufficient data to conclude whether trivalent chromium and its compounds have a harmful effect on the environment, the priority for the preceding recommendations is considered to be high.

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