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Draft Assessment

Aluminium-containing Substances Group

Environment and Climate Change Canada
Health Canada

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Canada

Synopsis

Pursuant to section 68 of the *Canadian Environmental Protection Act, 1999* (CEPA), the Minister of the Environment and the Minister of Health have conducted an assessment of 55 substances referred to collectively as the Aluminium[1]-containing Substances Group. The potential for cumulative effects was considered in this assessment by examining cumulative exposures to total aluminium.

There are both natural and anthropogenic sources of aluminium exposure to humans and the environment. Natural sources of aluminium include weathering and biogeochemical processes, which, in combination with deposition processes, contribute to a complex aluminium cycle in the environment. Anthropogenic sources include the production of aluminium (for example, smelting), cement production, metal mining, electric power generation, pulp and paper manufacturing, and the use of products and manufactured items containing aluminium compounds.

According to information submitted in response to CEPA section 71 surveys, of the 54 substances surveyed, 40 substances in the group were manufactured or imported above the reporting threshold of 0.1 tonnes in 2011 or 2015. Results of the surveys indicated that 8 of the 54 surveyed substances were manufactured in Canada above reporting thresholds and that 37 of the 54 surveyed substances were imported into Canada above reporting thresholds. Of the 40 substances manufactured or imported above the reporting threshold of 0.1 tonnes, 12 were manufactured or imported in quantities exceeding 1000 tonnes. These substances are used in a wide variety of products and applications including in arts, crafts and hobby materials; automotive care products; building and construction materials; cleaning products; food packaging; ink, toner, and colourants; self-care products (cosmetics, natural health products, and non-prescription drugs); paints and coatings; pest control products; plastics; textiles; and other industrial and commercial uses.

Substances in the Aluminium-containing Substances Group have the potential to dissolve, dissociate, or degrade through various transformation pathways and therefore potentially contribute to exposures to total aluminium. Therefore, total aluminium concentrations, modelled or measured, were used as a surrogate for the potential exposure from the 55 substances in the group. Ecological hazards were characterized accordingly in order to evaluate the potential for harm from exposure to total aluminium.

The ecological exposure assessment focuses on sectors with the highest commercial activity involving substances in the group as well as those with the largest releases of aluminium reported to the National Pollutant Release Inventory. Specifically, exposure scenarios were developed for primary aluminium manufacturing, cement manufacturing, metal mining, electric power generation, and pulp and paper manufacturing. Predicted

[1] The international spelling of “aluminium” is used throughout, except when referring to a name corresponding to the Chemical Abstracts Service Registry Number (CAS RN) (which, as property of the American Chemical Society, generally uses the American spelling “aluminum”) or to other regulatory list names that use American spelling.

environmental concentrations (PECs) were derived for each of these sectors, using monitoring data in the receiving environment, monitoring data in effluents, or industrial emission factors. Canadian long-term surface water quality monitoring data associated with land use classifications and available data on concentrations of bioavailable aluminium in soil were also used to characterize exposure.

The ecological hazard assessment for the aquatic compartment considers total concentrations of aluminium and the toxicity modifying factors such as pH, water hardness, and dissolved organic carbon. Predicted no effect concentrations (PNECs) were generated in alignment with the Federal Water Quality Guidelines for Aluminium. For soils, a PNEC was derived from published calcium chloride extractable aluminium thresholds. These accounted for bioavailability in soil; and on the basis of the data available, were considered to be protective of both terrestrial plants and soil invertebrates.

Aluminium is considered to be persistent in the environment, as are all elements, but may change speciation and cycle between environmental compartments. Although there are certain tolerant and hyper-accumulating plant species, aluminium is not generally considered to be bioaccumulative.

The analysis of aquatic risk quotients for each sector showed that PECs infrequently exceeded PNECs, suggesting a lower potential to cause ecological harm. For the soil compartment, the PNEC was compared with corresponding exposure data as a function of pH. In the range of pH values where anthropogenic releases of substances in the group would occur, the soil PNEC was not exceeded. For the sediment compartment, based on the qualitative lines of evidence considered, the 55 aluminium-containing substances were found to have a low potential to cause ecological harm.

Considering all available lines of evidence presented in this draft assessment, there is low risk of harm to the environment from the 55 aluminium-containing substances. It is proposed to conclude that the 55 aluminium-containing substances do not meet the criteria under paragraphs 64(*a*) or (*b*) of CEPA as they are not entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity or that constitute or may constitute a danger to the environment on which life depends.

Canadians may be exposed to substances in the Aluminium-containing Substances Group through environmental media (soil, house dust, and air), food, and drinking water. Traditional, subsistence, or country foods may be a source of aluminium exposure for certain Indigenous communities in Canada. People living near industrial facilities, such as primary aluminium smelters, may be exposed to elevated concentrations of aluminium from point source emissions. In addition, Canadians are exposed to aluminium from a variety of products and manufactured items available to consumers.

The systemic exposure of the general Canadian population over the age of 3 to substances in the Aluminium-containing Substances Group was characterized using nationally representative biomonitoring data. Aluminium was measured in biobanked samples of whole blood from Cycle 2 of the Canadian Health Measures Survey (CHMS). Aluminium content in whole blood samples provides a biologically relevant, integrated measure of systemic exposure that may occur across multiple routes (for example, oral ingestion, dermal contact, and inhalation) and sources (for example, natural and anthropogenic, environmental media, diet, and frequent or daily-use products). Aluminium levels were below the method reporting limit of 8 μg/L in 97.1% of the Canadian population (age group 3 to 79). For children under 3 years old, biomonitoring data from small-scale studies and intake estimates from environmental media, food, and drinking water were considered to characterize risk. Dietary intake estimates for certain Indigenous communities were also considered to characterize risk.

Inhalation exposure scenarios from the use of products available to consumers and ambient air concentrations, including in proximity to point sources of releases, were quantified separately in order to assess the potential risk of portal of entry effects in the lungs. Substances in the Aluminium-containing Substances Group are found in a range of aerosol, trigger spray, and loose powder products, the uses of which may result in inhalation exposure. These products include self-care products (that is, cosmetics, natural health products, and non-prescription drugs), paints and coatings, do-it-yourself products (for example, cement products, tile grout), and cleaning products. In addition, aluminum hydroxychloride (CAS RN[2] 1327-41-9) and aluminum chlorohydrate (CAS RN 12042-91-0) are used in aerosol and powdered antiperspirant and deodorant products.

Several international organizations have established health-based guidance values for aluminium (for example, tolerable weekly intakes), which were established on the basis of neurological, neurodevelopmental, and reproductive effects. Thus, to characterize human health risk, a whole blood biomonitoring equivalent (BE) was derived for daily intake levels associated with the provisional tolerable weekly intake (PTWI) established on the basis of a critical endpoint that was identified from a developmental and chronic neurotoxicity study by the Joint Food and Agriculture Organization/World Health Organization Expert Committee on Food Additives (JECFA). With respect to inhalation exposure, a no-observed adverse effect concentration (NOAEC) from a worker study was identified as a route-specific endpoint for the Aluminium-containing Substances Group. Additionally, repeated inhalation of aluminium chlorohydrate generates lung effects that are not observed after inhalation of other aluminium-containing substances. As a result, granulomatous pneumonia was selected as a route-specific critical health effect for aluminum hydroxychloride and aluminum chlorohydrate.

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Median and 95th percentile concentrations of total aluminium in whole blood from the CHMS were lower than the derived BE value. Average plasma aluminium concentrations in infants under 3 years old from small-scale biomonitoring studies were also lower than the derived BE value. Furthermore, intake estimates from environmental media, food, and drinking water for children under 3 years old as well as intake estimates from the consumption of country foods for certain Indigenous communities were lower than the daily intake level associated with the JECFA PTWI. Therefore, systemic exposure to the aluminium-containing substances is considered to be of low concern to the health of Canadians at current levels of exposure. In addition, the resulting margins of exposure estimated for inhalation exposure and the NOAEC for 53 of the 55 aluminium-containing substances were considered adequate to address uncertainties in the available health effects and exposure data used to characterized risk. Margins of exposure between levels of inhalation exposure from the use of aerosol antiperspirants and aerosol foot deodorant spray and the critical health effect for aluminum hydroxychloride (CAS RN 1327-41-9) and aluminum chlorohydrate (CAS RN 12042-91-0) were considered potentially inadequate to address uncertainties in the available health effects and exposure data used to characterize risk.

The human health assessment took into consideration those groups of individuals within the Canadian population who, due to greater susceptibility or greater exposure, may be more vulnerable to experiencing adverse health effects. For instance, age-specific exposures are routinely estimated and developmental and reproductive toxicity studies are evaluated for potential adverse health effects. Human biomonitoring data were available for infants, children and pregnant women and pregnant people. These subpopulations were taken into account in the risk assessment outcomes of aluminum-containing substances. In addition, exposure from consuming traditional, subsistence or country foods for certain Indigenous communities and exposure from outdoor air for people living near sources of release were examined.

Considering all the information presented in this draft assessment, it is proposed to conclude that aluminum hydroxychloride (CAS RN 1327-41-9) and aluminum chlorohydrate (CAS RN 12042-91-0) meet the criteria under paragraph 64(*c*) of CEPA as they are entering or may enter the environment in a quantity or concentration or under conditions that constitute or may constitute a danger in Canada to human life or health.

Considering all the information presented in this draft assessment, it is proposed to conclude that 53 of the 55 aluminium-containing substances do not meet the criteria under paragraph 64(*c*) of CEPA as they are not entering the environment in a quantity or concentration or under conditions that constitute or may constitute a danger in Canada to human life or health.

It is therefore proposed to conclude that aluminum hydroxychloride (CAS RN 1327-41-9) and aluminum chlorohydrate (CAS RN 12042-91-0) meet one or more of the criteria set out in section 64 of CEPA. It is proposed to conclude that the remaining 53 aluminium-containing substances do not meet any of the criteria set out in section 64 of

CEPA. It is also proposed to conclude that aluminum hydroxychloride and aluminum chlorohydrate meet the persistence criteria but not the bioaccumulation criteria as set out in the *Persistence and Bioaccumulation Regulations* of CEPA.

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List of Tables and Figures

1. Introduction

Pursuant to section 68 of the *Canadian Environmental Protection Act, 1999* (CEPA) (Canada 1999), the Minister of the Environment and the Minister of Health have conducted an assessment of a group of 55 substances (identified in Appendix A), referred to collectively as the Aluminium[3]-containing Substances Group, to determine whether these substances present or may present a risk to the environment or to human health. Forty-seven substances in this group were identified as priorities for assessment as they met categorization criteria, or were prioritized through other mechanisms (ECCC, HC [modified 2017]). Eight additional substances were identified for further consideration following prioritization of the Revised In Commerce List (R-ICL)[4] (ECCC, HC [modified 2017]; Health Canada [modified 2017]).

Health Canada and Environment Canada had previously published a Second Priority Substance List (PSL2) Assessment on three aluminium salts (aluminum chloride, aluminum nitrate, and aluminum sulphate) (EC, HC 2010). This assessment builds upon the PSL2 assessment and expands the scope to include additional substances. Other aluminium compounds present on the *Domestic Substances List* (DSL) have been addressed in separate reports (EC, HC 2010, 2013, 2016).

This assessment only considers the effects associated with aluminium and does not address other elements or moieties that may be present in the Aluminium-containing Substances Group (such as magnesium, fluoride, zirconium, or the organic components of organometallics and organic-metal salts). Some of these other elements or moieties have been addressed through previous assessments conducted as part of the Priority Substances List (PSL) program under CEPA or may be addressed via other initiatives of the Chemicals Management Plan (CMP). Engineered nanomaterials containing aluminium (1 nm to 100 nm) that may be present in environmental media or products are not explicitly considered in the exposure scenarios of this assessment, but measured concentrations of aluminium in the environment or human biomonitoring data could include aluminium from these sources. Similarly, this assessment does not explicitly consider ecological or health effects associated with nanomaterials containing aluminium. Aluminium is an adjuvant in certain vaccines and a main component in some non-prescription drugs used to neutralize gastric acid in heartburn or inflammation of the upper gastric tract (antacids). These uses are not considered in this assessment.

[3] The international spelling of “aluminium” is used throughout, except when referring to a CAS RN name (which, as property of the American Chemical Society, generally uses the American spelling “aluminum”) or to other regulatory list names that use American spelling.

[4] The Revised In Commerce List (R-ICL) is an administrative list of substances that are potentially used in products regulated under the *Food and Drugs Act* and that were in Canadian commerce between January 1, 1987, and September 13, 2001. The Government of Canada has prioritized these substances and is addressing them for their potential impact on human health and the environment in order to risk manage the substances, if required.

The ecological assessment for this group will focus on releases of aluminium related to the manufacture, import, and use of the 55 aluminium-containing substances, as well as incidental releases of aluminium. Environmental monitoring in the aquatic compartment, or predictive modelling where necessary, in conjunction with recently developed Federal Water Quality Guidelines serve as the primary lines of evidence for evaluating the potential for exposure and hazard of the substances in this group (ECCC 2022a).

Significant impacts of aluminium on freshwater ecosystems have been observed for decades as a consequence of acidic precipitation mobilizing naturally occurring metals, with ongoing issues particularly in areas that are part of the Canadian Precambrian Shield (Gensemer and Playle 1999; Wilson 2012; Adams et al. 2018; Canada [modified 2018a]; Rotteveel and Stirling 2020). However, this ecological assessment focuses on addressing potential concerns from industrial activities involving the 55 aluminium-containing substances. The potential impacts of environmental acidification are complex, including those involving the solubilization, bioavailaibility, and ecotoxicity of ambient aluminium. These impacts and the efforts to address them have been described elsewhere (Environment Canada 2004; Canada [modified 2013a], [modified 2013b], [modified 2018a]).

The human health risks of systemic effects were characterized using the Biomonitoring-based Approach 2 (Health Canada [modified 2016]), which compares human biomonitoring data (exposure) against biomonitoring guidance values (health effects) that are consistent with available health-based guidance values, such as biomonitoring equivalents (BEs), to identify whether substances are of low concern to human health. Additional exposure estimates were considered in order characterize the human health risks of systemic effects in groups of individuals not represented in the population level biomonitoring data. In addition, a route-specific approach was used to characterize portal of entry effects from the inhalation route of exposure for substances in the Aluminium-containing Substances Group.

This draft assessment takes into consideration information on chemical properties, environmental fate, hazards, uses, and exposures, including additional information submitted by stakeholders. Relevant data were identified up to July 2021. Empirical data from key studies as well as results from models were used to reach proposed conclusions. When available and relevant, information presented in assessments from other jurisdictions was considered.

This draft assessment was prepared by staff in the CEPA Risk Assessment Program at Health Canada and Environment and Climate Change Canada and incorporates input from other programs within these departments. The ecological and human health portions of this assessment have also undergone external review. Comments on the technical portions relevant to the environment were received from Mr. Geoff Granville (GCGranville Consulting Corp.), Dr. Claude Fortin (Institut national de la recherche scientifique), Dr. Scott Smith (Wilfrid Laurier University), the Technical Assessment and Standards Development Branch of the Ontario Ministry of Environment, Conservation and Parks, and the Water Protection & Sustainability Branch of the British Columbia

Ministry of Environment & Climate Change Strategy. Comments on the technical portions relevant to human health were received from Tetra Tech. In addition, the health portion of this assessment is based on the Biomonitoring-based Approach 2 Science Approach Document (SciAD) (published December 9, 2016), which was externally peer-reviewed and subject to a 60-day public comment period. While external comments were taken into consideration, the final content and outcome of the assessment remain the responsibility of Health Canada and Environment and Climate Change Canada.

Assessment focus on information critical to determining whether substances meet the criteria as set out in section 64 of CEPA by considering scientific information including information, if available, on subpopulations who may have greater susceptibility or greater exposure, vulnerable environments and cumulative effects[5], and by incorporating a weight of evidence approach and precaution[6]. This draft assessment presents the critical information and considerations on which the proposed conclusions are based.

2. Substance identity

The 55 aluminium-containing substances belong to various chemical categories including inorganic compounds, organic-metal salts, organometallic compounds, and unknown or variable composition, complex reaction products, or biological materials (UVCBs). The CAS RNs,[7] DSL names, and common names of the individual substances in the Aluminium-containing Substances Group are presented in Table A-1, Appendix A.

3. Physical and chemical properties

Aluminium is a metal belonging to Group 13 of the periodic table. Only the trivalent oxidation state of aluminium is relevant in the natural environment (Wilson 2012). Aluminium forms Al^{3+} ions by losing its valence p-electron and two valence s-electrons

[5] The consideration of cumulative effects under CEPA may involve an analysis, characterization and possible quantification of the combined risks to health or the environment from exposure to multiple chemicals.

[6] A determination of whether one or more of the criteria of section 64 of CEPA are met is based upon an assessment of potential risks to the environment and/or to human health associated with exposures in the general environment. For humans, this includes, but is not limited to, exposures from ambient and indoor air, drinking water, foodstuffs, and products available to consumers. A conclusion under CEPA is not relevant to, nor does it preclude, an assessment against the hazard criteria specified in the *Hazardous Products Regulations*, which are part of the regulatory framework for the Workplace Hazardous Materials Information System for products intended for workplace use. Similarly, a conclusion based on the criteria contained in section 64 of CEPA does not preclude actions being taken under other sections of CEPA or other acts.

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and may act as a substitute for silicon in ionic solids to define many minerals (Miessler et al. 2014). Aluminium oxides occur in various polymorphs, hydrated species, and hydroxides (Cotton and Wilkinson 1999). At environmentally relevant concentrations, aluminium forms water-soluble compounds with ions such as fluoride, sulfate, and organic chelators such as fulvic and humic acids (US EPA 2018).

The water solubilities of substances in the group range from very low to fully soluble. For example, reported water solubility values range from qualitatively insoluble to "greater than 10^6 mg/L." Vapour pressures are negligible for most substances, particularly where reported at physiologically and environmentally relevant temperatures. Water solubility and vapour pressure are less relevant for the organo-aluminium compounds, as these tend to be pyrophoric (may spontaneously ignite in air) and highly water-reactive (Sleppy 2007; Krause et al. 2012). A summary of available physical and chemical property data for substances in the group is presented in Appendix B.

4. Sources and uses

4.1 Natural sources

Aluminium is the most abundant metal and third most abundant element in the lithosphere, behind only oxygen and silicon (Haynes 2016). Aluminium is mostly associated with metamorphic and igneous rocks, mineral and clay deposits, and clay minerals in weathered soils. Through weathering and biogeochemical processes, small fractions of the total aluminium in the lithosphere enter a complex aluminium cycle in the environment (Driscoll and Postek 1996). Atmospheric deposition of aluminium is not a major natural source but may be important for isolated surface waters (Driscoll et al. 1994). The aluminium cycle will be discussed further in the environmental fate section.

Of the 55 aluminium-containing substances in the group, few are expected to have natural sources in the environment. Most of the substances are produced as downstream products of non-metallurgical aluminium hydroxide (that is, from the fraction of refined bauxite ore that is not used for smelting to aluminium metal) or from other sources (CEH 2021).

4.2 Anthropogenic sources

4.2.1 Aluminium production

Bauxite ore, the principal raw material used for aluminium production, is not produced in Canada. Rather, it is imported—for example, in quantities of approximately 3.7 million tonnes in 2015 (NRCan 2016). Bauxite ore is then refined to metallurgical alumina for smelting to aluminium metal, or to commodity and specialty aluminas for a variety of uses, including the production of other aluminium compounds (NRCan 2016; CEH 2021). As of 2016, there is one facility in Canada that produces metallurgical and non-

metallurgical alumina, and ten primary aluminium smelters. Nine aluminium smelters are located in Quebec and one in British Columbia (NRCan 2016).

4.2.2 Manufacture and imports

Of the 55 substances included in this assessment, 54 were included in surveys issued pursuant to section 71 of CEPA for the reporting years 2011 or 2015 (Canada 2012; Canada 2017b). Responses were received for 40 of the 54 substances surveyed (Table C-1, Appendix C). According to information submitted in response to the surveys, 8 of the 54 surveyed substances were manufactured in Canada and 37 of the 54 surveyed substances were imported to Canada above the reporting threshold of 0.1 tonnes. Of the 40 substances for which responses were received, 12 were manufactured or imported in quantities greater than 1000 tonnes. No manufacture or import activities above the reporting threshold of 0.1 tonnes were reported for the other 14 substances that were surveyed (Table C-2, Appendix C).[8] Aluminum hydroxide (CAS RN 21645-51-2) was the only substance in the current group that was prioritized for assessment and not surveyed under section 71 of CEPA for the reporting years of 2011 or 2015.

International trade data were also collected for substances in the Aluminium-containing Substances Group. Harmonized System (HS) codes relevant to 20 substances were identified for 2010 to 2013, and the annual import data are presented in Table C-3, Appendix C. The international trade data also indicated that aluminum hydroxide (HS code 2818.30.0000), not included in the surveys mentioned above, was imported in quantities greater than 1000 tonnes (CBSA 2016).

4.3 Uses

The uses or activities associated with the Aluminium-containing Substances Group on the basis of function codes submitted in response to CEPA section 71 surveys are presented in Table C-5 (Appendix C) (Canada 2012, 2017b). While approximately 100 unique sector and function codes were reported, the largest quantity uses and activities were aluminium production and cement manufacturing (Environment Canada 2013; ECCC 2017). These two activities represent approximately two-thirds and one-third of the overall reported quantities, respectively. However, when adjusted for the approximate percentage of aluminium in each substance, quantities related to aluminium production increase to approximately 90% of the overall reported quantities, with quantities related to cement manufacturing dropping to about 5%. All other uses and activities cumulatively account for less than 5% of reported overall aluminium-normalized quantities, although they still represent about 400 000 tonnes.

[8] Values reflect quantities reported in response to a survey conducted under CEPA section 71 (Canada 2012; Canada 2017b). See survey for specific inclusions and exclusions (schedules 2 and 3).

Other activities involving substances in the group with the largest associated reported quantities were petroleum production, refractory materials, adhesives and sealants, intermediates, lubricant additives, solids separation agents, abrasives, process regulators, fillers and flame retardants, laboratory substances, pigments, adsorbents and absorbents, anti-adhesive agents, catalysts, plastic films, and beer filtration (Environment Canada 2013; ECCC 2017). All additional reported uses beyond those listed here were either notified as confidential business information (CBI) or were associated with commercial quantities of less than 1000 tonnes per year. Substance-specific details on uses and sectors associated with the largest quantities according to information submitted in response to CEPA section 71 surveys are provided in Appendix C (Table C-5, Table C-6).

Further literature searches, market research, and international trade data generally corroborate the survey results presented above. However, they also suggest that Canadian consumption for filler and flame retardant, pulp and paper, water treatment, and pigment coating applications has grown significantly as compared to survey data reported for 2011 (Darragh and Ertell 2003; Helmboldt et al. 2012; Hudson et al. 2012; CBSA 2016; CEH 2021). Additionally, literature searches suggest the substances in the Aluminium-containing Substances Group may be present in a wide variety of products available to consumers including self-care products (that is, cosmetics, natural health products, and non-prescription drugs), paints and coatings, do-it-yourself (DIY) products (for example, cement products, tile grout), and cleaning products (see section 8.2.2). Additional uses of substances in the group are identified in Table 4-1.

In Canada, aluminium may be present in foods through the use of aluminum-containing food additives; as components in incidental additives used in food processing establishments; and as components in the manufacture of some food packaging materials (personal communication, email from the Food Directorate [FD], Health Canada, to the Existing Substances Risk Assessment Bureau [ESRAB], Health Canada, dated October 2, 2018, and March 31, 2022; unreferenced). Additionally, some substances in the Aluminium-containing Substances Group are present in registered pest control products in Canada as formulants (personal communication, email from the Pest Management Regulatory Agency [PMRA], Health Canada, to the ESRAB, Health Canada, dated June 18, 2018; unreferenced; Health Canada 2010).

Substances within the Aluminium-containing Substances Group are present in cosmetics according to notifications submitted under the *Cosmetic Regulations* (personal communication, emails from the Consumer and Hazardous Product Safety Directorate [CHPSD], Health Canada, to the ESRAB, Health Canada, dated June 25, 2018; unreferenced). The substances in this group are also present as medicinal or non-medicinal ingredients in disinfectants, human or veterinary drug products as well as natural health products (personal communication, email from the Therapeutic Products Directorate [TPD], Health Canada, to the ESRAB, Health Canada, dated June 11, 2018; unreferenced; DPD [modified 2022]; LNHPD [modified 2022]; NHPID [modified 2022]; personal communication, email from the Natural and Non-prescription Health Products Directorate [NNHPD], Health Canada, to the ESRAB, Health Canada, dated June 12,

2018; unreferenced). Some of the substances in the Aluminium-containing Substances Group are used as antiperspirant ingredients in self-care products available to consumers. Aluminum hydroxychloride (CAS RN 1327-41-9), aluminum chlorohydrate (CAS RN 12042-91-0), and aluminum zirconium complexes (CAS RNs 57158-29-9 and 90604-80-1) are identified as being restricted or prohibited for use in aerosol dispensers or prohibited in combination with other aluminium compounds on the Cosmetic Ingredient Hotlist under entries for aluminum zirconium complexes and aluminum chlorohydrate and its associated complexes (Health Canada [modified 2022a]).

Table 4-1. Additional uses in Canada for the Aluminium-containing Substances Group

Use	Aluminium-containing Substances Group
Food additive[a]	Y
Incidental additive[b]	Y
Food packaging materials[b]	Y
Medicinal or non-medicinal ingredients in disinfectant, human, or veterinary drug products[c]	Y
Medicinal or non-medicinal ingredients in natural health products[d]	Y
List of Prohibited and Restricted Cosmetic Ingredients[e]	Y
Present in cosmetics, according to notifications submitted under the *Cosmetic Regulations*[f]	Y
Formulant in registered pest control products[g]	Y

Abbreviations: Y, yes, use was reported for these substances

[a] In Canada, food additives are regulated under the *Canadian Food and Drug Regulations* (FDR) and associated Marketing Authorizations. Permitted food additives and their conditions of use are listed in the Lists of Permitted Food Additives. Ammonium Aluminum Sulphate, Potassium Aluminum Sulphate, Sodium Aluminum Sulphate, and Sodium Aluminum Phosphate are currently permitted for use in certain foods prescribed by one or more of the Lists (personal communication, from the FD, Health Canada, to the ESRAB, Health Canada, dated March 31, 2022; unreferenced).

[b] While not defined under the *Food and Drugs Act* (FDA), incidental additives may be regarded, for administrative purposes, as those substances which are used in food processing plants and which may potentially become adventitious residues in foods (for example, cleaners, sanitizers) (personal communication, email from the FD, Health Canada, to the ESRAB, Health Canada, dated October 2, 2018; unreferenced).

[c] Personal communication, email from the TPD, Health Canada, to the ESRAB, Health Canada, dated June 11, 2018; unreferenced; DPD [modified 2022].

[d] LNHPD [modified 2022]; personal communication, emails from the NNHPD, Health Canada, to the ESRAB, Health Canada, dated June 12, 2018; unreferenced; NHPID [modified 2022].

[e] Health Canada [modified 2022a]; Health Canada's Cosmetic Ingredient Hotlist is an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances may contravene the general prohibition found in section 16 of the *Food and Drugs Act* (FDA) (Canada 1985) or may contravene one or more provisions of the *Cosmetic Regulations* (Canada 2009). Aluminum hydroxychloride (CAS RN 1327-41-9), aluminum chlorohydrate (CAS RN 12042-91-0), and aluminum zirconium complexes (CAS RNs 57158-29-9 and 90604-80-1) are identified as being restricted on the Cosmetic Ingredient Hotlist. In July 2023, Health Canada published a proposed update to the entry for aluminum chlorohydrate and its associated complexes on the Cosmetic Ingredient Hotlist (Health Canada [modified 2023]). This revision proposes to prohibit aluminum chlorohydrate and its associated complexes in aerosol products.

[f] Personal communication, emails from the CHPSD, Health Canada, to the ESRAB, Health Canada, dated June 25, 2018; unreferenced.

[g] Health Canada 2010; personal communication, email from the PMRA, Health Canada, to the ESRAB, Health Canada, dated June 18, 2018; unreferenced.

5. Releases to the environment

Canada's National Pollutant Release Inventory (NPRI) reports annual releases to the environment, annual quantities recycled, and annual quantities disposed for specific physical forms of two aluminium-containing substances: aluminum (fume or dust only) and aluminum oxide (fibrous forms only).

NPRI data for 2013 to 2017 are presented in the tables and figures below. Industrial sectors were attributed as North American Industry Classification System (NAICS) codes at the 4-digit level, as notified to the NPRI. Aluminum (fume or dust only) and aluminum oxide (fibrous forms only) were released primarily to air for all sectors except pulp, paper and paperboard mills, where larger releases have been reported to water. Further details are provided in Appendix D.

Table 5-1. Total annual quantities of aluminum (fume or dust only) released to air, land, and water from 2013 to 2017

Year	Air (tonnes)	Land (tonnes)	Water (tonnes)	Unspecified media (tonnes)	Annual total (tonnes)
2013	137.8	0.0	3.3	0.2	141.3
2014	95.6	0.0	1.8	1.1	98.5
2015	125.2	0.0	6.0	1.7	132.9
2016	98.0	0.0	2.1	1.7	101.8
2017	93.8	0.0	2.8	2.1	98.7

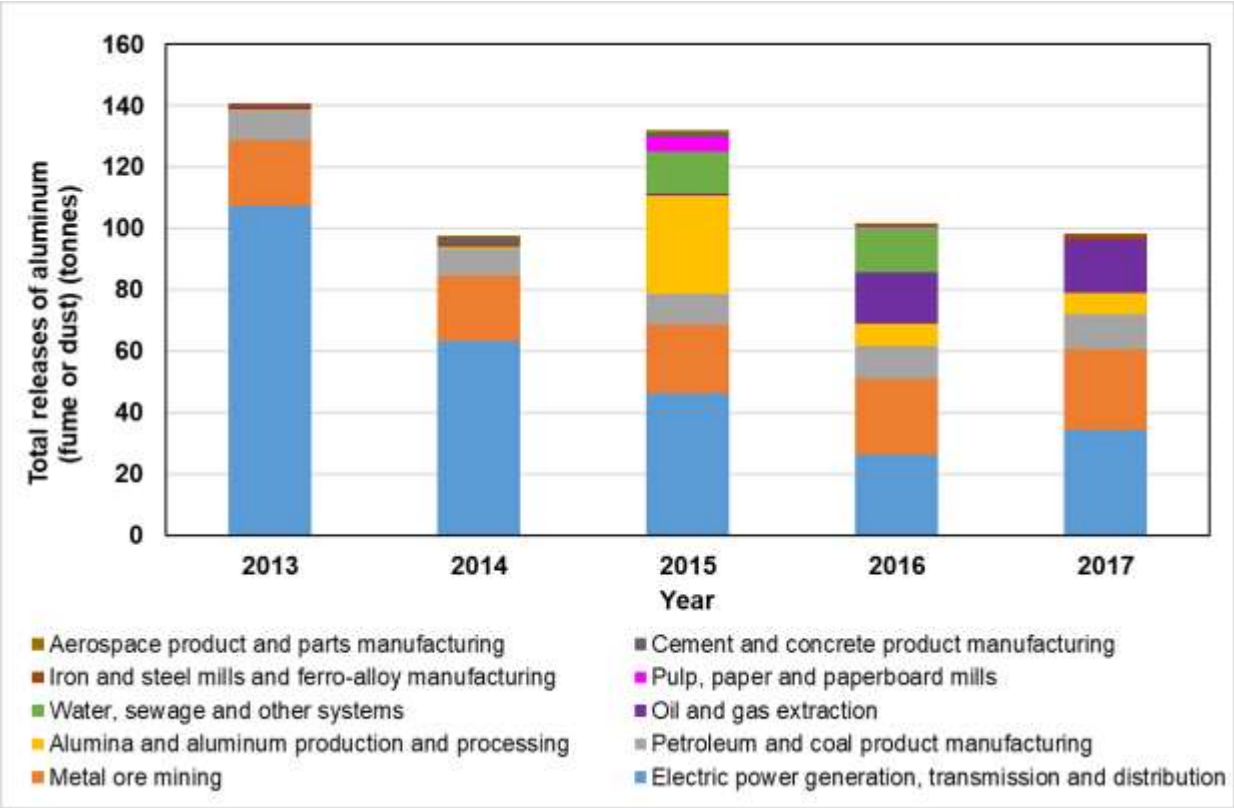

Figure 5-1. Sectors reporting the largest releases of aluminum (fume or dust only) to the National Pollutant Release Inventory from 2013 to 2017 (NPRI [modified 2022])

Figure 5-1 shows that the proportion of total releases of aluminum (fume or dust only) from the electrical power generation sector has generally been decreasing from 2013 to 2017, potentially correlated with closures, conversions to other fuel sources, technology improvements, or reductions of activity at some coal power plants (Ontario [modified 2021]; CER [modified 2022]). Releases from metal ore mining and petroleum and coal product manufacturing have remained relatively constant.

Table 5-2. Total annual quantities of aluminum oxide (fibrous forms only) released to air, land, and water from 2013 to 2017

Year	Air (tonnes)	Land (tonnes)	Water (tonnes)	Unspecified media (tonnes)	Annual total (tonnes)
2013	40.9	0.0	0.0	0.2	41.1
2014	43.2	0.0	0.0	1.9	45.1
2015	30.6	0.0	0.0	1.5	32.1
2016	0.0	0.0	0.0	1.3	1.3
2017	0.0	0.0	0.0	1.3	1.3

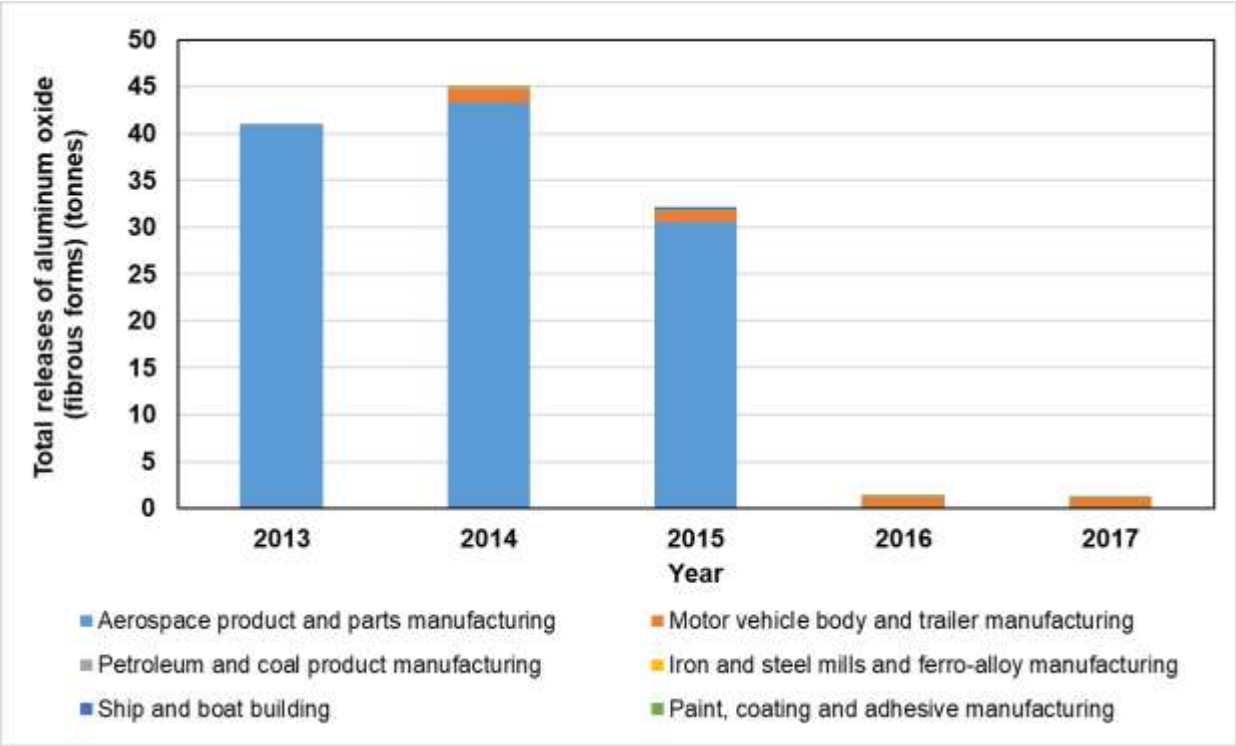

Figure 5-2. Sectors reporting the largest releases of aluminum oxide (fibrous forms only) to the National Pollutant Release Inventory from 2013 to 2017 (NPRI [modified 2022])

As indicated in Figure 5-2, reported releases of aluminum oxide (fibrous forms only) from the aerospace product and parts manufacturing sector ceased after 2015. The 2016 NPRI submission notes from the company involved (responsible for between 95% and over 99% of the total reported releases) suggest that the form of aluminum oxide released is not fibrous and, therefore, the releases are no longer reportable. Recent reported releases of aluminum oxide (fibrous forms only) are very modest compared to those reported for aluminum (fume or dust only).

Significant and highly variable quantities of aluminum (fume or dust only) and aluminum oxide (fibrous forms only) are disposed of both on-site and off-site by reporting facilities (Table D-2 and Table D-4). From 2013 to 2017, average total annual disposal of aluminum (fume or dust only) ranged from 149 tonnes to 10 727 tonnes, while aluminum oxide (fibrous forms only) ranged from 98 tonnes to 3 226 tonnes. For both substances, the largest disposals were generally to landfill. Cumulatively, the sectors with the highest disposal quantities were waste treatment and disposal, metal ore mining, pulp, paper and paperboard, and alumina and aluminum production.

6. Environmental fate and behaviour

Substances in the Aluminium-containing Substances Group belong to a wide variety of chemical categories (for example, inorganic, organometallic, and UVCBs), with a relatively wide range of chemical properties. Each substance has a unique environmental fate and behaviour when examined in isolation. However, while the reaction rates may vary substantially, each substance in the group is considered to have the potential to eventually dissolve, decompose, biodegrade, or otherwise transform to contribute to “total aluminium” in the environment. Recognizing the exclusions described in the Introduction, this approach is conceptually consistent with international guidance on the grouping of chemicals and a number of case studies from other jurisdictions (Worth and Patlewicz 2007; OECD 2014). Therefore, for the purposes of this assessment, the evaluation of environmental fate and behaviour will focus on the aluminium moiety.

6.1 Environmental distribution

Aluminium is found naturally in all environmental compartments: air, soil, water, sediment, and biota. Atmospheric deposition of aluminium to soils is mostly attributed to the deposition of dust particles and is generally low (Driscoll et al. 1994). Most of the substances in the group have negligible volatility at environmentally relevant temperature and pressure. Those that do have some volatility (for example, organoaluminium compounds) are pyrophoric and, therefore, are not expected to persist in the air (Krause et al. 2012). As a ubiquitous constituent of soil, the amount of aluminium present in the air compartment due to natural fluxes of windblown dust is expected to be much greater than that due to industrial releases of substances in the group (EC, HC 2010). For the same reason, long-range transport potential (LRTP) has not been evaluated in detail. Although aluminium-containing particles from natural erosion are known to travel long distances, it is suggested that aluminium-containing particles associated with industrial sources such as steel or cement manufacturing usually deposit near their source (Eisenreich 1980; Heimburger et al. 2012; Abril et al. 2014).

Aluminium in soil and sediment represent the largest pool of naturally occurring aluminium in the environment, principally in the form of aluminosilicates (Sparling and Lowe 1996). Aluminium has low mobility and is not very soluble in neutral soils, but it is soluble in acidic soils, with hydrolysis being the most important reaction in the chemical weathering of the common silicate minerals (Shiller and Frilot 1996; Courchesne and Hendershot 1997). Weathering and degradation processes mobilize aluminium from soil and sediment into the aquatic compartment. Conversely, processes such as precipitation and retention on surfaces and particles reduce the mobility of aluminium in the aquatic compartment. The net result of these opposing processes is a complex biogeochemical cycle, which, for aluminium, is particularly sensitive to pH and additional factors such as the availability of complexing ions and temperature (Lydersen 1990; Driscoll and Postek 1996).

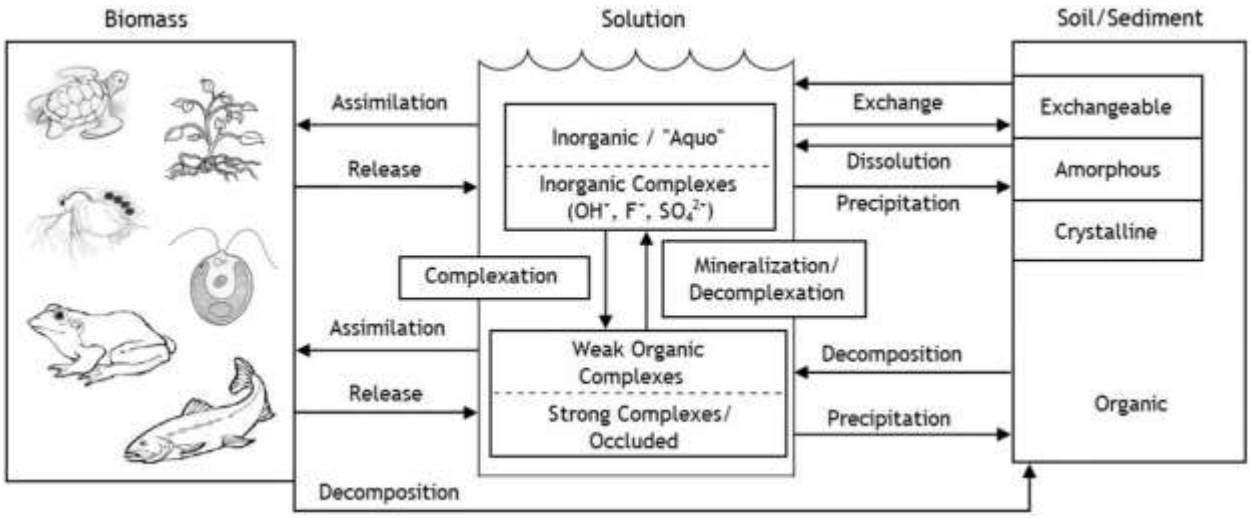

Figure 6-1. Representation of the aluminium cycle (adapted from Driscoll and Postek 1996)

The distribution of aluminium species in the aquatic compartment is an important consideration in ecological hazard and exposure assessment. Aluminium is relatively insoluble at and around neutral pH (for example, 6 to 8), with solubility at its minimum between pH 6.2 and 6.5 (Martin 1986; Driscoll and Schecher 1990; Gensemer and Playle 1999). As a hard Lewis acid, Al^{3+} has a high affinity for forming complexes with hard bases, including oxygen donor ligands, such as hydroxides, carboxylates (and by extension, fulvic and humic acids), and phosphates. Therefore, Al^{3+} can compete with biologically relevant cations of calcium, iron, magnesium, and potentially others for oxygen-rich binding sites and physiological oxoanions (Brothers and Ruggiero 2011). Al^{3+} will form complexes with other hard bases as well, such as fluoride (for example, AlF^{2+}, AlF_2^{+}) and sulfate (for example, $Al(SO_4)^{+}$) ligands (Driscoll and Postek 1996; Krstic et al. 2012).

Consequently, aluminium speciation in aquatic media, including in the interstitial water in soil and sediment, is highly complex. Along a gradient of increasing aluminium and hydroxide concentrations (that is, increasing pH), Al^{3+} will undergo mononuclear hydrolysis (for example, to $Al(OH)^{2+}$ and $Al(OH)_2^{+}$) and will "polymerize" to various polynuclear species (for example, $AlO_4Al_{12}(OH)_{24}(H_2O)_{12}^{7+}$, also known as "$Al_{13}$"), and then to macromolecules, colloidal forms, and ultimately to crystals of gibbsite (α-$AlOH_3$) (Poléo et al. 1994; Poléo 1995). The polynuclear species can be potent anion scavengers, and this polymerization process is key to the utility of aluminium compounds as flocculants in water treatment (Bertsch and Parker 1996). Temperature also plays an important role in the mobility of aluminium, indicating the importance of kinetics in this process (Vesely et al. 2003). Lydersen (1990) found that at the same pH, lower temperatures significantly increased the proportion of measured Al^{3+} relative to hydrolyzed cationic species ($Al(OH)^{2+}$, $Al(OH)_2^{+}$). At lower aluminium concentrations expected in the natural environment, anionic species such as phosphate, sulfate, and

fluoride may interfere with aluminium hydrolysis and polymerization (Jardine and Zelazny 1996).

The chemistry of aluminium adsorption to soil and sediments is also complex. Aluminosilicates are a common component of soils and sediments (Stumm and Morgan 1981). The retention of aluminium by sediments decreases as pH decreases (Dillon et al. 1988; Nilsson 1988), and sediments in acidified watersheds can provide a source of aluminium to the water column (Nriagu and Wong 1986; Wong et al. 1989). This has been demonstrated by rapidly increasing measured aluminium concentrations in response to experimental acidification of lakes and limnocorrals (Schindler et al. 1980). The release of aluminium hydroxide sludge from drinking water treatment and wastewater treatment systems[9] directly to surface waters is the primary anthropogenic pathway by which aluminium enters sediment. If water velocity is low at the point of discharge, much of the released sludge will settle onto the surface of local sediment. In Canada, the pH of waters receiving such discharges is typically nearly neutral; therefore, the solubility of aluminium species in the sludge is expected to remain low in general (EC, HC 2000).

In soil, adsorbed aluminium may be exchangeable to varying degrees with cations in solution, or it may be in various stages of precipitation from amorphous to more crystalline phases (Jardine and Zelazny 1996). In acidic soils, more aluminium is dissolved in the soil solution, and cationic nutrients (Ca^{2+}, Mg^{2+}, and K^{+}) may be replaced in the cation exchange complex (CEC) by Al^{3+}, which particularly tends to dominate in soils at pH values below 5 (Violante et al. 2010). In forest soils of cold and humid regions, such as those of eastern Canada, aluminium migrates from upper to lower mineral soil horizons by complexation with organic acids leached from foliage and the slow decomposition of organic matter in the forest floor (Courchesne and Hendershot 1997). Most dissolved aluminium in forest floor soil solutions is organically bound, and these aluminium-organic complexes become less abundant with increasing soil depth (Nilsson and Bergkvist 1983; David and Driscoll 1984; Driscoll et al. 1985).

6.2 Environmental persistence

Virtually all aluminium on Earth is of the stable isotope ^{27}Al (Haynes 2016). As noted above, processes such as hydrolysis and biodegradation will apply to varying extents and at varying rates for specific substances within the Group. The aluminium within each substance is considered persistent as it cannot degrade beyond the monomeric species (the hexahydro ion $Al(H_2O)_6^{3+}$, frequently abbreviated as Al^{3+}), although it can

[9] In this assessment, the term “wastewater treatment system” refers to a system that collects domestic, commercial, and/or institutional household sewage and possibly industrial wastewater (following discharge to the sewer), typically for treatment and eventual discharge to the environment. Unless otherwise stated, the term wastewater treatment system makes no distinction of ownership or operator type (municipal, provincial, federal, Indigenous, private, partnerships). Systems located at industrial operations and specifically designed to treat industrial effluents will be identified by the terms “on-site wastewater treatment systems” and/or “industrial wastewater treatment systems”.

transform into different chemical species, partition among different phases and environmental compartments, or both. The persistence of each potential parent compound has not been evaluated individually in the present assessment.

6.3 Potential for bioaccumulation

In the aquatic compartment, aluminium readily accumulates on fish gills and mucus during acute exposures but is slow to enter the blood and internal organs (Handy and Eddy 1989; Spry and Wiener 1991). Dussault et al. (2001) noted significant gill accumulation of aluminium in rainbow trout but low and dose-independent accumulation in the liver. Several species of algae can accumulate large amounts of aluminium without apparent damage (Sparling and Lowe 1996; Roy 1999). Factors such as pH and organic matter are important considerations for aluminium bioaccumulation studies, and measured accumulation is often a function of these variables. The effect of these factors on bioaccumulation by algae depends greatly on the species studied. Parent and Campbell (1994) reported suppressed accumulation of aluminium by *Chlorella pyrenoidosa* at lower pH. Roy and Campbell (1997) found that fulvic acid reduced the toxicity of aluminium to juvenile Atlantic salmon, while Winter et al. (2005) found that natural dissolved organic matter eliminated aluminium accumulation from juvenile rainbow trout gills across a range of pH values. Parent et al. (1996) found that soil fulvic acid may have promoted the accumulation of aluminium by *Chlorella pyrenoidosa*.

In a comprehensive literature review, the United States Environmental Protection Agency (US EPA) (2018) identified three acceptable aluminium bioaccumulation studies for aquatic organisms. Although measured aluminium residues were likely both surface-bound and internal, Cleveland et al. (1991) found that whole body kinetic bioconcentration factors (BCFs) in brook trout were inversely correlated with pH, with BCFs ranging from 215 at pH 5.3 to 36 at pH 7.2. Aluminium was rapidly depurated from all treatment groups, and residues had begun to decline before the end of the exposure period. Buckler et al. (1995) noted a similar decline in tissue concentrations in Atlantic salmon during the exposure period and hypothesized that the effect may be due to the decreased capacity of gills to adsorb aluminium as they become damaged, to modified physiological responses, or to growth dilution. Finally, Dobranskyte et al. (2004) measured aluminium accumulation in the digestive gland and other soft tissues of freshwater snails exposed to aluminium. Aluminium accumulated in both tissues and remained significantly higher in the treatment groups relative to control snails, although concentrations also started to decline during the exposure period. BCFs calculated from tissue concentrations at 30 days, compared with the average of water concentrations measured at 0 h, 24 h, and 48 h after each water change, were approximately 4.3 and 2.3 for the digestive gland and other soft tissues, respectively.

While BCF (and bioaccumulation factor, BAF) approaches often have limited usefulness in quantifying metal accumulation, these data show that aluminium has the potential to accumulate both on aquatic organisms, particularly on respiratory surfaces and associated mucus layers, and within aquatic organisms (McGeer et al. 2003). However,

aluminium is rapidly depurated when organisms are removed from exposure (Wilkinson and Campbell 1993).

Information is available on the accumulation of aluminium for plants and soil-dwelling organisms. The Agency for Toxic Substances and Disease Registry (ATSDR) (2008) reports an uptake factor (that is, the ratio of the concentration of aluminium in the plant to the concentration of aluminium in soil) of 0.004 for leafy vegetables, and 0.00065 for fruits and tubers (DOE 1984), although the specific species and pH values under which these factors were derived are unclear. In a bioaccumulation study by Zhao and Qiu (2010), earthworms (*Eisenia andrei*) were exposed to treatments of 20, 34, 50, and 100 mg $AlCl_3$/kg added to dry soil. Aluminium concentrations in earthworms reached a maximum at day 16, with concentrations ranging from 130 mg/kg to 170 mg/kg, which then decreased to between 70 mg/kg and 90 mg/kg by day 32. No information on the depuration phase was reported. After 32 days of exposure via soil, the BCF values based on total concentrations of aluminium in soil and earthworms ranged from approximately 1 to 3. From data presented in Bilalis et al. (2013), BCF values for the earthworm (*Octodrilus complanatus*) were estimated, which varied from 2 in the control group to 0.16 to 0.27 in the aluminium treatment groups. In another earthworm (*Eisenia fetida*) study by Tejada et al. (2010), the presented data suggest BCF values of approximately 0.04 in the control and values of 0.09 to 0.13 in the aluminium exposure groups.

Notwithstanding a number of tolerant and hyperaccumulating plant species (Jansen et al. 2002), the general bioaccumulation potential of aluminium in plants and invertebrates is low.

7. Potential to cause ecological harm

7.1 Ecological effects assessment

7.1.1 Mode/mechanism of action

Aluminium is a non-essential element without a known biological function (US EPA 2007; Wilson 2012; Gensemer et al. 2018). Aluminium is unlike most other metals in that the modes of action are conserved for both acute and chronic effects (Wilson 2012). Aluminium causes toxicity to freshwater organisms by disturbance of ionoregulatory processes, respiratory disruption, or both, depending primarily on pH and its effect on aluminium speciation (Wilson 2012; Cardwell et al. 2018; Gensemer et al. 2018). At acidic pH values where monomeric aluminium species start to prevail (for example, pH below ~5), aluminium (Al^{3+}) typically has an additive effect to the ionoregulatory disturbance caused by acidity alone by displacing calcium from anionic binding sites within intercellular junctions and interfering with enzymes such as gill sodium-potassium pumps (Na^+/K^+-ATPase) and carbonic anhydrase (Gensemer and Playle 1999; Wilson 2012). At very acidic pH (for example, pH ~3), aluminium (Al^{3+}) can technically begin to antagonize the ionoregulatory disturbance caused by acidity alone

by competing with H^+ for binding sites, but the effect can be temporary (Gensemer and Playle 1999; Skei and Dolmen 2006).

In fish, respiratory system toxicity predominates in moderately acidic water (for example, pH ~5 to 6), where aluminium accumulation on gill surfaces leads to excessive mucus production, inflammation, and thickening and shortening lamellae (Wilson 2012). Local increases in the pH of water at the gill surface due to continuous excretion of ammonia cause a shift in speciation towards cationic and polynuclear aluminium hydroxide species and precipitated $Al(OH)_3$, which may clog the gill surface by either chemical or physical adsorption (Playle and Wood 1989; Wilkinson and Campbell 1993; Gensemer and Playle 1999; Wilson 2012). This basic mechanism for acute toxicity also applies at a larger scale in mixing and liming zones, where sudden increases in pH induce rapid polymerization of aluminium species and create a zone of acute toxicity (Wilson 2012). Oxidative stress responses in common carp and grass carp have also been observed following acute aluminium exposures (Fernández-Dávila et al. 2012; Razo-Estrada et al. 2013).

In amphibians, a similar respiratory toxicity mechanism may also be relevant in moderately acidic water (for example, pH ~5 to 6) comparing the survival of a species with internal gills (*Bufo bufo*) with that of a species with external gills (*Triturus vulgaris*) (Skei and Dolmen 2006). Similar to fish, the effects of aluminium on amphibians at lower pH (for example, pH below ~5) are generally additive with the effects of acidity (H^+) through the ionoregulatory disturbance mechanism, although aluminium can antagonize the effects of acidity for some species not far below this pH range (Freda and McDonald 1990; Sparling and Lowe 1996; Dolmen et al. 2018).

Fewer mode of action data are available for invertebrates and algae, although ionoregulatory effects are the most commonly documented for invertebrates (Gensemer and Playle 1999; US EPA 2018). For example, Havas (1985) observed an increasing loss of sodium and chloride ion concentrations in *Daphnia magna* following acute exposures at low pH to increasing aluminium concentrations, which led to disrupted swimming and feeding behaviour and eventually death. Herrmann and Andersson (1986) proposed both “chemical” ionoregulatory disruption and “mechanical” respiration impedance by precipitated hydroxides and resulting mucus formation as mechanisms for respiratory effects observed in mayfly species. For aquatic plants and algae, aluminium may be internalized by the cell and interfere with metabolic processes (Crémazy et al. 2013). Surface precipitated aluminium species may interfere with the movement of flagella or act as photosynthetic shade (Lindemann et al. 1990; Golding et al. 2015). The potential for aluminium to bind phosphorus may also reduce the availability of this nutrient to primary producers (Sparling and Lowe 1996; US EPA 2018).

Save for certain tolerant and hyperaccumulating species, aluminium is generally phytotoxic to terrestrial plants, with the effects of aluminium exposure being most evident in root tissues (Jansen et al. 2002; Haridasan 2008; Santos et al. 2014). The most common effects of aluminium on sensitive plants include cessation of root

elongation, changes in morphology of the root system, decreased root respiration, and subsequent nutrient deficiencies due to impaired uptake (Ryan et al. 1994; Delhaize and Ryan 1995; Rout et al. 2001; Barabasz et al. 2002; Kochian et al. 2004; Yang et al. 2011; Kopittke et al. 2015; Kopittke and Blamey 2016). While root tissues are generally the site of action, whether effects on the cell wall, ion transport processes, or hormones and signalling are the causative or secondary mechanisms of aluminium toxicity is unclear (Brothers and Ruggiero 2011; He et al. 2012). Aluminium also has the potential to affect rhizobial symbiosis, including a reduction of rhizobial population, effects on rhizobial gene expression, and ultimately an impairment of the nitrogen fixation process (Jaiswal et al. 2018). Furthermore, simple determination of the total or exchangeable aluminium content in the soil solution is not a sufficient indicator to determine aluminium toxicity to plants, and therefore, quantification of the chemical species becomes necessary (Cunha et al. 2018). In general, the hazardous effects on root growth can be empirically related to different aluminium species in the following order: $Al^{3+} > Al(OH)_2^+ > Al(OH)^{2+} > Al(OH)_4^-$ (Nogueirol et al. 2015). However, compared to these mononuclear aluminium species, the polynuclear triskaideka aluminium species, Al_{13}, exhibits particularly strong phytotoxic properties (Parker et al. 1989; Kinraide 1990, 1997; Klöppel et al. 1997; US EPA 2003; Drábek et al. 2005; Manoharan et al. 2007). Finally, Bloom and Erich (1995) suggest that aluminium precipitated or complexed by organic compounds is apparently non-hazardous to plants.

7.1.2 Effects on aquatic organisms

Many studies are available on the acute and chronic toxicity of aluminium to aquatic organisms such as algae, plants, invertebrates, and fish. A Federal Water Quality Guideline (FWQG) for the protection of aquatic life from adverse chronic effects of total aluminium was developed in parallel with this assessment. The FWQG was selected to derive freshwater predicted no effect concentrations (PNECs) for the Aluminium-containing Substances Group because it includes recent scientific studies, integrates toxicity modifying factors (TMFs), and uses chronic toxicity data that are an indicator of harm from long-term exposure. On the basis of the substances in the group and the exposure scenarios considered in the assessment, an acute effects threshold for aluminium was not developed.

Details on the derivation of the FWQG, including the toxicity database, are available in ECCC (2022a). In brief, the FWQG proposes a multiple linear regression (MLR) approach to account for the influence of the TMFs pH, dissolved organic carbon (DOC), and hardness on aluminium toxicity. A pooled MLR relationship was developed between toxicity and TMF data for two species, the fathead minnow (*Pimephales promelas*) (n = 27) and the water flea (*Ceriodaphnia dubia*) (n = 32). The pooled MLR relationship was then used to normalize the acceptable chronic freshwater toxicity data for all species. Endpoints were combined and preferred endpoints were selected for each species following the Protocol for the Derivation of Water Quality Guidelines for the Protection of Aquatic Life (CCME 2007). The final normalized data set comprised of 52 EC_{10} endpoints for 3 fish, 8 invertebrates, and 2 aquatic plants/algae species. Species sensitivity distributions (SSDs) were fit for several cumulative distribution functions

using maximum likelihood estimation. A model averaged HC_5 was established using Akaike's information criterion and, with the slopes of the MLR relationship, the following FWQG equation was derived (ECCC 2022a):

$$\text{FWQG } (\mu g/L) = e^{([0.645\times\ln(\text{DOC})]+[2.255\times\ln(\text{hardness})]+[1.95\times\text{pH}]+[-0.284\times(\ln(\text{hardness})\times\text{pH})]-0.96)}$$

where DOC is expressed in mg/L, hardness in mg/L as $CaCO_3$, and pH is unitless. Final guideline values are rounded to two significant figures. For example, the FWQG at a hardness of 50 mg/L as $CaCO_3$, pH 7.5, and DOC concentration of 0.5 mg/L is 170 µg/L.

A calculation spreadsheet distributed as Appendix B of ECCC (2022a) was used to calculate PNECs for the assessment of the 55 aluminium-containing substances. PNECs were not extrapolated beyond the domain of the pooled MLR relationship, and the calculator spreadsheet replaces out of domain input values with the lower or upper boundary for that parameter, as appropriate. Domain of the pooled MLR includes pH from 6.0 to 8.7, DOC from 0.08 mg/L to 12 mg/L, and water hardness from 10 to 430 (as mg $CaCO_3$/L) (ECCC 2022a). Highest and lowest possible FWQGs based on model limits are not presented due to their limited meaningfulness, given the natural autocorrelation of input variables. Instead, representative central tendency values for ecozones were used in this assessment when faced with an absence of TMF measurements (described further in section 7.2.1 and Table E-2).

7.1.3 Effects on benthic organisms

Few toxicity studies on the biological effects of the 55 aluminium-containing substances on sediment-dwelling organisms were identified. Similarly, Crane et al. (2007) found the same in their work on environmental quality standards. More recently, Stanley et al. (2010) published a study on the sediment toxicity of nanoscale and micron-sized aluminium oxide. However, micron-sized aluminium oxide resulted in no observed toxicity in *T. tubifex* and *H. azteca*, and nanoscale aluminium oxide is not within the scope of this assessment. Woodburn et al. (2011) conducted a dietary study on freshwater crayfish fed aluminium chloride-spiked food (~1.5 g $AlCl_3$/kg food), observing an inflammatory response in the hepatopancreas but without an effect on the number of circulating hemocytes, hemolymph ion concentrations, or hemolymph protein levels. These data could not be used to develop a PNEC for the sediment medium.

The PSL2 assessment addressed the aluminium substances most implicated in potential exposure routes to benthic organisms (for example, drinking water treatment or the treatment of eutrophic lakes) (EC, HC 2010). The most significant potential effects on the benthic environment were described as the localized physical effects of blanketing and smothering benthic communities near the outfall of aluminium-containing sludges (EC, HC 2010; Reitzel et al 2013). Studies and reviews published in the intervening years remain supportive of the lines of evidence of the previous assessment (Wauer and Teien 2010; Landman and Ling 2011; Zamparas and Zacharias 2014).

Although the potential for local impacts on benthic organisms exists, there are relatively few reports of such damage, and available studies summarized in the PSL2 assessment did not observe adverse effects, or they demonstrated only low, temporally, and spatially limited effects (Lin et al. 1984; Lin 1989; George et al. 1991; George et al. 1995). Field studies of the potential effects on benthic organisms of eutrophic lake treatments using aluminium substances were also summarized, demonstrating minimal long-term effects (Connor and Martin 1989; Narf 1990; Smeltzer 1990).

7.1.4 Effects on terrestrial plants and soil-dwelling organisms

Measurements of total aluminium can provide useful information for soil characterization with respect to the mineralogical composition and weathering of soil (Bertsch and Bloom 1996). However, for expressing effects on soil-dwelling organisms, total aluminium measurements are rarely useful due to the absence of correlation with observed toxicity (Mulder et al. 1989; US EPA 2003). To better incorporate bioavailability, many studies measure an “exchangeable” or “extractable” aluminium, operationally defined on the basis of a number of extracting agents of varying strength (for example, 1 M KCl, 1 M NH_4Cl, 0.01 M $CuCl_2$). A review of the literature regarding these methods determined that extraction by $CaCl_2$ (0.01 M to 0.02 M) was the most appropriate for developing an aluminium effects threshold for the purposes of this assessment. Briefly, Shuman (1990) indicated that the activity of Al^{3+} in soil solution, aluminium saturation of the CEC, and 0.01 M $CaCl_2$-extractable aluminium were all well related to plant growth, with $CaCl_2$-extractable being the easiest to measure in the laboratory. Hoyt and Nyborg (1987) demonstrated that correlation with yield responses of barley and rapeseed was highest with 0.02 M $CaCl_2$-extractable aluminium for soils above pH 5. Wright et al. (1989) also identified $CaCl_2$ as a superior extractant for predicting aluminium phytotoxicity due to better correlation with free Al^{3+} activity. $CaCl_2$ extraction is becoming a method of choice in other jurisdictions for the determination of extractable aluminium in soils (Government of South Australia 2016; Venter 2017).

No classical guideline-based phytotoxicity studies were identified for the 55 aluminium-containing substances. However, multiple soil toxicity thresholds for aluminium, related largely to phytotoxicity determined instead by hydroponic and soil solution studies, have been published in the literature. For example, Hoyt et al. (1974) demonstrated little response of barley to lime when acidic Canadian soils contained between 1 mg/kg and 2 mg/kg extractable aluminium. Hoyt and Nyborg (1972) noted reductions in barley grain yield, while McKenzie and Nyborg (1984) observed impairment of alfalfa and barley root growth, when $CaCl_2$-extractable aluminium concentrations were between 2 mg/kg and 3 mg/kg (Singleton et al. 1987). In New Zealand, a threshold concentration of $CaCl_2$-extractable aluminium for toxicity to sensitive pasture legumes in the plant root zone has been suggested as 3 mg/kg (Moir and Moot 2014; Moir et al. 2016, 2018; Whitley et al. 2016). Additionally, the Government of South Australia (2016) suggested a low phytotoxicity potential when $CaCl_2$-extractable aluminium concentrations are less than 2 mg/kg. Overall, this information indicates that for terrestrial plants, soil toxicity thresholds for $CaCl_2$-extractable aluminium fall in the range of 1 mg/kg to 3 mg/kg.

While extensive empirical studies (albeit hydroponic studies, not classical terrestrial toxicity studies) are available on the chronic toxicity of aluminium to plants, a more modest data set exists for soil invertebrates. Several of the available studies were evaluated and summarized in the PSL2 assessment of three aluminium salts and its supporting document (Bélanger et al. 1999; van Gestel and Hoogerwerf 2001; EC, HC 2010). A literature update was conducted to identify additional toxicity studies on soil invertebrates, with further details provided in Appendix F. For soil invertebrates, the most sensitive, statistically derived endpoints for earthworms were LC_{50} values ranging from 316 mg/kg to 457 mg/kg in acidic soils and to 532 mg/kg in neutral soils, both expressed as total aluminium (van Gestel and Hoogerwerf 2001; Zhao and Qui 2010). The use of the acute-to-chronic approach is less certain for soil toxicity than it is for aquatic toxicity, but extrapolating from acute median mortality to chronic sub-lethal low effect concentrations (for example, a factor of 10) would result in concentrations of 29 mg/kg to 53 mg/kg considering the most sensitive endpoints in Appendix F. A result of similar magnitude would be found extrapolating from median to low-level effects (for example, a factor of 5) on the EC_{50}s for cocoon production also reported by van Gestel and Hoogerwerf (2001). Both predictions would be in agreement with supporting evidence presented in the other available invertebrate studies (for example, Rundgren and Nilsson 1997; Tejada et al. 2010), summarized in Appendix F.

For comparison between the invertebrate toxicity data (based on total aluminium) and the phytotoxicity data (based on $CaCl_2$-extractable aluminium), it is important to note that Zhao and Qui (2010) also indicated that the LC_{50} of 532 mg/kg, on the basis of total aluminium, corresponded to 1.7 mg/kg of monomeric aluminium and 2.5 mg/kg of water-soluble aluminium. Van Gestel and Hoogerwerf (2001) also reported $CaCl_2$-extractable aluminium concentrations on the order of 21 mg/kg in the control to 41 mg/kg in the first treatment group. However, these concentrations were determined using a substantially more concentrated extractant (1 M $CaCl_2$), which should have extracted a larger proportion of aluminium, making comparisons difficult relative to the 0.01 M $CaCl_2$ to 0.02 M $CaCl_2$ extractions noted above.

Therefore, as a conservative assumption, it is proposed that the soil PNEC for terrestrial organisms (plants and invertebrates) is 1 mg/kg to 3 mg/kg $CaCl_2$-extractable aluminium. Separate assessment factors accounting for species variation of mode of action were not applied for the soil PNEC, given the incorporation of bioavailability and the breadth of species considered in the derivations of thresholds described above.

7.2 Ecological exposure assessment

Ecological exposure scenarios were developed for the industrial activities corresponding to the largest quantities in commerce according to information submitted in response to CEPA section 71 surveys (section 4) and corresponding to the largest reported releases reported to the NPRI (section 5). Exposure scenarios were therefore developed for: 1) primary aluminium manufacturing, 2) cement manufacturing, 3) metal mining, 4) electric power generation, and 5) pulp, paper and paperboard mills. Exposure characterizations for these sectors were based on measured concentrations in receiving and reference

environments when data were available, or they were based on modelled concentrations from effluent monitoring, reported loading rates, or emission factors. Owing to the typically remote location of facilities in these sectors, modelled exposures assumed “direct” discharge to the receiving environment following any on-site treatment. In other words, exposures were not assumed to aggregate at common wastewater treatment systems for “indirect” discharge via releases to sewer systems. Predicted environmental concentrations (PECs) generated from these five scenarios are presented as supporting information (ECCC 2020a). Although not presented here, predictive models using generic exposure scenarios that are based on quantity and use information submitted in response to surveys were used to estimate exposures from other sectors (ECCC 2020b).

Measured surface water concentrations from a number of federal, provincial, and territorial programs were also used to estimate exposure. For certain sectors, sites from the Federal Water Quality Monitoring Network (FWQMN) have been associated with estimated land use classifications for the respective drainage area, which has added to the weight of evidence.

7.2.1 Background concentrations and toxicity modifying factors

Ranges and quantiles of measured ambient total aluminium concentrations in Canadian surface waters by province and territory, and for the Great Lakes, are presented in the FWQG (ECCC 2018, 2022a). Ranges of ambient concentrations for each province or territory were very large, spanning up to five orders of magnitude. However, interquartile ranges generally spanned only one order of magnitude.

Background concentrations of total aluminium in surface waters pertaining to specific Canadian terrestrial ecozones were also estimated by Kilgour & Associates (2016). Samples considered to be in reference condition by the conductivity-alkalinity approach outlined in Kilgour & Associates (2016) and Proulx et al. (2018) were used to calculate median background concentrations for use in exposure modelling. Data from a variety of federal and provincial surface water quality monitoring programs and other repositories were used for these calculations. Non-detects were substituted with one-half of the reported detection limit. This had a negligible impact on the analysis, given a total aluminium detection frequency of approximately 99%. Data sources and median background concentrations used in exposure modelling are presented in Appendix E (Table E-1).

The chronic MLR-based FWQG for total aluminium incorporates data for three TMFs: pH, DOC, and total hardness. For assessing aluminium exposures, paired TMF measurements were preferred in all analyses where available, followed by TMF data from the same monitoring station (that is, station averages). When neither of these were available, representative values for the ecozone were used. Representative ecozone TMF data were derived using data identified as being in reference condition by the approach above (Proulx et al. 2018). Geometric means for total water hardness, DOC, and hydronium ion concentration (that is, mean of pH) were calculated as a

representative measure of central tendency since these parameters typically follow log-normal distributions in the environment (Appendix E, Table E-2).

7.2.2 Primary aluminium manufacturing

An exposure scenario was developed for the primary aluminium manufacturing sector, using daily effluent monitoring data from 2011 for all 10 facilities that are currently engaged in primary aluminium manufacture (personal communication, data collected by the Mining and Processing Division, Environment and Climate Change Canada [ECCC], shared with the Ecological Assessment Division, Environment and Climate Change Canada, dated July 2019; unreferenced). The majority of the facilities reported effluent flow on a daily basis as well as measured aluminium concentrations three times per week, on average. However, one facility provided monthly averages, while another provided only an annual average concentration for composite and grab samples.

PECs were calculated from these effluent concentrations (C_{eff}) and effluent flow data (R_{eff}) using a conservative local exposure scenario. Briefly, PECs were calculated as:

$$PEC\ (\mu g/L) = C_b\ (\mu g/L) + \left(\frac{C_{eff}\ (\mu g/L) - C_b\ (\mu g/L)}{R_f\ (L/d)/R_{eff}\ (L/d)}\right)$$

where R_f is the 10th percentile receiving river flow that was obtained from the nearest hydrometric monitoring station (HYDAT database, ECCC 2020c), and C_b is the median background total aluminium concentration for the respective ecozone. As a standard approach, the effective dilution factor was limited at a value of 10, and an effective dilution factor of 10 was used for large lakes. Since TMFs in the receiving environments required to calculate corresponding PNECs were not reported, ecozone values were used (section 7.2.1).

7.2.3 Cement manufacturing

Wet kilns no longer operate in Canada (personal communication, Mining and Processing Division, Environment and Climate Change Canada, July 23, 2019; unreferenced; US EPA 1995). However, cement plants generate effluents through other activities, including the eventual disposal of re-used cooling water, cleaning of equipment and yards, dust suppression, and rainwater leachate from storage piles (CANMET and Radian Canada 1993; PCA 2006).

No recently measured concentrations in either the effluents or receiving environments of cement manufacturing facilities were identified. An emission factor approach was therefore applied to characterize exposure. A published emission factor for aluminium in liquid effluents ranged from 0.04 g/tonne to 1.08 g/tonne (average: 0.48) of cement produced, and concentrations ranged from 0.05 mg/L to 0.29 mg/L (average: 0.16) of effluent, on the basis of monitoring data from facilities in Ontario in the early 1990s (CANMET and Radian Canada 1993). The emission factor was calculated using the relatively low capacity utilization of the early 1990s (~60%). This emission factor was

revised in 2005 using the same monitoring data but was derived using higher capacity utilization values relevant to the late 1990s and early 2000s (~90%). The emission factor for aluminium in liquid effluents in this update ranged from 0.02 g/tonne to 0.61 g/tonne (average: 0.28) of cement manufactured, and concentrations ranged from 0.05 mg/L to 0.29 mg/L (average: 0.15) of effluent (Cement Association of Canada 2005).

As a conservative initial assumption, due to potential fluctuations in capacity and capacity utilization in more recent years, and in the absence of any other data, both the average and worst-case production-based emission factors and concentrations from CANMET and Radian Canada (1993) were used to calculate PECs. Cement production was estimated from USGS (2015) for all facilities in Canada, except for one which had commenced production more recently. Production from this facility was estimated from publicly available company literature. Annual days of operation were averaged from operational schedules minus the total number of shutdown days reported to the NPRI from 2013 to 2017.

Under Ontario Regulation 561/94, *Effluent Monitoring and Effluent Limits – Industrial Minerals Sector* (Ontario 1990a), some of the cement plants located in Ontario are required to report monthly effluent flow rates (among other parameters) to the Ontario Ministry of the Environment, Conservation and Parks. Average flow data between 2012 and 2016 were analyzed for each reporting facility (Ontario 2019). To estimate effluent flows for other Ontario facilities and facilities located elsewhere in Canada, an average value of effluent flow per tonne of production capacity at the reporting facilities was applied (R_{eff}). Average and worst-case PECs for each facility were calculated using both the production-based emission factor approach and effluent concentrations (C_{eff}) as:

$$PEC\ (\mu g/L) = C_b\ (\mu g/L) + \left(\frac{\frac{\text{Emission factor } (\mu g/t) \times \text{Production } (t/y)}{\text{Days of operation } (d/y) \times R_{eff}\ (L/d)} - C_b\ (\mu g/L)}{R_f\ (L/d)/R_{eff}\ (L/d)} \right)$$

and:

$$PEC\ (\mu g/L) = C_b\ (\mu g/L) + \left(\frac{C_{eff}\ (\mu g/L) - C_b\ (\mu g/L)}{R_f\ (L/d)/R_{eff}\ (L/d)} \right)$$

where R_f, the 10th percentile receiving river flow, and C_b, the median background aluminium concentration for the ecozone, were obtained as described above (sections 7.2.1 and 7.2.2). As a standard approach, the effective dilution factor was limited at a value of 10. TMFs in the specific receiving environments required to calculate corresponding PNECs were not reported and, therefore, ecozone values were used (section 7.2.1).

7.2.4 Metal mining

Viable deposits of bauxite ores do not presently exist in Canada (NRCan [modified 2022]). However, the potential for the incidental mobilization of aluminium from ores, overburden, or via processing activities at metal mines, coupled with large releases reported to the NPRI (relative to most other sectors, Figure 5-1) suggested the need to develop an exposure scenario for this sector. Ore extraction and concentration operations generate dust, which may escape and deposit nearby, and effluents, which may be stored in tailings ponds or treated and released to surface waters.

Canadian metal mines subject to the *Metal and Diamond Mining Effluent Regulations* (MDMER) under the *Fisheries Act* (Canada 2022) must conduct effluent and water quality monitoring under environmental effects monitoring (EEM), including measurement of total aluminium. While the mine is depositing effluent, water quality monitoring studies must be completed four times per year, with samples at least one month apart, in exposure areas surrounding the point of entry of effluents in receiving waters from each final discharge point (FDP) and from the related reference areas. According to the MDMER, “exposure area” means all fish habitat and waters frequented by fish that are exposed to effluent, whereas “reference area” means water frequented by fish that is not exposed to effluent and that, as far as practicable, is most similar to that of the exposure area (Canada 2022).

When effluents are deposited to freshwater or estuarine waters, pH and hardness are among the required parameters to be measured and reported for water quality monitoring, and these paired data were used as FWQG inputs to calculate PNECs corresponding to each total aluminium measurement. Although DOC is not a required parameter, measured values were still reported for approximately 10% of the data set. Therefore, most DOC values and missing pH and hardness values were substituted with station averages or ecozone values, according to the order of preference described above (section 7.2.1).

Concentrations of total aluminium measured between 2013 and 2017 in surface water samples from exposure and reference areas, as submitted to Environment and Climate Change Canada under the MDMER and EEM program, were analyzed. Additional data for selected FDPs from 2018 were extracted from available interpretive reports (EEM 2020). A total of 6118 total aluminium measurements were available, comprising 3281 exposure area measurements and 2837 reference area measurements (EEM 2018, 2020). Non-detects were replaced with one-half the reported detection limit, but with a detection frequency of greater than 95% across the data set, the choice of method for addressing non-detects had no significant impact on the analysis.

Total aluminium concentration data were available for a total of 189 FDPs belonging to 128 facilities. Summary statistics for total aluminium concentrations in exposure and reference areas for masked FDPs are available as supporting information (ECCC 2020a). In summary, exposure areas receiving metal mining effluents were not systematically increased in total aluminium concentrations relative to corresponding

reference areas. In most cases, median and average total aluminium exposure concentrations were evenly distributed above and below one when divided by their corresponding median and average reference concentrations (for example, “enrichment factors”), although the full distributions were somewhat skewed (Figure 7-1). Differences in median pH between exposure and reference sites were very modest on average, with exposure sites tending to be slightly more alkaline than the corresponding reference site. Overall, these data suggest a large natural (for example, seasonal) variability in measured total aluminium concentrations but do not preclude the potential for higher aluminium enrichment at certain sites, as suggested by the lognormal distributions in Figure 7-1.

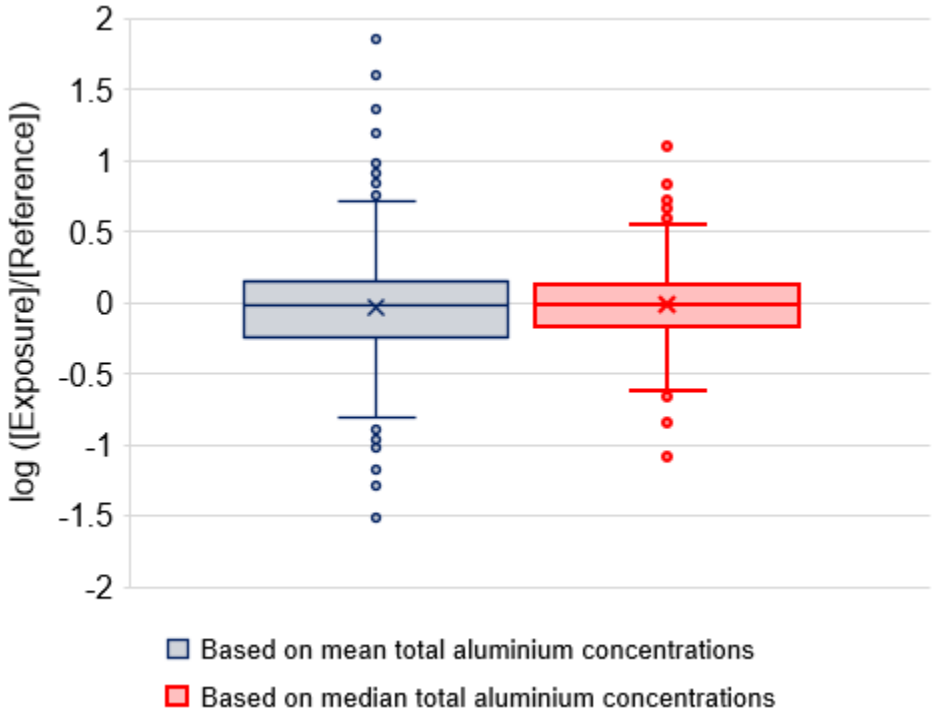

Figure 7-1. Total aluminium mean and median concentrations in exposure areas divided by corresponding concentrations in reference areas

7.2.5 Electric power generation

Under Ontario Regulation 215/95, *Effluent Monitoring and Effluent Limits – Electric Power Generation Sector* (Ontario 1990b), facilities in the electric power generation sector are required to report total aluminium loadings and effluent flow rates (among other parameters) to the Ontario Ministry of the Environment, Conservation and Parks. This data set includes representation from facilities using a variety of fuel types—nuclear, natural gas, oil, biomass, and coal—and was considered appropriate for read-across to facilities in other provinces. Monthly total aluminium loading and effluent flow data between 2012 and 2016 were analyzed for each reporting facility (Ontario 2019).

Since the regulation requires loadings to be determined from analytical results, monthly PECs for each facility were calculated as:

$$PEC\ (\mu g/L) = C_b\ (\mu g/L) + \left(\frac{\frac{\text{Maximum loading } (\mu g/d)}{R_{eff}\ (L/d)} - C_b\ (\mu g/L)}{R_f\ (L/d)/R_{eff}\ (L/d)} \right)$$

where R_{eff} is the monthly average of daily effluent flows, R_f is the 10th percentile receiving river flow, and C_b is the median background total aluminium concentration for the respective ecozone, obtained as described above (sections 7.2.1 and 7.2.2). As a standard conservative approach, the effective dilution factor was limited at a value of 10. Ecozone TMF values were used to calculate corresponding PNECs (section 7.2.1).

The Ontario (2019) data set does include some representation from coal-powered facilities. Although potential impacts from co-located coal mining complicate interpretation, water quality surveys conducted by the Alberta Ministry of the Environment at Wabamun Lake in 2002 and 2005 were also considered as a further realistic worst-case scenario for coal-powered generation facilities (Alberta Environment 2002, 2003a, 2006). Representative TMF values for calculating local PNECs were determined from Wabamun Lake water quality surveys conducted between 1999 and 2001 (Alberta Environment 2003b) and were notably similar to the representative values derived for the Boreal Plains ecozone.

7.2.6 Pulp and paper

The Canadian pulp and paper sector includes facilities (mills) that produce a range of products including paper, cardboard, newsprint, and pulp. These mills are subject to the *Pulp and Paper Effluent Regulations* (PPER) under the *Fisheries Act* (Canada 2018b). Effluent monitoring and Environmental Effects Monitoring (EEM) are conducted under Schedules II and IV.1 of the PPER, respectively. There is no requirement for mills to report aluminium concentrations in effluent or receiving areas. However, aluminium concentrations and concentrations of other metals are often measured as part of the "Investigation of Cause" phase after adverse effects of effluents have been observed (Environment Canada 2010).

Total aluminium concentrations in exposure and reference areas were identified from EEM interpretive reports for eight facilities between 2009 and 2018. Total aluminium non-detects were substituted with one-half the reported detection limit. This had limited impact on the analysis with a detection frequency of approximately 95% across the data set. Across eight facilities, 81 measurements in exposure areas and 48 measurements in reference areas were identified. Each facility reported at least one reference value. Corresponding pH, hardness, and DOC measurements were also collected or replaced with representative ecozone values when not reported in order to calculate aquatic PNECs. Measured pH and hardness were usually available (that is, <5% missing), while measured DOC was only available for about one-third of this data set.

Additionally, the National Council for Air Stream Improvement (NCASI) provided effluent and ambient (primarily raw intake) water quality measurements (NCASI 2020). Thirty facilities representing two mill process categories (mechanical and chemical pulping), different wood fibre sources, and covering four provinces and four ecozones participated in a sampling campaign (August 2018 to January 2019) and study sponsored by Canadian pulp and paper facilities, which was designed, coordinated, and managed by NCASI. Total and dissolved aluminium concentrations in effluent as well as aluminium, pH, and hardness in ambient waters were available in samples from 27 mills (16 chemical and 11 mechanical). Concentrations from three samples were provided for all but one of these mills. The effluent total aluminium concentrations within a mill were quite uniform, whereas the ambient concentrations were somewhat more variable. Therefore, PECs were calculated for each mill from their respective average total aluminium concentrations (C_{eff}) as follows:

$$PEC\ (\mu g/L) = C_b\ (\mu g/L) + \left(\frac{C_{eff}\ (\mu g/L) - C_b\ (\mu g/L)}{R_f\ (L/d)/R_{eff}\ (L/d)}\right)$$

where C_b was the average total aluminium concentration reported for the ambient (primarily raw intake) water, R_{eff} is the effluent flow, and R_f is the 10th percentile receiving river flow, as described above (sections 7.2.1 and 7.2.2). C_b was unavailable for one mill and was substituted with the corresponding ecozone median value. Although process type and ecozone were provided, specific facility identities were masked in NCASI (2020), rendering correlated values of R_{eff} and R_f unavailable. However, data collected for other recent assessments indicate that the vast majority of direct discharging pulp and paper mills are subject to the standard maximum effective dilution factor of 10. Therefore, the term R_f/R_{eff} in the equation above was replaced with a factor of 10 to calculate PECs for this sector. pH and hardness measurements for the ambient water were available as input TMFs for PNEC calculation. Because DOC measurements were not included in the study, representative ecozone values were used (section 7.2.1).

7.2.7 Canadian long-term surface water quality monitoring data

Land use classifications linked to certain monitoring stations in the Federal Water Quality Monitoring data set were available. These classifications considered variables such as population density, percentage of cropland, forest loss, and the number of facilities from the mining, forestry, oil sands, and shale gas sectors per unit area within the drainage area (Canada [modified 2017a]). For example, PECs classified on the presence of a facility in the same drainage area as a monitoring station provide somewhat less confidence than the facility-specific exposure characterizations described above, but they were still characterized for their contribution to the overall weight-of-evidence. Long-term water quality monitoring data for total aluminium from the most recent five years available in the data set (2011 to 2015) for stations with land use classifications were analyzed (personal communication, data prepared by the Water Quality Monitoring and Surveillance Division for the Ecological Assessment Division, Environment and Climate Change Canada [ECCC], dated February 17, 2016;

unreferenced). If paired measurements of pH, hardness, and DOC were not available in the data set, they were estimated using representative station values or ecozone values in the order of preference described above (section 7.2.1).

7.2.8 Bioavailable aluminium in Canadian soils

Most of the aluminium discharged from wastewater treatment systems is associated with sludge (Cheminfo Services 2008). Approximately one-third of this sludge is sent to landfill, while two-thirds are converted and applied to farmland, representing a significant source of anthropogenic aluminium exposure to agricultural soils in Canada (EC, HC 2010).

Bergman and Boots (1997) studied the potential for an increase in bioavailable aluminium in sludge-amended soils, finding that the application of 75 tonnes per hectare of alum sludge did not result in higher levels of the acid-extractable aluminium relative to control. Kluczka et al. (2017) found that, while total aluminium concentrations in alum sludge were 10 times higher than in fermented sewage sludge, the $BaCl_2$-extractable aluminium concentrations in soil mixed with alum sludge were similar to the $BaCl_2$-extractable aluminium concentrations in untreated soil. In contrast, Novak et al. (1995) found that acidic (pH 4.7 and 5.5) soils had higher concentrations of extractable aluminium after application of wastewater treatment system residuals compared to before. However, in a separate experiment, Novak et al. (1995) found no differences between the control and an acidic forest soil plot treated with a much more concentrated alum sludge after 30 months. Aluminium concentrations in seeds (Bergman and Boots 1997) and plants (Oladeji et al. 2006) from alum sludge-amended soils were not statistically different from those in control soil. According to Novak et al. (1995), statistical differences in aluminium concentrations were seen in corn, but not wheat or loblolly pine, grown on alum sludge-amended acidic soils compared to control.

High application rates of alum sludge, beyond what would be currently permissible in Canada, were used in most of the studies described above. Additionally, applications were most often made to soils more acidic than would be permitted in a sludge application scenario. For example, Oladeji et al. (2006) used an application rate approximately double, and Bergman and Boots (1997) used a rate approximately triple, that which would be allowed under the highest numerical maximum application rate found in Canada of 25 tonnes/hectare (once every three years) (Alberta Environment 2001). In addition, sludge application is usually only permitted when the pH is greater than 6.0 or when liming is done, if necessary (OMEE and OMAFRA 1996).

7.3 Characterization of ecological risk

The approach taken in this ecological assessment was to examine assessment information and develop proposed conclusions using a weight-of-evidence approach and precaution. Evidence was gathered to determine the potential for the 55 aluminium-containing substances to cause harm in the Canadian environment. Lines of evidence considered include those evaluated in this assessment that support the characterization

of ecological risk in the Canadian environment. Secondary or indirect lines of evidence are considered when available, including regulatory decisions and classification of hazard or fate characteristics made by other regulatory agencies. The potential for cumulative effects was considered in this assessment by examining cumulative exposures to total aluminium.

7.3.1 Risk quotient analysis

Risk quotient (RQ) analyses were performed by comparing the various realistic worst-case estimates of exposure and available measured environmental concentrations (PECs; see the Ecological Exposure Assessment section) with ecotoxicity information (PNECs; see the Ecological Effects Assessment section) to determine whether there is potential for ecological harm in Canada. Aquatic PNECs were calculated using the chronic MLR-based FWQG for the protection of aquatic life (ECCC 2022a) and paired TMF measurements for each total aluminium PEC, or substituted representative station or ecozone values if paired measurements were unavailable. RQs were calculated for the aquatic compartment by dividing PECs by the corresponding PNECs for the exposure scenarios described in section 7.2. Specifically, RQs were calculated for the aquatic compartment (surface freshwaters) for: 1) primary aluminium manufacturing, 2) cement manufacturing, 3) metal mining, 4) electric power generation, and 5) pulp, paper and paperboard mills. RQs associated with land use classifications from the Federal Water Quality Monitoring data set were also used to support the weight of evidence.

Box plots were generated to display the distribution of aluminium RQs at the facility level, where possible. The lower and upper edges of the box represent the first and third quartiles (Q1 and Q3, the 25th and 75th percentiles, respectively). The horizontal line within the box represents the median value (Q2, the 50th percentile). The mean is shown as an “x” within the box. The difference between the first and third quartiles is called the interquartile range (IQR). The upper whisker extends to the lowest data point that is within Q3 + 1.5 IQR, while the lower whisker extends to the largest data point that is within Q1 − 1.5 IQR. Individual data points outside of those ranges are represented by filled circles. Values outside of 1.5 ×IQR are considered “outliers” by some definitions, but statistical tests for outliers were not performed.

Primary aluminium manufacturing (smelting): Ecological risk characterization for this scenario was based on PECs modelled from 2011 effluent monitoring data and PNECs generated from ecozone values of TMFs. The distribution of resulting risk quotients is presented in Figure 7-2. For this sector, RQs were generally low, with only 0.4% of the approximately 1600 measurements representing a PNEC exceedance. Noting the conservative assumptions in the PEC calculation, Figure 7-2 demonstrates that there is low potential for ecological risk for aluminium from the primary aluminium smelting sector.

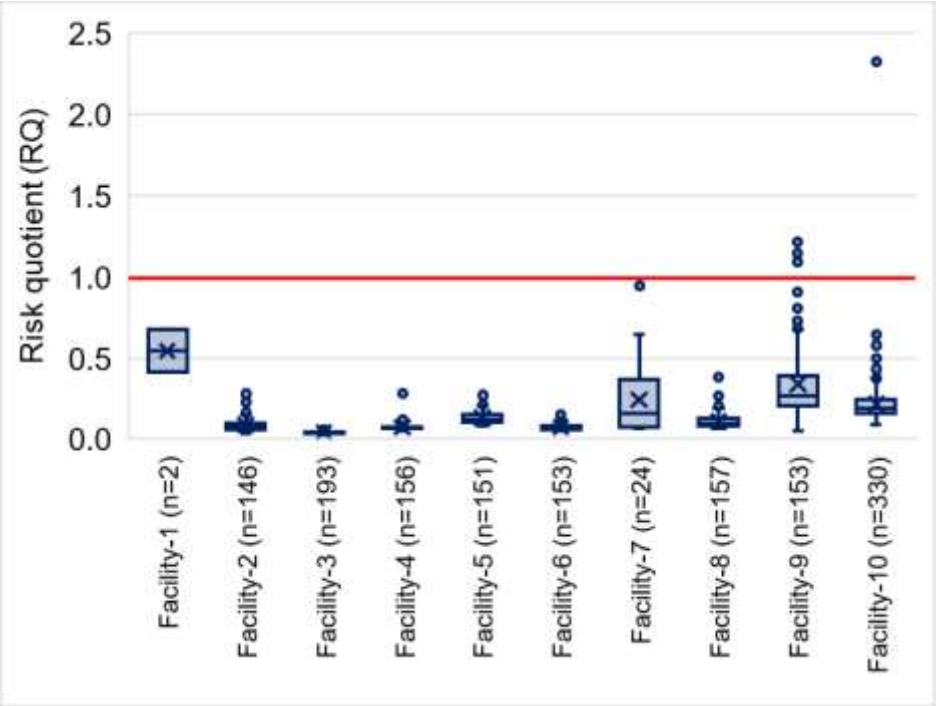

Figure 7-2. Primary aluminium smelting risk quotients for 10 facilities using effluent monitoring from 2011

Cement manufacturing: Limited data were available to generate aluminium RQs for the cement manufacturing sector. An emission factor approach was followed to estimate the amount of aluminium in cement plant effluents per unit of production (CANMET and Radian Canada 1993). The amount of effluent produced per tonne of cement production was estimated from data reported by 4 cement plants to the Government of Ontario (Ontario 2019) and was found to be comparable to other estimates (PCA 2006; Bezerra 2014). Covering most of the distribution of production capacities in Canada, these 4 cement plants were considered to be representative, and the ratio was applied to the other 15 currently active cement plants to estimate their effluent flow rates. Additionally, effluent concentrations from Ontario facilities in the early 1990s were used to calculate a second set of average and reasonable worst-case PECs. Aquatic PNECs were calculated for representative ecozone values of aluminium TMFs. Risk quotients for all cement plants with both average and reasonable worst-case emission factors and concentrations were generally low (Table 7-1).

Table 7-1. Summary of average and reasonable worst-case risk quotients obtained for cement manufacturing plants in Canada

Scenario	Minimum RQ	Q1 RQ	Average RQ	Median RQ	Q3 RQ	Maximum RQ
Average, production-based emission factor	0.030	0.036	0.14	0.13	0.14	0.53

Scenario	Minimum RQ	Q1 RQ	Average RQ	Median RQ	Q3 RQ	Maximum RQ
Worst case, production-based emission factor	0.041	0.056	0.21	0.19	0.28	0.65
Average, effluent concentration	0.032	0.032	0.13	0.098	0.13	0.51
Worst case, effluent concentration	0.042	0.042	0.17	0.15	0.19	0.57

Abbreviations: RQ, risk quotient; Q1, first quartile (25th percentile); Q3, third quartile (75th percentile)

Metal mining: Ecological risk characterization for the metal mining sector used surface water quality monitoring data submitted to the MDMER EEM program for samples collected from 2013 to 2017 (EEM 2018). The PECs consisted of measured total aluminium concentrations from effluent receiving environments (that is, exposure areas) and corresponding reference areas of metal mining facilities. Aquatic PNECs were calculated as described in section 7.2.4 with both exposure and reference area TMFs. Risk quotients were calculated for 189 FDPs belonging to 128 metal mining facilities. Approximately two-thirds of the exposure sites and one-half of the reference sites had no PNEC exceedances. To narrow down the number of metal mining exposure sites for closer analysis, a “frequency of exceedance differential” was calculated as the difference between the frequency of PNEC exceedance at the exposure site and the frequency of exceedance at the corresponding reference site.

Figure 7-3 presents a histogram of the differences in the frequency of PNEC exceedances for exposure sites where the sample size was greater than 10 and the median concentration of aluminium was greater than the median concentration in the corresponding reference.

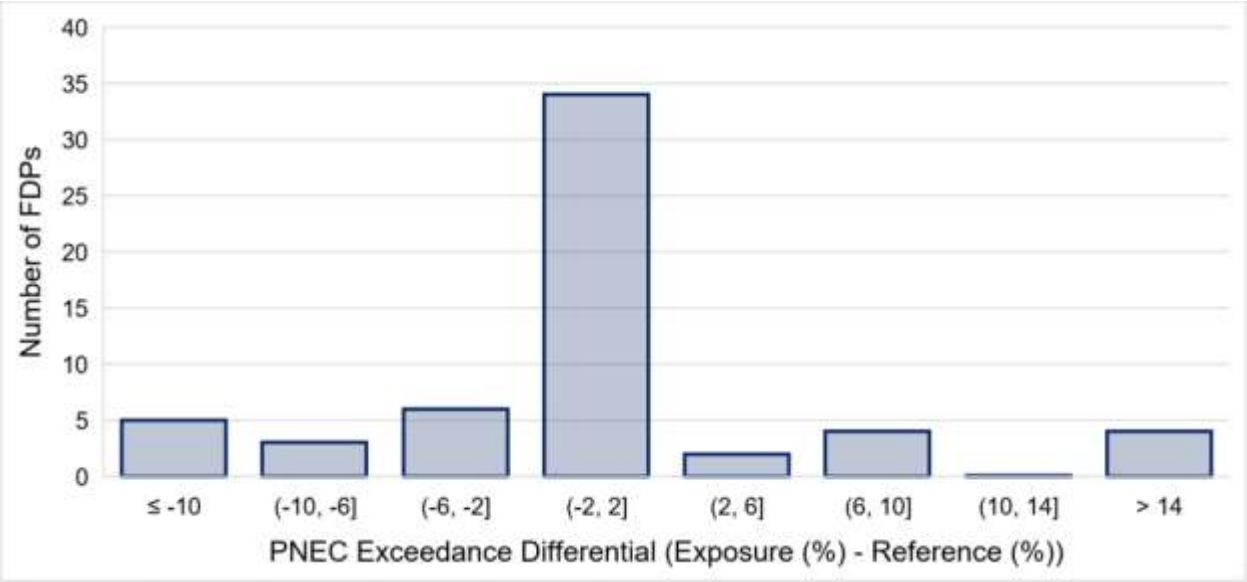

Figure 7-3. Frequency of PNEC exceedance at metal mining exposure sites (final discharge points) minus the frequency of PNEC exceedance at corresponding reference sites (%)

Figure 7-3 indicates that for the clear majority of FDPs, the frequency of PNEC exceedance in the exposure area is quite similar to the frequency of exceedance in the reference area. However, the histogram indicates that there are a small number of sites with a comparatively large frequency of exceedance differentials. In addition to sample size and enrichment of aluminium concentrations, further site selection criteria were applied. Specifically, the median and 95th percentile exposure RQs must exceed the corresponding median and 95th percentile reference RQs, respectively, and the 95th percentile exposure RQ must be greater than 1. Finally, the frequency of exceedance differential must be greater than 0% (that is, the PNEC must have been exceeded more often in the exposure area than it was in the corresponding reference area). Application of these criteria reduced 189 exposure sites to 6 sites of potential interest (3%). The distributions of total aluminium concentrations and corresponding RQs at the 6 sites are presented in Figure 7-4 and Figure 7-5.

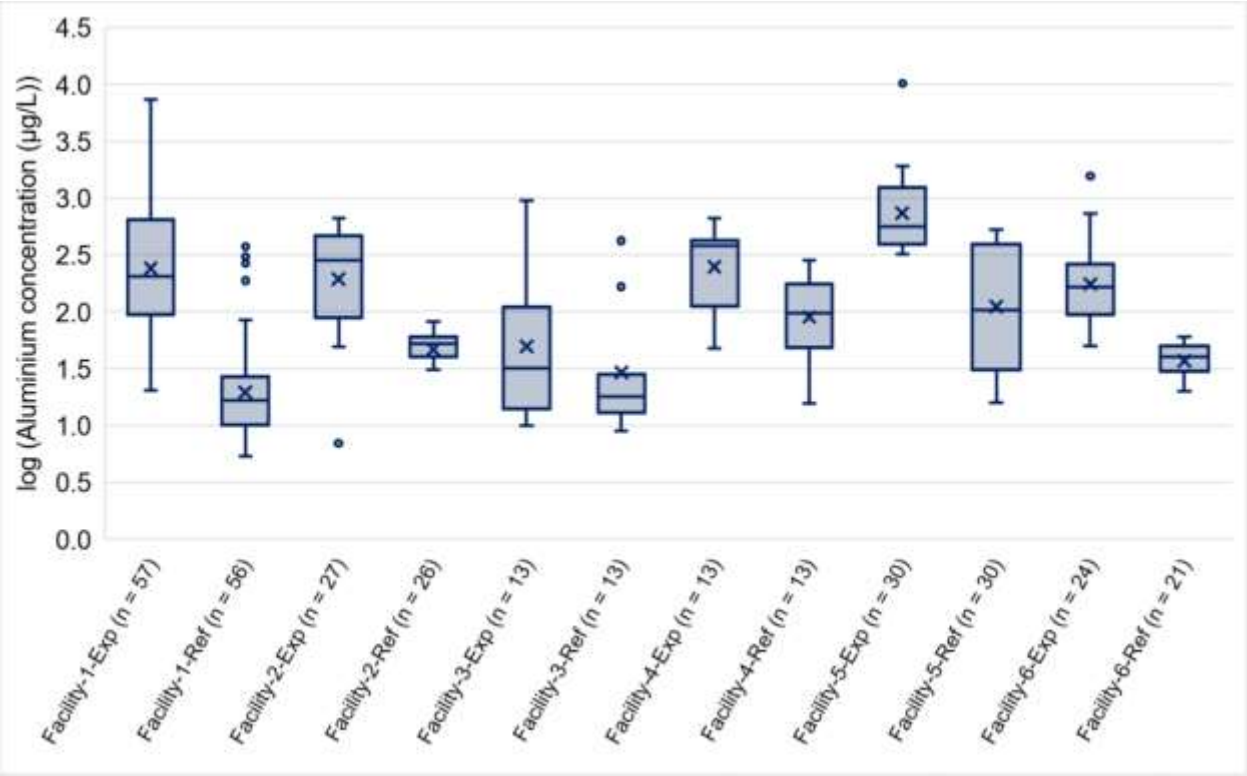

Figure 7-4. Measured total aluminium concentrations (µg/L) in exposure and reference areas for selected metal mining sites subject to the MDMER from 2013 to 2017 (EEM 2018)

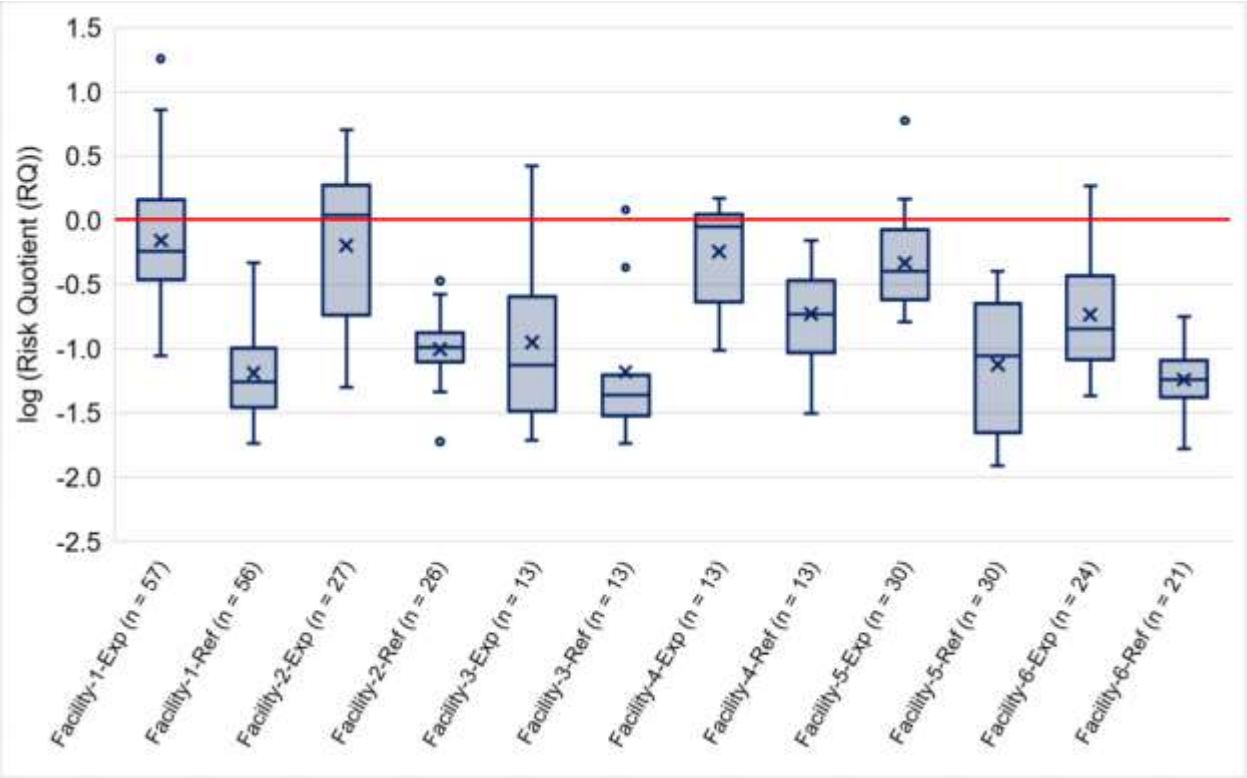

Figure 7-5. Risk quotients for selected metal mining sites subject to the MDMER from 2013 to 2017 (EEM 2018)

For the majority of the selected sites, the magnitude of RQs are relatively low. Sites F-1 and F-2 demonstrated some potential for ecological concern with relatively frequent exceedances in the exposure area but no exceedances in the corresponding reference areas. Interestingly, the median reference concentration for F-1 is less than half of the 5th percentile of the anticipated background concentration for its ecozone. Although perhaps statistically anomalous, this would typically magnify the concern with respect to the anthropogenic contribution at the corresponding exposure area. However, F-1 has been considered on suspended operations, and care and maintenance since 2013 (EEM 2020). Total aluminium concentrations in the effluent for F-1 during de-watering activities were approximately half non-detects and are generally much lower in magnitude than concentrations reported for the exposure area (EEM 2020).

Conversely, total aluminium concentrations for the F-2 reference are in close agreement with expected background concentrations for its respective ecozone. However, closer inspection of the most recent EEM interpretive report for F-2 indicated a surprising trend, with the concentrations of total aluminium in the receiving area being about an order of magnitude more concentrated than in its effluent, while hardness, alkalinity, and concentrations of other metals such as cadmium were being substantially diluted (EEM 2020). Approximately one-third of the measurements for the F-2 exposure area were outside of the pH domain of the MLR; all were too acidic, several by more than an order of magnitude (that is, pH ≤5). More than half of the RQs greater than 1 for the F-2

exposure area were associated with pH inputs below the domain of the MLR. In the F-2 reference area, pH was circumneutral and all measurements were within the domain of the MLR. Additionally, although water hardness was quite low, it was within the lower limits of the model. Comparing the exposure PEC to the reference PNEC resulted in fewer exceedances (ECCC 2020a), which suggests a broader potential pH issue at this site, rather than an aluminium-specific concern.

Given these considerations for the F-1 and F-2 exposure areas and noting that these sites represent a small fraction of the sector, these RQs provide insufficient evidence of ecological concern from the metal mining sector for aluminium.

Electric power generation: Risk quotients for the electric power generation sector were calculated from two sources. Firstly, from monthly total aluminium effluent loading and flow-rate data submitted to the Government of Ontario, covering reporting years from 2012 to 2016 (Ontario 2019). This data set was considered representative for Canada and included representation from electric power generation from a variety of fuel types: nuclear, natural gas, oil, biomass, and coal. Calculated RQs are presented in Figure 7-6 and suggest a low potential for ecological concern in the aquatic compartment.

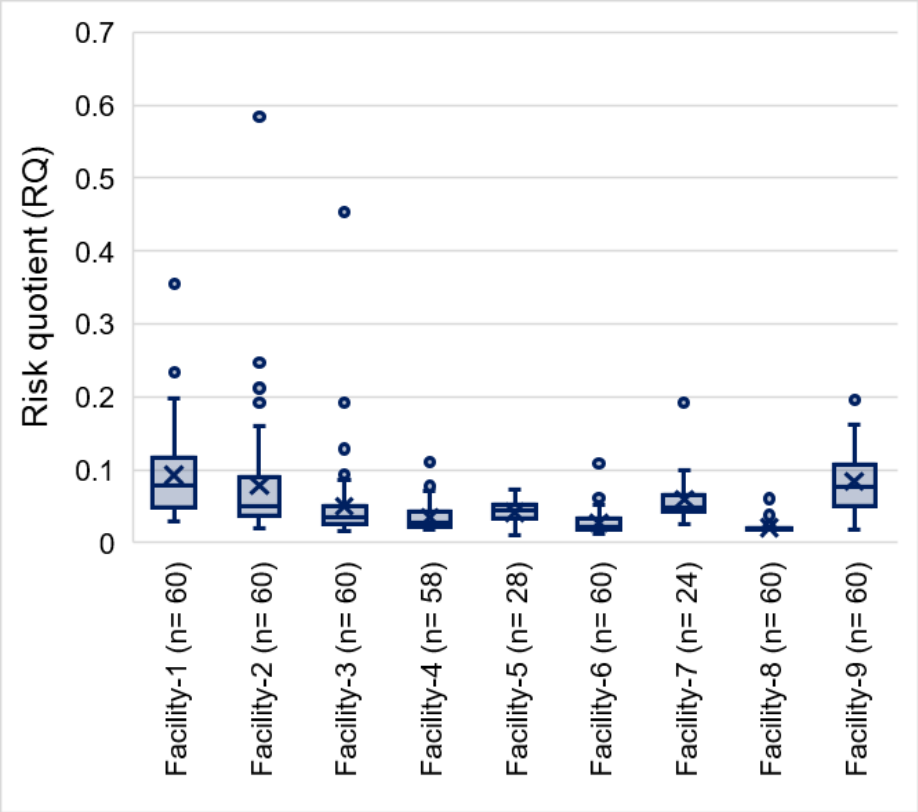

Figure 7-6. Risk quotients for electric power generation facilities between 2012 and 2016 (Ontario 2019)

The second source of risk quotients was the set of water quality monitoring studies from Wabamun Lake and a limited number of measurements in surrounding lakes (Alberta Environment 2002, 2003a, 2003b, 2006). Although somewhat dated and potentially confounded by the co-location of coal mining, these measured concentrations remain a reasonable worst-case scenario for the ecological risk characterization of coal-powered generation facilities. Although a small number of aluminium RQs in Wabamun Lake were slightly elevated relative to nearby lakes, low risk quotients were observed across the data set (Table 7-2).

Table 7-2. Summary of risk quotients in the Wabamun Lake area, Alberta

Location	Sample size	Minimum RQ	Q1 RQ	Average RQ	Median RQ	Q3 RQ	Maximum RQ
Nearby lakes	8	0.0018	0.0028	0.0085	0.0057	0.010	0.027
Wabamun Lake	125	0.0011	0.0075	0.021	0.010	0.018	0.22

Abbreviations: RQ, risk quotient; Q1, first quartile (25th percentile); Q3, third quartile (75th percentile)

Pulp and paper: Ecological risk characterization for the pulp and paper sector was based on two sources of information: reporting of aluminium concentrations in interpretive reports submitted to the EEM program under the PPER (Canada 2018b), and a voluntary sampling campaign and study organized by NCASI (2020).

Risk quotients calculated from data submitted to the EEM program under the PPER are presented in Figure 7-7. Of the eight facilities for which data were identified, only F-2 and F-3 demonstrated a high frequency of PNEC exceedances. However, concomitant exceedances in the respective reference areas suggest a low level of ecological concern. Furthermore, if only the most recent year of available data for F-3 are examined (n = 3), the risk quotients for exposure (average: 1.1) and reference (average: 1.3) areas are very similar.

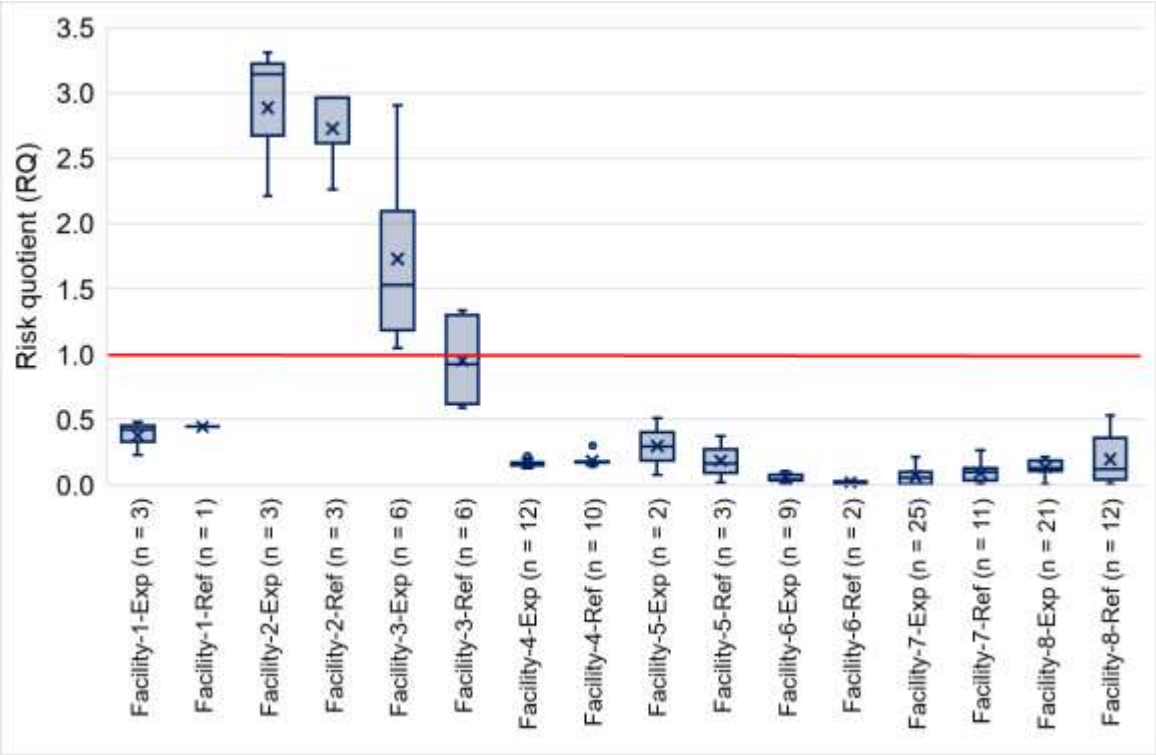

Figure 7-7. Risk quotients for pulp and paper facilities reporting aluminium concentrations to EEM under the PPER (Canada 2018b)

Risk quotients for the pulp and paper sector were also calculated from effluent and ambient water quality monitoring data submitted on samples collected from 27 Canadian mills from August 2018 to January 2019 (NCASI 2020). Mill-specific PNECs were calculated using pH and hardness data for the respective ambient waters and representative ecozone DOC values. RQs for the modelled exposure and ambient areas are presented in Figure 7-8. RQs were generally low and, at most, only very modestly increased in the estimated exposure areas relative to the corresponding ambient (primarily raw intake) waters, suggesting a low potential for ecological risk.

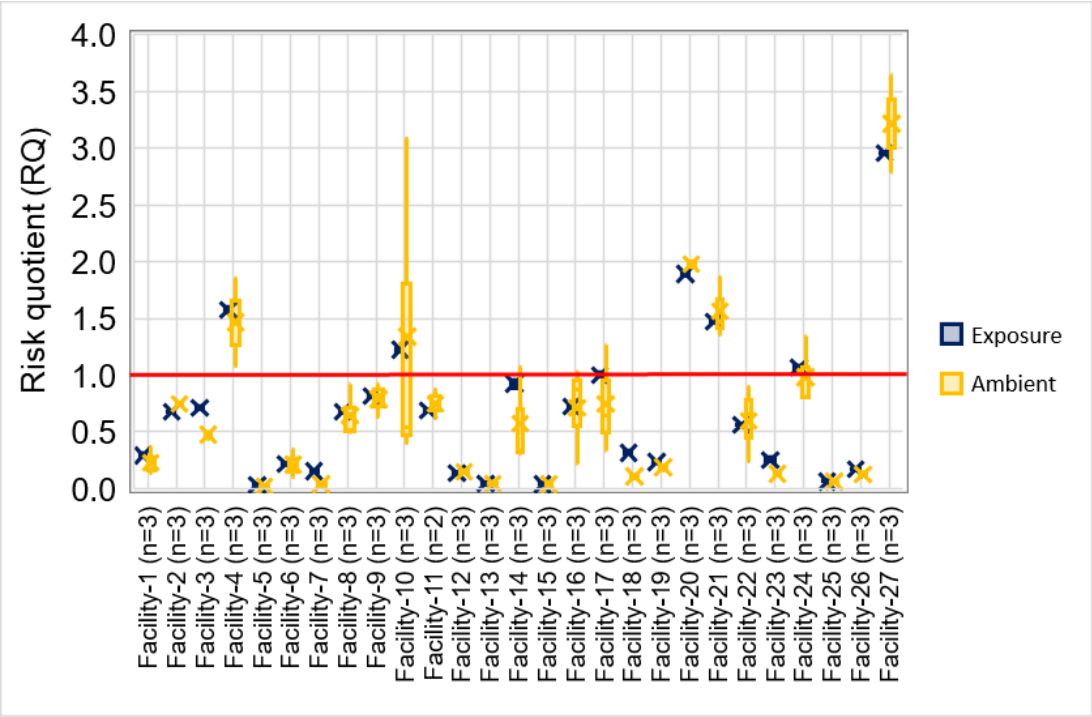

Figure 7-8. Pulp and paper sector risk quotients for 27 facilities releasing aluminium from August 2018 to January 2019 (NCASI 2020)

Canadian long-term surface water quality monitoring: To support the weight of evidence, aluminium RQs were calculated from long-term water quality monitoring data from 2011 to 2015 for stations with land use classifications available. Long-term water quality monitoring stations were assigned an approximate land use classification considering factors in the drainage basin. These included the percentage of cropland (>20%) and livestock intensity; the number of mines, mineral projects, and pulp, paper, or saw mills (either 0 or >0); the presence of upstream oilsands or shale gas areas; and the population density (<10 people/km^2 and >25 people/km^2 for remote and populated areas, respectively). Median RQs for stations with more than 10 measurements and more than 50% of their measurements within the domain of the FWQG are presented in Figure 7-9. Compared to remote stations, RQs are most elevated at stations classified for multiple industrial uses and areas classified as populated; however, exceedances are comparatively rare. The median RQ for one station classified as impacted by agriculture, forestry, and mining is greater than 1 (as are multiple stations classified as remote), but greater confidence is placed in the sector-specific exposure characterizations described above. Overall, this data set supports the weight of evidence for low potential for ecological concern.

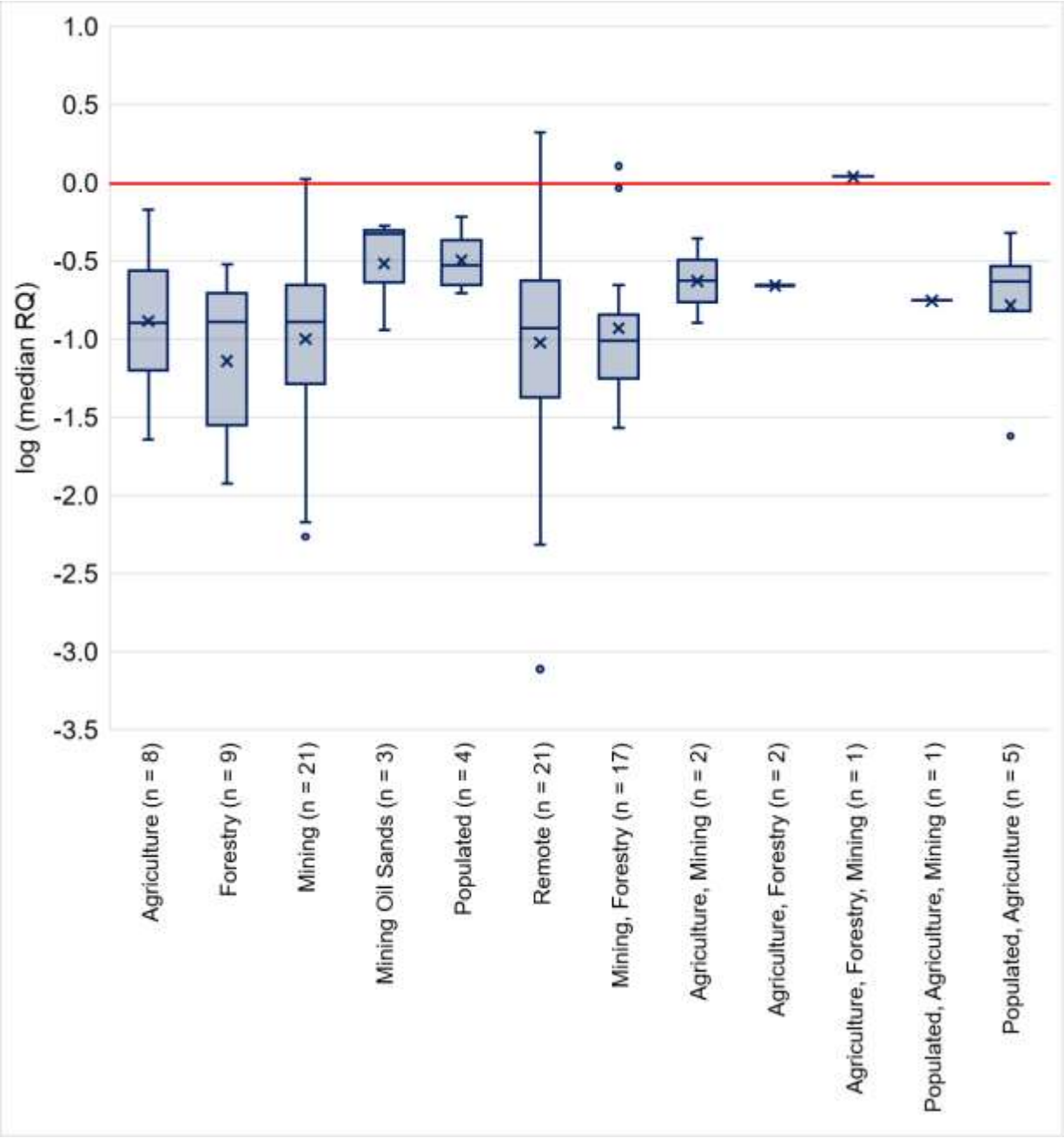

Figure 7-9. Median RQs for long-term water quality monitoring stations by land use classification (Canada [modified 2017a])

Bioavailable aluminium in Canadian soils: Considering the available phytotoxicity and soil invertebrate toxicity data, a soil PNEC of 1 mg/kg to 3 mg/kg $CaCl_2$-extractable aluminium was derived. While measured concentrations of total aluminium in Canadian soils are abundantly available and summarized in EC, HC (2010), concentrations of $CaCl_2$-extractable aluminium are comparatively rare, and no concentration data were available specific to any of the sectors described above for the aquatic compartment. However, Webber et al. (1977) reported a wide range of $CaCl_2$-extractable aluminium

concentrations in mostly acidic soils. Given the context of studying agricultural liming requirements, it was assumed that the samples from Webber et al. (1977) came from agricultural soils to which biosolids could potentially be applied. These data, presented in Figure 7-10, show that $CaCl_2$-extractable aluminium concentrations are not likely to exceed the PNEC as pH increases above 5.0. This would be consistent with US EPA (2003), which proposed to identify aluminium as a contaminant of potential concern only when soil pH is less than 5.5. More recent literature further supports the idea that aluminium toxicity is an important growth limiting factor for plants when pH is less than 5.0 to 5.5 (Rout et al. 2001; Bishop and Quin 2013; Ayeni et al. 2014).

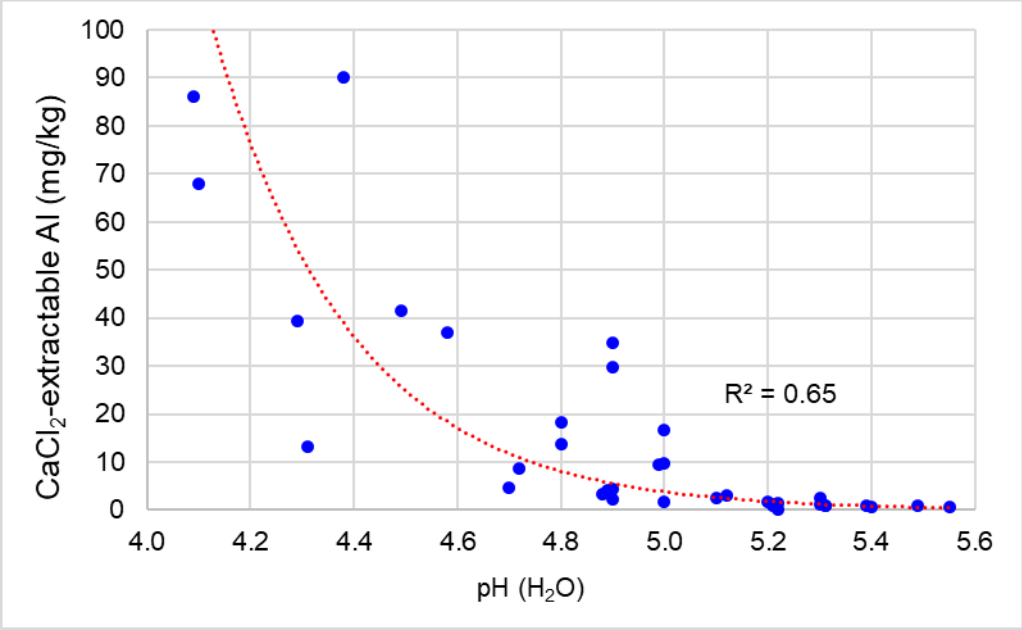

Figure 7-10. Bioavailable aluminium concentrations in Canadian soils (Webber et al. 1977)

While the applicability of the derived soil PNEC below pH ≤5.0 is debatable, application of a solely pH-based threshold for aluminium toxicity is also uncertain given the wide diversity in tolerance and properties of different species. For example, alfalfa yield is reduced below pH 6.0 not because of aluminium toxicity, but because of insufficient nitrogen fixation resulting from poor survival of rhizobium and inadequate nodulation (Rice 1975). Meanwhile, the desired pH for blueberries is 4.8; for corn, rye, grass hay, and pasture, the pH below which liming is beneficial is only 5.1 (OMAFRA 2018). Conversely, Whitley et al. (2016) showed that across many soil orders and climatic zones in New Zealand, $CaCl_2$-extractable aluminium concentrations in soils with pH from 5.5 to 5.7 could be as high as 5 mg/kg to 7 mg/kg. They also demonstrated that individual soil samples of up to pH 5.9 had $CaCl_2$-extractable aluminium concentrations that would exceed thresholds for sensitive pasture legumes.

Another important consideration is that ecological studies have tended not to be explicit about the types of acid soils used (Proctor 1999). In mineral soils with low pH, high

levels of bioavailable aluminium is indeed a major phytotoxicity factor. However, in organic acidic soils, mobile aluminium is virtually non-existent, and at very low pH, hydronium ions dominate the soil solution (Kidd and Proctor 2001). In other words, the direct toxicity of the hydronium ion is more likely the proximal cause of poor growth of non-tolerant plants in acidic organic soils, not the aluminium moiety (Kidd and Proctor 2001).

Nevertheless, the exposure scenario, which could result in the 55 aluminium-containing substances entering the soil compartment, would be constrained by the pH limitations placed on the application of water treatment system sludges. Additionally, most studies of aluminium exposure demonstrated minimal differences between sludge-amended soils at unrealistic rates of application and the corresponding controls (section 7.2.8). These lines of evidence support a low potential for ecological harm to the soil compartment from the 55 aluminium-containing substances.

7.3.2 Consideration of the lines of evidence

To characterize the ecological risk of the 55 aluminium-containing substances, technical information for various lines of evidence was considered (as discussed in the relevant sections of this report) and qualitatively weighted. The key lines of evidence supporting the proposed assessment conclusion are presented in Table 7-3, with an overall discussion of the weight of evidence provided in section 7.3.3. The level of confidence refers to the combined influence of data quality and variability, data gaps, causality, plausibility, and any extrapolation required within the line of evidence. The relevance refers to the impact the line of evidence has when determining the potential to cause harm in the Canadian environment. Qualifiers used in the analysis ranged from low to high, with the assigned weight having five possible outcomes.

Table 7-3. Weighted lines of key evidence considered to determine the potential for the 55 aluminium-containing substances to cause harm in the Canadian environment

Line of evidence	Level of confidence[a]	Relevance in assessment[b]	Weight assigned[c]
Environmental fate and behaviour	High	Moderate	Moderate to high
Persistence in the environment	High	Moderate	Moderate to high
Bioaccumulation in aquatic and terrestrial organisms	High	Low	Moderate
PNEC for freshwater aquatic organisms (chronic FWQG)	High	High	High
PNEC for soil-dwelling organisms	Moderate	Moderate	Moderate
Qualitative hazard analysis for sediment-dwelling organisms	Moderate	Low	Low to moderate

Line of evidence	Level of confidence[a]	Relevance in assessment[b]	Weight assigned[c]
PECs in surface water – primary aluminium manufacturing	Moderate	High	Moderate to high
PECs in surface water – cement manufacturing	Low	High	Moderate
PECs in surface water – metal mining	High	High	High
PECs in surface water – electric power generation	Moderate	High	Moderate to high
PECs in surface water – pulp and paper	Moderate	High	Moderate to high
PECs in surface water – long-term surface water quality monitoring	Moderate	Moderate	Moderate
PECs in soil ($CaCl_2$-extractable)	Low	Low	Low
RQs for surface water – primary aluminium	Moderate	High	Moderate to high
RQs for surface water – cement manufacturing	Low	High	Moderate
RQs for surface water – metal mining	High	High	High
RQs for surface water – electric power generation	Moderate	High	Moderate to high
RQs for surface water – pulp and paper	Moderate	High	Moderate to high
RQs for surface water – long-term surface water quality monitoring	Moderate	Moderate	Moderate
RQs for soil ($CaCl_2$-extractable)	Low	Moderate	Low to moderate

[a] Level of confidence is determined according to data quality, data variability, and data gaps (that is, are the data fit for purpose).
[b] Relevance refers to the impact of the evidence in the assessment.
[c] Weight is assigned to each line of evidence according to the overall combined weights for level of confidence and relevance in the assessment.

7.3.3 Weight of evidence for determining potential to cause harm to the Canadian environment

Once released into the environment, the 55 aluminium-containing substances have the potential to dissolve, dissociate, or degrade to contribute to total aluminium exposure. The fate and behaviour of substances in the group is governed by the physical and

chemical properties of the receiving environment (for example, pH, redox conditions, organic matter). A substance contributing significantly to total aluminium exposure under one set of conditions may provide an insignificant contribution under another set of conditions. The aluminium that can be mobilized from the 55 aluminium-containing substances is persistent; it will remain in the environment and partition among environmental compartments according primarily to pH and organic matter content. Aluminium is not a nutritionally essential element and, except for certain tolerant and hyperaccumulating plant species, is not considered bioaccumulative.

The chronic FWQG was selected as the PNEC for the aquatic environment. The aquatic toxicity of aluminium is strongly dependent upon factors such as pH, hardness, and DOC. These parameters, collectively referred to as TMFs, were taken into account in the chronic FWQG in a multiple linear regression (MLR) approach to normalizing the toxicity data set (ECCC 2022a). There was a high degree of confidence in the resulting PNECs when they were calculated with paired or site-specific TMF measurements (for example, metal mining, and pulp and paper sectors), and a moderate level of confidence when substitution with representative ecozone values was required.

Large quantities of the 55 aluminium-containing substances were reported in response to CEPA section 71 surveys for both import and manufacturing. These quantities, when passed through generic aquatic exposure scenarios and compared with the solely pH-dependent CCREM (1987) guideline, resulted in an initial ecological risk classification of higher potential for ecological concern (CCME 2008; ECCC 2020b). The present assessment, however, considered sector-specific monitoring data where available or refined exposure scenarios, resulting in a higher confidence in derived PECs. Alignment of the aquatic PNEC with the more recent FWQG also results in a higher degree of confidence as the FWQG considers more recent data and incorporates additional toxicity modifying factors. In light of these changes, risk quotients in the aquatic compartment for facilities in all sectors rarely exceeded unity or exceeded unity only by relatively small magnitudes, or exposure site exceedances were congruent with exceedances in corresponding reference areas.

The lines of evidence for the potential for the 55 aluminium-containing substances to cause ecological harm in the sediment compartment are of low relevance to the overall assessment. This is primarily because the aluminium substances with a greater likelihood of being used in activities resulting in exposures to the sediment compartment (that is, eutrophication treatments) have been previously addressed (EC, HC 2010). However, a literature search was conducted to identify any new lines of evidence from a “total aluminium” perspective, considering the possibility that some of the 55 aluminium-containing substances could be substitutes for these uses, even though no such uses were reported (Environment Canada 2013; ECCC 2017). Studies and reviews published in the intervening years were somewhat limited but remained supportive of the lines of evidence of the previous assessment.

Finally, the potential for the 55 aluminium-containing substances to cause ecological harm in the soil compartment is low when considering the available lines of evidence. In

the majority of studies reviewed, the application of aluminium-containing sludge did not significantly alter extractable (bioavailable) aluminium concentrations, uptake into vegetation relative to controls, or vegetation (crop) yield. The exposure scenario most likely to result in the 55 aluminium-containing substances entering the soil compartment would be further constrained by the pH and application rate limitations.

This information indicates that the 55 aluminium-containing substances have low potential to cause ecological harm in Canada.

7.3.4 Sensitivity of conclusion to key uncertainties

The chemical similarities of the substances in this group—namely their capacity to dissolve, dissociate, or degrade to contribute to the total exposure of organisms to aluminium—warranted using a read-across approach for ecotoxicity data. The potential for aluminium to cause adverse effects is also well documented. Therefore, additional empirical toxicity studies would not likely change the proposed conclusion. Additional information on the bioaccumulative potential of aluminium would similarly have a low impact on the proposed conclusion. However, certain organic-metal salts or organometallics, in addition to potentially contributing to total aluminium exposure, may also exert their own effects through another mode of action, for example, as a parent compound or through an organic transformation product. Additional information on the stability and effects of individual parent compounds could inform the need for more specific assessment of those substances (OECD 2015).

The choice of ecological exposure scenarios developed for the Aluminium-containing Substances Group was informed by the relative magnitude of commercial activity obtained from responses to CEPA section 71 surveys and relative magnitude of releases reported to the NPRI. In the absence of particular data, realistic assumptions were made in order to estimate PECs. For example, in the case of the cement manufacturing scenario, refinement of the emission factor or preferably, more recent monitoring of effluents or receiving environments for metals such as aluminium would help increase the certainty in the PECs and risk characterization. More recent effluent monitoring data or preferably, receiving environment concentrations for the primary aluminium manufacturing sector would similarly increase confidence.

PNECs for surface water were calculated using the MLR-based FWQG, incorporating pH, DOC, and hardness. Paired TMF measurements were preferred but not always available, necessitating the use of estimated values as described in section 7.2.1. The use of a central tendency to substitute for an unavailable or non-existent measured value is more representative of an average case than a realistic worst case and may lead to an underestimation of effects for certain areas. However, for modelled scenarios, where substitution was most often necessary, the use of a central tendency also reduces the likelihood of compounding with other conservative assumptions.

For the analysis of metal mining and pulp and paper EEM data, RQs were calculated by comparing exposure PECs to exposure PNECs and reference PECs to reference

PNECs. Exposure RQs calculated this way may be considered under-protective or erroneous as they do not take into account possible perturbations of TMFs also caused by the industrial activity. Conversely, the alternative method of calculating exposure RQs as exposure PECs divided by reference PNECs does not represent a currently existing scenario. To determine the potential implications of this choice on the conclusion, additional RQs comparing exposure PECs to corresponding reference PNECs were calculated and are presented as supporting information (ECCC 2020a). The distributions of these alternate RQs were not significantly different from those presented above, indicating that the conclusion would not be sensitive to this uncertainty.

8. Potential to cause harm to human health

8.1 Health effects assessment

Several national and international organizations have reviewed the health effects of exposure to aluminium substances in the general population (WHO 2010; US EPA 2006; JECFA 2007, 2012; ATSDR 2008; EFSA 2008; EC, HC 2010; RIVM 2020; SCCS 2020; Health Canada 2021a). In addition, there are several literature reviews that have assessed the human health risks of aluminium (Krewski et al. 2007; Willhite et al. 2014; Tietz et al. 2019). The health effects on workers exposed to aluminium under occupational settings were also assessed by several international organizations, including the American Conference of Governmental Industrial Hygienists (ACGIH) (2008), the International Agency for Research on Cancer (IARC) (2010), the Health Council of the Netherlands (2010), and the Senate Commission for the Investigation of Health Hazards of Chemical Compounds in the Work Area (MAK) (2014). These existing reports were used to inform the health effects section of this assessment.

The assessment approach for human health is outlined in the Science Approach Document Biomonitoring-based Approach 2 (Health Canada [modified 2016]). In this approach, human biomonitoring data are compared to biomonitoring guidance values that are consistent with available health-based guidance values in order to identify if substances are of low concern for human health. Additionally, portal-of-entry effects in the lungs are considered separately as the BE value is representative of systemic health effects. The health effects assessment is focused on the aluminium moiety. However, a literature search on health effects and toxicokinetic data available on individual substances within the Aluminium-containing Substances Group was conducted to assess the potential for increased toxicity above the toxicity of the aluminium moiety.

8.1.1 Toxicokinetics and adequacy of biomarker

Relatively few toxicokinetic studies are available for aluminium mainly due to analytical difficulties associated with aluminium measurement up until the 1990s (see section 8.2.3) (Priest 2004). Aluminium ingested via the oral route is poorly absorbed through the gastrointestinal tract. The absorption varies depending on physiological and

chemical factors, such as solubility of the aluminium substance, gastric pH, and presence of various dietary intakes (Krewski et al. 2007; EC, HC 2010). In humans, the absorption of soluble aluminium complexed with citrate, chloride, hydroxide, or lactate are in the range of 0.01% to 0.8% (Keith et al. 2002; Priest 2004; ATSDR 2008; RIVM 2020; Health Canada 2021a). The absorption of aluminium citrate was estimated at 2.18% in rabbits (RIVM 2020). The absorption of aluminium lactate was measured at 0.78% in humans, and some investigators considered this value to be representative of the aluminium absorption for infants (Keith et al. 2002; Mitkus et al. 2011).

Dermal absorption of aluminium is expected to be minimal. According to the data from a clinical study conducted on six female subjects, the SCCS (2020) established a mean dermal absorption of 0.00052% for aluminium.

About 90% of aluminium in plasma is bound to transferrin, and the rest is bound to low molecular weight molecules (mainly citrate) (Priest 2004; JECFA 2012; RIVM 2020). Cellular uptake of aluminium likely occurs as transferrin-bound aluminium by transferrin receptor-mediated endocytosis (JECFA 2012). Some investigators believe that aluminium bioavailability has an inverse relationship with iron status (ATSDR 2008). From blood, aluminium is slowly taken up by tissues and organs and mainly distributed to bones. For both adults and infants, bone is the primary long-term reservoir for systemic aluminium exposure via oral intake or injection (Mitkus et al. 2011). To a lesser extent, aluminium is distributed to the brain by crossing the blood-brain barrier and the blood–cerebrospinal fluid barrier (RIVM 2020; Health Canada 2021a). Aluminium is detected in most soft tissues and organs (Krewski et al. 2007; ATSDR 2008; EFSA 2008; EC, HC 2010; JECFA 2012; Willhite et al. 2014; Health Canada 2021a). According to data from intravenous (i.v.) studies, the elimination half-life from plasma varies from 14 hours to 85 days (Priest 2004; Klotz et al. 2019). It is widely accepted that aluminium is exclusively partitioned into plasma, and therefore, at steady state, serum and whole blood aluminium concentrations are approximately equal because almost all aluminium is found in plasma (Krewski et al. 2007; ATSDR 2008). Conversely, a review by Priest (2004) stated that approximately 10% of aluminium could be segregated to erythrocytes.

Aluminium has a high affinity for organic ligands; as a result, most of the aluminium in the body exists in the form of macromolecular complexes, which can be very stable (ATSDR 2008). Thus, aluminium metabolism is determined by its affinity to ligands and the type of complexes formed (Health Canada 2021a).

In humans, approximately 95% of absorbed aluminium is excreted in urine, while approximately 2% is excreted by biliary excretion via the feces (Krewski et al. 2007; EC, HC 2010; Health Canada 2021a). Urine elimination showed a multi-phasic elimination pattern, with an elimination half-life ranging from hours to years (Priest et al. 1995; Talbot et al. 1995; Priest 2004; Krewski et al. 2007). The initial elimination half-lives in rats, mice, rabbits, and dogs ranged from 2 to 5 hours, whereas for humans it was less than a day (JECFA 2012). There is no significant difference in urinary aluminium elimination in adults and infants (Mitkus et al. 2011). The elimination half-life of

aluminium from the brain and other soft tissues is >100 days. Elimination half-life from the lungs was approximately 100 days (Krewski et al. 2007). The rate of elimination depends primarily on the type of complexes formed; aluminium citrate complexes are more readily eliminated than transferrin-bound aluminium (Health Canada 2021a). Further details on aluminium toxicokinetics can be found in EC, HC (2010) and Health Canada (2021a).

Evidence from multiple studies indicates that inhaled aluminium and aluminium oxide particles are cleared from the lung by alveolar macrophages to the lymphatic system (Christie et al. 1963; Gross et al. 1973; Pigott et al. 1981; Thomson et al. 1986). Gross et al. (1973) noted macrophages filled with particles in satellite lymph nodes of guinea pigs, hamsters, and rats exposed to aluminium oxide or aluminium powder for 6 or 12 months. One year post-exposure, aluminium powder was partially or completely cleared from the airways in all three species (Gross et al. 1973). Christie et al. (1963) observed that histiocytes in the hilar lymph nodes of rats and hamsters contained cytoplasmic dust granules after exposure to 100 mg/hour of McIntyre Powder (80% aluminium oxide, 20% aluminium) for 13 months. Wet weight of the lungs increased significantly after exposure in comparison to the control animals; however, lung weights gradually declined 3, 6, and 10 months post-exposure. The same trend was observed for the ash and aluminium oxide content of the lungs. The authors concluded that McIntyre Powder is cleared from the lungs after cessation of exposure (Christie et al. 1963). Similar observations were made in an acute inhalation study in rats using aluminium powder (Thomson et al. 1986). Pathological examination performed 14 days post-exposure revealed aluminium particles contained within histiocytes in the hilar lymph nodes. Finally, a study that exposed rats to aluminium oxide fibres observed fibres contained within alveolar macrophages in the mediastinal lymph nodes after 86 weeks of exposure (Pigott et al. 1981). In comparison, animal studies have indicated that aluminium chlorohydrate is primarily retained in the lungs after inhalation exposure (ATSDR 2008). Rats and guinea pigs administered aluminium chlorohydrate for up to 24 months showed accumulation of aluminium primarily in the lungs (Steinhagen and Cavender 1978; Stone et al. 1979). Other organs with significant accumulation included the adrenal glands (Stone et al. 1979) and the peribronchial lymph nodes (Steinhagen and Cavender 1978; Stone et al. 1979). There was no significant accumulation of aluminium observed in the brain, heart, spleen, kidneys, or liver of either species (Steinhagen and Cavender 1978; Stone et al. 1979). A fraction of inhaled aluminium can be absorbed to the systemic circulation. On the basis of occupational studies that characterized the relationship between urinary aluminium excretion and airborne soluble aluminium, a fractional absorption of approximately 1.5% to 2% via the inhalation route was proposed by Yokel and McNamara (2001).

Biomarker adequacy

Aluminium is measured in serum, plasma, whole urine, hair, and feces. Aluminium in serum, plasma and whole blood is expected to be equivalent because the majority of aluminium is found in plasma (ATSDR 2008).

Older assessments, such as ATSDR (2008), did not consider biological matrices such as serum and urine to be sensitive enough as biomarkers of exposure. They considered that the exposure levels could not be accurately related to serum or urine concentrations due to the poor absorption of aluminium by any route. Additionally, aluminium oral absorption in particular can be affected by other concurrent intakes (ATSDR 2008). However, more recent investigators have suggested that these matrices may be useful biomarkers of exposure based on the analysis of occupational biomonitoring studies and review of kinetic data (Klotz et al. 2017, 2019; Ferguson et al. 2018).

Several epidemiological studies have measured both blood and urine aluminium concentrations in workers who were exposed to aluminium predominantly via the inhalation route in the workplace. These studies showed a significant increase in both urine and blood (measured in serum or plasma) aluminium concentrations with increased aluminium exposure in the workplace (Hosovski et al. 1990; Riihimäki et al. 2000; Polizzi et al. 2002; Kraus et al. 2006; Kiesswetter et al. 2007; Giorgianni et al. 2014; Zawilla et al. 2014). However, there were limited data available for internal concentrations in either blood or urine associated with increased exposure via the oral route.

Based on evidence from a systemic review of the available kinetic data, Ferguson et al. (2018) concluded that concentrations of aluminium in blood and urine (biomonitoring measurements) are likely suitable biomarkers for quantifying exposure when there is stable and continuous exposure to aluminium.

Daily or frequent exposure to aluminium in the general population of Canada is expected because the primary exposure source for the general population is environmental media (soil, drinking water, air) and food. Therefore, blood aluminium concentration in the general population is likely at steady state. Furthermore, aluminium concentration in whole blood has an added advantage as a biomarker because it provides aluminium concentrations closer to the target organs (that is, the nervous system).

Available evidence from kinetic studies and occupational epidemiology studies support the conclusion that the aluminium concentration in blood (that is, whole blood, plasma, and serum) is a suitable biomarker for quantifying exposure in the general population.

8.1.2 Health effects from oral route of exposure

There is a large data set that examines the association between aluminium exposure and health effects in experimental animals and in workers. In both humans and animals, repeated dose exposure to aluminium is linked to effects in bones and kidneys, neurological effects, and reproductive/developmental effects, including neurodevelopmental and neurobehavioural effects (Krewski et al. 2007; ATSDR 2008; Willhite et al. 2014; Tietz et al. 2019; Health Canada 2021a). The overall data set indicates that the nervous system is the most sensitive target organ for aluminium-

induced toxicity from systemic exposure in both experimental animals and humans (Krewski et al. 2007; ATSDR 2008; Willhite et al. 2014; Health Canada 2021a). Multiple mechanisms have been proposed for aluminium toxicity, including oxidative damage from reactive oxygen species generation and lipid peroxidation, inflammatory responses, changes in neuroskeletal proteins, and membrane effects due to metal-ion imbalance (ATSDR 2008). In a recent study, Tsialtas et al. (2020) indicated that the mechanism of aluminium chlorohydrate toxicity is associated with its ability to interfere with estrogen receptor signalling. Similar findings were reported by other authors (Gorgogietas et al. 2018). It should be noted that individuals with compromised kidney function are at an increased risk for systemic aluminium toxicity as the kidney is the primary route of elimination for absorbed aluminium (Willhite et al. 2014).

Acute toxicity

Acute toxicity of oral exposure to various aluminium salts has been studied in rats and mice. The LD_{50} values for the oral route of exposure have been reported to be between 162 mg/kg bw and 980 mg/kg bw (MAK 2014).

Short-term toxicity

There are fewer well-conducted short-term oral toxicity studies available compared to sub-chronic and chronic toxicity studies for aluminium. Neurological effects (degenerative changes in the prefrontal cortex) were reported in male rats exposed to 101 mg Al/kg bw/day in drinking water for 30 days (Akinola et al. 2015). When male mice were exposed to 0, 300, or 600 mg Al/kg bw/day as aluminium nitrate for 14 days, decreased motor coordination was reported at 600 mg Al/kg bw/day (Colomina et al. 1999).

Chronic oral exposure

Several international organizations have previously established health-based guidance values for aluminium in the general population exposed via food, products available to consumers, and environmental media (JECFA 2007, 2012; ATSDR 2008; EFSA 2008).

The European Food Safety Authority (EFSA) (2008) evaluated the safety of aluminium from dietary intake. When deriving their guidance values, the EFSA panel took into consideration that aluminium has the potential to produce neurotoxicity (in mice and rats), male reproductive system effects (in dogs), embryotoxicity (in mice), and neurodevelopmental effects (in mice and rats). The Panel therefore based its evaluation on the combined evidence from several studies, which resulted in lowest-observed-adverse-effect levels (LOAELs) for effects on neurotoxicity, reproductive toxicity, embryotoxicity, and neurodevelopmental toxicity of 52 mg Al/kg bw/day, 75 mg Al/kg bw/day, 100 mg Al/kg bw/day, and 50 mg Al/kg bw/day, respectively. Similarly, the lowest values for no-observed adverse-effect levels (NOAELs) were reported at 30 mg Al/kg bw/day, 27 mg Al/kg bw/day, 100 mg Al/kg bw/day, and between 10 mg Al/kg bw/day and 42 mg Al/kg bw/day for effects on neurotoxicity, reproductive toxicity,

embryotoxicity, and neurodevelopmental toxicity, respectively. The studies selected were conducted using aluminium lactate, which is a form of aluminium with relatively high solublility. Based on these data, the EFSA (2008) derived a tolerable weekly intake (TWI) of 1 mg Al/kg bw/week using a NOAEL and a LOAEL of 10 mg Al/kg bw/day and 50 mg Al/kg bw/day, respectively, based on neurodevelopmental toxicity in mice and rats. To derive a TWI of 1.2 mg Al/kg bw/week using the LOAEL of 50 mg Al/kg bw/day, the panel applied an uncertainty factor (UF) of 300 to account for interspecies variation (x10), intraspecies variation (x10), and the extrapolation from a LOAEL to a NOAEL (x3). To derive a TWI of 0.7 mg/kg bw/wk using the NOAEL (10 mg Al/kg bw/day), a UF of 100 was applied to account for interspecies variation (x10) and intraspecies variation (x10). The EFSA (2008) used the average of the TWI values from the NOAEL and the LOAEL to derive their final TWI of 1 mg Al/kg bw/week. The EFSA established a TWI rather than a tolerable daily intake (TDI) because of the cumulative nature of aluminium in the organism after dietary exposure.

ATSDR (2008) selected a LOAEL of 100 mg Al/kg bw/day based on neurological effects, significant decreases in forelimb and hindlimb grip strength, and a decrease in thermal sensitivity in mice exposed to 100 mg Al/kg/day as aluminium lactate in the diet during gestation, lactation, and postnatally until 2 years of age (Golub et al. 2000). A UF of 300 was applied to the LOAEL to account for interspecies variation (x10), intraspecies variation (x10), and extrapolation from a LOAEL to a NOAEL (x3). Additionally, a modifying factor of 0.3 was applied to account for the greater bioavailability of aluminium lactate compared to other aluminium compounds to which the general population is more likely to be exposed. The resulting minimal risk level (MRL), based on neurological effects and identified by ATSDR, was 1 mg Al/kg bw/day.

In 2010, Health Canada and Environment Canada published a PSL2 assessment on three aluminium compounds (aluminium chloride, aluminium nitrate, and aluminium sulphate) (EC, HC 2010). Similar to EFSA (2008), this assessment considered the fact that there is no single study that provides an adequate basis for characterizing the dose-response relationship of aluminium. Therefore, the PSL Report considered a composite LOAEL of 50 mg/kg bw/day as the critical point of departure (POD) (EC, HC 2010). Compared to lower doses, this dose level produced neurological, neurodevelopmental, and reproductive effects in laboratory animals (primarily rats and mice) more consistently under a wide range of experimental conditions (EC, HC 2010). In these studies, animals were exposed to various forms of aluminium, including aluminium chloride, aluminium citrate, aluminium lactate, aluminium hydroxide, and aluminium nitrate (EC, HC 2010).

The Joint FAO/WHO Expert Committee on Food Additives (JECFA) re-evaluated their health-based guidance value for aluminium in food additives in 2012 as 1 mg Al/kg bw. The critical POD for the derivation of the provisional tolerable weekly intake (PTWI) was identified from a developmental and chronic neurotoxicity study by Poirier et al. (2011). The study was a double-blind, vehicle-controlled randomized study conducted in accordance with good laboratory practice (GLP) and OECD guideline 426. In this study, aluminium citrate, which is the most soluble and the most bioavailable aluminium

compound that can cross the blood–brain barrier, was administered to male and female rats during gestation (*in utero*) through lactation, until one year of age, in drinking water at 0, 30, 100, or 300 mg Al/kg bw/day. In addition, animals were exposed to a low amount of aluminium from the feed since the feed contained approximately 10 µg/g aluminium. However, the dietary exposure was not added to the administered dose because dietary exposure was minimal compared to the tested aluminium dose. During the experiment, endpoints assessed in dams included: daily morbidity and mortality checks, Functional Observational Battery (FOB) examinations on gestational days 7 and 13, a clinical examination on the day of delivery, and FOB examinations on post-natal days (PNDs) 3 and 10. The endpoints assessed in both female and male pups included: behavioural (motor activity [PNDs 15 or 16, 17, 21, 62, 117 and 363]), T-maze (PND 22), auditory startle, FOB, grip strenth test, Morris swim maze- (PNDs 59, 61, 63 and 64; PNDs 114, 116, 118 and 119; and PNDs 357, 359, 361 and 362), brain weight, clinical chemistry, hematology, tissue/blood levels of aluminium, and neuropathology. FOB examinations were done on PNDs 5, 11, 22, 36, 45, 56, and biweekly thereafter until the week of PND 350.

The most prominant treatment-related effect observed in the pups in Poirier et al. (2011) was renal pathology, predominantly in the male pups. In addition, dose-dependent increment in deficits in neuromuscular functions, measured by hindlimb and forelimb grip strength test and landing foot splay, was reported. Aluminium exposure during gestation and/or lactation consistently resulted in decreases in forelimb and/or hindlimb grip strength and increased landing foot splay in pups; thus, grip strength and landing foot splay were considered to be the most sensitive endpoints of aluminium-induced neurotoxicity (ATSDR 2008).

Poirier et al. (2011) reported deficits in fore- and hindlimb grip strength, foot splay, and renal effects in animals exposed to 100 mg/kg bw/day. Thus, the dose level of 100 mg Al/kg bw/day was considered a LOAEL, and 30 mg Al/kg bw/day was considered a NOAEL. Based on this NOAEL and through the application of a safety factor of 100 to account for interspecies (x10) and intraspecies (x10) variation, the Committee established a PTWI of 2 mg Al/kg bw. The previous PTWI of 1 mg/kg bw was also withdrawn (JECFA 2007). The daily intake associated with the current JECFA guidance value is 0.3 mg Al/kg bw/day.

In 2021, Health Canada published a drinking water quality guideline for aluminium. To derive their maximum acceptable concentration (MAC) in drinking water, Health Canada (2021) used the same POD selected by JECFA (2012) from Poirier at al. (2011) (NOAEL = 30 mg Al/kg bw/day). A UF of 100 was applied to the POD to account for interspecies (x10) and intraspecies (x10) variation. The resulting MAC for total aluminium concentration in drinking water is 2.9 mg/L.

The German Federal Environmental Agency (Umweltbundesamt) has established provisional reference values for aluminium for the general population as <15 µg/L for urine, <5 µg/L for serum, and approximately 13 µg/L plasma to protect against early signs of aluminium-induced neurotoxicity (Klotz et al. 2017). However, because these

values were determined on the basis of background aluminium exposure in the general population, a health-based guidance value was not considered in the derivation (Klotz et al. 2017).

The JECFA guidance value of 0.3 mg Al/kg bw/day, which was based on the developmental and chronic neurotoxicity study by Poirier et al. (2011), is the most appropriate guidance value for the hazard characterization of systemic exposure to aluminium and was selected for the derivation of the BE (section 8.1.4).

Reproductive and developmental toxicity

Studies in experimental animals have indicated that oral exposure to aluminium substances can lead to reproductive toxicity in male animals (MAK 2014; Health Canada 2021a). Male rats exposed to 75 mg Al/kg bw/day as $AlCl_3 \cdot 6H_2O$ in drinking water for 12 weeks experienced decreased body weight gain, absolute and relative testis weight, absolute seminal vesicle weights, and changes in sexual behaviour and aggression. The changes in sexual behaviour, aggression, and the reduction in weights of reproductive organs were attributed to the simultaneous retardation in body weight gain (MAK 2014). There were no effects on fertility (Bataineh et al. 1998 as cited in MAK 2014). Other studies have shown reproductive effects in both males and females at lower doses; however, these studies were not considered adequately robust to determine critical PODs. Most of the developmental studies, including Poirier et al. (2011) (the critical study for chronic toxicity) were focused on aluminium-induced neurodevelopmental effects, and therefore, other developmental effects, such as malformations, were not well reported. A recent study indicated that *in utero* and postnatal exposure to aluminium (as aluminium sulfate) may result in hematological and immunological impairment in female offspring of rats; however, the study did not evaluate immunological effects in male offspring or the histopathology of primary and secondary lymph organs such as the thymus and spleen (Omran 2019). Additional details of the reproductive and developmental toxicity studies can be found in the 2010 and 2021 Health Canada assessments (EC, HC 2010; Health Canada 2021a).

Genotoxicity

Aluminium compounds are not generally considered to be mutagenic but appear to act as clastogens and likely act through indirect mechanisms of action. This is not expected to be relevant to the general population, given that it only occurs at relatively high levels of exposure. Details of genotoxicity can be found in ATSDR (2008), EFSA (2008), MAK (2013), and Health Canada (2021a).

Carcinogenicity

There is no evidence for carcinogenicity of aluminium in animals exposed via oral or inhalation routes (Health Canada 2021a). There are no carcinogenicity classifications attributed solely to aluminium compounds (Krewski et al. 2007). Further details on carcinogenicity are discussed under section 8.1.3.

Epidemiological data

Many investigators have studied the relationship between aluminium exposure and onset of Alzheimer's disease in humans as well as the relationship between aluminium exposure and breast cancer.

The current evidence related to the risk of Alzheimer's disease from elevated aluminium exposure is conflicting. In a meta-analysis, Wang et al. (2016) found an increased risk for Alzheimer's disease in individuals chronically exposed to aluminium in drinking water. In contrast, several studies found no association between significantly high aluminium exposure in occupational settings and Alzheimer's disease (Salib and Hillier 1996; Virk and Eslick 2015). Some studies reported elevated aluminium concentrations in the brains of Alzheimer's patients (Klotz et al. 2017; Lukiw et al. 2019). However, it is unclear whether aluminium is the cause of the change or whether the accumulation is due to the Alzheimer's pathology (Bhattacharjee et al. 2013; Klotz et al. 2017). Walton and Wang (2009) noted that aluminium accumulated in brain tissues can increase amyloid precursor protein (APP) levels in neural cells, leading to formation of amyloid plaques in the brain. While amyloid plaque formation is observed in Alzheimer's patients, amyloid plaques are generally absent from rat and mouse brains (Walton and Wang 2009). Overall, the epidemiological data set provides only uncertain indications of an association between aluminium exposure and Alzheimer's disease (Klotz et al. 2017, 2019). Although recent reviews and international assessments consistently conclude that there is insufficient evidence for a causal link between exposure to aluminium and Alzheimer's disease, there is also consensus that the hypotheses should not be dismissed (ATSDR 2008; EFSA 2008; EC, HC 2010; JECFA 2012; Willhite et al. 2014).

Aluminium chlorohydrate substances, which are active ingredients of antiperspirants, have been linked as a causative agent for breast cancer (Darbre 2005; Darbre et al. 2013; Klotz et al. 2017, 2019). Similar to Alzheimer's disease, the epidemiological evidence for aluminium-induced breast cancer is inconsistent. A retrospective study showed an earlier age of disease onset in breast cancer patients who had used aluminium-containing antiperspirants/deodorants accompanied by axillary shaving (McGrath 2003). Darbre (2005) showed that aluminium forms found in antiperspirant could interfere with the function of estrogen receptors of MCF7 human breast cancer cells both in terms of ligand binding and estrogen-regulated reporter gene expression.

In a series of studies, elevated levels of aluminium were observed in breast cancer tissues (Mannello et al. 2011; Darbre et al. 2013; Klotz et al. 2017). However, it is unclear whether aluminium is the trigger for breast tumours or whether aluminium has a preferential accumulation in tumour tissues, similar to other minerals (Manello et al. 2011; Klotz et al. 2017). A population-based case-control study did not identify a link between the use of antiperspirants or deodorants on shaved skin and the risk of breast cancer in women aged 24 to 74 years (Mirick et al. 2002). Similar findings were reported by Fakri et al. (2006) for antiperspirant (not deodorant) use based on a case-control study on 54 women with breast cancer. A systematic analysis of the published literature showed no increased risk of breast cancer associated with antiperspirant use (Namer et

al. 2008). In a study conducted to understand the mechanism of aluminium toxicity and its implications in estrogen receptor-related breast cancer development, Gorgogietas et al. (2018) reported that aluminium chlorohydrate induced a significant increase in estrogen receptor protein level, possibly via interference with estrogen receptor gene expression or estrogen receptor protein stability. However, the authors agreed that their findings do not provide conclusive evidence that aluminium is a breast carcinogen. Overall, the link between aluminium exposure and breast cancer is not consistently supported by scientific evidence (Klotz et al. 2017).

8.1.3 Health effects from inhalation route of exposure

A literature search for health effects specific to the inhalation route of exposure was conducted using 22 of the 55 aluminium-containing substances as search terms. According to available exposure information, these 22 substances have uses that could result in inhalation exposure to the general population. From the results of the literature search, it was determined that inhalation toxicity studies for the substances in the Aluminium-containing Substances Group were conducted primarily using aluminium chlorohydrate, aluminium oxide, or aluminium powder. Aluminium chlorohydrate is soluble, whereas aluminium oxide and aluminium powder are insoluble. In addition, repeated inhalation of aluminium chlorohydrate generates lung effects in rodents that are not observed after inhalation of aluminium powder or aluminium oxide. Therefore, two PODs for repeat-dose inhalation toxicity were selected: a POD for aluminum hydroxychloride and aluminum chlorohydrate and a POD for the Aluminium-containing Substances Group.

Acute toxicity

Thomson et al. (1986) was identified as the critical study for acute inhalation exposure to the Aluminium-containing Substances Group. In this study, Fischer 344 male rats (6/dose) were exposed to aluminium powder (mass median aerodynamic diameter [MMAD] of 1.58 µm) at 1, 10, 50, 100, 200, and 1000 mg/m^3 for 4 hours. At 24 hours, 14 days, 3 months, and 6 months post-exposure, rats were evaluated for acute lung injury. No changes in pulmonary function or adverse physiological responses were reported; however, multifocal granulomas in the lungs and hilar lymph nodes were observed at 200 mg/m^3 and 1000 mg/m^3. These pathological changes became evident 14 days after exposure and were still observed at 3 and 6 months post-exposure. Therefore, 100 mg/m^3 is considered a NOAEC for acute exposure for the Aluminium-containing Substances Group based on multifocal granulomas in the lungs. In its opinion on the safety of aluminium in cosmetic products, the Scientific Committee on Consumer Safety (SCCS) noted a NOAEC of 1000 mg/m^3 from the same study (SCCS 2019, 2020).

Repeat-dose toxicity

Minor lung reactions have been reported in inhalation studies exposing animals to aluminium oxide or aluminium powder (Christie et al. 1963; Gross et al. 1973; Pigott et al. 1981). Gross et al. (1973) exposed guinea pigs, hamsters, and rats to aluminium

powder or aluminium oxide. Animals were exposed to atomized, pyro, or flake aluminium powder at concentrations of 15 mg/m^3 or 30 mg/m^3 for 6 months (6 hours/day, 5 days/week), or 50 mg/m^3 or 100 mg/m^3 for 12 months (6 hours/day, 5 days/week). As a non-fibrogenic dust control, animals were exposed to 30 mg/m^3 or 75 mg/m^3 of aluminium oxide powder for 12 months (6 hours/day, 5 days/week). A significant number of spontaneous deaths occurred across all species after 12 months of exposure within both the experimental and control group. However, no trend was observed regarding mortality and inhaled dose of aluminium or aluminium oxide powder. The authors attributed the deaths to low airflow and the amount of crowding in the exposure chambers. All three species of animals exposed to aluminium powder developed alveolar proteinosis. The severity and extent of alveolar proteinosis was not consistently or clearly related to the type of aluminium powder or the severity of dust exposure. As the number of post-exposure months increased, the alveolar proteinosis improved, and aluminium powder was cleared from the lungs. After 1.5 years post-exposure, most animals showed little or no remaining alveolar proteinosis or aluminium powder in the airspaces. Rats developed granulomatous inflammation in response to aluminium powder exposure, resulting in small collagenous scars in the lung (that is, endogenous lipid pneumonitis). However, the granulomas were not associated with the aluminium powder itself but with areas where cholesterol crystals formed in the absence of proteinosis. Hamsters and guinea pigs did not experience any form of fibrosis. Exposure to the aluminium oxide control did not produce alveolar proteinosis or endogenous lipid pneumonitis.

Another study exposed rats and hamsters to 100 mg/hour of McIntyre Powder (80% aluminium oxide, 20% aluminium) for 8 hours/day for 13 months (Christie et al. 1963). Both species experienced lymphoid hyperplasia and focal areas of lipid pneumonia in peribronchial and subpleural areas. In rats, exposure also resulted in focal deposits of hyaline material in the alveolar walls. There was no evidence of fibrosis in either species. A study conducted by Pigott et al. (1981) exposed rats to 2.18 mg/m^3 or 2.45 mg/m^3 aluminium oxide fibres for 86 weeks. Focal necrosis and regeneration of olfactory epithelium was observed in nasal cavity post-exposure. Since this effect was not seen in other inhalation toxicity studies using aluminium oxide powders, it could be assumed that it was caused by the fibrous shape. No fibrosis was observed in the lungs of treated rats.

Aluminosis is the most commonly reported respiratory effect observed in workers exposed to very high concentrations of fine aluminium dusts (ATSDR 2008). Aluminosis is characterized by diffuse interstitial lung fibrosis, primarily in the upper and middle lobes. In advanced stages, it is characterized by subpleural bullous emphysema, with an increased risk of spontaneous pneumothorax (ACGIH 2008; MAK 2014; Sjögren et al. 2015). Multiple case studies have reported aluminosis in workers exposed to aluminium powders (Mitchell et al. 1961; Edling 1961; McLaughlin et al. 1962; Swensson et al. 1962). A series of case studies reported by Mitchell et al. (1961) identified pulmonary fibrosis in 6 out of 27 examined factory workers manufacturing pyro powder (approximately 81.4% aluminium, 17% aluminium oxide and aluminium hydroxide, 0.5% stearin, 0.5% silicon, 0.1% copper, magnesium, manganese, and iron).

Two out of six of these cases were fatal. Factory workers were exposed to concentrations ranging from 19 mg/m^3 to 114 mg/m^3 respirable aluminium dust for approximately 3.5 hours per day. Particle size measurements from this study estimated that 70% of aluminium particles were 5 µm or less in diameter. Pulmonary fibrosis was identified by performing chest X-rays of all workers and necropsies of deceased workers. No personal protective equipment (PPE) was used by affected workers prior to the development of pulmonary fibrosis (Mitchell et al. 1961).

Hunter et al. (1944) conducted an investigation of 92 individuals working as airplane propeller grinders. The workers were exposed to a mixture of aluminium and aluminium oxide powder at concentrations between 3 mg/m^3 and 100 mg/m^3. The concentration of aluminium and aluminium oxide powder varied depending on the job performed. The majority of particles were greater than 7 µm in diameter. The highest concentrations of particles less than 7 µm (4.1 mg aluminium and aluminium oxide/m^3) were observed near the polisher. Images of workers provided in the study suggest that no PPE was used. Adverse lungs effects due to inhalation of aluminium dusts were assessed using health records and chest X-rays. Machine shop workers in the same factory were treated as a control with respect to health records; however, they reported a higher number of absences due to illness than did the propeller grinders. The authors determined that there were no lung effects caused by aluminium dust exposure in propeller grinders.

The results from Hunter et al. (1944) are further supported by a more recent cross-sectional study (Musk et al. 2000), which reported no significant evidence of decreased lung function in alumina refinery workers (n = 2388) exposed to levels of up to 2.18 mg/m^3 (particle size not reported) alumina dusts (alumina refinery workers are primarily exposed to bauxite, alumina dusts, and caustic soda mist). Forced expiratory volume in 1 second, forced vital capacity, and work-related symptoms (wheeze, chest tightness, shortness of breath, and rhinitis) were used to assess lung function. Adjustments were made for age, smoking, atopy, and process group.

McIntyre powder (mixed aluminium and aluminium oxide) was used in the Canadian mining industry in the 1930s for preventative treatment of silicosis. It was recommended that miners inhale one gram of McIntyre powder per 1000 ft^3 (35.6 mg/m^3) for 10 minutes at the start of their work shift (Zarnke et al. 2019). Peters et al. (2013) determined that there was no significant difference in mortality from silicosis between workers exposed to McIntyre powder and those unexposed, suggesting that McIntyre powder had no protective effects against silicosis.

Aluminium is a known neurotoxicant via the oral route of exposure, as described under the oral health effects assessment section (section 8.1.2). Several international assessments have considered neurotoxicity as the critical effect following inhalation exposure (US EPA 2006; ACGIH 2008; SCCS 2019, 2020). Studies that assess the association between inhalation exposure to aluminium and neurotoxicity are limited to occupationally exposed populations (aluminium potroom and foundry workers, welders, and miners). The results from these studies are inconsistent and report only mild

neurological effects (Krewski et al. 2007; ATSDR 2008; EC, HC 2010; Health Council of the Netherlands 2010; Ferguson et al. 2018). In addition, aluminium-induced neurotoxicity, which is a systemic effect from elevated aluminium exposure, is addressed by the Biomonitoring-Based approach presented in section 8.1.2 of this assessment.

Based on exposure to aluminium powder by workers (Hunter et al. 1944), a NOAEC of 4.1 mg aluminium and aluminium oxide particles/m^3 based on the absence of lung effects was selected to characterize the risk of inhalation exposure to the Aluminium-containing Substances Group. This NOAEC is further supported by the results of other occupational studies (Mitchell et al. 1961; Musk et al. 2000) and the results of animal studies (Gross et al. 1973; Pigott et al. 1981). No significant lung effects were seen in animals at concentrations as high as 75 mg/m^3 (Gross et al. 1973). Pulmonary fibrosis due to inhalation of aluminium substances has been observed in workers exposed to aluminium powders at levels as low as 19 mg/m^3, but this was not observed in animal studies. This discrepancy may be due to concomitant exposure to other substances in an industrial setting. Nonetheless, the consistency in this finding across occupational studies supports the hypothesis that aluminium dusts may cause fibrosis at high levels of exposure. The critical POD of 4.1 mg aluminium and aluminium oxide particles/m^3 was converted to a continuous exposure concentration by adjusting for the number of hours per day (8/24) as well as the number of days per week (5/7) that the workers were exposed to respirable aluminium. The resulting continuous air concentration of aluminium powder associated with no observed adverse effects was calculated to be 0.98 mg aluminium and aluminium oxide particles/m^3.

The IARC (1984, 2010) concluded that “there is sufficient evidence in humans for the carcinogenicity of occupational exposures during aluminium production” and “sufficient evidence in experimental animals for the carcinogenicity of airborne particulate polynuclear organic matter from aluminium-production plants” (Group 1 – “carcinogenic to humans”). The report contained no implications that aluminium itself is a carcinogen. Occupational aluminium exposure is confounded by exposure to other carcinogenic substances (US EPA 2006; Krewski et al. 2007; ATSDR 2008; Willhite et al. 2014). Therefore, there is no clear evidence of cancer due to inhalation of aluminium substances (US EPA 2006; Krewski et al. 2007; Willhite et al. 2014).

Aluminium chlorohydrate substances: Aluminum hydroxychloride and aluminum chlorohydrate

Steinhagen and Cavender (1978) exposed rats and guinea pigs (10/sex/dose) to 0, 0.25, 2.5, or 25 mg/m^3 hydrated aluminium chlorohydrate (as dry powder) for 6 hours/day, 5 days/week for 6 months. The severity of pathological lesions observed in the lung was dose-dependent. At 0.25 mg/m^3, rats and guinea pigs (3/10) had slight increases in alveolar macrophages in the lung compared to the control group. At this dose level, a granulomatous change in the peribronchial lymph node was observed in one rat. At 2.5 mg/m^3 and above, multifocal granulomatous pneumonia was observed in all rats and guinea pigs. At 25 mg/m^3, the granulomatous reaction was more intense

and diffuse. A significant decrease in body weight was observed in rats, and increased lung weights and lung-to-body weight ratios occurred in both species exposed to 25 mg/m^3. At 2.5 mg/m^3 and 25 mg/m^3, granulomatous lesions were observed in the lower lung and peribronchial lymph nodes of both species (Steinhagen and Cavender 1978). The Health Council of the Netherlands (2010) used this study to derive a health-based recommended occupational exposure limit (HBROEL) for aluminium chlorohydrate of 0.05 mg/m^3 (inhalable dust) as an 8-hour time-weighted average. In comparison, the US EPA (2006) identified a NOAEC of 0.25 mg/m^3 for this study for the risk characterization of aluminium chlorohydrate and its associated complexes. In the current assessment, a NOAEC of 0.25 mg/m^3 aluminium chlorohydrate was selected for the risk characterization of inhalation exposure to aluminum hydroxychloride and aluminum chlorohydrate based on the occurrence of multifocal granulomatous pneumonia at the next dose. Aluminum hydroxychloride is considered to be an associated complex of aluminium chlorohydrate (Health Canada [modified 2022a]). This concentration was converted to a continuous exposure concentration by adjusting for the number of hours per day (6/24) as well as the number of days per week (5/7) that the animals were exposed to aluminium chlorohydrate. The resulting continuous air concentration at the NOAEC was calculated to be 0.045 mg/m^3.

8.1.4 Derivation of biomonitoring equivalent (BE)

In BE derivation, an internal concentration or range of concentrations of a chemical or its metabolites in a biological medium (that is, blood, urine, or other medium) that is consistent with an existing health-based guidance value such as a reference dose (RfD) or a tolerable daily intake (TDI) is derived using available kinetic data or by conducting regression analysis between exposure and blood or urine concentrations (Hays et al. 2008, 2016). Since there were no existing BE values or other human biomonitoring guidance values for aluminium, a whole blood BE was derived for the daily intake levels associated with the PTWI established by JECFA (2012).

As a first step, a three-compartmental PBPK model was developed to predict the steady-state whole blood concentration following oral exposure to aluminium (Poddalgoda et al. 2021). A three-compartment model was considered to provide a sufficient degree of complexity to characterize aluminium kinetics in the body. The model includes 1) plasma/blood; 2) rapid tissue compartment to represent liver, kidney, brain, etc.; and 3) slow tissue compartment to represent bone. In addition to the urine excretion (represented by kidney), a combined fecal and dermal excretion pathway was included to facilitate the use of available kinetic data (Priest et al. 1995; Talbot et al. 1995). A schematic diagram of the PBPK model is presented in Figure J-1 of Appendix J.

Two sets of kinetic data were used to develop the PBPK model, one based on a minimally bioavailable form of aluminium (that is, aluminium chloride) and the other one based on a highly bioavailable form of aluminium (that is, aluminium citrate). Steinhausen et al. (2004) examined the pharmacokinetic properties of aluminium chloride. A single oral or i.v. dose of radio-labelled aluminium (^{26}Al) as aluminium

chloride was administered to six healthy volunteers. Kinetic data for aluminium in blood and urine were reported for up to 64 days. The kinetics of aluminium citrate were studied by Fifield (1977). Fifield (1977) administered to one healthy volunteer a single oral dose of radio-labelled aluminium (^{26}Al) as aluminium citrate. Kinetic data were reported for 24 days. Additionally, kinetic data from two other volunteer studies (Priest et al. 1995; Talbot et al. 1995), in which the volunteers were exposed to aluminium citrate via i.v. injection, were also used in the PBPK model.

The kinetic modelling was performed using Microsoft Excel (version 16.9). When fitting the model, it was assumed that concentrations of aluminium in whole blood and plasma are equivalent, based on the kinetic data available in the literature, which indicate that most of the aluminium in blood is bound to plasma protein (Fifield 1997; Krewski et al. 2007). Although both i.v. and oral exposure data were used to develop the model, the model provided a better prediction of blood concentrations from oral dosing for both aluminium chloride and aluminium citrate. The models fitted to the oral and i.v. data for aluminium citrate are presented in Figure J-2 of Appendix J. It was observed that the steady-state blood aluminium concentration following an oral dose of aluminium citrate was approximately 4.5 times higher than the blood concentration following an equivalent dose of aluminium chloride (Poddalgoda et al. 2021). This difference is likely due to the relatively high solubility and bioavailability of aluminium citrate compared to aluminium chloride.

The developed PBPK model was then used to derive the BE, which is the steady-state blood concentration associated with the daily intake of 0.3 mg Al/kg bw/day. This daily intake was calculated using the critical POD (a NOAEL of 30 mg/kg bw/day) and an uncertainty factor of 100 identified by JECFA (2012) for the derivation of their PTWI. The BE value was derived using kinetic data from aluminium citrate because the POD used in JECFA (2012) was from a chronic animal toxicity study, where animals were administered aluminium citrate as the test material (Poirier et al. 2011; see section 8.1.2 for details).

Based on the current PBPK model, the whole blood BE is 16 µg/L (Poddalgoda et al. 2021). Although the BE was developed using data for the oral route, it is expected that it is protective of systemic exposure from the dermal route, which has a significantly lower bioaccessibility. The derived BE is not considered appropriate for interpreting portal-of-entry effects in the lungs from exposure to aluminium via the inhalation route because the critical POD for the BE derivation is associated with systemic effects. A urinary BE (123 µg/L, µg/g creatinine) was also derived for the daily intake associated with the JECFA PTWI using the mass balance approach (Poddalgoda et al. 2021).

8.2 Health exposure assessment

Canadians may be exposed to the substances in the Aluminium-containing Substances Group through their contribution to total levels of aluminium in environmental media (soil, drinking water, house dust, and air), food, and from the use of products available to consumers. Total aluminium has been measured in environmental media (soil,

drinking water, house dust, and air), food, and whole blood. These data provide concentrations of total aluminium in these media but not substance-specific data. In this assessment, total aluminium data are used as a surrogate for substance-specific exposure data to assess systemic exposure. Data on total aluminium are considered to be an acceptable, although protective, surrogate for CAS RN specific data. CAS RN specific data are used to assess potential inhalation exposure to the substances in the Aluminium-containing Substances Group.

8.2.1 Environmental Media, food, and drinking water

Total aluminium has been measured in food, drinking water distribution systems and tap water, household dust, soil, and in outdoor, indoor, and personal air samples (Table G-2, Appendix G).

Most foods, including plant and animal-based foods, contain a certain amount of aluminium originating from natural occurrence and environmental contamination from anthropogenic sources (EC, HC 2010). Aluminium may also be present in foods through the use of aluminium-containing food additives, from its potential use as a component in incidental additives used in food processing establishments, and from its potential use as a component in the manufacture of food packaging materials (personal communication, email from the FD, Health Canada, to the ESRAB, Health Canada, dated October 2, 2018; unreferenced). The use of aluminium cookware can result in the leaching of aluminium into food during preparation and is estimated to contribute an additional 10% to 20% to total daily aluminium intake, although the contribution depends on a number of factors including the quality and condition of the cookware, type of food being cooked, and cooking time (Health Canada [modified 2015]). Exposure to aluminium from the use of aluminium cookware is expected to be captured in available population level biomonitoring data, and therefore, quantitative exposure was not estimated from this pathway into food.

Dietary exposure to aluminium was estimated using the results from over 18 500 samples of foods (meats, seafood, dairy products, grain products, fruits and vegetables, eggs, processed foods, confectionary, nuts, beverages, and infant formula) analyzed as part of the Total Diet Study (TDS) (2008–2010 data) and by the Canadian Food Inspection Agency (CFIA) (2007–2013 data) (personal communication, email from the FD, Health Canada, to the ESRAB, Health Canada, dated March 26, 2020; unreferenced). Food consumption data from the Canadian Community Health Survey (CCHS) Cycle 2.2 (Statistics Canada 2004) were used to estimate usual dietary exposures, which were calculated using a probabilistic approach (that is, to reflect the long-term average of the daily intake). Mean exposures ranged from 0.025 mg/kg bw/day in 51- to 70-year-old males to 0.089 mg/kg bw/day in children 1 to 3 years of age (both sexes combined) (Table G-3, Appendix G). Grain-based foods are the main contributors to dietary aluminium exposure in Canadians aged 1 year old or older. In particular, baked goods such as pancakes, muffins, cakes, and waffles, to which aluminium-containing food additives may be added directly or in which these additives may be a component of baking powder used as an ingredient in these foods (at levels

consistent with good manufacturing practices [GMP]), contribute most notably to dietary aluminium exposure (personal communication, emails from the FD, Health Canada, to the ESRAB, Health Canada, November 2019; unreferenced).

Aluminium has been reported in human milk from Canadian mothers, which is a source of exposure for nursing infants (EC, HC 2010). The weighted mean aluminium concentration in human milk reported in two studies of Canadian women was approximately 0.1133 mg/L (0.11 mg/kg, adjusted assuming human milk density = 1.030 g/mL) (EC, HC 2010; US EPA 2011). More recently, from 2008 to 2011, aluminium was measured in human milk collected from Canadian women between 2 weeks and 10 weeks post-partum as part of the Maternal-Infant Research on Environmental Chemicals (MIREC) project (personal communication, email from the FD, Health Canada, to the ESRAB, Health Canada, dated March 26, 2020; unreferenced). Aluminium was detected at low levels in approximately 21% of the 847 samples analyzed (limit of detection [LOD] = 12 ng/mL). Conservatively setting values reported below the LOD to the LOD value results in a mean aluminium concentration of 0.0151 mg/L (personal communication, email from the FD, Health Canada, to the ESRAB, Health Canada, dated March 26, 2020; unreferenced). Using the median (127.95 g/kg bw/day) consumption figure for human milk reported in Arcus-Arth et al. (2005) for exclusively human milk-fed infants 0 to 6 months old, average exposure to aluminium from human milk was estimated to be 0.0019 mg/kg bw/day. It is assumed that human milk-fed infants under the age of 6 months consume only human milk, and therefore, the aluminium intake from human milk is their sole source of dietary exposure. Daily aluminium intake from human milk for children older than 6 months was not quantified, but it is expected to be lower than the aluminium intakes of formula-fed children or children of the same age consuming solid foods (Table G-3, Appendix G), given that infant formula generally contains higher concentrations of aluminium than human milk does (EC, HC 2010).

Milk- and soy-based infant formulas from CFIA data are included in the total dietary exposure estimates for aluminium (Table G-3, Appendix G). Mean concentrations of aluminium calculated from infant formula samples included in the 2016, 2017, and 2018 TDS are 94 ng/g and 442 ng/g in milk- and soy-based infant formulas, respectively (personal communication, email from the FD, Health Canada, to the ESRAB, Health Canada, dated March 26, 2020; unreferenced). The estimated mean dietary intake of aluminium for formula-fed infants under 1 year old is 0.086 mg/kg bw/day (Table G-3, Appendix G).

Aluminium is present in drinking water; it occurs naturally, is added during water treatment, and has been reported to leach from pipes or linings into drinking water (Health Canada 2021a). Health Canada recently published a MAC of 2.9 mg/L (2900 µg/L) and an operational guidance value (OG) of 0.100 mg/L (100 µg/L) for aluminium in drinking water (Health Canada 2021a). Water monitoring data from the provinces and territories presented in the drinking water guideline technical document indicated that concentrations of aluminium were generally low for raw, treated, and distributed water, but mean and 90th percentile levels of total aluminium in certain

municipal surface waters (treated and/or distributed) exceeded the OG value (Health Canada 2021a). The overall mean concentration of aluminium of 0.120 mg/L, reported in municipal treated surface water, was below the MAC but exceeded the OG (Health Canada 2021a). Mean aluminium concentrations in municipal treated groundwater, municipal distributed groundwater, municipal distributed surface water, and non-municipal water were lower (Table G-2, Appendix G). Furthermore, maximum concentrations for certain non-municipal supplies and municipal surface water exceeded the MAC (Health Canada 2021a). Concentration of aluminium in drinking water is highly variable depending on the type of source water, geographical location, and drinking water treatment processes. The mean concentration of aluminium in distributed surface water (0.111 mg/L) was used to estimate aluminium intake from drinking water for the general population as it is representative of average Canadian exposure (Table G-5, Appendix G) (personal communication, emails from the WAQB, Health Canada, to the ESRAB, Health Canada, dated March 4, 2020; unreferenced; Health Canada 2021a).

Aluminium concentrations in outdoor Canadian air vary depending on sampling location. Aluminium concentrations in approximately 10 000 samples of particulate matter with an aerodynamic diameter of 10 µm or less (PM_{10}) from provinces and territories across Canada ranged from below the study detection limit to up to 24.94 µg/m^3, with an estimated mean of 0.17 µg/m^3 (EC, HC 2010). Similarly, the aluminium concentration in approximately 20 000 samples of particulate matter with an aerodynamic diameter of 2.5 µm or less ($PM_{2.5}$) from provinces and territories across Canada ranged from below the study detection limit to up to 9.24 µg/m^3, with an estimated mean of 0.069 µg/m^3 (EC, HC 2010). Aluminium concentrations in various studies of Canadian outdoor air quality conducted by Health Canada between 2009 and 2013 reported a median PM_{10} aluminium concentration of 0.0714 µg/m^3 and median $PM_{2.5}$ aluminium concentrations of between 0.0022 µg/m^3 and 0.0788 µg/m^3 (personal communication, email from the WAQB, Health Canada, to the ESRAB, Health Canada, dated February 18, 2020; unreferenced). The median concentration of aluminium in outdoor air studied in Windsor, Ontario, was 0.0587 µg/m^3 in the $PM_{2.5}$ fraction and 0.152 µg/m^3 in the $PM_{10–2.5}$ fraction (Rasmussen et al. 2018). The mean concentration of aluminium in outdoor air $PM_{2.5}$ samples from the provinces and territories (0.069 µg/m^3) was used to estimate exposure to the general population (Appendix G, Table G-5) as well as to calculate mean daily air concentration (Table 8-1).

Primary production of aluminium is a major industry in Canada. As of 2016, there is 1 facility in Canada that produces metallurgical and non-metallurgical alumina, and 10 primary aluminium smelters (NRCan 2016). This included 10 facilities located in Quebec (QC), with the majority around the Saguenay-Lac-Saint-Jean region, and 1 in Kitimat, British Colombia (BC) (NRCan 2016; NPRI [modified 2019], [modified 2022]). Air quality monitoring data from National Air Pollution Surveillance (NAPS) stations close in proximity (within 25 km radius) to aluminium industry point sources identified through NPRI (NPRI [modified 2022]), collected between 2010 and 2017, reported daily average total $PM_{2.5}$ air concentrations of up to 105 µg/m^3 in QC, with an average of 6.95 µg/m^3, and concentrations of up to 47 µg/m^3 in BC, with an average of 4.13 µg/m^3 (NAPS

[modified 2022]). Taking into consideration the mass fraction of aluminium in total $PM_{2.5}$ of 4.05% reported in a study in close proximity to an aluminium smelter in Alma, QC (Boullemant 2011), the mean aluminium air concentration in $PM_{2.5}$ was estimated to be 0.28 µg Al/m^3 near point sources in QC and 0.17 µg Al/m^3 near point sources in BC (Boullemant 2011; NAPS [modified 2022]). In Canada, the aluminium air concentrations in areas close in proximity to point sources, such as aluminium smelters, may be elevated compared to national averages. In an effort to reduce air emissions, an environmental performance agreement for the achievement of base-level industrial emissions requirements for the aluminium and alumina sector was put into place in November 2017 (ECCC [modified 2018]). Since the publication of the Code of Practice to Reduce Emissions of Fine Particulate Matter ($PM_{2.5}$) from the Aluminium Sector in 2016 and the environmental performance agreement in 2017, $PM_{2.5}$ emissions near aluminium industry point sources have decreased (ECCC 2016; ECCC [modified 2018]; NPRI [modified 2022]). Aluminium has been measured in air through studies and monitoring initiatives in industrial areas within the vicinity of potential point sources of release, including ports, shale gas plants, steel mills, and oil sands (WBEA 2019, 2020; personal communication, email from the WAQB, Health Canada, to the ESRAB, Health Canada, dated February 18, 2020; unreferenced). The median concentrations of aluminium in outdoor air collected in the vicinity of point sources of release were between 0.0091 µg/m^3 and 0.1337 µg/m^3. Since ports, shale gas plants, steel mills, and oil sands are not identified as industries emitting the largest amount of aluminium in Canada, the air concentration—estimated using the study on particulate matter in close proximity to an aluminium smelter and air quality monitoring data from NAPS stations—is used to characterize exposure for individuals living in the vicinity of a point source of release (Table 8-1).

Aluminium was measured in studies of indoor air, personal air, and household dust (EC, HC 2010; Rasmussen et al. 2018). Aluminium concentrations in various studies of Canadian indoor air quality conducted by Health Canada between 2009 and 2013 reported median aluminium concentrations between 0.0122 µg/m^3 and 0.0224 µg/m^3 in the $PM_{2.5}$ fraction (personal communication, email from the WAQB, Health Canada, to the ESRAB, Health Canada, dated February 18, 2020; unreferenced). Additionally, matched indoor, outdoor, and personal monitoring air ($PM_{2.5}$ and $PM_{10–2.5}$) samples were collected from Windsor, Ontario, from 2005 to 2006 (Rasmussen et al. 2018). Aluminium concentrations in the coarse particle range size ($PM_{10–2.5}$) exceeded concentrations in fine particle range size ($PM_{2.5}$) in indoor, outdoor, and personal air samples. The highest median aluminium concentration in $PM_{10–2.5}$ was measured in personal air samples (173 ng/m^3), followed by outdoor and indoor air concentrations (median of 152 ng/m^3 and 102 ng/m^3, respectively). The highest median aluminium concentration in $PM_{2.5}$ was measured in outdoor air samples (58.7 ng/m^3), followed by indoor and personal air concentrations (median of 23.7 ng/m^3 and 19.0 ng/m^3, respectively). Similar trends were displayed in 95th percentile concentrations of aluminium in $PM_{10–2.5}$ and $PM_{2.5}$. These relationships displayed seasonal variability, which suggests that natural soil minerals as well as anthropogenic inputs, such as self-care products, paints, and textiles, are an important source of aluminium in indoor air (Rasmussen et al. 2018). The concentration of aluminium in the respirable fraction of

indoor air ($PM_{2.5}$) found in the study of Windsor homes (23.7 ng/m^3) was used to characterize intake of aluminium from indoor air (Table G-5, Appendix G) as well as to calculate mean daily air concentration (Table 8-1).

Aluminium was also measured in samples of $PM_{2.5}$ from subways and buses in large Canadian cities as part of Health Canada's urban transport exposure study conducted in Montreal, Ottawa, Toronto, and Vancouver (personal communication, email from the WAQB, Health Canada, to the ESRAB, Health Canada, dated February 18, 2020; unreferencedThe mean aluminium air concentrations on subways (0.249 µg/m^3) and buses (0.133 µg/m^3) were higher than average indoor and outdoor aluminium air concentrations, suggesting that public transit may be a point source of exposure to aluminium. Aluminium concentrations in $PM_{2.5}$ from inside and outside private vehicles were also measured in the urban transport exposure study, with median concentrations falling between 0.0192 µg/m^3 and 0.0778 µg/m^3, and 0.0445 µg/m^3 and 0.0574 µg/m^3, respectively (personal communication, email from the WAQB, Health Canada, to the ESRAB, Health Canada, dated February 18, 2020; unreferenced). Daily intake of aluminium from air on public transit was not factored into the daily intake estimates in this assessment but was considered as a point source of inhalation exposure in risk characterization (Table 8-1).

Aluminium concentrations in soil are variable throughout Canada depending on geology and anthropogenic inputs. Average soil concentrations of aluminium ranged from 12 000 mg/kg in Nova Scotia to 87 633 mg/kg in BC, with a mean total aluminium concentration of approximately 41 000 mg/kg (EC, HC 2010). In areas close in proximity to point sources of exposure, such as aluminium refineries and smelters, soil concentrations may be elevated from atmospheric fallout. The mean aluminium concentration of 41 000 mg/kg was used to estimate aluminium intake from soil (Appendix G, Table G-5).

Aluminium was also measured in household dust in Canada. The median concentration of aluminium in household dust measured from samples collected from Windsor homes was 11 453 µg/g (Rasmussen et al. 2018). This was found to be significantly correlated with concentrations of aluminium in PM_{10} in both indoor and personal air samples. The concentration of 11 453 µg/g in household dust was used to estimate aluminium intake from dust (Appendix G, Table G-5).

A study conducted in the southern Mackenzie Mountains, Northwest Territories (NWT), measured concentrations of elements in four large mammal species. Aluminium concentrations were measured in the muscle and organ meat of moose, mountain caribou, Dall's sheep, and mountain goat (Larter et al. 2016). Mean concentrations of aluminium ranged from 2.71 mg Al/kg to 16.7 mg Al/kg in muscle meat, with the highest concentration found in mountain caribou. In organ meat, concentrations ranged from 0.14 mg Al/kg in moose kidney to 0.36 mg Al/kg in mountain caribou kidney. The results of food frequency questionnaires given to individuals 6 to 79 years old, in nine communities of the Dehcho and Sahtú regions of NWT, indicated that these country foods are consumed by Indigenous peoples in the region (Ratelle et al. 2020a). The

average portion size of land animal muscle meat consumed was 143 g, 5.2 times per week. Portion sizes of large game organs averaged 100 g and were consumed 8.4 times per week. On the basis of the above concentrations and consumption amounts, intake of aluminium from the consumption of land animal meat and large game organs ranged from 2.46×10^{-2} mg/kg bw/day for adults to 7.90×10^{-2} mg/kg bw/day for children 4 to 8 years old (Appendix G, Table G-4). Canadian data on aluminium concentrations in country foods are limited. The estimated intakes derived from this data are limited to the consumption of land animals and organ meats by Indigenous communities in the Dehcho and Sahtú regions of the NWT.

Additionally, aluminium was measured in drinking water samples from 1516 households in First Nations communities across Canada as part of the First Nations Food, Nutrition and Environment Study (FNFNES) (Chan et al. 2019b). Concentrations in drinking water of 208 households across 23 First Nations communities exceeded the OG value of 100 µg/L. Twenty first draw water samples, all collected in one Manitoba Boreal Shield community, ranged from 6 680 to 33 100 µg/L, exceeding the MAC of 2 900 µg/L (Chan et al. 2019b; Schwartz et al. 2021). The elevated aluminium levels in the water samples from this community were a result of issues at the water treatment plant (Schwartz et al. 2021). A resampling of the water treatment plant two months later found that aluminium levels for this community were acceptable.

Daily intake of aluminium from environmental media and from country foods by people residing in Indigenous communities is expected to be highly variable depending on geographical location, proximity to point sources of exposure, food preparation methods, food consumption patterns, and water treatment methods. It is important to emphasize that some study authors working in these communities have noted that “the benefits of country food consumption generally outweigh contaminant risks” (Ratelle et al. 2019). Given the highly variable data and the potential benefits of country food consumption, the data available are insufficient to conduct a fulsome analysis of aluminium intakes from environmental media and country food in Indigenous communities at this time.

Aluminium intakes from environmental media, food, and drinking water are presented in Appendix G, Table G-5. The available data regarding bioavailability do not provide evidence for significant differences in relative oral bioavailabilities with respect to water, food, and soil intake (EC, HC 2010). The bioavailability via inhalation may be higher than oral bioavailability but would not significantly influence the estimated absorbed dose because of the low estimated concentrations of aluminium in ambient and indoor air. For these reasons, estimated values of bioavailability for different media and routes of exposure were not integrated into the estimates of aluminium intake from environmental media, food, and drinking water (Appendix G, Table G-5). Daily intake estimates from environmental media, food, and drinking water for the general population were highest in infants aged 6 months to 1 year at 0.16 mg/kg bw/day. Notwithstanding the limited data set, it was noted that, for specific Indigenous communities, intake of aluminium resulting from the consumption of certain country foods was highest in children aged 4 to 8 years at 0.0790 mg/kg bw/day. These exposure estimates were

brought forward for risk characterization. Mean daily air concentrations in ambient air, as well as air with point source influence and air with transit influence, presented in Table 8-1, were brought forward for risk characterization to assess the potential of portal-of-entry effects from inhalation exposure.

Table 8-1. Summary of aluminium air concentrations

Exposure scenario	Al daily air concentrations (mg/m^3)
Mean daily air concentration, ambient air in Canada ($PM_{2.5}$)[a]	3.0×10^{-5}
Mean daily ambient air concentrations with point source influence: aluminium smelter in QC ($PM_{2.5}$)[b]	2.8×10^{-4}
Mean daily air concentration with transit influence ($PM_{2.5}$)[c]	7.8×10^{-5}

Abbreviations: Al, aluminium; $PM_{2.5}$, particulate matter with an aerodynamic diameter of 2.5 µm or less

[a] Daily air concentration estimated using median 24-hr outdoor air sample $PM_{2.5}$ of 0.069 $\mu g/m^3$ (n=>10 000) (EC, HC 2010) and median 24-hr indoor air sample $PM_{2.5}$ of 0.0237 $\mu g/m^3$ (n=121) measured in Windsor, Ontario (Rasmussen 2018). Canadians are assumed to spend 3 hours outdoors and 21 hours indoors each day (Health Canada 1998). Daily air concentration = (concentration Al outdoor air × (3 hours / 24 hours)) + (concentration Al indoor air × (21 hours / 24 hours)).

[b] Daily air concentration, point source influence estimated using an ambient air concentration (0.28 µg Al/m^3, maximum mean concentration) estimated based on NAPS data from 2010 to 2017 (NAPS [modified 2022]), considering the weight fraction of aluminium particulate matter near a primary aluminium smelter (Boullemant 2011). In the absence of data on aluminium concentration in indoor air within the vicinity of a point source of release, the aluminium air concentration is assumed to be constant over a 24-hour period each day.

[c] Daily air concentration, transit influence estimated using a median 24-hr $PM_{2.5}$ personal air sample of 0.249 $\mu g/m^3$, taken from the subway (personal communication, email from the WAQB, Health Canada, to the ESRAB, Health Canada, dated February 18, 2020; unreferenced), and mean 24-hr outdoor air concentration in $PM_{2.5}$ of 0.069 $\mu g/m^3$ (n=>10 000) (EC, HC 2010). The largest median $PM_{2.5}$ aluminium concentration reported in the urban transport data sets from Ottawa, Toronto, Montreal, and Vancouver was assumed to represent median aluminium air concentration as a conservative assumption. For time not spent on transit, the aluminium concentration in outdoor air is used as a conservative assumption as it is higher than the aluminium concentration in indoor air. Individuals are assumed to spend 70 minutes on transit per day (van Ryswyk et al. 2017). Daily air concentration, transit influence = (concentration Al personal air, subway × (70 min / 1440 minutes)) + (concentration Al outdoor air x (1370 minutes / 1 440 minutes)).

8.2.2 Products available to consumers

Some substances in the Aluminium-containing Substances Group have widespread industrial, commercial, and consumer uses that contribute to daily exposure. As outlined in the sources and uses section (section 4.3), substances in the Aluminium-containing Substances Group are present in a range of products available to consumers. Exposures to products available to consumers are captured in the biomonitoring data where product use may contribute to systemic levels of aluminium; therefore, potential systemic exposures from their use have not been quantified (section 8.2.3). Inhalation exposure estimates were derived for products available to consumers since lung effects from the inhalation route were identified in the health effects data set (section 8.1.3).

Inhalation exposure from products available to consumers

In Canada, some substances in the Aluminium-containing Substances Group were found in a range of aerosol, trigger spray, and loose powder products where use may

result in inhalation exposure. These products include self-care products (that is, cosmetics, natural health products, and non-prescription drugs), paints and coatings, DIY products (for example, cement products, tile grout), and cleaning products (Household Products Database 1993-; Environment Canada 2013; ECCC 2017; LNHPD [modified 2022]; DPD [modified 2022]; personal communication, emails from the CHPSD, Health Canada, to the ESRAB, Health Canada, dates ranging from June 25, 2018 to November 5, 2019; unreferenced; personal communication, emails from the NNHPD and the TPD, Health Canada, to the ESRAB, Health Canada, dates ranging from June 11, 2018 to October 31, 2019; unreferenced). DIY welding applications may result in exposure to aluminium from welding fumes, which can consist of up to 40% aluminium (Sjögren et al.1996; US EPA 2006). Exposure of the general population to welding fumes from at-home welding applications is expected to be limited. Sentinel exposure scenarios (scenarios associated with the highest exposure) were identified in order to estimate exposure to consumers from the use of products containing aluminium where inhalation exposure was anticipated.

Air concentrations from the use of aerosol and spray self-care products were modelled using the Consumer Exposure Web Model (ConsExpo Web 2019), a computational modelling program intended to estimate exposure of the general population to common products available to consumers. Air concentrations were estimated for the use of self-care products formulated as aerosol deodorants and sunscreen (personal communication, email from the CHPSD, Health Canada, to the ESRAB, Health Canada, dates ranging from June 25, 2018, to January 13, 2020; unreferenced; emails from the NNHPD, Health Canada, to the ESRAB, Health Canada, dated June 12, 2018, and October 31, 2019; unreferenced). Concentrations of aluminium-containing substances, including aluminum hydroxychloride or aluminum chlorohydrate, were reported to be up to 25% in aerosol or trigger spray self-care products (personal communication, email from the CHPSD, Health Canada, to the ESRAB, Health Canada, dated June 25, 2018; unreferenced). Refinements to certain default model parameters for aerosol self-care product scenarios were made in order to generate the most realistic and relevant exposure estimates for each sentinel self-care product type (Appendix H, Table H-1).

The air concentrations generated from the use of aerosol antiperspirant products containing aluminum hydroxychloride or aluminum chlorohydrate were estimated using available empirical data. Empirical data were used as a refinement over ConsExpo Web estimates and to ensure that the best available exposure data were used. Aluminum hydroxychloride and aluminum chlorohydrate are used in concentrations of up to 25% within aerosol antiperspirant and deodorant products in Canada (personal communication, emails from the CHPSD, Health Canada, to the ESRAB, Health Canada, dates ranging from June 25, 2018, to November 5, 2019; unreferenced). In a study by Schwarz et al. (2018), an air concentration of 0.16 mg Al/m^3 in the thoracic size range (<10 µm) was measured during the simulated use of an antiperspirant spray containing 4.2% aluminum chlorohydrate (equal to 1.3% Al). The air concentration reported in that study was adjusted according to the highest concentration of aluminum chlorohydrate in self-care products available to consumers in Canada (25%) to obtain

an estimated event air concentration of 3.1 mg aluminum chlorohydrate/m^3 (Appendix H, Table H-1).

The air concentration generated from the use of an aerosol foot deodorant spray containing aluminum chlorohydrate was estimated using the same empirical data, assuming similar use conditions as aerosol antiperspirant products. To obtain an estimated event air concentration of 1.2 mg aluminum chlorohydrate/m^3 (Appendix H, Table H-1), the air concentration reported in the study by Schwarz et al. (2018) was adjusted based on the highest concentration of aluminum chlorohydrate in aerosol foot deodorant spray products available to consumers in Canada (10%). Estimated adjusted daily air concentrations generated from the use of aerosol self-care products ranged from 1.0×10^{-3} mg aluminum oxide/m^3 to 1.4×10^{-2} mg aluminum chlorohydrate/m^3 (Table 8-2 and Table 8-3).

Exposure was also estimated for self-care products formulated as loose powders with the potential for inhalation of respirable aluminium-containing particles. These products include powdered sunscreen products, loose powdered foot deodorants, and powdered face makeup (personal communication, emails from the CHPSD, Health Canada, to the ESRAB, Health Canada, dates ranging from June 25, 2018, to November 5, 2019; unreferenced; emails from the NNHPD, Health Canada, to the ESRAB, Health Canada, dated June 12, 2018, and October 31, 2019; unreferenced). Concentrations of aluminium-containing substances in these products ranged from 2.5% to 30% (personal communication, emails from the CHPSD, Health Canada, to the ESRAB, Health Canada, dates ranging from June 25, 2018, to November 5, 2019; unreferenced; emails from the NNHPD, Health Canada, to the ESRAB, Health Canada, dated June 12, 2018, and October 31, 2019; unreferenced). Self-care products formulated as pressed powders were not identified as a potential source of inhalation exposure of concern, because these formulations contain coarser particles and binders, such as oils or waxes, which help bind the particles together and do not lead to the formation of a “dust cloud” that can be inhaled.

Air concentrations of powdered self-care products were estimated using experimentally measured particulate matter air concentration data for poorly soluble particles from the use of loose powdered cosmetics. Several studies of air concentrations generated from the use of powdered self-care products were considered (Nazarenko et al. 2012; Anderson et al. 2017; Rasmussen et al. 2019). Nazarenko et al. (2012) measured the air concentration of particulate matter in particle number concentrations by simulating the use of cosmetic powders. Data from Nazarenko et al. (2012) were not used to estimate air concentrations of aluminium from the use of powdered self-care products in this assessment as the mass per volume air concentration data were not reported (only the number of particles per volume of air were reported).

Of the available data, Anderson et al. (2017) and Rasmussen et al. (2019) provide the best and most relevant available data to model the product scenarios included in this assessment report. This is because the data in these studies are reported in mass concentration and the products analyzed are representative of the product types

considered in this assessment. In the studies by Anderson et al. (2017) and Rasmussen et al. (2019), average PM_4 (particulate matter with an aerodynamic diameter of 4 µm or less) air concentrations of talc, a poorly soluble mineral common in loose body and face powdered products, were estimated from the use of such products. Average air concentrations from Anderson et al. (2017) were combined with the body and face replicates from Rasmussen et al. (2019) to obtain an overall average PM_4 event air concentration of 1.36 ± 0.97 mg/m^3 (ECCC, HC 2021). This value was then used to estimate adjusted air concentrations for self-care products containing aluminium compounds while considering the highest concentration of aluminium-containing substance present in the product. The use of talc PM_4 as a surrogate for the aluminium-containing substances is based on their physical similarities as poorly soluble particles and use in similar types of products available to consumers. Estimated adjusted daily air concentrations generated from the use of powdered products available to consumers ranged from 3.6×10^{-5} mg aluminum chlorohydrate/m^3 to 1.4×10^{-3} mg aluminum hydroxide/m^3 (Table 8-2 and 8-3).

The exposure scenarios for aerosol, trigger spray, and loose powdered products that resulted in the highest exposure concentrations are presented in Table 8-2 and Table 8-3. On the basis of the duration and nature of effects seen in the toxicity data used to characterize risk, the resultant mean event air concentrations of aluminium-containing substances in products with frequent use patterns were adjusted to a continuous exposure scenario by amortizing exposure over 24 hours, considering the duration of exposure and frequency of use (details in Appendix H, Table H-1). The details of all exposure scenarios and input values for the models are provided in Table H-1 of Appendix H, including the refinements made to defaults.

Additional use scenarios with the potential for inhalation exposure to substances in the Aluminium-containing Substances Group were considered—including as non-medicinal ingredients in natural health products and non-prescription drugs, cosmetics (for example, aerosol dry shampoo, aerosol nail polish, fragrance, aerosol face makeup, powdered dry shampoo, body powders, powdered nail products, intimate powder), paints, DIY products (for example, cement, tile grout), cleaning products, and automotive products—but they resulted in lower exposure estimates than those presented in Table 8-2 and Table 8-3.

Table 8-2. Estimated air concentrations of substances in the Aluminium-containing Substances Group from the use of self-care products

Exposure scenario	Age group[a]	Mean event air concentration (mg substance/m^3)	Adjusted daily air concentrations[b] (mg substance/m^3)
Aerosol deodorant, 3% potassium alum	Adult	1.7	7.6×10^{-3}
Aerosol sunscreen, 2.535% aluminum oxide	1 to 13 years old	1.8×10^{-1}	1.0×10^{-3}

Exposure scenario	Age group[a]	Mean event air concentration (mg substance/m^3)	Adjusted daily air concentrations[b] (mg substance/m^3)
Powdered face makeup, 30% aluminum hydroxide	14 years old to adult	4.1×10^{-1}	1.4×10^{-3}
Powdered sunscreen, 2.535% aluminum oxide	1 to 13 years old	3.4×10^{-2}	1.9×10^{-4}

[a] The age group(s) identified are those with the highest estimated daily exposure according to the event air concentration and frequency of use.
[b] Air concentrations were adjusted to a continuous exposure scenario by amortizing exposure over 24 hours based on duration of exposure and frequency of use (for further details, see Appendix H, Table H-1).

Table 8-3. Estimated air concentrations of aluminum hydroxychloride (CAS RN 1327-41-9) and aluminum chlorohydrate (CAS RN 12042-91-0) from the use of self-care products

Exposure scenario[b]	Age group	Mean event air concentration (mg substance/m^3)	Adjusted daily air concentration[a] (mg substance/m^3)
Aerosol antiperspirant, 25% aluminum chlorohydrate	Adult	3.1	1.4×10^{-2}
Aerosol antiperspirant, 25% aluminum chlorohydrate	14 to 18 years old	3.1	1.3×10^{-2}
Aerosol antiperspirant, 25% aluminum chlorohydrate	9 to 13 years old	3.1	1.2×10^{-2}
Aerosol foot deodorant spray, 10% aluminum chlorohydrate	2 years old to adult	1.2	3.2×10^{-3}
Loose foot powder, 1% aluminum chlorohydrate	3 years old to adult	1.4×10^{-2}	3.6×10^{-5}

[a] Air concentrations were adjusted to a continuous exposure scenario by amortizing exposure over 24 hours based on duration of exposure and frequency of use (for further details, see Appendix H, Table H-1).
[b] Aluminum hydroxychloride (CAS RN 1327-41-9) and aluminum chlorohydrate (CAS RN 12042-91-0) are included in the INCI name “aluminum chlorohydrate”; therefore, the term “aluminum chlorohydrate” is used in the exposure estimates and may refer to either CAS RN. Health Canada published a proposed update to the entry for aluminum chlorohydrate and its associated complexes on the Cosmetic Ingredient Hotlist in July 2023 (Health Canada [modified 2023]). This revision proposes to prohibit aluminum chlorohydrate and its associated complexes in aerosol products.

Less frequent exposure from the use of a temporary hair colour aerosol spray (30% C.I. Pigment Blue 29) was estimated using ConsExpo Web (2019) modelling and resulted in a mean event air concentration of 5.4 mg C.I. Pigment Blue 29/m^3, with an adjusted 4-hour air concentration of 0.1125 mg C.I. Pigment Blue 29/m^3 (Appendix H, Table H-1). Air concentrations were adjusted for comparison with the acute toxicological study

duration (Thomson et al. 1986). Exposure from other aerosol, spray, and loose powdered products with infrequent or intermittent use (for example, powdered nail cleaner, foot bath powder, DIY products) were considered, but these resulted in lower exposure than the temporary hair colour aerosol spray, according to product use patterns.

8.2.3 Biomonitoring data

In this assessment, the concentration of aluminium in whole blood was used to estimate systemic exposure of the general population to aluminium. Very limited CAS RN specific exposure data are available. All bioavailable forms of aluminium contribute to the total aluminium moiety, and aluminium concentrations in blood can be considered a biologically relevant, integrated measure of systemic exposure to all aluminium-containing CAS RNs that may occur across multiple routes (for example, oral, dermal, and inhalation) and sources, including environmental media, diet, and frequent or daily use products. Due to the availability of biomonitoring data, Biomonitoring-based Approaches were considered for use in this assessment to evaluate exposure and risk. The aluminium biomonitoring data did not meet the criteria for assessment using Biomonitoring-based Approach 1 as the limit of detection of the available biomonitoring data was not sufficiently low (Jayawardene et al. 2021); therefore, the Biomonitoring-based Approach 2 was used (Health Canada [modified 2016]).

Biomonitoring studies were carefully selected for inclusion in this risk assessment because of the inadvertent contamination that may occur due to the abundance of aluminium in the environment, which could cause contamination of sampling and storage devices (Morita et al. 1994; Moyer et al. 1991; Rodushkin and Ödman 2001). Methodological uncertainties related to the analysis of aluminium in biological samples were also identified (Riihimäki and Aitio 2012; Bertram et al. 2015; RIVM 2020). Aluminium is known to impart analytical challenges due to its widespread presence in laboratory analysis and sample collection materials such as glass collection tubes, rubber stoppers in standard evacuated blood tubes, vials, stainless steel venipuncture needles, and anticoagulants (Jayawardene et al. 2021). Some biomonitoring studies were not considered quantitatively in this assessment due to the methodological uncertainties associated with the measurement of aluminium. In particular, pooling samples can result in cumulative contamination (Bornhorst et al. 2005; Bowen et al. 2010; Rothhaar et al. 2016). As a result, studies of pooled blood samples were not considered quantitatively in this assessment.

Canadian population-level whole blood aluminium concentrations were generated in a recent CHMS biobank project (Jayawardene et al. 2021). In this project, whole blood samples, originally collected during Cycle 2 of the CHMS (5752 samples) and stored in the CHMS biobank, were analyzed by Inductively Coupled Plasma Mass Spectrometry (ICP-MS) at Health Canada's Health Products Laboratory in Longueuil, Quebec, for aluminium concentration (Health Canada 2013; Jayawardene et al. 2021). The CHMS is a national survey carried out by Statistics Canada in partnership with Health Canada and the Public Health Agency of Canada, which collects information from Canadians

about their general health (Health Canada [modified 2021b]). It is designed to be nationally representative[10] and includes a biomonitoring component. The CHMS is not a targeted survey, and thus, does not target individuals with high metal exposure or who live near point sources of exposure. The CHMS Cycle 2 samples were collected between 2009 and 2011 from Canadians aged 3 to 79 years old, including pregnant females and both fasting and non-fasting individuals, at 18 sites across Canada (Health Canada 2013). A summary of aluminium concentrations measured in whole blood collected during Cycle 2 of the CHMS is provided in Table 8-4.

Table 8-4. Whole blood aluminium concentrations (µg/L) measured in biobank samples from the CHMS - Cycle 2

Substance	Number of samples	MRL (µg/L)	Median (µg/L)	95th percentile (µg/L)	Percentage detected above MRL
Aluminium	5752	8	<8	<8	2.9

Abbreviation: MRL, method reporting limit

Aluminium was not detected at or above the method reporting limit of 8 µg/L in 97.1% of the Canadian population (age group 3 to 79 years old). Overall, the median and 95th percentile population-weighted aluminium concentrations were below the method reporting limit (Jayawardene et al. 2021). However, the 95th percentile aluminium concentration in children aged 6 to 19 years was in the range of 10 µg/L to 11 µg/L (Table I-1, Appendix I). Males 12 to 19 years old had the highest 95th percentile blood concentration at 12 µg/L (Table I-1, Appendix I). This elevated aluminium blood concentration in children and teens, compared to adults, is also observed in dietary intake estimates. Mean dietary intake estimates for the 4- to 18-year-old age groups (range from 0.032 mg/kg bw/day to 0.073 mg/kg bw/day) are higher than the mean for the adult age groups (range from 0.025 mg/kg bw/day to 0.032 mg/kg bw per day) (Table G-3, Appendix G). Similarly to the CHMS data, a study conducted in the UK on healthy subjects found that males (5.4 µg/L) had higher plasma aluminium concentrations than did females (4.0 µg/L) (Sharp et al. 1993).

Children under 3 years of age were not included in the CHMS. However, some blood data are available for infants aged 2 months to 23.5 months from studies in the United States examining the effects of vaccinations with aluminium-containing adjuvants (Movsas et al. 2013; Tsou et al. 1991). Median or average aluminium concentrations in pre-vaccine serum or plasma for infants aged 2.5 months to 13 months ranged from 4.3 µg/L to 11.1 µg/L. It should be noted that the Movsas et al. (2013) and Tsou et al. (1991) studies were limited by small sample numbers (n=15 and n=16, respectively).

[10] Cycle 2 of the Canadian Health Measures Survey covers the population 3 to 79 years of age living in the ten provinces and the three territories. Excluded from the survey's coverage are: persons living on reserves and other Indigenous settlements in the provinces; full-time members of the Canadian Forces; and the institutionalized population and residents of certain remote regions. Altogether, these exclusions represent less than 4% of the target population.

Additionally, a study of aluminium levels in pre-term and full-term infants (1 day to 88 days old, n=176) reported average plasma aluminium levels between 7.8 µg/L and 13.2 µg/L (0.29 µmol/L and 0.49 µmol/L, respectively) (Bougle et al. 1992).

In a small Canadian study conducted in British Columbia, serum aluminium concentrations were measured in 61 healthy, non-smoking adults (Clark et al. 2007). The mean serum aluminium concentration was 1.81 µg/L, and the 95th percentile was 10 µg/L. There was no significant difference between the serum aluminium concentrations in men and women and no age dependence (Clark et al. 2007).

Whole blood aluminium concentrations from the CHMS and the British Columbia study (Clark et al. 2007) are similar to whole blood, serum, or plasma aluminium concentrations reported in adult biomonitoring studies conducted in Sweden, France, Italy, UK, Germany, Belgium, Korea, and Australia (averages or medians ranged from 0.11 µg/L to 10.8 µg/L) (Minoia et al. 1990; Sharp et al. 1993; Rodushkin et al. 1999; Goullé et al. 2005; Kim et al. 2017; Nisse et al. 2017; Troisi et al. 2019; Heitland and Köster 2021; Hoet et al. 2021; Komarova et al. 2021).

Concentrations of aluminium were measured in the whole blood and urine of people residing in communities across the Dehcho and Sahtú regions of the Northwest Territories (Ratelle et al. 2019, 2020b). More than 80% of whole blood samples (n=276) from these communities had concentrations below the limit of detection (LOD=0.765 µg/L), whereas the 95th percentile was 44 µg/L. In the same study, aluminium was detected in 100% of the urine samples (n=198) (LOD=1.4 µg/L), with a mean and 95th percentile aluminium concentration of 14 µg/L and 41 µg/L, respectively.

A similar biomonitoring project measuring aluminium concentrations in whole blood and urine was conducted in Old Crow, Yukon (Drysdale et al. 2021). Aluminium was detected in 78% of the whole blood samples (n=54) (LOD=0.765 µg/L), with mean and 95th percentile whole blood aluminium concentrations of 19 µg/L and 539 µg/L, respectively. Aluminium was also detected in 100% of urine samples from this study population (n=44) (LOD=1.4 µg/L), with mean and 95th percentile aluminium concentrations of 11 µg/L and 30 µg/L, respectively.

Additionally, a pilot biomonitoring project measured aluminium in the urine of 29 Indigenous and non-Indigenous pregnant women from two communities near a natural gas exploitation point source in Northeastern British Columbia (Caron-Beaudoin et al. 2019). The mean and 95th percentile urinary aluminium concentrations in this population were 15.29 µg/L and 355 µg/L, respectively. However, there is uncertainty associated with the aluminium concentration measured in these studies. Typically, urine concentrations of aluminium are on average 2.7 times higher than blood concentrations (Poddalgoda et al. 2021). Data from these studies show inconsistent trends in aluminium concentrations measured in blood and urine within the same populations. Without additional information on sources of aluminium exposure in these communities, these concentrations could not be verified. Aluminium contamination during collection, storage, and analysis of samples is very common. Since these studies did not target

aluminium (instead, a large number of metals were measured using ICP-MS), it is unclear whether specific measures to reduce aluminium contamination were applied during analysis. This increases the uncertainty of these reported results.

The CHMS 95th percentile concentration for ages 3 to 79 years old, which was less than 8 µg/L, and the 95th percentile concentration of the highest exposed group (males 12 to 19 years old), which was 12 µg/L, were brought forward for risk characterization. Confidence is high in this data set as additional precautions were taken to minimize contamination during sample pre-treatment and analysis in order to ensure the accuracy of the reported results. Measures taken included using polystyrene centrifuge tubes (instead of glass tubes made with Al-borosilicate or polypropylene); pre-rinsing containers and accessories (for example, automatic pipette tips, vials) with a dilute nitric acid solution followed by ultrapure water; using a Teflon perfluoroalkoxy (PFA) automatic solution dispenser to prevent contamination during liquid transfer; pre-soaking polystyrene centrifuge tubes with ultrapure water and drying prior to use; and using high or ultrapure reagents, powderless nitrile gloves, and lint-free tissue (Kimwipe^{TM}). The auto-sampler was placed inside a Plexiglas enclosure, and additional Plexiglas cover sheets (placed approximately 30 cm from the ceiling ventilation near the workspace) were used to minimize airborne contamination of the samples during handling and analysis. To reduce aluminium contamination within the instrument, the ICP-MS inlet tubing was rinsed with an acid solution after replacement, and an acid solution was pumped for at least 1 hour to allow the aluminium signal to stabilize prior to carrying out the sample analysis. Despite these measures and in order to maintain reproducibility, a method reporting limit below 8 ug/L was not possible (Jayawardene et al. 2021). Confidence is high that reportable aluminium blood concentrations were not underestimated; however, it is possible that they could be overestimated despite the efforts to minimize contamination. In addition, the highest average concentration (13.2 µg/L) obtained from small-scale studies of infants under 3 years old was brought forward as part of the weight of evidence to characterize risk for Canadians under 3 years of age.

Other available biomonitoring data presented here, including those from populations that may be more vulnerable to experiencing adverse health effects due to greater exposure, were not brought forward for risk characterization, given the availability of more reliable exposure estimation data for these populations (for example, dietary intakes of country foods, point source air concentration data). Inconsistent trends between urine and blood concentrations and the absence of source attribution information have created uncertainties in the interpretation of the biomonitoring data from these studies.

8.3 Characterization of risk to human health

The human health assessment took into consideration those groups of individuals within the Canadian population who, due to greater susceptibility or greater exposure, may be more vulnerable to experiencing adverse health effects. For instance, age-specific exposures are routinely estimated and developmental and reproductive toxicity studies

are evaluated for potential adverse health effects. Human biomonitoring data were available for infants, children and pregnant women and pregnant people. These subpopulations were taken into account in the risk assessment outcomes of aluminum-containing substances. In addition, exposure from consuming traditional, subsistence or country foods for certain Indigenous communities and exposure from outdoor air for people living near sources of release were examined. The potential for cumulative effects was considered in this assessment by examining cumulative exposures to total aluminium.

Where adequate biomonitoring data was available, human health risks from systemic exposure to the aluminium-containing substances in this group were characterized using the Biomonitoring-based Approach 2 (Health Canada [modified 2016]). Systemic exposure to total aluminium in the Canadian population over 3 years of age was also characterized using biomonitoring data from the CHMS biobank project. Blood aluminium concentrations from the CHMS biobank are below the derived BE (Table 8-5).

No blood samples from children under the age of 3 were taken as part of the CHMS Cycle 2. Therefore, risk was quantified using aluminium concentrations in blood from smaller-scale biomonitoring studies of infants 0 to 23.5 months of age (Tsou et al. 1991; Bougle et al. 1992; Movsas et al. 2013). The highest average aluminium concentration reported in small-scale biomonitoring studies of infants is below the derived BE (Table 8-5). Estimates of aluminium intake from environmental media, food, and drinking water (Table G-5, Appendix G) were also considered in order to characterize the exposure of children under 3 years of age. The estimated average daily intakes of aluminium from environmental media, food, and drinking water are below the daily intake level derived from the JECFA PTWI (0.3 mg Al/kg bw/day) (JECFA 2012) for all age groups under 3 years of age (Table 8-5).

Available data, albeit limited, were used to estimate the exposure of certain Indigenous communities to aluminium from the consumption of specific country foods. Due to the limitations of available biomonitoring studies in Indigenous communities (section 8.2.3), dietary intake estimates derived from the consumption of these country foods (that is, land animals and organ meats) were used to characterize the exposure of certain Indigenous communities in the NWT. The highest estimated intake of aluminium from the consumption of certain country foods is below the daily intake level of 0.3 mg Al/kg bw/day, derived based on JECFA's PTWI (JECFA 2012) (Table 8-5).

Table 8-5. Relevant exposure values, hazard values, and determination of risk of systemic exposure to the Aluminium-containing Substances Group

Population	Exposure	Hazard guidance value	Exceedance (yes/no)[a]
General population biomonitoring, CHMS: 3 to 79	<8 µg/L[b]	16 µg/L[c]	No

years old, 95th percentile			
Biomonitoring, CHMS: males 12 to 19 years old, 95th percentile	12 µg/L[b, d]	16 µg/L[c]	No
Biomonitoring, (Bougle et al. 1992): infants 2.5 to 13 months	13.2 µg/L[e]	16 µg/L[c]	No
Daily intake from environmental media, food, and drinking water for the general population: 6 months to 1 year old[f]	0.16 mg/kg bw/day[g]	0.3 mg/kg bw/day[h]	No
Dietary intake from country foods: children 4 to 8 years old, Indigenous community, NWT[f]	0.0790 mg/kg bw/day[i]	0.3 mg/kg bw/day[h]	No

[a] Assessment to determine if the exposure value (that is, whole blood concentration or intake estimate) exceeds the hazard guidance value (that is, BE or JECFA reference dose).
[b] Jayawardene et al. 2021
[c] Whole blood BE of 16 µg/L derived from a PBPK model and corresponding to a daily intake of 0.3 mg Al/kg bw/day, based on the critical POD (a NOAEL of 30 mg/kg bw/day) and an uncertainty factor of 100 identified by JECFA (2012) for the derivation of their PTWI (Poddalgoda et al. 2021).
[d] Use with caution. Statistics Canada guidelines for release stipulate that coefficients of variation (CVs) between 16.6% and 33.3% are considered to have a high sampling variability and caution is recommended when using these data.
[e] Highest plasma or serum concentration reported in studies of infants 0 to 23.5 months old (Tsou et al. 1991; Bougle et al. 1992; Movsas et al. 2013).
[f] Based on age group(s) with the highest potential exposure in mg/kg bw/day.
[g] Details of intake estimates are presented in Appendix G, Tables G-3 and G-5.
[h] JECFA PTWI (2 mg Al/kg bw) converted to daily value (JECFA 2012).
[i] Based on concentrations of aluminium measured in land animals and organ meats from the NWT (Larter et al. 2016) and consumption rates reported in the NWT (Ratelle et al. 2020a). Further details of estimates are presented in Appendix G, Table G-4.

Overall, risk estimates for the Aluminium-containing Substances Group for systemic exposure of the Canadian population, including subpopulations of interest such as children, pregnant females, and Indigenous populations, are low enough to account for uncertainties in the health effects and exposure data used to characterize risk. Therefore, at current levels of exposure, systemic exposure to the Aluminium-containing Substances Group is considered to be of low concern for the health of the Canadian general population, as well as that of subpopulations of interest.

The risk associated with portal-of-entry effects from inhalation exposure to the Aluminium-containing Substances Group was assessed using traditional risk assessment methods. A NOAEC of 100 mg/m^3, which is based on multifocal granulomas in the lungs of rats, was selected for risk characterization from acute inhalation exposure (Thomson et al. 1986).

Table 8-6. Relevant exposure values, and margins of exposure, for determination of risk from acute inhalation exposure for the Aluminium-containing Substances Group

Exposure scenario	Adjusted exposure air concentration (mg/m^3)[a]	MOE[b,c]
Aerosol Temporary Hair Dye, 30% C.I. Pigment Blue 29 (4 years old to adult)	1.1×10^{-1}	909

[a] Adjusted to match exposure duration of toxicology study (4 hours).
[b] Based on age group(s) with highest potential exposure.
[c] Margin of exposure calculated using a no effect concentration of 100 mg/m^3 (Thomson et al. 1986).

From the available studies, a NOAEC of 4.1 mg aluminium and aluminium oxide particles/m^3 from a worker study was selected as the critical POD to characterize the risk of repeated inhalation exposure for the Aluminium-containing Substances Group (Hunter et al. 1944). Both the NOAEC and the estimated aluminium air concentrations were adjusted to represent continuous chronic exposure. This adjustment was made to address the differences in exposure duration between that in the critical health effects study (Hunter et al. 1944) and from actual use of self-care products available to consumers. The NOAEC of 4.1 mg/m^3 is equivalent to an adjusted concentration of 0.98 mg/m^3, as noted in the Health Effects section (8.1.3).

The adjusted air concentrations from the use of self-care products and the margins of exposure from products available to consumers, outdoor air, indoor air, and personal air in subways are presented in Table 8-7. The estimated air concentrations in Table 8-7 were compared to the critical POD identified for the 55 aluminium-containing substances, because the exposures do not result in exposure to aluminum hydroxychloride or aluminum chlorohydrate.

Table 8-7. Relevant exposure values, and margins of exposure, for determination of risk from daily inhalation exposure for the Aluminium-containing Substances Group

Chronic exposure scenario	Exposure air concentration (mg substance/m^3)	MOE[a]
Mean daily air concentration, ambient air in Canada (aluminium in $PM_{2.5}$)	3.0×10^{-5}	32 667
Mean daily ambient air concentrations with point source influence: aluminium smelter in QC (aluminium in $PM_{2.5}$)	2.8×10^{-4}	3 500

Mean daily air concentration with transit influence (aluminium in $PM_{2.5}$)	7.8×10^{-5}	12 564
Aerosol Deodorant, 3% Potassium Alum (Adult)[b]	7.6×10^{-3} [c]	129
Aerosol Sunscreen, 2.535% Aluminum Oxide (1 year old to 13 years old)[b]	1.0×10^{-3} [c]	980
Face Powder, 30% Aluminum Hydroxide (14 years old to adult)[b]	1.4×10^{-3} [c]	700
Powdered Sunscreen, 2.535% Aluminum oxide (1 year old to 13 years old)[b]	1.9×10^{-4} [c]	5158

Abbreviations: adj, adjusted; Al, aluminium; MOE, margin of exposure; $PM_{2.5}$, particulate matter with aerodynamic diameter of 2.5 µm or less.

[a] Margins of exposure calculated based on the adjusted no-effect level of 0.98 mg aluminium and aluminium oxide particles/m^3 (Hunter et al. 1944).

[b] Based on age group(s) with highest potential exposure.

[c] Adjusted to a continuous exposure scenario based on duration of exposure and frequency of use (for details see Appendix H, Table H-1).

A separate POD was selected for repeated inhalation exposure to aluminum hydroxychloride and aluminum chlorohydrate because of the differences in the effect observed in the lung as compared to other substances in the Aluminium-containing Substances Group. A NOAEC of 0.25 mg/m^3, based on multifocal granulomatous pneumonia in rats and guinea pigs, was selected for repeated inhalation exposure to aluminum hydroxychloride and aluminum chlorohydrate (Steinhagen and Cavender 1978).

Adjustments to represent continuous chronic exposure, similar to those discussed above, were made to the critical NOAEC (Steinhagen and Cavender 1978) and estimated exposure concentrations. The NOAEC of 0.25 mg/m^3 is equivalent to an adjusted concentration of 0.045 mg/m^3, as noted in the Health Effects section (section 8.1.3). The adjusted air concentrations from the use of self-care products (aerosol antiperspirants, aerosol foot deodorant spray, and loose foot powder) and the margins of exposure for the determination of risk of inhalation exposure to aluminum hydroxychloride and aluminum chlorohydrate are presented in Table 8-8.

Table 8-8**. Relevant exposure values, and margins of exposure, for determination of risk from inhalation exposure to aluminum hydroxychloride (CAS RN 1327-41-9) and aluminum chlorohydrate (CAS RN 12042-91-0) in antiperspirants and deodorant products**

Exposure scenario	Adjusted daily air concentrations (mg substance/m^3)[a]	MOE[b]
Aerosol antiperspirant, 25% aluminum chlorohydrate, adult	1.4×10^{-2}	3

Exposure scenario	Adjusted daily air concentrations (mg substance/m^3)[a]	MOE[b]
Aerosol antiperspirant, 25% aluminum chlorohydrate, 14 to 18 years old	1.3×10^{-2}	3
Aerosol antiperspirant, 25% aluminum chlorohydrate, 9 to 13 years old	1.2×10^{-2}	4
Aerosol foot deodorant spray, 10% aluminum chlorohydrate, 2 years old to adult	3.2×10^{-3}	14
Loose foot powder, 1% aluminum chlorohydrate, 3 years old to adult	3.6×10^{-5}	1250

[a] Adjusted to a continuous exposure scenario based on duration of exposure and frequency of use (for details, see Appendix H, Table H-1)..
[b] Margins of exposure calculated based on the adjusted no-effect level of 0.045 mg/m^3 (Steinhagen and Cavender 1978).

Given the use of a critical endpoint from a chronic inhalation study in humans, the calculated margins of exposure for 53 of the 55 aluminium-containing substances are considered adequate to address uncertainties in the health effects and exposure data used to characterize risk (Table 8-6, Table 8-7). However, the margins of exposure between critical effects and inhalation exposure to aluminum hydroxychloride (CAS RN 1327-41-9) and aluminum chlorohydrate (CAS RN 12042-91-0) resulting from the use of antiperspirant and deodorant aerosol products are considered potentially inadequate to address uncertainties in the health effects and exposure data used to characterize risk (Table 8-8).

8.4 Uncertainties in evaluation of risk to human health

The key sources of uncertainty within this assessment are summarized below.

Aluminum hydroxychloride and aluminum chlorohydrate belong to a broader group of “aluminum chlorohydrate and its associated complexes” as defined on the Cosmetics Ingredient Hotlist (Health Canada [modified 2022a]). Although the scope of this draft assessment is limited to aluminum hydroxychloride and aluminum chlorohydrate, similar risk is expected from the inhalation of aerosol deodorant and antiperspirant products containing the other substances listed under the Cosmetics Ingredient Hotlist entry for aluminium chlorohydrate and its associated complexes.

Confidence is high that the nervous system is the most sensitive target organ for aluminium-induced systemic toxicity. There is also high confidence that the critical study selected for the risk characterization of systemic exposure to aluminium would account for health effects in all age groups including children. However, there is some uncertainty in the health effects data set for inhalation exposure to the 55 aluminium-containing substances, because the substance-specific inhalation toxicity data are

limited. Additionally, there is uncertainty in the composition of aluminium dusts reported in the study used to inform the inhalation POD (Hunter et al. 1944).

There is uncertainty associated with the use of whole blood as a biomarker of aluminium exposure due to the analytical and methodological challenges of accurately measuring aluminium levels in blood and urine. The potential for contamination during sample collection, analysis, and study design (for example, sample pooling) from the ubiquitous presence of aluminium in the environment creates challenges for the interpretation of reported biomonitoring data. Due to the degree of uncertainty caused by potential contamination, data from multiple Canadian biomonitoring studies were not used quantitatively in this assessment (Alberta Health and Wellness 2008, 2010; Saskatchewan Ministry of Health 2019; Caron-Beaudoin et al. 2019; Ratelle et al. 2019, 2020b; Drysdale et al. 2021).

In the CHMS, population-weighted data are representative of 97% of the Canadian population, but the survey excludes people living on reserves or in other Indigenous communities in the provinces, residents of institutions, full-time members of the Canadian Forces, persons living in certain remote areas (such as Northern Canada), and persons living in areas with a low population density (Health Canada 2013). Albeit limited, available data indicate that certain Indigenous communities and remote communities in proximity to a point source of release may be exposed to elevated levels of aluminium compared to the general population (Saskatchewan Ministry of Health 2010; Larter et al. 2016; Caron-Beaudoin et al. 2019; Ratelle et al. 2019, 2020a, 2020b; Drysdale et al. 2021). However, due to the analytical challenges of accurately measuring aluminium in biological media and the absence of data on potential exposure source attribution (for example, dietary intake estimates or environmental monitoring in these communities), there are insufficient data to reliably interpret the results from biomonitoring studies in these communities. In addition, there are limited data available on the concentration of aluminium in country foods consumed by Indigenous peoples. The available data on the concentration of aluminium in land mammals and organ meats from the NWT were used to characterize risk from the consumption of country foods in this assessment. Estimates of intake from land mammals and organ meat may be conservative as weekly consumption throughout the year was assumed, even though the availability of large game is seasonal. However, there are insufficient data available to conduct a fulsome analysis of daily aluminium intakes from environmental media and country food in Indigenous communities at this time. Although the risk estimates generated from the available data are considered low enough to account for uncertainties in the health effects and exposure data used to characterize risk, further generation of data to investigate the potential for elevated aluminium exposure among people residing in Indigenous communities could be beneficial for reliably characterizing the exposure and potential risk of these populations.

There is also uncertainty associated with whole blood as a biomarker of aluminium exposure based on toxicokinetic considerations. Aluminium absorption and consequent blood levels can be influenced by factors unrelated to aluminium levels, such as the form of aluminium and the presence of other chemical constituents (for example, citrate)

in the GI tract. However, an analysis of the available kinetic data and epidemiological data on workers indicated that whole blood could act as an adequate biomarker of exposure when the aluminium exposure is stable and continuous.

In the absence of substance-specific kinetic, health effects, and exposure data, data available on the metal moiety was used as a surrogate. It is important to note that, compared to the metal moiety, there may be different bioavailability and health effects associated with a specific substance, particularly with soluble and insoluble substances.

Aggregate inhalation exposure estimates were not derived for self-care products containing aluminium, but exposure to these self-care products may occur concurrently, leading to some uncertainty in the inhalation exposure estimates. Information on use patterns of self-care products, market share of products available to consumers, duration of particle clouds generated from use, and lung clearance rates of various aluminium particles would be needed in order to consider aggregate exposure estimates.

9. Conclusion

Considering all available lines of evidence presented in this draft assessment, there is low risk of harm to the environment from the 55 aluminium-containing substances. It is proposed to conclude that the 55 aluminium-containing substances do not meet the criteria under paragraphs 64(*a*) or (*b*) of CEPA as they are not entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity or that constitute or may constitute a danger to the environment on which life depends.

Considering all the information presented in this draft assessment, it is proposed to conclude that aluminum hydroxychloride (CAS RN 1327-41-9) and aluminum chlorohydrate (CAS RN 12042-91-0) meet the criteria under paragraph 64(*c*) of CEPA as they are entering or may enter the environment in a quantity or concentration or under conditions that constitute or may constitute a danger in Canada to human life or health.

Considering all the information presented in this draft assessment, it is proposed to conclude that 53 of the 55 aluminium-containing substances do not meet the criteria under paragraph 64(*c*) of CEPA as they are not entering the environment in a quantity or concentration or under conditions that constitute or may constitute a danger in Canada to human life or health.

It is therefore proposed to conclude that aluminum hydroxychloride (CAS RN 1327-41-9) and aluminum chlorohydrate (CAS RN 12042-91-0) meet one or more of the criteria set out in section 64 of CEPA. It is proposed to conclude that the remaining 53 aluminium-containing substances do not meet any of the criteria set out in section 64 of CEPA. It is also proposed to conclude that aluminum hydroxychloride and aluminum

chlorohydrate meet the persistence criteria but not the bioaccumulation criteria as set out in the *Persistence and Bioaccumulation Regulations* of CEPA.

References

Abril GA, Wannaz ED, Mateos AC, Pignata ML. 2014. Biomonitoring of airborne particulate matter emitted from a cement plant and comparison with dispersion modelling results. Atmos Environ. 82:154-163.

[ACGIH] American Conference of Governmental Industrial Hygienists. 2008. Aluminum metal and insoluble compounds. 7th ed. Cincinnati (OH): ACGIH. p. 1-14.

Adams WJ, Cardwell AS, DeForest DK, Gensemer RW, Santore RC, Wang N, Nordheim E. 2018. Aluminum bioavailability and toxicity to aquatic organisms: Introduction to the special section. Environ Toxicol Chem. 37(1):34-35.

Akinola OB, Biliaminu SA, Adediran RA, Adeniye KA, Abdulquadir FC. 2015. Characterization of prefrontal cortex microstructure and antioxidant status in a rat model of neurodegeneration induced by aluminium chloride and multiple low-dose streptozotocin. Metab Brain Dis. 30(6):1531-1536.

Alberta Environment. 2001. Guidelines for the application of municipal wastewater sludges to agricultural lands. March 2001 (updated August 2009) [PDF]. Edmonton (AB): Alberta Environment.

Alberta Environment. 2002. Lake Wabamun water quality and sediment survey. Preliminary report. Edmonton (AB): Alberta Environment. [accessed 2016 Nov]. 37 p. ISBN 0-7785-2296-0.

Alberta Environment. 2003a. Spatial variability of water quality in Wabamun Lake (July 2002). Draft report. Edmonton (AB): Alberta Environment, Environmental Monitoring and Evaluation Branch.

Alberta Environment. 2003b. Wabamun Lake water quality 1982 to 2001. Edmonton (AB): Alberta Environment, Science and Standards Branch.

Alberta Environment. 2006. Wabamun Lake oil spill August 2005: Data report for water and sediment quality in the pelagic area of the lake (August 4-5 to September 15, 2005). Edmonton (AB): Alberta Environment. [accessed 2016 Nov]. 99 p. ISBN 0-7785-4589-X.

Alberta Health and Wellness. 2008. Alberta Biomonitoring Program: Chemicals in serum of pregnant women in Alberta (2005) [PDF]. Edmonton (AB): Government of Alberta. [accessed 2021 Sep 12].

Alberta Health and Wellness. 2010. Alberta Biomonitoring Program: Chemicals in serum of children in southern Alberta (2004–2006) - Influence of age and comparison to pregnant women [PDF]. Edmonton (AB): Government of Alberta. [accessed 2021 Sep 12].

Anderson EL, Sheehan PJ, Kalmes RM, Griffin JR. 2017. Assessment of health risk from historical use of cosmetic talcum powder. Risk Anal. 37(5):918-929.

Arcus-Arth A, Krowech G, Zeise L. 2005. Breast milk and lipid intake distributions for assessing cumulative exposure and risk. J Expo Anal Environ Epidemiol. 15(4):357-365.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2008. Toxicological profile for aluminum [PDF]. Atlanta (GA): US Department of Health and Human Services. [accessed 2019 Dec 19].

Ayeni O, Kambizi L, Laubscher C, Fatoki O, Olatunji O. 2014. Risk assessment of wetland under aluminium and iron toxicities: A review. Aquat Ecosyst Health Manag. 17(2):122-128.

Barabasz W, Albińska D, Jaśkowska M, Lipiec J. 2002. Ecotoxicology of aluminium. Pol J Environ Stud. 11(3):199-203.

Bélanger N, Fyles H, Hendershot W. 1999. Chemistry, bioaccumulation and toxicity of aluminum in the terrestrial environment – PSL2 assessment of aluminum salts. Unpublished report. Montreal (QC): Environment Canada.

Bergman JJ, Boots BF. 1997. Alum sludge management at Buffalo Pound WTP. Report prepared by Buffalo Pound Water Treatment Plant for Environment Canada. Unpublished report. Gatineau (QC): Environment Canada.

Bertram J, Brand P, Hartmann L, Schettgen T, Kossack V, Lenz K, Purrio E, Reisgen U, Kraus T. 2015. Human biomonitoring of aluminium after a single, controlled manual metal arc inert gas welding process of an aluminium-containing worksheet in nonwelders. Int Arch Occup Environ Health. 88(7):913-923.

Bertsch PM, Bloom PR. 1996. Chapter 18: Aluminum. In: Sparks DL, editor. Methods of Soil Analysis, Part 3, Chemical Methods. No. 5. Soil Sci. Soc. Amer, Inc. p. 517-550.

Bertsch PM, Parker DR. 1996. Aqueous polynuclear aluminum species. In: Sposito G, editor. The environmental chemistry of aluminum. 2nd ed. Boca Raton (FL): CRC Press. p. 117-168.

Bezerra SMC. 2014. Enhancing characterization of water use practices in cement manufacturing and related construction sectors [dissertation]. Guelph (ON): The University of Guelph.

Bhattacharjee S, Zhao Y, Hill JM, Culicchia F, Kruck TP, Percy ME, Pogue AI, Walton JR, Lukiw WJ. 2013. Selective accumulation of aluminum in cerebral arteries in Alzheimer's disease (AD). J Inorg Biochem. 126:35-37.

Bilalis D, Tzortzi I, Vavoulidou E, Karkanis A, Emmanouel N, Efthimiadou A, Katsenios N, Patsiali S, Dellaporta L. 2013. Effects of aluminum and moisture levels on aluminum bioaccumulation and protein content in the earthworm *Octodrilus complanatus*. J Soil Sci Plant Nutr. 13(4):845-854.

Bishop P, Quin BF. 2013. Undiagnosed metal phytotoxicity in soils: Measurement of soil pH micro-variability under Manawatu pastures, and an assessment of an alternative means of amelioration. Proceedings of the New Zealand Grassland Association. 75:179-184.

Bloom PR, Erich MS. 1995. The quantitation of aqueous aluminum. In: Sposito G, editor. The environmental chemistry of aluminum. 2nd ed. Boca Raton (FL): CRC Press. p.1-38.

Bornhorst JA, Hunt JW, Urry FM, McMillin GA. 2005. Comparison of sample preservation methods for clinical trace element analysis by inductively coupled plasma mass spectrometry. Am J Clin Pathol. 123(4):578-583.

Bougle D, Bureau F, Voirin J, Neuville D, Duhamel JF. 1992. A cross-sectional study of plasma and urinary aluminum levels in term and preterm infants. JPEN J Parenter Enteral Nutr. 16(2):157-159.

Boullemant A. 2011. PM2.5 emissions from aluminum smelters: Coefficients and environmental impact. J Air Waste Manag Assoc. 61(3):311-318.

Bowen RAR, Hortin GL, Csako G, Otañez OH, Remaley AT. 2010. Impact of blood collection devices on clinical chemistry assays. Clin Biochem. 43(1-2):4-25.

[BQMA] Banque de données sur la qualité du milieu aquatique [database]. 2015. Quebec (QC): Government of Quebec. Datasets for all stations monitoring metals. [accessed 2015 Nov]. (available in French only).

Brothers PJ, Ruggiero CE. 2011. Coordination and solution chemistry of the metals: Biological, medical and environmental relevance. In: Aldridge S, Downs AJ, editors. The Group 13 metals, aluminium, gallium, indium, and thallium: Chemical patterns and peculiarities. New York (NY): John Wiley and Sons. p. 519-611.

Buchan NI, Potemski RM, Kuech TF. 1991. Vapor pressure and vapor composition of diethylgallium chloride and diethylaluminum chloride. J Chem Eng Data. 36(4):372-374.

Buckler DR, Cleveland L, Little EE, Brumbaugh WG. 1995. Survival, sublethal responses, and tissue residues of Atlantic salmon exposed to acidic pH and aluminum. Aquat Toxicol. 31(3):203-216.

Canada. 1985. *Food and Drugs Act*. R.S.C. 1985, c.F-27.

Canada. 1999. *Canadian Environmental Protection Act, 1999*. S.C. 1999, c.33. Canada Gazette Part III, vol. 22, no. 3.

Canada. 2009. *Cosmetic Regulations.* C.R.C., c.869.

Canada, Dept. of the Environment. 2012. *Canadian Environmental Protection Act, 1999*: *Notice with respect to certain substances on the Domestic Substances List* [PDF]. Canada Gazette, Part I, vol. 146, no. 48, Supplement.

Canada. [modified 2013a]. Reducing acid rain. Government of Canada. [accessed 2020 Jan].

Canada. [modified 2013b]. Air pollution: Effects on soil and water. Government of Canada. [accessed 2020 Jan].

Canada. [modified 2017a]. Overview of freshwater quality monitoring and surveillance. [accessed 2020 Jan].

Canada, Dept. of the Environment. 2017b. *Canadian Environmental Protection Act, 1999: Notice with respect to substances included as part of the 2017 Inventory Update*. Canada Gazette, Part I, vol. 151, no. 2, p 89-161.

Canada. [modified 2018a]. Acid rain: Causes and effects. Government of Canada. [accessed 2020 Jan].

Canada. 2018b. *Fisheries Act: Pulp and Paper Effluent Regulations.* P.C. 1992-961, SOR/92-269, last amended September 26, 2018. [accessed 2020 Jan].

Canada. 2022. *Fisheries Act: Metal and Diamond Mining Effluent Regulations*. P.C. 2002-987, SOR/2002-222, last amended June 24, 2022. [accessed 2022 Oct 14].

[CBSA] Canada Border Services Agency. 2016. Information on imports corresponding to the HS Codes 2523.10.0000, 2523.21.0000, 2523.29.0000, 2523.30.0000, 2523.90.0010, 2523.90.0020, 2523.90.0030, 2523.90.0040, 2523.90.0090, 2818.20.0010, 2818.20.0020, 2818.20.0090, 2818.30.0000, 2826.12.0000, 2826.30.0000, 2827.49.0010, 2833.30.0010, 2833.30.0020, 2841.90.2010, 2842.90.0050, 2842.90.9930, 2931.00.1040, 2931.90.0010, 3206.41.0000, 3206.41.0010, 3206.41.0021, 3206.41.0029, 3802.90.0010,

3802.90.0040. Ottawa (ON): CBSA, Commercial and Trade Operations Division. Confidential information received by Environment and Climate Change Canada for calendar years 2010-2013.

[CANMET] Canada Centre for Mineral & Energy Technology and Radian Canada. 1993. Raw material balances, energy profiles and environmental unit factor estimates: Cement and structural concrete products. Report posted by the Athena Sustainable Materials Institute, Ottawa, Canada.

[CCME] Canadian Council of Ministers of the Environment. 2007. A protocol for the derivation of water quality guidelines for the protection of aquatic life [PDF]. Winnipeg (MB): Canadian Council of Ministers of the Environment.

[CCME] Canadian Council of Ministers of the Environment. 2008. Canadian Water Quality Guidelines [PDF]. Winnipeg (MB): Canadian Council of Ministers of the Environment.

[CCREM] Canadian Council of Resource and Environment Ministers. 1987. Canadian Water Quality Guidelines. Task Force on Water Quality Guidelines. Ottawa (ON): Environment Canada.

Cardwell AS, Adams WJ, Gensemer RW, Nordheim E, Santore RC, Ryan AC, Stubblefield WA. 2018. Chronic toxicity data of aluminum, at a pH of 6, to freshwater organisms: Empirical data for the development of international regulatory standards/criteria. Environ Toxicol Chem. 37(1):36-48.

Caron-Beaudoin É, Bouchard M, Wendling G, Barroso A, Bouchard MF, Ayotte P, Frohlich KL, Verner M-A. 2019. Urinary and hair concentrations of trace metals in pregnant women from Northeastern British Columbia, Canada: A pilot study. J Expo Sci Environ Epidemiol. 29(5):613-623.

Cement Association of Canada. 2005. Cement and structural concrete products: Life cycle inventory update #2 [PDF]. Prepared by Athena Sustainable Materials Institute. Ottawa, Canada.

[CER] Canada Energy Regulatory. [modified 2022]. Canada's retiring coal-fired power plants will be replaced by renewable and low-carbon energy sources. Calgary (AB): Government of Canada. [accessed 2022 Oct].

Chan L, Receveur O, Sharp D, Schwartz H, Ing A, Tikhonov C. 2011. First Nations Food, Nutrition and Environment Study (FNFNES): Results from British Columbia (2008/2009). Prince George (BC): University of Northern British Columbia. [accessed 2022 Aug 16].

Chan L, Receveur O, Sharp D, Schwartz H, Ing A, Fediuk K, Black A, Tikhonov C. 2012. First Nations Food, Nutrition and Environment Study (FNFNES): Results from Manitoba (2010) [PDF]. Prince George (BC): University of Northern British Columbia. [accessed 2022 Aug 16].

Chan L, Receveur O, Batal M, David W, Schwartz H, Ing A, Fediuk K, Black A, Tikhonov C. 2014. First Nations Food, Nutrition and Environment Study (FNFNES): Results from Ontario (2011/2012) [PDF]. Ottawa (ON): University of Ottawa. [accessed 2022 Aug 16].

Chan L, Receveur O, Batal M, David W, Schwartz H, Ing A, Fediuk K, Tikhonov C. 2016. First Nations Food, Nutrition and Environment Study (FNFNES): Results from Alberta (2013) [PDF]. Ottawa (ON): University of Ottawa. [accessed 2022 Aug 16].

Chan L, Receveur O, Batal M, David W, Schwartz H, Ing A, Fediuk K, Tikhonov C. 2017. First Nations Food, Nutrition and Environment Study (FNFNES): Results from the Atlantic Region (2014) [PDF]. Ottawa (ON): University of Ottawa. [accessed 2022 Aug 16].

Chan L, Receveur O, Batal M, Sadik T, Schwartz H, Ing A, Fediuk K, Tikhonov C. 2018. First Nations Food, Nutrition and Environment Study (FNFNES): Results from Saskatchewan (2015) [PDF]. Ottawa (ON): University of Ottawa. [accessed 2022 Aug 16].

Chan L, Batal M, Receveur O, Sadik T, Schwartz H, Ing A, Fediuk K, Tikhonov C, Lindhorst K. 2019a. First Nations Food, Nutrition and Environment Study (FNFNES): Results from Quebec (2016) [PDF]. Ottawa (ON): University of Ottawa. [accessed 2022 Aug 16].

Chan L, Batal M, Sadik T, Tikhonov C, Schwartz H, Fediuk K, Ing A, Marushka L, Lindhorst K, Barwin L,et al. 2019b. First Nations Food, Nutrition and Environment Study (FNFNES) final report for eight assembly of First Nations regions: Draft comprehensive technical report [PDF]. Assembly of First Nations, University of Ottawa, Université de Montréal. [accessed 2021 Oct 17].

[CEH] Chemical Economics Handbook. 2021. Aluminum chemicals. Wietlisbach S, Gao A, Funada C, Davis S. IHS Markit. [accessed 2022 Oct]. [restricted access].

Cheminfo Services. 2008. Characterization and analysis of aluminum salts and releases to the environment in Canada. Final report. Vancouver (BC). Prepared for Environment Canada, Environmental Stewardship Branch.

Christie H, MacKay RJ, Fisher AM. 1963. Pulmonary effects of inhalation of aluminum by rats and hamsters. Am Ind Hyg Assoc J. 24(1):47-56.

Clark NA, Teschke K, Rideout K, Copes R. 2007. Trace element levels in adults from the west coast of Canada and associations with age, gender, diet, activities, and levels of other trace elements. Chemosphere. 70(1):155-164.

Cleveland L, Buckler DR, Brumbaugh WG. 1991. Residue dynamics and effects of aluminum on growth and mortality in brook trout. Environ Toxicol Chem. 10(2):243-248.

Colomina MT, Sanchez DJ, Sanchez-Turet M, Domingo JL. 1999. Behavioral effects of aluminum in mice: Influence of restraint stress. Neuropsychobiology. 40(3):142-149.

Connor JN, Martin MR. 1989. An assessment of sediment phosphorus inactivation, Kezar Lake, New Hampshire. Water Resour Bull. 25(4):845-853.

[ConsExpo Web] Consumer Exposure Web Model. 2019. Bilthoven (NL): Rijksinstituut voor Volksgezondheid en Milieu [National Institute for Public Health and the Environment].

Cotton FA, Wilkinson G. 1999. Advanced inorganic chemistry. 5th ed. New York (NY): John Wiley and Sons.

Courchesne F, Hendershot WH. 1997. La genèse des podzols. Geogr Phys Quatern. 51(2):235-250.

Crane M, Atkinson C, Comber S, Sorokin N. 2007. Proposed EQS for Water Framework Directive. Annex VIII substances: Aluminium (inorganic monomeric). Science Report: SC040038/SR1; SNIFFER Report: WFD52(i). February 2007. Bristol (UK): Environment Agency. 89 p.

Crémazy A, Levy JL, Campbell PGC, Fortin C. 2013. Uptake and subcellular partitioning of trivalent metals in green alga: Comparison between Al and Sc. Biometals. 26(6):989-1001.

Cunha GOM, Almeida JA, Ernani PR, Pereira ER, Skoronski E, Lourenço LS, Brunetto G. 2018. Chemical species and aluminum concentration in the solution of acid soils cultivated with soybean and corn under liming. Rev Bras Cienc Solo. 42:e0170406.

Dabeka R, Fouquet A, Belisle S, Turcotte S. 2011. Lead, cadmium and aluminum in Canadian infant formulae, oral electrolytes and glucose solutions. Food Addit Contam Part A Chem Anal Control Expo Risk Assess. 28(6):744-753.

Darbre PD. 2005. Aluminium, antiperspirants and breast cancer. J Inorg Biochem. 99(9):1912-1919.

Darbre PD, Mannello F, Exley C. 2013. Aluminium and breast cancer: Sources of exposure, tissue measurements and mechanisms of toxicological actions on breast biology. J Inorg Biochem. 128:257-261.

Darragh KV, Ertell CA. 2003. Aluminum sulfate and alums. In: Kirk-Othmer Encyclopedia of Chemical Technology. Online version. New York (NY): John Wiley and Sons, Inc. [accessed 2020 Jan].

David MB, Driscoll CT. 1984. Aluminum speciation and equilibria in soil solutions of a Haplorthod in the Adirondack Mountains (New York, USA). Geoderma. 33(4):297-318.

Delhaize E, Ryan PR. 1995. Aluminum toxicity and tolerance in plants. Plant Physiol. 107(2):315-321.

[DOE] US Department of Energy. 1984. A review and analysis of parameters for assessing transport of environmentally released radionuclides through agriculture. ORNL-5786, September 1984. 150 p.

Dillon PJ, Evans HE, Scholer PJ. 1988. The effects of acidification on metal budgets of lakes and catchments. Biogeochemistry. 5:201-220.

Dobranskyte A, Jugdaohsingh R, Stuchlik E, Powell JJ, White KN, McCrohan CR. 2004. Role of exogenous and endogenous silicon in ameliorating behavioural responses to aluminium in a freshwater snail. Environ Pollut. 132(3):427-433.

Dolmen D, Finstad AG, Skei JK. 2018. Amphibian recovery after a decrease in acidic precipitation. Ambio. 47(3):355-367.

Drábek O, Mládková L, Borůvka L, Szakova J, Nikodem A, Nemecek K. 2005. Comparison of water-soluble and exchangeable forms of Al in acid forest soils. J Inorg Biochem. 99(9):1788-1795.

Driscoll CT, van Breemen N, Mulder J. 1985. Aluminum chemistry in a forested spodosol. Soil Sci Soc Am J. 49(2):437-444.

Driscoll CT, Otton JK, Iverfeldt Å. 1994. Trace metals speciation and cycling. In: Moldan B, Černý J, editors. Biogeochemistry of small catchments: A tool for environmental research. New York (NY): John Wiley and Sons. p. 299-322.

Driscoll CT, Postek KM. 1996. The chemistry of aluminum in surface waters. In: Sposito G, editor. The environmental chemistry of aluminum. 2nd ed. Boca Raton (FL): CRC Press. p. 363-418.

Driscoll CT, Schecher WD. 1990. The chemistry of aluminum in the environment. Environ Geochem Health. 12:28-49.

[DPD] Drug Product Database [database]. [modified 2022 Mar 10]. Ottawa (ON): Government of Canada. [accessed 2022 Oct 14].

Drysdale M, Ratelle M, Skinner K, Garcia-Barrios J, Gamberg M, Williams M, Majowicz S, Bouchard M, Stark K, Chalil D, et al. 2021. Human biomonitoring results of contaminant and nutrient biomarkers in Old Crow, Yukon, Canada. Sci Total Env. 760:143339.

Dussault ÈB, Playle RC, Dixon DG, McKinley RS. 2001. Effects of sublethal, acidic aluminum exposure on blood ions and metabolites, cardiac output, heart rate, and stroke volume of rainbow trout, *Oncorhynchus mykiss*. Fish Physiol Biochem. 25:347-357.

Edling NPG. 1961. Aluminium pneumoconiosis: A roentgendiagnostic study of five cases. Acta Radiol. 56:170-178.

Eisenreich SJ. 1980. Atmospheric input of trace metals to Lake Michigan (USA). Water Air Soil Pollut. 13:287-301.

[ECCC] Environment and Climate Change Canada. 2016. Code of practice to reduce emissions of fine particulate matter (PM2.5) from the primary aluminium sector [PDF]. Ottawa (ON): Government of Canada. ISBN 978-0-660-03883-4, Cat. No.: En14-241/2015E-PDF.

[ECCC] Environment and Climate Change Canada. 2017. DSL Inventory Update data collected under the *Canadian Environmental Protection Act, 1999, section 71: Notice with respect to substances included as part of the 2017 Inventory Update*. Data prepared by: Environment and Climate Change Canada, Health Canada; Existing Substances Program.

[ECCC] Environment and Climate Change Canada. 2018. National long-term water quality monitoring data. Open data. Ottawa (ON): Government of Canada. [accessed 2019 Jan].

[ECCC] Environment and Climate Change Canada. 2020a. Supporting documentation: Summary statistics and predicted environmental concentrations. Gatineau (QC): ECCC. Information in support of the draft assessment for the Aluminium-containing Substances Group. Available from substances@ec.gc.ca.

[ECCC] Environment and Climate Change Canada. 2020b. Science approach document: Ecological risk classification of inorganic substances. Ottawa (ON): Government of Canada. [updated 2020 Jan; accessed 2022 Oct 14].

[ECCC] Environment and Climate Change Canada. 2020c. HYDAT database. 2020. Water Survey of Canada, Environment and Climate Change Canada. [accessed 2020 Jan].

[ECCC] Environment and Climate Change Canada. 2022a. *Canadian Environmental Protection Act, 1999* - Federal environmental quality guidelines – Aluminium. Ottawa (ON): Government of Canada. [accessed 2022 Oct 14].

[ECCC] Environment and Climate Change Canada. [modified 2022b]. National Pollutant Release Inventory (NPRI) substance list by threshold. Ottawa (ON): Government of Canada. [accessed 2022 Oct 14].

[ECCC, HC] Environment and Climate Change Canada, Health Canada. 2013. Rapid screening of substances of lower concern. Ottawa (ON): Government of Canada. [accessed 2022 Aug 15].

[ECCC, HC] Environment and Climate Change Canada, Health Canada. 2016. Rapid screening of substances identified from phase two of the Domestic Substances List inventory update: Results of the screening assessment. Ottawa (ON): Government of Canada. [accessed 2022 Aug 15].

[ECCC, HC] Environment and Climate Change Canada, Health Canada. [modified 2017]. Categorization of chemical substances. Ottawa (ON): Government of Canada. [accessed 2020 Jan 5].

[ECCC, HC] Environment and Climate Change Canada, Health Canada. 2021. Screening assessment talc ($Mg_3H_2(SiO_3)_4$). Ottawa (ON): Government of Canada. [accessed 2021 Apr 17].

[EC, HC] Environment Canada, Health Canada. 2000. State of the science report for aluminum chloride, aluminum nitrate and aluminum sulphate. Ottawa (ON): Environment Canada, Health Canada.

[EC, HC] Environment Canada, Health Canada. 2010. Priority substances list assessment report - Follow-up to the state of the science report, 2000. Aluminum chloride (7446-70-0), aluminum nitrate (13473-90-0), aluminum sulphate (10043-01-3). Ottawa (ON): Government of Canada.

[ECHA] European Chemicals Agency. c2007-2019a. Registered substances database; search for CAS RN 12004-14-7. Helsinki (FI): ECHA. [accessed 2019 Mar].

[ECHA] European Chemicals Agency. c2007-2019b. Registered substances database; search for CAS RN 12042-91-0. Helsinki (FI): ECHA. [accessed 2019 Mar].

[ECHA] European Chemicals Agency. c2007-2019c. Registered substances database; search for CAS RN 13419-15-3. Helsinki (FI): ECHA. [accessed 2019 Mar].

[ECHA] European Chemicals Agency. c2007-2019d. Registered substances database; search for CAS RN 14782-75-3. Helsinki (FI): ECHA. [accessed 2019 Mar].

[ECHA] European Chemicals Agency. c2007-2019e. Registered substances database; search for CAS RN 15305-07-4. Helsinki (FI): ECHA. [accessed 2019 Mar].

[ECHA] European Chemicals Agency. c2007-2019f. Registered substances database; search for CAS RN 39290-78-3. Helsinki (FI): ECHA. [accessed 2019 Mar].

[ECHA] European Chemicals Agency. c2007-2019g. Registered substances database; search for CAS RN 54326-11-3. Helsinki (FI): ECHA. [accessed 2019 Mar].

[ECHA] European Chemicals Agency. c2007-2019h. Registered substances database; search for CAS RN 57158-29-9. Helsinki (FI): ECHA. [accessed 2019 Mar].

[ECHA] European Chemicals Agency. c2007-2019i. Registered substances database; search for CAS RN 65997-16-2. Helsinki (FI): ECHA. [accessed 2019 Mar].

[ECHA] European Chemicals Agency. c2007-2019j. Registered substances database; search for CAS RN 68425-65-0. Helsinki (FI): ECHA. [accessed 2019 Mar].

[ECHA] European Chemicals Agency. c2007-2019k. Registered substances database; search for CAS RN 68855-54-9. Helsinki (FI): ECHA. [accessed 2019 Mar].

[ECHA] European Chemicals Agency. c2007-2019l. Registered substances database; search for CAS RN 70131-50-9. Helsinki (FI): ECHA. [accessed 2019 Mar].

[EFSA] European Food Safety Authority. 2008. Safety of aluminium from dietary intake - Scientific opinion of the panel on food additives, flavourings, processing aids and food contact materials (AFC) [PDF]. EFSA Journal. 754:1-34. [accessed 2020 Jan 5].

Environment Canada. 2004. Canadian acid deposition science assessment. Catalogue No. En4-46/2004E-PDF. Toronto (ON): Government of Canada. [accessed 2022 Oct 14].

Environment Canada. 2010. Pulp and paper Environmental Effects Monitoring (EEM) technical guidance document [PDF]. Ottawa (ON): Environment Canada. [accessed 2022 Oct 14].

Environment Canada. 2013. DSL Inventory Update data collected under the *Canadian Environmental Protection Act, 1999, section 71: Notice with respect to certain substances on the Domestic Substances List*. Data prepared by: Environment Canada, Health Canada; Existing Substances Program.

Fakri S, Al-Azzawi A, Al-Tawil N. 2006. Antiperspirant use as a risk factor for breast cancer in Iraq. East Mediterr Health J. 12(3-4):478-482.

Ferguson G, Hopkins R, Poortinga K, McCallum L. 2018. Systematic review of occupational aluminum exposure and adverse health conditions. Mississauga (ON): Intrinsik.

Fernández-Dávila ML, Razo-Estrada AC, García-Medina S, Gómez-Oliván LM, Piñón-López MJ, Ibarra RG, Galar-Martínez M. 2012. Aluminum-induced oxidative stress and neurotoxicity in grass carp (Cyprinidae—*Ctenopharingodon idella*). Ecotoxicol Environ Saf. 76:87-92.

Fifield LK. 1997. Study of the kinetics of aluminium absorption and excretion in humans (with Day, Oldham and Carling (University of Manchester)). Annual Report 1997. Canberra (AU): Australian National University, Department of Nuclear Physics. p. 97-101.

Freda J, McDonald DG. 1990. Effects of aluminum on the leopard frog, *Rana pipiens*: Life stage comparisons and aluminum uptake. Can J Fish Aquat Sci. 47(1):210-216.

[FQMS] Freshwater Quality Monitoring and Surveillance. 2014. Environment and Climate Change Canada. Online data.

[FQMS] Freshwater Quality Monitoring and Surveillance. 2016. Environment and Climate Change Canada. Genie: Web Mapping Application. [restricted access].

Gensemer RW, Gondek JC, Rodriguez PH, Arbildua JJ, Stubblefield WA, Cardwell AS, Santore RC, Ryan AC, Adams WJ, Nordheim E. 2018. Evaluating the effects of PH, hardness, and dissolved organic carbon on the toxicity of aluminum to freshwater aquatic organisms under circumneutral conditions. Environ Toxicol Chem. 37(1):49-60.

Gensemer RW, Playle RC. 1999. The bioavailability and toxicity of aluminum in aquatic environments. Crit Rev Environ Sci Technol. 29(4):315-450.

George DB, Berk SG, Adams VD, Morgan EL, Roberts RO, Holloway CA, Lott RC, Holt LK, Ting RS, Welch AW. 1991. Alum sludge in the aquatic environment. Denver (CO): American Water Works Association Research Foundation. 224 p.

George DB, Berk SG, Adams VD, Ting RS, Roberts RO, Parks LH, Lott RC. 1995. Toxicity of alum sludge extracts to a freshwater alga, protozoan, fish, and marine bacterium. Arch Environ Contam Toxicol. 29:149-158.

Giorgianni CM, D'Arrigo G, Brecciaroli R, Abbate A, Spatari G, Tringali MA, Gangemi S, De Luca A. 2014. Neurocognitive effects in welders exposed to aluminium. Toxicol Ind Health. 30(4):347-356.

Golding LA, Angel BM, Batley GE, Apte SC, Krassoi R, Doyle CJ. 2015. Derivation of a water quality guideline for aluminium in marine waters. Environ Toxicol Chem. 34(1):141-151.

Golub MS, Germann SL, Han B, Keen CL. 2000. Lifelong feeding of a high aluminum diet to mice. Toxicology. 150(1-3):107-117.

Gorgogietas VA, Tsialtas I, Sotiriou N, Laschou VC, Karra AG, Leonidas DD, Chrousos GP, Protopapa E, Psarra A-M G. 2018. Potential interference of aluminum chlorohydrate with estrogen receptor signaling in breast cancer cells. J Mol Biochem. 7(1):1-13.

Goullé J-P, Mahieu L, Castermant J, Neveu N, Bonneau L, Lainé G, Bouige D, Lacroix C. 2005. Metal and metalloid multi-elementary ICP-MS validation in whole blood, plasma, urine and hair: Reference values. Forensic Sci Int. 153(1):39-44.

Government of South Australia. 2016. Department of Environment, Water and Natural Resources. Aluminium toxicity: Soil and land, fact sheet number 7. [accessed 2019 Dec].

Gross P, Harley RA Jr, deTreville RTP. 1973. Pulmonary reaction to metallic aluminum powders: An experimental study. Arch Environ Health. 26(5):227-236.

Handy RD, Eddy FB. 1989. Surface absorption of aluminium by gill tissue and body mucus of rainbow trout, *Salmo gairdneri*, at the onset of episodic exposure. J Fish Biol. 34(6):865-874.

Haridasan M. 2008. Nutritional adaptations of native plants of the cerrado biome in acid soils. Braz J Plant Physiol. 20(3):183-195.

Havas M. 1985. Aluminum bioaccumulation and toxicity to *Daphnia magna* in soft water at low pH. Can J Fish Aquat Sci. 42(11):1741-1748.

Haynes WM, editor. 2016. CRC handbook of chemistry and physics. 97th ed. Boca Raton (FL): CRC Press.

Hays SM, Aylward LL, LaKind JS, Bartels MJ, Barton HA, Boogaard PJ, Brunk C, DiZio S, Dourson M, Goldstein DA, et al. 2008. Guidelines for the derivation of Biomonitoring Equivalents: report from the Biomonitoring Equivalents Expert Workshop. Regul Toxicol Pharmacol. 51(3 Suppl):S4-S15.

Hays SM, Macey K, Poddalgoda D, Lu M, Nong A, Aylward L. 2016. Biomonitoring Equivalents for molybdenum. Regul Toxicol Pharmacol. 77:223-229.

He H, He L, Gu M. 2012. Interactions between nitric oxide and plant hormones in aluminum tolerance. Plant Signal Behav. 7(4):469-471.

Health Canada. 1998. Exposure factors for assessing total daily intake of priority substances by the general population of Canada. Unpublished report. Ottawa (ON): Government of Canada.

Health Canada. 2010. PMRA list of formulants [PDF]. Ottawa (ON): Government of Canada. [accessed 2018 Aug 20]. HC Pub. No.: 100460, Cat. No.: H114-22/2010E.

Health Canada. 2013. Second report on human biomonitoring of environmental chemicals in Canada. Results of the Canadian Health Measures Survey Cycle 2 (2009-2011). Ottawa (ON): Health Canada. [accessed 2019 Nov 23].

Health Canada. [modified 2015 Mar 18]. The safe use of cookware. Ottawa (ON): Government of Canada. [accessed 2020 Feb 18].

Health Canada. [modified 2016 Dec 9]. Science approach document. Biomonitoring-based approach 2 for barium-containing substances, molybdenum-containing substances, silver-containing substances,

thallium-containing substances and inorganic tin-containing substances. Ottawa (ON): Health Canada. [accessed 2019 Sep 3].

Health Canada. [modified 2017 Aug 10]. Results of the prioritization of the Revised In Commerce List. Ottawa (ON): Government of Canada. [accessed 2019 Apr 9].

Health Canada. 2018. Draft backgrounder document on default values for breast milk and formula intakes. Unpublished report. Ottawa (ON): Government of Canada.

Health Canada. 2020. Personal care products workbook [recommended defaults]. Internal draft. Unpublished report. Ottawa (ON): Health Canada, Existing Substances Risk Assessment Bureau. [updated 2020 Oct 19].

Health Canada. 2021a. Guidelines for Canadian drinking water quality: Guideline technical document – Aluminum. Ottawa (ON): Government of Canada. [accessed 2021 Apr 19].

Health Canada. [modified 2021b Dec 21]. The Canadian Health Measures Survey. Ottawa (ON): Health Canada. [accessed 2022 Oct 14].

Health Canada. [modified 2022a Aug 26]. Cosmetic Ingredient Hotlist: List of ingredients that are prohibited for use in cosmetic products. Ottawa (ON): Government of Canada. [accessed 2022 Oct 14].

Health Canada. [modified 2022b]. Concentration of contaminants and other chemicals in food composites: Trace elements. Canadian Total Diet Study. Ottawa (ON): Health Canada, Health Products and Food Branch. [accessed 2022 Oct 14].

Health Canada. [modified 2022c Feb 11]. Canadian exposure factors used in human health risk assessments. Ottawa (ON): Government of Canada. [accessed 2022 Oct 14].

Health Canada. [modified 2023 Jul 13]. Proposed updates to the Cosmetic Ingredient Hotlist: Prohibited and restricted ingredients. Ottawa (ON): Health Canada. [accessed 2023 Jul 14].

Health Council of the Netherlands. 2010. Aluminium and aluminium compounds: Health-based recommended occupational exposure limit [PDF]. The Hague (NL): Health Council of the Netherlands. [accessed 2019 Dec 14].

Heimburger A, Losno R, Triquet S, Dulac F, Mahowald N. 2012. Direct measurements of atmospheric iron, cobalt, and aluminum-derived dust deposition at Kerguelen Islands. Global Biogeochem Cycles. 26(4):GB4016.

Heitland P, Köster HD. 2021. Human biomonitoring of 73 elements in blood, serum, erythrocytes and urine. J Trace Elem Med Biol. 64:126706.

Helmboldt O, Hudson LK, Misra C, Wefers K, Heck W, Stark H, Danner M, Rösch N. 2012. Aluminum compounds, inorganic. In: Ullmann's encyclopedia of industrial chemistry. 7th ed., vol. 2. Weinheim (DE): Wiley-VCH. p. 569-584.

Herrmann J, Andersson KG. 1986. Aluminium impact on respiration of lotic mayflies at low pH. Water Air Soil Pollut. 30:703-709.

Hoet P, Jacquerye C, Deumer G, Lison D, Haufroid V. 2021. Reference values of trace elements in blood and/or plasma in adults living in Belgium. Clin Chem Lab Med. 59(4):729-742.

Hosovski E, Matelica Z, Sunderić D, Radulović D. 1990. Mental abilities of workers exposed to aluminium. Med Lav. 81(2):119-123.

Household Products Database [database]. 1993- . Bethesda (MD): US National Library of Medicine. [accessed 2019 Aug 20].

Hoyt PB, Nyborg M. 1972. Use of dilute calcium chloride for the extraction of plant-available aluminum and manganese from acid soil. Can J Soil Sci. 52(2):163-167.

Hoyt PB, Nyborg M. 1987. Field calibration of liming responses of four crops using soil pH, Al and Mn. Plant Soil. 102:21-25.

Hoyt PB, Nyborg M, Penney DC. 1974. Farming acid soils in Alberta and northeastern British Columbia. Ottawa (ON): Agriculture Canada. Publication 1521. 14 p.

Hudson LK, Misra C, Perrotta AJ, Wefers K, Williams FS. 2012. Aluminum oxide. In: Ullmann's encyclopedia of industrial chemistry. 7th ed., vol. 2. Weinheim (DE): Wiley-VCH. p. 607-645.

Hunter D, Milton R, Perry KMA, Thompson DR. 1944. Effect of aluminium and alumina on the lung in grinders of duralumin aeroplane propellers. Br J Ind Med. 1(3):159-164.

[IARC] IARC Working Group on Evaluation of Carcinogenic Risks to Humans. 1984. Polynuclear aromatic compounds, part 3: Industrial exposures in aluminium production coal gasification, coke production, and iron and steel founding. IARC Monogr Eval Carcinog Risks Hum. 34:1-219.

[IARC] IARC Working Group on Evaluation of Carcinogenic Risks to Humans. 2010. Occupational exposures during aluminium production. IARC Monogr Eval Carcinog Risks Hum. 100F:215-223.

[ICSC] International Chemical Safety Cards [database]. c1996-2018. Geneva (CH): International Labour Organization. [accessed 2022 Oct 14].

Jaiswal SK, Naamala J, Dakora FD. 2018. Nature and mechanisms of aluminium toxicity, tolerance and amelioration in symbiotic legumes and rhizobia. Biol Fertil Soils. 54(3):309-318.

Jansen S, Broadley MR, Robbrecht E, Smets E. 2002. Aluminum hyperaccumulation in angiosperms: A review of its phylogenetic significance. Bot Rev. 68:235-269.

Jardine PM, Zelazny LW. 1996. Surface reactions of aqueous aluminum species. In: Sposito G, editor. The environmental chemistry of aluminum. 2nd ed. Boca Raton (FL): CRC Press. p. 221-270.

Jayawardene I, Paradis J-F, Bélisle S, Poddalgoda D, Macey K. 2021. Multi-elemental determination of metals, metalloids and rare earth element concentrations in whole blood from the Canadian Health Measures Survey, 2009-2011. J Trace Elem Med Biol. 68:126830.

[JECFA] Joint FAO/WHO Expert Committee on Food Additives. 2007. Evaluation of certain food additives and contaminants [PDF]. Geneva (CH): World Health Organization. [accessed 2019 Jan 6]. (WHO Technical Report Series 940). Sixty-seventh report of the JECFA.

[JECFA] Joint FAO/WHO Expert Committee on Food Additives. 2012. Safety evaluation of certain food additives [PDF]. Geneva (CH): World Health Organization, International Programme on Chemical Safety. [accessed 2020 Jan 3]. (WHO Food Additive Series 65). Prepared by the seventy-fourth meeting of the

JECFA. On the cover: First draft prepared by Benford DJ, Agudo A, Baskaran C, DiNovi M, Folmer D, Leblanc J-C, Renwick AG.

Keith LS, Jones DE, Chou CHSJ. 2002. Aluminium toxicokinetics regarding infant diet and vaccinations. Vaccine. 20(Suppl. 3):S13-7.

Kidd PS, Proctor J. 2001. Why plants grow poorly on very acid soils: Are ecologists missing the obvious? J Exp Bot. 52(357):791-799.

Kilgour & Associates Ltd. 2016. Estimating background concentration ranges data to inform ecological chemical risk assessments. Unpublished report. Gatineau (QC): Environment and Climate Change Canada, Ecological Assessment Division.

Kim H-J, Lim H-S, Lee K-R, Choi M-H, Kang NM, Lee CH, Oh E-J, Park H-K. 2017. Determination of trace metal levels in the general population of Korea. Int J Environ Res Public Health. 14(7):702.

Kinraide TB. 1990. Assessing the rhizotoxicity of the aluminate ion, $Al(OH)_4^-$. Plant Physiol. 93(4):1620-1625.

Kinraide TB. 1997. Reconsidering the rhizotoxicity of hydroxyl, sulphate, and fluoride complexes of aluminium. J Exp Bot. 48(5):1115-1124.

Klöppel H, Fliedner A, Kördel W. 1997. Behaviour and ecotoxicology of aluminium in soil and water - Review of scientific literature. Chemosphere. 35(1-2):353-363.

Klotz K, Weistenhöfer W, Neff F, Hartwig A, van Thriel C, Drexler H. 2017. The health effects of aluminium exposure. Dtsch Arztebl Int. 114(39):653-659.

Klotz K, Meyer-Baron M, van Thriel C, Pallapies D, Nasterlack M, Letzel S, Roßbach B, Triebig G, Weistenhöfer W, Drexler H, et al. 2019. Addendum to Aluminium [BAT value documentation, 2019]. The MAK Collection for Occupational Health and Safety. 4(4):2279-2311.

Kluczka J, Zołotajkin W, Ciba J, Staroń M. 2017. Assessment of aluminum bioavailability in alum sludge for agricultural utilization. Environ Monit Assess. 189:422.

Kochian LV, Hoekenga OA, Pineros MA. 2004. How do crop plants tolerate acid soils? Mechanisms of aluminum tolerance and phosphorous efficiency. Annual Rev Plant Biol. 55:459-493.

Komarova T, McKeating D, Perkins AV, Tinggi U. 2021. Trace element analysis in whole blood and plasma for reference levels in a selected Queensland population, Australia. Int J Environ Res Public Health. 18(5):2652.

Kopittke PM, Moore KL, Lombi E, Gianoncelli A, Ferguson BJ, Blamey FPC, Menzies NW, Nicholson TM, McKenna BA, Wang P, et al. 2015. Identification of the primary lesion of toxic aluminum in plant roots. Plant Physiol. 167(4):1402-1411.

Kopittke PM, Blamey FPC. 2016. Theoretical and experimental assessment of nutrient solution composition in short-term studies of aluminium rhizotoxicity. Plant Soil. 406:311-326.

Kraus T, Schaller KH, Angerer J, Hilgers R-D, Letzel S. 2006. Aluminosis - Detection of an almost forgotten disease with HRCT. J Occup Med Toxicol. 1:4.

Krause MJ, Orlandi F, Saurage AT, Zietz JR Jr. 2012. Aluminum compounds, organic. In: Ullmann's encyclopedia of industrial chemistry. 7th ed., vol. 2. Weinheim (DE): Wiley-VCH. p. 586-605.

Krewski D, Yokel RA, Nieboer E, Borchelt D, Cohen J, Harry J, Kacew S, Lindsay J, Mahfouz AM, Rondeau V. 2007. Human health risk assessment for aluminium, aluminium oxide, and aluminium hydroxide. J Toxicol Environ Health B Crit Rev. 10(Suppl 1):1-269.

Krstic D, Djalovic I, Nikezic D, Bjelic D. 2012. Aluminium in acid soils: Chemistry, toxicity and impact on maize plants. In: Aladjadjiyan A, editor. Food production - Approaches, challenges and tasks. Rijeka (HR): InTech. p. 231-242.

Kiesswetter E, Schaeper M, Buchta M, Schaller KH, Rossbach B, Scherhag H, Zschiesche W, Letzel S. 2007. Longitudinal study on potential neurotoxic effects of aluminium: I. Assessment of exposure and neurobehavioural performance of Al welders in the train and truck construction industry over 4 years. Int Arch Occup Environ Health. 81(1):41-67.

Landman MJ, Ling N. 2011. Fish health changes in Lake Okaro, New Zealand: Effects of nutrient remediation, season or eutrophication? Hydrobiologia. 661:65-79.

Larter NC, Macdonald CR, Elkin BT, Wang X, Harms NJ, Gamberg M, Muir DCG. 2016. Cadmium and other elements in tissues from four ungulate species from the Mackenzie Mountain region of the Northwest Territories, Canada. Ecotoxicol Environ Saf. 132:9-17.

Lewis RJ, editor. 1997. Hawley's condensed chemical dictionary. 13th ed. New York (NY): John Wiley and Sons, Inc.

[LNHPD] Licensed Natural Health Products Database [database]. [modified 2022 Feb 12]. Ottawa (ON): Government of Canada. [accessed 2022 Oct 14].

Lin S, Evans RL, Schnepper D, Hill T. 1984. Evaluation of wastes from the East St. Louis water treatment plant and their impact on the Mississippi River. Springfield (IL): Department of Energy and Natural Resources, (ISWS/CIR-160/84:1-89, 1984).

Lin SD. 1989. No adverse environmental impacts from water plant discharges. Water Eng Manag. 136:40-41.

Lindemann J, Holtkamp E, Herrmann R. 1990. The impact of aluminium on green algae isolated from two hydrochemically different headwater streams, Bavaria, Germany. Environ Pollut. 67(1):61-77.

Liu X, Rua D, Wokovich A, Guo C, Keire D. 2019. Particle size distribution analysis of OTC aerosol or powder drug products with potential for inadvertent inhalation exposure to consumers. J Pharma Sci. 108(4):1506-1511.

Lukiw WJ, Kruck TPA, Percy ME, Pogue AI, Alexandrov PN, Walsh WJ, Sharfman NM, Jaber VR, Zhao Y, Li W, et al. 2019. Aluminum in neurological disease – A 36 year multicenter study. J Alzheimers Dis Parkinsonism. 8(6):457.

Lydersen E. 1990. The solubility and hydrolysis of aqueous aluminium hydroxides in dilute fresh waters at different temperatures. Nord Hydrol. 21(3):195-204.

MAK. 2014. Aluminium, dusts containing aluminium as metal, aluminium oxide and aluminium hydroxide [MAK Value Documentation, 2007] [PDF]. The MAK Collection for Occupational Health and Safety.

Mannello F, Tonti GA, Medda V, Simone P, Darbre PD. 2011. Analysis of aluminium content and iron homeostasis in nipple aspirate fluids from healthy women and breast cancer-affected patients. J Appl Toxicol. 31(3):262-269.

Manoharan V, Loganathan P, Tillman RW, Parfitt RL. 2007. Interactive effects of soil acidity and fluoride on soil solution aluminium chemistry and barley (*Hordeum vulgare* L.) root growth. Environ Pollut. 145(3):778-786.

Martin RB. 1986. The chemistry of aluminum as related to biology and medicine. Clin Chem. 32(10):1797-1806.

McGeer JC, Brix KV, Skeaff JM, DeForest DK, Brigham SI, Adams WJ, Green A. 2003. Inverse relationship between bioconcentration factor and exposure concentration for metals: Implications for hazard assessment of metals in the aquatic environment. Environ Toxicol Chem. 22(5):1017-1037.

McGrath KG. 2003. An earlier age of breast cancer diagnosis related to more frequent use of antiperspirants/deodorants and underarm shaving. Eur J Cancer Prev. 12(6):479-485.

McKenzie RC, Nyborg M. 1984. Influence of subsoil acidity on root development and crop growth in soils of Alberta and northeastern British Columbia. Can J Soil Sci. 64(4):681-697.

McLaughlin AIG, Kazantzis G, King E, Teare D, Porter RJ, Owen R. 1962. Pulmonary fibrosis and encephalopathy associated with the inhalation of aluminium dust. Br J Ind Med. 19(4):253-263.

Miessler GL, Fischer PJ, Tarr DA. 2014. Inorganic Chemistry. 5th ed. Upper Saddle River (NJ): Pearson Education Inc.

Minoia C, Sabbioni E, Apostoli P, Pietra R, Pozzoli L, Gallorini M, Nicolaou G, Alessio L, Capodaglio E. 1990. Trace element reference values in tissues from inhabitants of the European community. I. A study of 46 elements in urine, blood and serum of Italian subjects. Sci Total Environ. 95:89-105.

Mirick DK, Davis S, Thomas DB. 2002. Antiperspirant use and the risk of breast cancer. J Natl Cancer Inst. 94(20):1578-1580.

Mitchell J, Manning GB, Molyneux M, Lane RE. 1961. Pulmonary fibrosis in workers exposed to finely powdered aluminium. Br J Ind Med. 18(1):10-20.

Mitkus RJ, King DB, Hess MA, Forshee RA, Walderhaug MO. 2011. Updated aluminium pharmacokinetics following infant exposures through diet and vaccination. Vaccine. 29(51):9538-9543.

Moir JL, Moot DJ. 2014. Medium-term soil pH and exchangeable aluminium response to liming at three high country locations. Proceedings of the New Zealand Grassland Association. 76:41-46.

Moir JL, Jordan P, Moot DJ, Lucas R. 2016. Phosphorus response and optimum pH ranges of twelve pasture legumes grown in an acid upland New Zealand soil under glasshouse conditions. J Soil Sci Plant Nutr. 16(2):438-460.

Moir JL, Moot DJ, Whitley AE, Black AD, Hendrie DL. 2018. Soil acidity and aluminium in South Island high and hill country: New data and future needs. Journal of New Zealand Grasslands. 80:145-148.

Morita H, Kita T, Umeno M, Morita M, Yoshinaga, J, Okamoto K, 1994. Analysis of serum elements and the contaminations from devices used for serum preparation by inductively coupled plasma mass spectrometry. Sci Total Environ. 151(1):9-17.

Moyer TP, Mussmann GV, Nixon DE. 1991. Blood-collection device for trace and ultra-trace metal specimens evaluated. Clin Chem. 37(5):709-714.

Movsas TZ, Paneth N, Rumbeiha W, Zyskowski J, Gewolb I H. 2013. Effect of routine vaccination on aluminum and essential element levels in preterm infants. JAMA Pediatr. 167(9):870-872.

[MSDS] Material Safety Data Sheet. 1999. Lambdaplast Violet P-2R [PDF]. Dover (OH): Buckeye Color & Chemical Company, LLC. [accessed 2019 Mar]. [restricted access].

Mulder J, van Breemen N, Eijck HC. 1989. Depletion of soil aluminium by acid deposition and implications for acid neutralization. Nature. 337:247-249.

Musk AW, de Klerk NH, Beach JR, Fritschi L, Sim MR, Benke G, Abramson M, McNeil JJ. 2000. Respiratory symptoms and lung function in alumina refinery employees. Occup Environ Med. 57(4):279-283.

Namer M, Luporsi E, Gligorov J, Lokiec F, Spielmann M. 2008.The use of deodorants/antiperspirants does not constitute a risk factor for breast cancer. Bull Cancer. 95(9):871-880.

[NAPS] National Air Pollution Surveillance Network. 2015. Monitoring data for 2015. Unpublished data. Ottawa (ON): Environment Canada, Analysis and Air Quality Division.

[NAPS] National Air Pollution Surveillance Network. [modified 2022 Nov 13]. National Air Pollution Surveillance (NAPS) Program - ECCC Data Mart: Integrated $PM_{2.5}$ data sets (2010 to 2018). Ottawa (ON): Government of Canada. [accessed 2023 Jan 26].

Narf RP. 1990. Interactions of Chironomidae and Chaoboridae (Diptera) with aluminum sulfate treated lake sediments. Lake Reservoir Manag. 6(1):33-42.

Nazarenko Y, Zhen H, Han T, Lioy PJ, Mainelis G. 2012. Potential for inhalation exposure to engineered nanoparticles from nanotechnology-based cosmetic powders. Environ Health Perspect. 120(6):885-892.

[NCASI] National Council for Air Stream Improvement, Inc. 2020. Aluminum in Canadian pulp & paper mill effluents and ambient waters. Unpublished report and data tables submitted to Environment and Climate Change Canada.

[NIOSH] National Institute for Occupational Safety and Health. 2018. NIOSH pocket guide to chemical hazards: Sodium aluminum fluoride (as F). Centers for Disease Control and Prevention. [accessed 2019 Mar].

[NLTWQM] National Long-term Water Quality Monitoring. [modified 2022 Jul 17]. Newfoundland and Labrador long-term water quality monitoring data. Ottawa (ON): Government of Canada. [accessed 2022 Oct 14].

[NPRI] National Pollutant Release Inventory. [modified 2019 Nov 28]. NPRI sector overview: Aluminium. Ottawa (ON): Government of Canada. [accessed 2020 Feb].

[NPRI] National Pollutant Release Inventory. [modified 2022 March 2]. Bulk data set (1993 to present); Release data for aluminum (fume or dust only), 7429-90-5 and aluminum oxide (fibrous forms only), 1344-28-1. Ottawa (ON): Government of Canada. [accessed 2022 Oct 14].

[NHPID] Natural Health Products Ingredients Database [database]. [modified 2022 Aug 18]. Ottawa (ON): Government of Canada. [accessed 2022 Oct 14].

[NRCan] Natural Resources Canada. 2016. Minerals and metals fact book – 2016 [PDF]. Cat. No. M31-16E-PDF. 124 p.

[NRCan] Natural Resources Canada. [modified 2022 Feb 22]. Aluminum facts. Ottawa (ON): Natural Resources Canada. [accessed 2022 Oct 14].

Nilsson SI, Bergkvist B. 1983. Aluminium chemistry and acidification processes in a shallow podzol on the Swedish west coast. Water Soil Air Pollut. 20:311-329.

Nilsson R. 1988. Aluminium in natural waters – Toxicity to fish and man. In: Astruc M, Lester JN, editors. Heavy metals in the hydrological cycle. London (GB): Selper Ltd. p. 11-18.

Nisse C, Tagne-Fotso R, Howsam M, Richeval C, Labat L, Leroyer A. 2017. Blood and urinary levels of metals and metalloids in the general adult population of Northern France: The IMEPOGE study, 2008–2010. Int J Hyg Environ Health. 220(2, Pt B):341-363.

Nogueirol RC, Monteiro FA, Azevedo RA. 2015. Tropical soils cultivated with tomato: Fractionation and speciation of Al. Environ Monit Assess. 187(4):160.

Novak JT, Knocke WR, Geertsema W, Dove D, Taylor A, Mutter R. 1995. An assessment of cropland application of water treatment residuals. Denver (CO): American Water Works Association Research Foundation. 71 p.

Nriagu JO, Wong HKT. 1986. What fraction of the total metal flux into lakes is retained in the sediments? Water Soil Air Pollut. 31:999-1006.

O'Neil MJ, Smith A, Heckelman PE, Obenchain JR, Gallipeau JR, D'Arecca MA, editors. 2001. The Merck index. An encyclopedia of chemicals, drugs, and biologicals. Whitehouse Station (NJ): Merck & Co., Inc.

Oladeji OO, Sartain JB, O'Connor GA. 2006. Agronomic impact of water treatment residual co-applied with phosphorus sources to Florida sands. Soil Crop Sci Soc Florida Proc. 65:38-48.

Omran GA. 2019. Hematological and immunological impairment following *in-utero* and postnatal exposure to aluminum sulfate in female offspring of albino rats. Immunopharmacol Immunotoxicol. 41(1):40-47.

OMAFRA [Ontario Ministry of Agriculture, Food and Rural Affairs]. 2018. Soil fertility handbook. Munroe J, editor. Publication 611, 3rd ed. Toronto (ON): OMAFRA, Queen's Printer for Ontario. 236 p.

Ontario. 1990a. *Environmental Protection Act*: O. Reg. 561/94 *Effluent Monitoring and Effluent Limits – Industrial Minerals Sector*. R.S.O. 1990, c. E. 19.

Ontario. 1990b. *Environmental Protection Act*: O. Reg. 215/95 *Effluent Monitoring and Effluent Limits – Electric Power Generation Sector*. R.S.O. 1990, c. E. 19.

Ontario. 2019. Data Catalogue: Industrial wastewater discharges. Toronto (ON): Government of Ontario, Ministry of Environment, Conservation and Parks. [accessed 2022 Oct 14].

Ontario. [modified 2021 Aug 23]. The end of coal. (ON): Ministry of Energy, Northern Development and Mines. Government of Ontario. [accessed 2022 Oct 14].

[OMEE] Ontario Ministry of Environment and Energy, [OMAFRA] Ontario Ministry of Agriculture, Food, and Rural Affairs. 1996. Guidelines for the utilization of biosolids and other wastes on agricultural land. Ontario Ministry of Environment and Energy, Ontario Ministry of Agriculture, Food and Rural Affairs, March 1996.

[OECD] Organisation for Economic Co-operation and Development. 2014. guidance on grouping of chemicals, second edition. Paris (FR): OECD. 141 p. (OECD Series on Testing and Assessment; no. 194).

[OECD] Organisation for Economic Co-operation and Development. 2015. Guidance on selecting a strategy for assessing the ecological risk of organometallic and organic metal salt substances based on their environmental fate. Paris (FR): OECD. (OECD Series on Testing and Assessment; no. 212). 55 p.

Parent L, Campbell PGC. 1994. Aluminum bioavailability to the green alga *Chlorella pyrenoidosa* in acidified synthetic soft water. Environ Toxicol Chem. 13(4):587-598.

Parent L, Twiss MR, Campbell PGC. 1996. Influences of natural dissolved organic matter on the interaction of aluminum with the microalga *Chlorella*: A test of the free-ion model of trace metal toxicity. Environ Sci Technol. 30(5):1713-1720.

Parker DR, Zelazny LW, Kinraide TB. 1989. On the phytotoxicity of polynuclear hydroxy-aluminum complexes. Soil Sci Soc Am J. 53(3):789-796.

Peters S, Reid A, Fritschi L, de Klerk N, Musk AWB. 2013. Long-term effects of aluminium dust inhalation. Occup Environ Med. 70(12):864-868.

[PhysProp] Interactive PhysProp Database [database]. c2018. Syracuse (NY): SRC, Inc. [accessed 2018 Nov].

Pigott GH, Gaskell BA, Ishmael J. 1981. Effects of long term inhalation of alumina fibres in rats. Br J Exp Pathol. 62(3):323-331.

Playle RC, Wood CM. 1989. Water chemistry changes in the gill micro-environment of rainbow trout: Experimental observations and theory. J Comp Physiol B. 159:527-537.

Poddalgoda D, Hays SM, Kirman C, Chander N, Nong A. 2021. Derivation of Biomonitoring Equivalents for aluminium for the interpretation of population-level biomonitoring data. Regul Toxicol Pharmacol. 122:104913.

Poirier J, Semple H, Davies J, Lapointe R, Dziwenka M, Hiltz M, Mujibi D. 2011. Double-blind, vehicle-controlled randomized twelve-month neurodevelopmental toxicity study of common aluminum salts in the rat. Neuroscience. 193:338-362.

Poléo ABS. 1995. Aluminium polymerization – A mechanism of acute toxicity of aqueous aluminium to fish. Aquat Toxicol. 31(4):347-356.

Poléo ABS, Lydersen E, Rosseland BO, Kroglund F, Salbu B, Vogt RD, Kvellestad A. 1994. Increased mortality of fish due to changing Al-chemistry of mixing zones between limed streams and acidic tributaries. Water Air Soil Pollut. 75:339-351.

Polizzi S, Pira E, Ferrara M, Bugiani M, Papaleo A, Albera R, Palmi S. 2002. Neurotoxic effects of aluminium among foundry workers and Alzheimer's disease. Neurotoxicology 23(6):761-774.

[PCA] Portland Cement Association. 2006. Life cycle inventory of Portland Cement manufacture [PDF]. Marceau ML, Nisbet MA, VanGeem MG, PCA R&D Serial No. 2095b. Illinois (US): Portland Cement Association. 69 p.

Priest ND. 2004. The biological behaviour and bioavailability of aluminium in man, with special reference to studies employing aluminium-26 as a tracer: Review and study update. J Environ Monit. 6(5):375-403.

Priest ND, Newton D, Day JP, Talbot RJ, Warner AJ. 1995. Human metabolism of aluminium-26 and gallium-67 injected as citrates. Hum Exp Toxicol. 14(3):287-293.

Proctor J. 1999. Heath forests and acid soils. Bot J Scotl. 51(1):1-14.

Proulx CL, Kilgour BW, Francis AP, Bouwhuis RF, Hill JR. 2018. Using a conductivity-alkalinity relationship as a tool to identify surface waters in reference condition across Canada. Water Qual Res J Can. 53(4):231-240.

[PWQMN] Provincial Water Quality Monitoring Network. [modified 2020 Nov 30]. Annual datasets for 2005 to 2012. Ottawa (ON): Queen's Printer for Ontario. [accessed 2022 Oct 14].

Rasmussen PE, Levesque C, Chénier M, Gardner HD. 2018. Contribution of metals in resuspended dust to indoor and personal inhalation exposures: Relationships between PM10 and settled dust. Build Environ. 143:513-522.

Rasmussen P, Levesque C, Niu J, Gardner HD, Nilsson G, Macey K. 2019. Characterization of airborne particles emitted during application of cosmetic talc products. Int J Environ Res Public Health. 16(20):3830.

Ratelle M, Skinner K, Brandow D, Packull-McCormick S, Laird, B. 2019. Results report. Contaminant biomonitoring in the Northwest Territories Mackenzie Valley: Investigating the links between contaminant exposure, nutritional status, and country food use [PDF]. Waterloo (ON): University of Waterloo.

Ratelle M, Skinner K, Packull-McCormick S, Laird, B. 2020a. Food frequency questionnaire assessing traditional food consumption in Dene/Métis communities, Northwest Territories, Canada. Int J Circumpolar Health. 79(1):1760071.

Ratelle M, Packull-McCormick S, Bouchard M, Majowicz S, Laird B. 2020b. Human biomonitoring of metals in sub-Arctic Dene communities of the Northwest Territories, Canada. Environ Res. 190:110008.

Razo-Estrada AC, García-Medina S, Madrigal-Bujaidar E, Gómez-Oliván LM, Galar-Martínez M. 2013. Aluminum-induced oxidative stress and apoptosis in liver of the Common Carp, *Cyprinus carpio*. Water Air Soil Pollut. 224(5):1510.

[RAMP] Regional Aquatics Monitoring Program [database]. 2016. Regional Aquatics Monitoring Program water quality data. All parameters under conventional variables, dissolved metals, extractable metals, field, major ions, and total metals for 2005 to 2015. Regional Aquatics Monitoring Program. [accessed 2022 Oct 14].

Reitzel K, Jensen HS, Egemose S. 2013. pH dependent dissolution of sediment aluminum in six Danish lakes treated with aluminum. Water Res. 47(3):1409-1420.

Rice WA. 1975. Effects of $CaCO_3$ and inoculum level on nodulation and growth of alfalfa in an acid soil. Can J Soil Sci. 55(3):245-250.

Riihimäki V, Hänninen H, Akila R, Kovala T, Kuosma E, Paakkulainen H, Valkonen S, Engström B. 2000. Body burden of aluminium in relation to central nervous system function among metal inert-gas welders. Scand J Work Environ Health. 26(2):118-130.

Riihimäki V, Aitio A. 2012. Occupational exposure to aluminum and its biomonitoring in perspective. Crit Rev Toxicol. 42(10):827-853.

[RIVM] Rijksinstituut voor Volksgezondheid en Milieu [National Institute for Public Health and the Environment (NL)]. 2007. Do-it-yourself products fact sheet. Bilthoven (NL): RIVM. Report No.: 320104007/2007.

[RIVM] Rijksinstituut voor Volksgezondheid en Milieu [National Institute for Public Health and the Environment (NL)]. 2020. Human health risk assessment of aluminium. Bilthoven (NL): RIVM. Report No. 2020-0001.

Rodushkin I, Ödman F, Branth S. 1999. Multi-element analysis of whole blood by high resolution inductively coupled plasma mass spectrometry. Fresenius' J Anal Chem. 364(4):338-346.

Rodushkin I, Ödman F. 2001. Assessment of the contamination from devices used for sampling and storage of whole blood and serum for element analysis. J Trace Elem Med Biol. 15(1):40-45.

Rothhaar U, Klause M, Hladik B. 2016. Comparative delamination study to demonstrate the impact of container quality and nature of buffer system. PDA J Pharm Sci Technol. 70(6):560-567.

Rotteveel L, Stirling SM. 2020. Five aluminum seasonality regimes identified in chronically acidified rivers of Nova Scotia. Environ Sci Technol. 54(2):807-817.

Rout G, Samantaray S, Das P. 2001. Aluminium toxicity in plants: A review. Agronomie. 21(1):3-21.

Roy R. 1999. The chemistry, bioaccumulation and toxicity of aluminum in the aquatic environment for the PSL2 assessment of aluminum salts. Report prepared by Fisheries and Oceans Canada for Environment Canada. Unpublished report. Montreal (QC): Environment Canada. 110 p.

Roy RL, Campbell PGC. 1997. Decreased toxicity of Al to juvenile Atlantic salmon (*Salmo salar*) in acidic soft water containing natural organic matter: A test of the free-ion model. Environ Toxicol Chem. 16(9):1962-1969.

Rundgren S, Nilsson P. 1997. Sublethal effects of aluminium on earthworms in acid soil: The usefulness of *Dendrodrilus rubidus* (Sav.) in a laboratory test system. Pedobiologia. 41(5):417-436.

Ryan PR, Kinraide TB, Kochian LV. 1994. Al^{3+}-Ca^{2+} interactions in aluminum rhizotoxicity: I. Inhibition of root growth is not caused by reduction of calcium uptake. Planta. 192(1):98-103.

Salib E, Hillier V. 1996. A case-control study of Alzheimer's disease and aluminium occupation. Br J Psychiatry. 168(2):244-249.

Santos C, Silva S, Pinto-Carnide O. 2014. Aluminum phytotoxicity: Physiological approaches and tolerance. In: Fishbein JC, Heilman JM, editors. Advances in molecular toxicology, vol. 8. Amsterdam (NL): Elsevier BV. p. 203-236.

Saskatchewan Ministry of Health. 2019. Northern Saskatchewan prenatal biomonitoring study technical report [PDF]. Regina (SK): Saskatchewan Health Authority. [accessed 2021 Sep 12].

Schindler DW, Hesslein RH, Wagemann R, Broecker WS. 1980. Effects of acidification on mobilization of heavy metals and radionuclides from the sediments of a freshwater lake. Can J Fish Aquat Sci. 37(3):373-377.

Schwartz H, Marushka L, Chan HM, Batal M, Sadik T, Ing A, Fediuk K, Tikhonov C. 2021. Metals in the drinking water of First Nations across Canada. Can J Public Health. 112(Suppl 1):113-132.

Schwarz K, Pappa G, Miertsch H, Scheel J, Koch W. 2018. A methodology for the assessment of inhalation exposure to aluminium from antiperspirant sprays. Arch Toxicol. 92(4):1383-1392.

[SCCS] Scientific Committee on Consumer Safety. 2019. Opinion on the safety of aluminium in cosmetic products, preliminary version. SCCS/1613/19. [accessed 2020 Jan 30].

[SCCS] Scientific Committee on Consumer Safety. 2020. Opinion on the safety of aluminium in cosmetic products, final version [PDF]. SCCS/1613/19 Final Opinion. [accessed 2020 Apr 29].

[SDS] Safety Data Sheet. 2015. Portland Cement [PDF]. Irving (TX): Lehigh Hanson [accessed 2019 Feb]. [restricted access].

[SDS] Safety Data Sheet. 2016. Fillite [PDF]. Madrid (ES): Tolsa Group. [accessed 2019 Mar]. [restricted access].

[SDS] Safety Data Sheet. 2017a. Hycite 713 [PDF]. Mississauga (ON): BASF Canada Inc. [accessed 2019 Mar]. [restricted access].

[SDS] Safety Data Sheet. 2017b. Pass 10 Polyaluminum Silicate Sulphate [PDF]. Richmond (BC): Univar Canada Ltd. [accessed 2019 Mar]. [restricted access].

[SDS] Safety Data Sheet. 2018a. EADC [PDF]. Middlebury (CT): LANXESS Solutions US Inc. [accessed 2019 Mar]. [restricted access].

[SDS] Safety Data Sheet. 2018b. Sodium aluminum sulfate [PDF]. Avon (NY): Aldon Corporation. [accessed 2019 Mar]. [restricted access].

[SDS] Safety Data Sheet. 2018c. Bis(2-ethylhexanoata)hydroxyaluminum [PDF]. Portland (OR): TCI America. [accessed 2019 Mar]. [restricted access].

[SDS] Safety Data Sheet. 2018d. MPT-UB-431810 [PDF]. Dayville (CT): Miyoshi America Inc. [accessed 2019 Mar]. [restricted access].

Sharp CA, Perks J, Worsfold M, Day JP, Davie MW. 1993. Plasma aluminium in a reference population: the effects of antacid consumption and its influence on biochemical indices of bone formation. Eur J Clin Invest. 23:554-560.

Shiller AM, Frilot DM. 1996. The geochemistry of gallium relative to aluminum in Californian streams. Geochim Cosmochim Acta. 60(8):1323-1328.

Shuman LM. 1990. Comparison of exchangeable Al, extractable Al, and Al in soil fractions. Canadian J Soil Sci. 70(3):263-275.

Singleton PL, Edmeades DC, Smart RE, Wheeler DM. 1987. Soil acidity and aluminium and manganese toxicity in the Te Kauwhata area, North Island, New Zealand. New Zealand J Agric Res. 30(4):517-522.

Sjögren B, Iregren A, Frech A. 1996. Effects on the nervous system among welders exposed to aluminum and manganese. Occup Environ Med. 53:32-40.

Sjögren B, Iregren A, Montelius J, Yokel RA. 2015. Chapter 26: Aluminum. In: Nordberg GF, Fowler BA, Nordberg M, editors. Handbook on the Toxicology of Metals. 4th ed., vol. II. Cambridge (MA): Academic Press. p. 549-564.

Skei JK, Dolmen D. 2006. Effects of pH, aluminium, and soft water on larvae of the amphibians *Bufo bufo* and *Triturus vulgaris*. Can J Zool. 84:1668-1677.

Sleppy WC. 2007. Aluminum compounds, survey. In: Seidel, A, Editor. Kirk-Othmer Encyclopedia of Chemical Technology, Vol. 2, 5th Ed. Hoboken (NJ): Wiley-Interscience. p. 344-360.

Smeltzer E. 1990. A successful alum/aluminate treatment of Lake Morey, Vermont. Lake and Reservoir Management 6:9-19.

Sparling DW, Lowe TP. 1996. Environmental hazards of aluminum to plants, invertebrates, fish, and wildlife. Rev Environ Contam Toxicol. 145:1-127.

Spry DJ, Wiener JG. 1991. Metal bioavailability and toxicity to fish in low alkalinity lakes: A critical review. Environ Pollut. 71:243-304.

Stanley JK, Coleman JG, Weiss CA Jr, Steevens JA. 2010. Sediment toxicity and bioaccumulation of nano and micron-sized aluminum oxide. Environ Toxicol Chem. 29(2):422-429.

Statistics Canada. 2004. Canadian Community Health Survey – Nutrition. Detailed information for 2004 (Cycle 2.2). Ottawa (ON): Government of Canada.

Steinhagen WH, Cavender FL. 1978. Six month inhalation exposures of rats and guinea pigs to aluminum chlorhydrate. J Environ Pathol Toxicol. 1:267-277.

Steinhausen C, Kislinger G, Winklhofer C, Beck E, Hohl C, Nolte E, Ittle TH, Alvarez-Brückmann MJL. 2004. Investigation of the aluminium biokinetics in humans: ^{26}Al tracer study. Food Chem Toxicol. 42(3):363-371.

Stone CJ, McLaurin DA, Steinhagen WH, Cavender FL, Haseman JK. 1979. Tissue deposition patterns after chronic inhalation exposures of rats and guinea pigs to aluminum chlorhydrate. Toxicol Appl Pharm. 49(1):71-76.

Stumm W, Morgan JJ. 1981. Aquatic chemistry: An introduction emphasizing chemical equilibria in natural waters. 2nd ed. New York (NY): John Wiley and Sons.

Swensson Å, Nordenfelt A, Forssman S, Lundgren K-D, Öhman H. 1962. Aluminium dust pneumoconiosis: A clinical study. Int Arch Gewerbepath Gewerbehyg. 19:131-148.

Talbot RJ, Newton D, Priest ND, Austin JG, Day JP. 1995. Inter-subject variability in the metabolism of aluminium following intravenous injection as citrate. Hum Exp Toxicol. 14(7):595-599.

[TDS] Technical Data Sheet. 2005. Alcloxa. Coriano (IT): Arkema Fine Chemicals srl. [accessed 2019 Mar].

[TDS] Technical Data Sheet. 2019. Aldioxa. Coriano (IT): Arkema Fine Chemicals srl. [accessed 2019 Mar].

Tejada M, Gómez I, Hernández T, García C. 2010. Response of *Eisenia fetida* to the application of different organic wastes in an aluminium-contaminated soil. Ecotoxicol Environ Saf. 73(8):1944-1949.

Thomson SM, Burnett DC, Bergmann JD, Hixson CJ. 1986. Comparative inhalation hazards of aluminum and brass powders using bronchopulmonary lavage as an indicator of lung damage. J Appl Toxicol. 6(3):197-209.

Tietz T, Lenzner A, Kolbaum AE, Zellmer S, Riebeling C, Gürtler R, Jung C, Kappenstein O, Tentschert J, Giulbudagian M, et al. 2019. Aggregated aluminium exposure: Risk assessment for the general population. Arch Toxicol. 93(12):3503-3521.

Troisi J, Giugliano L, Sarno L, Landolfi A, Richards S, Symes S, Colucci A, Maruotti G, Adair D, Guida M, et al. 2019. Serum metallome in pregnant women and the relationship with congenital malformations of the central nervous system: A case-control study. BMC Pregnancy Childbirth. 19(1):471.

Tsialtas I, Gorgogietas VA, Michalopoulou A, Komninou A, Liakou E, Georgantopoulos A, Kalousi FD, Karra AG, Protopapa E, Psarra A-MG. 2020. Neurotoxic effects of aluminum are associated with its interference with estrogen receptors signaling. Neurotoxicology. 77:114-126.

Tsou VM, Young RM, Hart MH, Vanderhoof JA. 1991. Elevated plasma aluminium levels in normal infants receiving antacids containing aluminium. Pediatrics. 87(2):148-151.

Tugulea 2016. A national survey of disinfection by-products and selected drinking water contaminants in Canadian drinking water (2009-2010). Unpublished database. Ottawa (ON): Health Canada, Exposure and Biomonitoring Division.

[US EPA] United States Environmental Protection Agency. 1995. AP 42, fifth edition compilation of air pollutant emissions factors, volume 1: Stationary point and area sources, Chapter 11: Mineral products industry, section 11.6: Portland cement manufacturing. 26 p.

[US EPA] United States Environmental Protection Agency. 2003. Ecological soil screening level for aluminum. Interim final. OSWER Directive 9285.7-60 Washington (DC): US EPA, Office of Solid Waste and Emergency Response (OSWER). OSWER Directive 9285.7-60.

[US EPA] United States Environmental Protection Agency. 2006. Provisional peer reviewed toxicity values for aluminum (CASRN 7429-90-5) [PDF]. Cincinnati (OH): US EPA, Office of Research and Development. [accessed 2019 Dec].

[US EPA] United States. Environmental Protection Agency. 2007. Framework for metals risk assessment. Washington (DC): US EPA, Office of the Science Advisor, Risk Assessment Forum. EPA/120/R-07/001.

[US EPA] United States Environmental Protection Agency. 2011. Exposure factors handbook 2011 edition (final report). Washington (DC): US EPA, National Center for Environmental Assessment, Office of Research and Development. EPA/600/R-09/052F.

[US EPA] United States Environmental Protection Agency. 2018. Final aquatic life ambient water quality criteria for aluminum. Washington (DC): US EPA, Office of Water, Office of Science and Technology. 329 p. EPA-822-R-18-001.

[USGS] United States Geological Service. 2015. 2015 Minerals yearbook – Canada [advance release] [PDF]. Barry JJ, U.S. Department of the Interior, US Geological Survey. 23 p.

van Gestel CAM, Hoogerwerf G. 2001. Influence of soil pH on the toxicity of aluminium for *Eisenia Andrei* (Oligochaete: Lumbricidae) in an artificial soil substrate. Pedobiologia. 45(5):385-395.

van Ryswyk K, Anastasopolos AT, Evans G, Sun L, Sabaliauskas K, Kulka R, Wallace L, Weichenthal S. 2017. Metro commuter exposures to particulate air pollution and PM2.5-associated elements in three Canadian cities: The urban transportation exposure study. Environ Sci Technol. 51(10):5713-5720.

Venter HJ. 2017. Soil acidity in the Kakanui ranges. In: Currie LD, Hedley MJ, editors. Science and policy: Nutrient management challenges for the next generation. Occasional report no. 30. Palmerston North (NZ): Massey University, Fertilizer and Lime Research Center. 8 p.

Veselý J, Majer V, Kopáček J, Norton SA. 2003. Increasing temperature decreases aluminum concentrations in Central European lakes recovering from acidification. Limnol Oceanogr. 48(6):2346-2354.

Violante A, Cozzolino V, Perelomov L, Caporale AG, Pigna M. 2010. Mobility and bioavailability of heavy metals and metalloids in soil environments. J Soil Sci Plant Nutr. 10(3):268-292.

Virk SA, Eslick GD. 2015. Occupational exposure to aluminum and Alzheimer disease: A meta-analysis. J Occup Environ Med. 57(8):893-896.

Walton JR, Wang M-X. 2009. APP expression distribution and accumulation are altered by aluminum in a rodent model for Alzheimer's disease. J Inorg Biochem. 103(11):1548-1554.

Wang Z, Wei X, Yang J, Suo J, Chen J, Liu C, Zhao X. 2016. Chronic exposure to aluminium and risk of Alzheimer's disease: A meta-analysis. Neurosci Lett. 610:200-206.

Wauer G, Teien H-C. 2010. Risk of acute toxicity for fish during aluminium application to hardwater lakes. Sci Total Environ. 408(19):4020-4025.

Webber MD, Hoyt PB, Nyborg N, Corneau D. 1977. A comparison of lime requirement methods for acid Canadian soils. Can J Soil Sci. 57(3):361-370.

Whitley AE, Moir JL, Almond PC, Moot DJ. 2016. Soil pH and exchangeable aluminium in contrasting New Zealand high and hill country soils. Hill Country – Grassland Research and Practice Series. 16:169-172.

Wilkinson KJ, Campbell PGC. 1993. Aluminum bioconcentration at the gill surface of juvenile Atlantic salmon in acidic media. Environ Toxicol Chem. 12(11):2083-2095.

Willhite CC, Karyakina NA, Yokel RA, Yenugadhati N, Wisniewski TM, Arnold IMF, Momoli F, Krewski D. 2014. Systematic review of potential health risks posed by pharmaceutical, occupational and consumer exposures to metallic and nanoscale aluminum, aluminum oxides, aluminum hydroxide and its soluble salts. Crit Rev Toxicol. 44(Suppl 4):1-80.

Wilson RW. 2012. Aluminum. In: Wood CM, Farrell AP, Brauner CJ, editors. Homeostasis and toxicology of non-essential metals. Volume 31B. Amsterdam (NL): Academic Press. p. 67-123.

Winter AR, Nichols JW, Playle RC. 2005. Influence of acidic to basic water pH and natural organic matter on aluminum accumulation by gills of rainbow trout (*Oncorhynchus mykiss*). Can J Fish Aquat Sci. 62(10):2303-2311.

Wong HKT, Nriagu JO, McCabe KJ. 1989. Aluminum species in porewaters of Kejimkujik and Mountain Lakes, Nova Scotia. Water Air Soil Pollut. 46:155-164.

[WBEA] Wood Buffalo Environmental Association. 2019. Annual report – Volume 2: 2018 Integrated data [PDF]. Fort McMurray (AB): Wood Buffalo Environmental Association. [accessed 2022 Aug 16].

[WBEA] Wood Buffalo Environmental Association. 2020. Annual report – Volume 2: 2019 Integrated data [PDF]. Fort McMurray (AB): Wood Buffalo Environmental Association. [accessed 2022 Aug 16].

Woodburn K, Walton R, McCrohan C, White K. 2011. Accumulation and toxicity of aluminium-contaminated food in the freshwater crayfish, *Pacifastacus leniusculus*. Aquat Toxicol. 105(3-4):535-542.

[WHO] World Health Organization. 2010. Aluminium in drinking-water - Background document for development of WHO guidelines for drinking-water quality [PDF]. Geneva (CH): World Health Organization. [accessed 2022 Aug 16].

Worth A, Patlewicz G. 2007, editors. A compendium of case studies that helped to shape the REACH Guidance on Chemical Categories and Read Across. IHCP Report No. EUR 22481 EN. Luxembourg: European Communities.

Wright RJ, Baligar VC, Ahlrichs JL. 1989. The influence of extractable and soil solution aluminum on root growth of wheat seedlings. Soil Sci. 148(4):293-302.

Yang JL, Zhu XF, Peng YX, Zheng C, Li GX, Liu Y, Shi YZ, Zheng SJ. 2011. Cell wall hemicellulose contributes significantly to aluminum adsorption and root growth in Arabidopsis. Plant Physiol. 155(4):1885-1892.

Yokel RA, McNamara PJ. 2001. Aluminium toxicokinetics: An updated mini review. Pharmacol Toxicol. 88(4):159-167.

Zamparas M, Zacharias I. 2014. Restoration of eutrophic freshwater by managing internal nutrient loads. A review. Sci Tot Environ. 496:551-562.

Zarnke A, Rasmussen PE, David M-O, Eidi H, Kennedy K, Hedges K, Irick T, Thome C, Pirkkanen J, Boreham D. 2019. Physical and chemical characterization of McIntyre Powder: An aluminum dust inhaled by miners to combat silicosis. J Occup Environ Hyg. 16(11):745-756.

Zawilla NH, Taha FM, Kishk NA, Farahat SA, Farghaly M, Hussein M. 2014. Occupational exposure to aluminium and its amyloidogenic link with cognitive functions. J Inorg Biochem 139:57-64.

Zhang J, Yu J, Ouyang Y, Xu H. 2013. Responses of earthworm to aluminum toxicity in latosol. Environ Sci Pollut Res. 20:1135-1141.

Zhao L, Qiu JP. 2010. Aluminum bioaccumulation in the earthworm and acute toxicity to the earthworm. In: 4th International Conference on Bioinformatics and Biomedical Engineering, ICBBE 2010. 18-20 June 2010, Chengdu, China. p. 1-4.

Appendix A. Substance identity information

Table A-1. Identity information for the Aluminium-containing Substances Group

CAS RN	List	DSL or R-ICL name	Common name
75-24-1	DSL	Aluminum, trimethyl-	Trimethylaluminum
96-10-6	DSL	Aluminum, chlorodiethyl-	Diethylaluminum chloride
97-93-8	DSL	Aluminum, triethyl-	Triethylaluminum
300-92-5[a]	DSL	Aluminum, hydroxybis(octadecanoato-O)-	Aluminum distearate
563-43-9	DSL	Aluminum, dichloroethyl-	Ethylaluminum dichloride
1070-00-4	DSL	Aluminum, trioctyl-	Trioctylaluminum
1116-73-0	DSL	Aluminum, trihexyl-	Trihexylaluminum
1302-42-7	DSL	Aluminate (AlO_2^{1-}), sodium	Sodium aluminate
1317-25-5	DSL	Aluminum, chloro[(2,5-dioxo-4-imidazolidinyl)ureato]tetrahydroxy di-	Alcloxa; Aluminum chlorhydroxy allantoinate
1327-41-9[b]	DSL	Aluminum chloride, basic	Aluminum hydroxychloride
1328-04-7[b]	DSL	C.I. Pigment Violet 5:1	NA
1344-28-1	DSL	Aluminum oxide (Al_2O_3)	NA
5579-81-7	DSL	Aluminum, [(2,5-dioxo-4-imidazolidinyl)ureato]dihydroxy-	Aldioxa; Aluminum dihydroxy allantoinate
7784-18-1	DSL	Aluminum fluoride (AlF_3)	NA
7784-25-0	DSL	Sulfuric acid, aluminum ammonium salt (2:1:1)	Ammonium alum (anhydrous)
7784-26-1	DSL	Sulfuric acid, aluminum ammonium salt (2:1:1), dodecahydrate	Ammonium alum
7784-28-3	DSL	Sulfuric acid, aluminum sodium salt (2:1:1), dodecahydrate	Sodium alum
7785-88-8	DSL	Phosphoric acid, aluminum sodium salt	Sodium aluminum phosphate
10043-67-1	DSL	Sulfuric acid, aluminum potassium salt (2:1:1)	Potassium alum (anhydrous)
10102-71-3	DSL	Sulfuric acid, aluminum sodium salt (2:1:1)	Sodium alum (anhydrous)
10279-59-1[c]	R-ICL	Phosphoric acid, aluminum sodium salt (8:2:3)	Sodium aluminum phosphate (anhydrous)
10305-76-7[c]	R-ICL	Phosphoric acid, aluminum sodium salt (8:3:1), tetrahydrate	Sodium aluminum phosphate

CAS RN	List	DSL or R-ICL name	Common name
11097-59-9	DSL	Magnesium, [carbonato(2-)]hexadecahydroxybis(aluminum) hexa-	Synthetic hydrotalcite
11138-49-1[b]	DSL	Aluminum sodium oxide	Sodium aluminate
12004-11-4	DSL	Aluminate(8-), hexaoxo[sulfato(2-)]di-, calcium (1:4)	Aluminum calcium oxide sulfate ($Al_2Ca_4O_6(SO_4)$)
12004-14-7	DSL	Aluminate(12-), hexaoxotris[sulfato(2-)]di-, calcium (1:6)	Aluminum calcium oxide sulfate ($Al_2Ca_6O_6(SO_4)_3$)
12005-57-1	DSL	Aluminate ($Al_{14}O_{33}^{24-}$), calcium (1:12)	Aluminum calcium oxide ($Al_{14}Ca_{12}O_{33}$)
12042-68-1	DSL	Aluminate (AlO_2^{1-}), calcium (2:1)	Aluminum calcium oxide (Al_2CaO_4)
12042-78-3	DSL	Aluminate (AlO_3^{3-}), calcium (2:3)	Aluminum calcium oxide ($Al_2Ca_3O_6$)
12042-91-0	DSL	Aluminum chloride hydroxide ($Al_2Cl(OH)_5$)	Aluminum chlorohydrate
13419-15-3	DSL	Aluminum, (octadecanoato-*O*)oxo-	Aluminum oxystearate
14782-75-3	DSL	Aluminum, (ethyl 3-oxobutanoato-*O1'*,*O3*)bis(2-propanolato)-, (T-4)-	Aluminum diisopropoxide ethylacetoacetate
15096-52-3	DSL	Cryolite	NA
15305-07-4	DSL	Aluminum, tris(*N*-hydroxy-*N*-nitrosobenzenaminato-*O*,*O'*)-	Aluminum cupferronate
15876-39-8[c]	R-ICL	Spiro[isobenzofuran-1(3H),9'-[9H]xanthen]-3-one,2',4',5',7'-tetrabromo-3',6'-dihydroxy, aluminum salt (3:2)	C.I. Pigment Red 90:1
21645-51-2	DSL	Aluminum hydroxide ($Al(OH)_3$)	NA
30745-55-2	DSL	Aluminum, bis(2-ethylhexanoato-o)hydroxy-	Hydroxyaluminum bis(2-ethylhexanoate)
31142-56-0	DSL	Aluminum citrate	NA
39290-78-3	DSL	Aluminium chloride hydroxide sulfate	Polyaluminum chloride hydroxide sulfate
53810-32-5	DSL	Aluminum hydroxide sulfate ($Al_4(OH)_6(SO_4)_3$)	NA

CAS RN	List	DSL or R-ICL name	Common name
54182-58-0[c]	R-ICL	Aluminum, hexadeca-μ-hydroxytetracosahydroxy[μ8-[[1,3,4,6-tetra-O-sulfo-β-D-fructofuranosyl α-D-glucopyranoside tetrakis(sulfato-κO')](8-)]]hexadeca-	Sucralfate
54326-11-3	DSL	Aluminum, (benzoato-*O,O'*)hydroxy(octadecanoato-*O,O'*)-	Aluminum hydroxide benzoate stearate
56639-51-1[c]	R-ICL	Aluminum, hydroxybis(tetradecanoato-κO)-	Aluminum dimyristate
57158-29-9[b]	DSL	Aluminum zirconium chloride hydroxide	Aluminum zirconium complexes
57455-37-5[b]	DSL	C.I. Pigment Blue 29	NA
65997-15-1[b]	DSL	Cement, portland, chemicals	NA
65997-16-2[b]	DSL	Cement, alumina, chemicals	NA
68131-74-8[a,b]	DSL	Ashes (residues)	NA
68425-65-0	DSL	Aluminum, oxo(2-propanolato)-	NA
68475-50-3[c]	R-ICL	Aluminum, tris[5-amino-4-hydroxy-3-(phenylazo)-2,7-naphthalenedisulfonato(2-)]di-	NA
68647-58-5[b]	DSL	Aluminum, benzoate hydrogenated tallow fatty acid iso-Pr alc. complexes	NA
68855-54-9[b]	DSL	Kieselguhr, soda ash flux-calcined	Flux-calcined diatomaceous earth
70131-50-9[b]	DSL	Bentonite, acid-leached	NA
90604-80-1[c]	R-ICL	Zirconium, chloro glycine hydroxy aluminum complexes	Aluminum zirconium complexes
134375-99-8[c]	R-ICL	Aluminum zirconium trichlorohydrex gly	Aluminum zirconium complexes

Abbreviations: NA, not available; DSL, Domestic Substances List; R-ICL, Revised In Commerce List

[a] This substance was included in this assessment as it was considered a priority on the basis of other human health concerns.

[b] This CAS RN is a UVCB (unknown or variable composition, complex reaction products, or biological materials).

[c] The Revised In Commerce List (R-ICL) is an administrative list of substances that are potentially used in products that are regulated under the *Food and Drugs Act* and that were in Canadian commerce between January 1, 1987, and September 13, 2001. The Government of Canada has prioritized these substances and is addressing them for their potential impact on human health and the environment in order to risk manage the substances, if required.

Appendix B. Physical-chemical properties of the 55 aluminium-containing substances

Table B-1. Physical and chemical properties for the 55 aluminium-containing substances

CAS RN	Common name (or substance name)	DSL/R-ICL substance category	Molecular weight (g/mol)	Water solubility (mg/L)	Vapour pressure (Pa)
75-24-1	Trimethylaluminum	Organometallic	72.1	NA (reactive)	1653[a]
96-10-6	Diethylaluminum chloride	Organometallic	120.6	NA (reactive)	28[b]
97-93-8	Triethylaluminum	Organometallic	114.2	NA (reactive)	3.4[c]
300-92-5	Aluminum distearate	Organic-metal salt	611.1	Insoluble[d]	NA
563-43-9	Ethylaluminum dichloride	Organometallic	151.0	NA (reactive)	700[e]
1070-00-4	Trioctylaluminum	Organometallic	366.7	NA (reactive)	NA
1116-73-0	Trihexylaluminum	Organometallic	282.6	NA (reactive)	NA
1302-42-7	Sodium aluminate	Inorganic	82.0	Very soluble[d]	NA
1317-25-5	Alcloxa; Aluminum chlorhydroxy allantoinate	Organometallic	314.6	Soluble[f]	NA
1327-41-9	Aluminum hydroxychloride	UVCB-Inorganic	210.5	Fully soluble ("550 000")[g]	NA
1328-04-7	C.I. Pigment Violet 5:1	UVCB-Organic-metal salt	NA	Insoluble[h]	NA
1344-28-1	Aluminum oxide (Al2O3)	Inorganic	102.0	Insoluble[d]	NA
5579-81-7	Aldioxa; Aluminum dihydroxy allantoinate	Organometallic	218.1	Insoluble[i]	NA
7784-18-1	Aluminum fluoride (AlF3)	Inorganic	84.0	5000[d]	NA
7784-25-0	Ammonium alum (anhydrous)	Inorganic	237.2	Slightly soluble[d]	NA
7784-26-1	Ammonium alum	Inorganic	453.4	Soluble[d]	NA
7784-28-3	Sodium alum	Inorganic	462.4	397 000[d]	NA
7785-88-8	Sodium aluminum phosphate	Inorganic	285.9	Insoluble[j]	NA
10043-67-1	Potassium alum (anhydrous)	Inorganic	258.2	59 000[d]	NA
10102-71-3	Sodium alum (anhydrous)	Inorganic	242.1	29 100[k]	NA
10279-59-1	Sodium aluminum phosphate (anhydrous)	R-ICL	897.8	NA	NA
10305-76-7	Sodium aluminum phosphate	R-ICL	949.9	NA	NA
11097-59-9	Synthetic hydrotalcite	Inorganic	532.0	Insoluble[l]	NA
11138-49-1	Sodium aluminate	UVCB-Inorganic	82.0	Very soluble[d]	NA
12004-11-4	Aluminum calcium oxide sulfate (Al2Ca4O6(SO4))	Inorganic	406.3	NA	NA
12004-14-7	Aluminum calcium oxide sulfate (Al2Ca6O6(SO4)3)	Inorganic	678.6	620[m]	NA

12005-57-1	Aluminum calcium oxide (Al14Ca12O33)	Inorganic	1386.7	NA	NA
12042-68-1	Aluminum calcium oxide (Al2CaO4)	Inorganic	158.0	NA (reactive)[d]	NA
12042-78-3	Aluminum calcium oxide (Al2Ca3O6)	Inorganic	270.2	Insoluble[d]	NA
12042-91-0	Aluminum chlorohydrate	Inorganic	174.5	Soluble[n]	NA
13419-15-3	Aluminum oxystearate	Organometallic	326.5	Sparingly soluble[o]	Negligible[o]
14782-75-3	Aluminum diisopropoxide ethylacetoacetate	Organometallic	277.4	NA (reactive)[p]	386.6[p]
15096-52-3	Cryolite	Inorganic	210.0	400[q]	Negligible[q]
15305-07-4	Aluminum cupferronate	Organometallic	438.4	0.28[r]	Negligible[r]
15876-39-8	C.I. Pigment Red 90:1	R-ICL	1991.5	NA	NA
21645-51-2	Aluminum hydroxide (Al(OH)3)	Inorganic	78.0	Insoluble[d]	Negligible[s]
30745-55-2	Hydroxyaluminum 2-ethylhexanoate	Organic-metal salt	330.5	Insoluble[t]	NA
31142-56-0	Aluminum citrate	Organic-metal salt	216.1	NA	NA
39290-78-3	Polyaluminum chloride hydroxide sulfate	Inorganic	175.5	Fully soluble[u]	NA
53810-32-5	Aluminum hydroxide sulfate (Al4(OH)6(SO4)3)	Inorganic	498.2	Fully soluble[v]	NA
54182-58-0	Sucralfate; Sulcrate	R-ICL	2086.8	NA	NA
54326-11-3	Aluminum hydroxide benzoate stearate	Organometallic	448.6	Sparingly soluble[w]	NA
56639-51-1	Aluminum dimyristate	R-ICL	498.8	NA	NA
57158-29-9	Aluminum zirconium complexes	UVCB-Inorganic	170.7	Fully soluble[x]	NA
57455-37-5	C.I. Pigment Blue 29	UVCB-Inorganic	994.5	Insoluble[y]	NA
65997-15-1	Cement, portland, chemicals	UVCB-Inorganic	NA	NA (reactive); slightly soluble[z]	NA
65997-16-2	Cement, alumina, chemicals	UVCB-Inorganic	NA	NA (reactive); 640[aa]	Negligible[aa]
68131-74-8	Ashes (residues)	UVCB-Inorganic	NA	Insoluble[ab]	NA
68425-65-0	Aluminum, oxo(2-propanolato)-	Organometallic	102.1	NA (reactive)[ac]	NA
68475-50-3	Aluminum, tris[5-amino-4-hydroxy-3-(phenylazo)-2,7-naphthalenedisulfonato(2-)]di-	R-ICL	1318.2	NA	NA
68647-58-5	Aluminum, benzoate hydrogenated tallow fatty acid iso-Pr alc. complexes	UVCB-Biological	NA	NA	NA

68855-54-9	Flux-calcined diatomaceous earth	UVCB-Inorganic	NA	Sparingly soluble[ad]	NA
70131-50-9	Bentonite, acid-leached	UVCB-Inorganic	NA	Sparingly soluble[ae]	NA
90604-80-1	Aluminum zirconium complexes	R-ICL	NA	NA	NA
134375-99-8	Aluminum zirconium complexes	R-ICL	NA	NA	NA

Abbreviations: NA: not available; DSL, Domestic Substances List; R-ICL, Revised In Commerce List; UVCB, unknown or variable composition, complex reaction products, or biological materials

[a] Boublik et al. 1994 as cited in PhysProp 2018
[b] Buchan et al. 1991
[c] Daubert and Danner 1989 as cited in PhysProp 2018
[d] Haynes 2016
[e] SDS 2018a
[f] TDS 2005
[g] O'Neil et al. 2001
[h] MSDS 1999
[i] TDS 2019
[j] Lewis 1997
[k] SDS 2018b
[l] SDS 2017a
[m] ECHA c2007-2019a
[n] ECHA c2007-2019b
[o] ECHA c2007-2019c
[p] ECHA c2007-2019d
[q] NIOSH 2018
[r] ECHA c2007-2019e
[s] ICSC c1996-2018
[t] SDS 2018c
[u] ECHA c2007-2019f
[v] SDS 2017b
[w] ECHA c2007-2019g
[x] ECHA c2007-2019h
[y] SDS 2018d
[z] SDS 2015
[aa] ECHA c2007-2019i
[ab] SDS 2016
[ac] ECHA c2007-2019j
[ad] ECHA c2007-2019k
[ae] ECHA c2007-2019l

Appendix C. Quantities, activities, and uses of the 55 aluminium-containing substances as reported in response to CEPA section 71 surveys

Table C-1. Summary of information on the Canadian manufacture and import of the 55 aluminium-containing substances submitted in response to CEPA section 71 surveys (Environment Canada 2013; ECCC 2017)

CAS RN	Common name	Total quantity manufactured (tonnes)[a]	Total quantity imported (tonnes)[a]	Reporting year
75-24-1	Trimethylaluminum	Under 0.1	1 to 10	2011
96-10-6	Diethylaluminum chloride	Under 0.1	10 to 100	2011
97-93-8	Triethylaluminum	Under 0.1	1 000 to 10 000	2011
300-92-5	Aluminum distearate	0.1 to 1	10 to 100	2011
563-43-9	Ethylaluminum dichloride	Under 0.1	1 to 10	2011
1070-00-4	Trioctylaluminum	Under 0.1	1 to 10	2011
1116-73-0	Trihexylaluminum	Under 0.1	10 to 100	2011
1302-42-7	Sodium aluminate	1 000 to 10 000	100 to 1 000	2011
1327-41-9	Aluminum hydroxychloride	Under 0.1	1 000 to 10 000	2011
1344-28-1	Aluminum oxide	10 000 to 100 000	1 000 000 to 10 000 000	2011
7784-18-1	Aluminum fluoride	Under 0.1	10 000 to 100 000	2011
7784-26-1	Ammonium alum	Under 0.1	100 to 1 000	2011
7784-28-3	Sodium alum	Under 0.1	1 to 10	2011
7785-88-8	Sodium aluminum phosphate	Under 0.1	100 to 1 000	2011
10043-67-1	Potassium alum (anhydrous)	Under 0.1	Under 0.1	2011
10102-71-3	Sodium alum (anhydrous)	Under 0.1	100 to 1 000	2011
11097-59-9	Synthetic hydrotalcite	Under 0.1	100 to 1 000	2011
11138-49-1	Sodium aluminate	Under 0.1	1 to 10	2011
12004-14-7	Aluminum calcium oxide sulfate ($Al_2Ca_6O_6(SO_4)_3$)	Under 0.1	1 to 10	2011
12042-68-1	Aluminum calcium oxide (Al_2CaO_4)	Under 0.1	10 000 to 100 000	2011
12042-91-0	Aluminum chlorohydrate	100 to 1 000	100 to 1 000	2011

CAS RN	Common name	Total quantity manufactured (tonnes)[a]	Total quantity imported (tonnes)[a]	Reporting year
13419-15-3	Aluminum oxystearate	Under 0.1	0.1 to 1	2011
14782-75-3	Aluminum diisopropoxide ethylacetoacetate	Under 0.1	10 to 100	2011
15096-52-3	Cryolite	10 000 to 100 000	1 to 10	2011
15305-07-4	Aluminum cupferronate	Under 0.1	1 to 10	2011
39290-78-3	Polyaluminum chloride hydroxide sulfate	1 000 to 10 000	100 to 1 000	2011
53810-32-5	Aluminum hydroxide sulfate	1 000 to 10 000	Under 0.1	2011
54326-11-3	Aluminum hydroxide benzoate stearate	Under 0.1	10 to 100	2011
57158-29-9	Aluminum zirconium complexes	Under 0.1	10 to 100	2011
57455-37-5	C.I. Pigment Blue 29	Under 0.1	100 to 1 000	2011
65997-15-1	Cement, Portland, chemicals	1 000 000 to 10 000 000	100 000 to 1 000 000	2011
65997-16-2	Cement, alumina, chemicals	Under 0.1	1 000 to 10 000	2011
68131-74-8	Ashes (residues)	Under 0.1	10 to 100	2011
68425-65-0	Aluminum, oxo(2-propanolato)-	Under 0.1	10 to 100	2011
68855-54-9	Flux-calcined diatomaceous earth	Under 0.1	1 000 to 10 000	2011
70131-50-9	Bentonite, acid-leached	Under 0.1	1 to 10	2011
10305-76-7	Sodium aluminum phosphate	Under 0.1	100 to 1 000	2015
56639-51-1	Aluminum dimyristate	Under 0.1	Under 0.1	2015
90604-80-1	Aluminum zirconium complexes	Under 0.1	100 to 1 000	2015
134375-99-8	Aluminum zirconium complexes	Under 0.1	10 to 100	2015

[a] Values reflect quantities reported in response to surveys conducted under section 71 of CEPA (Canada 2012, 2017). See survey for specific inclusions and exclusions (schedules 2 and 3). Quantities are presented as a range of values.

Table C-2. Aluminium-containing substances for which no information was submitted in response to CEPA section 71 surveys (Environment Canada 2013; ECCC 2017)

CAS RN	Common name	List	Survey[a]	Reporting year

1317-25-5	Alcloxa	DSL	Canada 2012	2011
1328-04-7	C.I. Pigment Violet 5:1	DSL	Canada 2012	2011
5579-81-7	Aldioxa	DSL	Canada 2012	2011
7784-25-0	Ammonium alum (anhydrous)	DSL	Canada 2012	2011
12004-11-4	Aluminum calcium oxide sulfate ($Al_2Ca_4O_6(SO_4)$)	DSL	Canada 2012	2011
12005-57-1	Aluminum calcium oxide ($Al_{14}Ca_{12}O_{33}$)	DSL	Canada 2012	2011
12042-78-3	Aluminum calcium oxide ($Al_2Ca_3O_6$)	DSL	Canada 2012	2011
30745-55-2	Aluminum 2-ethylhexanoate	DSL	Canada 2012	2011
31142-56-0	Aluminum citrate	DSL	Canada 2012	2011
68647-58-5	Aluminum, benzoate hydrogenated tallow fatty acid iso-Pr alc. complexes	DSL	Canada 2012	2011
10279-59-1	Sodium aluminum phosphate (anhydrous)	R-ICL	Canada 2017b	2014 or 2015
15876-39-8	C.I. Pigment Red 90:1	R-ICL	Canada 2017b	2014 or 2015
54182-58-0	Sucralfate	R-ICL	Canada 2017b	2014 or 2015
68475-50-3	Aluminum, tris[5-amino-4-hydroxy-3-(phenylazo)-2,7-naphthalenedisulfonato(2-)]di-	R-ICL	Canada 2017b	2014 or 2015

Abbreviations: DSL, Domestic Substances List; R-ICL, Revised In Commerce List

[a] Surveys conducted under section 71 of CEPA (Canada 2012, 2017). See survey for specific inclusions and exclusions (schedules 2 and 3).

Aluminum hydroxide (CAS RN 21645-51-2) was the only substance amongst the 55 aluminium-containing substances not included in a CEPA section 71 survey, given that trade data were available from the CBSA for the Harmonized System (HS) code 2818.30 (aluminum hydroxide). Table C-3 presents HS codes related to the Aluminium-containing Substances Group, along with average annual import quantities between 2010 and 2013 (CBSA 2016). The column entitled “HS code years” in Table C-3 indicates whether an HS code was available throughout 2010 to 2013, or only before or after the 2012 update.

Table C-3. Canadian import data for Harmonised System (HS) codes relevant to the Aluminium-containing Substances Group (CBSA 2016)

Harmonized system (HS) code	HS code description	HS code years	Average annual quantity imported (tonnes)	Related CAS RN(s)
2523.10.0000	Cement clinkers	2010 to 2013	10 000 to 100 000	65997-15-1
2523.21.0000	Portland cement: White cement, whether or not artificially coloured	2010 to 2013	10 000 to 100 000	65997-15-1
2523.29.0000	Portland cement: Other	2010 to 2013	1 000 000 to 10 000 000	65997-15-1
2523.30.0000	Aluminous cement, ciment fondu	2010 to 2013	100 000 to 1 000 000	65997-16-2
2523.90.0010	Other hydraulic cements: Masonry cement	2010 to 2013	100 000 to 1 000 000	65997-15-1
2523.90.0020	Other hydraulic cements: Oilwell cement	2010 to 2013	10 000 to 100 000	65997-15-1
2523.90.0030	Other hydraulic cements: Slag cement	2010 to 2013	100 000 to 1 000 000	65997-15-1
2523.90.0040	Other hydraulic cements: Supersulphated cement	2010 to 2013	1 to 10	65997-15-1
2523.90.0090	Other hydraulic cements: Other	2010 to 2013	1 000 000 to 10 000 000	65997-15-1
2818.20.0010	Alumina, prepared for processing into aluminum	2010 to 2013	1 000 000 to 10 000 000	1344-28-1
2818.20.0020	Activated alumina	2010 to 2013	1 000 to 10 000	1344-28-1
2818.20.0090	Aluminum oxide, nes	2010 to 2013	100 000 to 1 000 000	1344-28-1
2818.30.0000	Aluminum hydroxide	2010 to 2013	10 000 to 100 000	21645-51-2
2826.12.0000	Aluminum fluorides	2010 to 2013	10 000 to 100 000	7784-18-1
2826.30.0000	Sodium hexafluoroaluminat	2010 to 2013	10 to 100	15096-52-3

	e (synthetic cryolite)			
2827.49.0010	Aluminum chloride hydroxide	2010 and 2011	100 to 1 000	12042-91-0
2833.30.0010	Aluminum ammonium sulphate	2010 and 2011	10 to 100	7784-25-0; 7784-26-1
2833.30.0020	Aluminum sodium sulphate	2010 and 2011	100 to 1 000	7784-28-3; 10102-71-3
2841.90.2010	Sodium aluminate	2010 and 2011	100 to 1 000	1302-42-7; 11138-49-1
2842.90.0050	Sodium aluminum phosphate	2012 and 2013	100 to 1 000	7785-88-8; 10279-59-1; 10305-76-7
2842.90.9930	Sodium aluminum phosphate	2010 and 2011	100 to 1 000	7785-88-8; 10279-59-1; 10305-76-7
2931.00.1040	Triethylaluminum	2010 and 2011	1 000 to 10 000	97-93-8
2931.90.0010	Triethylaluminum	2012 and 2013	1 000 to 10 000	97-93-8
3206.41.0000	Ultramarine and preparations based thereon	2012 and 2013	100 to 1 000	57455-37-5
3206.41.0010	Ultramarine colouring matter	2010 and 2011	100 to 1 000	57455-37-5
3206.41.0021	Dispersions, based on ultramarine colouring matter	2010 and 2011	1 to 10	57455-37-5
3206.41.0029	Preparations, based on ultramarine colouring matter, nes	2010 and 2011	100 to 1 000	57455-37-5
3802.90.0010	Activated clay	2010 to 2013	100 000 to 1 000 000	70131-50-9
3802.90.0040	Activated diatomite	2010 to 2013	1 000 to 10 000	68855-54-9

Abbreviations: nes, not elsewhere specified.

As part of the CEPA section 71 notice published in 2017 (Canada 2017b), information on exports of select substances including the substances in the Aluminium-containing Substances Group was collected. This allows an estimate of apparent consumption (import plus manufacture minus export) to be made. Since there was a requirement to

report quantities of substances in products,[11] the potential difference between apparent consumption and total consumption should be modest. Apparent consumption of surveyed substances within the Aluminium-containing Substances Group for which responses were received is shown in Table C-4.

Table C-4. Apparent consumption for the 55 aluminium-containing substances according to information submitted in response to a CEPA section 71 survey (Canada 2017b)

CAS RN	Common name	Apparent consumption (tonnes)[a]	Reporting year
10305-76-7	Sodium aluminum phosphate	100 to 1000	2015
134375-99-8	Aluminum zirconium complexes	10 to 100	2015
56639-51-1	Aluminum dimyristate	Under 0.1	2015
90604-80-1	Aluminum zirconium complexes	100 to 1000	2015

[a] Values reflect quantities reported in response to a survey conducted under section 71 of CEPA (Canada 2017b). See survey for specific inclusions and exclusions (schedules 2 and 3).

Table C-5. Uses or functions associated with the largest apparent consumption of the 55 aluminium-containing substances according to information submitted in response to CEPA section 71 surveys (Environment Canada 2013; ECCC 2017)

CAS RN	Common name	Use associated with largest quantity[a]	Use associated with second largest quantity[a]	Use associated with third largest quantity[a]
75-24-1	Trimethylaluminum	CBI	CBI	NA
96-10-6	Diethylaluminum chloride	Process regulator	NA	NA
97-93-8	Triethylaluminum	Process regulator	Intermediate	CBI
300-92-5	Aluminum distearate	Processing aid	CBI	Paint and coating additive
563-43-9	Ethylaluminum dichloride	CBI	NA	NA
1070-00-4	Trioctylaluminum	CBI	CBI	NA
1116-73-0	Trihexylaluminum	Process regulator	CBI	Intermediate
1302-42-7	Sodium aluminate	By-product	Solids separation agent	Processing aid

[11] See survey (Canada 2017b) for specific inclusions and exclusions (schedules 2 and 3).

1327-41-9	Aluminum hydroxychloride	Solids separation agent	Processing aid	CBI
1344-28-1	Aluminum oxide	Primary material in aluminum production	Adhesives and sealants	Lubricants and additives
7784-18-1	Aluminum fluoride	Process regulators	CBI	Processing aid
7784-26-1	Ammonium alum	CBI	CBI	NA
7784-28-3	Sodium alum	Baking powder	NA	NA
7785-88-8	Sodium aluminum phosphate	Food leavening agent	Baking powder	NA
10043-67-1	Potassium alum (anhydrous)	CBI	NA	NA
10102-71-3	Sodium alum (anhydrous)	CBI	Food leavening agent	NA
11097-59-9	Synthetic hydrotalcite	Plastic additive	Processing aid	CBI
11138-49-1	Sodium aluminate	CBI	Solids separation agent	CBI
12004-14-7	Aluminum calcium oxide sulfate ($Al_2Ca_6O_6(SO_4)_3$)	Mortar	NA	NA
12042-68-1	Aluminum calcium oxide (Al_2CaO_4)	Refractory	CBI	CBI
12042-91-0	Aluminum chlorohydrate	Solids separation agent	Processing aid	Antiperspirant and deodorant
13419-15-3	Aluminum oxystearate	Paint and coating additive	NA	NA
14782-75-3	Aluminum diisopropoxide ethylacetoacetate	Paint and coating additive	NA	NA
15096-52-3	Cryolite	Aluminum electrolysis process	NA	NA
15305-07-4	Aluminum cupferronate	CBI	NA	NA

39290-78-3	Polyaluminum chloride hydroxide sulfate	Solids separation agent	Processing aid	Coagulant
53810-32-5	Aluminum hydroxide sulfate	Solids separation agent	Processing aid	NA
54326-11-3	Aluminum hydroxide benzoate stearate	Lubricants and additives	NA	NA
57158-29-9	Aluminum zirconium complexes	Antiperspirant and deodorant	NA	NA
57455-37-5	C.I. Pigment Blue 29	Pigment	CBI	CBI
65997-15-1	Cement, Portland, chemicals	Cement manufacturing	Construction material	CBI
65997-16-2	Cement, alumina, chemicals	Adhesives and sealants	Mortar	Filler
68131-74-8	Ashes (residues)	CBI	NA	NA
68425-65-0	Aluminum, oxo(2-propanolato)-	CBI	NA	NA
68855-54-9	Flux-calcined diatomaceous earth	Beer filtration	Anti-adhesive agent	Plastic films
70131-50-9	Bentonite, acid-leached	CBI	NA	NA
10305-76-7	Sodium aluminum phosphate	CBI	NA	NA
56639-51-1	Aluminum dimyristate	Cosmetics	NA	NA
90604-80-1	Aluminum zirconium complexes	Antiperspirant	NA	NA
134375-99-8	Aluminum zirconium complexes	NA	NA	NA

Abbreviations: CBI, confidential business information; NA, not applicable.
[a] Information reported in response to surveys conducted under section 71 of CEPA (Canada 2012, 2017). See survey for specific inclusions and exclusions (schedules 2 and 3).

Table C-6. Sectors of activity associated with the largest apparent consumption of the 55 aluminium-containing substances based on reporting to CEPA section 71 surveys (Environment Canada 2013; ECCC 2017)

CAS RN	Common name	Sector (NAICS4, abbreviated) associated with largest quantity[a]	Sector (NAICS4, abbreviated) associated with second largest quantity[a]	Sector (NAICS4, abbreviated) associated with third largest quantity[a]
75-24-1	Trimethylaluminum	CBI	CBI	NA

96-10-6	Diethylaluminum chloride	Basic chemical manufacturing	CBI	NA
97-93-8	Triethylaluminum	Basic chemical manufacturing	Chemical and allied product wholesalers	CBI
300-92-5	Aluminum distearate	Basic chemical manufacturing	Chemical and allied product wholesalers	Resin and synthetic rubber manufacturing
563-43-9	Ethylaluminum dichloride	CBI	NA	NA
1070-00-4	Trioctylaluminum	CBI	CBI	NA
1116-73-0	Trihexylaluminum	CBI	Basic chemical manufacturing	Chemical and allied product wholesalers
1302-42-7	Sodium aluminate	Basic chemical manufacturing	Other chemical product manufacturing	Pulp, paper and paperboard mills
1327-41-9	Aluminum hydroxychloride	Basic chemical manufacturing	Chemical and allied product wholesalers	CBI
1344-28-1	Aluminum oxide	Alumina and aluminum production	Iron and steel mills and ferro-alloy manufacturing	Non-ferrous metal (except aluminum) production
7784-18-1	Aluminum fluoride	Alumina and aluminum production	CBI	NA
7784-26-1	Ammonium alum	CBI	CBI	NA
7784-28-3	Sodium alum	Chemical and allied product wholesalers	NA	NA
7785-88-8	Sodium aluminum phosphate	Food manufacturing	Chemical and allied product wholesalers	NA
10043-67-1	Potassium alum (anhydrous)	CBI	NA	NA
10102-71-3	Sodium alum (anhydrous)	CBI	Chemical and allied product wholesalers	NA
11097-59-9	Synthetic hydrotalcite	CBI	Motor vehicle manufacturing	Chemical and allied product wholesalers

11138-49-1	Sodium aluminate	CBI	Chemical and allied product wholesalers	NA
12004-14-7	Aluminum calcium oxide sulfate ($Al_2Ca_6O_6(SO_4)_3$)	Building supplies wholesalers	NA	NA
12042-68-1	Aluminum calcium oxide (Al_2CaO_4)	Iron and steel mills and ferro-alloy manufacturing	Clay product and refractory manufacturing	Petroleum and coal product manufacturing
12042-91-0	Aluminum chlorohydrate	Basic chemical manufacturing	Pharmaceutical, toiletries and cosmetics wholesalers	Other chemical product manufacturing
13419-15-3	Aluminum oxystearate	Chemical and allied product wholesalers	Paint, coating and adhesive manufacturing	NA
14782-75-3	Aluminum diisopropoxide ethylacetoacetate	Other chemical product manufacturing	Paint, coating and adhesive manufacturing	NA
15096-52-3	Cryolite	Alumina and aluminum production	Iron and steel mills and ferro-alloy manufacturing	Spring and wire product manufacturing
15305-07-4	Aluminum cupferronate	CBI	NA	NA
39290-78-3	Polyaluminum chloride hydroxide sulfate	Basic chemical manufacturing	CBI	CBI
53810-32-5	Aluminum hydroxide sulfate	Basic chemical manufacturing	NA	NA
54326-11-3	Aluminum hydroxide benzoate stearate	Petroleum and coal product manufacturing	Other chemical product manufacturing	NA
57158-29-9	Aluminum zirconium complexes	Pharmaceutical, toiletries and cosmetics wholesalers	Chemical and allied product wholesalers	NA
57455-37-5	C.I. Pigment Blue 29	Plastic product manufacturing	Other chemical product manufacturing	Paint, coating and adhesive manufacturing
65997-15-1	Cement, Portland, chemicals	Cement and concrete product manufacturing	CBI	Chemical and allied product wholesalers

65997-16-2	Cement, alumina, chemicals	Iron and steel mills and ferro-alloy manufacturing	Building supplies wholesalers	CBI
68131-74-8	Ashes (residues)	Paint, coating and adhesive manufacturing	NA	NA
68425-65-0	Aluminum, oxo(2-propanolato)-	CBI	NA	NA
68855-54-9	Flux-calcined diatomaceous earth	Beverage manufacturing	Rubber product manufacturing	Plastic product manufacturing
70131-50-9	Bentonite, acid-leached	CBI	NA	NA
90604-80-1	Aluminum zirconium complexes	Grocery stores	NA	NA

Abbreviations: CBI, confidential business information; NAICS, North American Industry Classification System; NA, not applicable.

[a] Information reported in response to surveys conducted under section 71 of CEPA (Canada 2012, 2017). See survey for specific inclusions and exclusions (schedules 2 and 3).

Appendix D. Releases reported to the NPRI from 2013 to 2017 for aluminum (fume or dust only) and aluminum oxide (fibrous forms only)

Two aluminium-containing substances have been reportable to the NPRI in recent years: aluminum (fume or dust only) (as CAS RN 7429-90-5) and aluminum oxide (fibrous forms only) (as CAS RN 1344-28-1). Reporting is mandatory for facilities that manufacture, process, or otherwise use these substances at a concentration greater than or equal to 1% by weight (except for by-products and mine tailings) and in a quantity of 10 tonnes or more, and where employees work 20 000 hours or more per year (ECCC [modified 2022b]).

Although the specification of fume or dust only for aluminum and fibrous forms only for aluminum oxide may be limiting in the context of the broader Aluminium-containing Substances Group, NPRI data for these two substances were considered as part of the lines of evidence as they could potentially connect elevated measured environmental concentrations of aluminium to industrial releases and sectors. NPRI data for the five most recent years available (2013 to 2017) are presented in the tables below. Industrial sectors were attributed as North American Industry Classification System (NAICS) codes at the 4-digit level (NAICS4), as notified to the NPRI.

Table D-1. Sectors reporting the largest releases of aluminum (fume or dust) to the National Pollutant Release Inventory from 2013 to 2017 (NPRI [modified 2022])

Industrial sector (NAICS4)	Releases to air (tonnes)	Releases to land (tonnes)	Releases to water (tonnes)	Releases to unspecified media (tonnes)	Total releases (per year) (tonnes)
Electric power generation, transmission and distribution	26.4 to 107.3	0	0	0	26.4 to 107.3
Metal ore mining	20.2 to 25.8	0	0 to 1.4	0 to 0.4	21.2 to 26.3
Petroleum and coal product manufacturing	7.5 to 8.9	0	1.2 to 2.8	0	8.7 to 11.4
Alumina and aluminum production and processing	0.6 to 32.1	0	0	0	0.6 to 32.1
Oil and gas extraction	0 to 16.7	0	0 to 0.7	0 to 0.5	0.5 to 17.1
Water, sewage and other systems	0 to 14.7	0	0	0 to 0.02	0 to 14.7
Pulp, paper and paperboard mills	0 to 0.4	0	0 to 4.1	0 to 0.1	0 to 4.6

Iron and steel mills and ferro-alloy manufacturing	0 to 1.1	0 to 0.01	0	0.2 to 0.6	0.4 to 1.7
Cement and concrete product manufacturing	0 to 2.1	0	0	0	0 to 2.1
Aerospace product and parts manufacturing	0 to 0.7	0	0	0 to 0.5	0 to 0.7

As shown in Table D-1, the majority of aluminum (fume or dust only) is released to the air from electrical power generation, extraction (of metal ore and fossil fuels), and aluminum production. Annual aluminum (fume or dust only) disposals are reported in **Error! Reference source not found.**.

Table D-2. Total disposal quantities of aluminum (fume or dust only) from 2013 to 2017 (NPRI [modified 2022])

Year	Land treatment (tonnes)	Landfill (tonnes)	Storage (tonnes)	Tailings management (tonnes)	Underground injection (tonnes)	Waste rock management (tonnes)	Annual total (tonnes)
2013	0	4 535	214	5 432	546	0	10 727
2014	0	139	10	0	0	0	149
2015	622	286	11	0	0	0	920
2016	591	735	13	0	0	0	1 339
2017	277	1 154	20	0	0	102	1 554

NAICS4 sectors reporting the largest disposals of aluminum (fume or dust only) over this time period were waste treatment and disposal, metal ore mining, pulp, paper and paperboard mills, and alumina and aluminum production.

Table D-3. Sectors reporting the largest releases of aluminum oxide (fibrous forms) to the National Pollutant Release Inventory from 2013 to 2017 (NPRI [modified 2022])

Industrial sector (NAICS4)	Releases to air (tonnes)	Releases to land (tonnes)	Releases to water (tonnes)	Releases to unspecified media (tonnes)	Total releases (per year) (tonnes)
Aerospace product and parts manufacturing	0 to 43.2	0	0	0	0 to 43.2
Motor vehicle body and trailer manufacturing	0	0	0	0 to 1.5	0 to 1.5

Petroleum and coal product manufacturing	0	0	0	0.1 to 0.2	0.1 to 0.2
Iron and steel mills and ferro-alloy manufacturing	0	0	0	0 to 0.1	0 to 0.1
Ship and boat building	0	0	0	0 to 0.1	0 to 0.1
Paint, coating and adhesive manufacturing	0	0	0	0 to 0.1	0 to 0.1

As indicated in **Error! Reference source not found.**, the majority of reported releases of aluminum oxide (fibrous forms only) have been to air, from the aerospace product and parts manufacturing sector. However, these reports ceased after 2015 due to new information demonstrating that the form released was not fibrous and thus, no longer reportable.

Table D-4. Total disposal quantities of aluminum oxide (fibrous forms only) from 2013 to 2017 (NPRI [modified 2022])

Year	Land treatment (tonnes)	Landfill (tonnes)	Storage (tonnes)	Tailings management (tonnes)	Underground injection (tonnes)	Waste rock management (tonnes)	Annual total (tonnes)
2013	0	98	0	0	0	0	98
2014	0	1005	0	0	0	0	1005
2015	0	1974	0	0	0	0	1974
2016	0	3226	0	0	0	0	3226
2017	0	1935	0	0	0	0	1935

NAICS4 sectors reporting the largest disposals of aluminum oxide (fibrous forms only) over this time period were waste treatment and disposal, oil and gas extraction, waste collection, and metal ore mining.

Appendix E. Background concentrations and toxicity modifying factors

Table E-1. Median total aluminium background concentrations for selected Canadian ecozones

Region	Median (µg/L)	Sample size
Boreal Cordillera	258	304
Boreal Plains	120	645
Boreal Shield	62	1947
Mixedwood Plains	27	4444
Montane Cordillera	34	1951
Pacific Maritime	111	1467
Prairies	311	334
Taiga Cordillera	195	21

Kilgour & Associates Ltd. 2016. Data from BQMA 2015; FQMS 2014; FQMS 2016; PWQMN [modified 2020]; RAMP 2016; personal communication, data prepared by the Water Stewardship Division, Province of Manitoba, for the Ecological Assessment Division, Environment and Climate Change Canada, dated February 24 2016; unreferenced; personal communication, data prepared by the Environmental and Municipal Management Services, Saskatchewan Water Security Agency, for the Ecological Assessment Division, Environment and Climate Change Canada, dated February 25 2016; unreferenced.

Table E-2. Representative toxicity modifying factor values for selected Canadian ecozones

Region	Total hardness sample size	Geometric mean of total hardness (mg/L)	pH sample size	Mean of pH	DOC sample size	Geometric mean of DOC (mg/L)
Atlantic Maritime	5	32	110	7.2	35	4.4
Boreal Cordillera	305	79	283	8.0	210	1.5
Boreal Plains	643	120	656	8.0	486	19
Boreal Shield	1655	40	1981	7.8	1009	7.4
Mixedwood Plains	4941	150	5154	8.3	1394	5.3
Montane Cordillera	1936	61	1858	7.9	1070	1.2
Pacific Maritime	1490	19	1475	7.3	837	1.4
Prairies	369	260	420	8.2	20	10
Taiga Cordillera	22	110	22	8.0	20	10
Taiga Shield	175	7.4	176	6.9	160	3.6

Abbreviation: DOC, dissolved organic carbon.

BQMA 2015; FQMS 2016; NLTWQM [modified 2022]; PWQMN [modified 2020]; RAMP 2016; personal communication, data prepared by the Water Stewardship Division, Province of Manitoba, for the Ecological

Assessment Division, Environment and Climate Change Canada, dated February 24, 2016; unreferenced; personal communication, data prepared by the Environmental and Municipal Management Services, Saskatchewan Water Security Agency, for the Ecological Assessment Division, Environment and Climate Change Canada, dated February 25, 2016; unreferenced.

Appendix F. Potential to cause harm to soil invertebrates

Error! Reference source not found. summarizes the key soil toxicity studies identified. Endpoints from only the first two studies were statistically derived and therefore, were the only endpoints considered for the derivation of the soil PNEC (van Gestel and Hoogerwerf 2001; Zhao and Qiu 2010). Endpoints for the remaining studies were estimated on the basis of the concentrations reported and serve to support the weight of evidence.

Table F-1. Key studies considered in selecting a critical toxicity value for soil

Test organism	Endpoint	Test substance	pH	Value (mg/kg)	Reference
Earthworm (*Eisenia andrei)*	14-day LC_{50}	Al_2O_3	2.4 to 7.1 (KCl)	>5000	van Gestel and Hoogerwerf (2001)
Earthworm (*Eisenia andrei)*	14-day LC_{50}	$AlCl_3$	3.2 to 3.5 (KCl)	316	van Gestel and Hoogerwerf (2001)
Earthworm (*Eisenia andrei)*	14-day LC_{50}	$AlCl_3$	3.7 to 4.4 (KCl)	359	van Gestel and Hoogerwerf (2001)
Earthworm (*Eisenia andrei)*	14-day LC_{50}	$AlCl_3$	4.4 to 6.7 (KCl)	>1000	van Gestel and Hoogerwerf (2001)
Earthworm (*Eisenia andrei)*	14-day LC_{50}	$Al_2(SO_4)_3$	3.24 (KCl)	457	van Gestel and Hoogerwerf (2001)
Earthworm (*Eisenia andrei)*	14-day LC_{50}	$Al_2(SO_4)_3$	4.86 to 7.2 (KCl)	>4000	van Gestel and Hoogerwerf (2001)
Earthworm (*Eisenia andrei)*	EC_{50}, growth	$Al_2(SO_4)_3$	3.4 (KCl)	189	van Gestel and Hoogerwerf (2001)
Earthworm (*Eisenia andrei)*	EC_{50}, growth	$Al_2(SO_4)_3$	4.3 to 7.3 (KCl)	>1000	van Gestel and Hoogerwerf (2001)
Earthworm (*Eisenia andrei)*	EC_{50}, cocoon production	$Al_2(SO_4)_3$	3.4 (KCl)	294	van Gestel and Hoogerwerf (2001)
Earthworm (*Eisenia andrei)*	EC_{50}, cocoon production	$Al_2(SO_4)_3$	4.3 (KCl)	529	van Gestel and Hoogerwerf (2001)
Earthworm (*Eisenia andrei)*	EC_{50}, cocoon production	$Al_2(SO_4)_3$	7.3 (KCl)	291	van Gestel and Hoogerwerf (2001)
Earthworm (*Eisenia andrei)*	NOEC, growth	$Al_2(SO_4)_3$	4.3; 7.3 (KCl)	100	van Gestel and Hoogerwerf (2001)
Earthworm (*Eisenia andrei)*	NOEC, cocoon production	$Al_2(SO_4)_3$	3.4 to 7.3 (KCl)	100	van Gestel and Hoogerwerf (2001)
Earthworm (*Eisenia andrei)*	14-day LC50	$AlCl_3$	7.0	532	Zhao and Qiu 2010
Earthworm (*Eisenia andrei)*	28-day LOEC, survival	$AlCl_3$	4.3	100	Zhang et al. 2013
Earthworm (*Eisenia andrei)*	28-day LOEC, avoidance	$AlCl_3$	4.3	25	Zhang et al. 2013
Earthworm (*Octodrilus complanatus*)	50-day LOEC, weight	$Al(OH)_3$	7.55 (H_2O)	2000	Bilalis et al. 2013
Earthworm (*Octodrilus complanatus*)	50-day LOEC, protein %	$Al(OH)_3$	7.4 (H_2O)	1000	Bilalis et al. 2013
Earthworm (*Eisenia andrei)*	LOEC, sub-lethal effects	$Al(NO_3)_3$	4.6 (H_2O)	50	Tejada et al. 2010
Earthworm (*Dendrodrilus rubidus*)	NOEC, sub-lethal effects	$AlCl_3$	4.9 (KCl)	10	Rundgren and Nilsson 1997
Earthworm (*Dendrodrilus rubidus*)	LOEC, sub-lethal effects	$AlCl_3$	4.8 (KCl)	25	Rundgren and Nilsson 1997

Abbreviations: EC_{50}, median effect concentration; LC_{50}, median lethal concentration; LOEC, lowest observed effect concentration; NOEC, no observed effect concentration.

Appendix G. Exposure to environmental media, food, and drinking water

Table G-1. General human exposure factors for different age groups in scenarios[a]

Age groups	Body weight (kg)	Inhalation rate (m^3/day)	Soil ingestion rate (µg/day)	Dust ingestion rate (µg/day)
0 to 5 months	6.3	3.7	N/A	21.6
6 to 11 months	9.1	5.4	7.3	27.0
1 year	11	8.0	8.8	35.0
2 to 3 years	15	9.2	6.2	21.4
4 to 8 years	23	11.1	8.7	24.4
9 to 13 years	42	13.9	6.9	23.8
14 to 18 years	62	15.9	1.4	2.1
Adults (19+)	74	15.1	1.6	2.6

[a] Health Canada [modified 2022c]

Table G-2. Concentrations of aluminium in environmental media in Canada

Media	Median	95th percentile	n	Reference
Drinking water – non-municipal	Mean 0.02 mg/L	Max. 14.00 mg/L (SK)	4321	Health Canada 2021a; personal communication, emails from the WAQB, Health Canada, to the ESRAB, Health Canada, dated March 4, 2020; unreferenced
Drinking water – ground treated and surface water	Mean 0.008 mg/L	Max. 2.030 mg/L (SK)	1844	Health Canada 2021a; personal communication, emails from the WAQB, Health Canada, to the ESRAB, Health Canada, dated March 4, 2020; unreferenced
Drinking water – distributed water – ground and surface water	Mean 0.015 mg/L	Max. 1.420 mg/L (SK)	4307	Health Canada 2021a; personal communication, emails from the WAQB, Health Canada, to the ESRAB, Health Canada, dated March 4, 2020; unreferenced
Drinking water – surface treated	Mean 0.120 mg/L	Max. 7.970 mg/L (MB)	2469	Health Canada 2021a; personal communication, emails from the WAQB, Health Canada, to the ESRAB, Health Canada, dated March 4, 2020; unreferenced

Drinking water – surface distribution	Mean 0.111 mg/L	Max. 6.600 mg/L (NL)	7208	Health Canada 2021a; personal communication, emails from the WAQB, Health Canada, to the ESRAB, Health Canada, dated March 4, 2020; unreferenced
Drinking water – municipal (source)	0.025 mg/L	0.2085 mg/L	124	Tugulea 2016
Drinking water – municipal (distributed water - D2)	0.013 mg/L	0.122 mg/L	97	Tugulea 2016
Drinking water, on reserve in Atlantic region, AB, BC, ON, MB, QC, and SK	NA	Range 0.105 to 33.1 mg/L[a]	NA	FNFNES Chan et al. 2011, 2012, 2014, 2016, 2018, 2019a, 2019b
NAPS outdoor air $PM_{2.5}$	16 ng/m^3	329 ng/m^3	969	NAPS 2015
NAPS outdoor air $PM_{2.5}$	18.5 ng/m^3	50.9 ng/m^3	820	NAPS [modified 2022]
NAPS outdoor air $PM_{2.5}$	14.4 ng/m^3	41.9 ng/m^3	1334	NAPS [modified 2022]
NAPS outdoor air $PM_{2.5}$	17.7 ng/m^3	53.6 ng/m^3	1189	NAPS [modified 2022]
Outdoor air $PM_{2.5}$	58.7 ng/m^3	190 ng/m^3	121	Rasmussen et al. 2018
Outdoor air $PM_{2.5}$	Mean 69.0 ng/m^3	Max. 9 240 ng/m^3	Approx. 20 000	EC, HC 2010
Outdoor air $PM_{2.5}$	14.7 ng/m^3	55.9 ng/m^3	595	Personal communication, email from the WAQB, Health Canada, to the ESRAB, Health Canada, dated February 18, 2020; unreferenced
Outdoor air $PM_{2.5}$	NA	32.6 ng/m^3	125	Personal communication, email from the WAQB, Health Canada, to the ESRAB, Health Canada, dated February 18, 2020; unreferenced
Outdoor air $PM_{2.5}$	2.2 ng/m^3	62.8 ng/m^3	131	Personal communication, email from the WAQB, Health Canada, to the ESRAB, Health Canada, dated February 18, 2020; unreferenced
Outdoor air $PM_{2.5}$, port	9.1 ng/m^3	47.8 ng/m^3	512	Personal communication, email from the WAQB, Health Canada, to the ESRAB, Health Canada, dated

				February 18, 2020; unreferenced
Outdoor air $PM_{2.5}$, steel mill	78.8 ng/m^3	338.7 ng/m^3	105	Personal communication, email from the WAQB, Health Canada, to the ESRAB, Health Canada, dated February 18, 2020; unreferenced
Outdoor air $PM_{2.5}$, shale gas plant	133.7 ng/m^3	199.1 ng/m^3	55	Personal communication, email from the WAQB, Health Canada, to the ESRAB, Health Canada, dated February 18, 2020; unreferenced
Outdoor air $PM_{2.5}$, oil sands	15 to 46 ng/m^3	65 to 230 ng/m^3	230	WBEA 2019
Outdoor air $PM_{2.5}$, oil sands	13 to 51 ng/m^3	50 to 240 ng/m^3	302	WBEA 2020
Outdoor air $PM_{10–2.5}$	152 ng/m^3	348 ng/m^3	121	Rasmussen et al. 2018
Outdoor air PM_{10}	Mean 170 ng/m^3	Max. 24 940 ng/m^3	Approx. 10 000	EC, HC 2010
Outdoor air PM_{10}, port	71.4 ng/m^3	292.3 ng/m^3	271	Personal communication, email from the WAQB, Health Canada, to the ESRAB, Health Canada, dated February 18, 2020; unreferenced
Indoor air $PM_{2.5}$	23.7 ng/m^3	141 ng/m^3	121	Rasmussen et al. 2018
Indoor air $PM_{2.5}$	12.2 ng/m^3	53.4 ng/m^3	610	Personal communication, email from the WAQB, Health Canada, to the ESRAB, Health Canada, dated February 18, 2020; unreferenced
Indoor air $PM_{2.5}$	22.4 ng/m^3	119.1 ng/m^3	133	Personal communication, email from the WAQB, Health Canada, to the ESRAB, Health Canada, dated February 18, 2020; unreferenced
Indoor air $PM_{2.5}$	17.0 ng/m^3	131.1 ng/m^3	79	Personal communication, email from the WAQB, Health Canada, to the ESRAB, Health Canada, dated February 18, 2020; unreferenced
Indoor air $PM_{10–2.5}$	102 ng/m^3	449 ng/m^3	121	Rasmussen et al. 2018
Indoor air PM_{10}	Mean 1 490 µg/m^3	NA	Approx. 2 900	PTEAM study Riverside, California 1990 as cited in EC, HC 2010
Personal air $PM_{2.5}$	19.0 ng/m^3	111 ng/m^3	78	Rasmussen et al. 2018

Personal air $PM_{10–2.5}$	173 ng/m^3	910 ng/m^3	78	Rasmussen et al. 2018
Personal air $PM_{2.5,}$ subways	39.0 to 495.8 ng/m^3	193.7 to 577.2 ng/m^3	54	Personal communication, email from the WAQB, Health Canada, to the ESRAB, Health Canada, dated February 18, 2020; unreferenced
Personal air $PM_{10,}$ subways	249.6 to 1 106 ng/m^3	736.5 to 2 088 ng/m^3	54	Personal communication, email from the WAQB, Health Canada, to the ESRAB, Health Canada, dated February 18, 2020; unreferenced
Personal air $PM_{2.5,}$ buses	60.5 to 105.1 ng/m^3	168.1 to 1 323 ng/m^3	54	Personal communication, email from the WAQB, Health Canada, to the ESRAB, Health Canada, dated February 18, 2020; unreferenced
Personal air $PM_{10,}$ buses	519 to 1 308 ng/m^3	1 445 to 3 513 ng/m^3	54	Personal communication, email from the WAQB, Health Canada, to the ESRAB, Health Canada, dated February 18, 2020; unreferenced
Indoor air $PM_{2.5,}$ Private Car	19.2 to 77.8 ng/m^3	35.6 to 155.6 ng/m^3	22	Personal communication, email from the WAQB, Health Canada, to the ESRAB, Health Canada, dated February 18, 2020; unreferenced
Indoor air $PM_{10,}$ Private Car	59.4 to 308.8 ng/m^3	103.3 to 555.3 ng/m^3	22	Personal communication, email from the WAQB, Health Canada, to the ESRAB, Health Canada, dated February 18, 2020; unreferenced
Outdoor air $PM_{2.5,}$ Private Car	37.4 to 102.7 ng/m^3	110 to 1 533 ng/m^3	22	Personal communication, email from the WAQB, Health Canada, to the ESRAB, Health Canada, dated February 18, 2020; unreferenced
Outdoor air $PM_{10,}$ Private Car	248.9 to 1 257 ng/m^3	1 627 to 1 818 ng/m^3	22	Personal communication, email from the WAQB, Health Canada, to the ESRAB, Health Canada, dated February 18, 2020; unreferenced
Settled house dust	11 453 ug/g	NA	60	Rasmussen et al. 2018
Soil (10 provinces)	Mean 41 000 mg/kg	Max. 87 633 mg/kg	>40 studies	EC, HC 2010 as cited in Health Canada 2021a

Infant formula, milk-based	NA	Range 0.040 to 0.171 µg/g	NA	Health Canada [modified 2022b]
Infant formula, milk-based	440 ng/g	Range 0.0010 to 3.400 µg/g	437	Dabeka et al. 2011
Infant formula, soy-based	NA	Range 0.258 to 0.476 µg/g	NA	Health Canada [modified 2022b]
Infant formula, soy-based	0.73 µg/g	Range 0.230 to 1.100 µg/g	437	Dabeka et al. 2011
Human milk	Mean 0.1133 mg/L	NA	17	EC, HC 2010
Human milk	<0.00012 mg/L	0.0151 mg/L	847	Ppersonal communication, email from the FD, Health Canada, to the ESRAB, Health Canada, dated March 26, 2020; unreferenced

Abbreviations: MB, Manitoba; NA, not available; NL, Newfoundland; PM_1, Particulate matter of aerodynamic diameter of 1 µm or less; $PM_{2.5}$, Particulate matter of aerodynamic diameter of 2.5 µm or less; $PM_{10\text{-}2.5}$, Particulate matter of aerodynamic diameter between 2.5 µm and 10 µm; PM_{10}, Particulate matter of aerodynamic diameter of 10 µm or less; SK, Saskatchewan.

[a] Reported samples with aluminium above the guidance value of 100 µg/L.

Table G-3. Mean dietary exposure estimates (95% confidence intervals) (Food Directorate, personal communication, 2020, unreferenced)

Age (years) and gender	Mean, usual exposure (mg/kg bw per day)
Under 1 – M & F (formula fed)	0.086
1 to 3 – M & F	0.089 (0.085,0.093)
4 to 8 – M & F	0.073 (0.072,0.075)
9 to 13 – M	0.053 (0.051,0.056)
9 to 13 – F	0.049 (0.046,0.052)
14 to 18 – M	0.034 (0.033,0.035)
14 to 18 – F	0.032 (0.030,0.034)
19 to 30 – M	0.027 (0.026,0.029)
19 to 30 – F	0.028 (0.027,0.030)
31 to 50 – M	0.026 (0.024,0.028)
31 to 50 – F	0.029 (0.028,0.031)
51 to 70 – M	0.025 (0.024,0.026)
51 to 70 – F	0.030 (0.029,0.033)
71 or more – M	0.030 (0.028,0.032)
71 or more – F	0.032 (0.031,0.035)

Abbreviations: F, females; M, males

Table G-4. Estimated intake of aluminium by Indigenous communities in the Sahtú and Dehcho regions of the Northwest Territories (NWT) from the consumption of certain country foods

Age	Intake from consumption of land animals (mg/kg bw/day)[a]	Intake from consumption of large game organs (mg/kg bw/day)[b]	Intake from consumption of land animals and large game organs (mg/kg bw/day)[c]
4 to 8 years old	7.71×10^{-2}	1.88×10^{-3}	7.90×10^{-2}
9 to 13 years old	4.22×10^{-2}	1.03×10^{-3}	4.33×10^{-2}
14 to 18 years old	2.86×10^{-2}	6.97×10^{-4}	$2.93 \times x\ 10^{-2}$
Adults 19+	2.40×10^{-2}	5.84×10^{-4}	2.46×10^{-2}

[a] Conservatively assuming the concentration of aluminium is equal to the highest mean aluminium concentration reported in a study of land animals by Larter et al. (2016) (16.7 mg/kg in Mountain Caribou). Indigenous persons in the Sahtú and Dehcho regions in the NWT were assumed to consume 143 g of land animals 5.2 times/week (Ratelle et al. 2020a).
[b] Conservatively assuming the concentration of aluminium is equal to the highest mean aluminium concentration reported in a study of land animal organs by Larter et al. (2016) (0.36 mg/kg in Mountain Caribou kidney). Indigenous persons in the Sahtú and Dehcho regions in the NWT were assumed to consume 100 g of large game organs 8.4 times/week (Ratelle et al. 2020a).
[c] This is an estimate of intake from the reported country food commodities in Indigenous communities in the Sahtú and Dehcho regions in the NWT. As there are limited available data, the intake estimates presented are not representative of the mean dietary exposure of this community or mean exposure of other Indigenous communities.

Table G-5. Estimated average daily intake (mg/kg bw/day) of aluminium by the general population in Canada from environmental media, food, and drinking water

Route of exposure	0 to 5 months[a] (human milk-fed)[b]	0 to 5 months[a] (formula-fed)[c]	6 to 11 months[c,d]	1 year	2 to 3 years	4 to 8 years	9 to 13 years	14 to 18 years	≥19 years
Ambient air[e]	5.1×10^{-6}	5.1×10^{-6}	5.1×10^{-6}	6.3×10^{-6}	5.3×10^{-6}	4.2×10^{-6}	2.9×10^{-6}	2.2×10^{-6}	1.8×10^{-6}
Indoor air[f]	1.2×10^{-5}	1.2×10^{-5}	1.2×10^{-5}	1.5×10^{-5}	1.3×10^{-5}	1.0×10^{-5}	6.9×10^{-6}	5.3×10^{-6}	4.2×10^{-6}
Drinking water[g]	N/A	1.5×10^{-2}	9.3×10^{-3}	3.6×10^{-3}	3.2×10^{-3}	2.6×10^{-3}	2.0×10^{-3}	2.0×10^{-3}	2.3×10^{-3}
Food and beverages[h]	1.9×10^{-3}	8.6×10^{-2}	8.6×10^{-2}	8.9×10^{-2}	8.9×10^{-2}	7.3×10^{-2}	5.3×10^{-2}	3.4×10^{-2}	3.2×10^{-2}
Soil[i]	N/A	N/A	3.3×10^{-2}	3.3×10^{-2}	1.7×10^{-2}	1.6×10^{-2}	6.7×10^{-3}	9.3×10^{-4}	8.9×10^{-4}
Dust[j]	3.9×10^{-2}	3.9×10^{-2}	3.4×10^{-2}	3.6×10^{-2}	1.6×10^{-2}	1.2×10^{-2}	6.5×10^{-3}	3.9×10^{-4}	4.0×10^{-4}
Total intake (mg/kg bw/day)	0.041	0.140	0.16	0.16	0.13	0.10	0.066	0.036	0.032

Abbreviations: N/A, not applicable
[a] It is assumed that no soil ingestion occurs due to typical caregiver practices.

[b] The mean aluminium concentration in human milk is assumed to be 0.0151 mg/L (personal communication, email from the FD, Health Canada, to the ESRAB, Health Canada, dated March 26, 2020; unreferenced). This aluminium concentration was adjusted for the density of human milk (1.030 g/mL) (US EPA 2011). Exclusively for human milk-fed infants, assumed to consume 127.95 g/kg bw/day (0.783 L) of human milk per day, and human milk is assumed to be the only dietary source for infants under 6 months (Arcus-Arth et al. 2005; Health Canada 2018).
[c] Milk and soy-based infant formulas from CFIA data are included in the total dietary exposure estimates for aluminium (Table G-3, Appendix G) (personal communication, email from the FD, Health Canada, to the ESRAB, Health Canada, dated March 26, 2020; unreferenced). Exclusively for formula-fed infants who are assumed to drink 0.826 L of water per day (Health Canada 2018), where water is used to reconstitute formula.
[d] Daily aluminium intakes from human milk for children older than 6 months were not quantified but are expected to be lower than the aluminium intakes of formula-fed children or children of the same age consuming solid foods.
[e] Intake estimated using average outdoor air concentration $PM_{2.5}$ of 0.069 µg/m^3 (n = >10 000) (EC, HC 2010). Canadians are assumed to spend 3 hours outdoors each day (Health Canada 1998).
[f] Intake estimated using the median 24-hr indoor air sample $PM_{2.5}$ of 23.7 ng/m^3 (n = 121) measured in Windsor, Ontario (Rasmussen et al. 2018). Canadians are assumed to spend 21 hours indoors each day (Health Canada 1998).
[g] Intake estimated using mean concentration in distributed surface water of 0.111 mg/L (n = 7208) (Health Canada 2021a).
[h] Dietary exposure to aluminium was estimated using results from over 18 500 samples of food analyzed as part of the Total Diet Study (2008–2010 data) and by the Canadian Food Inspection Agency (2007–2013 data). Food consumption data from the Canadian Community Health Survey (CCHS) Cycle 2.2 (Statistics Canada 2004) were used to estimate usual dietary exposures, which were calculated using a probabilistic approach. When age groups were not comparable, the highest estimate was taken from the applicable age groups; details in Table G-3.
[i] Intake estimated using the average aluminium concentration of 41 000 mg/kg in soil from 10 provinces (EC, HC 2010). Aluminium bioaccessibility factor from soil was not applied to the estimated intake (see section 8.2.1).
[j] Intake estimated using the median aluminium concentration of 11 453 ug/g measured in 60 homes in Windsor, Ontario (Rasmussen et al. 2018). Aluminium bioaccessibility factor from dust was not applied to the estimated intake (see section 8.2.1).

Appendix H. Inhalation exposures to humans from products available to consumers

Exposure estimates were derived for multiple age groups; however, only estimates for the age group with the highest exposure estimate are presented here. Exposure estimates were derived using the highest concentration (weight fraction) of aluminium-containing substances found per product type or scenario, unless otherwise noted. The concentration of aluminium-containing substances in products available to consumers was obtained through information notified to Health Canada under the Cosmetic Notification System, the internal LNHPD [modified 2022], and the internal DPD [modified 2022] as noted in section 8.2.2.

Product amount, retention factor, and frequency of use in self-care product estimates were assumed from internal defaults, unless otherwise noted (Health Canada 2020). The values used for product amount, retention factors, exposure frequency (that is, frequency of use), and retention factors were developed through a process established for CMP assessments (Health Canada 2020). This process includes a review of available data on product amount, frequency of use, and retention factors of self-care products for comprehensiveness of the study or survey, the relevance of the data collected, and the type of information collected. The highest central tendency value from the studies with the highest quality rating is selected for use in CMP assessments, and underlying studies are cited.

Default inputs from the ConsExpo Web application and associated fact sheets (RIVM 2007) were used to estimate exposure from spray products with aluminium-containing substances, unless otherwise noted in Table H-1 below. Exposure from the use of powder self-care products and aluminium chlorohydrate containing sprays was estimated on the basis of data from exposure studies as described in section 8.2.2.

Table H-1. Exposure factors for estimating air concentrations via use of products available to consumers

Product	Exposure factors	Mean event exposure estimate (mg/m^3)	Adjusted exposure
Aerosol spray deodorant, potassium alum	From factsheet "Cosmetics: Deodorant cosmetics: Deodorant spray" Scenario "Application" Exposure model "Exposure to spray - spraying" Age group: adults Concentration: 3% potassium alum Exposure frequency: 1.3/day Spray duration: 0.17 min Exposure duration: 5 min Room volume: 10 m^3 Room height: 2.5 m Cloud volume: 0.0625 m^3 Ventilation rate: 2 per hour Mass generation rate: 0.45 g/s Airborne fraction: 0.9 Density non-volatile: 1.8 g/cm^3 Inhalation cut-off diameter: 10 µm[a] Aerosol diameter: normal Mean diameter: 27.8 µm[b] Standard deviation: 18.3[b] Maximum diameter: 130 µm[b] Spraying towards person: yes Adjusted daily air concentration (mg/m^3) = mean event air concentration (mg/m^3) × [exposure duration (min) × exposure frequency (/day) / 1440 min/day]	1.7	Adjusted daily air concentration: 7.6×10^{-3} mg substance/m^3 (Adult)
Aerosol sunscreen,	From factsheet "Cosmetics: Deodorant cosmetics: Deodorant spray" Scenario "Application"	0.18	Adjusted daily air concentration:

Product	Exposure factors	Mean event exposure estimate (mg/m^3)	Adjusted exposure
aluminium oxide	Exposure model “Exposure to spray - spraying” Age group: 1 year old to 13 years old Concentration: 2.535% aluminium oxide Exposure frequency: 1.6/day Spray duration: 0.2633 min[c] Exposure duration: 5 min Room volume: 10 m^3 Room height: 2.5 m Ventilation rate: 2 per hour Cloud volume: 0.0625 m^3 Mass generation rate: 0.45 g/s Airborne fraction: 0.9 Density non-volatile: 1.5 g/cm^3 Inhalation cut-off diameter: 10 µm[a] Aerosol diameter: normal Mean diameter: 57.9 µm[b] Standard deviation: 21.0[b] Maximum diameter: 138 µm[b] Spraying towards person: yes Adjusted daily air concentration (mg/m^3) = mean event air concentration (mg/m^3) × [exposure duration (min) × exposure frequency (/day) / 1440 min/day]		9.4×10^{-4} mg substance/m^3 (1 year old to 13 years old)
Aerosol temporary hair colour, C.I. Pigment Blue 29	From factsheet “Cosmetics: Hair care: Hair dye spray ” Scenario “Application” Exposure model “Exposure to spray - spraying” Age group: 4 years old to adult Concentration: 30% C.I. Pigment Blue 29 Spray duration: 0.24 min Exposure duration: 5 min Room volume: 10 m^3 Room height: 2.5 m Ventilation rate: 2 per hour Cloud volume: 0.0625 m^3 Mass generation rate: 0.4 g/s	5.4	Adjusted 4-hour air concentration: 1.1×10^{-1} mg substance/m^3 (4 years old to adult)

Product	Exposure factors	Mean event exposure estimate (mg/m^3)	Adjusted exposure
	Airborne fraction: 0.2 Density non-volatile: 1.5 g/cm^3 Inhalation cut-off diameter: 10 µm[a] Aerosol diameter: log-normal Mean diameter: 46.5 µm Arithmetic coefficient of variation: 2.1 Maximum diameter: 50 µm Spraying towards person: yes Adjusted 4-hour air concentration (mg/m^3) = mean event air concentration (mg/m^3) × [exposure duration (min) / 240 min]		
Face makeup powder, aluminium hydroxide	Algorithm: mean event air concentration (mg/m^3) = average talc study concentration (mg/m^3) × maximum aluminium-containing substance concentration in product (fraction) Age group: adult Average talc study concentration: 1.36 mg/m^3 [d] Concentration: 30% aluminium hydroxide Exposure duration: 5 min[e] Exposure frequency: 1/day Adjusted daily air concentration (mg/m^3) = mean event air concentration (mg/m^3) × [exposure duration (min) × exposure frequency (/day) / 1440 min/day]	0.41	Adjusted daily air concentration: 1.4×10^{-3} mg substance/m^3 (Adult)
Powdered sunscreen, aluminium oxide	Algorithm: mean event air concentration (mg/m^3) = average talc study concentration (mg/m^3) × maximum aluminium-containing substance concentration in product (fraction) Age group: 1 year old to 13 years old Average talc study concentration: 1.36 mg/m^3 [d] Concentration: 2.5% aluminium oxide Exposure duration: 5 min[e] Exposure frequency: 1.6/day	3.5×10^{-2}	Adjusted daily air concentration: 1.9×10^{-4} mg substance/m^3 (1 year old to 13 years old)

Product	Exposure factors	Mean event exposure estimate (mg/m^3)	Adjusted exposure
	Adjusted daily air concentration (mg/m^3) = mean event air concentration (mg/m^3) × [exposure duration (min) × exposure frequency (/day) / 1440 min/day]		
Loose foot powder, aluminium chlorohydrate	Algorithm: mean event air concentration (mg/m^3) = average talc study concentration (mg/m^3) × maximum aluminium-containing substance concentration in product (fraction) Age group: 3 years old to adult Average talc study concentration: 1.36 mg/m^3 [d] Concentration: 1% aluminium chlorohydrate Exposure duration: 5 min[e] Exposure frequency: 0.75/day Adjusted daily air concentration (mg/m^3) = mean event air concentration (mg/m^3) × [exposure duration (min) × exposure frequency (/day) / 1440 min/day]	1.4×10^{-2}	Adjusted daily air concentration: 3.6×10^{-5} mg substance/m^3 (3 years old to adult)
Aerosol antiperspirant, aluminium chlorohydrate	Algorithm: mean event air concentration (mg/m^3) = ACH study air concentration (mg/m^3) × (maximum ACH concentration in product / ACH study concentration) Age group: adult ACH study air concentration: 0.518 mg/m^3 [f] Maximum ACH concentration in product: 25% ACH study concentration: 4.2% [g] Exposure duration: 5 min[h] Exposure frequency: 1.3/day Age group: 14 to 18 years old ACH study air concentration: 0.518 mg/m^3 [f] Maximum ACH concentration in product: 25% ACH study concentration: 4.2%[g] Exposure duration: 5 min[h]	3.1 (Adult, 14 to 18 years old and 9 to 13 years old)	Adjusted daily air concentration: 1.4×10^{-2} mg substance/m^3 (Adult) Adjusted daily air concentration: 1.3×10^{-2} mg substance/m^3 (14 to 18 years old) Adjusted daily air concentration:

Product	Exposure factors	Mean event exposure estimate (mg/m^3)	Adjusted exposure
	Exposure frequency: 1.2/day Age group: 9 to 13 years old ACH study air concentration: 0.518 mg/m^3 [f] Maximum ACH concentration in product: 25% ACH study concentration: 4.2%[g] Exposure duration: 5 min[h] Exposure frequency: 1.1/day Adjusted daily air concentration (mg/m^3) = mean event air concentration (mg/m^3) × [exposure duration (min) × exposure frequency (/day) / 1440 min/day]		1.2×10^{-2} mg substance/m^3 (9 to 13 years old)
Foot deodorant spray, aluminium chlorohydrate	Algorithm: mean event air concentration (mg/m^3) = ACH study air concentration (mg/m^3) × (maximum ACH concentration in product / ACH study concentration) Age group: 2 years old to adult ACH study air concentration: 0.518 mg/m^3 [f] Maximum ACH concentration in product: 10% ACH study concentration: 4.2%[g] Exposure duration: 5 min[h] Exposure frequency: 1/day Adjusted daily air concentration (mg/m^3) = mean event air concentration (mg/m^3) × [exposure duration (min) × exposure frequency (/day) / 1440 min/day]	1.2	Adjusted daily air concentration: 3.2×10^{-3} mg substance/m^3 (2 years old to adult)

Abbreviations: ACH, aluminium chlorohydrate

[a] ConsExpo default for inhalation cut-off diameter is 15 µm; this has been adjusted to 10 µm, which is the default typically used by the Existing Substances Risk Assessment Bureau (Health Canada).

[b] Particle size distribution of aerosol skin care products (deodorant, sunscreen, etc.) is based on particle size distributions of over-the-counter aerosol drug products (that is, sunscreen and antiperspirants) measured by Liu et al. (2019).

[c] Spray duration is adjusted on the basis of the ratio of product amount of spray sunscreen used compared to product amount of spray deodorant used (Health Canada 2020).

[d] Overall average PM_4 event air concentration is from Anderson et al. 2017 and Rasmussen et al. 2019.

[e] Exposure time for powder face makeup, body powder, and foot powder is 5 minutes, considering the duration of particle cloud, study sampling duration, formation of secondary exposure clouds, and median time spent in the bathroom following a shower or bath (RIVM 2007; US EPA 2011; ECCC, HC 2021).

[f] Schwarz et al. 2018; study air concentration of ACH calculated assuming a mass fraction of 30.9% aluminium in ACH (CAS RN 12042-91-0) (MW = 174 g/mol, $Al_2ClH_5O_5$). Study air conc. ACH = 0.16 mg Al/m^3 / 0.309.
[g] ACH study concentration = Al study concentration from Schwarz et al. (2018) / mass fraction of 30.9% aluminium in ACH.
[h] Exposure time is 5 minutes for aerosol deodorant spray and aerosol foot spray on the basis of default exposure time for deodorant spray scenario from the RIVM cosmetics factsheet and median time spent in the bathroom following a shower or bath (RIVM 2007; US EPA 2011; ECCC, HC 2021).

Appendix I. Aluminium biomonitoring data

Table I-1. Concentrations of total aluminium in whole blood (µg/L) from the CHMS biobank project (Jayawardene et al. 2021)

Age Group	Sex	n	Median	P95
3 to 79 years old	M+F	5752	<8	<8
3 to 79 years old	M	2801	<8	<8
3 to 79 years old	F	2951	<8	<8
3 to 5 years old	M+F	475	<8	<8
6 to 11 years old	M+F	921	<8	11 (7.4–14)
6 to 11 years old	M	468	<8	11[a] (6.8–15)
6 to 11 years old	F	453	<8	11[a] (6.5-15)
12 to 19 years old	M+F	960	<8	9.9[a] (6.3–14)
12 to 19 years old	M	507	<8	12[a] (6.5–18)
20 to 39 years old	M+F	1214	<8	<8
20 to 39 years old	M	511	<8	<8
20 to 39 years old	F	703	<8	<8
40 to 59 years old	M+F	1148	<8	<8
40 to 59 years old	M	583	<8	<8
40 to 59 years old	F	565	<8	<8
60 to 79 years old	M+F	1034	<8	<8

Abbreviations: F, females; M, males; n, sample size; NA, not available.
[a] Use with caution. Statistics Canada guidelines for release stipulate that CVs between 16.6% and 33.3% are considered to have high sampling variability, and caution is recommended when using these data.

Appendix J. PBPK model

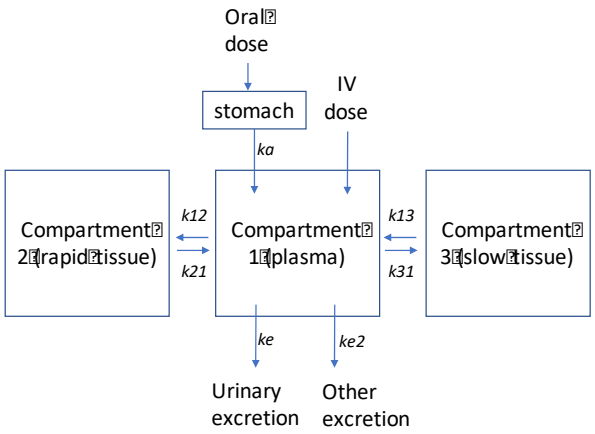

Figure J-1. Structure of three-compartment pharmacokinetic model for aluminium (from Poddalgoda et al. 2021)

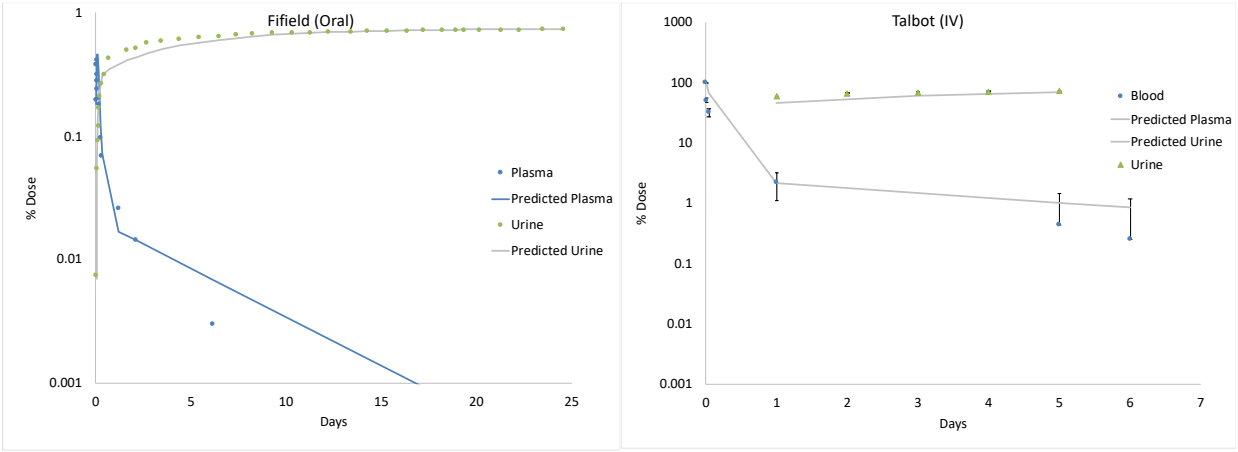

Figure J-2. Model fitted to data collected from volunteers administered aluminium citrate via a single oral dose (Fifield et al. 1997) and a single i.v. dose (Talbot et al. 1995) (from Poddalgoda et al. 2021).