Draft guidelines for Canadian drinking water quality

Chlorite and chlorate

Guideline technical document
for public consultation

Consultation period ends
April 14, 2026

Health Canada Santé Canada

Canada

Draft guidelines for Canadian drinking water quality, Chlorite and chlorate

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Purpose of consultation

This guideline technical document outlines the evaluation of the available information on chlorite, chlorate and chlorine dioxide, with the intent of updating the guideline values for chlorite and chlorate in drinking water. The purpose of this consultation is to solicit comments on the proposed guidelines, on the approaches used for their development, and on the potential impacts of implementing them.

The existing guideline technical document on chlorite and chlorate was developed in 2008. The maximum acceptable concentration (MAC) of 1 mg/L (1 000 µg/L) for chlorite was based on neurodevelopmental and liver effects seen in a two-generation study in rats. The MAC of 1 mg/L (1 000 µg/L) for chlorate was based on thyroid gland effects in rats from a 90-day study. No MAC was developed for chlorine dioxide since it quickly breaks down into mostly chlorite, and lesser amounts of chlorate in drinking water and is also quickly metabolized to chlorite and chlorate in laboratory animals.

This document proposes to keep the MAC of 1 mg/L (1 000 µg/L) for chlorite in drinking water, based on effects seen in the aforementioned two-generation study in rats. The MAC for chlorate of 1 mg/L (1 000 µg/L) based on thyroid effects would also remain the same but it would be based on a new, long-term study instead of a 90-day study. The approach for chlorine dioxide (no MAC) would remain unchanged.

This document is available for a 60-day public consultation period.

Please send comments (with rationale, where required) to Health Canada via email: water-consultations-eau@hc-sc.gc.ca.

All comments must be received before April 14, 2026. Comments received as part of this consultation will be shared with members of the Federal-Provincial-Territorial Committee on Drinking Water (CDW), along with the name and affiliation of their author. Authors who do not want their name and affiliation shared with CDW members should provide a statement to this effect along with their comments.

It should be noted that this guideline technical document will be revised following the evaluation of comments received, and drinking water guidelines will be established, if required. This document should be considered as a draft for comment only.

Proposed guideline values

The proposed MAC for chlorite in drinking water is 1 mg/L (1 000 µg/L). The proposed MAC for chlorate in drinking water is 1 mg/L (1 000 µg/L). A MAC for chlorine dioxide is not required because of its rapid reduction to chlorite and, to a lesser extent, chlorate in drinking water.

Drinking water treatment systems should make every effort to meet the guidelines; however, any method of control employed must not compromise the effectiveness of water disinfection.

Executive summary

This guideline technical document was prepared in collaboration with CDW and assesses all relevant information on chlorite and chlorate as well as chlorine dioxide. It assesses the health risks associated with chlorite and chlorate in drinking water, taking into account new studies and approaches, as well as available treatment technologies.

Exposure

Chlorite and chlorate are disinfection by-products of chlorine dioxide. Chlorine dioxide is an unstable gas used as a primary disinfectant or biocide in municipal water treatment and to control for taste, odour and colour. It must be generated onsite from either sodium chlorite or sodium chlorate as it rapidly breaks down into chlorite and chlorate. Under certain conditions, hypochlorite solutions used to treat drinking water can degrade and form chlorate. Chlorine dioxide and its disinfection by-products, chlorite and chlorate, are not naturally present in the environment.

Although the use of chlorine dioxide by drinking water systems in Canada is limited, drinking water is the main source of exposure to chlorite and chlorate for the general population of Canada.

Canadian data indicate that chlorite and chlorate levels found in drinking water are generally well below the proposed maximum acceptable concentration (MAC)s and are mostly below detection limits.

Health effects

Chlorine dioxide rapidly degrades to mostly chlorite, and lesser amounts of chlorate, in drinking water and is also quickly metabolized to chlorite and chlorate in laboratory animals. The health effects of chlorine dioxide are similar to those of chlorite, its major metabolite, and somewhat similar to chlorate.

The epidemiological databases for chlorite and chlorate were limited and unsuitable to assess long-term (chronic) risk from exposure. In animals, chlorite adversely affected neurodevelopment and development, brain and liver weights and altered thyroid hormone levels. Chlorate mainly caused thyroid and hematological effects, with thyroid effects being the most sensitive endpoint. No studies were found on neurodevelopmental effects from chlorate.

Analytical and treatment considerations

The development of a drinking water guideline takes into consideration the ability to both measure the substance and remove it from drinking water supplies. Several analytical methods are available for measuring chlorite, chlorate and chlorine dioxide concentrations, both at water treatment facilities and in the field.

Drinking water systems use chlorine dioxide largely to oxidize iron and manganese, control taste and odour and reduce total trihalomethanes formation. Chlorite can be removed after formation using ferrous salts, sulphur compounds and granular activated carbon, but chlorate is very difficult to effectively remove. Therefore, treatment strategies are focused on prevention as a best approach.

Distribution system

Although chlorine dioxide is a relatively strong disinfectant, it is not frequently used as a distribution system disinfectant for two reasons: 1) its residual does not last as long as that of other disinfectants and 2) it breaks down quickly into chlorite (predominantly). Chlorine dioxide decay in the distribution system is the result of auto-decomposition reactions and reactions with organic and inorganic compounds, including biofilms, pipe materials and scales. It is also subject to photolytic decomposition.

Application of the guidelines

Note**:** Specific guidance related to the implementation of drinking water guidelines should be obtained from the appropriate drinking water authority.

All water utilities should implement a comprehensive, up-to-date risk management water safety plan. A source-to-tap approach should be taken that ensures water safety is maintained. This approach requires a system assessment to characterize the source water; describe the treatment barriers that prevent or reduce contamination; identify the conditions that can result in contamination; and implement control measures. Operational monitoring is then established and operational/management protocols are instituted (for example, standard operating procedures, corrective actions and incident responses). Compliance monitoring is determined and other protocols to validate the water safety plan are implemented (for example, record keeping and consumer satisfaction). Operator training is also required to ensure the effectiveness of the water safety plan at all times.

There is significant complexity in dosing chlorine dioxide and monitoring this disinfectant and its by-products. The chlorine dioxide feed dose should not exceed 1.2 mg/L to minimize chlorite formation and limit potential exposure to chlorine dioxide. Given the disinfection efficacy of chlorine dioxide for drinking water, a higher dose is typically not necessary. Limiting the dose also provides a control measure to ensure that chlorite and chlorate concentrations do not exceed their proposed guidelines.

Table of Contents

1.0 Exposure considerations

1.1 Substance identity

Chlorite and chlorate are very soluble and reactive inorganic anions (Table 1). They will combine with metal ions to form solid salts (for example, sodium chlorite and sodium chlorate, respectively) (ATSDR 2004; OEHHA 2009; WHO 2016).

Chlorine dioxide (ClO_2) is an unstable gas at ambient temperature and pressure that is readily soluble in water (ATSDR 2004). It is stable in pure water in the absence of light but reacts quickly in alkaline solutions in the presence of sunlight to form ions such as chlorite, chlorate and chloride (WHO 2016).

Although used to control aesthetic issues, ClO_2 can also cause odour problems when it evaporates suddenly from cold water at the tap (less than 8 to 10 °C) (Suffet et al. 1995; WHO 2016). ClO_2 has a taste threshold of 0.20 to 0.25 ppm (equivalent to 0.20 to 0.25 mg/L) and odour threshold of 0.2 ppm (equivalent to 0.2 mg/L) (Suffet et al. 1995). Other reported threshold values ranged from 0.2 to 0.4 mg/L (Roche and Benanou 2007; U.S. NRC 1987). Customers describe the smell as chlorinous, kerosene-like and cat-urine-like (Dietrich and Hoehn 1991; Suffet et al. 1995). The odour problem can be mitigated by lowering the dose to below 1 mg/L (Dietrich and Hoehn 1991).

Technical grade sodium chlorite is approximately 80% pure. Its main impurities are sodium chloride and sodium chlorate salts (OEHHA 2009). Toxicological studies conducted on chlorite and chlorate typically use their sodium salts (OEHHA 2009).

Table 1: Physical and chemical properties of chlorine dioxide, sodium chlorite and sodium chlorate relevant to their presence in drinking water

Property	Chlorine dioxide[a]	Chlorite (sodium)[a]	Chlorate (sodium)[b]
CAS RN	10049-04-4	7758-19-2	7775-09-0
Synonyms	alcide, anthium dioxcide, chlorine oxide, chlorine peroxide, chloroperoxide, chloroperoxyl, chloryl radical	chlorite ion, chlorite anion, chlorine dioxide ion(1-), chlorous acid, ion(1-)[c]	chlorate ion, chlorate anion and chlorine oxide[c]
Molecular formula	ClO_2	$NaClO_2$	$NaClO_3$
Molecular weight (g/mol)	67.452	90.45	106.4
Appearance and physical state	Yellow to reddish gas	White solid	White or colourless crystals or granules

Property	Chlorine dioxide[a]	Chlorite (sodium)[a]	Chlorate (sodium)[b]
Melting point (°C)	-59	180 to 200 decomposes[d]	248
Boiling point (°C)	11	decomposes	> 300 decomposes
Log octanol-water partition coefficient (log K_{ow})	No data	low[c]	-7.08[c]
Henry's law constant	No data	Not applicable	Not applicable
Vapour pressure (volatility)	> 1 atm at 25 °C 142.13 kPa at 20 °C (very high)	Not applicable[c]	Not applicable[c]
Density	1.640 g/ml (0 °C liquid) 1.614 g/ml (10 °C liquid)	2.468 g/ml[d]	2.5 g/cm^3 (0 °C)
Water solubility	3.01 g/L at 25 °C and 34.5 mm Hg[a,d] highly soluble unstable, estimated half-life about 25 min.[d]	390 g/L at 30 °C very soluble dissociates into sodium and chlorite ions	101 g/L at 20 °C very soluble dissociates into sodium and chlorate ions

CAS RN – Chemical Abstracts Service Registry Number.
[a] ATSDR (2004) – unless otherwise stated, information for chlorine dioxide and sodium chlorite is from ATSDR (2004).
[b] WHO (2016) – unless otherwise stated, information for sodium chlorate is from WHO (2016).
[c] Health Canada (2008a)
[d] U.S. EPA (2006a)

1.2 Uses, sources and environmental fate

Uses/Sources: ClO_2 is a powerful antimicrobial and bleaching agent that is primarily used in pulp and paper mills. ClO_2 also has applications in industrial and municipal wastewater treatment, in the food processing and textile industries, and in healthcare settings (ATSDR 2004; Health Canada 2008a; U.S. EPA 2000; WHO 2016).

In municipal water treatment, it is used as a primary disinfectant or biocide and to control for taste, odour and colour (Volk et al. 2002). As a strong oxidizing agent (oxidation power relative to chlorine = 0.94) in water, ClO_2 disinfection requires less contact time and lower dose levels than chlorine for comparable coliform reductions (Aieta et al. 1980; Volk et al. 2002).

Due to its volatile and reactive nature, ClO_2 has a very limited shelf life and must be generated onsite. The main use of sodium chlorite and sodium chlorate is the generation of ClO_2 (ATSDR 2004; Health Canada 2008a). Sodium chlorite is preferred as a precursor when high purity (chlorine-free) water is needed, such as for water treatment purposes (ATSDR 2004). When high purity water is not required, like for pulp bleaching, sodium chlorate is typically used (ATSDR 2004). The majority of ClO_2 added to drinking water will eventually form chlorite and, to a lesser extent, chlorate as disinfection by-products (DBPs) (ATSDR 2004; NHMRC 2011). Chlorate is also formed during the decomposition of hypochlorite solutions that are stored for long periods, particularly at warm temperatures (WHO 2016).

Sodium chlorate and sodium chlorite are regulated by Health Canada's Pest Management Regulatory Agency (PMRA) for use in controlling slime-forming microbes in pulp and paper process waters and in recirculating cooling tower systems. Unlike the United States, they are not registered for use in agriculture (Health Canada 2008a; 2008b; 2017; U.S. EPA 2006a; 2006b).

Environmental fate: ClO_2 is not naturally occurring (Bates et al. 2002). Most releases of ClO_2 to the environment will be as gaseous industrial emissions or in wastewater. As a very reactive compound, ClO_2 will not exist in the environment for long periods of time (ATSDR 2004). In air, ClO_2 breaks down into chlorine gas and oxygen in the presence of sunlight or with exposure to mild heat, noise, flame or minor pressure (ATSDR 2004). Mobility and partitioning of ClO_2 in soils and sediments is insignificant as ClO_2 in solution will rapidly volatilize from soil surfaces or be reduced by oxidizable soil matter (ATSDR 2004; Bates et al. 2002). In water, ClO_2 will degrade, within a few hours to a few days, to chlorite, chlorate and chloride with the concentration of each ion dependent on factors such as temperature, pH, exposure to light and the presence of naturally occurring organic material (Bates et al. 2002).

Chlorite and chlorate are more likely to be released into the environment than their parent compound, ClO_2 (Baribeau et al. 2002). Since chlorite and chlorate are ions, they are not expected to volatize from moist soil or water surfaces and will not exist in the atmosphere in the vapour phase but will exist primarily in water (ATSDR 2004). Under environmental conditions, chlorite is unlikely to sorb onto suspended particles, sediment, or clay surfaces. It will be mobile in soils and leach into groundwater where it may undergo oxidation-reduction reactions with components in soils, suspended particles and sediments (for example, ferrous ions, manganese ions) (ATSDR 2004; WHO 2016). In sunlight, chlorite undergoes photolytic decomposition to form chlorate (Griese et al. 1992). Both chlorite and chlorate undergo reduction by bacteria under anaerobic conditions in anoxic groundwater, sediments and some soils (Logan 1998). The reduction of chlorite results in the formation of chloride ions (ATSDR 2004). Sodium chlorite and sodium chlorate are not expected to bioaccumulate or to persist in the environment (Health Canada 2008a). Aerobic soil half-lives for both chlorate and chlorite are expected to be below 180 days (Health Canada 2008a).

1.3 Exposure

Based on the physical-chemical properties of chlorite and chlorate and given the lack of data regarding their presence in food, air, soil and consumer products, chlorite and chlorate are not expected to be present in significant levels in environmental media other than drinking water treated with ClO_2 or hypochlorite (OEHHA 2009). Although few drinking water treatment plants (DWTPs) in Canada currently use ClO_2, in those facilities where it is used, drinking water would be the main source of exposure. Therefore, an allocation factor of 80% is considered appropriate (Krishnan and Carrier 2013). Exposure to ClO_2 is expected to be negligible because of its rapid breakdown into chlorite, chlorate and chloride (WHO 2016).

1.3.1 Water

During water treatment, if ClO_2 and chlorite are not removed prior to secondary disinfection with chlorine, they will react with free chlorine to form chlorate. Once chlorate is present in water, it is very persistent and difficult to remove. As the use of ClO_2 disinfection is not widespread among Canadian drinking water systems, only a few jurisdictions provided data. Based on limited provincial data, as well as First Nations water systems data, levels of chlorite and chlorate are generally low (see Table 2, Table 3 and Table 4).

Table 2: Chlorite and chlorate levels in treated and distributed water samples from Quebec, Ontario and Saskatchewan

Parameter	Chlorine dioxide used?	Water type	MRL (mg/L)	Detects/ samples	% Detect	Mean (mg/L)	Median (mg/L)	90th percentile (mg/L)
Chlorite (2018–2023)	Yes	Ground or unspecified - Distributed or unspecified	0.01	135/135	100	0.332	0.350	0.596
Chlorite (2018–2023)	No	Ground and/or surface/unspecified - Raw/treated/distributed/unspecified	0.01	188/748	25	0.042	0.050	0.050
Chlorite (2018–2023)	Seasonal	Surface - Raw or treated	0.01	4/14	28	NC	NC	NC
Chlorate (2018–2023)	Yes	Ground or unspecified - Distributed or unspecified	0.01	135/135	100	0.141	0.100	0.270
Chlorate (2018–2023)	No	Ground and/or surface/unspecified - Raw/treated/distributed/unspecified	0.01	429/751	57	0.204	0.078	0.552
Chlorate (2018–2023)	Seasonal	Surface - Treated	0.01	6/14	43	NC	NC	NC

MRL – method reporting limit; NC – not calculated due to statistically insignificant number of samples.

The analyzed data show that chlorite, as a DBP, is primarily relevant for drinking water systems using ClO_2. In the past, Quebec has been identified as the Canadian jurisdiction where ClO_2 is most frequently used for drinking water treatment purposes (Aranda-Rodriguez et al. 2008;

Health Canada 2005). Chlorate is likely to be a more widespread issue since it can impact systems applying chlorination as well as systems using ClO_2.

Table 3: Occurrence of chlorite in First Nations water systems

Jurisdiction	Chlorine dioxide used?	Water type	DL (mg/L)	Detects/ samples	% Detect	Mean (mg/L)	Median (mg/L)	90th percentile (mg/L)
Atlantic-FNIHB (2018–2023)[a]	No	Ground and/or surface - Treated	0.1	0/19	0.0	< DL	< DL	< DL
Atlantic-FNIHB (2018–2023)[a]	No	Ground and/or surface – Distributed	0.1–1.0	0/18	0.0	< DL	< DL	< DL
Ontario-FNIHB (2018–2023)[a]	Yes	Surface – Distributed	0.01–0.05	22/29	75.9	0.75	0.76	1.49
Ontario-FNIHB (2018–2023)[a]	Yes	Surface - Treated	0.10	16/18	88.9	0.94	1.09	1.55
Ontario-FNIHB (2018–2023)[a]	No	Ground and/or surface - Distributed	0.01–0.05	1/3	NC	NC	NC	NC
Quebec-FNIHB (2018–2023)[a]	No	Ground and/or surface - Distributed	0.08	0/84	0.0	< DL	< DL	< DL
Quebec-FNIHB (2018–2023)[a]	No	Ground and/or surface - Treated	0.08	0/85	0.0	< DL	< DL	< DL
Quebec-FNIHB (2018–2023)[a]	No	Ground and/or surface - Unspecified	0.08	0/19	0.0	< DL	< DL	< DL
British Columbia-FNHA (2012–2019)[a]	No	Ground and/or surface - Distributed	0.1–0.5	0/4	0.0	< DL	< DL	< DL

DL – detection limit (method detection limit or method reporting limit); FNHA – First Nations Health Authority; FNIHB – First Nations and Inuit Health Branch; NC – not calculated; Unspecified – sample not specified whether from treated or distributed water.

[a] Indigenous Services Canada (2023)

First Nations water systems data analyzed in Table 3 show chlorite levels above the detection limit were seen only in Ontario. In systems that did not use ClO_2, chlorite levels above the detection limit were recorded in approximately one third of distributed ground and/or surface water samples in the Ontario First Nations and Inuit Health Branch (FNIHB) region. The 90th percentile values of about 1.5 mg/L for samples taken from systems using ClO_2 were higher than the proposed chlorite guideline value of 1 mg/L.

Table 4: Occurrence of chlorate in First Nations water systems

Jurisdiction	Chlorine dioxide used?	Water type	DL (mg/L)	Detects /samples	% Detect	Mean (mg/L)	Median (mg/L)	90th Percentile (mg/L)
Atlantic- FNIHB (2018–2023)[a]	No	Ground and/or surface - Distributed	0.1–1.0	3/18	17	0.18	< DL	0.50
Atlantic-FNIHB (2018–2023)[a]	No	Ground and/or surface - Treated	0.1–0.5	11/19	58	0.18	0.15	0.36
Ontario-FNIHB (2018–2023)[a]	Yes	Surface - Distributed	NP	29/29	100	0.29	0.27	0.41
Ontario-FNIHB (2018–2023)[a]	Yes	Surface - Treated	NP	18/18	100	0.31	0.30	0.42
Ontario-FNIHB (2018–2023)[a]	No	Ground and/or surface - Distributed	NP	3/3	NC	NC	NC	NC
Quebec-FNIHB (2018–2023)[a]	No	Ground and/or surface - Distributed	0.08	51/84	60.7	0.15	0.11	0.35
Quebec-FNIHB (2018–2023)[a]	No	Ground and/or surface - Treated	0.08	69/85	81.2	0.24	0.16	0.55
Quebec-FNIHB (2018–2023)[a]	No	Ground and/or surface - Unspecified	0.08	7/19	36.8	0.06	< DL	0.11
British Columbia-FNHA (2012–2019)[a]	No	Ground and/or surface - Distributed	0.1–0.5	2/4	NC	NC	NC	NC

DL – detection limit (method detection limit or method reporting limit); FNHA – First Nations Health Authority; FNIHB – First Nations and Inuit Health Branch; NC – not calculated; NP – not provided; Unspecified – sample not specified whether from treated or distributed water.

[a] Indigenous Services Canada (2023)

Table 4 shows that detected chlorate levels were about 17% for groundwater and/or distributed surface water samples from systems not using ClO_2 in the Atlantic FNIHB region. In contrast, all of the treated/distributed surface water and groundwater samples from systems in the Ontario FNIHB region had detectable chlorate levels, regardless of whether they used ClO_2 or not. It is noteworthy that the two highest 90th percentile values identified for chlorate samples were from systems not using ClO_2. As previously noted, chlorate, as a DBP, may be an issue even in systems practising simple chlorination.

1.3.2 Food

Sodium chlorite and sodium chlorate are not currently registered in Canada for use on food or feed crops. However, the use of ClO_2, chlorite and chlorate in the food processing and packaging industries suggests that they may be present in foodstuffs (CMA 1989; WHO 2016). No Canadian data on the levels of chlorite and chlorate in foods were found; however, a maximum residue limit of 0.1 ppm would apply to any agricultural products imported into Canada (Health Canada 2008b).

,retain cooking water such as rice. In a study by Asami et al. (2013), the contribution of tap water to the total daily intake of chlorate ranged from 47% to 58% in populations with high rice consumption (WHO 2016).

Infant formula prepared using tap water treated with ClO_2 is unlikely to contain chlorite. Ascorbic acid, a component of infant formula, can react with and remove chlorite and ClO_2 (Ozawa and Kwan 1987). At concentrations of up to 20 mg/L of chlorite added to infant formula, 30% to 35% of chlorite was reduced to chloride within the first 5 minutes of contact while no chlorite remained after a 30-minute contact time (Busch 2006; Tran 2006). As reactive chemicals, ClO_2 and chlorite are also unlikely to be found in breast milk (ATSDR 2004). A study by Li et al. (2022) measuring chlorate in breast milk, infant formula, baby supplemental foods and tap water found mean chlorate levels of 1.73, 2.48 and 2.67 µg/L and no detections, respectively. The findings suggest that the contribution of tap water to chlorate exposure in infants is negligible.

The United States Environmental Protection Agency (U.S. EPA) estimated diet-only chlorate exposure at 2.7 µg/kg body weight (bw) per day for all populations, 4.5 µg/kg bw per day for under 1 year of age and 8.4 µg/kg bw per day for 1 to 2 years of age. Estimates were based on modelled data and used default concentration factors (U.S. EPA 2006b; WHO 2016). It concluded that risk from exposure through food only was below the level of concern for the general population and various subpopulations and attributed less than 20% of dietary intake to food only for the general population (U.S. EPA 2006b; WHO 2016).

International mean dietary exposures from acidified sodium chlorite-treated foods have been conservatively estimated to be between 0.2 to 0.7 µg/kg bw per day for chlorite and 0.1 to 0.6 µg/kg bw per day for chlorate, whereas national estimates for European Union countries of mean to 95th percentile dietary exposures in the general population were 0.9 to 3 µg/kg bw per day for chlorite and 0.3 to 0.6 ug/kg bw per day for chlorate (Joint FAO/WHO 2008). However, available data show that chlorate and chlorite residues in foods treated with acidified sodium chlorite declined with time to levels below the detection limits (Joint FAO/WHO 2008).

1.3.3 Air

Both chlorite and sodium chlorite are non-volatile and unlikely to be found in air (OEHHA 2009). Exposure to ClO_2 gas in air is unlikely outside of occupational settings (WHO 2016).

1.3.4 Other exposure pathways

No information on levels of chlorite, chlorate or their salts in soils was found. Swimming pool water disinfected with ClO_2 or hypochlorite solutions can be a source of dermal, inhalation and ingestion exposures to chlorate (Righi et al. 2014), and to a much lesser extent, chlorite. In an Italian study (Righi et al. 2014), mean levels of chlorate and chlorite were 3 661 µg/L and 149 µg/L in pool water (n = 24). The highest level of chlorate measured in pools (n = 33) in a German study was 40 mg/L while chlorite was not detected (Erdinger et al. 1999).

Dialysis water is routinely tested for total residual chlorine, chloramines and ClO_2; however, the method used does not detect residual chlorite or chlorate. Casey et al. (2017) found that granulated activated charcoal (GAC) filters remove chlorite. However, the GAC filters can become saturated before their scheduled replacement date. The authors suggest that dialysis providers closely monitor GAC filters, replacing them more frequently when ClO_2 is present in municipal or hospital water. Dialysis treatment providers at all levels—large facilities/hospitals, small community facilities, mobile units, providers for independent/home dialysis—should be notified that water is disinfected using ClO_2 and that it may contain chlorite and chlorate.

2.0 Health considerations

ClO_2 rapidly degrades to chlorite, and, to a lesser extent, chlorate, in drinking water (Michael et al. 1981) and is quickly metabolized to chlorite and chlorate in laboratory animals (Abdel-Rahman et al. 1982). Therefore, relevant scientific literature on ClO_2 as well as on chlorite and chlorate was evaluated to determine the most appropriate studies and toxicological endpoints for calculating the health-based values (HBVs).

Note: Most toxicological studies used sodium chlorite and sodium chlorate.

2.1 Kinetics

Following ingestion, ClO_2 is rapidly reduced to chloride in the saliva and stomach of monkeys (Bercz et al. 1982). Based on studies in rats, ingested radiolabelled chlorine incorporated into chlorite, chlorate and ClO_2 is rapidly absorbed from the gastrointestinal tract and appears quickly in the bloodstream (Abdel-Rahman et al. 1980; 1982; 1984a). The radiolabelled chlorine is then widely distributed throughout the bodies of the rats (Abdel-Rahman et al. 1980; 1982; 1984a). As reactive chemicals, ClO_2 and chlorite are unlikely to be found in neonatal blood, amniotic fluid, meconium or breast milk (ATSDR 2004). Abdel-Rahman et al. (1980) found no difference in the rate of absorption between rats given a single dose of ClO_2 and those ingesting water containing ClO_2 for 15 days.

In rats, all compounds were rapidly metabolized, with chloride being the major metabolite. Other metabolites included chlorite and chlorate (Abdel-Rahman et al. 1982; 1984a). Excretion was mainly in the urine and, to a lesser extent, in the feces but not in exhaled air (Abdel-Rahman et al. 1980; 1982; 1984a). Excretion half-life for ClO_2 decreased with repeat dosing, which the authors attributed to saturation of protein binding (Abdel-Rahman et al. 1980). Data

from Abdel-Rahman et al. (1984a) indicate that chlorate elimination is biphasic, with the initial alpha-phase being rapid followed by a slower beta-phase while chlorite elimination is likely a single phase. See Table 5 for more information.

Dermal absorption is not a significant route of exposure. Dermal absorption of sodium chlorate ranged from 0.30% to 1.05% of the total dose applied to human skin and from 2.17% to 14.42% of the total dose applied to rat skin in an in vitro study (ECHA 2006).

No physiologically-based pharmacokinetic (PBPK) models were found for ClO_2, chlorite or chlorate.

Table 5: Kinetics of chlorine dioxide, chlorite and chlorate in rats

Kinetic information	Chlorine dioxide	Chlorite	Chlorate
Absorption[a]	Rapidly absorbed from gastrointestinal tract	Rapidly absorbed from gastrointestinal tract	Rapidly absorbed from gastrointestinal tract
Absorption half-life in hours[a]	0.18 (single dose) 0.22 (15 days of dosing)	3.50	1.74
Time to reach peak plasma levels[a]	Not determined	2 hours	30 minutes
Percent absorbed[b]	30	35	Not determined
Metabolites (as percentage of initial dose)[a]	chloride (26.93) chlorite (3.46) chlorate (0.73)	chloride (31.55) chlorite (6.0)	chloride (20.50) chlorite (3.95) chlorate (8.2)
Distribution (as radiolabelled chlorine ions)[a]	Widely distributed throughout the body	Widely distributed throughout the body; highest concentration in blood	Widely distributed throughout the body; highest concentration in blood
Excretion (as percentage of recovered dose)[a]	Urine: 75 Feces: 25	Urine: 87 Feces: 13	Urine: 76 Feces: 24
Excretion (as percentage of initial dose)[a]	Urine: 30.81 Feces: 10.10	Urine: 33.16 to 35.86 Feces: 3.78 to 5.71	Urine: 38.02 to 42.25 Feces: 2.18 to 4.10
Elimination half-life in hours[a]	43.9 (single) 31.0 (15 days of dosing)	35.2	36.7
Excreted as[a]	Chloride Chlorite Chlorate	Chloride Chlorite	Chloride Chlorite Chlorate

[a] Abdel-Rahman et al. (1980; 1982; 1984a)
[b] U.S. EPA (2000)

2.2 Health effects

In humans, chlorite, chlorate and ClO_2, at levels typically found in drinking water, did not alter clinical chemistry, hematology or thyroid hormone levels in either a prospective study or in a study using volunteers (Lubbers et al. 1981; 1982; Michael et al. 1981). However, effects on red blood cells have been observed in in vitro studies using human cell lines exposed to sodium

chlorite and in cases of intentional ingestion of large amounts of chlorite or chlorate (Ali and Mahmood 2017; Burke et al. 2014; Gebhardtova et al. 2014; Lardieri et al. 2021; Lubbers et al. 1981; 1982; Michael et al. 1981; Parikh et al. 2021; Ranghino et al. 2006; Romanovsky et al. 2013; U.S. NRC 1982). Reproductive and developmental effects were either absent or were inconsistently seen (Källen and Robert 2000; Kanitz et al. 1996; Tuthill et al. 1982; Wright and Rivera-Núñez 2011) in humans. No effects on thyroid hormone levels, including triiodothyronine (T3), thyroxine (T4) and free T4, or on neonatal hypothyroidism were observed (Bercz et al. 1982; Källen and Robert 2000; Ouhoummane et al. 2004).

In animal studies, chlorite, chlorate and ClO_2 targeted the thyroid while chlorite and ClO_2 also targeted the liver and pup development, including neurodevelopment (Abdel-Rahman et al. 1984b; Bercz et al. 1982; Carlton et al. 1991; CMA 1996; Daniel et al. 1990; Gill et al. 2000; NTP 2005; Orme et al. 1985; Suh et al. 1984). Neurodevelopmental studies were not conducted using chlorate. All three parameters showed varying degrees of hematological effects with chlorate causing the most pronounced and consistent effects (Abdel-Rahman et al. 1980; Carlton et al. 1987; CMA 1996; Gill et al. 2000; Harrington et al. 1995a; Kurokawa et al. 1986; Moore and Calabrese 1982; NTP 2005).

2.2.1 Effects in humans

Most human studies investigating ClO_2 were not quantitative. Although a few volunteer studies detailed amounts of ClO_2, chlorite or chlorate ingested, their short duration (12 weeks), limited measured outcomes and lack of adverse effects make them unsuitable to assess risk from chronic exposure.

Drinking water containing ClO_2, chlorite or chlorate did not cause methemoglobinemia, hemolysis or other hematological effects, clinical chemistry effects or thyroid hormone level disturbances in either a prospective study or in studies using volunteers (Lubbers et al. 1981; 1982; Michael et al. 1981). Glucose-6-phosphate dehydrogenase (G6PD) activity, an enzyme critical to the production of glutathione (GSH), was also unaffected (Lubbers et al. 1981; 1982; Michael et al. 1981). However, accidental or intentional ingestion of high doses of chlorite (especially as commercial sodium chlorite solutions sold as Miracle Mineral Solution) or chlorate caused hemolysis and methemoglobinemia while ingestion of large amounts of chlorate caused decreased G6PD activity (Burke et al. 2014; Gebhardtova et al. 2014; Lardieri et al. 2021; Li et al. 2022; Parikh et al. 2021; Ranghino et al. 2006; Romanovsky et al. 2013; U.S. NRC 1982). ClO_2, chlorite and chlorate caused central nervous system effects, gastrointestinal effects and kidney damage when given in gram amounts (Arellano-Gutiérrez et al. 2021; Burke et al. 2014; Gebhardtova et al. 2014; Lardieri et al. 2021; Li et al. 2022; Medina-Avita et al. 2021; Parikh et al. 2021; Ranghino et al. 2006; Romanovsky et al. 2013; U.S. NRC 1982).

Reproductive and developmental effects were either absent or inconsistently seen. Generally, studies did not measure concentrations of ClO_2 in water or estimate the amount consumed by mothers. ClO_2 had no effect on rates of Caesarean section (Kanitz et al. 1996) while ClO_2, chlorite and chlorate had no effect on preterm delivery (Aggazzotti et al. 2004; Kanitz et al. 1996; Tuthill et al. 1982; Wright and Rivera-Núñez 2011). Only one of four studies found an

association between neonatal jaundice and maternal exposure to ClO_2 (Källen and Robert 2000; Kanitz et al. 1996; Tuthill et al. 1982; Wright and Rivera-Núñez 2011). Shorter body length and smaller head circumference were associated with maternal ClO_2 exposure in a study by Kanitz et al. (1996) but not in a study by Tuthill et al. (1982). Maternal ClO_2 exposure was not associated with small size for gestational age, lowered birth weight, fetal and neonatal mortality, Apgar score, neonatal hypothyroidism or cancer (Aggazzotti et al. 2004; Källen and Robert 2000; Kanitz et al. 1996; Tuthill et al. 1982).

Birth defects were not seen in Kanitz et al. (1996) or Källen and Robert (2000). However, a study by Cedergreen et al. (2002) found that cardiac defects in newborns were associated with maternal exposure to both ClO_2 and hypochlorite simultaneously through municipal drinking water supplies but not with other water treatment methods. The authors concluded that ClO_2 was the likely causative agent as exposure to hypochlorite alone was not associated with cardiac defects. Exposure to ClO_2 alone was not examined. In a case-control study examining 1 917 different congenital anomalies, birth defects (renal defects, abdominal wall defects, cleft palate, obstructive urinary defects and spina bifida) were associated with exposure to chlorite and chlorate (Righi et al. 2012).

A study by Ouhoummane et al. (2004) found no significant effect on thyroid stimulating hormone (TSH) levels or on the incidence of congenital hypothyroidism in newborns from 11 Quebec municipalities using ClO_2 to disinfect drinking water compared to 15 municipalities using chlorine as a disinfectant. Chlorite concentrations of drinking water, categorized as low, medium and high, had no effect on TSH levels in newborns. TSH levels were higher in newborns with low birthweight; however, because few babies were identified with low birthweight, this result may have been due to chance.

2.2.1.1 Populations that may be disproportionately impacted

The epidemiological database is insufficient to identify populations that may be disproportionately impacted.

2.2.2 Effects in animals

Toxicological studies for chlorite, chlorate and ClO_2 are summarized here and presented in more detail in Appendices B, C, and D.

2.2.2.1 Acute toxicity

Acute animal toxicity data for ClO_2, chlorite and chlorate are limited to oral lethal dose (LD_{50}) values (Table 6). Single gavage doses of 100, 250, 500 or 750 mg/kg bw of sodium chlorate given to male Wistar rats significantly decreased antioxidant defence enzymes, altered levels of enzymes involved in carbohydrate metabolism and were associated with damage in the intestinal villi and kidney (Ali et al. 2017; 2018).

Table 6: Acute oral toxicity values for chlorite, sodium chlorite, sodium chlorate and chlorine dioxide

Chemical	Species	LD_{50} (mg/kg bw)	Reference
Chlorite	Rat	79–133	OEHHA (2009)
Sodium chlorite	Rat	105–177	Musil et al. (1964); Seta et al. (1991)
Sodium chlorite	Rat	165	National Center for Biotechnology Information (2025c)
Sodium chlorite	Mouse	350	National Center for Biotechnology Information (2025c)
Sodium chlorite	Guinea pig	300	National Center for Biotechnology Information (2025c)
Sodium chlorate	Rat	1 200	National Center for Biotechnology Information (2025b)
Sodium chlorate	Mouse	8 350	National Center for Biotechnology Information (2025b)
Chlorine dioxide	Rat	94	National Center for Biotechnology Information (2025a)
Chlorine dioxide	Rat	292	National Center for Biotechnology Information (2025a)

bw – body weight; LD_{50} – oral lethal dose; OEHHA – Office of Environmental Health Hazard Assessment.

2.2.2.2 Chlorite

Neurodevelopment, development (delayed preputial separation) and the liver were the most sensitive endpoints of chlorite toxicity with no-observed-adverse-effect level (NOAEL) and lowest-observed-adverse-effect level (LOAEL) of 2.9 and 5.9 mg/kg bw per day of chlorite, respectively, seen in a two-generation reproductive/developmental study (CMA 1996; Gill et al. 2000). Other developmental endpoints were also affected, including crown-rump length, number of resorbed and dead fetuses, and day of vaginal opening or eye opening, but they occurred at higher doses than those causing neurodevelopmental effects or delayed preputial separation (CMA 1996; Couri et al. 1982a; Gill et al. 2000). However, soft tissue and skeletal malformations were not observed (CMA 1996; Couri et al. 1982a; Gill et al. 2000; Harrington et al. 1995b). The thyroid was also a target of chlorite toxicity. Chlorite altered thyroid hormone levels of rat pups (NOAELs of 0.75 and 3 mg chlorite/kg bw per day and LOAELS of 75 and 6 mg chlorite/kg bw per day) but had little to no effect on thyroid hormone levels of adult monkeys or adult rats (Bercz et al. 1982; Carlton et al. 1987; CMA 1996; Gill et al. 2000; Harrington et al. 1995b; Mobley et al. 1990).

Hematological changes were seen in some, but not all studies, with rats being the most sensitive species. Methemoglobinemia was either absent or inconsistently seen in studies measuring methemoglobin levels (Abdel-Rahman et al. 1980; Carlton et al. 1987; CMA 1996; Gill et al. 2000; Harrington et al. 1995b; Kurokawa et al. 1986; Moore and Calabrese 1982). Decreased fertility was seen in one drinking water study using rats but not in other studies using rats, mice or rabbits (Carlton et al. 1987; Couri et al. 1982a; Gill et al. 2000; Harrington et al. 1995b; Mobley et al. 1990; Moore and Calabrese 1980). Although sperm morphology and

motility were affected at doses greater than or equal to 7.5 mg/kg bw per day of chlorite in a drinking water study using rats, fertility was not affected (Carlton et al. 1987). Altered kidney pathology was see in two-year study but it was attributed to nonspecific salt effects by the author (Haag 1949). Studies are provided in greater detail in Appendix B: Summaries of animal studies using chlorite and sodium chlorite.

Liver: In a two-generation rat study, decreased absolute and relative liver weights were seen in F0 females and in F1 males given greater than or equal to 70 ppm of sodium chlorite (equivalent to greater than or equal to 7.9 mg chlorite/kg bw per day and greater than or equal to 5.9 mg chlorite/kg bw per day) and in F1 females given 300 ppm of sodium chlorite (equivalent to 28.6 mg chlorite/kg bw per day) in drinking water (CMA 1996). A shorter-term, 13-week study found no effect on liver weights in rats given doses of up to 80 mg/kg bw per day of sodium chlorite (equivalent to up to 59.7 mg/kg bw per day of chlorite) (Harrington et al. 1995b).

Bercz et al. (1986) measured serum liver enzymes in African green monkeys given up to 400 mg/L of sodium chlorite (equivalent to 58.4 mg/kg bw per day of chlorite) in drinking water. Statistically significant, dose-dependent increased serum glutamic pyruvic transaminase was observed but not serum glutamic oxaloacetic transaminase. Histopathology was not conducted on the liver, so the significance of the increase could not be determined.

Thyroid: One-generation rat studies by Carlton et al. (1987) and Mobley et al. (1990) found statistically significant decreased thyroid hormone levels in pups exposed to 7.5 and 6 mg/kg bw per day of chlorite or higher, respectively, but not in adults exposed to the same dose levels. In the Carlton et al. (1987) study, thyroid hormone levels were taken on lactation day 7, 21 and 40 for male pups (10/dose) and lactation day 21 and 40 for female pups (10/dose). T3 levels were decreased in female pups on day 21 and in male pups on day 40 while T4 levels were decreased in both male and female pups on day 40. In the Mobley et al. (1990) study, blood samples for thyroid hormone levels were taken from male pups on post-conception days 37, 38 and 42. Free and total T3 and total T4 levels were unaffected in pups, but free T4 was significantly increased on day 42 in high-dose pups (6 mg/kg bw per day of chlorite). However, F1 pups from a two-generation study receiving higher doses of chlorite (22.7 or 28.6 mg/kg bw per day for males and females, respectively) showed no effect on thyroid hormone levels. The two-generation study evaluated one pup per sex per litter (from 19 to 21 F1 litters per treatment group) and took samples on post-natal day (PND) 25 only, which may explain the lack of a response (CMA 1996; Gill et al. 2000).

Relative and absolute thyroid weights were unaffected in adult Crl:CD(SD)BR rats of both sexes gavaged with up to 80 mg/kg bw per day of sodium chlorite (equivalent to greater than or equal to 59.7 mg/kg bw per day of chlorite) for 13 weeks. Thyroid hormone levels were not measured (Harrington et al. 1995b).

Slightly decreased T4 levels were seen in African green monkeys (5 males and 7 females per dose) given up to 400 mg/L sodium chlorite (calculated as up to 58.4 mg/kg bw per day of

chlorite) in drinking water for 30 to 60 days. Although the decrease was dose-dependent, it lacked statistical significance (Bercz et al. 1982).

Hematological: In a two-generation rat study, hematological changes were seen starting at 35 ppm in male and female F1 parental animals (equivalent to 2.9 and 3.8 mg chlorite/kg bw per day, respectively) and in F1 female pups (equivalent to 3.8 mg chlorite/kg bw per day) and at 70 ppm in F1 male pups (equivalent to 5.9 mg chlorite/kg bw per day). However, hematological changes were within historical ranges for results seen at 35 and 70 ppm but not 300 ppm. Methemoglobin levels were increased in F1 female pups only, starting at 35 ppm. The NOAEL for hematological parameters was 2.9 mg chlorite/kg bw per day in F1 male parental animals. Hematological parameters were not measured in the F0 or F2a/F2b generations (CMA 1996; Gill et al. 2000).

Rats gavaged with 10, 25, or 80 mg/kg bw per day of sodium chlorite (equivalent to 7.4, 18.6 or 59.7 mg/kg bw per day of chlorite) for 13 weeks had increased white blood cell counts, decreased red blood cell counts and increased spleen and adrenal weights beginning at 25 mg/kg bw per day. Multiple changes in red blood cells and their associated indices were seen in both sexes at the highest dose, with the effects more pronounced in males. Changes in methemoglobin levels were inconsistent. Trend analysis showed statistically higher levels in mid-dose males only and statistically significant decreased levels in high-dose females. A NOAEL of 10 mg/kg bw per day of sodium chlorite (equivalent to 7.4 mg/kg bw per day of chlorite) and a LOAEL of 25 mg/kg bw per day of sodium chlorite (equivalent to 18.6 mg/kg bw per day of chlorite) were determined based on increased white blood cells in males, and increased adrenal and spleen weights and decreased red blood cell counts in females (Harrington et al. 1995b).

Hemolysis was seen in pregnant rats given 2% solutions of sodium chlorite (equivalent to 610 mg/kg bw per day of chlorite) in drinking water from gestation day (GD) 8 to 15 but not at lower doses. However, decreased maternal body weight, accompanied by decreased water and food intakes, were observed starting at the 0.5% dose (Couri et al. 1982b). In a study by Abdel-Rahman et al. (1980), male rats were given chlorite in drinking water at 10 or 100 mg/L [equivalent to 1 or 10 mg/kg bw per day (OEHHA 2009)] for 4 months. A statistically significant decrease in hemolysis was observed in the 100 mg/L group at 2 months but not at 4 months, while blood GSH was statistically decreased at both 2 and 4 months. Methemoglobin was measured for, but not detected, in the study.

Statistically significant, dose-dependent decreased red blood cell count and cell indices were observed in African green monkeys dosed with up to 400 mg/L of sodium chlorite (equivalent to up to 58.4 mg/kg bw per day of chlorite). The lack of detail in the presentation of the data does not allow for the identification of threshold doses. Other dose-dependent hematological (reticulocyte count, methemoglobin, hemoglobin) changes were noted but were not statistically significant (Bercz et al. 1982).

Doses of up to 100 mg/L of sodium chlorite (equivalent to 15 mg/kg bw per day of chlorite) in drinking water for 30 days had no effect on 11 hematological parameters in two strains of mice.

The NOAEL was 100 mg/L of sodium chlorite (equivalent to 15 mg/kg bw per day of chlorite) (Moore and Calabrese 1982). Similarly, sodium chlorite at doses up to 100 ppm (equivalent to 7.5 mg/kg bw per day of chlorite) had no effect on hematological parameters, including methemoglobin levels, in a rat reproductive/developmental study (Carlton et al. 1987). Hematological changes were also absent in rats and mice given up to 600 ppm and 500 ppm of sodium chlorite in drinking water for 85 weeks, respectively (Kurokawa et al. 1986).

Kidney: Altered kidney pathology (distended glomerular capsule, material in renal tubules) was seen in a 2-year carcinogenicity study in which a small number of rats were given 100 mg/L or greater (equivalent to greater than or equal to 9.3 mg/kg bw per day) of chlorite in drinking water. However, the author attributed the changes to nonspecific salt effects (Haag 1949). In a subchronic gavage study, rats were given 10 to 80 mg/kg bw per day for 13 weeks. Increased kidney weights were seen in females at 80 mg/kg bw per day of sodium chlorite (equivalent to 59.7 mg/kg bw per day of chlorite) but not in males (Harrington et al. 1995b). No effects on body weight gain, absolute and relative kidney weights, kidney histology or water consumption were observed in mice given 4 to 100 ppm sodium chlorite (3 to 75 ppm chlorite) in drinking water for 30 or 90 days (Moore and Calabrese 1982).

Developmental: Chlorite caused delays in developmental (day of eye opening, preputial separation and vaginal opening) and neurodevelopmental (exploratory activity, maximum response amplitude) milestones, as well as decreased growth (crown-rump length, body weights) but did not cause statistically significant increases in the incidence of soft tissue or skeletal malformations.

A two-generation rat study reported a LOAEL of 70 ppm of sodium chlorite (equivalent to 5.9 mg/kg bw per day of chlorite) and a NOAEL of 35 ppm of sodium chlorite (equivalent to 2.9 mg/kg bw per day of chlorite) based on decreased maximum response amplitude in an acoustic startle habituation test in F2b pups and on delayed preputial separation in F1 pups (CMA 1996; Gill et al. 2000). In F1 pups, a significant decrease in absolute but not relative brain weights was observed in males at 300 ppm on PND 11 but not at greater than or equal to 25 as reported by Gill et al. (2000); however, the unpublished Chemical Manufacturers Association (CMA) (1996) study as reported by Toxicology Excellence for Risk Assessment (TERA) (1998) found decreased absolute brain weights in F1 and F2 males and females at 70 ppm. There were no gross or macroscopic lesions in the brains or spinal cords. In F1 pups, preputial separation was delayed starting at 70 ppm while vaginal opening was delayed at 300 ppm. Eye opening was delayed in F2 pups at 300 ppm. No effects were seen in the functional observation battery, motor activity (figure-of-eight), swim maze learning or on T3 and T4 levels. Chlorite had no effect on anogenital distance, gross external malformations or timing of ear opening in F1, F2a and F2b pups.

In a one-generation rat study, pups of dams given 20 or 40 ppm (equivalent to 3 or 6 mg/kg bw per day) of chlorite in drinking water starting at 10 days pre-mating until post-conception day 42 had decreased exploratory activity. No effect was seen on pup weight or weight gain, or day of eye opening. The LOAEL was 20 ppm (3 mg/kg bw per day) of chlorite (Mobley et al. 1990).

Pregnant Sprague-Dawley rats given 0.1% to 2% of sodium chlorite (estimated at 70 to 610 mg/kg bw per day of chlorite) in drinking water had an increased number of resorbed and dead fetuses and fetuses with significantly decreased crown-rump lengths. Fetal weights, number of soft tissue and skeletal malformations, and postnatal pup growth were unaffected by treatment. The study LOAEL was 70 mg/kg bw per day of chlorite based on decreased fetal crown-rump length and number of resorptions (Couri et al. 1982b). Fetal crown-rump length was also affected in a study by Suh et al. (1983) at 10 mg/L (equivalent to 1.0 mg/kg bw per day) of chlorite in drinking water.

No effects were seen on median day of eye opening or observed vaginal patency, or on body weight or organ-to-body weight ratios in a one-generation study in which pregnant rats were given up to 100 ppm of sodium chlorite (equivalent to up to 7.5 mg/kg bw per day of chlorite) in drinking water. At 100 ppm, T3 and T4 levels were decreased in pups but not in parental animals. The LOAEL was 7.5 mg/kg bw per day of chlorite based on decreased T3 and T4 levels in pups (Carlton et al. 1987). Harrington et al. (1995b) also found no effect on the incidence of major and minor external, visceral or skeletal fetal abnormalities in New Zealand white rabbits given up to 1 200 ppm of sodium chlorite (estimated at 39.6 mg/kg bw per day of chlorite) in drinking water from GD 7 to 19.

In a study by Moore et al. (1980), average pup weaning weight and birth-to-weaning growth rate were significantly decreased in pups from pregnant A/J mice (10/dose) given 100 ppm of sodium chlorite [equivalent to 23 mg chlorite/kg bw per day in (ATSDR 2004)] in drinking water from presence of vaginal plug until weaning at 28 days when compared to controls.

Reproductive: Sodium chlorite caused decreased fertility (increased number of resorbed and dead fetuses) in rats (Couri et al. 1982b) when given in drinking water as a 0.1% solution or greater (equivalent to greater than or equal to 70 mg chlorite/kg bw per day). However, it had no statistically significant effect on fertility in rats (Carlton et al. 1987; Gill et al. 2000; Mobley et al. 1990), mice (Moore et al. 1980) or rabbits (Harrington et al. 1995b) when given in drinking water at doses up to 1 200 ppm of sodium chlorite (equivalent to 39.6 mg chlorite/kg bw per day). Decreased sperm motility and increased abnormal sperm morphology were seen in male Long-Evans rats given greater than or equal to 100 ppm of sodium chlorite (estimated as greater than or equal to 7.5 mg/kg bw per day of chlorite) in drinking water but fertility was not affected (Carlton et al. 1987). Other studies found no effect on sperm morphology and motility. Sperm parameters were unaffected in a two-generation study using doses of up to 300 ppm (equivalent to 22.7 mg/kg bw per day of chlorite) in drinking water (CMA 1996; Gill et al. 2000). Sodium chlorite did not cause sperm head abnormalities in male B6C3F1mice (Meier et al. 1985).

2.2.2.3 Chlorate

In animals, chlorate primarily caused thyroid and hematological effects. Thyroid gland follicular cell hypertrophy and altered thyroid enzymes were commonly seen in short- and long-term drinking water studies using sodium chlorate. The lowest reported LOAEL was 125 mg/L of

sodium chlorate (equivalent to 5 mg chlorate/kg bw per day) based on thyroid effects in male rats (NTP 2005). Rats were more sensitive to thyroid effects than mice in lifetime studies (NTP 2005). Studies in rats, mice and rabbits did not find any effect on reproductive parameters following exposure to sodium chlorate. Only one study showed a developmental effect, that is, increased crown-rump length. No neurodevelopmental studies were located in the published literature. Studies are provided in greater detail in Appendix C: Summaries of animal studies using chlorate and sodium chlorate.

Thyroid: The LOAEL for increased thyroid gland follicular cell hypertrophy was 125 mg/L (equivalent to 5 mg chlorate/kg bw per day) in rats and 2 000 mg/L (equivalent to 120 mg chlorate/kg bw per day) in mice. The NOAEL for mice was 1 000 mg/kg (60 mg/kg bw per day of chlorate). A NOAEL for rats could not be determined (NTP 2005).

In a two-year study conducted by the United States National Toxicology Program (NTP) (2005), rats were exposed to sodium chlorate in drinking water at 125, 1 000 or 2 000 mg/L (equivalent to 5, 35 or 75 and 5, 45 or 95 mg/kg bw per day of chlorate for males and females, respectively) while mice were exposed at 500, 1 000, or 2 000 mg/L (equivalent to 40, 80 or 160 and 30, 60 or 120 mg/kg bw per day of chlorate for males and females, respectively). Significantly increased thyroid gland follicular cell hypertrophy was seen in male rats at 125 mg/L and in females at 1 000 mg/L. Follicular cell mineralization was also significantly increased in female rats starting at 1 000 mg/L. In mice, thyroid gland follicular cell hypertrophy was significantly increased at 2 000 mg/L in females but was not observed in males (NTP 2005). The incidences of thyroid gland follicular cell adenoma and/or carcinoma exceeded the historical control ranges in rats of both sexes at 2 000 mg/L, but were not significantly different from the control group used in the study (NTP 2005). Thyroid hormone levels were measured in rats at 4 days, 3 weeks and 14 weeks but were not evaluated in mice. T4 and T3 levels were significantly reduced in rats of both sexes starting at 1 000 mg/L on day 4 and at 2 000 mg/L on week 3 but not on week 14. TSH levels were significantly increased at greater than or equal to 1 000 mg/L on day 4 in both sexes and on week 3 in males. At week 14, TSH levels were significantly higher in both sexes at 2 000 mg sodium chlorate/L only (NTP 2005). The LOAEL for increased thyroid gland follicular cell hypertrophy was 125 mg/L (equivalent to 5 mg chlorate/kg bw per day) in rats and 2 000 mg/L (equivalent to 120 mg chlorate/kg bw per day) in mice. The NOAEL for mice was 1 000 mg/kg (60 mg/kg bw per day of chlorate). An NOAEL for rats could not be determined (NTP 2005).

Hooth et al. (2001) gave sodium chlorate at 0.05, 1.0, 2.0, 4.0 or 6.0 g/L to rats and mice in drinking water for 105 days. In rats, thyroid gland colloid was significantly decreased, and the incidence and severity of follicular cell hyperplasia was significantly increased starting at 2.0 g/L. At 6.0 g/L, thyroid hypertrophy was also increased in rats. Thyroid histopathology was unaffected in mice. Similar thyroid effects were seen in male rats given sodium chlorate in drinking water for 90 days (Hooth et al. 2001). Decreased thyroid colloid and increased hypertrophy were seen starting at the lowest dose tested (0.001 g/L) of sodium chlorate while the incidences and severity of thyroid gland follicular cell hyperplasia was increased starting at 1.0 g/L.

In a one-generation study in which rats were dosed with 40 to 1 000 mg/kg bw per day of sodium chlorate (equivalent to 31 to 780 mg chlorate/kg bw per day), thyroid epithelial cell hyperplasia was seen in males starting at 200 mg/kg bw per day (equivalent to 156 mg chlorate/kg bw per day) and in females at 1 000 mg/kg bw per day (equivalent to 780 mg chlorate/kg bw per day). The NOAEL for thyroid effects was 40 mg/kg bw per day of sodium chlorate (equivalent to 31 mg chlorate/kg bw per day) (EFSA 2015). Thyroid follicular hyperplasia was also seen in F0 and F1 rats in a two-generation gavage study at the highest dose tested (390 mg chlorate/kg bw per day). The NOAEL was 10 and 70 mg sodium chlorate/kg bw per day for males and females, respectively (equivalent to 8 and 55 mg chlorate/kg bw per day) (EFSA 2015).

Thyroid gland colloid depletion was also seen in a 90-day rat study starting at doses of 3.0 mM of sodium chlorate (estimated as 30 and 42 mg chlorate/kg bw per day for males and females, respectively) in drinking water (McCauley et al. 1995).

Rats, but not mice, given up to 2 000 mg/L of chlorate in drinking water for 3 weeks had significantly increased incidences of thyroid gland follicular cell hypertrophy starting at 500 mg/L for males and 1 000 mg/L for females. This lesion did not occur in control rats (NTP 2005).

F344 rats given 10, 100, or 1 000 mg/L of sodium chlorate per day (equivalent to 0, 2.5, 11.9, 93.1 mg/kg bw of sodium chlorate) in drinking water for 7 days showed no effect on serum T4 or serum T3 levels (Khan et al. 2005). However, TSH was increased in the high-dose group. Thyroid follicular epithelial cell hypertrophy was statistically significant in all treated groups. The highest dose group showed thyroid colloid depletion and hyperplasia of follicular epithelial cells but these changes were not statistically significant (Khan et al. 2005).

Thyroid hormone levels were unaffected in monkeys given 25 to 400 mg/L of sodium chlorate (equivalent to 4–58.4 mg chlorate/kg bw per day) for 30 to 60 days (Bercz et al. 1982).

Hematology: In a two-year National Toxicology Program (NTP) study using rats and mice, male rats given sodium chlorate at 2 000 mg/L (equivalent to 75 mg chlorate/kg bw per day) had increased hematopoietic cell proliferation in spleen and hyperplasia in bone marrow at greater than or equal to 1 000 mg/L (equivalent to 35 mg chlorate/kg bw per day). Bone marrow hyperplasia was also increased in female mice exposed to 500 to 2 000 mg/L of sodium chlorate (equivalent to 30 to 120 mg/kg bw per day of chlorate) in drinking water for two years. The LOAEL for bone marrow hyperplasia was 30 mg chlorate/kg bw per day for female mice and 35 mg chlorate/kg bw per day for male rats. Hematological evaluations were not conducted (NTP 2005).

In 90-day studies, rats given 3.0 to 48.0 mM of sodium chlorate/kg bw per day (equivalent to 30 to 800 mg chlorate/kg bw per day) in drinking water or gavaged with 19 to 1 000 mg/kg bw per day of sodium chlorate (equivalent to 8 to 788 mg/kg bw per day of chlorate) showed decreased

hematocrit, red blood cell counts and hemoglobin (Bio/dynamics 1987a; McCauley et al. 1995). The NOAEL and LOAEL were 100 and 510 mg/kg bw per day of chlorate in the drinking water study and 79 and 788 mg/kg bw per day of chlorate in the gavage study.

In a 3-week study, rats and mice were given 125 to 2 000 mg/L of sodium chlorate in drinking water. Decreased hematocrit and red blood cell counts were seen in rats while decreased hemoglobin was seen in both rats and mice (NTP 2005). The LOAELs based on hemoglobin changes were 300 and 350 mg chlorate/kg bw per day for male rats and male mice, respectively. Absolute and relative heart weights were also decreased in male rats at 2 000 mg/L (equivalent to 300 mg chlorate/kg bw per day) but not in mice. Rats of both sexes had a dose-dependent decrease in segmented neutrophils starting at 125 mg/kg bw per day of sodium chlorate (equivalent to 20 mg/kg bw per day of chlorate); however, the cause for the decrease could not be determined by the authors (NTP 2005).

In a series of studies, small groups of male rats were given doses of 10 or 100 mg/L of chlorate in drinking water for up to 12 months (Abdel-Rahman et al. 1980; 1984b; Couri and Abdel-Rahman 1980). Blood samples were taken at various intervals throughout each study. In a 4-month study by Abdel-Rahman et al. (1980), methemoglobin was not detected at either 2 or 4 months. Red blood cell counts, hematocrit and hemoglobin were decreased at both doses after 9 months of dosing (Abdel-Rahman et al. 1984b). In general, GSH levels were significantly decreased at up to 9 months of dosing (Abdel-Rahman et al. 1980; 1984b). However, Couri and Abdel-Rahman (1980) found that GSH concentrations were increased after 12 months of dosing.

Reproductive: Chlorate had no effect on reproductive parameters in either rats or rabbits or on sperm morphology in mice. No effects on maternal survival or body weight, pregnancy rate, number of implantations, or number of live, resorbed or dead fetuses were seen in female rats given either 1 or 10 mg/L of chlorate in drinking water for 10 weeks pre-mating until GD 20 (Suh et al. 1983) or in female rats gavaged with 10 to 1 000 mg/kg bw per day of sodium chlorate (equivalent to 7.8 to 780 mg chlorate/kg bw per day) on GD 6 to 15 (Bio/dynamics 1987b). Reproductive effects were also absent in rats gavaged with 40 to 1 000 mg/kg bw per day of sodium chlorate (equivalent to 31 to 780 mg chlorate/kg bw per day) starting at 6 weeks of age until end of mating in males and until end of lactation in females in a one-generation study (Gaoua, 2004a). Effects on female reproductive parameters or on sperm parameters were not seen in either F0 or F1 rats in a two-generation reproductive study using doses of 10 to 500 mg/kg bw per day of sodium chlorate (equivalent to 8 to 390 mg chlorate/kg bw per day) (Gaoua, 2004b). Maternal weight and body weight gain as well as gravid uterine weights were unaffected in rabbits gavaged with sodium chlorate doses of 100 to 475 mg/kg bw per day (equivalent to 78 to 372 mg chlorate/kg bw per day) from GD 6 to 29 (NTP 2005).

Chlorate did not cause sperm head abnormalities in male B6C3F1 mice (Meier et al. 1985).

Developmental: Crown-rump length was significantly increased in male fetuses of female rats given 10 mg/L of chlorate (equivalent to 1 mg chlorate/kg bw per day) in drinking water for 10 weeks pre-mating until GD 20 while the incidences of external, visceral or skeletal

malformations were unaffected (Suh et al. 1983). The incidences of fetal malformations were also unaffected in the prenatal gavage study using rabbits and rats (Bio/dynamics 1987b; NTP 2002). Lower fetal body weights and decreased body weight gain were seen at the highest dose tested (equivalent to 780 mg chlorate/kg bw per day) in a one-generation study in which rats were gavaged with 40 to 1 000 mg/kg bw per day of sodium chlorate (Gaoua, 2004a). Survival and development were unaffected in both progeny of F0 and F1 rats in a two-generation gavage study using doses of up to 500 mg sodium chlorate/kg bw per day (equivalent to 390 mg chlorate/kg bw per day) (Gaoua, 2004b).

2.2.2.4 Chlorine dioxide

Toxicological effects observed for ClO_2 are consistent with those observed for its primary metabolites, chlorite and, to a lesser extent, chlorate. Overall, ClO_2 consistently affected thyroid function as well as development and neurodevelopment in rat pups with reported lowest LOAELs of 100 mg/L (equivalent to 14 mg/kg bw per day) and 5 mg/kg bw per day of ClO_2 for altered thyroid hormones and decreased vaginal weights in pups, respectively (Carlton et al. 1991; Orme et al. 1985). However, thyroid effects in adult animals were mostly sporadic or absent in adult rats and were reversible in adult monkeys (Bercz et al. 1982; Carlton et al. 1991). Hematological effects were absent in studies using monkeys and mice (Bercz et al. 1982; Moore and Calabrese 1982) but were present in rats in a study by Abdel-Rahman et al. (1984b). Liver effects were not seen in monkeys but were seen in rats and included altered liver enzymes and decreased liver weights. However, liver weight changes were mostly associated with decreased water intake and decreased body weights (Bercz et al. 1982; Daniel et al. 1990; Suh et al. 1984). The only effect seen in a chronic 2-year study in rats was a decrease in mean life span that occurred at the highest dose tested (Haag 1949). Studies are provided in greater detail in Appendix D: Summaries of animal studies using chlorine dioxide.

Hematology: Hematological and circulatory effects were inconsistently seen in monkeys, mice and rats. The lowest LOAEL for hematological effects was 1 mg/L of ClO_2 (equivalent to 0.1 mg/kg bw per day) based on statistically significant, dose-dependent decreased hematocrit and hemoglobin in a 12-month drinking water study in rats (Abdel-Rahman et al. 1984b). At 10 and 100 mg/L of ClO_2 (equivalent to 1 and 10 mg/kg bw per day), increased mean corpuscular hemoglobin concentration and decreased red blood cell counts were also seen. However, statistically significant changes in other hematological parameters were inconsistent over time and did not appear to be dose related (Abdel-Rahman et al. 1984b). The same study showed decreased osmotic fragility of red blood cells, indicative of unusually flattened red cells, which decreases the volume-to-surface area ratio, starting at 10 mg/L per day (estimated at 1 mg/kg bw per day) but mean corpuscular volume (MCV) was unaffected (Abdel-Rahman et al. 1984b). A previous study by the same authors found no effect on osmotic fragility in rats exposed to much higher doses (up to 1 000 mg/L per day; estimated at 342 mg/kg bw per day) of ClO_2 for 4 months (Abdel-Rahman et al. 1980). Daniel et al. (1990) reported decreased spleen weights in female rats given 25-200 mg/L (calculated as 2.4 to 14.9 mg/kg bw per day) of ClO_2 in drinking water for 90 days but not males, and no clear, dose-dependent hematological changes were seen. Drinking water intake was also statistically decreased in all treatment groups (Daniel et al. 1990).

ClO_2 had no effect on blood parameters, such as methemoglobin levels, hematocrit, osmotic fragility, hemoglobin levels, red blood cell counts and mean corpuscular hemoglobin concentration, in African green monkeys given 30 or 100 mg/L (calculated as 3.5 or 9.0 mg/kg bw per day) of ClO_2 in drinking water for up to 60 days (Bercz et al. 1982; Harrington et al. 1986), or in mice given 100 ppm of ClO_2 (equivalent to 15 mg/kg bw per day) in drinking water for 30 days (Moore and Calabrese 1982). Methemoglobin levels, blood glutathione and osmotic fragility were also unaffected in male Sprague-Dawley rats given up to 1 000 mg/L of ClO_2 in drinking water for 4 months (Abdel-Rahman et al. 1980).

Liver: Liver effects were observed in male rats but not in female rats or African green monkeys. Liver weight was decreased in male Sprague-Dawley rats receiving greater than or equal to 50 mg/L (equivalent to greater than or equal to 3.6 mg/kg bw per day) of ClO_2 in drinking water for 90 days but was accompanied by decreased water intake (Daniel et al. 1990). At greater than or equal to 100 mg/L (equivalent to greater than or equal to 6.2 mg/kg bw per day), liver enzymes were altered in males (Daniel et al. 1990). Drinking water intake was also decreased in female rats but liver weights and enzyme levels were unaffected. At the highest dose tested (200 mg/L; equivalent to 11.5 and 14.9 mg/kg bw per day in males and females, respectively), decreased final body weights and body weight gain were seen in both sexes, while food intake was decreased in males (Daniel et al. 1990). ClO_2 altered liver enzymes in male rats. It caused a statistically significant decrease in aminopyrine demethylase, which was not dose-dependent, at the mid-dose only (10 mg/L; calculated as 1 mg/kg bw per day) and a statistically significant and dose-dependent increased aniline hydroxylase at the high dose (Suh et al. 1984). Liver weight and function were unaffected in monkeys dosed up to 100 mg/L (equivalent to 9.5 mg/kg bw per day) of ClO_2 in drinking water for up to 60 days (Bercz et al. 1982).

Thyroid: Statistically significant decreased T4 and increased T3 levels were observed in rat pups indirectly exposed to 100 mg/L (calculated as 14 mg/kg bw per day) of ClO_2 via gestation and lactation. However, thyroid hormone levels were unaffected in treated dams (Orme et al. 1985).

In a one-generation study, adult male, but not female, Long-Evans rats gavaged with 10 mg/kg bw per day of ClO_2 had significantly lower T4 levels. Sporadic changes in T3 and T4 levels were observed at 2.5 and 5.0 mg/kg bw per day; however, the changes lacked statistical significance and were not dose-dependent. At 10 mg/kg bw per day, T4 levels were significantly increased in male pups on postnatal day (PND) 17 but not on PND 28 or PND 40 (Carlton et al. 1991).

ClO_2 given in drinking water for 30 to 60 days caused reversible decreases in T4 levels (at greater than or equal to 30 mg/L, calculated 3.5 mg/kg bw per day) in adult monkeys (Bercz et al. 1982).

Developmental: In a neurodevelopmental study in rats given ClO_2 at doses of 2 to 100 mg/L (equivalent to 1 to 14 mg/kg bw per day) in drinking water, locomotor levels in the pups were consistently lower than controls. Although the decrease was not statistically significant, it was seen at 100 mg/L, a dose that also caused statistically significant changes in thyroid hormone levels in pups. However, thyroid hormone levels were unaffected in treated dams (Orme et al.

1985). The authors attributed the lack of significance to the degree of variability between control litters. In the same study, pups from untreated dams were gavaged with 14 mg/kg bw per day of ClO_2 from PND 5 to 20. They had statistically significant decreased T4 levels and depressed locomotor activity compared to control pups. Age of eye opening and pup birth weight were unaffected by route of exposure or dose level (Orme et al. 1985). The study used a positive control group dosed with propylthiouracil, a thyroid hormone suppressant, to assess neonatal hypothyroidism. Depressed thyroid hormone levels, delayed eye opening and decreased body weights and activity were seen in litters from the positive control group (Orme et al. 1985).

Litter size, pup viability and pup weights were unaffected in a one-generation study in which parental rats were gavaged with 2.5 to 10.0 mg/kg bw per day of ClO_2 (Carlton et al. 1991). All measured parameters in the F1 generation were unaffected by treatment, except vaginal weights, which were decreased in the high-dose group. The authors could not readily explain this observation (Carlton et al. 1991). Decreased median day of eye opening was also seen in high-dose pups but the effect was not dose-dependent and, according to the authors, lacked biological significance (Carlton et al. 1991).

Reproductive: ClO_2 had no effect on reproductive parameters in parental rats gavaged with 2.5 to 10.0 mg/kg bw per day of ClO_2 in a one-generation study (Carlton et al. 1991). In male B6C3F1 mice, ClO_2 did not cause sperm head abnormalities (Meier et al. 1985).

2.3 Genotoxicity and carcinogenicity

Chlorite, chlorate and ClO_2 are unlikely to be genotoxic or carcinogenic at levels that caused the non-carcinogenic effects reported in section 2.2.2.

Overall, there was no evidence of genotoxicity in in vivo oral animal studies using either chlorite, chlorate or ClO_2. With the exception of chlorite, in vitro mutagenicity studies showed no evidence of mutagenicity. There was no evidence of carcinogenicity reported for sodium chlorite at doses of up to 71 mg chlorite/kg bw per day in mice and 40.9 mg chlorite/kg bw per day in rats (Kurokawa et al. 1986). NTP (2005) concluded that sodium chlorate exposure resulted in non-neoplastic lesions in the thyroid and bone marrow and had equivocal effects on pancreatic islet cell adenoma and carcinoma. ClO_2 did not increase the incidence of tumours in a two-year drinking water study in rats given up to 100 mg/L (equivalent to 13 mg/kg bw per day) of ClO_2 in drinking water (Haag 1949).

In a study by Hayashi et al. (1988), both sodium chlorite and ClO_2 induced micronucleated polychromatic erythrocytes (MNPCE), but not polychromatic erythrocytes (PCE), in bone marrow when given as single intraperitoneal injections to mice. Except for a slight decrease seen at the highest dose tested, the frequencies of MNPCEs were statistically significant increased in a dose-dependent manner at all doses of ClO_2 (3.2 to 25 mg/kg bw) used. For sodium chlorite, only the mid-dose levels (15 and 30 mg/kg bw) showed statistically significant increased MNPCEs but not at the highest dose tested (60 mg/kg bw). Further testing of sodium

chlorite at doses of 37.5 to 300 mg/kg bw via gavage by the authors did not produce positive results. Similarly, a study by Meier et al. (1985) showed sodium chlorite, sodium chlorate and ClO_2 given to mice as a single dose (up to 400 mg/L for ClO_2 and 1 000 mg/L for chlorite and chlorate) via gavage did not cause abnormalities in sperm heads or chromosomal aberrations, or increase the frequency of micronuclei or PCE in bone marrow. Sodium chlorate given in drinking water to mice for 3 weeks did not increase the frequency of micronucleated normochromic erythrocytes in peripheral blood or PCEs (NTP 2005).

In an Ames test, both sodium chlorite and ClO_2 caused reverse mutations in only one of six strains of *Salmonella typhimurium* (TA100) tested. Sodium chlorite tested positive with metabolic activation while ClO_2 was positive without metabolic activation (Ishidate et al. 1984). Sodium chlorate was not mutagenic in five strains of *S. typhimurium*, including TA100, in the absence of metabolic activation (NTP 2005).

Sodium chlorite, but not ClO_2, tested positive in chromosomal aberrations tests using Chinese hamster fibroblast cells (Ishidate et al. 1984). In a cytogenetics assay using Chinese hamster ovary cells (CHO), sodium chlorite increased chromosomal aberrations both with and without metabolic activation (Ivett and Myhr 1986). Sodium chlorite also increased mutation frequency in a mouse lymphoma forward mutation assay (using L5178Y $TK^{+/-}$) both with and without metabolic activation but only at doses that were cytotoxic (Cifone and Myhr 1986).

No significant increase in tumours was seen in a carcinogenicity study in which sodium chlorite was administered in drinking water to B6C3F1 mice (50/sex/dose) at doses up to 500 ppm (equivalent to 71 mg chlorite/kg bw per day) and to F344 rats (50/sex/dose) at doses up to 600 ppm (equivalent to 40.9 mg chlorite/kg bw per day) for 85 weeks. Although treated male mice exhibited an increased incidence of lung and liver tumours, tumour rates were within historical ranges for control mice. Furthermore, increases in liver tumours were seen only for benign tumours and did not display a typical dose-response pattern (Kurokawa et al. 1986). Harrington et al. (1995b) found squamous epithelial hyperplasia with hyperkeratosis, ulceration, chronic inflammation and edema in the stomachs of rats dosed with 59.7 mg/kg bw per day of chlorite, which they attributed to the irritant property of sodium chlorite.

Exposure to sodium chlorate for two years in drinking water resulted in non-neoplastic lesions in the thyroid gland of male and female rats and female mice, bone marrow of male rats and female mice, and spleen of male rats (NTP 2005). Groups of rats and mice were given sodium chlorate in drinking water at doses of 125, 1 000 or 2 000 mg/L and 500, 1 000 or 2 000 mg/L, respectively, for 2 years. There were positive trends in the incidences of thyroid gland follicular cell carcinoma in male rats and in follicular cell adenoma or carcinoma (combined) in both male and female rats with the incidences of follicular cell adenoma or carcinoma (combined) in the 2 000 mg/L group exceeding the historical control ranges. The incidences of follicular cell hypertrophy were increased in male rats at all dose levels, in female rats starting at 1 000 mg/L and in female mice at 2 000 mg/L. Changes in thyroid hormone levels were also seen in rats. The NTP considered these findings to show “some evidence of carcinogenic activity” in rats based on increased incidences of thyroid gland neoplasms. In mice, there was a positive, albeit

marginal, trend in the incidences of pancreatic islet cell adenoma or carcinoma in female mice with the incidences exceeding historical controls in the 2 000 mg/L group. However, the decreased incidences of hyperplasia with increasing dose did not support this effect and the NTP concluded that the effect provided "equivocal evidence of carcinogenic activity". The incidences of hepatocellular carcinoma were also significantly increased in female mice at 500 and 1 000 mg/L but not at 2 000 mg/L, but, when combined with the incidences of hepatocellular adenoma, there was no effect. The NTP concluded that sodium chlorate did not induce hepatocellular carcinomas but caused non-neoplastic lesions in the thyroid, bone marrow and spleen (NTP 2005).

ClO_2 did not increase the incidence of tumours in a 2-year drinking water study in rats given up to 100 mg/L (equivalent to 13 mg/kg bw per day) of ClO_2 in drinking water (Haag 1949). Although hyperplasia and inflammation of the nasal turbinates were observed in a 90-day study in which Sprague-Dawley rats (10/sex/dose) were given ClO_2 in drinking water at concentrations ranging from 0 to 200 mg/L (0 to 12 mg/kg bw per day for males and 2 to 15 mg/kg bw per day for females), the effects were likely caused from inhalation of ClO_2 vapours rather than ingestion of the drinking water (Daniel et al. 1990).

The International Agency for Research on Cancer has not assessed chlorate or ClO_2 for carcinogenicity. It considers sodium chlorite as "Group 3 – not classifiable" as to its carcinogenicity to humans (IARC 1991). The U.S. EPA has classified both chlorite and ClO_2 as "Class D – not classifiable as to human carcinogenicity" but has not assessed chlorate for carcinogenicity (U.S. EPA 2018).

2.4 Mode of action

Chlorite, chlorate and ClO_2 can affect the thyroid by interfering with the uptake of dietary iodine in the gastrointestinal tract (Bercz et al. 1986). As strong oxidizers, they can oxidize dietary iodide to a reactive species that binds to tissues of the digestive tract preventing its absorption and creating a state of iodine deficiency. Iodine deficiency leads to a decrease in T4 and to a lesser extent, in T3 thereby increasing TSH. Overstimulation of the thyroid by TSH can cause morphological changes in the thyroid leading to goitre in humans and follicular cell hypertrophy and/or hyperplasia in rodents (Huisinga et al. 2020; Zimmermann and Boelaert 2015).

Thyroid hormones are also essential to the normal development of the brain and nervous system (ATSDR 2004). T4 deficiencies during critical periods of fetal development can cause delayed maturation of neuronal and glial cells in the neonatal brain, which can lead to behavioural impairments, including motor skills and activity, and delayed eye opening and decreased body weights (Orme et al. 1985).

The liver plays an important role in maintaining thyroid hormone levels by activating and inactivating thyroid hormones, and transporting and metabolizing them. Conversely, thyroid hormones impact the activities of the liver (Piantanida et al. 2020). Xenobiotics have been shown to induce liver enzymes in rats increasing the rate of T4 and T3 metabolism and

elimination. The result is decreased serum T4 and increased serum TSH concentrations (Huisinga et al. 2020).

Hematological effects of ClO_2, chlorite and chlorate are related to their strong oxidizing properties (ATSDR 2004). Sodium chlorite has been shown to increase reactive oxygen species that damage the membrane and cellular components of red blood cells (Ali and Mahmood 2017).

2.5 Selected key studies

The epidemiological database is insufficient to use as the basis for a risk assessment for either chlorite, chlorate or ClO_2 or to identify populations that may be disproportionately impacted. Most available human studies were related to ClO_2 exposure and did not quantify the exposure but compared it to other drinking water treatment methods. Although a few volunteer studies detailed the amounts ingested, they showed no change to hematological parameters, clinical chemistry or physical symptoms (Lubbers et al. 1981; 1982). Their short durations (12 weeks), limited measured outcomes and lack of adverse effects make them unsuitable to assess any risks from chronic exposure.

2.5.1 Chlorite

Based on animal and in vitro studies, chlorite is not likely to be genotoxic or carcinogenic at levels causing non-carcinogenic effects (see section 2.3). Adverse effects on development, including neurodevelopment, as well as liver weight changes, were identified in animal studies as the critical effects and were observed at doses lower than those causing hematological or reproductive effects (see section 2.2.2). The rat was the most sensitive species (CMA 1996; Couri et al. 1982b; Gill et al. 2000; Mobley et al. 1990). Chlorite also affected thyroid hormone levels in pups but not adults (Bercz et al. 1982; Carlton et al. 1987; CMA 1996; Gill et al. 2000; Harrington et al. 1995a; Mobley et al. 1990). Mode of action information (see section 2.4), including decreased iodine uptake and liver effects leading to changes in thyroid hormone levels, support the selection of the critical endpoints—neurodevelopmental and developmental effects seen in rat pups and altered liver weights in F0 and F1 animals (Marty et al. 2022; Piantanida et al. 2020).

A two-generation study showing neurodevelopmental and developmental effects, as well as changes in liver weights, was selected as the key study (CMA 1996; Gill et al. 2000). It reported a NOAEL of 35 ppm of sodium chlorite (equivalent to 2.9 mg/kg bw per day of chlorite) and a LOAEL of 70 ppm of sodium chlorite (equivalent to 5.9 mg chlorite/kg bw per day). This LOAEL was the lowest LOAEL available in the literature for developmental and neurodevelopmental effects. It was based on developmental delays in F1 and F2 pups, including lower auditory startle amplitude, delayed preputial separation and decreased absolute brain weight, and on decreased altered liver weights in F0 and F1 generations. The study tested a range of outcomes in both pups and parents, including neurodevelopmental and developmental endpoints, and administered sodium chlorite via the most relevant route of exposure—drinking water.

In a two-generation study, the parental generation (F0) of Sprague-Dawley rats (30/sex/dose) were given drinking water containing 0, 35, 70 or 300 ppm of sodium chlorite (equivalent to 0, 3.0, 5.6 or 20.0 mg chlorite/kg bw per day for males and 0, 3.8, 7.5 or 28.6 mg chlorite/kg bw per day for females) for 10 weeks and then mated. Males were exposed throughout mating, then sacrificed (CMA 1996; Gill et al. 2000). Exposure for females continued throughout mating, pregnancy and lactation until necropsy following weaning of their litters. Twenty-five males and females from each of the first 25 litters to be weaned in a treatment group were chosen to produce the F1 generation. The F1 pups were continued on the same treatment regimen as their parents (equivalent to 0, 2.9, 5.9 or 22.7 mg chlorite/kg bw per day for males and 0, 3.8, 7.9 or 28.6 mg chlorite/kg bw per day for females). At approximately 14 weeks of age, F1 rats were mated to produce the F2a generation. Because of a reduced number of litters in the 70 mg/L F2a generation, F1 animals were remated following weaning of the F2a generation to produce the F2b generation. F2a and F2b generations were maternally exposed to sodium chlorite only and did not receive treatment beyond weaning at PND 21. Animals used for neurotoxicological testing were dosed until weaning and the study was terminated on PND 60 after final testing of F2b animals.

No effects were seen in the functional observation battery, motor activity (figure-of-eight), or swim maze learning that were assessed in F1 pups only. Neuropathology was also assessed in F1 pups, but no gross or macroscopic lesions in the brains or spinal cords were found. No changes were seen in T3 or T4 levels in F1 parents or pups. Thyroid hormone levels were not assessed in either the F0 or F2 generations.

Acoustic startle habituation testing was performed on F2b pups. No effects were seen on maximum startle response; however, maximum response amplitude was significantly decreased in both sexes starting at 70 ppm on PND 24 but not 60. The discontinuation of dosing at PND 21 may have played a role in the lack of effects seen on PND 60. Decreased absolute brain weights were seen in F1 and F2 pups of both sexes starting at 70 ppm.

In F1 pups, preputial separation was delayed starting at 70 ppm while vaginal opening was delayed at 300 ppm. Eye opening was delayed in F2a pups at 300 ppm. Chlorite had no effect on anogenital distance, gross external malformations or timing of ear opening in F1, F2a and F2b pups. Although statistically significant hematological changes were seen in F1 pups and adults starting at 35 ppm, they were within historical ranges for the 35 and 70 ppm groups. Pup body weights were decreased at 300 ppm for both sexes and in F1, F2a and F2b pups. The study NOAEL was 35 ppm of sodium chlorite (equivalent to 2.9 mg/kg bw per day of chlorite) while the LOAEL was 70 ppm sodium chlorite (5.9 mg/kg bw per day of chlorite) and were based on neurodevelopmental toxicity (lower auditory startle amplitude in F2b pups, decreased absolute brain weights in F1 and F2 pups), developmental delays (delayed preputial separation in F1 male pups) and decreased absolute and relative liver weights in F0 females and F1 males and females.

Neurodevelopmental effects seen in CMA (1996) and Gill et al. (2000) are supported by a one-generation study by Mobley et al. (1990) that had very similar NOAEL and LOAEL values. In that

study, rat pups exposed to 3 and 6 mg chlorite/kg bw per day had decreased exploratory activity post-conception days 36-37 and 36-39, respectively. The delays were considered slight at 3 mg/kg bw per day and significant at 6 mg/kg bw per day. The NOAEL was 3 mg chlorite/kg bw per day and LOAEL was 6 mg chlorite/kg bw per day. Developmental delays, including decreased pup weights and growth weights, and changes in fetal crown-rump length were seen at the lowest dose tested in other reproductive/developmental studies (Couri et al. 1982b; Moore et al. 1980; Suh et al. 1983).

The thyroid hormones play an important role in neurodevelopment (Marty et al. 2022; Orme et al. 1985). Changes in pup thyroid levels support the selection of neurodevelopmental toxicity as one of the key endpoints. Studies by Carlton et al. (1987) and Mobley et al. (1990) showed chlorite altered thyroid hormone levels of rat pups (NOAELs of 0.75 and 3 mg chlorite/kg bw per day and LOAELS of 75 and 6 mg chlorite/kg bw per day, respectively). Alterations in T3 and T4 levels were not seen in the key study; however, their levels were not measured in F2b rats undergoing auditory startle testing and were only measured in F1 pups on PND 25 and parents at 13 weeks of age. Studies showed changes in thyroid hormone levels sampled at different time periods than the key study, which may have impacted the results (Carlton et al. 1987; Mobley et al. 1990). Additionally, pups were dosed continuously with chlorite in the studies showing thyroid hormone level changes alone or in conjunction with neurobehavioural testing, contrary to pups undergoing neurobehavioural testing in the key study, which were treated with sodium chlorite only until weaning (Gill et al. 2000).

2.5.2 Chlorate

Chlorate was not a reproductive toxicant and was not mutagenic or genotoxic in in vitro and in vivo studies and caused only slight developmental toxicity (Section 2.2.2.3). The NTP (2005) concluded that sodium chlorate showed “some evidence of carcinogenic activity” based on increased incidences of thyroid gland neoplasms in rats of both sexes. Thyroid gland follicular tumours are common in rats, particularly in males, and likely result from increased TSH production and overstimulation of the thyroid (Huisinga et al. 2020). A similar mode of action has not been seen in humans. Based on available data, no chemical is known to increase the incidence of thyroid tumours in humans by overstimulating TSH production (Bartsch et al. 2018). Rodent follicular thyroid tumours are not relevant to humans (Bartsch et al. 2018; Huisinga et al. 2020). However, thyroid hyperplasia and hypertrophy have been observed in both rats and humans (Bartsch et al. 2018; Lewandowski et al. 2004).

Thyroid and hematological effects were consistently seen in both short- and long-term animal studies. Thyroid effects occurred at lower doses than hematological effects. Increased incidences and severity of thyroid gland follicular cell hypertrophy, mineralization, hyperplasia and decreased thyroid colloid were reported in published literature, in addition to changes in thyroid levels. Thyroid gland follicular cell hypertrophy was selected as the critical effect and the 2-year NTP (2005) was selected as the critical study. Rats were more sensitive to thyroid effects than mice, and male rats were more sensitive than female rats (NTP 2005). Thyroid hyperplasia was also seen in one- and two-generation studies using rats, with NOAELs of 156 and 390 mg chlorate/kg bw per day, respectively (Gaoua, 2004a; 2004b). The lowest available LOAEL for

thyroid effects was 5 mg/kg bw per day of chlorate for male rats and 120 mg/kg per day of chlorate for female mice (NTP 2005).

In the 2-year study conducted by NTP (2005), rats were exposed to sodium chlorate in drinking water at 125, 1 000 or 2 000 mg/L (equivalent to 5, 35 or 75 and 5, 45 or 95 mg/kg bw per day of chlorate for males and females, respectively), while mice were exposed at 500, 1 000, or 2 000 mg/L (equivalent to 40, 80 or 160 and 30, 60 or 120 mg/kg bw per day of chlorate for males and females, respectively). Significantly increased thyroid gland follicular cell hypertrophy was seen in male rats at 125 mg/L and in females at 1 000 mg/L. Follicular cell mineralization was also significantly increased in female rats starting at 1 000 mg/L. In mice, thyroid gland follicular cell hypertrophy was significantly increased at the top dose of 2 000 mg/L in females but not males (NTP 2005). The incidences of thyroid gland follicular cell adenoma and/or carcinoma exceeded the historical control ranges in rats of both sexes at 2 000 mg/L, but were not significantly different from the control group used in the study (NTP 2005). Thyroid hormone levels were measured in rats at 4 days, 3 weeks and 14 weeks but were not evaluated in mice. T4 and T3 levels were significantly reduced in rats of both sexes starting at 1 000 mg/L on day 4 and at 2 000 mg/L on week 3 but not on week 14. TSH levels were significantly increased at greater than or equal to 1 000 mg/L on day 4 in both sexes and on week 3 in males. At week 14, TSH levels were significantly higher in both sexes at 2 000 mg sodium chlorate/L only (NTP 2005). The LOAEL for increased thyroid gland follicular cell hypertrophy was 125 mg/L (equivalent to 5 mg chlorate/kg bw per day) in rats and 2 000 mg/L (equivalent to 120 mg chlorate/kg bw per day) in mice. The NOAEL for mice was 1 000 mg/kg (60 mg/kg bw per day of chlorate). A NOAEL for rats could not be determined (NTP 2005).

2.5.3 Chlorine dioxide

ClO_2 caused developmental effects in rat pups exposed perinatally, including lowered locomotor levels, decreased vaginal weights, increased fetal weights and altered T4 levels (Carlton et al. 1991; Orme et al. 1985). However, at low doses, there will be minimal human exposure to ClO_2 because it is rapidly converted to chlorite, chlorate and chloride.

Rat pups directly exposed to 14 mg/kg bw per day of ClO_2 via gavage had signs of neonatal hypothyroidism, including decreased body weights, locomotor activity and T4 levels. Pups indirectly exposed via maternal exposure to 100 mg/L (calculated as 14 mg/kg bw per day) of ClO_2 showed statistically significant decreased T4 and increased T3. Although locomotor activity was depressed, the change was not statistically significant. Altered thyroid function in the indirectly exposed pups was seen in the absence of measurable effects in the dam. Therefore, it appears that thyroid function of neonatal rats is more sensitive to the effects of ClO_2 than adults. The NOAEL and LOAEL were 3 and 14 mg/kg bw per day of ClO_2, respectively (Orme et al. 1985).

3.0 Derivation of the health-based values (HBVs)

3.1 Chlorite

The most critical effects were developmental delays in F1 and F2 pups, including lower auditory

startle amplitude, delayed preputial separation, decreased absolute brain weight and decreased altered liver weights in F0 and F1 generations, which were seen at the lowest dose tested in a two-generation rat study. The study NOAEL of 2.9 mg/kg bw per day was selected as the point of departure (POD). It is the same key study and approach used by Health Canada's PMRA to calculate an acceptable daily intake (ADI) (CMA 1996; Gill et al. 2000).

An intraspecies uncertainty factor of 10 was used to account for variability between the average human response and the response of the most sensitive individuals. An interspecies uncertainty factor of 10 was also used. The key study spans two generations and evaluated the entire developmental and reproductive life. Furthermore, the database contains chronic (2-year) and long-term (85-week) studies; therefore, an uncertainty factor to account for the use of a less-than-lifetime study was unnecessary. A total uncertainty factor of 100 (10 intraspecies X 10 interspecies) was considered appropriate. Using the NOAEL of 2.9 mg/kg bw per day of chlorite and the total uncertainty factor of 100, an ADI can be calculated as follows:

$$\text{ADI for chlorite} = (2.9\ \text{mg/kg bw per day})/100$$

$$\text{ADI for chlorite} = 0.029\ \text{mg/kg bw per day}$$

where:

- 2.9 mg/kg bw per day is the NOAEL based on lower startle amplitude, delayed preputial separation, decreased absolute brain weight and altered liver weights in a two-generation rat study (CMA 1996; Gill et al. 2000)
- 100 is the uncertainty factor, comprising 10 for interspecies and 10 for intraspecies differences

The ADI above of 0.029 mg/kg bw per day and source allocation factor of 80% (see section 1.3), along with adult values for body weight and drinking water intake, were used to calculate an HBV. Although the critical effects include neurodevelopmental and developmental endpoints, the selection of adult values is justified based on:

- the HBV set for chronic, lifetime exposure
- decreased liver weights seen in adult animals (F0)
- reduced brain weight and slight delays in preputial separation that may have been associated with decreased pup body weights (Joint FAO/WHO 2008)
- decreased maximum response amplitude seen on PND 24 but not PND 60, which may have been caused by:
    - discontinuation of dosing at PND 21 for animals undergoing acoustic startle habituation testing
    - perturbed habituation in the control animals (Joint FAO/WHO 2008), and/or
    - reversibility of effect following discontinuation of dosing
- the lack of chlorite in infant formula and breast milk (see section 1.3.2)

Additionally, the use of adult values is in keeping with the approach used by other national and

international organizations that set their guideline values based on the CMA (1996) and Gill et al. (2000).

Using the ADI, the HBV can be calculated:

$$\text{HBV for chlorite} = (0.029\ \text{mg/kg bw per day} \times 74\ \text{kg} \times 0.80)/(1.53\ \text{L/day})$$

$$\text{HBV for chlorite} = 1\ \text{mg/L (rounded)}$$

where:

- 0.029 mg/kg bw per day is the ADI for chlorite
- 74 kg is the average body weight of an adult human (Health Canada 2021)
- 0.80 is the allocation factor as drinking water is the main source of exposure (Krishnan and Carrier 2013)
- 1.53 L/day is the drinking water intake of an adult human (Health Canada 2021)

3.2 Chlorate

Health Canada's PMRA has established an ADI of 0.03 mg/kg bw per day for chlorate based on increased thyroid gland follicular cell hypertrophy and follicular cell mineralization in a chronic rat study (NTP 2005). The study LOAEL was 5 mg/kg bw per day. A NOAEL was not identified in the study; therefore, a benchmark dose analysis was performed and a benchmark dose level ($BMDL_{10}$) of 28 mg sodium chlorate/L (equivalent to 22 mg chlorate/L) was calculated, which corresponds to an oral chlorate dose of 0.9 mg/kg bw per day (U.S. EPA 2016). The interspecies default uncertainty factor of 10 can be subdivided into subfactors of 3 (rounded from 3.16) each for toxicokinetics and toxicodynamics. Because of several important quantitative differences between rats and humans with respect to thyroid function, such as the half-life of T4 (12 hours in rats vs 5 to 9 days in humans), the toxicodynamics subfactor can be reduced to 1, resulting in an interspecies uncertainty factor of 3 (Bartsch et al. 2018; Dellarco et al. 2005; Döhler et al. 1979; U.S. EPA 2016). The default intraspecies uncertainty factor of 10 was also used, resulting in an uncertainty factor of 30 (3 for interspecies and 10 for intraspecies was used) (Health Canada 2008a).

The database includes subchronic, chronic, developmental and reproductive studies but lacks neurodevelopmental studies. Chlorate decreases iodine uptake, potentially causing thyroid hormone deficiencies. Thyroid hormones are important in fetal and neonatal neurodevelopment (see section 2.4). In fetuses, thyroid hormones are initially supplied from the mother via the placenta, while neonates synthesize their own hormones (EFSA 2015). Neonates may be particularly susceptible to chemicals disrupting thyroid hormone synthesis. Iodine deficiencies in infants is unlikely in Canada as fortification with iodine of human milk fortifiers and infant formulas is mandatory and fortification of infant cereal products is voluntary (CFIA 2025). Additionally, the NOAEL for thyroid epithelial cell hyperplasia in the parental generation of a one-generation study was 40 mg sodium chlorate/kg per day (equivalent to 31 mg chlorate/kg bw per day), much lower than the NOAEL for developmental toxicity of 200 mg sodium chlorate/kg bw per day (equivalent to 156 mg chlorate/kg bw per day) seen in the same

study (EFSA 2015), suggesting that the selected ADI would be protective of developmental effects. For the reasons stated above, an uncertainty factor for database deficiencies was unnecessary.

The ADI was calculated as follows:

$$\text{ADI for chlorate} = (0.9 \text{ mg/kg bw per day})/30$$

$$\text{ADI for chlorate} = 0.03 \text{ mg/kg bw per day}$$

where:

- 0.9 mg/kg bw per day is the $BMDL_{10}$, based on increased thyroid gland follicular cell hypertrophy and follicular cell mineralization in a chronic rat study (Health Canada 2008a; NTP 2005; U.S. EPA 2016)
- 30 is the uncertainty factor, comprising 3 for interspecies differences and 10 for intraspecies differences

Based on the ADI for chlorate, an HBV can be calculated as follows:

$$\text{HBV for chlorate} = 0.03 \text{ mg/kg bw per day} \times 74 \text{ kg} \times 0.80) / 1.53 \text{ L/day}$$

$$\text{HBV for chlorate} = 1 \text{ mg/L (rounded)}$$

where:

- 0.3 mg/kg bw per day is the ADI for chlorate
- 74 kg is the average body weight of an adult human (Health Canada 2021)
- 0.80 is the allocation factor (Krishnan and Carrier 2013)
- 1.53 L/day is the drinking water intake of an adult human (Health Canada 2021)

3.3 Chlorine dioxide

An HBV has not been calculated for ClO_2 because of its rapid reduction to chlorite and, to a lesser extent, chlorate. The lowest NOAEL and LOAEL observed for ClO_2 are 3 and 14 mg/kg bw per day, respectively, and are based on thyroid hormone changes associated with decreased locomotor activity (Orme et al. 1985). They are similar to the NOAEL of 2.9 mg/kg bw per day used to derive the tolerable daily intake (TDI) for chlorite, a major metabolite of ClO_2 (CMA 1996; Gill et al. 2000). The MAC for chlorite is considered adequately protective for potential toxicity from ClO_2.

4.0 Analytical methods

4.1 Standardized methods

Standardized methods available for the analysis of chlorite and chlorate in drinking water and their respective method detection limits (MDLs) are summarized in Table 7. Similarly, Table 8 provides the analytical methods available and respective MDLs for ClO_2. MDLs are dependent on the sample matrix, instrumentation and selected operating conditions, and will vary between

individual laboratories. These methods are subject to a variety of interferences that are outlined in the respective references and briefly described in the tables below.

Select accredited laboratories in Canada were contacted to determine the MDLs and the method reporting limits for chlorite and chlorate analysis. Reporting limits were between 0.0001 mg/L and 0.5 mg/L for chlorite and between 0.0002 mg/L and 0.5 mg/L for chlorate based on stated methods (Lee 2023). Drinking water treatment systems should discuss sampling requirements with the accredited laboratory conducting the analysis to ensure that quality control procedures are met. Method reporting limits must be low enough to ensure accurate monitoring at concentrations below the MAC.

Table 7: Standardized analytical methods for the analysis of chlorite and/or chlorate in drinking water

Method (reference)	Methodology	Chlorite MDL (mg/L)	Chlorate MDL (mg/L)	Interferences/Comments
EPA 300.0 Rev. 2.1 (U.S. EPA 1993)	Ion chromatography	0.01	3	ClO_2 must be quenched immediately upon sampling.
EPA 300.1 Rev. 1.0 (U.S. EPA 1997)	Ion chromatography	0.00089	0.00131	Any ClO_2 must be purged at time of sample collection.
EPA 317.0 Rev. 2.0 (U.S. EPA 2001)	Ion chromatography (conductivity detection with PCR online)	0.00089	0.00062	ClO_2 must be quenched immediately upon sampling.
EPA 326.0 Rev. 1.0 (U.S. EPA 2002)	Ion chromatography	0.002	0.0017	ClO_2 must be quenched immediately upon sampling.
EPA 327.0 Rev. 1.1 (U.S. EPA 2005)	Lissamine Green B and Horseradish Peroxidase (with detection by visible spectrometry)	0.078	N/A	Laboratory glassware and ClO_2 in sample may interfere.
SM 4500-ClO_2 E (APHA et al. 2023b)	Amperometric Method II	0.00144	0.00255	Manganese, copper and nitrite may interfere.
SM 4500-ClO_2 C (APHA et al. 2017)	Amperometric Method I	0.02	N/A	Iodide, bromide, chloride, ferricyanide, ferric ion, chromate and dichromate may interfere.
D-6581-18 A (ASTM 2018)	Ion Chromatography (chemically suppressed)	0.005	0.005	Method technically equivalent to Part B of U.S. EPA Method 300.1.2.
D-6581-18 B (ASTM 2018)	Ion Chromatography (electronically suppressed)	0.02	0.02	Method technically equivalent to Part B of U.S. EPA Method 300.1.2.
ISO 10304-4:2022(E) (ISO 2022)	Ion chromatography	0.01	0.03	Organic acids, dissolved organics, fluoride, carbonate, nitrite and nitrate, high levels of chloride and bromide can interfere.

APHA – American Public Health Association; ASTM – ASTM International; ISO – International Organization for Standardization; N/A – not applicable; PCR – polymerase chain reaction; U.S. EPA – United States Environmental Protection Agency.

Table 8: Standardized analytical methods for the analysis of chlorine dioxide in drinking water

Method (reference)	Methodology	MDL (mg/L)	Interferences/Comments
EPA 327.0 Rev. 1.1 (U.S. EPA 2005)	Lissamine Green B and Horseradish Peroxidase (with detection by visible spectrometry)	NA	Laboratory glassware and ClO_2 in sample may interfere.
SM 4500-ClO_2 E (APHA et al. 2023b)	Amperometric Method II	NA	Low titrated solution pH of 1.0 to 2.0 may trigger interferences.
SM 4500-ClO_2 C (APHA et al. 2017)	Amperometric Method I	NA	Iodide, bromide, chloride, ferricyanide, ferric ion, chromate and dichromate may interfere.
SM 4500-ClO_2 E-00 (APHA et al. 2000)	Amperometry	0.02	This method is only available online.

APHA – American Public Health Association; MDL – method detection limit; NA – Not available; U.S. EPA – United States Environmental Protection Agency.

4.2 Sample preservation and preparation

The analytical methods in Table 7 measure the concentration of chlorite and chlorate in the sample. Sample processing considerations for analysis of chlorite and chlorate in drinking water (for example, sample preservation and storage) using standardized or U.S. EPA-approved methods can be found in the reference documents indicated in the table. Appropriate sample handling procedures are essential to obtain accurate, precise and reliable data on chlorite and chlorate occurrence and formation.

Once a sample is taken, it is critical to quickly stop further ClO_2 reaction, which can continue to form more chlorite and/or chlorate. This is generally achieved by purging the sample with an inert gas (helium, argon, or nitrogen). Sparging must be conducted prior to preservation and at time of sample collection.

4.3 Online and portable meters

Numerous field-test kits for ClO_2 have been identified on the market. Some of these devices also measure chlorite. Among these test kits, some have been identified as using U.S. EPA-approved analytical methods (U.S. EPA 2024), including one that measures chlorite and ClO_2 using an electrochemical sensor (amperometric method) as indicated in Table 9 (APHA et al. 2023a).

To accurately measure chlorite and chlorate using these units, drinking water treatment systems should develop a quality assurance and quality control program such as those outlined in SM 3020 (APHA et al. 2023a). In addition, periodic verification of results using an accredited laboratory is recommended. Drinking water treatment systems should check with the responsible drinking water authority to determine whether results from these units can be used for compliance reporting.

Table 9: Chlorite and/or chlorine dioxide field test kits based on EPA-approved methods

Method (reference)	Methodology	Parameter measured
SM 4500-ClO_2 E (APHA et al. 2023a)	Amperometric method (using an electrochemical sensor)	Chlorite and ClO_2
ChlordioX Plus, Rev. 1.1 (Palintest 2020)	Amperometry (using disposable sensors)	Chlorite and ClO_2
ChlordioX Plus, Rev. 1.0 (Palintest 2013)	Amperometry (using disposable sensors)	Chlorite and ClO_2

APHA – American Public Health Association.

5.0 Treatment considerations

ClO_2 is a broad-spectrum powerful oxidant and disinfectant. Drinking water systems use ClO_2 largely to oxidize iron and manganese, control taste and odour and reduce trihalomethanes formation (Hoehn et al. 1992). It is also used to remove colour and inactivate chlorine-resistant microorganisms (such as protozoa). Pathogen inactivation with ClO_2 is much less affected by pH in the 6.0 to 8.5 range than with chlorine. ClO_2 yields lower levels of organic DBPs (such as trihalomethanes) in comparison to free chlorine (U.S. EPA 1999; 2011). ClO_2 is a highly selective oxidant due to its one-electron transfer mechanism where it is reduced to chlorite (ClO_2^-) (U.S. EPA 1999). ClO_2 can be combined with ferrous ions, activated carbon, ozone, ultraviolet (UV), visible light, or persulfate processes (Xu et al. 2022). However, ClO_2 can produce inorganic DBPs, namely chlorite and chlorate, during the oxidation process (Freese and Nozaic 2007). Treatment strategies are focused on prevention of their formation as the best approach; once formed, removal of chlorite and chlorate can be challenging. Chlorite can be removed after formation using ferrous salts, sulphur compounds and GAC, but chlorate is very difficult to effectively remove.

5.1 Municipal-scale treatment

Chlorite and chlorate formation in water is directly related to the application of ClO_2. Chlorite formation can vary between 30% and 70% and chlorate formation between 5% and 25% when using ClO_2 (Korn et al. 2002; Richardson et al. 2009; Yang et al. 2013). Therefore, many treatment strategies are focused on prevention, such as:

- optimizing the ClO_2 generation process to maximize ClO_2 purity
- controlling treatment processes to minimize disinfectant demand
- limiting the feed dose of ClO_2
- minimizing chlorate formation by:
    - using high quality hypochlorite solutions and
    - removing chlorite precursors

5.1.1 Chlorine dioxide generation process

There are various methods to generate ClO_2, which may result in ClO_2 of varying purity (Gordon 2001). ClO_2 is produced on site for water treatment as a dissolved gas in aqueous solutions. Depending on generator design, it is typically generated at concentrations of 3 g/L or less (Black & Veatch 2010).

ClO_2 can be generated via a number of processes (Black & Veatch 2010; Gates et al. 2009), including:

- reaction of an acid with sodium chlorite solution (acid-chlorite)
- reaction of chlorine (in solution or gas phase) with chlorite (in solution or solid phase) (chlorine-chlorite)
- use of an electrochemical or photochemical generation process using a sodium chlorite solution (chlorite-based)
- use of chlorate-based systems, which may use a variety of reducing agents (chlorate-based)

The generation of ClO_2 is complex but, assuming a chlorine-chlorite reaction, a simplified equation can be written as (Gates 1999):

$2NaClO_2 + Cl_2\ (g) \rightarrow 2ClO_2\ (g) + 2NaCl$

Note that this equation does not describe the formation of potential by-products, such as chlorite or chlorate.

Sodium chlorite ($NaClO_2$) can also react with hypochlorous acid or hydrochloric acid to form ClO_2 and other by-products (Gates 1999). The purity of the ClO_2 generated will depend on the specific generator used. It is recommended that the equipment specifications for generators include a requirement that they produce ClO_2 that is consistently at least 95% pure. Purity is determined by dividing the concentration of ClO_2 by the sum of the concentrations of ClO_2, chlorite, chlorate and free available chlorine (FAC) (Black & Veatch 2010).

5.1.1.1 Generator optimization

ClO_2 generator design and performance significantly impact the amount of chlorite ion formed during ClO_2 production. Precise generator operation, proper maintenance and the generation technology used have a large bearing on the ClO_2 production efficiency and the rate at which chlorite and other undesirable by-products (including chlorate, hydrogen peroxide and perchlorate) are formed and fed into the water when dosing the ClO_2. A properly tuned generator will yield high purity ClO_2, thus limiting the presence of contaminants that can carry through into the distribution system and increase the total concentration of chlorate and chlorite. Proper balance and control of ClO_2 generators are required to prevent the formation and carry-through of impurities such as chlorate ion, perchlorate ion and chlorine (Gordon 2001).

Unreacted sodium chlorite feedstock can pass through the generator into the treated water. This can be minimized by controlling the feedstock, frequently checking and enhancing generator performance, and proper generator design and operation (Black & Veatch 2010). It is recommended that designers and operators consult with individual suppliers or manufacturers of ClO_2 generators to determine optimal operating conditions and practices.

ClO_2 generators can produce free chlorine as a side-product or allow unreacted free chlorine reagent to enter the treated water. Yang et al. (2022) reported that aliphatic amines reacted rapidly with ClO_2 to form significant amounts of FAC and related DBPs. Their study investigated the formation of FAC when ClO_2 reacts with six model aliphatic amines (including primary amines, secondary amines and tertiary amines). FAC was formed immediately upon the addition of ClO_2 to the precursor solution. While free chlorine and chlorite can react to reform ClO_2, this mixture can also result in the formation of chlorate (Gan et al. 2016).

5.1.1.2 Use of high-quality reagents

The quality of the chemicals used to generate ClO_2 will have a direct effect on the performance of the generator, including by-products formed. Hypochlorite solutions can add chlorate and chlorite ions to treated water. The handling and storage of hypochlorite solutions are important to minimize the potential for chlorate and chlorite addition to drinking water. Light, warmer temperatures, organic matter and certain heavy metal cations, such as copper, nickel and cobalt, accelerate the decomposition of the chlorine in the hypochlorite solution (AWWA 2024).

AWWA B303-95, Standard for Sodium Chlorite, outlines quality parameters for the chemical (Gates 1999). Storing sodium chlorite solutions beyond their recommended shelf life or exposing them to high temperatures may cause them to degrade, increasing their chlorate content (Black & Veatch 2010). Gan et al. (2016) emphasize that chlorate is present in a free chlorine solution due to auto-decomposition of free chlorine during manufacture, shipment and storage. The authors found 0.14 mg/L chlorate in 1 mg/L free chlorine. Thus, when ClO_2 is used as a pre-oxidant before chlorination, chlorate may result from the addition of chlorine.

There are currently three standards that address precursor feedstock specifications: (1) sodium chlorite (AWWA 2018b), (2) liquid chlorine (AWWA 2018a) and (3) hypochlorites (AWWA 2024). Chemicals used for disinfection should be certified as meeting NSF International/American National Standards Institute/Canada (NSF/ANSI/CAN) Standard 60: Drinking Water Treatment Chemicals - Health Effects, which is the recognized health effects standard for chemicals used to treat drinking water and includes certification criteria for chlorine and hypochlorites. NSF/ANSI/CAN Standard 60 sets maximum concentrations for impurities, such as chlorate and chlorite, that may be present in chemicals that are directly added to drinking water as part of the treatment process. This maximum concentration is known as the single product allowable concentration. The single product allowable concentration represents the maximum concentration that can be contributed to drinking water when hypochlorite is dosed at its typical use level as established under this standard (NSF/ANSI/CAN 2024).

In a bench-scale study conducted to determine the impacts of using ClO_2 contaminated with free chlorine, surface water from Lake Ontario (pH 7.6; 1.05 mg/L total organic carbon [TOC]) was dosed with 1.0 mg/L of ClO_2 and then “contaminated” with either 0 mg/L, 0.1 mg/L, or 0.25 mg/L of free chlorine (0%, 10%, or 20% contamination by chlorine mass). After 6 hours of contact time, the non-contaminated solution was observed to have higher chlorite levels than the contaminated solutions. In contrast, the chlorate levels were consistently higher for the contaminated solutions throughout the duration of the tests, with the highest chlorate

formation observed in the 20% contaminated solution. This solution showed almost twice as much chlorate formation than the non-contaminated solution (estimated graphically: at 1 hour, approximately 0.03 mg/L and 0.06 mg/L chlorate, respectively). The authors attributed the higher chlorate levels to the oxidation of chlorite by free chlorine (Hofmann et al. 2004).

5.1.2 Reducing disinfectant demand

Drinking water must, first and foremost, be microbiologically safe to prevent waterborne diseases. Therefore, any method used to reduce the ClO_2 demand (such as removing organic precursors or pre-oxidation) must not compromise the effectiveness of disinfection or increase other DBPs (for example, haloacetic acids, trihalomethanes). One effective way of minimizing DBP formation is through reducing disinfectant demand, which will lower the dose required. This approach enables both the removal of DBP precursor compounds and results in less ClO_2 present in solution to decompose into chlorite and, possibly, chlorate.

Natural organic matter (NOM) has a chemical oxidant (for example, ClO_2) demand that must be satisfied before pathogen log inactivation requirements can be met (Health Canada 2020). Grunert et al. (2018) identified that the disinfectant concentration decayed rapidly when dissolved organic carbon (DOC) concentrations were above or equal to 2 mg/L. Disinfection is typically applied after treatment processes that remove NOM to ensure efficient inactivation of pathogens and to minimize the formation of DBPs. Further information on the effect of NOM on chemical oxidant demand, decay and disinfection is available in other publications (Health Canada 2009; 2018).

Health Canada has published guidance on NOM in drinking water (Health Canada 2020), highlighting the impacts of climate change and source water changes on NOM. Temporal variations in the concentration and character of NOM can have a significant influence on the selection, design and operation of water treatment processes (Särkkä et al. 2015). More variable weather patterns associated with climate change will place increased importance on proper process selection (Huck and Coffey 2004) and day-to-day process control (Wright et al. 2016). Drinking water treatment systems should integrate strategies for managing risks related to changes in climate (for example, algal blooms, drought, forest fires and flooding) into treatment processes to maximize the reliability, robustness and resilience of their systems (Emelko et al. 2011; Irias 2019). A comprehensive review of the expected impacts of climate change on the treatability of NOM can be found elsewhere (Ritson et al. 2014).

5.1.2.1 Impact of pre-oxidation on chlorine dioxide demand and formation of chlorite and chlorate

Table 10 summarizes studies on the impact of pre-oxidation on ClO_2 demand and the formation of chlorite and chlorate in different types of water.

Table 10: Summary of impact of pre-oxidation studies

Reference	Water characteristics	Study specifics	Study objective	Results
Conio et al. (2009)	Surface water high in manganese	Change of pre-oxidant from ClO_2 to $KMnO_4$ Maintained ClO_2 for final disinfection	Regulatory compliance: to reduce chlorite levels	- Chlorite levels in DS reduced from 0.75 mg/L to 0.45 mg/L (40% reduction)
Sorlini et al. (2014)	Partially treated groundwater (through biofilter)	Pre-oxidation with either $KMnO_4$, ClO_2, or NaOCl	To lower ClO_2 consumption	- Pre-oxidation with ClO_2 resulted in lowest demand of secondary disinfectant (also ClO_2) - Chlorite levels lowest when using $KMnO_4$ as pre-oxidant
Sorlini et al. (2014)	Drinking water	Ferric chloride vs aluminum sulphate coagulation following pre-oxidation step	To investigate NOM impact on ClO_2 demand	- With ferric chloride, ClO_2 demand was similar regardless of oxidant used for pre-oxidation - With aluminum sulphate, ClO_2 demand was impacted by oxidant used at pre-oxidation step - Pre- and post-treatment PAC use found to reduce ClO_2 demand by 50% and lower chlorite/chlorate levels by 20 to 40%

DS – distribution system; NOM – natural organic matter; PAC – powdered activated carbon.

5.1.2.2 Impact of removal of organic precursors on chlorine dioxide demand and formation of chlorite and chlorate

Table 11 summarizes studies on the impact of organic precursor removal on ClO_2 demand and the formation of chlorite and chlorate in different types of water.

Table 11: Summary of organic precursor removal studies

Reference	Water characteristics	Study specifics	Study objective	Results
Sorlini et al. (2016)	Artificial lake water	- Bench-scale study	To evaluate treatment processes for effect on ClO_2 consumption and chlorite/chlorate formation	- Adding ferrous iron during coagulation decreased chlorite levels by 90% - Further treatment with GAC filtration decreased chlorite levels to below DL of 0.01 mg/L - Chlorate levels not affected by coagulation with or without ferrous iron
Arora et al. (2001)	Samples treated by a pilot-scale test unit	- Pre-oxidation with ClO_2 at 2.5 mg/L, coagulation,	To evaluate PAC for chlorite and chlorate removal	- PAC had no effect on chlorate

Reference	Water characteristics	Study specifics	Study objective	Results
		flocculation and sedimentation, and mixed for 30 minutes - Samples then passed through 0.45 µm filter		
Schmidt et al. (2000)	Surface water pretreated with activated carbon or with ozone followed by activated carbon	- Investigating ClO_2 transformation rate into chlorite	To investigate NOM impact on chlorite formation	- Chlorite formation found to be initially dependent on amount of NOM remaining after pretreatment; for longer reaction times, concentration of chlorite formed consistent when using ozone/activated carbon regardless of DOC levels - For activated carbon pre-treatment alone, chlorite concentration even highest at lowest DOC level - 40% to 50% of ClO_2 transformed in first 2 hours; 25% more transformed between 2 and 72 hours - ClO_2 reaction with NOM main source of chlorite formation but auto-decomposition reactions do contribute
Gonce and Voudrias (1994)	Drinking water	- Test solutions had 2.3 mg/L TOC, initial chlorate concentration of 5 mg/L and were pH-adjusted to 5 or 7 - Solutions were passed through a column with 4 g of GAC - Influent solution replaced with buffered solution containing no chlorate	To test chlorate removal using GAC at pH 5 and 7	- No chloride was detected in the effluents of these experiments, indicating that removals were due to sorption rather than chemical reduction - For pH 5 solution, after 7 hours of operation, concentration of chlorate in the effluent was equal to that of the influent, indicating that no more chlorate was being removed - Most of the chlorate (98.3%) desorbed from the GAC over 7 hours - Results for the pH 7 solution were similar, except that total filter breakthrough occurred within 90 minutes of operation. - GAC sorption capacity was 4.9 and 0.5 mg chlorate/g GAC at pH 5 and pH 7, respectively

DL – detection limit; DOC – dissolved organic carbon; GAC – granulated activated charcoal; NOM – natural organic matter; PAC – powdered activated carbon; TOC – total organic carbon.

5.1.3 Impact of ferrous iron addition on chlorite and chlorate removal

The addition of reduced iron compounds (such as ferrous chloride) has been used to lower chlorite levels by reducing chlorite (ClO_2^-) to chloride (Gates 1999). Ferrous iron reduces chlorite and forms solid ferric hydroxide (Hurst and Knocke 1997; Sorlini and Collivignarelli 2005):

$$4Fe^{2+} + ClO_2^- + 10H_2O \rightarrow 4Fe(OH)_{3\,(s)} + Cl^- + 8H^+$$

The ferric hydroxide formed is insoluble and can therefore be removed via sedimentation or filtration in the treatment train. It may also act as a coagulant, potentially reducing the required coagulant dose (Sorlini and Collivignarelli 2005). A summary of studies on the impact of ferrous iron on chlorite and chlorate removal is found in Table 12.

Table 12: Summary of studies of chlorite and chlorate removal using ferrous iron

Reference	Water characteristics	Study specifics	Study objective	Results
Sorlini and Collivignarelli (2005)	3 waters with different organic matter concentrations	- Bench-scale study - Ferrous iron as $FeCl_2$ (stoichiometric ratio of 3.31) mg Fe^{2+}/mg chlorite	- To test chlorite removal using ferrous iron - To evaluate impact of 3 starting pH conditions (pH 7.0, 7.86 and 8.5)	- Reaction complete in 60 seconds with 98% to 100% chlorite removal - Highest chlorite removals obtained at pH 7.0, with decreasing efficacy as pH increased - No chlorate removal
Hurst and Knocke (1997)	Drinking water	- Bench-scale study	- To evaluate removal using ferrous iron-chlorite under alkaline conditions (up to pH 10) and aerated (> 3 mg/L $O_{2\,(aq)}$) and deaerated conditions (< 1 mg/L $O_{2\,(aq)}$)	- Dosing ferrous iron 10% to 20% above theoretical stoichiometric requirement allowed for efficient chlorite removals at pH 8 to 10 - Resulted in greater chlorite concentrations at pH 9 and 10 as DOC increased - No chlorate removal
Cassol et al. (2022)	Drinking water	- Mixing ferrous iron (as ferrous sulphate) with $Al_2(SO_4)_3$ coagulant - Study used initial ClO_2 dose of 1.0 mg/L as pre-oxidant	- To remove chlorite formed from using ClO_2 as a pre-oxidant	- Molar yield of chlorite formed after pre-oxidation was 72.3% - Coagulation with $Al_2(SO_4)_3$ resulted in no chlorite removal; using ferrous iron with $Al_2(SO_4)_3$ transformed chlorite to chloride
Conio et al. (2009)	Surface water	- Two full-scale DWTPs using ClO_2 as a pre-oxidant - Experienced high chlorite levels	- To remove chlorite formed when ClO_2 used as a pre-oxidant with addition of $FeCl_2$	- Final chlorite concentrations leaving plants averaged 0.2 to 0.4 mg/L during study period - No chlorate removal

Reference	Water characteristics	Study specifics	Study objective	Results
Sorlini et al. (2016)	Artificial surface water	- Bench-scale study - Processes: pre-oxidation ($KMnO_4$, NaOCl, ClO_2), coagulation and flocculation (PACl with/without $FeCl_2$), GAC adsorption (10-minute EBCT), final disinfection with ClO_2	- To evaluate different treatment processes for their effect on chlorite and chlorate removal	- Adding ferrous iron during coagulation decreased chlorite levels by 90% - No chlorate removal
Arora et al. (2001)	Surface water	- Pilot-scale, conventional treatment unit - ClO_2 applied at 2.5 mg/L - Coagulation with PACl (7 mg/L) or $FeCl_3$ (6 mg/L)	- To evaluate the effect of ferrous sulfate on chlorite and chlorate removals	- Chlorite levels (ranging from 0.9 to 1.5 mg/L) were reduced to below DL with a ferrous ion dose of 5 mg/L or higher - Kinetics of chlorite removal with ferrous ion was complete in less than 30 seconds - Mass of chlorite removed per mass of ferrous ion was calculated to be a ratio of 2.7 on average (ranging from 2.4 to 3.0)

DL – detection limit; DOC – dissolved organic carbon; DWTPs – drinking water treatment plants; EBCT – empty bed contact time; GAC – granulated activated charcoal; PACl – polyaluminum chloride.

5.1.3.1 Sulphur compounds

A study by Gordon et al. (1990) proposed using sulphur dioxide-sulphite chemistry as a means of removing chlorite. After application of ClO_2, sulphur dioxide-sulphite ion can be added to remove residual ClO_2 and chlorite. Free chlorine could then be added to remove residual sulphur dioxide-sulphite ion in the treated water. The study used bench-scale experiments to react sodium sulphite (Na_2SO_3) with sodium chlorite ($NaClO_2$). The authors determined that, in the pH range of 7 to 7.5, the below reaction was likely occurring. They also determined that the experimental stoichiometry deviated by ± 8% from this equation.

$$2SO_3^{2-} + ClO_2^- \leftrightarrow 2SO_4^{2-} + Cl^-$$

However, above pH 8 and in the presence of air, the experimental stoichiometry deviated from the equation, likely due to increasing competition from the sulphite ion-oxygen reaction. The authors determined that, with chlorite levels between 0.5 and 7 mg/L and using a tenfold excess of sulphur dioxide-sulphite, chlorite removal can be completed in less than 1 minute at pH 5 and below, and in less than 15 minutes at pH 6.5.

Arora et al. (2001) used a pilot-scale, conventional treatment unit to evaluate the effect of sulphite ion (in the form of sodium sulphite) on chlorite and chlorate removal. The water tested was surface water, ClO_2 was applied at 2.5 mg/L and the coagulant was polyaluminium chloride

(PACl) (7 mg/L) or ferric chloride (6 mg/L). Chlorite removal was better at lower pH (6.7 versus 7.9) when coagulating with PACl. Sulphite ion doses at 50 mg/L were required for approximately 50% chlorite removal.

5.1.3.2 Activated carbon

Chlorite removal by GAC occurs by either adsorption or chemical reduction of chlorite to chloride (see the below equation) (Collivignarelli et al. 2006). Table 13 summarizes studies on the removal of chlorite and chlorate by activated carbon.

$$ClO_2^- + HOCl \rightarrow ClO_3^- + Cl^- + H^+$$

Table 13: Summary of identified activated carbon-based chlorite/chlorate removal studies

Reference	Water characteristics	Study specifics	Study objective	Results
Collivignarelli et al. (2006)	Distilled water	Batch experiments with 20 g GAC preloaded with 5 mg/L of chlorite; solution of distilled water with 500 mg/L nitrate ions and 5 mg/L chlorite	To evaluate GAC for chlorite removal	Chlorite removal was complete within 6 to 7 minutes
Collivignarelli et al. (2006)	400 mg/L of nitrate added to distilled water	GAC preloaded with nitrate ions (0, 100, 180, or 240 mg/L); solution of distilled water with nitrate ions (0, 60, 260, or 400 mg/L) and 5 mg/L chlorite	To demonstrate the negative effect of inorganic ions on chlorite removal	Time required for chlorite removal increased from 7 to 15 minutes in the presence of nitrate
Collivignarelli et al. (2006)	Raw river water	Bench-scale column test; raw water spiked with 5 mg/L chlorite; GAC capacity 30 mg chlorite/g GAC; EBCT of 10 minutes	To determine chlorite removal efficacy of thermally regenerated carbon	Regenerated carbon had 100% chlorite removal efficacy for approximately 20 days, with filter breakthrough after 475 hours of operation
Collivignarelli et al. (2006)	Raw river water	Exhausted GAC (where the iodine value is less than 600 mg/L) raw water spiked with 5 mg/L chlorite; GAC capacity 30 mg chlorite/g GAC; EBCT of 10 minutes	To explore chlorite removal efficacy of exhausted GAC	Exhausted GAC achieved 50% to 60% removal during study
Sorlini et al. (2017)	Drinking water	Laboratory-scale small column tests and pilot-scale study	To predict activated carbon removal of	Pilot plant test shows GAC provides good chlorite removal, close to 55%

Reference	Water characteristics	Study specifics	Study objective	Results
			chlorite at full-scale level	Laboratory-scale small column tests useful to predict pilot column and full-scale plant performance
Arora et al. (2001)	Water treated at pilot scale and filtered (0.45 µm filter)	PAC slurry (≤ 30 mg/L) added to filtered samples after pre-oxidation with 2.5 mg/L ClO_2, followed by coagulation, flocculation and sedimentation	To evaluate PAC for chlorite and chlorate removal	Reductions in chlorite were minimal (< 11%)
Gonce and Voudrias (1994)	Drinking water	GAC column test with 4 g GAC with 5 mg/L chlorite solution passed through column with 0.3 or 2.3 mg/L of TOC	To explore the effect of NOM on chlorite removals	Study showed limited GAC effectiveness at removing chlorite in presence of NOM Chlorite removal at hour 24 was 66% and 52% for the 0.3 and 2.3 mg/L TOC, respectively (14% differential) By hour 120, this difference had shrunk to 8%[a]
Gonce and Voudrias (1994)	Drinking water	Effect of varying pH was studied at pH 5, 7, and 8	To explore the effect of pH on chlorite removals	pH 5 solution showed complete removal during first 60 hours of operation (10 000 bed volumes) At 60 hours, GAC capacity was 100 mg chlorite/g GAC with EBCT of 0.34 minutes At pH values 7 and 8, filter breakthrough noted within 2 hours Chlorine atoms (either as chlorite or chloride) recovery in effluent was 96% or higher, indicating that the chlorine species were mostly not sorbed onto the GAC[b]
Sorlini et al. (2017)	Partially treated water (collected at outlet of GAC columns), followed by pre-oxidation with ClO_2 then, coagulated, flocculated, settled and filtered through	RSSCTs conducted at lab-scale Columns were fed 1 mg/L chlorite solution; each column operated with 5-minute EBCT at 1 L/hour flow rate; columns operated continuously GAC preloaded by mixing for 20 days in	To compare mineral and vegetal GAC under virgin and preloaded conditions To evaluate influence of preloaded GAC on chlorite removal To demonstrate ability of	Virgin mineral and vegetal GACs demonstrated complete chlorite removals for three months and one month, respectively After 6 months, virgin mineral GAC and virgin vegetal GAC produced chlorite concentrations of about 0.02 mg/L and 0.045 mg/L respectively Comparing the preloaded GACs, mineral GAC had the higher removal efficacy during the first

Reference	Water characteristics	Study specifics	Study objective	Results
	a pilot sand column without backwashing	a solution of methylene blue, iodine and phenol	laboratory- and pilot-scale tests to predict GAC removal at full-scale performance for chlorite	month; however, the vegetal GAC achieved removals almost twice that of the mineral GAC after 80 days of operation Preloaded GAC removed chlorite 30% to 40% less than virgin GAC Activated carbon proved effective at chlorite removal with virgin activated carbon achieving 80% removal, and preloaded carbon having an efficacy of 19%
Hoehn et al. (2003)	Drinking water	Full-scale study DWTP using ClO_2 for pretreatment Average ClO_2 dose was 0.4 mg/L	To evaluate GAC effectiveness in removing chlorite	GAC achieved average overall chlorite removal of 63%, with removals declining with increasing volume throughput
Sanchez-Cano et al. (2024)	Drinking water	2 granulated activated carbons modified with 5 different alkyl quaternary ammonium-based surfactants with high affinity for inorganic anions	To investigate potential full-scale application of 2 modified granulated activated carbons for chlorite and chlorate removal	GAC modified with hexadecyl pyridinium chloride monohydrate is an efficient adsorbent that achieves high chlorite and chlorate removal in 2 hours (≥ 99 and 80 ± 0.5% of chlorite and chlorate, respectively)
Sanchez-Cano et al. (2024)	Drinking water	Drinking water from actual DWTP and 12-minute EBCT	To evaluate chlorite/chlorate removal using a continuous flow under real-world conditions	Showed high chlorite and chlorate removal efficacy for four cycles of 160 hours each showing promise for full-scale use in drinking water applications.

DWTP – drinking water treatment plant; EBCT – empty bed contact time; GAC – granulated activated charcoal; LTOC – low total organic carbon; NOM – natural organic matter; PAC – powdered activated carbon; RSSCT – rapid small-scale column tests; TOC – total organic carbon.

[a] This suggested that the GAC's reactive sites were taken up by the organics and/or exhausted by the reaction.

[b] This suggested the pathway for chlorite removal is the redox reaction wherein chlorite is reduced to chloride.

5.1.3.3 UV photolysis

A study investigated the mechanism for UV photolysis of ClO_2^- and the production of reactive species. UV photolysis primarily transforms chlorite to ClO_2, and then to hypochlorous acid/hypochlorite ion, chlorate and chloride. The formation of DBPs and total organic chlorine is not significant during UV/ClO_2^- treatment, and over 90% of total organic chlorine is unknown DBPs (Zheng et al. 2023).

Sulphite radicals-based advanced reduction processes have shown promise in efficiently removing chlorite. Contributions of sulphite radicals and hydrated electrons in a UV/sulphite [UV/S(IV)] system for chlorite reduction were investigated. The UV/S(IV) system achieved

greater than 81.0% removal of 13.5 mg/L chlorite to chloride within 60 minutes in real water using 160 mg/L S(IV) and UV doses of 6.0 mW/cm^2 (Xiao et al. 2024).

5.1.3.4 Magnetic ion exchange

A study using response surface methodology-directed adsorption of chlorite and chlorate onto a magnetic ion exchange (MIEX) resin showed that this substrate can be an effective adsorbent for chlorite and chlorate. When the resin dose is increased from 2 to 10 mL/L, the removal rates of chlorite and chlorate increase. At the optimal stirring strength of 250 rpm and neutral pH, the highest removal of chlorite and chlorate by MIEX was achieved. Co-occurring anions will impact the removal of chlorite and chlorate in the following order: $SO4^{2-} > CO_3^{2-} > HCO_3^- > Cl^-$ for chlorite, and $SO4^{2-} > Cl^- > HCO_3^- > CO_3^{2-}$ for chlorate. The Box-Behnken design method used adsorbent dose, reaction time and initial concentration to establish a quadratic polynomial mode to successfully predict the removal efficacy for chlorite and chlorate. The adsorbent dose had a strong correlation with the chlorite or chlorate concentration and reaction time. These factors impact the optimization of the chlorite and chlorate removal process by MIEX. The adsorption capacities of chlorite and chlorate through MIEX were established by this study as being 398 mg/L and 525 mg/L, respectively (Yang et al. 2020).

5.1.4 Prevention of chlorate formation in hypochlorite solutions

Although the formation of chlorate is most often associated with the use of ClO_2, the treatment of drinking water with either NaOCl or calcium hypochlorite ($Ca(OCl)_2$) can also increase the concentration of chlorate in finished water. Hypochlorite solutions contain oxyhalide species such as perchlorate, chlorate and bromate that form during and after treatment. Chlorate is known to increase in concentration during storage as a function of time, temperature and a suite of chemical factors (Snyder et al. 2009; Stanford et al. 2011).

The long-term storage of hypochlorite solutions may lead to their decomposition and the formation of chlorate. The formation of chlorate ion in a hypochlorite solution is influenced by storage conditions such as pH, temperature, length of time in storage, presence of UV light, concentration of solution and presence of transition metals. Solid forms of hypochlorite are not affected by this decomposition (Gordon et al. 1993). Appropriate management of hypochlorite solutions must be undertaken to minimize decomposition into inorganic contaminants such as chlorite and chlorate. Effective and proactive practices related to purchase, storage and handling of hypochlorite solutions in order to minimize formation of chlorine-based DBPs should be in place and applied at all times. Hypochlorite solutions should (AWWA 2024):

- contain less than 1 500 mg chlorate/L
- have a pH greater than 12
- be used within a relatively short time frame after delivery (within 3 months)
- be stored in a cool dry location where the temperature does not exceed 30 °C, away from sunlight
- contain less than 0.08 mg/L of transition metals

Manufacturers are able to produce hypochlorite that has a lower initial concentration of chlorate. Drinking water treatment systems should specify hypochlorite solutions with a chlorate concentration as low as possible to ensure that they will meet the proposed guideline for chlorate and chlorite in finished water. Drinking water treatment systems should also plan to have minimum handling and storage time between product manufacturing and product delivery (Aranda-Rodriguez et al. 2017; Asami et al. 2009; AWWA 2024; AWWA and WRF n.d.; Coulombe et al. 2019). Snyder et al. (2009) developed recommendations for the handling and storage of hypochlorite solutions to help drinking water treatment systems minimize the concentrations of contaminants in hypochlorite solutions. Detailed recommendations can be found in Appendix E: Recommendations for the handling and storage of hypochlorite solutions.

In a case study of the purity of feedstock, Garcia-Villanova et al. (2010) found that it is not economically feasible to remove chlorate ions from treated water. The authors recommended monitoring reagent supplies delivered to the DWTP for chlorite, chlorate and bromate.

5.2 Residential-scale treatment

ClO_2 is intended to be used at the municipal scale in light of the complexity of operation. However, residential-scale treatment information may be relevant for small systems but not for individual residences supplied by a private well. Therefore, homeowners are unlikely to have this issue since they would not use ClO_2. In rare cases where small systems might need to treat for chlorite or chlorate, the same technologies (for example, GAC or MIEX) as larger systems can be used. Treatment devices classified as residential-scale may have a rated capacity to treat volumes greater than that needed for a single residence. Thus, these devices may also be used in small systems. As chlorite and chlorate are not included in the applicable certification standard, small system users should ensure that, at minimum, devices meet the relevant materials safety standard (NSF/ANSI/CAN 61).

Before a treatment unit is installed, the water should be tested to determine the general water chemistry and total chlorite and chlorate concentration in the water supply. Periodic testing by an accredited laboratory should be conducted on both the water entering the unit and the treated water, to verify that the treatment unit is effective. Units can lose removal capacity through use and time, and need to be maintained and/or replaced. Consumers should verify the expected longevity of the components in the treatment unit according to the manufacturer's recommendations and service it when required. Choosing a unit with a warning system (for example, an alarm or light indicator) will indicate when servicing is required.

Health Canada does not recommend specific brands of drinking water treatment units. However, it is strongly recommended that consumers use units that have been certified by an accredited certification body. The certification ensures that the drinking water treatment unit meets the appropriate NSF International/American National Standards Institute (NSF/ANSI) standards. The purpose of these standards is to establish minimum requirements for the materials, design and construction of drinking water treatment units. The certification of treatment units is conducted by a third party. This certification ensures that materials in the unit

do not leach contaminants into the drinking water (in other words, material safety). In addition, the standards include performance requirements that specify the removal that must be achieved for specific contaminants (for example, reduction claim) that may be present in water supplies.

Certification organizations (in other words, third parties) provide assurance that a product conforms to applicable standards and must be accredited by the Standards Council of Canada (SCC). Accredited organizations in Canada (SCC 2025) include:

- CSA Group
- NSF International
- Water Quality Association
- UL Solutions
- Bureau de normalisation du Québec (available in French only)
- International Association of Plumbing and Mechanical Officials
- ALS Laboratories Inc. (former Truesdail Laboratories Inc.)

An up-to-date list of accredited certification organizations can be obtained from SCC.

Currently, chlorite and chlorate are not included in the performance requirements (for example, reduction claims) of NSF/ANSI standards. Therefore, there are no residential treatment devices certified for chlorite or chlorate removal at this time. Technologies that have proven effective at the municipal scale should also be effective at the residential scale. For this reason, chlorite removal options are limited solely to adsorption through a GAC filter.

The chlorate ion is very difficult to remove from drinking water and GAC is not effective for its removal. No residential-scale treatment technology is currently available to remove it from residential tap water once it has been formed (Gallagher et al. 1994).

5.3 Distribution system considerations

Although ClO_2 is a relatively strong disinfectant, it is not frequently used as a distribution system disinfectant for two reasons: 1) its residual does not last as long as that of other disinfectants and 2) it breaks down quickly into chlorite (predominantly).

ClO_2 decay in the distribution system is the result of auto-decomposition reactions and reactions with organic and inorganic compounds, including biofilms, pipe materials and scales. It is also subject to photolytic decomposition. Some studies using ClO_2 as a secondary disinfectant in full-scale distribution systems have shown that residuals can be maintained throughout these specific systems without booster stations (Andrews et al. 2001; Volk et al. 2002). Other studies have demonstrated the opposite, that residuals disappear at the ends of the system without booster addition (Gates 1999). Disinfectant residuals decrease faster as the water temperature increases, and the size and complexity of the distribution system also increase. ClO_2 is generally not effective for secondary disinfection because the residual dissipates quite rapidly and the distribution systems are usually not biologically stable (Black & Veatch 2010).

5.3.1 Chlorite and chlorate in the distribution system

Chlorite and chlorate behaviour and fate within distribution systems have been investigated by numerous studies in recent years. Table 14 summarizes the findings of some of these studies under various conditions.

Table 14: Summary of studies on chlorite and chlorate in the distribution system

Reference	Water characteristics	Study specifics	Study objective	Results
Baribeau et al. (2002)	Drinking water	- ClO_2 use for secondary disinfection - Compared chloride and chlorate levels in small diameter pipes (6") (mostly unlined grey iron) and larger steel-reinforced concrete mains (14" to 24") - Cold (0–4 °C) and warmer water conditions (8–24 °C) - MDL for chlorate and chlorite = 0.01 mg/L	- To track chlorite and chlorate concentrations in DSs as a function of residence time, pipe diameter and materials, temperature, and post-disinfectant - To investigate the impact of using free chlorine as residual disinfectant	**Chlorite** - Generally, chlorite ion concentrations decreased with increasing water residence time[a] - Chlorite ion concentrations always below the MDL when using free chlorine in post-disinfection - Samples from small diameter pipes had lower and more variable chlorite levels than those from larger main pipes **Chlorate** - Chlorate levels decreased slightly in warm water with increasing residence time - Chlorate concentrations were significantly variable when using free chlorine in post-disinfection - Chlorate levels were below MDL or remained stable at low levels (0.09 mg/L) in cold water following ClO_2 post-disinfection[b] - Generally, chlorate levels were equal to or lower in small-diameter pipes than in main pipes
Rungvetvuthiv itaya et al. (2019)	Drinking water	- Buffered water at pH 7–9; temperature 15—35 °C; NOM present; presence of: ammonia, nitrite, nitrate - Chloramines present	- To investigate chlorite behaviour and fate under various DS conditions	- Chlorite is stable under typical DS conditions for this chloraminated system (buffered water at pH 7–9; temperature between 15 °C and 35 °C; and in the presence of NOM,

Reference	Water characteristics	Study specifics	Study objective	Results
				ammonia, nitrite and nitrate) - Chlorite decays in the presence of chloramines
Aranda-Rodriguez et al. (2008)	Drinking water	- Municipal DWS using ClO_2	- To assess the behaviour and fate of chlorite/chlorate ions in a municipal DS using ClO_2	- Chlorite ion levels decreased along DS whereas chlorate levels remained relatively constant - Study suggested mild temperature effects
Boano et al. (2015)	Drinking water	- DWS using ClO_2 as secondary disinfectant	- To conduct modelling study on chlorate in DWS using ClO_2	- Chlorate formation in DS minimal and related to initial amount of chlorate released by DWTPs
Hoehn et al. (2003)	Drinking water	- Full-scale study - DWTP using ClO_2 for pretreatment - Average raw water ClO_2 dose 0.4 mg/L	- To assess chlorite and chlorate levels prevalent in DWS using ClO_2 as secondary disinfectant	- Chlorite levels in DS were mostly less than 0.2 mg/L and decreased with distance from the DWTP - Chlorate levels did not exceed 0.1 mg/L
Sorlini et al. (2016)	Drinking water	- Full-scale reaction kinetics/stability case study - DS monitored 2006 to 2011 - U.S. EPA EPANET 2.0 modelling software applied to DS	- To analyze chlorite and chlorate levels in local municipal DS and conduct associated modelling	- Chlorite concentration was over 0.7 mg/L for 12% to 16% of results in the first 2 years, for 48% of results in 2008 and for 1% to 8% of results from 2009 to 2011 - Chlorite exceeded 0.7 mg/L at DS locations far away from the DWTP - Conversely, chlorate levels were always below 0.2 mg/L
Liu et al. (2013)	Drinking water	- DWS using ClO_2 as secondary disinfectant	- To assess the impact of metal oxides on chlorite/chlorate behaviour and fate in DWSs using ClO_2	- Metal oxides generally enhance ClO_2 decay into chlorite and chlorate - Particularly relevant for copper pipes in DSs
Reuter and Lastoskie (2021)	Drinking water	- DWS using ClO_2 as secondary disinfectant	- To assess the impact of lead oxide, lead carbonate and cupric oxide on chlorite/chlorate	- ClO_2 decay is enhanced by lead oxide, lead carbonate and cupric oxide - Decay rate significantly higher with lead oxide than cupric oxide

Reference	Water characteristics	Study specifics	Study objective	Results
			behaviour and fate in DS	- Only chlorite is produced when lead minerals are present but both chlorite and chlorate are produced when cupric oxide is present
Li et al. (2015)	Drinking water	- DWS using ClO_2	- To investigate chlorite and chlorate adsorption in DSs	- Chlorite and chlorate were adsorbed by calcium carbonate in the DS: 23.8% to 41.3% of chlorite and 16.7% to 28.7% of chlorate in the water was adsorbed within 3 hours - Adsorption decreased as pH and temperature increased
Gagnon et al. (2005)	Drinking water	- DWS using ClO_2 as secondary disinfectant	- To investigate impact of chlorite on controlling bacteria in DSs	- Chlorite in low concentrations is not effective at controlling heterotrophic bacteria in DSs - ClO_2 residual had to be maintained
Liyanage et al. (1997)	Drinking water	- DWS using ClO_2 as secondary disinfectant	- To investigate chlorite/chlorate effectiveness as disinfecting agents	- ClO_2 is an effective disinfectant, but its by-products (including chlorite and chlorate) are not effective for disinfection
Eisnor and Gagnon (2004)	Drinking water	- Bench-scale study	- To evaluate unlined cast-iron water distribution pipes	- Corrosion rates decreased when chlorite was used - ClO_2 did not impact corrosion

DS – distribution system; DWS – drinking water system; DWTP – drinking water treatment plant; MDL – method detection limit; NOM – natural organic matter.

[a] The authors suggested these decreases were attributed to ferrous iron in pipe material or metabolism by microbial biomass on pipe walls.

[b] This decrease was attributed to the presence of microbial biomass.

6.0 Management strategies

All drinking water treatment systems should implement a comprehensive, up-to-date risk management water safety plan. A source-to-tap approach should be taken to ensure water safety is maintained (CCME 2004; WHO 2017; 2024). These approaches require a system assessment to characterize the source water, describe the treatment barriers that prevent or reduce contamination, identify the conditions that can result in contamination and implement control measures.

Operational monitoring is then established, and operational/management protocols are instituted (for example, standard operating procedures, corrective actions and incident responses). Compliance monitoring is determined, and other protocols are implemented to validate the water safety plan (for example, recordkeeping, consumer satisfaction). Operator training is also required to ensure the effectiveness of the water safety plan (Smeets et al. 2009).

Management of chlorite and chlorate is principally focused on minimizing their formation by limiting the feed dose, optimizing the ClO_2 generator efficiency, removing residual ClO_2 and chlorite prior to chlorination and using high-quality hypochlorite solutions and their proper storage and handling.

The available treatment options to reduce chlorite ion concentrations in drinking water at the municipal scale are activated carbon, sulphur-reducing agents and iron-reducing agents, and optimizing the ClO_2 generator (WHO 2005). Currently, there is no known treatment available to remove chlorate ion once it has been formed in drinking water.

Removing residual ClO_2 and chlorite from solution before post-chlorination minimizes the formation of chlorate by removing key reactants (Griese et al. 1992). ClO_2 can react with chlorine (Cl_2) or hypochlorous acid (HOCl) to form chlorate and chloride ions when both ClO_2 and chlorine are used together, sequentially, in a single treatment process (Gordon et al. 1990; Richardson et al. 2009).

$$ClO_2^- + HOCl \rightarrow ClO_3^- + Cl^- + H^+$$

In summary, in order to control persistent DBP formation, it is important to minimize production of chlorate ion in the ClO_2 generation process and to remove the chlorite ion before adding post-chlorine (Gallagher et al. 1994; WHO 2005). Chlorate can also be generated when ClO_2 is used as a pretreatment prior to a UV-chlorine advanced oxidation process, as the by-product chlorite undergoes oxidation.

6.1 Treatment and distribution system considerations

The preferred control strategies should include methods to minimize chlorite and chlorate formation during treatment and within the distribution system. Effective management of chlorite and chlorate requires a good understanding of the disinfectant demand/decay. Drinking water treatment systems should make every effort to meet the guidelines; however, any method of control employed must not compromise the effectiveness of water disinfection. Impacts on the distribution system from any control strategy implementation should be considered.

6.1.1 Treatment and distribution system control options

The application of ClO_2 disinfection is complex and requires a high level of technical skill from operators. There are safety concerns associated with ClO_2 (ATSDR 2004). In light of these

hazards, an operator should exercise caution when handling ClO_2. It is also important to note that, in high concentrations, ClO_2 can be corrosive to certain materials used in water treatment systems. This can lead to accelerated wear and tear of equipment, increasing maintenance costs and potentially causing unexpected downtime (Palintest 2025).

Management of chlorite and chlorate is principally focused on minimizing their formation by limiting the feed dose, maximizing the efficiency of the ClO_2 generator, removing residual ClO_2 and chlorite prior to chlorination, using high-quality hypochlorite and taking steps to ensure its proper storage and handling. To minimize the amount of chlorate and chlorite added to treated water, drinking water treatment systems should purchase hypochlorite treatment chemicals that are certified as meeting NSF/ANSI/CAN Standard 60 (NSF/ANSI/CAN 2024).

Several aspects need to be taken into consideration, at the DWTP and within distribution systems, for controlling chlorite and chlorate resulting from the use of ClO_2. The need to address disinfectant demand and/or limit the ClO_2 dose are important considerations in the selection of this disinfectant for both small and large systems. During drinking water treatment, chlorite is the predominant reaction by-product, with 25% to 70% of the reacted ClO_2 converting to chlorite and 30% converting to chlorate or chloride (Korn et al. 2002; Richardson et al. 2009; Stevens 1982; U.S. EPA 2002; Yang et al. 2013).

A high ClO_2 dose can lead to an exceedance of the proposed chlorite and chlorate MACs since the disinfectant will ultimately react to produce these DBPs. The larger the system, the greater the need to apply a high ClO_2 dose at the DWTP to ensure a detectable residual at the extremities. This increased dose makes it more difficult to both maintain the residual and control resulting chlorite and chlorate levels.

ClO_2 is very reactive and must be generated on site. DWTPs using ClO_2 as primary disinfectant should not exceed a maximum dose of 1.2 mg/L. Limiting the dose of ClO_2 will ensure that chlorite and chlorate concentrations do not exceed the proposed guidelines. This approach also limits potential exposure to ClO_2 and represents a reasonable control measure. Given the disinfection efficacy of ClO_2 for drinking water, a higher dose is typically not necessary.

As much as 35% of the chlorate concentration found in a distribution system can be attributed to the type and performance of ClO_2 generator (WHO 2005). Optimizing (or maximizing the efficacy) of the ClO_2 generator is an important control measure for limiting the formation of chlorite and chlorate. Hypochlorite contains chlorate, which is known to increase in concentration during storage as a function of time, temperature and a suite of chemical factors. Best practices to address these factors should be implemented to minimize chlorate during and after the use of ClO_2.

For small systems using ClO_2 as a secondary disinfectant, the size of the distribution system is an important consideration. Since the ClO_2 reaction is rapid, ensuring a detectable residual at the extremities may not be possible unless it is a shorter distribution system. A longer distribution system may require a larger dose of ClO_2, which may not be an acceptable solution. Small

systems should also implement best practices to maintain stable chemical and biological water quality conditions throughout the system. These conditions are generally the most favourable for allowing the dissolved ClO_2 radical (ClO_2) to linger in the system and maintain a disinfectant residual (U.S. EPA 1999).

Removing residual ClO_2 and chlorite from solution before post-chlorination minimizes the formation of chlorate (Griese et al. 1992). Since ClO_2 reacts with chlorine (Cl_2) or hypochlorite to form chlorate and chloride ions, this is relevant when ClO_2 and chlorine are used sequentially (Gordon et al. 1990; Richardson et al. 2009).

The available treatment options to reduce chlorite ion concentrations in drinking water at the municipal scale are activated carbon, sulphur-reducing agents and iron-reducing agents (WHO 2005). Currently, there is no known treatment available to remove chlorate ion once it has been formed in drinking water.

6.2 Monitoring

Careful monitoring of ClO_2 and chlorite levels is very important. It ensures the safety and health of those in the vicinity, maintains operational efficiency, protects equipment, and safeguards the environment. Regular, precise monitoring and a proactive approach to water management are essential in mitigating the risks associated with elevated levels of these chemicals (Palintest 2025).

Monitoring programs should be designed to consider risk factors that contribute to chlorite and chlorate formation. Programs should verify that control strategies are operating as intended. Trend analyses will allow for forecasting water quality changes in advance and provide early warning signals. This monitoring will allow for the undertaking of control and/or proactive measures (Tomperi et al. 2016). In most cases, drinking water quality monitoring in distribution systems is undertaken for reasons related to achieving, maintaining, and/or verifying/demonstrating regulatory compliance; establishing and maintaining the most favourable operational conditions for the machinery and processes; or water system or parameter characterization. Since disinfection of water with ClO_2 and the use of hypochlorite solutions are currently considered the primary sources of chlorite and chlorate in drinking water, monitoring should focus on these processes.

Daily monitoring of ClO_2, chlorite and chlorate is recommended for water leaving the DWTP. For systems using hypochlorite solutions, levels of chlorate should also be monitored in the treated water at the plant.

6.2.1 Operational

Operational monitoring for chlorite and chlorate should take into account measures aiming at minimizing the formation of these by-products in the finished and distributed water while achieving and maintaining the most effective disinfection process. Appropriate operational monitoring also entails considering all identified conditions or factors that can inform the

frequency or location of monitoring (for example, depending on whether ClO_2 or sodium hypochlorite is used for secondary disinfection, seasonal variations of water temperature and other quality parameters, the quality of reagents).

For facilities using hypochlorite solutions, process monitoring is also needed to ensure water is adequately disinfected and DBP formation is minimized. Procedures regarding the purchasing, handling and storage of hypochlorite solutions are also needed to minimize the chlorite/chlorate concentration in the treated water. It is also important to verify the available chlorine in hypochlorite solutions upon delivery as this concentration affects the hypochlorite dose required to achieve disinfection targets and, by association, the chlorite/chlorate concentration in the treated water. Drinking water treatment systems should measure ClO_2 levels at the entry of the distribution to monitor its ongoing disinfection efficacy and ensure it does not exceed 1.2 mg/L.

Ferrous ion can reduce chlorite, but pH adjustment is required to minimize chlorate formation. The use of a reducing agent like ferrous ion can add complexity in the application of secondary disinfectants (including ClO_2) (U.S. EPA 2001). It is also important to take into consideration the aesthetic objective (≤ 0.1 mg/L) for total iron (Health Canada 2024) when dosing ferrous iron in order to ensure that this guideline is not exceeded.

6.2.2 Compliance

A locational annual average of a minimum of quarterly samples taken in the distribution system for chlorite and chlorate should be calculated. These values are then compared against the respective MAC. Sampling should occur at a point or at points in the distribution system where chlorite and chlorate concentrations are expected to be the highest. For systems using ClO_2, the locations where the maximum levels of chlorite and chlorate are usually found are mid-system and end of system (or extremities), respectively. For chlorite, consideration should be given to potential interferences from cast iron materials. Chlorite should be sampled in areas of the distribution system where the pipes are not made of cast iron.

For both chlorite and chlorate, locations with the highest water age, after booster chlorination stations, reservoir storage and areas with the longest disinfectant retention time, should also be considered. The locations of high concentrations may vary seasonally and temporally.

Increased frequency may be required for facilities using surface water sources (including groundwater sources that are under the direct influence of surface water) during periods where water quality may undergo significant changes. Such periods may include spring runoff, heavy rainfall events or periods of overland flooding or prolonged droughts.

7.0 International considerations

Other national and international organizations have drinking water guidelines, standards and/or guidance values for chlorite and chlorate in drinking water. Variations in these values can be attributed to the age of the assessments or to differing policies and approaches, including the

choice of key study and the use of different consumption rates, body weights and source allocation factors (see Table 15).

The World Health Organization (WHO) (2016) guidelines for chlorite and chlorate are considered provisional because the use of ClO_2 as a disinfectant may result in exceedances of the guideline values. However, difficulties in meeting the guideline values must never be a reason for compromising adequate disinfection. Although WHO (2016) calculated an HBV of 0.3 mg/L based on a $BMDL_{10}$ of 1.1 mg/kg bw per day for non-neoplastic effects on the thyroid of male rats (NTP 2005) and an uncertainty factor of 100, it retained the previous provisional guideline value of 0.7 mg/L from 2005 (WHO 2005) since, in some circumstances, it may not be possible to adequately disinfect potable water and maintain chlorate concentrations at or below the HBV, as chlorate is a by-product of hypochlorite.

The approach by the European Union (EU) for chlorite and chlorate is to have Member States take the measures necessary to ensure that water intended for human consumption complies with the parametric value of 0.25 mg/L set out in Part B of Annex I for chlorate, chlorite by January 12, 2026. However, a parametric value of 0.70 mg/L shall be applied where a disinfection method that generates chlorate or chlorite, in particular ClO_2, is used for disinfection of water intended for human consumption. Where possible, without compromising disinfection, Member States shall strive for a lower value. These parameters shall be measured only if such disinfection methods are used. Within the EU, a limit for chlorate and chlorite in potable water has been agreed upon, under the new Drinking water Directive, of 0.25 mg/L, with an exception of 0.7 mg/L (the WHO guideline level) where a disinfection method that generates chlorate, in particular ClO_2, is used. The basis for the use of 0.25 mg/L was not located.

For ClO_2, the U.S. EPA has set a maximum residual disinfectant level (MRDL). An MRDL is a health-based drinking water standard that specifies the highest level of disinfectant allowed in drinking water. It is enforceable and similar to the maximum contaminant level (MCL) (U.S. EPA 1998b).

Table 15: National and international drinking water values for chlorine dioxide, chlorite and chlorate

Chemical	**Agency** (year)	**Value** mg/L	**NOAEL and Key endpoint** (reference)	**UF**	**ADI** mg/kg bw/d	**BW** kg	**DW** L/d	**AF** %	**Comments**
Chlorine dioxide	U.S. EPA (1998b) - MRDL	0.8 mg/L (800 ppb)	3 mg/kg bw/day based on neurodevelopment (CMA 1996; U.S. EPA 1998a, 1998b)	100	0.03	70	2	80	No additional comments.

Chemical	Agency (year)	Value mg/L	NOAEL and Key endpoint (reference)	UF	ADI mg/kg bw/d	BW kg	DW L/d	AF %	Comments
Chlorine dioxide	Health Canada **proposed**	NA	NA	NA	NA	NA	NA	NA	A MAC has not been proposed for chlorine dioxide because of its rapid reduction to chlorite and, to a lesser extent, chlorate. The MAC for chlorite is considered protective for potential toxicity from chlorine dioxide.
Chlorine dioxide	WHO (2016)	NA	NA	NA	NA	NA	NA	NA	Any chlorine dioxide remaining at the consumer's tap will be reduced to chlorite and chloride upon ingestion. Consequently, a guideline value for chlorine dioxide has not been established. The provisional guideline values for chlorite and chlorate are adequately protective for potential toxicity from chlorine dioxide. The taste and odour threshold for chlorine dioxide is 0.2-0.4 mg/L.
Chlorine dioxide	Australia (2011)	0.4 mg/L AO	NA	NA	NA	NA	NA	NA	A health-based guideline value has not been established due to chlorine dioxide's rapid hydrolysis to chlorite and chlorate. The guideline for chlorite is adequately protective for potential toxicity from chlorine dioxide.
Chlorine dioxide	EU (2020)	NA	NA	NA	NA	NA	NA	NA	Not assessed.
Chlorite	Health Canada **proposed**	1 mg/L	2.9 mg/kg bw/day based on altered development, decreased liver weights (CMA 1996;	100	0.029	74	1.53	80	No additional comments.

Chemical	Agency (year)	Value mg/L	NOAEL and Key endpoint (reference)	UF	ADI mg/kg bw/d	BW kg	DW L/d	AF %	Comments
			Gill et al. 2000)						
Chlorite	WHO (2016)[a]	0.7 mg/L	3 mg/kg bw/day based on decreased liver weights (Gill et al. 2000)	100	0.03	60	2	80	No additional comments.
Chlorite	Australia (2011)	0.8 mg/L	2.9 mg/kg bw/day based on unspecified endpoint (CMA 1997; TERA 1998)	100	0.029	70	2	80	No additional comments.
Chlorite	EU (2020)[b]	0.25 mg/L; 0.7 mg/L	NOAEL of 30 mg/kg bw/day and other related values in table were used to calculate parametric value of 0.7 mg/L based on WHO 2005, 2016	100	0.03	60	2	80	No additional comments.
Chlorite	U.S. EPA (1994, 1998b)	MCL 1.0 mg/L	NA	NA	NA	NA	NA	NA	The MCL was set at 1 mg/L and was based on the lowest level considered practicably achievable by typical systems using chlorine dioxide, from both treatment and monitoring perspectives (U.S. EPA 1994).
Chlorate	Health Canada **proposed**	1 mg/L	$BMDL_{10}$ 0.9 mg/kg bw/day based on increased thyroid gland follicular cell hypertrophy and follicular cell mineralization in a chronic rat study (NTP 2005)	30	0.03	74	1.53	80	No additional comments.

Chemical	Agency (year)	Value mg/L	NOAEL and Key endpoint (reference)	UF	ADI mg/kg bw/d	BW kg	DW L/d	AF %	Comments
Chlorate	WHO (2016)[a]	0.7 mg/L[c]	30 mg/kg bw/day based on thyroid gland colloid depletion in 90-day rat study (McCauley et al. 1995)	1 000	0.03	60	2	80	No additional comments.
Chlorate	EU (2020)[b]	0.25 mg/L; 0.7 mg/L	NOAEL of 30 mg/kg bw/day and other related values in table were used to calculate parametric value of 0.7 mg/L based on WHO 2005, 2016	1 000	0.03	60	2	80	No additional comments.
Chlorate	Australia (2011)	NA	NA	NA	NA	NA	NA	NA	Data are currently considered insufficient to set a guideline value for chlorate in Australian drinking water supplies.
Chlorate	U.S. EPA	NA	NA	NA	NA	NA	NA	NA	No drinking water values have been set for chlorate.

ADI – acceptable daily intake; AF – allocation factor; AO – aesthetic objective; BMDL – benchmark dose level; BW – body weight; CMA – Chemical Manufacturers Association; DW – drinking water intake; EU – European Union; MAC – maximum acceptable concentration; MCL – mean contaminant level; MRDL – maximum residual disinfectant level; NA – not available; NOAEL – no-observed-adverse-effect level; NTP – National Toxicology Program; TERA – Toxicology Excellence for Risk Assessment; UF – uncertainty factor; U.S. EPA – United States Environmental Protection Agency; WHO – World Health Organization.

[a] WHO (2016) Provisional guideline value

[b] EU (2020) - The parametric value of 0.25 mg/L applies except where a disinfection method generates chlorate or chlorite, in particular chlorine dioxide, and is used for disinfection of water intended for human consumption. In such cases, a parametric value of 0.7 mg/L is used.

[c] Retained the previous provisional guideline value of 0.7 mg/L from 2005 (WHO 2005).

8.0 Rationale

Chlorite and chlorate are DBPs of ClO_2. Under certain conditions, the use of hypochlorite solutions to treat drinking water can also result in the introduction of chlorate. ClO_2 and its DBPs, chlorite and chlorate, are not present in the natural environment. Canadian data indicate that chlorite and chlorate levels found in drinking water are generally well below the MACs and are mostly below detection limits.

Drinking water treated with ClO_2 is the main source of exposure to chlorite and chlorate for the general population of Canada. An HBV and associated MAC were not calculated for ClO_2 as it rapidly degrades mainly to chlorite and minor amounts of chlorate in drinking water and is also quickly metabolized to chlorite and chlorate in laboratory animals. Furthermore, health effects seen in studies using ClO_2 were similar to those seen in studies using chlorite, its major metabolite, and, to a lesser extent, those using chlorate. The MAC for chlorite is considered adequately protective for potential toxicity from ClO_2. This approach is similar to the approach taken by other national and international organizations such as the WHO and Australia.

Health Canada, in collaboration with the Federal-Provincial-Territorial Committee on Drinking Water, is proposing a MAC of 1 mg/L (1 000 µg/L) each for chlorite and chlorate based on the following considerations:

- An HBV of 1 mg/L (1 000 µg/L) for chlorite based on developmental delays in F1 and F2 pups, including lower auditory startle amplitude, delayed preputial separation, decreased absolute brain weight and decreased altered liver weights in F0 and F1 generations from a two-generation developmental reproductive study
- An HBV of 1 mg/L (1 000 µg/L) for chlorate based on increased thyroid gland follicular cell hypertrophy and follicular cell mineralization in a 2-year study
- The proposed MAC for chlorite is considered protective for potential toxicity from ClO_2
- The availability of several analytical methods to measure chlorite, chlorate and ClO_2 concentrations both at water treatment facilities and in the field
- Drinking water treatment technologies are available to remove chlorite to below the MAC as well as to prevent its formation
- Chlorate can be reduced by controlling or minimizing its formation

For compliance with the guideline, a locational annual average of four quarterly samples (not running average) is recommended due to the highly variable NOM levels in source waters from climate change impacts. Issues with NOM are becoming more prominent as a result of forest fires, flooding and related climate events. To address climate change impacts, a running average would not capture the perturbations caused by climate change. A locational annual average would allow drinking water treatment systems to capture variations through monitoring, allowing for punctual process improvement.

As part of its ongoing guideline review process, Health Canada will continue to monitor new research in this area and recommend any change to this guideline technical document that it deems necessary.

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Appendix A: List of abbreviations

ADI	acceptable daily intake
ALT	alanine aminotransferase
ANSI	American National Standards Institute
AST	aspartate aminotransferase
ASTM	ASTM International
ATSDR	Agency for Toxic Substances and Disease Registry
A/J, B6C3F1, F344 and similar	mouse and rat strains
AWWA	American Water Works Association
BMDL	benchmark dose level
bw	body weight
CAS RN	Chemical Abstracts Service Registry Number
CDW	Federal-Provincial-Territorial Committee on Drinking Water
CHO, L5178Y $TK^{+/-}$	cell lines
ClO_2	chlorine dioxide
ClO_2^-	chlorite
ClO_3^-	chlorate
CMA	Chemical Manufacturers Association
CSA	CSA Group or Canadian Standards Association
DBP	disinfection by-product
DL	detection limit
DOC	dissolved organic carbon
DS	distribution system
DW	drinking water
DWS	drinking water system
DWTP	drinking water treatment plant
EBCT	empty bed contact time
FAC	free available chlorine
FNIHB	First Nations and Inuit Health Branch
G6PD	glucose-6-phosphate dehydrogenase
GAC	granulated activated charcoal
GD	gestation day
GSH	glutathione

HBV	health-based value
IARC	International Agency for Research on Cancer
ISO	International Organization for Standardization
LDH	lactate dehydrogenase
LOAEL	lowest-observed-adverse-effect level
LTOC	low total organic carbon
MAC	maximum acceptable concentration
MCHC	mean corpuscular hemoglobin concentration
MCL	maximum contaminant level
MDL	method detection limit
MIEX	magnetic ion exchange
MNPCE	micronucleated polychromatic erythrocytes
MRDL	maximum residual disinfectant level
MRL	method reporting limit
NOAEL	no-observed-adverse-effect level
NOM	natural organic matter
NSF	NSF International
NTP	National Toxicology Program
PAC	powdered activated carbon
PACl	polyaluminum chloride
PCD	post-conception day
PCE	polychromatic erythrocytes
PCR	polymerase chain reaction
PMRA	Pest Management Regulatory Agency
PND	post-natal day
RSSCT	rapid small-scale column tests
SCC	Standards Council of Canada
T3	triiodothyronine
T4	thyroxine
TSH	thyroid stimulating hormone
U.S. EPA	United States Environmental Protection Agency
UF	uncertainty factor
UV	ultraviolet
WHO	World Health Organization

Appendix B: Summaries of animal studies using chlorite and sodium chlorite

NOAEL/LOAEL in mg chlorite/kg bw per day (reference)	Study type: Duration method	Dose in mg chlorite/kg bw per day: Statistically significant effects
0.7/9.3 (Haag 1949)	**Chronic cancer: 2 years**	**≥ 9.3:** Altered renal pathology

NOAEL/LOAEL in mg chlorite/kg bw per day (reference)	Study type: Duration method	Dose in mg chlorite/kg bw per day: Statistically significant effects
	Rats, (7/sex/dose) given **chlorite** at 0, 1, 2, 4, 8, 100 or 1 000 mg/L (equivalent to 0, 0.09, 0.18, 0.35, 0.7, 9.3, or 81 mg/kg bw per day) in drinking water for 2 years	**Note:** No carcinogenic effects were seen. Authors concluded renal effects were a nonspecific salt effect.
7.4/18.6 (Harrington et al. 1995a)	**Subchronic: 13 weeks** Rats, Crl:CD(SD)BR (15/sex/dose) gavaged with 0, 10, 25 or 80 mg **sodium chlorite**/kg bw per day (equivalent to 7.4, 18.6 or 59.7 mg **chlorite**/kg bw per day) for 13 weeks	**≥ 18.6:** Increased white blood cell counts in males; decreased red blood cell counts and increased adrenal and spleen weights in females **59.7:** Decreased red blood cell indices, morphological changes in red blood cells in both sexes; increased adrenal and spleen weights in males; increased kidney weights in females **Note:** No effect on terminal body weight or body weight gain, thyroid weights or clinical chemistry. Ulceration of stomach related to irritant properties of sodium chlorite.
71/ND (Kurokawa et al. 1986)	**Subchronic: 85 weeks** Mice, B6C3F1 (50/sex/dose), given **sodium chlorite** at 0, 250 or 500 ppm (0, 36 or 71 mg **chlorite**/kg bw per day) in drinking water	**Note:** No effect on hematological parameters. Increased combined incidences of liver hyperplastic nodules and hepatocellular carcinomas of the liver in low dose group and lung adenomas and adenocarcinomas in high-dose group were statistically significant but within historical control values.
32.1/ND (Kurokawa et al. 1986)	**Subchronic: 85 weeks** Rats, F344 (50/sex/dose), given **sodium chlorite** at 0, 300 or 600 ppm (equivalent to 0, 18.0 or 32.1 mg **chlorite**/kg bw per day in males and 0, 28.3 or 40.9 mg **chlorite**/kg bw per day in females) in drinking water for 85 weeks	**Note:** No effect on hematological parameters or incidence of tumours. Viral infection of all groups led to early study termination.
1/10 (Abdel-Rahman et al. 1980)	**Subchronic: 4 months** Rats, Male Sprague-Dawley (50/sex/dose), given **chlorite** at 0, 10 or 100 mg/L (equivalent to 0, 1 or 10 mg/kg bw per day) in drinking water for 4 months	**10:** Decreased blood glutathione at 2 and 4 months **Note:** Hemolysis was seen in the 10 mg/kg bw per day group at 2 months only. No effect on methemoglobin.
15/ND (Moore and Calabrese 1982)	**Subacute: 30 days** Mice, A/J and C57L/J (14-16/strain/dose), given **sodium chlorite** at 0, 1.0, 10 or 100 mg/L (equivalent to 0, 0.15, 1.5 or 15 mg **chlorite**/kg bw per day) in drinking water for 30 days	**Note:** Although statistically significant differences were observed between mouse strains, chlorite had no effect on all 11 hematologic parameters tested.
ND/ND (Bercz et al. 1982)	**Subacute: 30–60 days** Monkeys, African green (5 males, 7 females) given **sodium chlorite** in	Statistically significant, dose-dependent decreased red blood cell indices and increased serum transaminase were observed but

NOAEL/LOAEL in mg chlorite/kg bw per day (reference)	Study type: Duration method	Dose in mg chlorite/kg bw per day: Statistically significant effects
	exponentially rising step doses of 0, 25, 50, 100, 200 or 400 mg/L (top dose equivalent to 58.4 mg **chlorite**/kg bw per day) in drinking water for 30–60 days	presentation of data did not permit identification of threshold doses **Note:** No effects on thyroid hormones.
2.9/5.9 **(CMA 1996; Gill et al. 2000)** **KEY STUDY**	**Two-generation** Rats, Sprague-Dawley given **sodium chlorite** at 0, 35, 70, 300 ppm in drinking water **F0:** (30/sex/dose) equivalent dose Male/Female - 0/0, 3.0/3.8, 5.6/7.5, 20.0/28.6 mg **chlorite**/kg bw per day starting 10 weeks prior to breeding, throughout mating (males sacrificed) and to weaning (females only) **F1:** (25/sex/dose) received same dosing regime as parents (equivalent to Male/Female - 0/0, 2.9/3.8, 5.9/7.9, 22.7/28.6 mg **chlorite**/kg bw per day); F1 rats were mated at 14 weeks of age to produce F2a. Owing to the reduced number of litters, F1 rats were re-paired following weaning of F2a to produce F2b. Dosing was discontinued at weaning for F1, F2a and F2b pups undergoing neurotoxicological testing.	**F0 – Females** **≥ 7.5:** Decreased absolute and relative liver weights **F1 – Males** **≥ 2.9:** Altered red blood cell indices in parents* **≥ 5.9:** Delayed preputial separation in pups; decreased absolute and relative liver weights; decreased absolute brain weight; changes in red blood cell indices in pups***** **22.7:** Decreased white blood cell counts; altered red blood cell parameters; decreased final body weight **F1 – Females** **≥ 3.8:** Changes in red blood cell indices in pups and parents*****; decreased white blood cell counts in pups*****; increased methemoglobin in pups***** **≥ 7.9:** Decreased absolute brain weight **28.6:** Decreased absolute and relative liver weights; delayed vaginal opening in pups; decreased white blood cell counts in parents; altered red blood cell indices in parents and pups **F2a – Both sexes** **22.7/28.6:** Delayed eye opening **F2b – Both sexes** **≥ 5.9/7.9:** Decreased maximum response amplitude in auditory startle test on post-natal day 24 but not 60 **F2 – Both sexes** **≥ 5.9/7.9:** Decreased absolute brain weight **Note:** F0 or F1 parental animals showed no effect on clinical signs, mortality, estrous cycle, sperm motility or morphology, mating or fertility indices, or reproductive tissues. No changes in number of pups born, pup sex ratio, live birth index or pup survival indices, or anogenital distance or gross external malformations in pups. No effect on total T3 or T4 levels in F1 parents or pups or on functional observation battery, motor activity or swim maze in F1 pups (both sexes) *****Although hematological changes seen at 35 and 70 ppm were statistically significant, they were within historical ranges.

NOAEL/LOAEL in mg chlorite/kg bw per day (reference)	Study type: Duration method	Dose in mg chlorite/kg bw per day: Statistically significant effects
3/6 **(Mobley et al. 1990)** **SUPPORT FOR KEY STUDY**	**One-generation** Rats, female Sprague-Dawley (12/dose) given **chlorite** at 0, 20 or 40 ppm (equivalent to 0, 3 or 6 mg **chlorite**/kg bw per day) in drinking water starting at 10 days pre-mating until post-conception day (PCD) 42; males exposed only during 5-day cohabitation	**3:** Decreased exploratory activity at PCD 36 and 37 but not on PCD 38-40 **6:** Decreased exploratory activity at PCD 36-39 but not PCD 40; increased free T4 on PCD 42 **Note:** No effect on litter size, sex ratios, pup weight or weight gain, or day of eye opening.
39.6/ND **(Harrington et al. 1995b)**	**Reproductive/developmental** Rabbits, New Zealand White, (16 pregnant dams/ dose) given **sodium chlorite** at 0, 200, 600 or 1 200 ppm (estimated at 0, 9.7, 26.5 or 39.6 mg **chlorite**/kg bw per day) in drinking water from GD 7 to day 19; dams sacrificed on GD 28	**≥ 9.7:** Decreased maternal water intake associated with palatability **≥ 39.6:** Decreased maternal food consumption; transient decrease in maternal weight gain **Note:** No clinical signs, changes in absolute body weight, reproductive parameters or histopathology in maternal animals. No external, visceral or skeletal fetal abnormalities.
ND/70 **(Couri et al. 1982a)**	**Reproductive** Rat, Sprague Dawley (13 pregnant dams/dose) given 0.1, 0.5 or 2% **sodium chlorite** [estimated as 70, 440 or 610 mg/kg bw per day of **chlorite** (ATSDR 2004)] in drinking water or gavaged with **sodium chlorite** at 200 mg/kg (equivalent to 157 mg **chlorite**/kg bw) from GD 8–15	**≥ 70:** Decreased fetal crown-rump length; increased number of resorbed and dead fetuses **≥ 440:** Hemolysis **157 (gavage):** Vaginal and urethral bleeding in dams **Note:** No effect on fetal weights, postnatal growth or soft tissue and skeletal malformations or maternal mortality.
0.75/7.5 **(Carlton et al. 1987; U.S. EPA 1998)**	**Reproductive/developmental** Rat, Long-Evans (12 males/dose; 24 females/dose) given 0, 1, 10 or 100 ppm of **sodium chlorite** [estimated as 0, 0.075, 0.75 or 7.5 mg **chlorite**/kg bw per day (U.S. EPA 1998)] in drinking water during pre-mating, mating until 10 post breeding for males and until weaning on lactation day 21 for females	**7.5:** Decreased T3 and T4 in pups **Note:** No effect on parental body weight, water intake, fertility, reproductive tracts (both sexes), sperm concentration and morphology, or hematological parameters. No effect on pup median day of vaginal patency or eye opening.
0.75/7.5 **(Carlton et al. 1987; U.S. EPA 1998)**	**Reproductive** Rat, Long-Evans (12 males/dose) given 0, 10, 100 or 500 ppm of **sodium chlorite** (estimated as 0, 0.75, 7.5 or 27 mg **chlorite**/kg bw per day of **chlorite** by U.S. EPA 1998) in drinking for 72–76 days	**≥ 7.5:** Decreased sperm motility and increased abnormal sperm morphology **Note:** No effect on body weight.
ND/ND **(Moore et al. 1980)**	**Reproductive**	**23:** Decreased average weight of pups at weaning and average birth to weaning growth rate

NOAEL/LOAEL in mg chlorite/kg bw per day (reference)	Study type: Duration method	Dose in mg chlorite/kg bw per day: Statistically significant effects
	Mouse, A/J (10/dose) given **sodium chlorite** at 0 or 100 ppm (equivalent to 0 or 23 mg **chlorite**/kg bw per day) from conception day 1 until weaning at 28 days	**Note:** No effect on gestation time, litter size.
0.1/1.0 **(Suh et al. 1983)**	**Reproductive** Rat, Sprague-Dawley (6–9 females/dose) given **chlorite** at 0, 1 or 10 mg/L (equivalent to 0, 0.1 or 1.0 mg **chlorite**/kg bw per day) in drinking water for 2.5 months pre-mating until GD 20	**1.0:** Increased crown-rump length **Note:** Incidences of skeletal anomalies were increased but were not statistically significant. No effect on fetal weight or reproductive parameters.

ATSDR – Agency for Toxic Substances and Disease Registry; CMA – Chemical Manufacturers Association; GD – gestation day; LOAEL – lowest-observed-adverse-effect level; ND – not determined; NOAEL – no-observed-adverse-effect level; PCD – post-conception day; T3 – triiodothyronine; T4 – thyroxine; U.S. EPA – United States Environmental Protection Agency.

Appendix C: Summaries of animal studies using chlorate and sodium chlorate

NOAEL/LOAEL in mg/kg bw per day unless otherwise stated (references)	Study type: Duration method	Dose in mg/kg bw per day unless otherwise stated: Statistically significant effects
ND/5 **(NTP 2005)** **KEY STUDY**	**Chronic: 2 years** Rats, F334/N (50/sex/dose) given **sodium chlorate** at 0, 125, 1 000 or 2 000 mg/L (equivalent to 0, 5, 35, or 75 mg **chlorate**/kg bw per day for males and 0, 5, 45, or 95 mg **chlorate**/kg bw per day for females) in drinking water for 2 years	**Males** **≥ 5:** Increased thyroid gland follicular cell hypertrophy **≥ 35:** Increased bone marrow hyperplasia **75:** Increased hematopoietic cell proliferation in spleen **Females** **≥ 45:** Increased thyroid gland follicular cell hypertrophy and mineralization **Both sexes** **75/95:** Increased TSH **Note:** No effect on survival, drinking water intake or mean body weights or clinical signs. T3 and T4 levels were decreased at day 4 (≥ 34/45) and week 3 (≥ 75/95) but not at week 14.
ND/30	**Chronic: 2 years**	**Females**

NOAEL/LOAEL in mg/kg bw per day unless otherwise stated (references)	Study type: Duration method	Dose in mg/kg bw per day unless otherwise stated: Statistically significant effects
(NTP 2005)	Mice, B6C3F1 (50/sex/dose) given **sodium chlorate** at 0, 500, 1 000 or 2 000 mg/L (equivalent to 0, 40, 80, or 160 mg **chlorate**/kg bw per day for males and 0, 30, 60, or 120 mg **chlorate**/kg bw per day for females) in drinking water for 2 years	**≥ 30:** Increased bone marrow hyperplasia; decreased body weights **120:** Increased thyroid gland follicular cell hypertrophy **Note:** No effects seen in males, including incidences of tumours. For females, no effect on survival or drinking water intake in females but positive trend in incidences of pancreatic islet cell adenoma or carcinoma (combined).
1.0/2.0 (in g/L) (Hooth et al. 2001)	**Subchronic: 105 days** Rats, female F344 (6/dose) given **sodium chlorate** at 0, 0.05, 1.0, 2.0, 4.0 or 6.0 g/L in drinking water for 105 days	**≥ 2.0 g/L:** Decreased thyroid colloid, increased incidence and severity of follicular cell hyperplasia **6.0 g/L:** Increased thyroid hypertrophy
6.0/ND (in g/L) (Hooth et al. 2001)	**Subchronic: 105 days** Mice, B6C3F1(6/dose) given **sodium chlorate** at 0, 0.05, 1.0, 2.0, 4.0 or 6.0 g/L in drinking water for 105 days	**Note:** No effect on thyroid histopathology.
ND/0.001 (in g/L) (Hooth et al. 2001)	**Subchronic: 3 months** Rats, male F344 (10/dose) given **sodium chlorate** at 0, 0.001, 0.01, 0.1, 1.0, 2.0 g/L in drinking water for 90 days	**≥ 0.001 g/L:** Decreased thyroid colloid, increased thyroid hypertrophy **≥ 1.0 g/L:** Increased incidences and severity of thyroid gland follicular cell hyperplasia
30/100 (McCauley et al. 1995)	**Subchronic: 90 days** Rats, Sprague-Dawley males given **sodium chlorate** at 0, 3.0, 12.0, or 48.0 mM (equivalent to at 0, 30, 100 or 510 mg **chlorate**/kg bw per day for males and 0, 42, 164 or 800 mg **chlorate**/kg bw per day for females) in drinking water for 90 days	**Males** **510:** Decreased relative heart, kidneys and liver weights; increased relative testes weights; decreased AST, ALT, calcium, creatinine and phosphorus; increased serum cholesterol **Females** **800:** Decreased relative adrenals, thymus and spleen weights; pituitary changes **Both sexes** **≥ 100/164:** Thyroid gland colloid depletion **510/800:** Decreased final body weight gain; increased relative brain weight; decreased hematocrit and red blood cell counts; decreased white blood cell count

NOAEL/LOAEL in mg/kg bw per day unless otherwise stated (references)	Study type: Duration method	Dose in mg/kg bw per day unless otherwise stated: Statistically significant effects
		Note: No clinical or behavioural effects in any group.
79/788 **(Bio/dynamics Inc. 1987a)**	**Subchronic: 3 months** Rats, Sprague-Dawley (14/sex/dose) gavaged with **sodium chlorate** at 0, 19, 100 or 1 000 mg/kg bw per day (equivalent to 0, 8, 79 or 788 mg **chlorate**/kg bw per day) for 3 months	**788:** Decreased red blood cell count, hemoglobin, hematocrit **Note:** No effect on mortality, behaviour, physical appearance, body weight, food consumption, clinical chemistry, gross necropsy or organ histopathology.
ND/ND **(Bercz et al. 1982)**	**Subacute: 30–60 days** Monkeys, African green (5 males, 7 females) given sodium **chlorate** in exponentially rising step doses of 0, 25, 50, 100, 200 or 400 mg/L [equivalent to 4, 75, 30 or 58.4 mg chlorate/kg bw per day (IPCS 2000)] in drinking water for 30–60 days	Statistically significant, dose-dependent decreased red blood cell count and cell indices and increased serum transaminase were observed but presentation of data did not identify threshold doses **Note:** No effects on thyroid hormones.
ND/20 **(NTP 2005)**	**Subacute: 3 weeks** Rats, F334/N (51/sex/dose) given **sodium chlorate** at 0, 125, 250, 500, 1 000 or 2 000 mg/L (equivalent to 0, 20, 35, 75, 170, or 300 mg **chlorate**/kg bw per day for males and 0, 20, 40, 75, 150, or 340 mg **chlorate**/kg bw per day for females) in drinking water for 3 weeks	**Males** **≥ 75:** Increased incidences and severity of thyroid gland follicular cell hypertrophy and hyperplasia; decreased thyroid colloid **300:** Decreased relative and absolute heart weights; decreased absolute kidney weight; decreased hematocrit, red blood cell counts, platelets and monocytes **Females** **≥ 150:** Increased incidences and severity of thyroid gland follicular cell hypertrophy and hyperplasia; decreased thyroid colloid **Both sexes** **≥ 20:** Decreased segmented neutrophil counts in both sexes **300/340:** Decreased hemoglobin **Note:** No effect on survival, water intake or mean body weights, clinical findings or clinical chemistry were observed.
175/350 **(NTP 2005)**	**Subacute: 3 weeks** Mice, B6C3F1 (10/sex/dose) given **sodium chlorate** at 0, 125, 250, 500, 1 000 or 2 000 mg/L (equivalent to 0, 20, 45, 90, 175 or 350 mg	**Males** **350:** Decreased eosinophils **Both sexes** **350/365:** Decreased hemoglobin

NOAEL/LOAEL in mg/kg bw per day unless otherwise stated (references)	Study type: Duration method	Dose in mg/kg bw per day unless otherwise stated: Statistically significant effects
	chlorate/kg bw per day for males and 0, 20, 45, 95, 190 or 365 mg **chlorate**/kg bw per day for females) in drinking water for 3 weeks	**Note:** No effect on survival, mean body weights, organ weights, water consumption or exposure-related lesions.
ND/2.5 **(Khan et al. 2005)**	**Subacute: 7 days** Rats, Male F344 (10/dose) given **sodium chlorate** at 0, 10, 100, 1 000 mg/L (equivalent to 0, 2.5, 11.9, 93.1 mg **chlorate**/kg bw per day) in drinking water **Purity:** > 99%	**≥ 2.5:** Increased thyroid follicular epithelial cell hypertrophy **93.1:** Increased TSH **Note:** No signs of clinical toxicity or mortality. No effect on body weight, water intake or T3 or TSH levels.
0.1/1 **Suh et al. 1983**	**Reproductive** Rats, female Sprague-Dawley (8–9/dose), given **chlorate** at 0, 1 or 10 mg/L [equivalent to 0.1 or 1 mg chlorate/kg bw per day (NTP 2002)] in drinking water for 10 weeks pre-mating until GD 20	**Male pups** **1:** Increased crown-rump length **Note:** No affect on maternal survival, body weight, pregnancy rate, total number of implantations per dam, or number of live, resorbed or dead fetuses. Incidences of external, visceral or skeletal fetal malformations were unaffected.
780/ND **(Bio/dynamics Inc. 1987b)**	**Reproductive** Rats, female Sprague-Dawley (6–9/dose), gavaged with **sodium chlorate** at 0, 10, 100 or 1 000 mg/kg bw per day (equivalent to 0, 7.8, 78, 780 mg **chlorate**/kg bw per day) on GD 6–15	**Note:** No maternal deaths, no effect on maternal body weight or weight gain or food consumption, clinical signs, number of uterine implantations or gross necropsy. Fetal body weight and sex ratio, and external, visceral, or skeletal abnormalities were unaffected.
31/156 (thyroid) **156/780 (fetal effects)** **780/ND (reproductive performance)** **(EFSA 2015)**	**One-generation** Rats, Sprague-Dawley (6/sex/dose), gavaged with **sodium chlorate** at 0, 40, 200 or 1 000 mg/kg bw per day (equivalent to 0, 31, 156, 780 mg **chlorate**/kg bw per day) from age 6 weeks through 10 weeks pre-mating and during mating (both sexes) and through pregnancy and lactation (females); litters culled to 4/sex on post partum day 4	**F0** **≥ 156:** Thyroid epithelial cell hyperplasia in parental males **780:** Thyroid epithelial cell hyperplasia in females; increased incidence and severity of vacuolated pituitary gland cells **F1** **780:** Decreased fetal body weight and body weight gain **Note:** In parental animals, no clinical signs relate to treatment and no adverse effects on reproduction were seen.
8/55 (thyroid)	**Two-generation**	**55:** Increased incidences of thyroid follicular hyperplasia and follicular

NOAEL/LOAEL in mg/kg bw per day unless otherwise stated (references)	Study type: Duration method	Dose in mg/kg bw per day unless otherwise stated: Statistically significant effects
390/ND (development) (EFSA 2015)	Rats, Sprague-Dawley (25/sex/dose), gavaged with **sodium chlorate** at 0, 10, 70 or 500 mg/kg bw per day (equivalent to 0, 8, 55, 390 mg **chlorate**/kg bw per day) from age 6 weeks, through 10 weeks pre-mating and during mating (both sexes) and through pregnancy and lactation (females); F1 pups were culled to 4/sex on postpartum day 4 and weaned on postpartum day 21 and randomly selected to produce F2 litters	hyperactivity in both sexes of F0 and F1 rats **Note:** No change in estrus cycle, female reproductive parameters or sperm parameters in F0 and F1 parents. No effect on survival or development in F0 or F1 pups. No thyroid lesions in F2 pups.
372/ND (NTP 2002)	**Developmental** Rabbits, female New Zealand white (24/dose), gavaged with **sodium chlorate** at 0, 100, 250 or 475 mg/kg bw per day (equivalent to 0, 78, 196 or 372 mg **chlorate**/kg bw per day) on GD 6–29	**Note:** No effect on maternal body weight, body weight gain, or liver and gravid uterine weight. No effect on resorptions, fetal viability, body weight, or external, visceral or skeletal alterations. No developmental toxicity.

ALT – alanine aminotransferase; AST – aspartate aminotransferase; EFSA – European Food Safety Authority; GD – gestation day; LOAEL – lowest-observed-adverse-effect level; ND – not determined; NOAEL – no-observed-adverse-effect level; NTP – National Toxicology Program; T3 – triiodothyronine; T4 – thyroxine; TSH – thyroid stimulating hormone.

Appendix D: Summaries of animal studies using chlorine dioxide

NOAEL/LOAEL in mg/kg bw per day (reference)	Study type: Duration method	Dose in mg/kg bw per day: Statistically significant effects
1.3/13* **(Haag 1949)** ***Frank effect level**	**Chronic cancer: 2 years** Rats, (7/sex/dose) given **ClO_2** at 0, 0.5, 1, 5, 10 or 100 mg/L (equivalent to 0, 0.07, 0.13, 0.7, 1.3, or 13 mg/kg bw per day) in drinking water for 2 years	**13:** Decreased survival rate and mean life span **Note:** Treated animals did not show any histopathological effects or increased incidences of tumours.
1/10 **(Suh et al. 1984)**	**Chronic: 1 year** Rats, male Sprague-Dawley given **ClO_2** at 0, 1, 10 or 100 mg/L (equivalent to 0, 0.1, 1 or 10 mg/kg bw per day) in drinking water for 1 year	**10:** Increased liver enzyme activity (aniline hydroxylase)
ND/0.1 **(Abdel-Rahman et al. 1984a)**	**Chronic: 1 year** Rats, male Sprague-Dawley (4/dose) given **ClO_2** at 0, 1, 10, 100 or 1 000 mg/L (equivalent to 0, 0.1, 1, 10 or 100 mg/kg bw per day by U.S. EPA, 2000) in drinking water for 12 months; blood sampled at 2, 4, 7 and 9 months	**≥ 0.10:** Decreased hematocrit and hemoglobin; decreased body weight **≥ 1:** Decreased red blood cell osmotic fragility **≥ 10:** Increased MCHC **100:** Decreased red blood cell count **Note:** Other statistically significant hematological changes were inconsistent over

		time and/or did not appear to be dose related.
ND/2.4 **(Daniel et al. 1990)**	**Subchronic: 90 days** Rats, Sprague-Dawley (10/sex/dose) given **ClO_2** at 0, 25, 50, 100 or 200 mg/L (equivalent to 0, 1.9, 3.6, 6.2, 11.5 mg/kg bw per day in males and 0, 2.4, 4.6, 8.2 or 14.9 mg/kg bw per day in females) in drinking water for 90 days	**Males** **≥ 3.6:** Decreased liver weights; decreased water intake **≥ 6.2:** Increased creatinine; decreased AST; decreased LDH **11.5:** Decreased final body weights and body weight gain; decreased food intake **Females** **≥ 2.4:** Decreased spleen weights; decreased water intake **14.9:** Decreased final body weights and body weight gain
ND/3.5 **(Bercz et al. 1982)**	**Subacute: 30–60 days** Monkeys, African green (5 males, 7 females) given **ClO_2** in exponentially rising step doses of 0, 30, 100, 200 mg/L (equivalent to 0, 3.5, 9.5, 9*) in drinking water for 30–60 days *dose discontinued after 1 week due to mucosal irritation	**≥ 3.5:** Decreased T4 **Note:** Thyroid effects were reversible once exposure stopped. No effects on drinking water intake (low and medium dose groups), on hematological parameters, liver/kidney function, or body weights.
15/ND **(Moore and Calabrese 1982)**	**Subacute: 30 days** Mice, A/J and C57L/J (10/strain/dose), given **ClO_2** at 0 or 100 mg/L (equivalent to 0 or 15 mg/kg bw per day) in drinking water for 30 days	**Note:** Although statistically significant differences were observed between mouse strains, ClO_2 had no effect on all 11 hematologic parameters tested.
5/10 **(Carlton et al. 1991)**	**One-generation** Rats, Long-Evans (12 males/dose; 24 females/dose) gavaged with **ClO_2** in water at 0, 2.5, 5 or 10 mg/kg bw per day for up to 73 days; Pups dosed until weaning on lactation day 21	**10:** Depressed absolute and relative vaginal weights in female F1 pups **Note:** No clinical signs, no effect on sperm or reproductive parameters in parental generation. No effect on litter size, pup viability or pup weight. No effect on thyroid parameters.
3/14 **(Orme et al. 1985)** **Aligns with chlorite key study for effects**	**Neurodevelopmental** Indirect pup dosing Rats, pregnant Sprague-Dawley (8 male pups/litter) given **ClO_2** at 0, 2, 20 or 100 mg/L (equivalent to 1, 3 or 14 mg/kg bw per day) in drinking water from gestation to weaning at PND 21 Direct pup dosing pups from unexposed dams were gavaged with **ClO_2** 14 mg/kg bw per day from age 5 to 20 days; Locomotor activity was assessed between ages 14–21 days **Note:** Positive control dosed with propylthiouracil, which suppresses thyroid hormone production.	**Indirect pup dosing** **14:** Decreased T4; increased T3 **Note:** Pup locomotor levels were consistently lower than controls but were not statistically significant. **Direct pup dosing** **14:** Depressed locomotor activity PND 18 and 19; decreased T4; decreased pup body weight on PND 14 and 21 **Note:** Maternal body weights and T4 and T3 levels were unaffected in exposed dams. Route of exposure had no effect on day of pup eye opening or pup birth weight.

0.7/7 **Suh et al. 1983**	**Reproductive** Rats, female Sprague-Dawley, given **ClO_2** at 0, 1, 10 and 100 mg/L (equivalent to 0, 0.07, 0.7 or 7 mg/kg bw per day) of ClO_2 in drinking water for 10 weeks pre-mating until GD 20	**7:** Increased fetal weights **Note:** No clinical signs or increased mortality in dams. Decreased, non-statistically significant maternal body weight gain was seen at ≥ 0.7 mg/kg bw per day and was associated with decreased water intake. No litter anomalies were seen. Skeletal defects seen were not statistically different from control group. At 7 mg/kg bw per day non-significant slight decrease in mean number of implants per dam and number of live fetuses were seen and showed statistically significant dose-dependent trends.

AST – aspartate aminotransferase; ClO_2 – chlorine dioxide; GD – gestation day; LDH – lactate dehydrogenase; LOAEL – lowest-observed-adverse-effect level; MCHC – mean corpuscular hemoglobin concentration; NOAEL – no-observed-adverse-effect level; ND – not determined; PND – post-natal day; T3 – triiodothyronine; T4 – thyroxine.

Appendix E: Recommendations for the handling and storage of hypochlorite solutions

Several key factors have been identified that impact the formation of perchlorate, bromate, and other contaminants in hypochlorite solutions. The major factors impacting perchlorate formation parallel those that also affect the decomposition of hypochlorite: temperature, ionic strength, concentration, and pH. By using the information gathered in the study referenced subsequently and by applying the "Predictive Model" to hypothetical liquid hypochlorite storage scenarios, several quantitative and qualitative recommendations can be made:

1. Dilute stored hypochlorite solutions upon delivery. The decomposition of hypochlorite and subsequent formation of chlorate and perchlorate is dependent upon hypochlorite concentration and ionic strength. Higher ionic strength and hypochlorite concentration will drive the reaction towards a greater production of chlorate and perchlorate while also increasing the rate of decomposition of hypochlorite. By diluting a 2 M hypochlorite solution by a factor of 2, the rate of perchlorate formation decreases by a factor of 7 because of the combination of concentration and ionic strength effects. A fourfold dilution of a hypochlorite solution will decrease the rate of formation by a factor of 36. A tenfold dilution of a hypochlorite solution will decrease the rate of perchlorate formation by a factor of 270.

2. Store the hypochlorite solutions at lower temperatures. Higher temperatures speed up the chemical decomposition of hypochlorite and the subsequent formation of chlorate and perchlorate. Every 5 °C (9 °F) reduction in storage temperature will reduce the rate of perchlorate formation by a factor of approximately 2. To minimize temperature increases, the product should be stored out of direct sunlight.

3. Control the pH of stored hypochlorite solutions at pH 11–13 even after dilution. Storage of concentrated hypochlorite solutions at pH values lower than 11 is not recommended because of

accelerated decomposition of hypochlorite ion/hypochlorous acid and the subsequent formation of chlorate, even though lower pH can reduce the amount of perchlorate formed. When the pH is higher than 13, perchlorate formation is enhanced because of the ionic strength effect. As such, utilities should continue to insist that manufacturer specifications include pH control in the range of 11−13. Given the typical pH range of on-site generation (OSG) hypochlorite (9−10), such solutions should be used as soon as possible after manufacture and should not be stored for more than one to two days.

4. Control the removal of transition metal ions by purchasing filtered hypochlorite solutions and by using low-metal ion concentration feedwater for the OSG systems and dilution water. The presence of transition metal ions results in an increased degradation rate of hypochlorite. While this degradation is concomitant with reduced perchlorate formation, the free available chlorine concentration is also reduced, forcing a utility to use a higher volume of a hypochlorite solution, which results in higher mass loading of contaminants such as perchlorate, chlorate, and bromate.

5. Use fresh hypochlorite solutions when possible. Hypochlorites will naturally decompose to produce oxygen, chlorate, and perchlorate. Less storage time will minimize the formation of these contaminants in the hypochlorite solution. Rotate stock and minimize the quantity of aged product in storage tanks before the delivery of new product. A fresh hypochlorite solution will also contain a higher concentration of hypochlorite, thereby reducing the amount of solution required to obtain the target chlorine residual. Again, higher hypochlorite concentration in a fresh hypochlorite solution will correspond to lower concentrations of contaminants dosed.

6. For utilities using OSG hypochlorite, use a low-bromide salt to minimize the amount of bromide present in the brine. Bromate formation will occur rapidly in hypochlorite solutions in the presence of bromide. By controlling the amount of bromide in the salt and source water used for OSG, bromate formation can be minimized.