



Canadian Water Quality Guidelines for the Protection of Aquatic Life

REACTIVE CHLORINE SPECIES

The chemistry of chlorine in fresh and marine waters is very complex, and numerous terms describing the various reactive forms of chlorine species are used in the literature, often defining related aspects of the chemistry. Simplifications and generalizations are often used in the analysis, toxicity testing, and reporting, potentially leading to confusion, oversimplifications, and errors in the interpretation of the analytical and toxicological results.

Reactive chlorine (RC) is the combined concentration of various chlorine species able to react and interconvert in a given system. It is essentially synonymous with total residual chlorine (TRC), combined residual chlorine (CRC), and total available chlorine (TAC). It includes free available chlorine (FAC; hypochlorous acid [HOCl] and the hypochlorite ion [OCl⁻]; also referred to as free residual chlorine [FRC]) and combined available chlorine (CAC; organic and inorganic chloramines [NH₂Cl, NHCl₂, and NCl₃] or *N*-chloramides). In ambient freshwater, the dominant reactive chlorine species are either hypochlorous acid and its associated hypochlorite anion or monochloramine, if a sufficient source of nitrogen/ammonia is present. In marine or estuarine water, the presence of bromide ions (Br⁻) causes rapid partial or complete replacement of chlorine by bromine (i.e., hypobromous acid [HOBr] and its hypobromite anion [OBr⁻], bromamines, and bromochloramines). The collection of these reactive chlorine and bromine species is called chlorine-produced oxidants (CPO). Hypochlorous acid, the hypochlorite ion, and chloramines are chemically interconvertible and can co-occur in the environment; the respective dominant species is determined by site-specific conditions. The species-specific analytical determination, especially at the low levels of toxicological relevance (i.e., low microgram per litre concentrations), is difficult and sometimes not possible or practical. Therefore, the development of separate guidelines for hypochlorous acid, indicative of FAC, and monochloramine, indicative of CAC, was deemed inappropriate. Instead, a guideline for reactive chlorine species, i.e., TRC, the combination of FAC and CAC, was derived for freshwater, and a guideline for chlorine-produced oxidants (CPO) was derived for marine waters.

The addition of FAC to waters containing ammonia produces predominantly monochloramine, but

dichloramine and, more rarely, trichloramine can also be found, i.e., CAC. If sufficient FAC is added, the available ammonia is sequentially oxidized into monochloramine, dichloramine, and trichloramine, and then into nitrogen gas or nitrate (Environment Canada 1999). Excess chlorine then exists as FAC. Site-specific conditions, such as elevated temperature, high pH, intense light, elevated organic carbon or organic nitrogen content are positively correlated to the degradation rate of reactive chlorine species.

Residual chlorine concentrations are reduced through several chemical and physical pathways, such as reactions in the water column, volatilization, photodegradation, adsorption, and interactions with aquatic biota and the benthos. Temperature, pH, stream depth, flow rate, ice cover, sediment composition, turbidity, etc., all influence the natural decay. The degradation rates of reactive chlorine vary considerably, ranging from seconds, to minutes and hours, and even days, depending on the kind of effluent (drinking water, wastewater effluent, cooling water, etc.), and the local physical, chemical, and biological characteristics and conditions of the receiving environment (e.g., river, creek, lake, ocean).

Reactive chlorine species in runoff from common domestic water uses (e.g., lawn watering, car washing, swimming pool draining, and driveway or road flushing) can be carried for some distance and can result in measurable concentrations in the storm sewer system both from chlorine and chloramine. Considerable concentrations (0.5 mg·L⁻¹ with chloraminated water, 37%

Table 1. Water quality guidelines for reactive chlorine species (freshwater) and chlorine-produced oxidants (marine water) for the protection of aquatic life (Environment Canada 1999).

Aquatic life	Guideline value (µg·L ⁻¹)
Freshwater	
Reactive chlorine species	0.5*
Marine	
Chlorine-produced oxidants [†]	0.5*

* If chemical species-specific identification is available, the guideline applies to the sum of all reactive chlorine species concentrations (i.e., hypochlorous acid, monochloramine, and others).

[†] The marine water quality guideline is expressed as chlorine-produced oxidants, which are essentially the reactive chlorine species formed under marine conditions.

of the initial concentration; and 0.14 mg·L⁻¹ with chlorinated water, 21% of the initial concentration) were still measurable close to 400 m and 200 m, respectively, from the use sites (Norecol Environmental Consultants Ltd. and Dayton and Knight Ltd. 1992). During a spill from a broken drinking water main pipe, chloraminated drinking water flowed over a road, ground surface, and storm sewer until it ultimately entered a stream approximately 1 km from the break. Even though the water carried a large, albeit unquantified, load of organic sediment (left on the road from a land-clearing operation), the chloraminated water (concentrations were not measured) caused 100% fish mortality in the small stream (Nikl and Nikl 1992).

In marine and estuarine waters, the presence of bromide ions and the higher reactivity of bromine compared to chlorine leads to the formation of CPO. Reactive chlorine (as FAC or TRC) in marine waters can be reduced to CPO in <10 s (Abarnou and Miossec 1992). In freshwater, bromide concentration is usually low and ammonia concentration generally higher, so monochloramine formation predominates. In marine waters, the bromide concentration is higher (67 mg·L⁻¹ [0.8 mM] in 35‰ seawater), and hypobromous acid (HOBr) is the main species initially produced (Bousher et al. 1989; Bousher et al. 1990). Depending on the ammonia concentrations, monobromamine, dibromamine (NHBr₂), tribromamine, and bromochloramine (NHBrCl) are also formed. Bromamines are less stable than monochloramine or bromine, with faster decomposition rates (Cooper et al. 1985) and higher toxicity toward aquatic organisms (Fisher et al. 1999). The overall result is an enhanced reactivity exhibited by chlorine in marine and estuarine waters (Morris 1978). In estuarine waters, intermediate situations may arise, as salinity and, hence, bromide concentration will be lower, and elevated ammonia levels due to pollution are more common. The formation of monochloramine is thus more likely than in marine waters (Bousher et al. 1989).

The four main sources of reactive chlorine species to the environment are treated wastewater effluents, chlorinated cooling water effluents, spills due to breaks in the drinking water distribution system, and uncollected releases of drinking water.

In 1992, approximately 61% of the effluents from 2800 municipal wastewater treatment plants in Canada (>1 × 10⁷ m³·d⁻¹) were chlorinated for disinfection purposes before discharge, mostly in Ontario, Manitoba, and British Columbia. The reactive chlorine species levels observed in the final effluents generally are below

1 mg·L⁻¹, but can range up to 3–7 mg·L⁻¹ under certain circumstances (Government of Canada 1993; Environment Canada 1998).

Very little information is available on residual chlorine levels in industrial wastewater effluents. Chlorination of wastewater and/or cooling water is practiced, among others, in meat processing plants, fish and poultry processing plants, natural gas plants, petroleum refineries, pulp and paper, and petrochemical industries. Levels, however, are expected to be low (Government of Canada 1993).

Chlorination of cooling water for industrial plants (mostly power generating stations) is commonly done in Newfoundland, Nova Scotia, Quebec, Ontario, Alberta, and British Columbia. Reactive chlorine is applied as an antifouling agent and for zebra mussel control. It is either added to the cooling water continuously at a concentration of approximately 1 mg·L⁻¹, or intermittently (ranging from 30-min to 2-h periods in regular or irregular daily, weekly, monthly, or seasonal intervals) at concentrations of up to 3 mg·L⁻¹ to maintain a minimum reactive chlorine species level of 0.5 mg·L⁻¹ in the system (Orvos et al. 1986; Abarnou and Miossec 1992). Release of the treated cooling water is either continuous or intermittent during short time intervals, sometimes two or three times per day, and in most cases after a total chlorine application time of <2 h (Orvos et al. 1986; Meyer et al. 1995). The reactive chlorine species level at the outfall is generally below 0.4 mg·L⁻¹, but can be as high as 3 mg·L⁻¹ (Government of Canada 1993; Meyer et al. 1995). Upon mixing with the receiving waters, site-specific conditions will determine the final concentration, composition, and further persistence. The toxicity of such releases has been extensively documented through numerous observed fish and phytoplankton kills in the receiving waters (Mattice and Zittel 1976).

Free available chlorines are mainly used as primary disinfectants in drinking water purification, while the longer-lived chloramines are mainly used for secondary disinfection to maintain an effective chlorination potential within the drinking water distribution network. Typical concentrations for reactive chlorine species range from approximately 0.8 to 4.0 mg·L⁻¹ at the plant, ensuring a minimum concentration of approximately 0.2–2.8 mg·L⁻¹ at every tap in the distribution system. The actual concentrations vary depending on the amount added at the treatment plant, temperature of the water, side reactions while in the pipes, and the length of time the water is in the pipes (Environment Canada 1998, 1999).

Spent drinking water is usually collected with the municipal wastewater stream, and any residual chlorine will soon be used up by the organic loading and, therefore, will not be released to the ambient environment. However, direct release of drinking water into the ambient environment can occur through pipeline breaks in the drinking water distribution system. In these cases, chlorinated or chloraminated water with concentrations of 0.2–4.0 mg·L⁻¹ Cl₂ can enter watercourses with considerable local effects.

Release of chlorinated drinking water into the ambient environment can also occur through noncontained uses such as lawn watering, car washing, driveway flushing, water main cleaning, road flushing, and fire fighting. Other sources include draining of private swimming pools, overflows from water storage reservoirs, and direct discharges by industrial/commercial water uses. In some cases, the released water can still contain reactive chlorine species at concentrations up to the original drinking water concentration range of 0.2–4.0 mg·L⁻¹ Cl₂.

Water Quality Guideline Derivation

The Canadian water quality guidelines for reactive chlorine species for the protection of aquatic life were developed based on the 1991 protocol (CCME 1991). For more details, see the supporting document (Environment Canada 1999).

Freshwater Life

As the different chlorine species can exist simultaneously, but are difficult, if not impossible, to reliably differentiate currently at low concentrations, the guideline has been derived for reactive chlorine species. A guideline of 0.5 µg·L⁻¹ for reactive chlorine species is recommended for the protection of freshwater life. If chemical species-specific identification is available, the guideline applies to the sum of all reactive chlorine species (i.e., hypochlorous acid, monochloramine concentrations, and others).

Percent compositions of CAC versus FAC in toxicity tests were rarely documented. It should be noted that the few studies that attempted to examine FAC exclusively often noted CAC contamination in the test water. This is mainly due to the waste products of the test organisms (Brooks and Bartos 1984; Cairns et al. 1990; Fisher et al. 1994). Therefore, the separation of toxicity results as presented in the following text may be misleading and must be considered with caution.

Hypochlorous Acid

For hypochlorous acid, the lowest reported acute toxicity studies for fish are 24h and 96h LC₅₀s for coho salmon (*Oncorhynchus kisutch*) of 16 and 4 µg·L⁻¹, respectively (Rosenberger 1971), and 6h and 11h LC₅₀s for brown trout (*Salmo trutta*) of 50 and 20 µg·L⁻¹, respectively (Pike 1971). However, the 96-h LC₅₀ of 4 µg·L⁻¹ for coho salmon reported by Rosenberger 1971 was not used for the guideline derivation due to reservations with respect to the analytical methodology and detection limit. More recently, Fisher et al. (1999) reported 96h LC₅₀s of 59 µg·L⁻¹ for rainbow trout (*O. mykiss*) and 304 µg·L⁻¹ for golden shiners (*Notemigonus crysoleucas*). Furthermore, a 48h LC₅₀ (2-h exposure, mortality assessed 48 h later) for emerald shiners (*Notropis atherinoides*) of 100 µg·L⁻¹ (Brooks et al. 1982), and an 11h LC₆₀ for rainbow trout (*O. mykiss*) of 108 µg·L⁻¹ (Basch and Truchan 1974) have been observed. Larson et al. (1978) reported several 96h LC₅₀s for juvenile cutthroat trout (*S. clarki*) of 75, 82, and 94 µg·L⁻¹.

The lowest reported acute toxicity studies for invertebrates are 24h LC₅₀s for water flea neonates (*Ceriodaphnia dubia*) of 5 and 6 µg·L⁻¹ (Taylor 1993). However, the reported detection limit in the study was 10 µg·L⁻¹, and the toxicity concentrations were estimated by dilution from a stock solution. Therefore, the study by Taylor (1993) was not used for the guideline derivation. Also, a 48h LC₅₀ for the amphipod *Gammarus minus* of 23 µg·L⁻¹ (Gregg 1974) and 48h LC₅₀s for the mayflies *Ephemerebella lata* and *Isonychia* sp. and for the stonefly

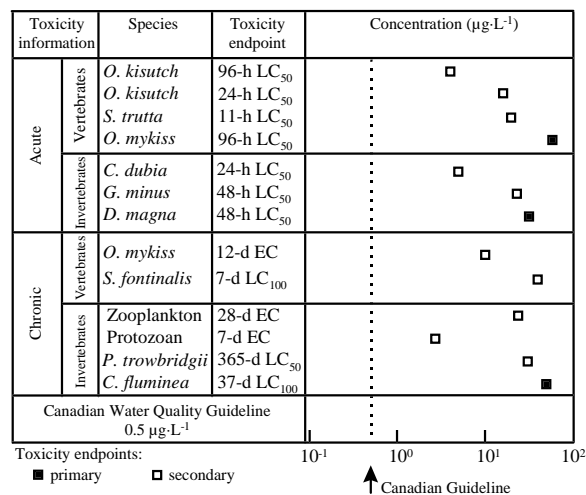


Figure 1. Select freshwater toxicity data for hypochlorous acid.

Peltaperla maria of 27, 9.3, and 20 µg·L⁻¹, respectively (Gregg 1974), have been reported. Fisher et al. (1999) reported a 48h LC₅₀ of 32 µg·L⁻¹ for the water flea *Daphnia magna* and a 96h LC₅₀ of 78 µg·L⁻¹ for the amphipod *Hyalella azteca*.

The aquatic plant water milfoil (*Myriophyllum spicatum*) showed significantly reduced growth after a 96h exposure to 50 µg·L⁻¹ (Watkins and Hammerschlag 1984), while *Chlorella pyreniodosa* showed 50% reduced growth after a 24h exposure to 180 µg·L⁻¹ (Kott et al. 1966).

The lowest reported chronic toxicity studies for fish are a 12-d mortality study for rainbow trout (*O. mykiss*) of 10 µg·L⁻¹ (Sprague and Drury 1969) and depressed activity and an LC₁₀₀ for brook trout (*Salvelinus fontinalis*) of 5 and 40 µg·L⁻¹, respectively, after 7-d exposure (Dandy 1972). However, these studies were not used for the guideline derivation due to reservations with respect to the analytical methodologies and testing protocols.

The lowest reported chronic toxicity studies for invertebrates are reduced zooplankton density in the field and reduced protozoan species numbers in the lab after exposure for 28 d to 24 and 25 µg·L⁻¹, respectively (Pratt et al. 1988), and reduced protozoan species richness after exposure for 7 d to 2.7 µg·L⁻¹ (Cairns et al. 1990). However, the study by Cairns et al. (1990) was not used for the guideline derivation due to reservations with respect to the analytical methodology, toxicant and endpoint determination, and the suitability of the endpoint for guideline derivation. Furthermore, a 365-d LC₅₀ for the crayfish *Pacifastacus trowbridgii* of 31 µg·L⁻¹ (Larson et al. 1978) and a 12-d LT₅₀ and a 37-d LC₁₀₀ for the Asiatic clam *Corbicula fluminea* of 50 µg·L⁻¹ (Belanger et al. 1991) have been reported. Also, Pratt et al. (1988) reported significantly depressed chlorophyll *a* levels in chlorophyta green algae exposed for 28 d to 2.1 µg·L⁻¹. However, this study was not used for the guideline derivation due to reservations with respect to the analytical methodology and the suitability of the endpoint for guideline derivation.

Monochloramine

For monochloramine, the lowest reported acute toxicity studies for fish are 96h LC₅₀s of 14–29 µg·L⁻¹ for rainbow trout (*O. mykiss*) (MDNR 1971), 57 µg·L⁻¹ for coho salmon (*O. kisutch*) (Larson et al. 1977a), and 82 µg·L⁻¹ for brook trout fry (*Salvelinus fontinalis*) (Larson et al. 1977b). An LT₅₀ of 90 µg·L⁻¹ for channel

catfish (*I. punctatus*) (Roseboom and Richey 1977) has also been observed. A 48-h LC₅₀ (2-h exposure) of 80 µg·L⁻¹ for emerald shiners (*N. atherinoides*) (Brooks and Bartos 1984) and a 96h LOEC for lethargic swimming behaviour, reduced growth, and delayed fry stage of 23 µg·L⁻¹ for coho salmon alevins (*O. kisutch*) (Larson et al. 1977a) have also been reported. It should also be noted, that Bogardus et al. (1978) reported avoidance behaviour after a 15-min exposure to 2 µg·L⁻¹ for alewife (*Alosa pseudoharengus*), 10 µg·L⁻¹ for yellow perch (*Perca flavescens*), and 30 µg·L⁻¹ for rainbow smelt (*Osmerus mordax*). Avoidance behaviour, however, is not an acceptable endpoint for guideline derivation.

Acute toxicity studies with low endpoints for invertebrates are a 48h LC₅₀ of 78 µg·L⁻¹ for the Asiatic clam *C. fluminea* (Belanger et al. 1991), a 24h EC₅₀ (growth) of 42 µg·L⁻¹ and a 24h LC₅₀ of 84 µg·L⁻¹ for the glochidia (larvae) of the mollusc *Villosa iris* (Goudreau et al. 1993), as well as 96h LC₅₀s of 89 and 80 µg·L⁻¹ for the copepods *Cyclops thomasi* and *C. bicuspedatus*, respectively (Beeton et al. 1976). Also, 24h LC₅₀s of 12 and 16 µg·L⁻¹ (in static and continuous flow-through tests, respectively) for *Ceriodaphnia dubia* neonates (Taylor 1993), as well as 24h LC₅₀s of 11 and 60 µg·L⁻¹ (Kaniewska-Prus 1982) and a 1h LC₅₀ of 63 µg·L⁻¹ for the water flea *Daphnia magna* (Mattice et al. 1981) have been observed. More recently, Fisher et al. (1999) reported a 48h LC₅₀ of <18 µg·L⁻¹ (i.e., lowest concentration tested) for *D. magna*. As part of a comprehensive study on the fate and

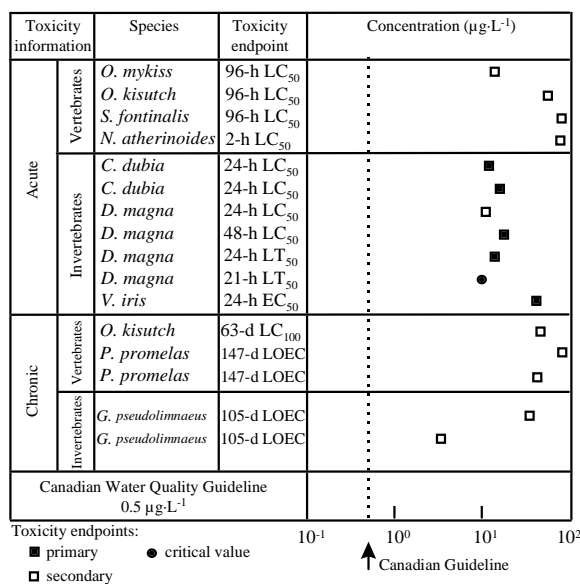


Figure 2. Select freshwater toxicity data for monochloramine.

behaviour, chemistry, analytical detection, and toxicity of monochloramine, the Pacific Environmental Science Centre (Environment Canada 1998) determined LT_{50} s and LT_{100} s for *D. magna* neonates. The LT_{50} s were reported as 8–24 h and 21–24 h when exposed to nominal concentrations of $20 \mu\text{g}\cdot\text{L}^{-1}$ (the measured concentrations were $14 \mu\text{g}\cdot\text{L}^{-1}$ at 24 h and $<10 \mu\text{g}\cdot\text{L}^{-1}$ at 21 h, respectively (detection limit $10 \mu\text{g}\cdot\text{L}^{-1}$). The LT_{100} s were 72 and 48 h, respectively.

Grossnickle (1974) reported a 1h LC_{50} of $32 \mu\text{g}\cdot\text{L}^{-1}$, a 4h LC_{50} of $27 \mu\text{g}\cdot\text{L}^{-1}$, and a 24h LC_{50} of $13.5 \mu\text{g}\cdot\text{L}^{-1}$ for the rotifer *Keratella cochlearis*. However, these LC_{50} s are questionable since control mortality was not accounted for, and reservations exist regarding the analytical methodology and detection limit.

Chronic toxicity studies for fish are a 63-d LC_{100} of $47 \mu\text{g}\cdot\text{L}^{-1}$ for coho salmon alevins (*O. kisutch*) (Larson et al. 1977a) and 147-d LOECs of $85 \mu\text{g}\cdot\text{L}^{-1}$ (survival and growth) and $43 \mu\text{g}\cdot\text{L}^{-1}$ (reduced spawning success) for fathead minnows (*Pimephales promelas*) (Arthur and Eaton 1971). Arthur and Eaton (1971) also reported 105-d LOECs of $35 \mu\text{g}\cdot\text{L}^{-1}$ (survival) and $3.4 \mu\text{g}\cdot\text{L}^{-1}$ (approximately 90% reduction in fecundity) for the invertebrate amphipod *Gammarus pseudolimnaeus*. Carlson (1976) reported reduced growth in a mixed periphyton community exposed to $1 \mu\text{g}\cdot\text{L}^{-1}$ monochloramine. However, the latter two studies were not used for the guideline derivation due to reservations with respect to the analytical methodologies and testing protocols.

A recent review, combined with new toxicity studies (Environment Canada 1998) for the Canadian Environmental Protection Act Priority Substances List chloramine assessment with meta analysis of published and unpublished toxicity data for chinook salmon, coho salmon, brook trout, cutthroat trout, and rainbow trout, showed that coho salmon and early life stages of rainbow trout seem to be the most sensitive. Acute and chronic LC_{50} s were in the 40 to $50 \mu\text{g}\cdot\text{L}^{-1}$ range. Similar data analysis for invertebrates showed that, in general, invertebrates seem to be more sensitive than fish and that the most sensitive invertebrates seem to be water fleas. *C. dubia* showed a 24h LC_{50} of approximately 15 – $20 \mu\text{g}\cdot\text{L}^{-1}$. However, the quality of this data was ranked as low. The water flea *D. magna* appears to be more sensitive than *C. dubia*, as exposure to $<10 \mu\text{g}\cdot\text{L}^{-1}$ measured concentration of chloramine (detection limit $10 \mu\text{g}\cdot\text{L}^{-1}$) for 21 h caused 100% mortality after 28 h (unpublished data). Toxicity testing below $10 \mu\text{g}\cdot\text{L}^{-1}$ would be required to determine the toxic effect threshold

for *D. magna*. However, this is hampered by the difficulty in analyzing reliably for monochloramine at these levels. Additional toxicity testing and analytical methodology developments are currently under way (Environment Canada 1998).

Guideline Derivation for Reactive Chlorine

Even though reactive chlorine species have been studied extensively, considerable uncertainty in the toxicological and analytical data, especially at low exposure concentrations, still exists because of the reactive nature and the complex chemistry of these compounds. For example, some studies reported toxicity values based on nominal concentrations, others were based on measured concentrations. However, most reported concentrations, especially in older studies, were derived from analytical methods having inadequate detection limits (i.e., 100 – $500 \mu\text{g}\cdot\text{L}^{-1}$). The lowest reliable detection limit currently is approximately $10 \mu\text{g}\cdot\text{L}^{-1}$ (Taylor 1993; Environment Canada 1998; Fisher et al. 1999). Also, there is no standardized reporting protocol. Toxicity values were reported as CAC, CRC, FAC, FRC, monochloramine, etc., while the actual detection limits, reactivity, interconvertibility, and co-existence of the different species were often not adequately considered.

Nevertheless, the collective evidence points to a toxic effects threshold range for both hypochlorous acid (FAC) and monochloramine (CAC) in the 1 to $10 \mu\text{g}\cdot\text{L}^{-1}$ range, i.e., below the currently achievable detection limit. This is also supported by the recent Canadian Environmental Protection Act Priority Substances List chloramine assessment (Environment Canada 1998) in its literature review and accompanying toxicity testing, and by several recently published studies (e.g., Fisher et al. 1999; Taylor 1993).

The Canadian water quality guideline for reactive chlorine species for the protection of freshwater life was derived based on the LT_{50} for *D. magna* after exposure to monochloramine for 21–24 h at $<10 \mu\text{g}\cdot\text{L}^{-1}$ (with concentration measured at 21 h) (Environment Canada 1998). This endpoint was chosen as it is the lowest measured endpoint recently reported and was, therefore, deemed most reliable considering the challenges posed by reactive chlorine species at these low concentrations. The LT_{50} of $<10 \mu\text{g}\cdot\text{L}^{-1}$ was multiplied by the acute application factor for nonpersistent substances (0.05), yielding a guideline value of $0.5 \mu\text{g}\cdot\text{L}^{-1}$ for the protection of freshwater life. It should be noted that the lowest reliable limit of detection reported is $10 \mu\text{g}\cdot\text{L}^{-1}$, a value that is

higher than the Canadian water quality guideline for the protection of aquatic life. Therefore, any detection of reactive chlorine species in aquatic systems is an indication that aquatic life is potentially being negatively affected. According to the formal protocol (CCME 1991), a water quality guideline is preferentially derived from an acceptable chronic exposure study. This is based on the understanding that such a study also provides adequate protection during short-term exposure scenarios. However, it was deemed that this was not the case with reactive chlorine species; therefore, the guideline derivation is based on an acute exposure study.

Marine Life

A guideline of 0.5 µg·L⁻¹ chlorine-produced oxidants is recommended for the protection of marine life.

The already complex freshwater chlorine chemistry is further complicated in marine water due to the presence of bromide ions (Br⁻). Therefore, the marine water quality guideline is derived for the group of chlorine-produced oxidants.

The lowest reported acute toxicity studies for fish are two 96h LC₅₀s for plaice (*Pleuronectes platessa*) of 24 µg·L⁻¹ (larvae) and 28 µg·L⁻¹ (adult) (Alderson 1970), a 48h and 96h LC₅₀ for inland silversides (*Menidia beryllina*) of 37 µg·L⁻¹ (Roberts et al. 1975) and 128 µg·L⁻¹ (Fisher et al. 1994), respectively, and a 48h LC₅₀ for striped bass (*Morone saxatilis*) of 40 µg·L⁻¹ (Middaugh 1977). Holland et al. (1960) also reported a 96h LC₅₀ for juvenile pink salmon (*Oncorhynchus gorbusha*) of 50 µg·L⁻¹ and a 24h LC₄₀ of 50 µg·L⁻¹ and a 72h LC₁₀₀ of 100 µg·L⁻¹ for juvenile chinook salmon (*O. tshawytscha*). Fisher et al. (1999) reported a 96h LC₅₀ of 65 µg·L⁻¹ for inland silversides (*M. beryllina*).

The lowest reported acute toxicity studies for invertebrates are 5-min EC₅₀s (reduced egg fertility success) for sand dollar sperm (*Dendraster excentricus*) of 2 µg·L⁻¹ and green sea urchin sperm (*Strongylocentrotus droebachiensis*) of 5 µg·L⁻¹ (Dinnel et al. 1981), a 48-h LC₅₀ for the eastern oyster larvae (*Crassostrea virginica*) of 5 µg·L⁻¹, and a 48-h EC₅₀ (percent straight hinge larvae) for hard clam larvae (*Mercenaria mercenaria*) of 6 µg·L⁻¹ (Roberts et al. 1975), an 8-min LC₂₀ and a 96h LC₅₀ for blue crab juveniles (*Callinectes sapidus*) of 10 µg·L⁻¹ (Patrick and McLean 1971), and a 96h LC₅₀ for crab larvae (*Panopeus herbstii*) of 24 µg·L⁻¹ (Roberts 1977). Also, 48h LC₅₀s for the copepod *Acartia tonsa* of 29 µg·L⁻¹ and for eastern oyster larvae (*Crassostrea*

virginica) of 26 µg·L⁻¹ (Roberts and Gleeson 1978) and a 96h LC₅₀ for mysid shrimp (*Mysidopsis bahia*) of 73 µg·L⁻¹ (Fisher et al. 1994) have been reported. More recently, Fisher et al. (1999) reported a 96h LC₅₀ of 92 µg·L⁻¹ for mysid shrimp (*M. bahia*). Similarly, Capuzzo (1979) reported 30-min LC₅₀s and LC₁₀₀s for American oyster larvae (*C. virginica*) of 10 µg·L⁻¹ and 160 µg·L⁻¹, respectively, and for the rotifer *Brachionus plicatilis* of 10 µg·L⁻¹ and 190 µg·L⁻¹, respectively.

The lowest reported chronic toxicity studies for fish are a 9-d LC₅₀ of 80 µg·L⁻¹ for coho salmon (*O. kisutch*) (Holland et al. 1960) and an 8-d LC₅₀ of 120 µg·L⁻¹ for plaice eggs (*P. platessa*) (Alderson 1970). Chronic toxicity studies for invertebrates include a 239-d EC₅₀ (inhibition of shell growth) of 25 µg·L⁻¹ for littleneck clams (*Protothaca staminea*) (Hillman et al. 1980) and a 25-d LC₁₀ of 140 µg·L⁻¹ for eastern oysters (*C. virginica*) (Scott and Middaugh 1978).

The lowest reported toxicity studies for phytoplankton communities include a 24h EC₅₀ and a 24h EC₂₀ (inhibition of photosynthesis) of 10 µg·L⁻¹ (Eppley et al. 1976) and 30 µg·L⁻¹ (Krock and Mason 1971), respectively. Maruyama et al. (1988) reported a 10-d EC₅₀ (growth) of 16 µg·L⁻¹ and a 10-d LC₅₀ of 30 µg·L⁻¹ for a marine red algae (*Porphyra yezoensis*). Furthermore, Sanders and Ryther (1980) reported a shift in the community composition for phytoplankton after exposure for 30 d to 50 µg·L⁻¹, and Erickson and Freeman (1978)

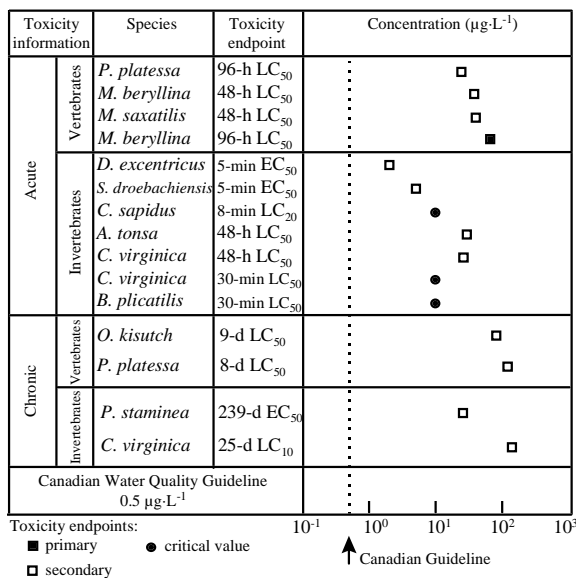


Figure 3. Select marine toxicity data for chlorine-produced oxidants.

reported EC₅₀s (cell division) of 125 µg·L⁻¹ for the algae *Skeletonema costatum*, *Thalassiosira pseudonana*, *Isochrysis galbana*, and *Glenodinium halli*.

A water quality guideline is preferentially derived from a chronic toxicity study, however, a survey of the available toxicity data base indicates that information on chronic effects is limited and that the available acute effects thresholds are generally below reported chronic effects thresholds. Therefore, the guideline for chlorine-produced oxidants in marine waters is based on acute effects thresholds. However, the four lowest reported acute effects thresholds, reduced egg fertilization successes for sand dollar and green sea urchin sperm at 2 and 5 µg·L⁻¹, respectively (Dinnel et al. 1981), the 48-h LC₅₀ for the eastern oyster larvae of 5 µg·L⁻¹, and the 48-h EC₅₀ for hard clam larvae of 6 µg·L⁻¹ (Roberts et al. 1975), were not considered acceptable due to reservations with respect to the analytical methodologies and testing protocols. Several studies covering invertebrates and plants are reporting toxic effects at the next higher level, 10 µg·L⁻¹, for example, blue crabs (Patrick and McLean 1971), American oysters (Capuzzo 1979), the rotifer *B. plicatilis* (Capuzzo 1979), and phytoplankton (Eppley et al. 1976). The guideline is derived by multiplying this effects threshold by the acute application factor for nonpersistent substances (0.05), yielding a guideline value of 0.5 µg·L⁻¹ for the protection of marine life.

It should be noted that the lowest reliable limit of detection reported is 10 µg·L⁻¹, a value that is higher than the Canadian water quality guideline for the protection of aquatic life. Therefore, any detection of reactive chlorine species in aquatic systems is an indication that aquatic life is potentially being negatively affected.

References

- Abarnou, A., and L. Miossec. 1992. Chlorinated waters discharged to the marine environment: Chemistry and environmental impact. An overview. *Sci. Total Environ.* 126:173–197.
- Alderson, R. 1970. Effects of low concentrations of free chlorine on eggs and larvae of plaice (*Pleuronectes platessa* L.). In: *Marine pollution and sea life*. M. Riuvo, ed. FAO, Fishing News (Books) Ltd., Surrey, England.
- Arthur, J.W., and J.G. Eaton. 1971. Chloramine toxicity to the amphipod (*Gammarus pseudolimnaeus*) and the fathead minnow (*Pimephales promelas*). *J. Fish. Res. Board Can.* 28:1841–1845.
- Basch, R.E., and J.G. Truchan. 1974. Calculated residual chlorine concentrations safe for fish. Michigan Water Resources Commission, Bureau of Water Management, Water Quality Appraisal Section, Lansing, MI.
- Beeton, A.M., P.K. Kovacic, and A.S. Brooks. 1976. Effects of chlorine and sulphite reduction on Lake Michigan invertebrates. EPA-600/3-76-036. U.S. Environmental Protection Agency, Washington, DC.
- Belanger, S.E., D.S. Cherry, J.L. Farris, K.G. Sappington, and J. Cairns Jr. 1991. Sensitivity of the Asiatic clam to various biocidal control agents. *Am. Water Works Assoc. J.* October:79–87.
- Bogardus, R., D. Boies, T. Teppen, and F. Horvath. 1978. Avoidance of monochloramine: Test tank results for rainbow trout, coho salmon, alewife, yellow perch, and spottail shiner. In: *Water chlorination: Environmental impact and health effects*, Vol. 2, R.L. Jolley, H. Gorchev, and D.H. Hamilton Jr., eds. Ann Arbor Science Publishers Inc., Ann Arbor, MI.
- Bousher, A., P. Brimblecombe, and D. Midgley. 1989. Kinetics of reactions in solutions containing monochloramine and bromide. *Water Res.* 23:1049–1058.
- . 1990. Bromate production in chlorinated waters: Reaction of monochloramine and hypobromite. *Water Res.* 24:1285–1294.
- Brooks, A., and J. Bartos. 1984. Effects of free and combined chlorine and exposure duration on rainbow trout, channel catfish, and emerald shiners. *Trans. Am. Fish. Soc.* 113:786–793.
- Brooks, A.S., J.M. Bartos, and P.T. Danos. 1982. Effects of chlorine on freshwater fish under various time and chemical conditions: Toxicity of chlorine to freshwater fish. DE82-905841. Nat. Tech. Info. Serv., Springfield, VA.
- Cairns, J., B. Niederlehner, and J. Pratt. 1990. Evaluation of joint toxicity of chlorine and ammonia to aquatic communities. *Aquat. Toxicol.* 16:87–100.
- Capuzzo, J.M. 1979. The effect of temperature on the toxicity of chlorinated cooling waters to marine animals—A preliminary review. *Mar. Pollut. Bull.* 10:45–47.
- Carlson, A.R. 1976. Reproductive behaviour of the threespine stickleback exposed to chloramines. M.Sc. thesis. Oregon State University, Corvallis, OR.
- CCME (Canadian Council of Ministers of the Environment). 1991. Appendix IX—A protocol for the derivation of water quality guidelines for the protection of aquatic life. (April 1991). In: *Canadian water quality guidelines*, Canadian Council of Resource and Environment Ministers. 1987. Prepared by the Task Force on Water Quality Guidelines. [Updated and reprinted with minor revisions and editorial changes in *Canadian environmental quality guidelines*, Chapter 4, Canadian Council of Ministers of the Environment, 1999, Winnipeg.]
- Cooper, W.J., R.G. Zitka, and M.S. Steinhauer. 1985. Bromide-oxidant interactions and THM formation: A literature review. *Am. Water Works Assoc. J.* April:116–121.
- Dandy, J.W.T. 1972. Activity response to chlorine in the brook trout (*Salvelinus fontinalis*) (Mitchill). *Can. J. Zool.* 50:405–410.
- Dinnel, P.A., Q.J. Stober, and D.H. DiJulio. 1981. Sea urchin sperm bioassay for sewage and chlorinated seawater and its relation to fish bioassays. *Mar. Environ. Res.* 5:29–39.
- Environment Canada. 1998. Development of a workplan for the Tier II and III risk assessment for the ecological assessment of inorganic chloramine. Proceedings of the workshop, May 28–29, 1998, Vancouver, BC. Prepared by Environment Canada, Pacific and Yukon Region, Vancouver.
- . 1999. Canadian water quality guidelines for reactive chlorine species: Hypochlorous acid and monochloramine. Supporting document. Environment Canada, Environmental Quality Branch, Guidelines and Standards Division, Ottawa. Unpub. draft doc.
- Eppley, R.W., E.H. Renger, and P.M. Williams. 1976. Chlorine reactions with sea-water constituents and the inhibition of photosynthesis of natural marine phytoplankton. *Estuarine Coastal Mar. Sci.* 4:147–161.
- Erickson, S.J., and A.E. Freeman. 1978. Toxicity screening of fifteen chlorinated and brominated compounds using four species of marine phytoplankton. In: *Water chlorination: Environmental impact and health effects*, Vol. 2, R.L. Jolley, H. Gorchev, and D.H. Hamilton Jr., eds. Ann Arbor Science Publishers Inc., Ann Arbor, MI.

- Fisher, D.J., D.T. Burton, L.T. Yonkos, S.D. Turley, B.S. Turley, G.P. Ziegler, and E. J. Zillioux. 1994. Acute and short-term chronic effects of continuous and intermittent chlorination on *Mysidopsis bahia* and *Menidia beryllina*. *Environ. Toxicol. Chem.* 13:1525–1534.
- Fisher, D.J., D.T. Burton, L.T. Yonkos, S.D. Turley, and G.P. Ziegler. 1999. The relative acute toxicity of continuous and intermittent exposures of chlorine and bromine to aquatic organisms in the presence and absence of ammonia. *Water Res.* 33:760–768.
- Goudreau, S.E., R.J. Neves, and R. J. Sheehan. 1993. Effects of wastewater treatment plant effluents on freshwater mollusks in the upper Clinch River, Virginia, USA. *Hydrobiol.* 252:211–230.
- Government of Canada. 1993. Chlorinated wastewater effluents. Canadian Environmental Protection Act Priority Substances List Assessment Report. Environment Canada and Health and Welfare Canada, Ottawa.
- Gregg, B.C. 1974. The effects of chlorine and heat on selected stream invertebrates. Ph.D. thesis. Virginia Polytechnic Institute and State University, Blacksburg, VA.
- Grossnickle, N.E. 1974. The acute toxicity of residual chloramine to the rotifer *Keratalla cochlearis* (Gosse) and the effect of dechlorination with sodium sulfite. M.Sc. thesis. University of Wisconsin, Milwaukee, WI.
- Hillman, R.E., C.I. Gibson, P. Wilkinson, and D.L. Woodruff. 1980. Growth and histological effects on *Protothaca staminea* (Littleneck clam) of long-term exposure to chlorinated seawater. In: *Water chlorination: Environmental impact and health effects*, Vol. 3, R.L. Jolley, W.A. Brungs, and R.B. Cumming, eds. Ann Arbor Science Publishers Inc., Ann Arbor, MI.
- Holland, G.A., J.E. Lasater, E.D. Newman, and W.E. Eldridge. 1960. Chlorine and chloramine experiments. Part of toxic effects of organic and inorganic pollutants on young salmon and trout. *Res. Bull. Wash. St. Dept. Fish.* 5:198–216.
- Kaniewska-Prus, M. 1982. The effect of ammonia, chlorine, and chloramine toxicity on the mortality of *Daphnia magna* Straus. *Pol. Arch. Hydrobiol.* 29(3–4):607–624.
- Kott, Y., G. Hershkovitz, A. Shemtob, and J.B. Sless. 1966. Algicidal effect of bromine and chlorine on *Chlorella pyrenoidosa*. *Appl. Microbiol.* 14(1):8–11.
- Krock, H.J., and D.T. Mason 1971. Bioassays of lower trophic levels. In: *A study of toxicity and biostimulation in San Francisco Bay–Delta waters*, Vol. 6, State Water Rescues Control Board, ed. California Department of Water Resources, San Francisco.
- Larson, G.L. F.E. Hutchins, and L.P. Lamperti. 1977a. Laboratory determination of acute and sublethal toxicities of inorganic chloramines to early life stages of coho salmon (*Oncorhynchus kisutch*). *Trans. Am. Fish. Soc.* 106:268–277.
- Larson, G.L., F.E. Hutchins, and D.A. Schlesinger. 1977b. Acute toxicity of inorganic chloramines to early life-stages of brook trout (*Salvelinus fontinalis*). *J. Fish Biol.* 11:595–598.
- Larson, G.L., C.W. Warren, F.E. Hutchins, L.P. Lamperti, D.A. Schlesinger, and W.K. Siem. 1978. Toxicity of residual chlorine compounds to aquatic organisms. EPA-600/3-78-023. U.S. Environmental Protection Agency, Corvallis, OR.
- Maruyama, T., K. Ochiai, A. Miura, and T. Yoshida. 1988. Effects of chloramine on the growth of *Porphyra yezoensis* (Rhodophyta). *Nippon Suisan Gakkaishi* 54(10):1829–1834.
- Mattice, J.S., and H.E. Zittel. 1976. Site-specific evaluation of power plant chlorination. *J. Water. Pollut. Control Fed.* 48:2284–2308.
- Mattice, J.S., S.C. Tsai, and M.B. Burch. 1981. Comparative toxicity of hypochlorous acid and hypochlorite ions to mosquitofish. *Trans. Am. Fish. Soc.* 110:519–525.
- MDNR (Michigan Department of Natural Resources). 1971. Chlorinated municipal waste toxicities to rainbow trout and fathead minnows. *Water Pollut. Control Res. Serv.*, 18050 GZZ. U.S. Environmental Protection Agency, Washington, DC.
- Meyer, J.S., D. D. Gulley, M.S. Goodrich, D.C. Szmania, and A. S. Brooks. 1995. Modeling toxicity due to intermittent exposure of rainbow trout and common shiners to monochloramine. *Environ. Toxicol. Chem.* 14:165–175.
- Middaugh, D.P. 1977. Responses of early life history stages of the striped bass, *Morone saxatilis*, to chlorination. *Chesapeake Sci.* 18:141.
- Morris, J.C. 1978. The chemistry of aqueous chlorine in relation to water chlorination. In: *Water chlorination: Environmental impact and health effects*, Vol. 2, R.L. Jolley, H. Gorchev, and D.H. Hamilton Jr., eds. Ann Arbor Science Publishers Inc., Ann Arbor, MI.
- Nikl, L., and D. Nikl. 1992. Environmental impacts associated with monochloramine-disinfected municipal potable water. National Conference on Drinking Water, Winnipeg.
- Norecol Environmental Consultants Ltd. and Dayton and Knight Ltd. 1992. Greater Vancouver water district environmental impact assessment of proposed secondary disinfection of drinking water. Prepared for the Greater Vancouver Regional District. Baseline (Stage I) Report. Vancouver.
- Orvos, D.R., C.M. Sellers, and N.E. Garrison. 1986. Effect of a single residual chlorine pulse upon subsequent in vitro protein synthesis by rainbow trout (*Salmo gairdneri*) liver. *Hydrobiol.* 131:249–255.
- Patrick, R., and R. McLean. 1971. Entrainment simulation studies on some estuarine organisms for the Potomac Electric Power Company. Academy of Natural Sciences, Philadelphia, Department of Limnology, Philadelphia.
- Pike, D.J. 1971. Toxicity of chlorine to brown trout. *N. Z. Wildl.* 33: 43.
- Pratt, J.R., N.J. Bowers, B.R. Niederlehner, and J. Cairns Jr. 1988. Effects of chlorine on microbial communities in naturally derived microcosms. *Environ. Toxicol. Chem.* 7:679–687.
- Roberts, M.H., Jr. 1977. Effects of chlorinated seawater on decapod crustaceans. In: *Water chlorination: Environmental impact and health effects*, Vol. 1, R.L. Jolley, ed. Ann Arbor. Science Publishers Inc., Ann Arbor, MI.
- Roberts, M.H., Jr., and R.A. Gleeson. 1978. Acute toxicity of bromochlorinated seawater to selected estuarine species with a comparison to chlorinated seawater toxicity. *Mar. Environ. Res.* 1:19–30.
- Roberts, M.H., Jr., R.J. Diaz, M.E. Bender, and R.J. Huggett. 1975. Acute toxicity of chlorine to selected estuarine species. *J. Fish. Res. Board Can.* 32:2525–2528.
- Roseboom, D.P., and D.L. Richey. 1977. Acute toxicity of residual chlorine on bluegill and channel catfish in Illinois. *Trans. Ill. State Acad. Sci.* 69:385.
- Rosenberger, D.R. 1971. The calculation of acute toxicity of free chlorine and chloramines to coho salmon by multiple regression analysis. M.Sc. thesis. Michigan State University, East Lansing, MI.
- Sanders, J.G., and J.H. Ryther. 1980. Impact of chlorine on the species composition of marine phytoplankton. In: *Water chlorination; environmental impact and health effects*, Vol. 3, R.L. Jolley, W. Brungs, and B. Cumming, eds. Ann Arbor. Science. Publishers Inc., Ann Arbor, MI.
- Scott, G.I., and D.P. Middaugh. 1978. Seasonal chronic toxicity of chlorination to the american oyster, *Crassostrea virginica* (G). In: *Water chlorination: Environmental impact and health effects*, Vol. 2, R.L. Jolley, H. Gorchev, and D.H. Hamilton Jr., eds. Ann Arbor Science Publishers Inc., Ann Arbor, MI.

Sprague, J.B., and D.E. Drury. 1969. Avoidance reactions of salmonid fish to representative pollutants. In: Advances in water pollution research. Proc. 4th International Conference, Water Pollut. Res., Pergamon Press, London.

Taylor, P. 1993. An evaluation of the toxicity of various forms of chlorine to *Ceriodaphnia dubia*. Environ. Toxicol. Chem. 12:925-930.
Watkins, C.H., and R.S. Hammerschlag. 1984. The toxicity of chlorine to a common vascular aquatic plant. Water Res. 18:1037-1043.

Reference listing:

Canadian Council of Ministers of the Environment. 1999. Canadian water quality guidelines for the protection of aquatic life: Reactive chlorine. In: Canadian environmental quality guidelines, 1999, Canadian Council of Ministers of the Environment, Winnipeg.

For further scientific information, contact:

Environment Canada
Guidelines and Standards Division
351 St. Joseph Blvd.
Hull, QC K1A 0H3
Phone: (819) 953-1550
Facsimile: (819) 953-0461
E-mail: ceqg-rcqe@ec.gc.ca
Internet: <http://www.ec.gc.ca>

For additional copies, contact:

CCME Documents
c/o Manitoba Statutory Publications
200 Vaughan St.
Winnipeg, MB R3C 1T5
Phone: (204) 945-4664
Facsimile: (204) 945-7172
E-mail: spccme@chc.gov.mb.ca