Ambient Water Quality Guidelines for Chlorate

Technical Background Report

Prepared pursuant to Section 2(e) of the *Environment Management Act, 19981*

Original signed by Assistant Deputy Minister, February 2002

CANADIAN CATALOGUING IN PUBLICATION

National Library of Canada Cataloguing in Publication Data
Warrington, P. D. (Patrick Douglas), 1942-
Ambient water quality guidelines for chlorate

Author: Pat Warrington. Cf.
Available on the Internet.
Includes bibliographical references.
ISBN 0-7726-4734-8

1. Water quality - Standards - British Columbia.
2. Chlorates - Environmental aspects - British Columbia.
I. British Columbia. Environmental Quality Branch. II. Title.

TD227.B7W37 2002 363.739'462'09711 C2002-960058-8

Updated: February 25, 2002

Summary

This document is one of a series which establishes ambient water quality guidelines for British Columbia. The guidelines represent safe conditions or levels of a variable which have province-wide application and are set to protect various water uses. This report sets guidelines for chlorates to protect marine and freshwater algae and drinking water for humans, wildlife and livestock. Guidelines were not set for other uses due to the low sensitivity of other organisms to chlorate, presently known ambient and effluent levels and lack of good data. CCREM (CCME) has not set a guideline for this water use. No data were available on the effects of chlorates on marine fish or invertebrates. Toxicity to marine brown algae is the effect which is manifested at the lowest chlorate concentration; this effects occur in the microgram/L concentration range; for fresh water green algae measured effects occur only at the 100's of mg/L level. There are two main economic uses of marine brown algae. Kelp is a raw material source for the production of agar, the laboratory growth medium, and is the preferred spawning site for herring which supports a roe fishery for the export market.

A major use of the guidelines is to set site-specific ambient water quality objectives. These objectives are the guidelines, adopted or modified to meet specific local conditions, applied to a particular body of water

Ministry of Environment | Water Protection and Sustainability Branch, Environmental Sustainability and Strategic Policy Division | Mailing Address: PO Box 9362 Stn Prov Govt Victoria BC V8W 9M2 | Telephone: 250 387-9481 Facsimile: 250 356-1202 Website: www.gov.bc.ca/water

to protect the most sensitive designated water use. The guidelines and objectives do not have legal standing, but are used in the preparation of Waste Management Permits, Orders or Approvals, which do have legal standing.

Microorganisms can adapt their metabolic processes to use virtually any source of carbon, including chlorates, for growth. There is evidence for anaerobic bacterial degradation of chlorates in nature. If the organisms have never been exposed to chlorates, there will be an initial adaptation period but once the adaptive phase is over and a large microbial population has been established, breakdown of chlorates is rapid. Subsequent additions of chlorates to the environment would be quickly degraded, if the concentrations were not excessive.

The guideline proposed for freshwater life, to protect the most sensitive species, which appear to be green algae, is 30 mg/L. For marine life, the most sensitive species are brown algae which includes the kelps (i.e. Macrocystus) and Fucus (bladder wrack). The recommended marine guideline is 5 µg/L. The raw human drinking water guideline is 2.4 mg/L; for wildlife and livestock drinking water it is 3 mg/L.

Table 9. Summary of Chlorate Guidelines

Water Use	Guideline
Raw Drinking Water	2.4 milligrams/L
Wildlife and Livestock	3 milligrams/L
Freshwater Aquatic Life	30 milligrams/L
Marine Aquatic Life	5 micrograms/L

Preface

THE MINISTRY OF ENVIRONMENT, LANDS AND PARKS (now called Ministry of Water, Land and Air Protection) develops province-wide ambient water quality guidelines for variables that are important in the surface waters of British Columbia. This work has the following goals:

1. to provide guidelines for the evaluation of data on water, sediment, and biota
2. to provide guidelines for the establishment of site-specific ambient water quality objectives

Ambient water quality objectives for specific waterbodies will be based on the guidelines and also consider present and future uses, waste discharges, hydrology/limnology/oceanography and existing background water quality. The process for establishing water quality objectives is more fully outlined in

Principles for Preparing Water Quality Objectives in British Columbia, copies of which are available from Water Quality Section of the Environmental Quality Branch.

Neither guidelines nor objectives which are derived from them, have any legal standing. The objectives, however, can be used to calculate allowable limits or levels for contaminants in waste discharges. These limits are set out in waste management permits and thus have legal standing. The objectives are not usually incorporated as conditions of the permit.

The definition adopted for a guideline is:

> ***A maximum and/or a minimum value for a physical, chemical or biological characteristic of water, sediment or biota, which should not be exceeded to prevent specified detrimental effects from occurring to a water use, including aquatic life, under specified environmental conditions.***

The guidelines are province-wide in application, are use-specific, and are developed for some or all of the following specific water uses:

- Raw drinking, public water supply and food processing
- Aquatic life and wildlife
- Agriculture (livestock watering and irrigation)
- Recreation and aesthetics
- Industrial (water supplies)

The guidelines are set after considering the scientific literature, guidelines from other jurisdictions, and general conditions in British Columbia. The scientific literature gives information on the effects of toxicants on various life forms. This information is not always conclusive because it is usually based on laboratory work which, at best, only approximates actual field conditions. To compensate for this uncertainty, guidelines have built-in safety factors which are conservative but reflect natural background conditions in the province.

The site-specific water quality objectives are, in most cases, the same as guidelines. However, in some cases, such as when natural background levels exceed the guidelines, the objectives could be less stringent than the guidelines. In relatively rare instances, for example if the resource is unusually valuable or of special provincial significance, the safety factor could be increased by using objectives which are more stringent than the guidelines. Another approach in such special cases is to develop site-specific guidelines by carrying out toxicity experiments in the field. This approach is costly and time-consuming and therefore seldom used.

Guidelines are subject to review and revision as new information becomes available, or as other circumstances dictate.

> **The guidelines apply to the ambient raw water source before it**

is diverted or treated for domestic use.
The Ministry of Health regulates the quality of water for domestic use after it is treated and delivered by a water purveyor.

Guidelines relating to public health at bathing beaches are the same as those used by the Ministry of Health which regulates the recreation and aesthetic use.

Introduction

Chlorates have been used historically as herbicides to kill all terrestrial plants except mosses; this literature has been reviewed by McIntyre and Noton (1990). The only other major source of chlorate input to the environment is effluent from pulp mills using chlorine dioxide bleaching. Minor amounts of chlorate are also produced when chlorine dioxide is used to disinfect drinking water. Chlorate toxicity is linked to nitrate concentrations and aerobic conditions. The most sensitive species are marine brown algae, the kelps (i.e. Macrocystis) and Fucus, which are vital components of the coastal ecosystem.

It appears that the only aquatic organisms at risk from chlorate toxicity are the brown marine algae of coastal habitats. Under anaerobic conditions, or with ammonia nitrogen (as opposed to nitrate nitrogen) as the available nitrogen source, when the nitrate reductase system is not active, there should be little risk of chlorate toxicity. There is a good literature review by Wijk and Hutchinson (1995) on the toxicity of chlorate to aquatic organisms. If pulp mills remove un-reacted chlorate from their effluent in coastal, marine habitats, there should be minimal environmental concern. Discharges to large rivers with long residence times prior to entering the ocean should not cause a problem.

Chemistry and Effects

Properties of Chlorates

Sodium chlorate is a colourless, cubic or trigonal crystal or a white powder. The crystals are also reported to be pale yellow or white which may be a function of purity. The compound is odourless and tastes salty. The boiling point is 122 degrees Celsius and the melting point is 248 degrees Celsius. The molecular weight of chlorate is 106.44 and the density is 2.490 g/mL at 15 degrees Celsius. Sodium chlorate is corrosive to zinc and mild steel. An aqueous solution has a neutral pH.

Chlorate is very soluble in fresh water, 80 g/100 mL at 0 degrees Celsius and 96 g/100 mL at 20 degrees Celsius. It is unlikely to adsorb to sediments, organics or food particles or to bio-accumulate. It is a

strong oxidant and should be non-persistent in water with organic material present. The chlorate anion is unstable in water and decomposes to form OCl- and oxygen (Environment Canada, 1985; CRC Handbook, 1993; Hayes, 1982; Herbicide Handbook, 1983; Worthing and Walker, 1987; Armour, 1991; Merck Index, 1989; NRC, 1987; Spencer, 1982).

Mechanism of Action of Chlorates

The chlorate anion is not directly toxic; the mechanism of chlorate toxicity in plants is indirect. In simple terms an enzyme system in plants, evolved to reduce nitrate, also reduces chlorate to a toxic intermediate product, apparently chlorite (ClO_2-) or possibly hypochlorite (OCl). These are reduction products of chlorate produced by the nitrate reductase enzyme system in plants and the chlorite inactivates the nitrate reductase system. This was demonstrated in wheat seedlings, *Triticum vulgare*, by Aberg (1947). The formation of chlorite was confirmed by Goksoyr (1952) in *Escherichia coli.*

Liljeström and Aberg (1966) concluded that the toxicity of chlorate is coupled to its reduction to chlorite and that this reduction is linked to an active, functioning nitrate reductase system. Solomonsson and Vennesland (1972) also suggested that chlorite is the toxic agent based on observations that the addition of chlorite inactivated some enzymes to the same extent as chlorate.

The nitrate reductase enzyme system in plants is an inducible enzyme system which requires a certain minimum threshold level of nitrate in order to become activated. It then reduces nitrate to nitrite and ultimately to ammonium, the preferred form of nitrogen for use by the plant. If there is sufficient nitrate in the growth medium, the nitrate reductase system is activated and takes up chlorate, reducing it to the toxic chlorite. There is competition for the active sites on the enzyme system and if nitrate is abundant, it prevents too much chlorate from being reduced and toxicities remain low. If nitrate concentrations are just high enough to induce the nitrate reductase system, but not high enough to out-compete chlorate for all the active sites, then chlorate may be reduced to chlorite at a maximal rate and toxicity is observed. This was demonstrated in work by Goksoyr (1951), Fahraeus (1952) and Liljestrom and Aberg (1966).

Generally, in BC, nitrate levels are low in large rivers and coastal marine waters which tend to be nitrogen limited systems; rarely would there be enough nitrate available to tie up all the active sites in the nitrate reductase enzyme system. Often there will not even be enough nitrate to induce the enzyme system to activity.

It is important therefore to know the level of nitrate in any experimental or natural system in which chlorate toxicity is being measured. Nitrate concentrations determine the activity of the nitrate reductase system and in turn the level of conversion of chlorate to chlorite. Chlorate is not very toxic in ammonium-based systems. Strains of Cytophaga did not show any depression in microbial activity when grown with ammonium phosphate as the nitrogen source, even when the chlorate concentration was up to 8350 mg/L. If a nitrate level of 70 mg/L was introduced, then microbial activity was depressed at 2100 mg/L chlorate.

Increasing the nitrate concentration to 140 or 280 mg/L eliminated this depression in microbial activity, demonstrating competition for the active sites on the enzymes by nitrate and chlorate (Fahraeus, 1952). Similar results have been shown in tomato plants, *Lycopersicon esculentum* (Hofsta, 1977).

The mode of action of chlorate toxicity was also studied in the green alga *Chlorella vulgaris* with toxicity related to growth of the algae in media containing nitrate as the nitrogen source (Solomonsson and Vennesland, 1972). Chlorate did not cause toxicity when the algae were grown in a medium with the ClO_3-/NO_3- ratio lower than 2:1 or when nitrate was absent from the culture medium. This indicates again that the nitrate reductase system, which needs a minimal amount of nitrate to become activated, is involved in chlorate toxicity and that there is competition for active sites on the enzyme by chlorate and nitrate. Similarly there is a report of a mutant chlorate-resistant strain of blue-green alga, Nostoc muscorum, which lacked nitrate-reductase activity and utilized nitrite as a nitrogen source (Singh et al., 1977).

Balch (1987) experimented with 36Cl ClO_3- as a tracer to study nitrate uptake in marine phytoplankton. In *Skeletonema costatum* and *Nitzschia closterium* chlorate was actively transported into the diatom cells, but no such active transport was seen in the dinoflagellate *Gonyaulax polyedra*. At equimolar concentrations of nitrate and chlorate, nitrate uptake rates were 1000 times higher than the chlorate uptake rates. Small amounts of nitrate in the growth medium can protect cells from chlorate toxicity by competition for the active sites on the enzymes. In the laboratory chlorate is used to select nitrate-reductase deficient bacteria from anaerobic cultures; only those mutants unable to reduce nitrate are resistant to chlorate (John, 1977).

Some species, particularly the brown algae, are very sensitive to chlorate while most other species are relatively insensitive. It may be that in non-sensitive species the reduction does not stop at the toxic chlorite but this chlorite is further reduced and does not accumulate to toxic levels. Perhaps in the sensitive species there is incomplete reduction and toxic intermediate products, probably chlorite, accumulate. This is speculative, the exact mechanism which distinguishes sensitive from non-sensitive species is not known and from a practical point-of-view, brown algae are the only sensitive species. However, brown algae are ubiquitous along coastal BC, are dominant components of the ecosystem and are the preferred sites for herring to spawn.

In eukaryotic organisms the reduction of chlorate to the toxic chlorite, by the nitrate assimilation and reductase system, is apparently an artifact; chlorate is not a naturally occurring compound (Malmqvist et al., 1991). However, in bacteria there are specific chlorate reductase enzyme systems, distinct from the nitrate reductase systems, and chlorate can be used by these organisms as an electron acceptor. In *Proteus mirabilis*, which is capable of reducing nitrate to nitrogen gas, there is a specific chlorate reductase enzyme system which can reduce chlorate, probably to chlorine (Oltmann et al., 1976; Quastel et al., 1925; Bryan and Rohlich, 1954; Malmqvist et al., 1991; van Ginkel et al., 1995).

Biological Effects of Chlorates

There is no evidence of chlorates being mutagenic, carcinogenic or teratogenic (Environment Canada, 1985).

Occurrence of Chlorates

Uses of Chlorates

Chlorate, ClO_3-, has historically been used for weed control in agriculture, at concentrations of 20 to 40 g/L , and is still used as a defoliant (Crafts and Robins, 1962, Agaev et al., 1986). Chlorates are being used increasingly as the raw material for the on-site production of chlorine dioxide used in bleaching of pulp and paper. Chlorates are used in the manufacture of matches and explosives, dyeing and printing of fabrics and tanning and finishing of leather (McKee and Wolf, 1963). When aqueous solutions, such as those used in weed control, are allowed to evaporate, the concentrated chlorate becomes a fire hazard (Goudey, 1946).

The commercial names of chlorates used as herbicides include the following: Asex; Atratol; B-Herbatox; Chlorate De Sodium; Chlorate Salt of Sodium; Chloric Acid Sodium Salt; Chlorsaure; Defol; De-Fol-ate; Dervan; Desolet; Drexel Defol; Dropleaf; Drop-Leaf; Evau-Super; Fall; Grain Sorghum Harvest Aid; Granex-O; KM; Kusa-Tohru; Kusatol; Leafex 2; Leafex 3; Natrium Chloraat; Natrium Chlorat; Ortho C ! Defoliant; Ortho C-1 Defoliant and Weed Killer; Oxycil; Shed-A-Leaf; Shed-A-Leaf 'L'; Soda Chlorate; Sodio (Chlorato DI); Sodium (Chlorate DE); Sodium Chlorate (NACLO3); Travex; Tumbleaf; Tumbleleaf; United Chemical Defoliant NO 1; Val Drop; Weed Killer.

Commercial Chlorate Production

Sodium chlorate is manufactured by acidifying, to pH 7, a saturated solution of sodium chloride in soft water. This brine is treated in electrolysis cells to produce hydrogen gas and sodium chlorate. The sodium chlorate is concentrated by evaporation and collected by filtration and drying. About 75% of the brine is converted to sodium chlorate and the remainder of the brine is recycled back to the electrolysis cell. The overall yield is about 95% (Environment Canada, 1985).

$$\mathbf{NaCl + 3H_2O = NaClO_3 + 3H_2}$$

Canadian production of sodium chlorate in 1983 was 294,000 tons; about 64% of which was used for pulp bleaching in domestic pulp and paper mills and 33% exported for other uses (Environment Canada, 1985).

Sources of Input to the Environment

The use of chlorates to generate chlorine dioxide, on-site, at pulp and paper mills has led to large amounts of chlorate being discharged in kraft pulp mill effluent (Germgard et al., 1981; Rosemarin et al., 1990). Chlorates occur in low concentrations in sodium hypochlorite, and in industrial and domestic disinfectants. In the chlor-alkali industry, producers are switching from mercury-based to membrane cell technology, resulting in the discharge of chlorates instead of mercury.

Since ClO2 is gaseous, and decomposes explosively at concentrations as low as 10% in air, it can not be transported but needs to be manufactured on-site from $NaClO_3$ and a reducing agent (often sulphur dioxide or methanol) under acidic conditions. A common reaction mechanism is:

$$\mathbf{6NaClO_3 + CH_3OH + 4H_2SO_4 = 6ClO_2 + CO_2 + 5H_2O + 2Na_3H(SO_4)_2}$$

The acid and caustic soda are recovered for reuse. During the bleaching process, 30 to 40% of the ClO_2 is converted to ClO_3-. Much of this chlorate is often discharged directly to the environment. In Sweden the concentration of chlorate in kraft mill effluent ranges from 1 to 70 mg/L, depending upon the amount of chlorine dioxide used and the effluent treatment process in place. Biological treatment may reduce chlorate to chloride (Lehtinen et al., 1988; Perrin, 1992; Perrin and Bothwell, 1992).

Levels of Chlorates Found in the Environment

Recent monitoring data indicates that typical surface water concentrations of chlorate are about 0.04 mg/L in the River Meuse and about 0.02 mg/L for the Rhine and Ijssel rivers in the Netherlands (Versteegh et al., 1993). There are few data on chlorate levels in brackish or marine waters. In the Baltic Sea, mean values of up to 53 mg ClO_3-/L were recorded in kraft pulp mill effluent discharged into coastal waters (Rosemarin et al., 1994).

The expected discharge from the Celgar Mill in the Kootenays is up to 70 mg/L in a total discharge of 76,000 to 93,600 m^3/d. Resulting dilution ratios would be 0.001 to 0.002, at high and low flows, in the Columbia River after complete mixing. Downstream chlorate levels would be between 0.07 and 0.15 mg/L These levels are within the range of values that are toxic to marine brown algae but are not known to affect freshwater life (Perrin, 1992; Perrin and Bothwell, 1992).

The Fate of Chlorates in the Environment

Physical and Chemical Processes

The fate of chlorates and their toxicity is linked closely to nitrate levels in the water. These are usually low in marine waters unless pollution is present, but may be elevated in some fresh waters. The Baltic Sea, in areas near pulp mill discharges, had up to 0.039 mg/L of nitrate nitrogen but in a British estuary nitrate-nitrogen levels ranged from 0.14 to 3.0 mg/L with 0.08 to 2.0 mg/L of ammonia nitrogen. In European fresh waters, nitrate-nitrogen levels ranged from 0.2 to 8.5 mg/L (Rosemarin et al, 1986, ,1990, 1994; Royal Commission, 1992; Herbert, 1982). In the Columbia River nitrate levels are generally in excess of 120 µg/L of nitrogen except in late summer and early fall when they drop to 40 µg/L. The critical nitrate levels appear to range from 5 µg/L, where chlorate transport is rapid, to 80 µg/L, which almost completely inhibits nitrate transport (Perrin, 1992; Perrin and Bothwell, 1992).

At 300 kg/ha sodium chlorate gives weed control for six months but is leached by high rainfall. It may remain in the soil for up to five years depending upon the rate of application, soil type, fertility, organic matter content, moisture level and weather conditions (Worthing and Walker, 1987; Hartley and Kidd, 1987).

Biological Processes

Chlorate is readily biodegraded by microorganisms under anaerobic conditions (Malmqvist et al., 1991; van Ginkel et al., 1995). There is no tendency for chlorate to accumulate or bioconcentrate in organisms and it is not magnified in the food chain. In low-nitrate waters, there is virtually no toxicity, even in sensitive species (Environment Canada, 1985).

Chlorate levels of 60 to 70 mg/L can be reduced to less than 2 mg/L with two hours of anaerobic pre-treatment and eight hours of aerobic treatment in a laboratory scale activated sludge system. A denitrifying culture can not remove chlorate under these conditions but a de-chlorating culture can reduce nitrate (Malyk, 1992).

Chlorate can be removed from bleach plant effluents by reduction with sulphur dioxide but this is an incomplete process and consumes large quantities of chemicals. Anaerobic fixed-film processes show that bacteria can remove chlorates from kraft bleach effluent with less than one hour retention time. Several bacterial isolates were found which were gram-negative, catalase-positive, oxidase-positive, motile rods. None could ferment glucose and all could grow aerobically using nitrate as an electron acceptor (Malmqvist and Welander, 1992).

Drinking Water

General

Chlorine dioxide is an attractive alternative to chlorine in drinking water disinfection since it inactivates bacteria and viruses over a wide range of pH values, has residual effect, controls taste and odour compounds, oxidizes iron and manganese and does not produce trihalomethanes (Werdehoff and Singer, 1987). In nearly neutral conditions chlorine dioxide reactions in water result in reduction to the chlorite ion (Miller et al., 1978) but at alkaline pH values a disproportionation reaction produces chlorite and chlorate ions (Gordon et al., 1972). Photolytic decomposition by sunlight and fluorescent lighting also produces chlorate from chlorine dioxide (Griese et al., 1992). Aqueous chlorine reacts with chlorine dioxide to produce chlorate ion with a very long half-life (15 to 20 days) and also produces chlorate in a reaction with chlorite ion under drinking water conditions. Treating raw drinking water with ClO2 results in chlorate in the finished water at levels up to 660 µg/L. This production of chlorate is the main disadvantage of chlorine dioxide as a water disinfection agent (Couri et al, 1982).

If chlorine dioxide is to be a viable water treatment alternative, chlorate and chlorite ions need to be reduced or removed. The amount of chlorate produced can be reduced by using hypochlorite solutions of less than 10% available chlorine in the disinfection step (Gordon et al., 1993; Bolyard et al., 1993). Granular activated carbon reversibly sorbs chlorate from drinking water but does not reduce the chlorate. There is no formation of chlorate, in the dark, from the chlorite/activated carbon system (Gonce and Voudrias, 1994).

Effects

Severe sodium chlorate poisoning occurred after a suicidal ingestion of 150 to 200 grams. There was hemolysis and coagulation, treated with transfusions, heparin and fresh plasma. Complete renal failure occurred after one hour and dialysis was required for several weeks. Survival after ingestion of 45 and 100 g doses resulted after treatment with sodium thiosulphate (2 to 5 grams in 200 mL of 5% sodium bicarbonate) and dialysis when renal failure occurred. A 29 year old man ingested 20 g of sodium chlorate (230 mg chlorate/kg) and became cyanotic and his hemoglobin level dropped to 11 g/100 mL. He was anuric for two weeks and finally improved and was released after six weeks.

There were 14 cases of sodium chlorate poisoning reported in patients between three and 55 years old. Doses in excess of 100 g (79 g chlorate ion) were fatal. A 46 year old woman died in 20 hours after a dose of 15 g (218 mg chlorate/kg). This was the lowest fatal dose. Another woman died in five days after ingesting 30 g (436 mg chlorate/kg) in spite of treatment. An 18 year old man survived 100 g (1.45 g chlorate/kg) after receiving extensive treatment.

Toxicity has been reported after ingestion of match heads (Steffen and Seitz, 1981; Ellenhorn and Barceloux, 1988; NRC, 1987). A dose of five to ten grams can prove fatal in adults and two grams in small children (Hartley and Kidd, 1987). Dermal absorption of chlorate used as a herbicide will not result in systemic poisoning, large oral doses are required (Hayes and Laws, 1991).

Guidelines From the Literature

The USEPA had recommended standards of 60 µg/L for chlorine dioxide and 7 µg/L for chlorite plus chlorate in drinking water (Smith, 1989). These appear to be very conservative values and it is estimated that even dialysis patients would not be harmed by these levels; the chlorate is a minor component and the regulations were primarily to protect against the more prevalent chlorite and chlorine dioxide. Patients would get more chlorate from their drinking water than from their dialysis treatments. The USEPA recommended that the combined residuals of chlorine dioxide, chlorite and chlorate not exceed 1 mg/L in the water distribution system (Gordon et al., 1990; Pontius, 1990). Again, this is designed to protect against chlorite and chlorine dioxide, primarily, and not chlorate. Chlorite and chlorine dioxide are regulated individually under the EPA's new Disinfection-Disinfection By-products Rule (Pontius, 1990; Lykins et al., 1992; EPA, 1993). There is no specific chlorate guideline under disinfection by-products and likely no need since the chlorate levels formed by these processes are relatively minor compared to other oxychlorines.

Recommended Guidelines

The recommended interim guideline for chlorate in drinking water is 2.4 mg/L to protect 5 kg infants since they drink more water in proportion to their body weight than adults. The maximum daily intake rate from all sources is 1 mg/kg body weight. Water consumption is assumed to be 1.5 L/day. Not all of this intake is permitted in the water; there must be some allowance reserved for intake from food and other sources. In the table below a ratio of about 70% in the water and 30% from other sources is assumed.

Table 8. Body Weight and Safe Chlorate Concentration in the Drinking Water

body weight in kilograms	5	10	20	30	50	70	90
[chlorate] in mg/L	2.4	4.8	9.6	14.4	24	33.6	43.2

The calculation is: 1 mg/Kg * 5 Kg = 5 mg / 1.5 L/day = 3.4 mg * 0.7 = 2.38 mg/L rounded to 2.4 mg/L for 5 Kg infants. This leads to overprotection for adults who are heavier but tend to drink about the same amount of water.

Rationale

Doses in excess of 100 mg/kg, 7 grams for a 70 kg adult human or 500 mg for a 5 Kg baby, are generally fatal; doses of this magnitude are unlikely from ambient exposures. The lowest doses reported as fatal in humans are 2 g in small children and 5 g in adults (Hartley and Kidd, 1987) or about 100 mg/kg. Applying a factor of 0.01 to derive an NOEL from the acute fatally toxic threshold gives a guideline of 1 mg/kg. A further factor of 0.7 is applied to reserve some of the maximum daily dose for food and other sources and permit only 70% in the water.

The normally accepted drinking water consumption rate for average people in north temperate climates is 1.5 L/day. Under these conditions 5 kg infants would receive their maximum dose of 5 mg chlorate in water with 3.4 mg/L chlorate, 10 kg children would receive their maximum dose of 10 mg chlorate in water with 6.8 mg/L chlorate, 30 kg children would receive their maximum dose of 30 mg chlorate in water with 20 mg/L chlorate and 70 kg adults would receive their maximum dose of 70 mg chlorate in water with 46.6 mg/L chlorate.

In both controlled studies and in actual community water supplies where chlorine dioxide is being used as a disinfectant, there have been no adverse health effects reported (Smith, 1989). The only sources of chlorate in raw drinking water would be drift or runoff from herbicide applications and discharges from pulp mills into the drinking water supply streams. The 'worst-case' situation would be a pulp mill discharging 100 mg/L of chlorate into a river with minimal dilution of 20:1 for a final concentration of 5 mg/L in the raw water. Calculations for the Columbia River, after complete mixing downstream from the Celgar Mill, indicate that chlorate levels would be between 70 and150 µg/L. Drinking water intakes should not be located immediately downstream of pulp mill discharges. Levels in drinking water are probably determined by residues from chlorine dioxide disinfection rather than from raw water levels.

Terrestrial Life

Plants

Chlorate is rapidly absorbed via roots and leaves and is translocated throughout the plant causing respiration increases, catalase activity decreases and depletion of food reserves. Chlorate is used as a herbicide and as a pre-harvest defoliant but at much higher concentrations (greater than 20 grams/L) than would be encountered in effluent discharges or irrigation water.

Animals

An unspecified amount of sodium chlorate applied to a field to kill weeds poisoned 15 cattle and killed another six. In another case two cattle died and several others were affected after 400 g of sodium chlorate was scattered on a field to kill thistles. Sheep appear to be less susceptible and have grazed unharmed on heavily treated plots. In a horse the lethal dose was 250 g, 500 g was lethal in a cow: 0.26 g/kg had no effect on a calf and in a cow 0.1 to 0.25 g/kg caused symptoms but was not fatal. A cow receiving 0.06 to 0.18 g/kg for three days was sick but survived, sheep get methemoglobinemia at 1 g and 5 g/kg is lethal to chickens. The minimum lethal dose is about 1 g/kg in cattle, 1.5 to 2.5 g /kg in sheep and 5 g/kg in chickens (Clarke et al., 1981; Buck et al., 1976; Humphreys, 1988). Wildlife should respond at similar concentrations, which are well above expected effluent discharge levels.

African Green Monkeys, *Cercopithecus aethiops*, were given $NaClO_3$ in their water at up to 60 mg/kg/day for several months. There was no change in serum thyroxine or any hematological changes in the animals (Bercz et al., 1982). The rat oral LD_{50} is 1200 mg/kg and for the mouse it is 596 mg/kg (Hayes, 1982; Hayes et al., 1991). The oral LD_{50} in rats is 12000 mg/kg (Springer, 1957).

Intoxication with chlorate salts is characterized by methaemoglobin formation, haemolysis and renal insufficiency. The toxic effects on the erythrocytes can be reproduced in-vitro. Incubation of erythrocytes with chlorates induces concentration-dependent oxidation of haemoglobin. This methaemoglobin formation is followed by denaturation of the globin, cross-linking of erythrocyte membrane proteins and inactivation. In-vivo, an oral dose of sodium chlorate at 1000 mg/kg to rabbits resulted in high serum and urine levels but methaemoglobin was not formed and no nephrotoxic effects could be observed.

In dogs the lethal oral dose of sodium chlorate is about 2000 mg/kg; 1000 mg/kg causes severe methemoglobinemia. The LD50 is about 500 to 2000 mg/kg in two to four daily doses (Buck et al., 1976; Sheahan et al, 1971).

Guidelines From the Literature

No literature guidelines were found for chlorate on terrestrial life.

Recommended Guidelines

An interim drinking water guideline of 2.9 mg/L is proposed for wildlife and livestock.

Rationale

For dogs applying the safety factor of 0.01 to an LD_{50} of 500 mg/kg gives a guideline of 5 mg/kg. In mice applying the safety factor of 0.01 to an LD_{50} of 596 mg/kg gives a guideline of 6 mg/kg. The standard allometric equations indicate water intake at about 80 mL/kg for these animals. At 5 mg/kg chlorate and 80 mL/kg water intake the chlorate concentration in the water should not exceed 62.5 mg/L for an adult animal. Using the same ratio of chlorate intake for neonate animals to adult animals, of about 1:15, as used in humans, the chlorate level in animal drinking water to protect neonate animals should be about 4.166 mg/L. Applying the same allocation of the maximum chlorate dose to water and food sources as was applied to humans, 70% in the water and 30% in the food, the water guideline becomes 2.916 mg/L, rounded to 3 mg/L. This is close to the 2.4 mg/L proposed for human drinking water.

The calculation is: 5 mg/Kg / 0.080 L = 62.5 mg/L / 15 = 4.166 mg/L * 0.7 = 2.916 mg/L which is rounded to 3 mg chlorate/L of drinking water.

Aquatic Life

Marine Algae

Brown algae ecosystems in the Baltic Sea were studied to document the effects of chlorate in kraft pulp mill effluent (Rosemarin *et al*.; 1986, 1990, 1994; Lehtinen *et al*., 1988). The coastal area was subjected to pulp mill effluent containing high levels of chlorate and there were adverse effects on local algal communities. The main species studied was *Fucus vesiculosus*, a common, ecologically significant, large brown alga. Excised fronds exposed to chlorate for 70 hours in toxicity tests exhibited reduced photosynthetic rates at chlorate concentrations down to 0.4 mg ClO_3- per litre. Both chlorate alone and pulp mill effluent containing chlorate were tested, confirming that it was the chlorate causing the effect. The Baltic Sea area was low in nitrate nitrogen, less than 0.039 mg/L.

Mesocosm experiments with *Fucus vesiculosus* transplants were carried out for six months using chlorate alone or pulp mill effluent containing various concentrations of chlorate. These experiments resulted in a one month EC_{50} for frond growth, of 0.10 mg ClO_3- /L. The six month trials gave a LOEC of 0.015 (0.010-0.020) mg ClO_3- /L and a NOEC of 0.005 mg ClO_3- /L. The effects on *Fucus serratus* were also measured and resulted in a one month EC_{50} for frond growth, of 0.13 mg ClO_3- /L. Transplantation to sites affected by the pulp mill effluent gave similar results. Once the chlorate was removed from the effluent the *Fucus* re-grew on its former sites indicating that it was the chlorate and not the effluent that was responsible for the toxicity. Other sensitive species of large brown algae included *Chorda filum*, *Ectocarpus siliculosus* and *Pilayella littoralis*. The blue-green algae, green algae (*Cladophora rupestri* and *Spirogyra*) and red alga (*Ceramium*) were all much less sensitive to chlorate. Removal of the chlorate from the effluent resulted in the re-colonization of the sites by the brown algae.

In 1998 Stauber reported on experiments with the marine microalgae, *Nitzschia closterium* and *Dunaliella tertiolecta*. When nitrate was limiting, less than 0.005 mg/L the 72 hour EC50 values for chlorate were 1.9 and 11 mg/L, respectively. At higher nitrate levels of 1 mg/L both algae were much less sensitive to chlorate with their respective EC_{50}'s being 10 and 11 mg/L, respectively. At nitrate levels of 15 mg/L both algae were insensitive to chlorate with their respective EC_{50}'s being over 500 and over

1000 mg/L, respectively. In *Dunaliella* only, when the molar concentration of chlorate exceeded that of nitrate, nitrate uptake was reduced due to inhibition of nitrate reductase activity. This was likely due to the chlorate being being converted by the nitrate reductase to the toxic chlorite. These microalgae are not as sensitive to chlorate as the larger kelps.

The lowest effect level, an LOEC for primary productivity in mixed marine phytoplankton, found was 50 mg/L of chlorate at less than 0.039 mg/L nitrate (Rosemarin *et al*., 1994).

Marine Invertebrates and Fish

There is no suitable published data on the effects of chlorate on marine fish or invertebrates. Marine organisms should not react any differently from freshwater organisms, given the mechanism of chlorate toxicity.

Freshwater Life

Chlorate appears to be non-toxic (LC_{50} is greater than 100 mg/L) to freshwater life (Wijk and Hutchinson, 1995); there are no sensitive brown algae in these habitats. An experiment was carried out in some experimental troughs at the South Thompson River facility at Chase BC. Nitrate-nitrogen concentrations were between 5.8 and 11.5 µg/L and KH_2PO_4 was added to each trough to give a phosphorus concentration of 5 µg/L. This ensured that the diatom-dominated community would be nitrogen limited. $NaClO_3$ was added to give final chlorate concentrations of 0, 25, 50, 100, 200 and 500 µg/L. Another experiment was run at a constant chlorate concentration of 300 µg/L and $NaNO_3$ was added to give nitrate-nitrogen concentrations of 0, 25, 50, 100 and 200 µg/L. There was no change in the diatom community nor in the specific growth rates of the diatoms (Perrin, 1992; Perrin and Bothwell, 1992).

Five species of insects, *Isoperla transmarina*, *Baetis tricaudatus*, *Tricorythodes minutus*, *Dasycorixa hybrida* and *Haliplus sp*. were exposed for 10 days to chlorate at 0.5, 5.0, 50.0 and 100.0 mg/L. There was no difference in survival from the controls at any of these chlorate concentrations. The 48 hour LC_{50} for Daphnia magna was estimated at 3162 mg/L. Effluent chlorate levels from a bleached pulp mill using 100% chlorine dioxide ranged from less than 0.002 to 75 mg/L at the point where the effluent entered the river. Maximum levels at 25 and 127 km downstream reached 3.25 and 1.00, respectively (Dosdall et al., 1997).

The lowest effect level found, an LOEC toxicity threshold, to an aquatic microorganism (*Entosiphon sulcatum*), was 817 mg/L of chlorate at 27 mg/L nitrate (Bringmann and Kühn, 1980). For freshwater algae the lowest effect level, an LOEC for growth inhibition in *Chlorella vulgaris*, was 334 mg/L chlorate at 28 mg/L nitrate (Perrin and Bothwell, 1992). The lowest effect level, a 24-hour LC_{50} to an aquatic invertebrate (the water flea, *Daphnia magna*) was 880 mg/L of chlorate at an unspecified nitrate level (Bringmann and Kühn, 1977). The lowest effect level, a 96-hour LC_{50} to a freshwater fish, (larval cherry salmon, *O. masou*) was 863 mg/L of chlorate at an unspecified nitrate level (AQUIRE, 1984).

Guidelines From the Literature

No guidelines for chlorates affecting marine or freshwater life were found in the literature.

Recommended Guidelines

For freshwater life a guideline is set to protect the most sensitive species, which appear to be green algae. The recommended guideline is 30 mg/L of chlorate ion (37 mg/L sodium chlorate).

For marine life, a guideline is set to protect the most sensitive species, which are the brown algae. These include the kelps (*Macrocystis*) and *Fucus* (bladder wrack). The recommended guideline is 5 µg/L of chlorate ion (6 µg/L sodium chlorate).

Rationale

In freshwater situations the lowest effect level, a LOEC for growth inhibition in *Chlorella vulgaris*, was 334 mg/L chlorate at 28 mg/L nitrate (Perrin *et al*., 1992). Applying a LOEL to NOEL factor of 0.1 results in a guideline of 33.4 mg/L, which hasa been rounded to 30 mg/L. The highest reported NOEL, for growth inhibition in *Chlorella vulgaris*, was also 334 mg/L chlorate but at 280 mg/L nitrate (Perrin et al., 1992).

In the coastal marine habitat pulp mill discharges occur in the same zone where the sensitive brown algae grow. This guideline is based on the NOEC of 5 µg/L chlorate which resulted from a 6 month chronic study on the growth of Fucus vesiculosus (Lehtinen et al., 1988; Rosmarin et al., 1994). The LOEC from these studies was 15 µg/L chlorate and the EC_{50} was 100 µg/L chlorate. The reported values from these studies confirm the standard 20:1 ratio of EC_{50} (or LC_{50}) to NOEC that is generally applied in deriving guidelines.

Irrigation, Recreation and Industrial Uses

Chlorates are used as herbicides but at concentrations over 20 gram/L. In fresh water situations the worst case situation would be a pulp mill discharging 100 mg/L of chlorate into a river with a minimal dilution of 20:1 for a final concentration of 5 mg/L. This should not affect plant growth. There is no evidence, given the mode of action of chlorates, to suspect that they would have any affect on recreational activities at the expected concentrations in fresh or marine waters. There are no known literature guidelines or documented effects on irrigation, recreation or industrial uses and none are anticipated at the maximum expected concentrations. No guidelines are set for these water uses.

Research

The one major area where more data is required is in drinking water guidelines where chlorates are made during the disinfection stage with chlorine dioxide. There is insufficient data on the appropriate organisms to use the preferred CCME protocol to develop CCME guidelines.

References

Aberg, B. 1947. On the Mechanism of the Toxic Action of Chlorates and some related substances upon young Wheat plants. Kungl. Lantbrukshogsk. Ann. 15: 37-107.

Agaev, R. A., V. B. Danilov, V. A. Khachaturov, B. Kh. Kasymov and B. Sh. Tishabaev. 1986. The Toxicity to Warm-blooded Animals and Fish of new Defoliants based on Sodium and Magnesium Chlorates. Uzb. Biol. Zh. 1: 40-43.

Aieta, E. M. and J. D. Berg. 1986. A Review of Chloride dioxide in Drinking Water Treatment. J. Am. Wat. Wks. Ass. 78: 62.

Alabaster, J. S. 1969. Survival of Fish in 164 Herbicides, Insecticides, Fungicides, Wetting Agents and miscellaneous substances. Int. Pest. Control. 11: 29-35.

Anderson, B. G. 1946. The Toxicity Thresholds of Various Sodium Salts Determined by the use of Daphnia magna. Sewage Works Journal 18: 82.

AQUIRE. 1984. USEPA, Duluth, MN. 55804.

Armour, M. A. 1991. Hazardous Laboratory Chemicals Disposal Guide. CRC Press. Boca Raton, Florida.

Balch, W. M. 1987. Studies of Nitrate Transport by Marine Phytoplankton using 36Cl-ClO3- as a Transport Analogue. J. Phycol. 23: 107-118.

Bercz, J. P., L. Jones, L. Garner, D. Murray and D. A. Ludwig. 1982. Subchronic Toxicity of Chlorine Dioxide and Related Compounds in Drinking Water in the Nonhuman Primate. Environmental Health Perspectives 46: 47-55.

Bolyard, M., F. P. Snyder and D. P. Hautman. 1993. Sources of Chlorate Ion in US Drinking Water. Journ. Amer. Water Works Assoc. 85(9): 81-88.

Bringmann, G. and R. Kuhn. 1980. Comparison of the Toxicity Thresholds of Water Pollutants to Bacteria, Algae and Protozoa in the Cell Multiplication Inhibition Test. Water Res. 14: 231-241.

Bringmann, G. and R. Kuhn. 1977. Befunde der Schadwirkung wassergefahrdender Stoffe gegen Daphnia magna. Wasser Abwasser-Forsch. 10: 161-166.

Bryan, E. H. and A. G. Rohlich. 1954. Biological Reduction of Sodium Chlorate as applied to measurement of Sewage BOD. Sewage Ind. Wastes. 26: 1315-1324.

Buck et al. 1976. Clinical and Diagnostic Veterinary Toxicology, 2nd. Edition.

Clarke, M. L., D. G. Harvey and D. J. Humphreys. 1981. Veterinary Toxicology. 2nd. Edition. Bailliere Tindall, London.

Couri, D. M. S. Abdel-Rahman and R. J. Bull. 1982. Toxicological Effects of Chlorine dioxide, Chlorite and Chlorate. Envir. Health Perspect. 46: 13.

Crafts, A. S. and W. W. Robins. 1962. Weed Control. McGraw-Hill, NY.
CRC Handbook. 1993. CRC Handbook of Chemistry and Physics. CRC Press Inc., Boca Raton, Florida.

Dosdall, L. M., L. R. Goodwin, R. J. Casey and L. Noton. 1997. The Effect of Ambient Concentrations of Chlorate on Survival of Freshwater Aquatic Invertebrates. Water Quality Research Journal of Canada. 32(4): 839-854.

Ellenhorn, M. J. and D. G. Barceloux. 1988. Medical Toxiciology-Diagnosis and Treatment of Human Poisoning. Elsevier Science Publishing Co., Inc. NY.

Ellis, M. M. 1937. Detection and Measurment of Stream Pollution (Related Principally to Fish Life). US Dept. Com., Bur. Fish. Bull. 22.

Environment Canada. 1985. Sodium Chlorate: Environmenmtal and Technical Information for Problem Spills. Technical Services Branch, Environmental Protection Service, Ottawa, Canada.

EPA. 1993. Draft Phase 1-D/DBP Rule for Regulatory Negatiations Process.

Fahraeus, G. 1952. Influence of Nitrate concentration upon Chlorate Toxicity in Microorganisms. Acta Chem. Scand. 5: 1416-1417.

Germgard, U., A. Teder and D. Tormund. 1981. Chlorate formation during Chlorine dioxide Bleaching of Softwood Kraft Pulp. Paperi Ja Puu 61: 127-133.

Goksoyr, J. 1951. On the Effect of Chlorate upon the Nitrate Reduction of Plants. I. Experiments with Aspergillus oryzae. Physiol. Plant. 4: 498-513.

Goksoyr, J. 1952. On the Effect of Chlorate upon the Nitrate Reduction of Plants. II. The Effect upon the Nitrate-reducing system in Escherichia coli. Physiol. Plant 5: 228-240.

Gonce, Nancy and Evangelos A. Voudrias. 1994. Removal of Chlorite and Chlorate Ions from Water using Granulated Activated Carbon. Water Research 28(5): 1059-1069.

Gordon, G., R. G. Keiffer and D. H. Rosenblatt. 1972. The Chemistry of Chlorine dioxide. In: Progress in Inorganic Chemistry. S. J. Lippard, Editor. Vol. 15: 201-286. Wiley, NY.

Gordon, G., B. Slootmaekers, S. Tachiyashiki and D. W. Wood. 1990. Minimizing Chlorite ion and Chlorate ion in Water treated with Chlorine dioxide. J. Am. Wat. Wks. Ass. 79: 64.

Gordon, G., L. C. Adam, B. P. Bubnis, B. Hoyt, S. J. Gillette and A. Wilczak. 1993. Controlling the Formation of Chlorate Ion in Liquid Hypochlorite Feedstocks. Journ. Amer. Wqater Works Assoc. 85(9): 89-97.

Goudey, R. F. 1946. Chemical Weed Control. Jour. AWWA. 38: 186.

Griese, M. J., J. J. Kaczur and G. Gordon. 1992. Combining Methods for the reduction of Oxychlorine Residuals in Drinking Water. J. Am. Wat. Wks. Ass. 84: 69.

Hartley, D. and H. Kidd. Editors. 1987. The Agrochemical Handbook. 2nd. Edition. The Royal Society of Chemistry. Lechworth, Herts, England.

Hayes, W. J. 1982. Pesticides Studied in Man. Baltimore/London, Williams and Wilkins.

Hayes, W. J. and E. R. Laws. 1991. Handbook of Pesticide Toxicology. Volume 2. Classes of Pesticides. Academic Press, Inc. NY.

Herbert, R. A. 1982. Nitrate dissimilation in Marine and Estuarine Sediments. In: Nedwell, D. B and C. M. Brown. Editors. Sediment Microbiology. Academic Press, London. p 54-71.

Herbicide Handbook. 1983. Weed Science Society of America. Herbicide Handbook. 5th. Edition. Weed Science of America. Champaign, Ill.

Hofstra, J. J. 1977. Chlorate Toxicity and Nitrate Reductase Activity in Tomato Plants. Physiol. Plant 41: 65-69.

Humphreys, D. J. 1988. Veterinary Toxicology. 3rd. Edition. Bailliere Tindell, London.

Hutchinson, T. H. 1994a. Sodium Chlorate: Toxicity to the Freshwater Alga Scenedesmus subspicatus. Zeneca Brixham Environmental Laboratory Internal Report BLS 1742/B.

Hutchinson, T. H. 1994b. Sodium Chlorate: Toxicity to the Marine Diatom Phaeodactylum tricornutum. Zeneca Brixham Environmental Laboratory Internal Report BLS 1741/B.

John, P. 1977. Aerobic and anaerobic Bacterial Respiration monitored by Electrodes. J. Gen. Microbiol. 98: 231-238.

Juhnke, I. and D. Luedemann. 1978. Ergebnisse der Unterschung von 200 Chemischen Verbindungen auf akute Fischtoxizitat mit dem Goldorfentest. Z. Wasser Abwasser-Forsch. 11:161-164.

Jones, J. R. E. 1941. A Study in the Relative Toxicity of Anions, with Polycelis nigra (Planarian) as test Animal. Jour. Exp. Biol. 18: 170.

Kroon, A. G. M. 1993. Toxicity of Sodium Chlorate to the Freshwater Alga Scenedesmus quadricauda. Akzo Research Laboratories. Arnhem. Internal Report CRL F 93115.

Lehtinen, K. J., M. Notini, J. Mattson and L. Landner. 1988. Disappearance of Bladder-wrack (Fucus vesciculosus L.) in the Baltic Sea: Relation to Pulp Mill Chlorate. Ambio 17: 387-393.

Liljeström, S. and B. Aberg. 1966. Studies on the Mechanism of Chlorate Toxicity.

Lantbrukshogsk. Ann. 32: 93-107.

Lykins, B. W. et al. 1992. Chemical, Microbiological and Mutagenic effects of using Alternative Disinfectants for Drinking Water Treatment. Proc. 42nd. Envir. Engng. Conf. Lawrence, Kansas.

Malmqvist, A., T. Welander and L. Gunnarsson. 1991. Anaerobic Growth of Microorganisms with Chlorate as an electron acceptor. Appl. Environ. Microbiol. 57: 2229-2232.

Malmqvist, A. and T. Welander. 1992. Anaerobic Removal of Chlorate from Bleach Effluents. Water Science and Technology 25(7): 237-242.

Malyk, W. 1992. M. Eng. Thesis, McMaster University.

Mark, U. E. and I. C. M. Arends. 1993. Acute Toxicity of Sodium Chlorate to Pimephales promelas. Akzo Research Laboratories Arnhem. Internal Report CRL F 93115.

Mark, U. E. andE. Hantink-de Rooij. 1991. Acute Toxicity of Sodium Chlorate to Brachydanio rerio. Akzo Research Laboratories Arnhem. Internal Report CRL F 91163.

Matida, Y., S. Kumura, H. Kumada and M. Yokote. 1974. Effects of some Herbicides applied in the Forest to Freshwater Fishes and other Aquatic Organisms. I. Bull. Freshwater Fish. Res. Lab. 25: 41-53.

Matida, Y., Y. Furuta, H. Kumada, H. Tanake, M. Yokote and S. Kumura. 1975. Effects of some Herbicides applied in the Forest to Freshwater Fishes and other Aquatic Organisms. II. Effects of Sodium Chlorate and Ammonium Sulfamate to the Aquatic Organisms in an Artificial Stream. Bull. Freshwater Fish. Res. Lab. 26: 79-84.

Matida, Y., Y. Furuta, H. Kumada, H. Tanake and M. Yokote. 1976. Effects of some Herbicides applied in the Forest to Freshwater Fishes and other Aquatic Organisms. III. Experiments on the assessment of Acute Toxicity. Bull. Freshwater Fish. Res. Lab. 26: 79-84.

McIntyre, L. and L. Noton. 1990. Chlorates: A Literature Review of aspects relevant to the Aquatic Environment. Manuscript Report. Alberta Environment.

McKee, J. E. and H. W. Wolf. 1963. Water Quality Criteria. Pub. No. 3-A, State Water Resources Control Board, California.

Meinck, F., H. Stoof and H. Kohlschütter. 1956. Industrial Waste Waters (Industrie-Abwasser). Second Edition. Gustav Fischer Verlag. Stuttgart536 48 D. M.

Merck Index. 1989. The Merck Index-Encyclopedia of Chemicals, Drugs and Biologicals. Merck and Co. Inc., Rahway, N. J.

Miller, G. W., R. G. Rice, C. M. Robson, W. Kuhn and H. Wolf. 1978. An Assessment of Ozone and Chlorine dioxide technologies for treatment of Municipal Water Supplies. p. 9-57 to 9-89 in Report of EPA Grant R804385-01. Municipal Environmental Research Laboratory. Office of Water Supply. USEPA, Cincinnati, Ohio.

Nishiuchi, Y. 1980. Effect of pH on the Toxicity of Pesticides to Aquatic Animals. II. Seitai Kagaku 3: 73-79.

Nishiuchi, Y. 1982. Toxicity of Pesticides to Freshwater Organisms. LXXIX. Effects of Water Temperature on Pesticide Susceptibility of Daphnids. Suyisan Zoshoku 30:158-162.

Nishiuchi, Y. and H. Iwamura. 1983. The Toxicity of Pesticides to some Freshwater Organisms. LXXXIV. Toxicity to Rainbow Trout. Suisan Zoshoku 30: 222-225.

NRC. 1987. Drinking Water and Health. Volume 7. National Research Council, Wash. DC. National Academy Press.

Oltmann, L. F., W., N. M. Reijnders and A. H. Stouthamer. 1976. Characterization of purified Nitrate reductase A and Chloraste reductase C from Proteus mirabilis. Arch. Microbiol. 111: 25-35.

Perrin, C. J. 1992. The Growth and Structure of a Lotic Periphyton Community in relation to additions of Chlorate: Completion Report. Limnotek Research and Development Inc., Vancouver, BC. Submitted to Dept. Fisheries and Oceans, Vancouver, BC.

Perrin, C. J. and M. L. Bothwell. 1992. Chlorate Discharges from Pulp Mills: An Examination of Effects on River Algal Communities. Water Poll. Res. J. Canada. 27(3): 473-485.

Pontius, F. W. 1990. Complying with the new Drinking Water Quality Regulations. J. Am. Wat. Wks. Ass. 82: 32.

Quastel, J. H., M. Stephenson and M. D. Whetham. 1925. Some Reactions of resting Bacteria in relation to Anaerobic growth. Biochem. J. 19: 304-317.

Rosemarin, A., J. Mattsson, K. J. Lehtinen, M. Notini and E. Nylen. 1986. Effects of Pulp Mill Chlorate (ClO_3^-) on Fucus vesiculosus: A Summary of Projects. Ophelia suppl. 4: 219-224.

Rosemarin, A., J. Mattsson, K. J. Lehtinen, M. Notini and E. Nylen. 1990. Effects of treated and untreated Softwood Pulp Mill Effluents on Baltic Sea Algae and Invertebrates in model Ecosystems. Nord. Pulp Pap. Res. J. 2: 83-87, 103.

Rosemarin, A., K. Lehtinen, M. Notini and J. Mattson. 1994. Effects of Pulp Mill Chlorate on Baltic Sea Algae. Environ. Pollut. 85: 3-13.

Royal Commision on Environmental Pollution. 1992. Freshwater Quality-16th Report. Her Majesty's Stationary Office (HMSO), London.

Sheahan, B. J. et al. 1971. Res. Vet. Sci. 12(4): 387-389.

Shifrer, C. C., E. J. Middlebrooks, D. B. Porcella and W. F. Sigler. 1974. Effects of Temperature on the Toxicity of Oil Refinery Waste, Sodium Chlorate and Treated Sewage to Fathead Minnows. Centre for Water Resources Research, Utah State University, Logan, Utah. Report # PRWG 105-4 (NTIS PB-237 516).

Singer, P. C. and W. K. O'Neil. 1987. Technical Note: The formation of Chlorate from the reaction of Chlorine and Chlorite in dilute Aqueous solution. J. Am. Wat. Wks. Ass. 79: 75.

Singh, H. N., K. C. Sonie and H. R. Singh. 1977. Nitrate regulation of the Heterocyst differentiation and Nitrogen Fixation in a Chlorate-resistance mutant of the blue-green alga, Nostoc muscorum. Mutat. Rtes. 42: 447-452.

Smith, R. P. 1989. Chlorine Dioxide and Hemodialysis. Government Reports, Announcements and Index. Issue 2, 1991. NTIS/DE90015912. Dept. Energy, Wash. DC.

Solomonsson, L. P. and B. Vennesland. 1972. Nitrate reductase and Chlorate Toxicity in Chlorella vulgaris Beijerinck. Plant Physiol. 50: 421-424.
Spencer, E. Y. 1982. Guide to the Chemicals Used in Crop Protection. 7th. Edition. Publication 1093. Research Institute, Agriculture Canada, Ottawa.

Springer, P. F. 1957. Effects of Herbicides and Fungicides on Wildlife. North Carolina Pesticide Manual. 87. North Carolina State College, Raleigh, NC.

Stauber, Jennifer L. 1998. Aquatic Toxicology. 41: 213-227.

Steffen, C. and R. Seitz. 1981. Arch. Toxic. 48(4): 281-288.

Steffen, C. and E. Wetzel. 1993. Chlorate Poisoning: Mechanism of Toxicity. Toxicology 84: 217-231.

van Ginkel, C. G., C. Plugge and C. Stroo. 1995. Microbial Reduction of Chlorate with various Energy substrates under Anaerobic conditions. In Press: referenced from Wijk, D. J. and T. H. Hutchinson. 1995.

Versteegh, J. F. M., J. Neele and R. F. M. J. Cleven. 1993. Chloriet en Chloraat in drinkwater: een desinfectie-of een milieuprobleem? H2O 26: 680-686.

Werdehoff, K. S. and P. C. Sainger. 1987. Chlorine dioxide effects on THMFP, TOXFP andf the formation of inorganic by-products. J. Am. Wat. Wks. Ass. 79: 107.

Wijk, D. J. and T. H. Hutchinson. 1995. The Ecotoxicity of Chlorate to Aquatic Organisms: A Critical Review. Ecotoxicology and Environmental Safety. 32: 244-253.

Worthing, C. R. and S. B. Walker. 1987. The Pesticide Manual-A World Compendium. 8th. Edition. Thorton Heath, UK. The British Crop Protection Council.