CONSULTATION PACKAGE

ON

TRIHALOMETHANES

Proposed Guideline

for the

Guidelines for Canadian

Drinking Water Quality

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Improved removal of organics prior to disinfection and/or the relocation of the point of disinfection are trihalomethane control options available at most water treatment plants. The effectiveness of these control options is a function of the nature of both the water supply and the treatment facility. In general, however, these strategies can reduce trihalomethane levels below 150 μg/L at existing facilities and normally below 50 μg/L at facilities that have been expanded or designed to minimize trihalomethane production. Efforts to control trihalomethanes using these strategies may require increased chemical usages that may, in turn, lead to increased coagulant levels in the treated water (e.g., aluminum), increased treated water turbidity and increased water use for filter back-washing. Also, relocating the point of chlorination can reduce disinfection times at some plants, thus reducing the level of protection provided by the disinfection process.

A second option is to use other disinfectants, such as chloramines, chlorine dioxide or ozone. The use of chloramines is fairly widely practised in North America and is a relatively inexpensive option for controlling trihalomethanes. It is, however, a less effective disinfectant than chlorine and, therefore, not recommended as a primary disinfectant. Also, the health effects of chloramine and its by-products have not been well assessed.

Chlorine dioxide is also fairly widely used as a disinfectant and taste and odour control chemical in water treatment. Although it is an effective disinfectant, there are concerns regarding its health effects and those of its two by-products (i.e., chlorite and chlorate), which require further investigation.

Ozone is a relatively new disinfection technology in North America. Although ozone is a very effective disinfectant, its technology is more sophisticated than chlorination and more difficult to operate and maintain. Ozonation of water can result in an increase in the amount of biodegradable organics by altering the amount of non-biodegradable organics. These organics, unless removed, can cause bacterial growth problems in distribution systems. Ozone reacts with organics in water to produce by-products that may be of concern. Also, because ozone has no residual, another disinfectant must be added after treatment to protect the water in the distribution system. This secondary disinfectant can react with the organics to form additional by-products.

For the removal of trihalomethanes, the most common methods of special treatment are air stripping and adsorption. More information on these treatment methods can be found in Appendix B, Section 2.2—"Special Treatment".

In general, all disinfectants and disinfection technologies have advantages and disadvantages. Care must be exercised when controlling trihalomethanes to ensure that such controls do not result in or encourage the use of alternative disinfectants that produce risks or problems greater than those posed by the trihalomethane issue they resolve.

2.4 Need for Guideline

Because trihalomethanes are formed in drinking water primarily as a result of

chlorination of organic matter present in raw water supplies, it is important to recognize the substantial benefits to health associated with disinfection by chlorination. The use of chlorine has virtually eliminated water-borne microbial diseases (e.g., typhoid) as a result of its ability to kill or inactivate essentially all enteric pathogenic micro-organisms, including viruses and bacteria from the human intestinal tract. Chlorine is the most convenient and easily controlled disinfectant; it is a strong oxidant for which a residual can be maintained in the distribution system to prevent bacterial recontamination.

Studies have shown that some trihalomethanes (e.g., chloroform) cause cancer in laboratory animals. Also, some epidemiological studies have shown evidence of higher cancer rates in people who drink chlorinated water. Based on these studies, chloroform is classified as a chemical that is probably carcinogenic to humans, and a guideline value needs to be established in the same range as that of other chemicals having a similar classification. A more detailed health risk assessment may be found in Appendix A.

2.5 Existing Standards

The concentration of trihalomethanes in any water supply depends on numerous factors, such as the nature of the watershed from which the water supply is drawn, the time of year, the effectiveness of the treatment process at removing organics, the point or points of chlorine application, the degree of chlorination, the temperature and pH of the water and the time between chlorination and water sampling or consumption.

Standards or guidelines may have to be based on an average of a number of samples over a period of time with sampling location specified. With this in mind, the following are four current values for trihalomethanes in drinking water set by different authorities.

The existing trihalomethanes guideline for Canada is 350 μg/L and is a one-time maximum value (e.g., not to be exceeded).

The United States Environmental Protection Agency has set a maximum contaminant level for trihalomethanes at 100 μg/L. This is an average value based on quarterly samples and is enforceable. This value is, however, currently under review.

The European Economic Community developed a set of drinking water standards in the form of a directive issued in 1980. Under this directive, haloform concentrations are to be as low as possible, but it is not enforceable. The World Health Organization set a guideline for chloroform of 30 μg/L, but with a note that disinfection efficiency must not be compromised when controlling chloroform content of the water.

Although the European guidelines for trihalomethanes are lower than those in North America, European water treatment practices do not rely entirely on chlorine for disinfection. Because of this, trihalomethane formation is not as significant a problem as in North America, and guideline values can be more stringent without affecting water treatment practices and costs.

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Trihalomethanes

Consultative Guideline

The proposed maximum acceptable concentration (MAC) for chloroform or total trihalomethanes in drinking water is 0.05 mg/L (50 µg/L), expressed as an annual average of quarterly samples.

1.0 Identity, Use and Sources in the Environment

Trihalomethanes (THMs) are halogen-substituted single-carbon compounds with the general formula CHX_3, where X may be fluorine, chlorine, bromine or iodine, or combinations thereof. These compounds are formed in drinking water primarily as a result of chlorination of organic matter present naturally in raw water supplies. The THMs most commonly present in drinking water are chloroform ($CHCl_3$), bromodichloromethane ($CHBrCl_2$), chlorodibromomethane ($CHClBr_2$) and bromoform ($CHBr_3$); consideration of information relevant to derivation of drinking water guidelines for THMs will be restricted to these compounds.

The four compounds considered here are liquids at room temperature. They are relatively to extremely volatile, with vapour pressures at 25°C ranging from 0.80 kPa for bromoform to 23.33 kPa for chloroform. The THMs are only slightly soluble in water, with solubilities less than 1 mg/mL at 25°C. Their log octanol-water partition coefficients range from 1.97 (chloroform) to 2.38 (bromoform). All of these compounds will decompose upon exposure to air and/or light.

Chloroform, the THM detected most frequently and at highest concentrations in drinking water, is the only THM produced commercially in Canada; however, data on volumes of production and consumption are not available. In 1987, 2712 tonnes were imported into Canada for use in the manufacture of other chemicals and as a solvent and degreasing agent.[1] Bromodichloromethane is used in the synthesis of other chemicals and as a solvent, whereas chlorodibromomethane is an intermediate in the manufacture of refrigerants, pesticides, propellents and other organic chemicals.[2] Bromoform is used in the synthesis of pharmaceuticals, as a solvent and in the aircraft and shipbuilding industries. Trihalomethanes are released into the environment from industrial sources as well as through indirect production in the chlorination of drinking water, municipal sewage and cooling water.

2.0 Exposure

Trihalomethanes result primarily from reactions between chlorine or bromine and naturally occurring organic compounds via the "classical" haloform reaction mechanism.[3] The rate and degree of THM formation increase as a function of the chlorine and humic acid concentration, temperature, pH and bromide ion concentration.[3,4] In the presence of bromides, brominated THMs are formed preferentially and chloroform concentrations decrease proportionally.[5] The point of chlorination may also have a significant effect on THM production. There are seasonal variations in THM concentrations in drinking water, with levels being considerably lower in the winter months.[6-9]

In a national survey of the water supplies of 70 communities serving about 38% of the population in Canada, conducted in the winter of 1976/77, concentrations of chloroform in treated water of the distribution system 0.8 km from the treatment plant, determined by the gas sparge technique, averaged 22.7 μg/L and ranged from 0 to 121 μg/L. Levels of the other THMs were considerably lower, averaging 2.9 μg/L for bromodichloromethane (range: 0 to 33 μg/L), 0.4 μg/L for chlorodibromomethane (range: 0 to 6.2 μg/L) and 0.1 μg/L for bromoform (range: 0 to 1.0 μg/L). Using the direct aqueous injection technique, average concentrations of most of the THMs were higher: 30.8 μg/L for chloroform (range: 0 to 150 μg/L), 3.1 μg/L for bromodichloromethane (range: 0 to 44 μg/L) and 0.4 μg/L for chlorodibromomethane (range: 0 to 7 μg/L).[9] In Ottawa, where sampling was conducted on several occasions over a one-year period, chloroform concentrations, determined by both gas sparge and direct aqueous injection methods, were approximately two to 10 times higher in the summer than in the winter.[9]

Concentrations of THMs have been determined in drinking water supplies at a considerable number of locations across Canada. However, the methods of sampling and analysis vary considerably and are often not well described in reports submitted by the provinces.

On the basis of data obtained from the provinces and from the 1976/77 national survey, it can be concluded that annual mean chloroform levels in Canadian drinking water supplies determined by the most suitable method of analysis (i.e., purge and trap) are generally less than 50 μg/L, with single values ranging up to several hundred micrograms per litre. Annual mean concentrations of both bromodichloromethane and chlorodibromomethane are generally less than 10 μg/L, with individual values ranging up to or somewhat greater than 50 μg/L. Mean concentrations of bromoform are generally less than the detection limit, or approximately 0.1 μg/L; individual values are less than 10 μg/L. Annual mean total THM concentrations are

generally less than 60 μg/L, with single values ranging up to several hundred micrograms per litre.

Information on the levels of THMs in Canadian foodstuffs was not found, although some relevant data are available from other countries. Total THM concentrations in six different cola and non-cola beverages (five samples of each) in New Jersey ranged from 3.2 to 44.8 μg/L.[10] Concentrations of chloroform and bromodichloromethane in unspecified beverage composites from the United States averaged 32 and 1.0 μg/L, respectively.[11] Chloroform concentrations are approximately 10 times higher in cola soft drinks than in non-cola soft drinks, even for similar water sources.[10-12] This may be due to the method of extraction of the cola and/or the presence of caramel in these soft drinks.

Maximum concentrations of 2200 μg/kg chloroform and 3 μg/kg bromodichloromethane were detected in the fat of nine species of fish from six areas of the Norwegian coastline that were contaminated principally by discharges from pulp and paper plants, but also by agricultural runoff, chemical plants and other industries. Bromoform and chlorodibromomethane were detected in only one sample at concentrations of 115 and 9 μg/kg, respectively.[13] Neither chloroform nor bromodichloromethane was detected in composite samples of meat/fish/poultry (quantitation limits were 18 and 4.5 ng/g, respectively) or oil/fat composite samples (quantitation limits were 28 and 8.3 ng/g, respectively) of 39 different foods in the United States.[12] In the composite of dairy foods, concentrations of chloroform and bromodichloromethane were 17 and 1.2 μg/L, respectively.

Concentrations of chloroform in the air of both Montreal and Toronto, sampled for a one-year period beginning in 1984, averaged 0.3 $\mu g/m^3$. Maximum concentrations detected were 0.4 $\mu g/m^3$ (49 samples) and 0.8 $\mu g/m^3$ (105 samples) in Montreal and Toronto, respectively. Local sources did not appear to appreciably influence airborne levels. Bromoform was also detected in the air in Toronto at a maximum concentration of 0.1 $\mu g/m^3$ (54 samples).[14]

Most of the limited data available indicate that the principal source of intake of THMs is drinking water and/or beverages produced with treated water. Because of its volatility, there is also potential for exposure in the home to airborne chloroform released from tap water. One recent study suggested that showering for 10 minutes could result in chloroform exposures (dermal and inhalation) greater than those observed through ingestion.[15] Although the presence of chloroform in dentifrices, liniments and antitussives contributed to the exposure of Canadians in the past, the use of chloroform in these products has now been banned under the Hazardous Products Act.

3.0 Analytical Methods and Treatment Technology*

As mentioned previously, THMs are formed in drinking water primarily as a result of chlorination of organic matter present in raw water supplies. It is therefore important, in assessing the risks associated with the ingestion of THMs in drinking water, to recognize the substantial benefits to health associated with disinfection by chlorination. The use of chlorine has virtually eliminated water-borne microbial diseases, because of its ability to kill or inactivate essentially all enteric pathogenic micro-organisms, including viruses and bacteria from the human intestinal tract. Chlorine is the most convenient and easily controlled disinfectant; it is a strong oxidant for which a residual can be maintained in the distribution system to prevent bacterial recontamination.[16,17]

Total THM-forming potential in drinking water samples can be determined by direct aqueous injection gas chromatography with appropriate detection. However, the preferred method is determination of individual THMs in preserved samples by purge and trap followed by gas chromatographic-mass spectrometric analysis. Concentrations determined by purge and trap are often significantly lower than those measured by direct aqueous injection as total potential THMs. The practical quantitation limit (PQL) (based on the capability of laboratories to measure THMs within reasonable limits of precision and accuracy) by the purge and trap method is 1 to 2 μg/L.[18]

Conventional control technologies for reduction of THM concentrations include optimization of precursor removal using conventional treatment, such as coagulation and sedimentation, use of alternative water supplies, modification of prechlorination practices and relocation of the points of chlorination. Based on the experience with these control technologies in Canada, mean annual concentrations of total THMs produced in the treatment of highly coloured waters can be reduced to less than 100 μg/L.

Total THM formation can also be reduced significantly by the use of alternative disinfectants, such as chloramines and ozone. Chloramines are a much weaker disinfectant than chlorine and are not recommended as a primary disinfectant, especially where virus or parasite cyst contamination may be present.[19] Moreover, although chloramines do not form significant levels of THMs, they are capable of producing halogen substitution to form organic compounds and thus may produce significant quantities of total organic halogen. Little is known about these oxidant residuals. The nature and

*For more detailed information on treatment technology, the reader is referred to Appendix B.

toxicity of products formed from the organic base precursor fractions, particularly the organic chloramine portion of the chlorine residual, have also not been characterized.

Ozone has also been used as a primary disinfectant in water treatment plants in some parts of Canada and Europe. Although it is an excellent disinfectant (even though it must be used in combination with a secondary disinfectant to maintain a residual in the distribution system) and does not form chlorinated by-products, the nature and toxicity of by-products of ozonation of natural organics have not been well studied. The U.S. National Academy of Sciences has concluded, however, that "the admittedly inadequate studies now available point to lower toxicities of ozonated water than chlorinated water."[19]

The optimum approach at present for reduction of THMs in drinking water is considered to be improvement of specific conventional water treatment processes to remove organic compounds prior to disinfection and addition of special processes such as carbon adsorption and preoxida-tion.[19] Initial removal of organic precursors precludes the need for reducing contact time, thus improving the efficiency of the disinfection process while still minimizing formation of chlorinated organic by-products. THM formation potential can be reduced by granular activated carbon filtration, with reduction a function of the type and adsorbability of the organic matter in the water and also the process design criteria (e.g., empty bed contact time and filter depth).[20]

4.0 Health Effects

4.1 Kinetics and Metabolism

The THMs are rapidly and efficiently absorbed following ingestion, metabolized primarily to carbon dioxide and/or carbon monoxide and rapidly exhaled. Because of their lipophilicity, THMs accumulate most in tissues with the highest lipid content, i.e., adipose tissue > brain > kidney > blood.

The haloforms are metabolized *in vitro* and *in vivo* through the formation of dihalocarbonyl compounds to carbon monoxide and carbon dioxide via the cytochrome P-450 dependent hepatic mixed-function oxidase system. Chloroform, bromodichloromethane, chlorodibromomethane and bromoform are metabolized to dichlorocarbonyl (phosgene), predominantly dichlorocarbonyl, predominantly chlorobromocarbonyl and dibromocarbonyl intermediates, respectively.[21-24] As halogens of higher molecular weight are better leaving groups, the rate of metabolism of these compounds to carbon monoxide both *in vivo* and *in vitro* generally follows the halide order, namely, bromoform >> chlorodibromomethane > bromodichloromethane ≈ chloroform; the carcinogenic potential of these compounds in the liver and kidney of experimental animals is reversed.[25] This may be due

to variations in the cellular lifetime of active intermediates.[25]

Available data indicate that there are distinct differences in the extent of metabolism of the THMs between rats and mice.[26] Within eight hours following intragastric administration of 150 mg/kg bw (rats) or 100 mg/kg bw (mice) in corn oil, 4% to 18% and 40% to 81% of total radiolabelled THMs were eliminated as carbon dioxide through the lungs in expired air in rats and mice, respectively. In the same experiment, 41% to 67% and 5% to 26% of the parent compound were eliminated unchanged in rats and mice, respectively. Less than 10% of the total radiolabel for each of the chemicals was detected in the urine of both species 36 to 48 hours post-exposure; the proportion excreted in the urine for both species was greatest for chloroform, followed, in descending order, by bromoform, bromodichloromethane and chlorodibromomethane. The authors considered the metabolism of these compounds in the mouse to be four- to ninefold greater than that in the rat; however, it should be noted that the administered doses were high and that metabolism in both species is more complete following administration of lower, more relevant doses.

Limited available data indicate that the metabolism of chloroform in man is similar to that in rodents. Based on comparison of the formation of reactive metabolites as measured by binding of radioactivity from $[^{14}C]CHCl_3$ (0 to 10 mmoles) in rat and human liver microsomes, it was concluded that the metabolism in these species is similar, although less efficient in humans.[27] In eight human volunteers ingesting gelatin capsules containing chloroform (500 mg in olive oil), a maximum of 68.3% and 50.6% of the dose was found in the expired air as chloroform and carbon dioxide, respectively, eight hours post-administration.[19,28] There was an inverse relationship between the adipose tissue content of the body and pulmonary elimination of chloroform.[28]

Available data indicate that the vehicle of administration affects the bioavailability of chloroform. Based on the calculated area under blood concentration-time curves (five hours), uptake of chloroform following administration of 75 mg/kg bw by intragastric intubation in aqueous solution was 8.7 times greater than that for a similar dose administered in corn oil in paired Wistar rats.[29] The mean times to peak concentrations in blood for the two vehicles of administration were similar: 39.3 μg/L for water and 5.9 μg/L for oil.

4.2 Effects in Humans

In several ecological (correlational) epidemiological studies, associations between the ingestion of chlorinated surface water or groundwater and various cancers have been reported, including cancers of the oesophagus, pancreas, urinary tract (white females), stomach and rectum

(males).[30-32] In general, there was no association between cancers at various sites and THM concentrations in drinking water, with the exception of a significant increase in pancreatic cancer in white males,[30] cancer of the rectum in males only[32] and stomach cancer in both sexes.[32] The association between stomach cancer and rectal cancer and recent THM concentrations observed by Tuthill and Moore, however, was not apparent when population migration patterns were taken into consideration.[32] Moreover, the results of a correlational study conducted in Iowa indicate that water quality parameters other than THMs may be associated with cancer. Independent of chlorination status, there were significant associations between nickel and 1,2-dichloroethane in non-chlorinated groundwater and cancer of the bladder and lung, and colon and rectum, respectively. These parameters in themselves may not be causal agents but rather indicators of contamination from external sources.[33]

Therefore, although there is evidence of associations between exposure to THMs in drinking water and cancers at several sites in correlational studies conducted to date, the associations have not been observed consistently. Moreover, the lack of data on exposure of the individuals in populations in ecological studies and the resulting inability to adjust rigorously for confounding factors and population mobility limit their usefulness in assessing cause-effect relationships.

In inherently more sensitive case-control studies that were based on mortality and did not involve interviews, there have been associations between ingestion of chlorinated drinking water and cancer at several specific sites, including the colon,[34-36] rectum,[34,37-39] breast,[37-39] lung in white males,[37,38] brain[35-39] and bladder.[40] However, in general, these associations were not observed consistently in various subgroups of the population by degree of urbanization,[34] and there was no exposure-response relationship.[35-36] Moreover, in several of these studies, population mobility was not assessed,[34-36] and several of the cancers, particularly those of the breast, lung and brain, may have been attributable to confounding factors, including family size (breast) and occupational exposure (brain, lung).[35-39] In a small (395 cases) but well-conducted investigation in which water source and treatment were recorded for cases and controls for a 20-year period at home or work prior to death and cumulative chloroform exposure was modelled from previous THM surveys, there was no association between colon or rectal cancers and cumulative exposure after control by logistic regression for average source type, population density, marital status, age or year of death.[41]

In the epidemiological studies with the inherently most sensitive protocols conducted to date (i.e., case-control based on personal interviews), there have been associations between ingestion of chlorinated drinking water and the incidences of colon cancer for

inherently most sensitive studies conducted to date, however, bladder cancer was associated with both level of intake and duration of exposure.

(3) **Strength:** The association between colon and bladder cancer and ingestion of chlorinated drinking water is weak to moderate.[47,48]

(4) **Specificity:** Data from the largest and inherently most sensitive studies conducted to date indicate that the most plausible associations between ingestion of chlorinated drinking water and cancer are restricted to those of the bladder and colon.

(5) **Temporal relationship:** In the inherently most sensitive and largest studies, observed associations have been limited to those greater than 60 years of age[42] or to those with greatest duration of exposure.[43]

(6) **Plausibility and supporting experimental data:** The observation of an association between ingestion of chlorinated drinking water and increased cancer risk is plausible, based on the positive results for several of the THMs observed in carcinogenicity bioassays in animal species.

Available data are, therefore, at least consistent with the hypothesis that ingestion of chlorinated drinking water, if not THMs specifically, may be causally related to cancers of the bladder and colon.

4.3 Toxicological Studies

4.3.1 Acute Toxicity

In acutely toxic doses, chloroform causes central nervous system depression and cardiac effects. In rats, the clinical signs of acute toxicity for all of the THMs are similar and include piloerection, sedation, flaccid muscle tone, ataxia and prostration.[49] LD_{50}s for chloroform, bromodichloromethane, chlorodibromomethane and bromoform were 908, 916, 1186 and 1388 mg/kg bw, respectively, in male rats and 1117, 969, 848 and 1147 mg/kg bw, respectively, in female rats. In surviving animals, there were a variety of effects, including reduced food intake, growth retardation, increased liver and kidney weights, haematological and biochemical effects and histological changes in the liver and kidney.[49]

4.3.2 Subchronic Toxicity

In contrast to the results reported above following administration of acutely toxic doses, the liver and thyroid rather than the liver and kidney were the organs most affected following administration of the THMs in a subchronic study by the same investigators.[50,51] Groups of 20

male and female rats (strain unspecified) ingested drinking water containing 5, 50, 500 or 2500 mg/L chloroform, bromodichloromethane, chlorodibromomethane or bromoform for 90 days; estimated doses were 0.11 to 0.17, 1.2 to 1.6, 8.9 to 14 and 29 to 55 mg/d per rat, respectively.[50,51] Ten animals in each group were killed at the end of exposure, and the remaining animals were sacrificed 90 days later.

The growth rate was suppressed in animals administered 2500 mg/L chloroform and bromodichloromethane at the end of exposure but not following the 90-day recovery period. Food consumption was also depressed during both exposure and recovery periods in groups receiving 2500 mg/L chloroform, chlorodibromomethane and bromodichloromethane. Food consumption in males was depressed during exposure to 2500 mg/L bromoform but was normal at the end of the recovery period. Lymphocyte counts were decreased at the end of the recovery period in groups receiving 500 mg/L chloroform, 2500 mg/L chlorodibromomethane and 2500 mg/L bromoform. Mild, reversible histological changes in the liver and thyroid of exposed groups were reported, with the hepatotoxicity being greatest for bromoform, followed by, in descending order, dichlorobromomethane, dibromochloromethane and chloroform; however, the incidence of the lesions was not dose-related, although the frequency of more severe changes was greater in higher dose groups (statistical significance not reported). As the histological effects were mild and reversible and the haematological effects observed in chloroform-exposed animals were not dose-related, the no-observed-adverse-effect level (NOAEL) for all of the THMs in this study is considered to be 500 mg/L; the lowest-observed-adverse-effect level (LOAEL) is considered to be 2500 mg/L.

In a 90-day study in which CD-1 male and female mice (seven to 12 animals of each sex per treatment group) received 50, 125 or 250 mg/kg bw per day chloroform by intubation in emulphor deionized water, there was a dose-related increase in liver weights and a decrease in hepatic microsomal activities in high-dose males and in females at all dose levels.[52] Hexobarbital sleeping times were also increased in mid- and high-dose females. Blood glucose was increased in the high-dose groups of both sexes, and humoral immunity decreased in high-dose males and mid- and high-dose females. Cellular immunity was decreased in high-dose females. The authors also reported slight histopathological changes in the kidney and liver of both sexes but did not provide information on the prevalence, severity or dose-response relationship. The LOAEL for female mice in this study is considered to be 50 mg/kg bw; for males, the LOAEL is 250 mg/kg bw and the NOAEL 125 mg/kg bw. The absence in this investigation of an increase in serum glutamic-pyruvic transaminase and serum glutamic-oxaloacetic transaminase observed in the high-dose groups in a 14-day study

with a similar dosing regime by the same investigators led the authors to conclude that some tolerance to the hepatotoxic action of chloroform may develop following long-term exposure.

The importance of the vehicle of administration in the toxicity of chloroform was demonstrated in a study in which groups of 80 male and female B6C3F$_1$ mice were exposed to 60, 130 or 270 mg/kg bw per day by gavage in corn oil or a 2% emulphor suspension for 90 days. Chloroform caused more marked hepatotoxic effects when administered in corn oil than in aqueous suspension as determined by body and organ weights, serum chemistry and histopathological examination.[53]

4.3.3 Chronic Toxicity and Carcinogenicity

Chloroform has been carcinogenic in two animal species in the most extensive bioassays conducted to date. In an early study conducted by the National Cancer Institute (NCI), chloroform was administered by gavage in corn oil to groups of 50 male and 50 female Osborne-Mendel rats and B6C3F$_1$ mice. Male rats received 0, 90 or 180 mg/kg bw five times per week for 78 weeks; female rats received 0, 125 or 250 mg/kg bw five times per week for the first 22 weeks and the same doses as the males thereafter. In the first 18 weeks, doses of 0, 100 or 200 mg/kg bw were administered to male mice, and 0, 200 or 400 mg/kg bw were administered to female mice. After 18 weeks, the doses were changed to 0, 150 and 300 mg/kg bw for male mice and 0, 250 and 500 mg/kg bw for female mice for the remainder of the exposure period.[54]

In male rats, there was a statistically significant dose-related increase in the incidence of carcinomas of the kidney (0/99, 4/50, 12/50 for control, low and high doses, respectively). These tumours were not observed in female rats, although there was a non-significant increase in tumours of the thyroid (adenocarcinomas and carcinomas) in this sex.[54]

Highly significant increases in hepatocellular carcinomas were observed in both sexes of mice (males: 1/18, 18/50, 44/45; females: 0/20, 36/45, 39/41 for control, low and high doses, respectively). Nodular hyperplasia was also observed in low-dose males.[54] It should be noted, however, that the weight loss in exposed animals was greater than 10%.

Upon re-examination of tissue samples from the NCI carcinogenesis bioassay, Reuber also reported increases in the incidence of several types of benign and malignant tumours of the liver in female rats and malignant lymphomas in both sexes of mice.[55]

In a more recent and larger study, 0, 200, 400, 900 or 1800 mg/L chloroform was administered in drinking water (a more appropriate vehicle than that used in the NCI bioassay described above) to male Osborne-Mendel rats (50 to 330

animals per group) and female $B6C3F_1$ mice (50 to 430 animals per group) for 104 weeks; the time-weighted average doses on a body weight basis ranged from 19 to 160 mg/kg per day for the rats and from 34 to 263 mg/kg per day for the mice.[56] To increase the sensitivity for detecting low response rates, group sizes were larger for the lower doses; there were two control groups (n=330 and n=50), one of which (n=50) was matched for water intake with the high-dose groups.

In rats, there were dose-related decreases in water consumption and body weight gain that persisted in the two highest dose groups; survival increased with dose, probably as a result of leaner body composition in the higher dose groups (e.g., after 104 weeks, only 12% of controls had survived, whereas 66% of the animals in the high-dose group were still alive; this is a common occurrence in such studies). Consistent with the results of the NCI bioassay described above, there was also a dose-related increase in the incidence of kidney tumours. The incidence of tubular cell adenomas and adenocarcinomas combined was slightly lower than that in the NCI bioassay: 1/50, 4/313, 4/148, 3/48 and 7/50 in the matched control and increasing dose groups, respectively. Although there were increases in other neoplastic lesions in rats, including neurofibromas, leukaemias, lymphomas and circulatory system tumours, they were not considered to be treatment-related because of a lack of a clear dose-response relationship or statistical significance or because they appeared to be attributable to the longer survival of the chloroform-treated animals.

With respect to the non-neoplastic histopathological changes in the kidney in this study, the authors commented only that "nontumour pathology of the kidney was high in all animals regardless of treatment." As a result, "it was not possible to relate tumour pathology with other tissue damage on either an individual animal or across-group basis." The incidence of nephropathy was 91% in the control group, 90% in the matched control and 95%, 95%, 100% and 92% in the increasing dose groups, respectively.

In mice, drinking water consumption was markedly depressed, leading to the death of about 25% of the two highest dose groups and 6% of the next highest dose group in the first week; after this initial period, survival did not differ significantly among groups. In contrast to the NCI bioassay described above, in which hepatic tumours in both sexes of mice were observed, there were no treatment-related increases in the incidence of any tumours in female mice in this study. Jorgenson *et al.* suggested that the hepatic tumours in mice in the NCI study may have been attributable to the interaction of chloroform with the corn oil vehicle.[56]

In three different studies in which four strains of mice (C57Bl, CBA, CF/1 and ICI) were exposed to chloroform in toothpaste (0, 17 or 60 mg/kg per day in ICI male and female mice) or in toothpaste or arachis oil

(0 or 60 mg/kg per day in males of all four strains), there were no treatment-related effects on the incidence of any type of tumour in males of three of the four strains (C57Bl, CBA and CF/1 mice). There was, however, an increase in the incidence of epithelial tumours of the kidney at 60 mg/kg per day in male mice, which was greater when chloroform was administered in arachis oil than in toothpaste.[57]

Several other studies on the potential carcinogenicity of chloroform have been conducted. In $B6C3F_1$ male mice (35 animals per group) ingesting chloroform in drinking water (0, 600 or 1800 mg/L) for periods up to 52 weeks, there were no increases in tumour incidence.[58] However, these results may have been a function of the short observation period and/or small group sizes. The potential of chloroform to promote tumours induced by known initiators was also investigated in this study. Mice of the same strain (35 animals per group) ingested drinking water containing 10 mg/L diethylnitrosamine (DENA) for four weeks followed by 600 or 1800 mg/L chloroform for up to 52 weeks. There were two control groups: after DENA treatment, the positive control group ingested drinking water containing phenobarbital (500 mg/L), while the vehicle control group received untreated drinking water. The induction of liver tumours was enhanced by exposure to phenobarbital but not by exposure to chloroform after DENA treatment. In contrast, in a study conducted by Deml and Oesterle, chloroform administered in corn oil (100, 200 and 400 mg/kg bw, twice weekly for 11 weeks, one week after administration of a single dose of 8 mg DENA) promoted the development of DENA-initiated pre-neoplastic foci liver tumours in Sprague-Dawley rats.[59]

In an inadequate bioassay, a significantly increased incidence of neoplastic nodules in female Wistar rats ingesting drinking water containing 2.9 g/L chloroform or 2.4 g/L bromodichloromethane (dose halved at 72 weeks) was reported.[60] However, the site of the nodules was not specified, the compounds were administered in city drinking water for which concentrations of other THMs had not been determined and documentation of the study protocol was inadequate.

In the only adequate carcinogenesis bioassay conducted to date for bromodichloromethane, groups of 50 male and 50 female F344/N rats and $B6C3F_1$ mice were administered the compound by gavage in corn oil, five days per week for 102 weeks. Rats received 0, 50 or 100 mg/kg bw per day; male mice received 0, 25 or 50 mg/kg bw per day, while female mice received 0, 75 or 150 mg/kg bw per day.[61]

In rats, there was some decrease in body weight gain in the high-dose groups of both sexes (statistical significance not specified), increased incidence of cytomegaly of the renal tubular epithelial cells in males (both doses), nephrosis in the high-dose group of females and hepatic changes, including necrosis, clear cell

change, eosinophilic cytoplasmic change, focal cellular change and fatty metamorphosis, in both sexes but predominantly in the high-dose group of females. There was clear evidence of carcinogenicity in male and female rats, with increases in the incidence of tubular cell adenomas and adenocarcinomas of the kidney (combined incidence in control, low- and high-dose groups of males, 0/50, 1/50 and 13/50; in females, 0/50, 1/50 and 15/50) and rare tumours (adenomatous polyps and adenocarcinomas) of the large intestine (combined incidence in males, 0/50, 13/50 and 45/50; in females, 0/46, 0/50 and 12/47). Increased incidence of skin neoplasms in low- but not high-dose male rats was also observed but was not considered to be compound-related. The neoplasms of the kidney in rats in this bioassay were not similar to those observed for other compounds, such as 1,4-dichlorobenzene, for which tumours occurred principally in males and were associated with severe nephropathy and increased incidence of calcification and hyaline droplet formation, associated with reabsorption of alpha-2-microglobulin.[62]

There was a decrease in body weight gain of female mice, and survival was significantly lower than that of controls, due partly to ovarian abscesses not considered to be treatment-related. The incidence of renal cytomegaly and hepatic fatty metamorphosis in male mice was also increased. Pathological changes in the thyroid gland and testis were also observed but were not considered to be treatment-related. There was also clear evidence of carcinogenicity in male and female $B6C3F_1$ mice, based on increased incidence of adenomas and adenocarcinomas (combined) of the kidney in males (incidence in control, low- and high-dose groups, 1/49, 2/50 and 9/50, respectively) and of hepatocellular adenomas and carcinomas (combined) in female mice (incidence in females, 3/50, 18/48 and 29/50).

In a recently conducted National Toxicology Program (NTP) carcinogenesis bioassay, 0, 40 or 80 mg/kg bw chlorodibromomethane was administered by gavage in corn oil five times per week for 104 weeks to groups of 50 male and female F344/N rats. In addition, 0, 50 or 100 mg/kg bw per day was administered in similar fashion to groups of 50 male and female $B6C3F_1$ mice five days per week for 105 weeks. Body weight gain in the high-dose group of male rats was decreased, and there was a dose-related increase in lesions (fatty metamorphosis and ground-glass cytoplasmic changes) of the liver in both sexes and nephrosis of the kidney (dose-related) in females. There was, however, no evidence of carcinogenicity in rats.[63]

In male mice, survival was significantly lower in both dose groups, and 35 animals in the low-dose group were accidentally killed during weeks 58 to 59. In both sexes, the incidences of hepatic lesions were increased, including fatty metamorphosis (both sexes), hepatocellular necrosis (dosed

males), hepatocytomegaly (high-dose males) and calcification of the liver (high-dose females). Nephrosis (high dose) and renal calcification in males and follicular cell hyperplasia of the thyroid gland (possibly related to a bacterial infection) in females were also increased. There was equivocal evidence of carcinogenicity in male $B6C3F_1$ mice based on an increased incidence of hepatocellular carcinomas but only a marginal increase in hepatocellular adenomas or carcinomas (combined) (incidence of hepatocellular carcinomas in control and high-dose groups, 10/50 and 19/50, respectively; incidence of hepatocellular adenomas and carcinomas combined, 23/50 and 27/50, respectively). The number of surviving animals in the low-dose group of male mice, however, was inadequate for analysis of tumour incidence, owing to a dosing error. There was also some evidence of carcinogenicity in female mice, based on an increased incidence of hepatocellular adenomas and hepatocellular adenomas or carcinomas (combined). The incidence of hepatic adenomas and carcinomas (combined) in the control, low- and high-dose groups was 6/50, 10/49 and 19/50, respectively.

In a recently completed NTP carcinogenesis bioassay, 0, 100 or 200 mg/kg bw bromoform was administered by gavage in corn oil five days per week for 103 weeks to groups of 50 F344/N rats of each sex and to female $B6C3F_1$ mice.[25] Male $B6C3F_1$ mice were administered 0, 50 or 100 mg/kg bw on the same schedule. In rats, there was a reduction of body weight gain in low- and high-dose males and high-dose females; survival in the high-dose group of males was also significantly lower than that in controls. As well, dose-related, non-neoplastic effects in the salivary gland (squamous metaplasia and chronic active inflammation in both sexes), prostate (squamous metaplasia), forestomach (ulcers in the males), lung (chronic active inflammation--males only) and spleen (pigmentation--high-dose females) were also observed, although the lesions of the salivary gland and lung were characteristic of infection by rat corona virus, to which a positive serological reaction was observed early in the study. There was some evidence of carcinogenicity in male rats and clear evidence in female rats, based on increased incidences of uncommon neoplasms (adenomatous polyps and adenocarcinomas of the large intestine) in both sexes. The incidences of these tumours (combined) in the control, low- and high-dose groups of females were 0/50, 1/50 and 8/50, respectively; in males, the comparable values were 0/50, 0/50 and 3/50. Although the incidence of these tumours in females was similar to that observed in the NTP bioassay for bromodichloromethane, the incidence in males was much less. Reduced survival in the high-dose group of male rats administered bromoform may, however, have lowered the sensitivity of the bioassay for detecting a carcinogenic response. The incidence

of neoplastic nodules in low-dose female rats was also greater than that in controls, but it was not considered to be a chemically induced neoplastic effect, as the lesions did not fit the current NTP criteria for hepatocellular adenomas, nor was the incidence significantly increased in high-dose female rats or in dosed male rats.

In female mice, there was a decrease in body weight gain and survival (partially attributable to utero-ovarian infection) and increases in the incidence of follicular cell hyperplasia of the thyroid (high dose) and fatty change of the liver (both doses). There was no evidence of carcinogenicity in male or female mice.[25]

4.3.4 Mutagenicity

In a recent extensive review, it has been concluded that although the results of most assays for genotoxicity of chloroform have been negative, the data are inconclusive because of inadequacies in the experimental protocols, particularly with respect to the use of exogenous activation systems.[64] However, the author further concluded that chloroform may indeed be mutagenic, based on the results of studies in which binding to macro-molecules, DNA damage and mitotic arrest have been examined. More recently, Varma *et al.* reported that chloroform was mutagenic in *Salmonella typhimurium* without metabolic activation, although a mixture of chloroform (85%) and bromoform (15%) was not mutagenic in the same assay with or without metabolic activation.[65]

Results concerning the mutagenicity of bromodichloromethane, chlorodibromomethane and bromoform in *S. typhimurium*, with or without metabolic activation, have been inconsistent.[25] However, all four THMs have induced sister chromatid exchanges in human lymphocytes *in vitro* (bromoform > chlorodibromo-methane > bromodichloromethane > chloroform) and in mouse bone marrow cells *in vivo*.[66]

4.3.5 Teratogenicity and Reproductive Effects

Available data on the teratogenicity of the THMs are confined principally to chloroform. In studies conducted to date, chloroform has not been teratogenic in rats, rabbits or mice at doses up to 400 mg/kg bw following administration by gavage in corn oil or emulphor:saline.[67-69] Foetotoxic effects (e.g., decreased body weights and sternebral and interparietal malformations) were sometimes observed, but only at doses that were toxic to the mothers.

The teratogenicity of other THMs considered here was investigated in one study in which 50, 100 or 200 mg/kg bw bromodichloro-methane, chlorodibromomethane or bromoform and 100, 200 or 400 mg/kg bw chloroform was administered to groups of 15 pregnant Sprague-Dawley rats by oral intubation in corn oil on days 6 to 15 of gestation.[68] Maternal

weight gain was depressed in the high-dose groups (200 mg/kg) receiving bromodichloromethane and chlorodibromomethane, but to a lesser extent than that in the high-dose group for chloroform (400 mg/kg). Maternal liver weight was also increased at the highest dose of bromodichloromethane (200 mg/kg). Bromodichloromethane and bromoform were considered to be foetotoxic, based on the observation of interparietal anomalies, although the statistical significance of the observed increases was not reported. (These compounds also appeared to increase the incidence of aberrations of the sternebrae.)

5.0 Classification and Assessment

As chloroform is the THM present in greatest concentration in drinking water, and as any guideline developed for this compound will be protective with respect to the other THMs, the proposed maximum acceptable concentration (MAC) is based on this compound and is also suitable for total THMs.

Although not complete, available epidemiological data are at least consistent with the hypothesis that ingestion of chlorinated drinking water, if not THMs specifically, may be causally related to cancers of the bladder and colon (chloroform and other THMs account for up to 50% by weight of the total chlorination by-products in drinking water).[70]

Chloroform has been classified in Group II--probably carcinogenic to man (inadequate evidence in man but sufficient data in animals). Cancer risks have been estimated on the basis of the results of the study by Jorgenson *et al.*, in which chloroform was administered in the most appropriate vehicle (i.e., drinking water) to large groups of male rats and female mice.[56] The nature of the vehicle appears to be an important factor in the toxicity and carcinogenicity of chloroform. More marked hepatotoxic effects and increased incidence of liver tumours in rats and mice are observed following administration of chloroform in corn oil compared with drinking water, probably as a result of the major shift in the nature of the caloric intake associated with the former vehicle.

A surface area correction was not incorporated, on the basis that chloroform is an indirect-acting carcinogen and that the rate of metabolism appears to be similar in both rodents and man. Using the robust linear extrapolation model for the increase in renal tubular cell adenomas or adenocarcinomas (combined) observed in male Osborne-Mendel rats in the Jorgenson *et al.* study, the calculated unit lifetime risk for ingestion of 1 μg/L chloroform in drinking water is 3.64×10^{-8}*. The

*Average adult body weight = 70 kg; average daily intake of drinking water = 1.5 L.

calculated unit lifetime risk for a second analysis in which incidence in the high-dose group was compared with that in a control group matched for water consumption was similar (3.50×10^{-8}*) (because of the low water consumption of the high-dose group, it was necessary to have a second control group that was matched for water consumption). The concentrations in drinking water corresponding to lifetime cancer risks of 10^{-5}, 10^{-6} and 10^{-7} for these tumours based on the model described above are as follows*:

Lifetime risk	**Concentrations in drinking water (μg/L)**
10^{-5}	270
10^{-6}	27
10^{-7}	2.7

Owing to limitations of the Jorgenson *et al.* study[56] (i.e., that only one sex of each species was examined and that the mortality rate in several of the groups of mice was high), cancer risks have also been estimated on the basis of the increase in renal tumours in male rats observed in the NCI bioassay in which chloroform was administered by gavage in corn oil to Osborne-Mendel rats and $B6C3F_1$ mice.[54] An increase in malignant tumours of the liver in female rats and in male and female mice was also observed in this study; however, because these increases have not been confirmed in bioassays in which chloroform has been administered in a more appropriate vehicle (i.e., drinking water), they have not been used as a basis for quantitative estimation of lifetime cancer risks. Using the robust linear extrapolation model for the increase in renal tumours observed in male rats in the NCI study, the calculated unit lifetime risk for ingestion of 1 μg/L chloroform in drinking water is within the same order of magnitude as that estimated on the basis of the Jorgenson *et al.* study.[56]

It is not possible to assess, on the basis of available data, whether the kidney tumours in male rats observed in the NCI[54] and Jorgenson *et al.*[56] bioassays may be species- and sex-specific responses associated with the reabsorption of alpha-2-microglobulin; additional data on this aspect are desirable. It is of interest in this regard that the neoplasms of the kidney observed in the NTP bioassay for bromodichloromethane[61] were not similar to those observed for other hydrocarbons for which tumours occurred principally in males and were associated with severe nephropathy and increased incidence of calcification and hyaline droplet formation.

Bromodichloromethane is the only other THM considered here that

*Average adult body weight = 70 kg; average daily intake of drinking water = 1.5 L.

has been classified in Group II--probably carcinogenic to man (sufficient evidence in animals; inadequate data in man). To ensure that a recommended maximum concentration developed on the basis of data for chloroform is sufficiently protective with respect to this compound, cancer risks have been estimated on the basis of the results of the only adequate carcinogenesis bioassay in F344/N rats, which was conducted by the NTP.[61] It should be noted, however, that the compound was administered by gavage in corn oil in this bioassay and that quantitative risks may be overestimated. Although there has been one carcinogenesis bioassay in which bromodichloromethane was administered in a more appropriate vehicle (i.e., drinking water),[60] it was considered inadequate for quantitative risk estimation, based on the limitations mentioned in the Health Effects section. Moreover, the increases in adenomas and adenocarcinomas in the kidney of male mice and in hepatocellular adenomas and carcinomas in female mice in the NTP bioassay have not been used for quantitative estimation of the cancer risks, as these increases were confined to one sex, and owing to the possible contribution of the corn oil vehicle to the induction of liver tumours in mice. Using the robust linear extrapolation model for the tumours that were significantly increased in F344/N rats in the NTP bioassay (intestinal adenomatous polyps and adenocarcinomas; renal tubular cell adenomas and adenocarcinomas), the calculated unit lifetime risks for ingestion of 1 μg/L bromodichloromethane in drinking water range from 3.36×10^{-8}* (based on intestinal adenomatous polyps or adenocarcinomas [combined] in female rats) to 17.76×10^{-8} (based on intestinal adenomatous polyps or adenocarcinomas [combined] in male rats). The estimated ranges of concentrations corresponding to lifetime cancer risks of 10^{-5}, 10^{-6} and 10^{-7} for these tumour types based on the model described above are as follows*:

Lifetime risk	**Concentrations in drinking water (μg/L)**
10^{-5}	56.5 - 299
10^{-6}	5.65 - 29.9
10^{-7}	0.565 - 2.99

Chlorodibromomethane has been classified in Group IIIB--possibly carcinogenic to man on the basis of limited evidence of carcinogenicity in animals (one species; equivocal evidence in one sex and limited evidence in the other) and inadequate data in man. Similarly, bromoform has been classified in Group IIIB--possibly

*Average adult body weight = 70 kg; average daily intake of drinking water = 1.5 L.

carcinogenic to man on the basis of limited evidence in animals (one species; some evidence in one sex and clear evidence in the other sex) and inadequate data in man.

6.0 Rationale

Because THMs are formed in drinking water primarily as a result of chlorination of organic matter present in raw water supplies, it is important to recognize the substantial benefits to health associated with disinfection by chlorination. The use of chlorine has virtually eliminated water-borne microbial diseases, because of its ability to kill or inactivate essentially all enteric pathogenic micro-organisms, including viruses and bacteria from the human intestinal tract. Chlorine is the most convenient and easily controlled disinfectant; it is a strong oxidant for which a residual can be maintained in the distribution system to prevent bacterial recontamination.[16,17]

Because chloroform is classified in Group II (probably carcinogenic to man), the proposed MAC is derived based on consideration of the best available treatment technology and estimated lifetime cancer risks. Because the MAC must also be measurable by available analytical methods, the PQL is also taken into consideration in derivation of the MAC.

A proposed MAC of 0.05 mg/L (50 μg/L) for chloroform or total THMs was established, therefore, on the basis of the following considerations:

(1) The estimated maximum unit lifetime cancer risk associated with the ingestion of 1 μg/L chloroform in drinking water is 3.64×10^{-8} (based on renal tubular cell adenomas or adenocarcinomas [combined] in rats administered chloroform in drinking water). Therefore, the estimated lifetime risk associated with the ingestion of 50 μg/L chloroform (i.e., 1.82×10^{-6}) is within a range that is considered to be "essentially negligible."

(2) The PQL (based on the ability of laboratories to measure chloroform within reasonable limits of precision and accuracy) is 1 to 2 μg/L.

(3) By optimum methods (i.e., improvement of specific conventional water treatment processes to remove organic compounds prior to disinfection and addition of such processes as carbon adsorption and preoxidation), total THM concentrations can be reduced to below 50 μg/L.

The proposed MAC is expressed as an annual average of quarterly samples. The frequency of sampling may be reduced where historical data indicate that maximum values will not exceed the MAC. Samples should be representative of exposure; protocols should, therefore, specify inclusion of samples from the extremities of the distribution system.

7.0 References

1. Unpublished data. Data disk "Canadian International Trade Classification" for 1986-1987. Import/ Export Volumes, Statistics Canada (1987).

2. Keith, L.H. and Walters, D.B. (eds.). Compendium of safety data sheets for research and industrial chemicals. VCH Publishers (1985).

3. Stevens, A.A., Slocum, C.J., Seeger, D.R. and Robeck, G.G. Chlorination of organics in drinking water. J. Am. Water Works Assoc., 68: 615 (1976).

4. Amy, G.L., Chadik, P.A. and Chowdhury, Z.K. Developing models for predicting trihalomethane formation potential and kinetics. J. Am. Water Works Assoc., 79: 89 (1987).

5. Aizawa, T., Magara, Y. and Musashi, M. Effect of bromide ions on trihalomethane (THM) formation in water. Aqua, 3: 165 (1989).

6. Otson, R. Purgeable organics in Great Lakes raw and treated water. Int. J. Environ. Anal. Chem., 31: 41 (1987).

7. Otson, R., Williams, D.T., Bothwell, P.D. and Quon, T.K. Comparison of trihalomethane levels and other water quality parameters for three treatment plants on the Ottawa River. Environ. Sci. Technol., 15(9): 1075 (1981).

8. Otson, R., Williams, D.T. and Bothwell, P.D. Volatile organic compounds in water at thirty Canadian potable water treatment facilities. J. Assoc. Off. Anal. Chem., 65(4): 1370 (1982).

9. Williams, D.T., Otson, R., Bothwell, P.D., Murphy, K.L. and Robertson, J.L. Trihalomethane levels in Canadian drinking water. Environ. Sci. Res., 16: 503 (1980).

10. Abdel-Rahman, M.S. The presence of trihalomethanes in soft drinks. J. Appl. Toxicol., 2(3): 165 (1982).

11. Wallace, L.A., Pellizzari, E., Hartwell, T., Rosenzweig, M., Erickson, M., Sparacino, C. and Zelon, H. Personal exposure to volatile organic compounds. I. Direct measurements in breathing-zone air, drinking water, food, and exhaled breath. Environ. Res., 35: 319 (1984).

12. Entz, R.C., Thomas, K.W. and Diachenko, G.W. Residues of volatile halocarbons in foods using headspace gas chromatography. J. Agric. Food Chem., 30: 846 (1982).

13. Ofstad, E.B., Drangsholt, H. and Carlberg, G.E. Analysis of volatile halogenated organic compounds in fish. Sci. Total Environ., 20: 205 (1981).

14. Environment Canada. Ambient air concentrations of volatile organic compounds in Toronto and Montreal. Unpublished report, Pollution Measurement Division (1986).

15. Jo, W.K., Weisel, C.P. and Lioy, P.J. Chloroform exposure and the health risk associated with multiple uses of chlorinated tap water. Risk Anal., 10(4): 581 (1990).

16. Orme, J., Mullin, C.S. and Ohanian, E.V. Regulatory effects on the practice of drinking water treatment. In: Water chlorination: Chemistry, environmental impact and health effects. Vol. 6. R.L. Jolley, L.W. Condie, J.D. Johnson, S. Katz, R.A. Minear, J.S. Mattice and V.A. Jacobs (eds.). Lewis Publishers, Chelsea, MI (1990).

17. U.S. Environmental Protection Agency. Health effects of drinking water technologies. Chap. 3. Disinfection. Drinking Water Health Effects Task Force, Office of Drinking Water. Lewis Publishers, Chelsea, MI. p. 56 (1989).

18. Otson, R. and Chan, C. Sample handling and analysis for 51 volatile organics by an adapted purge and trap GC-MS technique. Int. J. Environ. Anal. Chem., 30: 275 (1987).

19. National Academy of Sciences. Drinking water and health. Disinfectants and disinfectant by-products. Vol. 7. National Academy Press, Washington, DC (1987).

20. American Water Works Association. Water quality and treatment. 4th edition. McGraw-Hill, New York, NY (1990).

21. Ahmed, A.E., Kubic, V.L. and Anders, M.W. Metabolism of haloforms to carbon monoxide. I. *In vitro* studies. Drug Metab. Dispos., 5(2): 198 (1977).

22. Anders, M.W., Stevens, J.L., Sprague, R.W., Shaath, Z. and Ahmed, A.E. Metabolism of haloforms to carbon monoxide. II. *In vivo* studies. Drug Metab. Dispos., 6(5): 556 (1978).

23. Stevens, J.L. and Anders, M.W. Metabolism of haloforms to carbon monoxide. III. Studies on the mechanism of the reaction. Biochem. Pharmacol., 28: 3189 (1979).

24. Stevens, J.L. and Anders, M.W. Metabolism of haloforms to carbon monoxide. IV. Studies on the reaction mechanism *in vivo*. Chem. Biol. Interact., 37: 365 (1981).

25. National Toxicology Program. Toxicology and carcinogenesis studies of tribromomethane (bromoform) (CAS No. 75-25-2) in F344/N rats and B6C3F$_1$ mice (gavage studies). NTP TR 350. U.S. Department of Health and Human Services, Research Triangle Park, NC (1989).

26. Mink, F.L., Brown, T.J. and Rickabaugh, J. Absorption, distribution, and excretion of ^{14}C-trihalomethanes in mice and rats. Bull. Environ. Contam. Toxicol., 37: 752 (1986).

27. Cresteil, T., Beaune, P., Leroux, J.P., Lange, M. and Mansuy, D. Biotransformation of chloroform by rat and human liver microsomes; in vitro effect on some enzyme activities and mechanism of irreversible binding to macromolecules. Chem. Biol. Interact., 24: 153 (1979).

28. Fry, B.J., Taylor, T. and Hathway, D.E. Pulmonary elimination of chloroform and its metabolite in man. Arch. Int. Pharmacodyn. Ther., 196: 98 (1972).

29. Withey, J.R., Collins, B.T. and Collins, P.G. Effect of vehicle on the pharmacokinetics and uptake of four halogenated hydrocarbons from the gastrointestinal tract of the rat. J. Appl. Toxicol., 3: 249 (1983).

30. Carlo, G.L. and Mettlin, C.J. Cancer incidence and trihalomethane concentrations in a public drinking water system. Am. J. Public Health, 70(5): 523 (1980).

31. Cech, I., Holguin, A.H., Littell, A., Henry, J.P. and O'Connell, J. Health significance of chlorination byproducts in drinking water: the Houston experience. Int. J. Epidemiol., 16(2): 198 (1987).

32. Tuthill, R.W. and Moore, G.S. Drinking water chlorination: a practice unrelated to cancer mortality. J. Am. Water Works Assoc., 72: 570 (1980).

33. Isacson, P., Bean, J.A., Splinter, R., Olson, D.B. and Kohler, J. Drinking water and cancer incidence in Iowa. III. Association of cancer with indices of contamination. Am. J. Epidemiol., 121(6): 856 (1985).

34. Brenniman, G.R., Vasilomanolkis-Lagos, J., Amsel, J., Namekata, T. and Wolff, A.H. Case-control study of cancer deaths in Illinois communities served by chlorinated or non-chlorinated water. In: Water chlorination: Environmental impact and health effects. Ann Arbor Science Publishers, Ann Arbor, MI. p. 1043 (1980).

35. Young, T.B., Kanarek, M.S. and Tsiatis, A.A. Epidemiologic study of drinking water chlorination and Wisconsin female cancer mortality. J. Natl. Cancer Inst., 67(6): 1191 (1981).

36. Kanarek, M.S. and Young, T.B. Drinking water treatment and risk of cancer death in Wisconsin. Environ. Health Perspect., 46: 179 (1982).

37. Gottlieb, M.S., Carr, J.K. and Morris, D.T. Cancer and drinking water in Louisiana: colon and rectum. Int. J. Epidemiol., 10(2): 117 (1981).

38. Gottlieb, M.S., Carr, J.K. and Clarkson, J.R. Drinking water and cancer in Louisiana. A retrospective mortality study. Am. J. Epidemiol., 116(4): 652 (1982).

39. Gottlieb, M.S. and Carr, J.K. Case-control cancer mortality study and chlorination of drinking water in Louisiana. Environ. Health Perspect., 46: 169 (1982).

40. Zierler, P.H.S., Feingold, L., Danley, R.A. and Craun, G. Bladder cancer in Massachusetts related to chlorinated and chloraminated drinking water: A case-control study. Arch. Environ. Health, 43(2): 195 (1988).

41. Lawrence, C.E., Taylor, P.R., Trock, B.J. and Reilly, A.A. Trihalomethanes in drinking water and human colorectal cancer. J. Natl. Cancer Inst. 72(3): 563 (1984).

42. Cragle, D.L., Shy, C.M., Struba, R.J. and Siff, E.J. A case-control study of colon cancer and water chlorination in North Carolina. In: Water chlorination: Chemistry, environmental impact and health effects. Vol. 5. R.L. Jolley, R.J. Bull, W.P. Davis, S. Katz, M.H. Roberts and V.A. Jacobs (eds.). Lewis Publishers, Chelsea, MI. p. 153 (1985).

43. Cantor, K.P., Hoover, R., Hartge, P., Mason, T.J., Silverman, D.T. and Levin, L.I. Drinking water source and risk of bladder cancer: a case-control study. In: Water chlorination: Chemistry, environmental impact and health effects. Vol. 5. R.L. Jolley, R.J. Bull, W.P. Davis, S. Katz, M.H. Roberts and V.A. Jacobs (eds.). Lewis Publishers, Chelsea, MI. p. 145 (1985).

44. Cantor, K.P., Hoover, R., Hartge, P., Mason, T.J., Silverman, D.T., Altman, K., Austin, D.F., Child, M.A., Key, C.R., Marrett, L.D., Myers, M.H., Narayana, A.S., Levin, L.I., Sullivan, J.W., Swanson, G.M., Thomas, D.B. and West, D.W. Bladder cancer, drinking water source and tap water consumption: a case-control study. J. Natl. Cancer Inst., 79(6): 1269 (1987).

45. Young, T.B., Wolf, D.A. and Kanarek, M.S. Case-control study of colon cancer and drinking water trihalomethanes in Wisconsin. Int. J. Epidemiol., 16(2): 190 (1987).

46. Wilkins, J.R. and Comstock, G.W. Source of drinking water at home and site-specific cancer incidence in Washington County, Maryland. Am. J. Epidemiol., 113(2): 178 (1981).

47. Murphy, P.A. Review of previous epidemiologic studies reporting associations between drinking water disinfection and cancer. Presented at the 6th Conference on Water chlorination: Environmental impact and health effects, Oak Ridge, TN (1987).

48. Craun, G.F. Surface water supplies and health. J. Am. Water Works Assoc., 80: 40 (1988).

49. Chu, I., Secours, V., Marino, I. and Villeneuve, D.C. The acute toxicity of four trihalomethanes in male and female rats. Toxicol. Appl. Pharmacol., 52: 351 (1980).

50. Chu, I., Villeneuve, D.C., Secours, V.E., Becking, G.C. and Valli, V.E. Trihalomethanes: II. Reversibility of toxicological changes produced by chloroform, bromodichloromethane, chlorodibromomethane and bromoform in rats. J. Environ. Sci. Health, B17(3): 225 (1982).

51. Chu, I., Villeneuve, D.C., Secours, V.E., Becking, G.C. and Valli, V.E. Trihalomethanes: I. Toxicity of trihalomethanes: I. The acute and subacute toxicity of chloroform, bromodichloromethane, chlorodibromomethane and bromoform in rats. J. Environ. Sci. Health, B17(3): 205 (1982).

52. Munson, A.E., Sain, L.E., Sanders, V.M., Kauffmann, B.M., White, K.L., Jr., Page, D.G., Barnes, D.W. and Borzellecal, F. Toxicology of organic drinking water contaminants: Trichloromethane, bromodichloromethane, dibromochloromethane, and tribromomethane. Environ. Health Perspect., 46: 117 (1982).

53. Bull, R.J., Brown, J.M., Meierhenry, E.A., Jorgenson, T.A., Robinson, M. and Stober, J.A. Enhancement of the hepatotoxicity of chloroform in B6C3F$_1$ mice by corn oil: implications for chloroform carcinogenesis. Environ. Health Perspect., 69: 49 (1986).

54. National Cancer Institute. Carcinogenesis bioassay of chloroform. Carcinogen Bioassay and Program Resources Branch, Bethesda, MD, March (1976).

55. Reuber, M.D. Carcinogenicity of chloroform. Environ. Health Perspect., 31: 171 (1979).

56. Jorgenson, T.A., Meierhenry, E.F., Rushbrook, C.J., Bull, R.J. and Robinson, M. Carcinogenicity of chloroform in drinking water to male Osborne-Mendel rats and female $B6C3F_1$ mice. Fundam. Appl. Toxicol., 5: 760 (1985).

57. Roe, F.J.C., Palmer, A.K., Worden, A.N. and van Abbe, N.J. Safety evaluation of toothpaste containing chloroform. I. Long-term studies in mice. J. Environ. Pathol. Toxicol., 2: 799 (1979).

58. Klaunig, J.E., Ruch, R.J. and Pereira, M.A. Carcinogenicity of chlorinated methane and ethane compounds administered in drinking water to mice. Environ. Health Perspect., 69: 89 (1986).

59. Deml, E. and Oesterle, D. Dose-dependent promoting activity of chloroform in rat liver foci bioassay. Cancer Lett., 29: 59 (1985).

60. Tumasonis, C.F., McMartin, D.N. and Bush, B. Lifetime toxicity of chloroform and bromodichloromethane when administered over a lifetime in rats. Ecotoxicol. Environ. Saf., 9: 233 (1985).

61. National Toxicology Program. Toxicology and carcinogenesis studies of bromodichloromethane (CAS No. 75-27-4) in F344/N rats and $B6C3F_1$ mice (gavage studies). NTP TR 321. U.S. Department of Health and Human Services, Research Triangle Park, NC (1987).

62. Charbonneau, M., Strasser, J., Jr., Lock, E.A., Turner, M.J., Jr. and Swenberg, J.A. Involvement of reversible binding to alpha-2-microglobulin in 1,4-dichlorobenzene-induced nephrotoxicity. Toxicol. Appl. Pharmacol., 99: 122 (1989).

63. National Toxicology Program. Toxicology and carcinogenesis studies of chlorodibromomethane (CAS No. 124-48-1) in F344/N rats and $B6C3F_1$ mice (gavage studies). NTP TR 282. U.S. Department of Health and Human Services, Research Triangle Park, NC (1985).

64. Rosenthal, S.L. A review of mutagenicity of chloroform. Environ. Mol. Mutagen., 10: 211 (1987).

65. Varma, M.M., Ampy, F.R., Verma, K. and Talbot, W.W. *In vitro* mutagenicity of water contaminants in complex mixtures. J. Appl. Toxicol., 8(4): 243 (1988).

66. Morimoto, K. and Koizumi, A. Trihalomethanes induce sister chromatid exchanges in human lymphocytes *in vitro* and mouse bone marrow cells *in vivo*. Environ. Res., 32: 72 (1983).

67. Thompson, D.J., Warner, S.D. and Robinson, V.B. Teratology studies of orally administered chloroform in the rat and rabbit. Toxicol. Appl. Pharmacol., 29: 348 (1974).

68. Ruddick, J.A., Villeneuve, D.C., Chu, I. and Valli, V.E. A teratological assessment of four trihalomethanes in the rat. J. Environ. Sci. Health, B18(3): 333 (1983).

69. Burkhalter, J.E. and Balster, R.L. Behavioral teratology evaluation of trichloromethane in mice. Neurobehav. Toxicol., 1: 199 (1979).

70. Krasner, S.W., McGuire, M.J., Jacangelo, J.G., Papnia, N.L., Reagan, K.M. and Aieta, E.M. The occurrence of disinfection by-products in US drinking water. J. Am. Water Works Assoc., 81: 41 (1989).

Trihalomethanes

1.0 Background Data

Trihalomethanes (THMs) are volatile, halogenated organic compounds that are often formed during chlorine disinfection in potable water treatment as a result of the reaction of chlorine with certain types of organic material present in the raw water. Humic and fulvic acids are the most common organic materials that react with chlorine to form THMs. Certain species of algae have also been implicated as THM precursors. Generally, groundwaters contain little organic material and are not susceptible to THM formation.[1]

The THM reaction is not instantaneous and may continue for several days, depending on the conditions. Increased pH, temperature and chlorine dosage tend to increase the rate of THM formation and the amount of THMs formed.[1]

In general, only four THMs occur in measurable amounts in treated drinking water supplies: chloroform (or trichloromethane, $CHCl_3$), bromodichloromethane (or dichlorobromomethane, $CHCl_2Br$), chlorodibromomethane (or dibromochloromethane, $CHClBr_2$) and bromoform (or tribromomethane, methyl tribromide, $CHBr_3$).[1-3] The sum of the concentrations of these four THMs is generally referred to as total trihalomethanes (TTHM). Chloroform is usually the most abundant THM, often accounting for greater than 90% of the TTHM concentration. If there is a significant amount of bromide in the raw water, the brominated THMs may be dominant.[1]

Several properties of these THMs may affect their behaviour during water treatment processes. Some of them are shown Table 1.

2.0 Water Treatment

The control of THMs in drinking water can be approached in three ways: precursor removal, the use of alternative disinfectants that do not form THMs and removal of THMs themselves.

2.1 Conventional Treatment

The major mechanism for THM control in conventional water treatment is usually THM precursor removal in the coagulation/sedimentation process. A stoichiometric relationship between the concentration of humic material and the required coagulant dosage for its removal has been reported. Organics removal by coagulation has been found to be optimum at pH 4 to 6. Aluminum and iron salts have both been reported to be effective for removal of humic and fulvic acids. Removal of organic material by coagulation using aluminum and iron salts has been reported to vary from 25% to 90% between source waters.[1]

Table 1 Properties of Trihalomethanes

Property	$CHCl_3$	$CHCl_2Br$	$CHClBr_2$	$CHBr_3$
Molecular weight[3,4]	119.38	163.8	208.3	252.8
Density[2,4]	1.485 g/mL	1.971 g/mL	2.44 g/mL	2.887 g/mL 2.98 g/mL
Melting point[3]	-63.5°C	-57.1°C	<-20.0°C	8.3°C 6–7°C
Boiling point[2,3,4]	61.7°C at 101.3 kPa	90°C at 101.3 kPa 89.2–90.6°C	119–120°C at 99.7 kPa 118–122°C	149.5°C at 101.3 kPa 150.3–151.2°C
Flash point[2]	-	-	-	9°C
Vapour pressure[3]	20.1 kPa at 20°C	6.7 kPa at 20°C	2.0 kPa at 10.5°C	1.3 kPa at 34°C
Octanol/water partition coefficient (log)[3]	1.97	1.88	2.09	2.30
Henry's law constant[1]	0.152	0.095	0.035	0.024

For the coagulation process to be most effective for THM control, the initial point of chlorine application should be after the coagulation/sedimentation process to allow for as much precursor removal as possible prior to chlorination. Reductions in THM production of up to 75% in full-scale plants have been reported as a result of moving the initial chlorination application point past the coagulation/sedimentation process.[1]

Oxidation of THM precursors with chemical oxidants other than chlorine is a possible method of THM control. Potential oxidants include ozone, chlorine dioxide, potassium permanganate, hydrogen peroxide and ultraviolet (UV) radiation; reduction of THM precursor concentrations of up to 90% has been reported with the use of ozone oxidation.[5] Researchers have also reported enhanced capability for THM production following ozone oxidation.[6,7] Chlorine dioxide has shown some limited potential for THM precursor oxidation.[8,9] Chlorine dioxide also has the capability to inhibit the chlorine precursor reaction. The use of potassium permanganate for THM precursor oxidation has had

limited success.[1] Hydrogen peroxide, UV radiation and combinations of UV radiation and hydrogen peroxide have been investigated for precursor oxidation with mixed results.[10] Glaze and co-workers successfully used UV radiation in combination with ozonation for precursor oxidation in a pilot-scale experimental program.[11,12]

Adsorption of THM precursors with activated carbon is a potential THM control alternative. Granular activated carbon (GAC) adsorption of THM precursor material is usually very effective initially, but breakthrough often occurs rather quickly. It is generally concluded that pilot plant studies are required for the design of each individual GAC installation because of variability of the precursor material. The use of powdered activated carbon (PAC) for THM precursor adsorption has also been investigated with varying results.[1]

Replacing chlorine with an alternative disinfectant that does not form THMs is a possible method of THM control. Ozone, chlorine dioxide and chloramines are the most common alternatives to chlorine. Ozone is an excellent disinfectant but does not provide a long-lasting residual. Ozone must, therefore, be used in combination with another disinfectant. Chlorine dioxide is usually considered to be as effective a disinfectant as chlorine. A major concern regarding the use of chlorine dioxide is related to the potential adverse health effects of its inorganic by-products--chlorate and chlorite. Chloramines have seen considerable use in North America but are considered inferior to chlorine as disinfectants. Although it has been demonstrated that ozone, chlorine dioxide and chloramines do not form THMs, it is generally accepted that some organic by-products are formed, the health effects of which are not well known.

2.2 Special Treatment

Special treatment for THM control usually focuses on the removal of THMs after their formation. Air stripping and adsorption are the most common methods of THM removal.

Symons *et al.* calculated the theoretical minimum air/water ratios required to achieve complete removal in a perfect tower aeration system: 6.7:1, 10.2:1, 28:1 and 41:1 for chloroform, bromodichloromethane, chlorodibromomethane and bromoform, respectively.[1]

Symons and co-workers reviewed a number of research efforts using both packed tower aeration and diffused air aeration.[1] They indicated that the best removals of chloroform were by countercurrent packed towers (>90%), although their use was relatively inefficient. The batch-scale diffused air experiments used the air more efficiently, but the chloroform removal (~50%) was less. These investigators suggested that aeration is a feasible approach to THM removal; however, removal becomes more difficult with the increased molecular weight of the brominated THMs. This normally should not be a major factor, as chloroform usually dominates the TTHMs.

Symons *et al.* reviewed the use of PAC for THM adsorption and found that PAC was relatively ineffective for

chloroform removal.[1] The bromine-containing THMs were much more adsorbable.

Studies involving GAC adsorption of THMs have been reviewed by Symons and co-workers.[1] A number of studies were reviewed that used varying empty bed contact times (EBCTs), influent THM concentrations and GAC media. The EBCT used in the various studies ranged from 3.2 to 46.3 minutes. The influent THM concentrations were as high as 156 μg/L. The authors noted that EBCT, contaminant influent concentration and the fraction of THMs containing bromine all influence the GAC bed's service life to exhaustion. Generally, except for very long EBCTs, the service life to exhaustion was short. Symons *et al.* concluded that GAC for THM removal may not be recommended because of the high reactivation frequency required.[1]

Symons *et al.* also reviewed the size of synthetic resins for THM adsorption.[1] From the review, they concluded that synthetic resins were generally unsatisfactory for THM adsorption; only one resin was tested, showing a high capacity for THM adsorption.

An inherent problem with removal of THMs after their formation is that further THMs can continue to form as long as free chlorine is available.

3.0 Conclusions

Current treatment technology for THM control usually focuses on removal of THM precursors by coagulation/ sedimentation prior to chlorination. GAC adsorption for precursor removal can also be effective, but the useful life of the GAC is often short.

Replacing chlorine with an alternative disinfectant that does not form THMs is a viable option for THM control. Ozone, chlorine dioxide and chloramines are the most common alternatives. Each of these alternatives has specific advantages and disadvantages.

Removal of THMs after their formation is also a possible THM control alternative. Air stripping and GAC adsorption are the most common technologies for THM removal. Rapid exhaustion of the media is often a problem when using GAC for THM adsorption.

4.0 References

1. Symons, J.M., Stevens, A.A., Clark, R.M., Geldreich, E.E., Love, O.T. and DeMarco, J. Treatment techniques for controlling trihalomethanes in drinking water. American Water Works Association, Denver, CO (1982).

2. Sax, N.I. and Lewis, R.J., Sr. (eds.). Hawley's condensed chemical dictionary. 11th edition. Van Nostrand Reinhold Co., New York, NY (1987).

3. U.S. Environmental Protection Agency. Water-related environmental fate of 129 priority pollutants. Vol. II. Halogenated aliphatic hydrocarbons, halogenated ethers, monocyclic aromatics, phthalate esters, polycyclic aromatic hydrocarbons, nitrosamines, miscellaneous compounds. EPA-440/4-79-029b, Office of Water Planning and Standards (WH-553), Washington, DC (1979).

4. Sax, N.I. Dangerous properties of industrial materials. 5th edition. Van Nostrand Reinhold Co., New York, NY (1979).

5. Trussell, R.R. and Umphres, M.D. The formation of trihalomethanes. J. Am. Water Works Assoc., 70(11): 604 (1978).

6. Reckhow, D.A. and Singer, P.C. The removal of organic halide precursor by preozonation and alum coagulation. J. Am. Water Works Assoc., 76(4): 151 (1984).

7. Reckhow, D.A. and Singer, P.C. Mechanisms of halide formation during fulvic acid chlorination and implications with respect to preozonation. In: Water chlorination: Chemistry, environmental impact and health effects. Vol. 5. R.L. Jolley, R.J. Bull, W.P. Davis, S. Katz, M.H. Roberts and V.A. Jacobs (eds.). Lewis Publishers, Chelsea, MI (1985).

8. Aieta, E.M. and Berg, J.D. A review of chlorine dioxide in drinking water treatment. J. Am. Water Works Assoc., 78(6): 62 (1986).

9. Werdehoff, K.S. and Singer, P.C. Chlorine dioxide effects of THMFP, TOXFP, and the formation of inorganic by-products. J. Am. Water Works Assoc., 78(11): 83 (1987).

10. McLeod, B.R. Trihalomethane precursor reduction in natural water. MSc thesis, University of Saskatchewan, Saskatoon (1989).

11. Glaze, W.H., Peyton, G.R., Huang, F.Y., Burleson, J.L., Jones, P.C., Prengle, N.W., Nall, A.E. and Joshi, D.S. Oxidation of water supply refractory species by ozone with ultraviolet radiation. EPA-600/2-80-110, U.S. Environmental Protection Agency, Cincinnati, OH (1980).

12. Glaze, W.H., Peyton, G.R. and Sohm, B. Pilot scale evaluation of photolytic ozonation for trihalomethane precursor removal. U.S. Environmental Protection Agency, Cincinnati, OH (1984).

1.005.

ALBERTA

Sampling/Analytical Program

During the period January 1, 1987 to July 31, 1990, Alberta Environment sampled a total of 225 Alberta municipal drinking waters (227 individual sampling sites) for trihalomethanes (THMs). The sampling protocol consisted of sampling from within the distribution system at a location distant from the treatment facility; collecting samples in duplicate in specially prepared 40 mL septum cap vials containing sodium thiosulphate crystals to quench further possible THM formation. All samples were analyzed using the "purge and trap" technique. Larger centres (eg. Edmonton, Calgary, Red Deer and Lethbridge) were sampled on a monthly basis with small communities sampled quarterly or less.

Provincial Exposure

Levels of total THMs averaged 70.1 μg/L and ranged from 0 to 528 μg/L. Chloroform was the principal THM observed, with bromodichloromethane also observed in the majority of samples, but at concentrations at least 2 to 60 times lower. Chlorodibromomethane and bromoform were observed only occasionally and then generally at or near the method detection level of 1 μg/L.

Treatment Technology

There are over 270 municipally owned water treatment systems in Alberta with 121 using ground water and 150 drawing from surface waters. Chlorination is provided in 81% of the ground water systems and in all of the surface water supplies. Surface water systems also provide a minimum of filtration with around 66% of the systems providing full conventional treatment (ie. coagulation, flocculation, sedimentation, filtration and disinfection).

*Presented (unedited) in the language in which it was submitted.

ALBERTA (continued)

Mean total THM values at individual municipal sites ranged from 0 to a high of 360 μg/L. It was noted that of the 227 municipal sites tested, 126 locations (56% of municipalities) had mean total THM values less than 50 μg/L; 51 additional locations had mean total THM values between 50 μg/L to 100 μg/L; and finally, there were 50 sites with mean total THM values exceeding 100 μg/L (50 > 100 μg/L, 31 > 150 μg/L, and 7 > 250 μg/L). Where multiple sampling data was available at a given site, it was generally noted that summer/fall values increased, which was possibly due to the biological activity (algal and general plant growth) in surface waters during late summer and fall which results in increased THM precursor concentrations in drinking water supplies.

Provincial Impact

Optimization of treatment processes to remove dissolved organics prior to chlorination and using chloramines instead of free chlorine for disinfection are strategies that have been used successfully in Alberta to minimize the production of total THMs. It is estimated that the total cost to reduce THM levels at the 44% of Alberta municipalities that currently would be exceeding a 50 μg/L THM limit would be in the tens of million dollars range.

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BRITISH COLUMBIA

Sampling/Analytical Program

In 1983, the Ministry of Health carried out a survey of selected water systems in British Columbia and based upon the findings at that time concluded that there were virtually no problems with meeting the present trihalomethane (THM) guideline of 0.35 mg/L. Of 190 water samples analyzed in 1983, from 63 waterworks systems, six percent (11 samples) exceeded 0.05 mg/L and two percent (4 samples) exceeded 0.1 mg/L. No samples exceeded 0.35 mg/L. However, since 1983 there have been concerns regarding parasitic contamination of surface water supplies used as drinking water sources and disinfection levels commonly followed in British Columbia in 1983 were lower in comparison to the levels necessary to control *Giardia*, a parasitical problem that has only recently become apparent. Thus, there has been minimal monitoring for THMs since 1983 and only recently have water purveyors in higher risk areas begun to disinfect at increased chlorine levels. Monitoring for THMs to assess this impact is only now beginning at the Health Unit level.

Provincial Exposure

See "Sampling/Analytical Program" section. There is very little recent information available on THM levels with the exception of self-monitoring data from the Greater Vancouver Water District which has levels averaging 0.03 mg/L and the Greater Victoria Water District which because it uses chloramines for disinfection has low THMs. These two supplies provide about one-half of the drinking water supply for British Columbia residents.

Treatment Technology

Approximately eighty three percent (83%) of the residents of British Columbia on community water supplies rely on surface water supplies. With the exception of a few water systems, all rely on simple disinfection as the only barrier to microbiological contamination. There are about 1,200 water systems with 600 using surface sources and 600 mainly relying on groundwater. Most surface water source users rely on chlorine as disinfectant without the benefit of filtration or any other physical or chemical renovation. Thus, if THMs are found to be elevated, the costs

BRITISH COLUMBIA (continued)

of resolution could exceed simple or minor improvements to the disinfection process and could include major upgrading costs. All surface water sources are impacted to some extent by periodic turbidity from precipitation and some sources are affected by plant and other biological growth which can enhance THM production.

Provincial Impact

The possible costs to reduce THMs to levels below 0.05 mg/L are unknown but there is a high potential for some cost impacts because of the traditional use of surface waters in British Columbia with disinfection only (chlorination). Increasing chlorine concentrations as a strategy to resolve health risks from *Giardia*, will undoubtedly have an impact on some water supplies and result in increased THM production. The use of chloramines as an alternative is not considered appropriate as it is inadequate as a primary disinfectant for *Giardia* control and it has recently been considered as a concern for use in areas where spills from water systems could cause fish kills in sensitive areas. Chlorine at higher levels is considered to be the only appropriate short-term strategy for *Giardia* control where there are no other easily obtainable sources of water that are free from *Giardia*. A reduction in the THM guideline could have major cost implications in the future, as *Giardia* concerns are addressed, and the resolution (more effective disinfection) causes THM levels to increase.

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MANITOBA

Sampling/Analytical Program

There are currently 290 public water systems being monitored in Manitoba. Of these, 135 utilities use a surface water supply or an infiltration gallery as a source of raw water. Chlorination is mandatory for all public water systems in the Province and The Public Health Act (Manitoba) requires that a minimum of 20 minutes of chlorine contact time be provided at all facilities using a surface water source prior to distribution.

In October of 1986, 38 of these utilities were selected for sampling for the presence of trihalomethanes (THMs) in product water. Samples were taken for general chemistry (inorganic), THMs and total organic carbon. The sampling protocol for THMs consisted of collecting duplicate samples at the water treatment plant, after the required contact time and prior to distribution. Samples were collected in specially prepared 25 or 40 millilitre septum seal screw-cap amber-glass bottles. The reducing agent, sodium thiosulphate, was added to each sample bottle at the laboratory prior to shipment to quench further THM formation after sampling. All samples were analyzed using the "Liquid-Liquid Extraction Gas Chromatographic Method" as described in the 16th edition of Standard Methods for the Examination of Water and Wastewater.

The principal THMs identified were chloroform ($CHCl_3$) followed by bromodichloromethane ($CHBrCl_2$), chlorodibromomethane ($CHClBr_2$), then bromoform ($CHBr_3$), the latter two being detected only occasionally at or near the detection level. Total THM levels observed ranged from <1.2 μg/L to 757.2 μg/L with a mean value of 103.7 μg/L. Mean level of THMs at utilities using prechlorination was 129 μg/L, while utilities not prechlorinating had a mean THM level of 71 μg/L. Distribution of the 38 utilities was as follows:

Total THMs <50 (μg/L)	$50<$ Total THMs <100 (μg/L)	Total THMs >100 (μg/L)
16 (42%)	10 (26%)	12 (32%)

Only one utility exceeded the existing guideline value of 350 μg/L.

*Presented (unedited) in the language in which it was submitted.

MANITOBA (continued)

Monitoring Program

Routine monitoring for THMs at public water supply systems was initiated in June 1989. In the 1989-1990 fiscal year, 59 utilities were sampled for THMs. Sampling was limited to surface water supplies and shallow aquifers susceptible to surface activities. Samples were collected in specially prepared 40 millilitre septum seal screw-cap amber-glass bottles. Preservatives were not added to the sample bottles such that maximum THM formation could be measured. All samples were analyzed using the "Liquid-Liquid Extraction Gas Chromatographic Method" as described in the 16th edition of Standard Methods for the Examination of Water and Wastewater. Total THM levels observed ranged from <1.2 μg/L to 519.0 μg/L with a mean value of 94.7 μg/L. Distribution of the 59 utilities was as follows:

Total THMs <50 (μg/L)	$50<$ Total THMs <100 (μg/L)	Total THMs >100 (μg/L)
23 (39%)	16 (27%)	20 (34%)

Only one utility exceeded the existing guideline value of 350 μg/L.

Contribution of various THMs to the total THM value at the 59 utilities monitored are:

% of Total THM	$CHCl_3$	$CHBrCl_2$	$CHClBr_2$	$CHBr_3$
< detection	5	5	5	5
< 10%		29	45	52
10 to 20%		14	3	2
21 to 30%		8	4	
> 30%	54	3	2	

*Presented (unedited) in the language in which it was submitted.

MANITOBA (continued)

Sampling for maximum THM formation potential will continue through the 1990-1991 and 1991-1992 sampling years. Multiple sampling data is still limited due to shortages in sampling and analytical resources. Once all available data has been compiled and a new THM guideline has been finalized, only those utilities identified as being near or in exceedance of the new guideline value will receive a more intensive investigation of THM levels (quarterly sampling at extremities of the distribution system) to determine actual levels in the system.

Provincial Impact

Treatment process trains are being reviewed on a limited, ongoing basis in order to optimize removal of THM precursors. Where utilities are upgrading their treatment processes, THM precursor reduction is strongly encouraged. Manitoba is maintaining its current legislated position on the disinfection of product water with chlorine for all public water supplies. Compliance with the THM guideline must be achieved through process optimization or system upgrading and not through the use of alternative disinfectants. It is noted that these process improvements are not THM specific, water quality can also be significantly improved from both health and aesthetic perspectives in addition to THM reduction.

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NEW BRUNSWICK

Sampling/Analytical Program

During the summer months (June, July, August) of 1990, 171 sources of drinking water in communities of various sizes were sampled once for the analysis of water chemistry, bacteriological contamination and the presence of volatile organic chemicals (VOC's) to provide a baseline for a continuing program of monitoring water quality. For the VOC's, samples were collected within the distribution system from a "well-run" tap into a laboratory-prepared, solvent-washed 60 millilitre glass bottle, the full bottles being kept cold until delivered for extraction by "purge and trap" technique and analysis by gas chromatography mass spectrometer for a screening of 32 VOC's.

Provincial Exposure

Of the 157 samples assayed for VOC's, 45 (29%) were positive for chloroform (detection limit = 0.2 μg/L). Trace amounts of bromodichloromethane were also detected but at levels up to 60-fold lower than for chloroform. Concentrations of chloroform ranged from 0.2 to 193 μg/L with an average of approximately 40 μg/L.

Treatment Technology

There are approximately 260 drinking water systems in New Brunswick. In the study to date, 157 systems have been examined, 136 of these using ground water and 21 using surface water. Chlorination is provided regularly for 26% of all systems with standby chlorination devices being available for an additional 11.5% of the systems. For systems drawing on surface waters, chlorination is provided for 90% on a regular or standby basis.

Based on a single (or duplicate) sampling/analysis for chloroform, 45 of the 157 sites had measurable or detectable levels (0.2 μg/L), with 35 sites being below 50 μg/L although none exceeded the current federal/provincial guideline of 350 μg/L. As all of these were "summer" samples taken during hot, sunny weather, they

*Presented (unedited) in the language in which it was submitted.

NEW BRUNSWICK (continued)

constituted a worst-case scenario. Re-sampling of positive systems yielded lower levels of chloroform in the autumn and following spring, supporting the concept that excessive biological activity in the surface waters coupled with increased chlorine usage contributed to the generation of trihalomethanes.

Provincial Impact

To date, no alternative strategies (removal of dissolved organic, chloramination rather than free chlorine, etc.) have been used in New Brunswick as the trihalomethanes have not exceeded the existing guideline value of 350 μg/L. However, should the guideline be reduced to 100 or 50 μg/L, this would seriously affect the compliance to the guideline in 3 or 10 community systems, respectively, and would require a re-examination of possible techniques for disinfection.

NEWFOUNDLAND

Sampling/Analytical Program

Sampling for trihalomethanes (THMs) in Newfoundland water supplies was carried out as part of a larger Federal-Provincial Toxic Chemical Survey of Municipal Drinking Water Sources. The program was carried out between 1985 and 1988 with 10 community water supplies tested each year for a total of 40 supplies. For THMs, duplicate samples were taken from the raw water source and at some point in the distribution system. In all cases the water supplies were from surface sources. Each system was sampled twice in the year of sampling - once in the spring and once in the fall. Samples for THMs were preserved in the field and were analyzed using purge and trap gas chromatography mass spectrometer techniques.

Provincial Exposure

Levels of total THMs ranged as follows for each study year:

1985 - Not detected to 80 μg/L
1986 - Not detected to 130 μg/L
1987 - Not detected to 110 μg/L
1988 - Not detected to 180 μg/L

Only three (3) sites were reported to have no chlorination at the time of sampling. However, chlorine residual levels were not recorded at all sampling runs and it is possible that some of the systems that reported having chlorination in place may have had a very low level of application. There was no attempt to correlate the level of chlorine residual with the level of THM formation.

Treatment Technology

In Newfoundland there are approximately 260 municipally owned water systems with the large majority (approximately 90%) using surface water as a source. In most supplies chlorine is the only treatment applied to the water. Chlorination of public water supplies has been mandatory since 1972. Only ten (10) communities treat their water by coagulation and/or filtration, while approximately another ten (10) adjust their pH in addition to the normal chlorine treatment.

*Presented (unedited) in the language in which it was submitted.

NEWFOUNDLAND (continued)

In addition to the municipally owned water systems there are approximately another 200 systems servicing the unincorporated areas of the province. These systems use a combination of ground and surface waters and for the most part only utilize chlorine for disinfection purposes.

Provincial Impact

To date there has been very little effort to focus generally on treatment for THMs in Newfoundland. Individual considerations are given to such treatment as the need arises to expand water systems and/or make alterations for other reasons.

Based on the preliminary information provided in the above noted Federal Provincial Survey and the general knowledge of the presence of highly coloured water, it is conceivable that a number of water supplies in Newfoundland would not meet a 50 μg/L THM limit. The impact of having to meet such a limit could be significant, both in the appropriation of capital and operating expenditures as well as having to develop a core of skilled operating personnel from a current very low availability of such.

NORTHWEST TERRITORIES

Sampling/Analytical Program

Trihalomethane (THM) sampling is not done routinely and very limited information exists on current water supplies. Currently, THM values have only been collected on 36 of 60 communities with only one sample collected from each supply. However, extrapolating on this limited data, approximately fifty percent (50%) of the communities will not be able to meet a THM limit of 50 μg/L. If the limit was lowered to 100 μg/L, only about ten percent (10%) of the communities would exceed this limit.

Provincial Impact

Based on this limited data and a 50 μg/L limit, a level four cost estimate has been completed. It is estimated $ 15,000,000 capital plus $ 6,000,000 in increased annual operating and maintenance funds are required. This cost represents about fifty percent (50%) of the Territories' annual construction budget and twenty five percent (25%) of the annual maintenance budget.

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NOVA SCOTIA

Sampling/Analytical Program

Municipal water supplies throughout Nova Scotia have been monitored since 1989 for trihalomethanes (THMs).

In addition, a detailed study of the Sydney water supply was conducted in 1989-90. This study was initiated because of the high readings obtained during the routine sampling program. Samples were collected on a monthly basis, taking into consideration the amount of chlorine added, chlorine residual, pH, temperature, etc.

The analytical procedure used to monitor THMs is method 6210 C, the "Purge and Trap Capillary Column Gas Chromatography-Mass Spectrometry. This method is described in detail in Standard Methods for the Examination of Water and Wastewater, 1989 17th Edition, AWWA APHA WPCF, also EPA 624.

Provincial Exposure

The most recent quarterly sampling for a one year period of 81 public drinking supplies in the province, of which 18 have ground water sources (ie. no THM precursors) indicated that of the remaining 63 surface water supplies, 38 had an average chloroform reading of >50 μg/L, 18 had THM readings which averaged over 100 μg/L, and 9 averaged over 150 μg/L.

Present Research Work

Presently, a detailed research study is underway in Nova Scotia involving Centre for Water Resources Studies (TUNS), Fenwick Laboratories Limited, and a Master of Applied Science (Civil Engineering) student at the Technical University of Nova Scotia.

*Presented (unedited) in the language in which it was submitted.

NOVA SCOTIA (continued)

This study consists of two phases. The first phase takes the form of an optimization study to determine the most effective type of coagulant (and dose of that coagulant) which would yield maximum removal of total organic carbon (and therefore THM precursors) at a given pH. The second phase of this research project involves a detailed study of the THM precursors using high-pressure liquid chromatography (HPLC-MS).

Four sites in Nova Scotia are under study, from which samples will be drawn and analyzed in each of the four seasons.

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ONTARIO

Sampling/Analytical Program

The most reliable trihalomethane (THM) data on drinking water supplies in Ontario are provided through the Drinking Water Surveillance Program (DWSP). The DWSP officially began in 1986, and now extends to almost 100 of the approximately 490 municipal water supply systems in Ontario. The municipal water supplies that are currently monitored through the DWSP are the larger supplies in the Province, and they provide more than 85 percent of Ontario's municipal water supply.

Through the DWSP, supplies are monitored either monthly or every second month for THM's, with unquenched samples of treated water being collected at the water treatment plant, and, as of 1991, quenched samples of treated water being collected and analyzed from representative sites in the distribution system.

All samples are analyzed using the "purge and trap" technique.

Provincial Exposure

Trihalomethane levels in drinking water supplies in Ontario vary greatly depending upon numerous factors including the type of raw water supply (i.e. lake, river or groundwater), the organic content of each supply, and the treatment processes applied. Water treatment plants that use the Great Lakes or groundwater as a source of raw water tend to have lower THM levels in the water than do supplies that use other inland lakes and rivers as a source of raw water.

For the purposes of this summary, DWSP data from 1991 are compiled. Ninety-six municipal water supplies were assessed in 1991. Annual mean THM values ranged from less than 5 μg/L to 194 μg/L for samples collected at water treatment plants, while, for samples collected in the distribution system, annual mean values ranged from less than 5 μg/L to 177 μg/L.

Between 65 and 70 supplies had annual mean THM levels less than 50 μg/L, and approximately 30 supplies had mean levels exceeding that level. Data are summarized in Table 1.

*Presented (unedited) in the language in which it was submitted.

ONTARIO (continued)

Table 1: 1991 DWSP Data for THMs

	Annual Mean THM Level (µg/L)		
	Less Than 50	Greater than 50 and less than 100	Greater than 100
Distribution System Samples (Quenched)*	69	19	8
Water Treatment Plant Samples (Unquenched)**	64	16	16

NOTES:

* Samples may be "quenched" by the addition of sodium thiosulphate to halt any further formation of THMs in the sample bottle itself. THM levels in "quenched" distribution system samples are considered more representative of the actual THM levels in water at the tap.

** "Unquenched plant samples give an indication of the potential for THM formation.

Treatment Technology

A wide range of treatment technologies are currently used in Ontario, ranging from simple filtration and disinfection systems to elaborate coagulation-flocculation, filtration systems employing various disinfection mechanisms. The type of treatment employed is dependent upon several factors including quality and characteristics of the raw water supply, size of the system and population served, economics, etc.

A reduction in THM levels in treated drinking water may be achieved by removing precursor material through processes such as chemically-assisted coagulation or granular activated carbon filtration prior to disinfection. THMs may also be removed after disinfection through the use of granular activated carbon filtration, but this is very expensive and difficult to operate with optimal efficiency over extended periods of time. Reverse osmosis systems may also be used to remove trihalomethanes.

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ONTARIO (continued)

Alternative disinfectants, such as ozone and chlorine dioxide may reduce THM production and levels, but great care must be taken to ensure that disinfection efficiency is not compromised. Where chlorination is used, the addition of ammonia to create a combined chlorine residual will also reduce THM levels.

Chlorine is still the preferred disinfectant for drinking water treatment in Ontario.

If THM levels have to be reduced in numerous water distribution systems, it will be necessary to assess each system on a case-by-case basis in order to determine the optimal treatment process and ensure optimization of the procedures.

Provincial Impact

At this time, it is impossible to accurately estimate the impact of a significantly lowered THM objective for drinking water as reliable data are not currently available on many smaller drinking water supplies and the cost of case-by-case assessment and treatment modification is unknown.

However, it can be generally estimated that if all municipal water supplies had to meet a 50 μg/L level for THMs, the total cost to the Province of Ontario would likely range from 500 million to a billion dollars.

QUÉBEC

Campagnes d'Échantillonnage, Prélevements et Analyses

Depuis 1985, le programme des micropolluants permet d'exercer une surveillance sur la présence de plusieurs composés volatils dont les THM dans les réseaux de distribution d'eau potable. Dix-huit (18) municipalités couvrant une population totalisant 2 400 000 h. ont fait partie des campagnes biannuelles d'été et d'hiver, depuis 1985. Les teneurs de THM présentes dans les systèmes de distribution ont été déterminées plus particulièrement lors des campagnes de février 1986, juillet 1986, juillet 1987 et à l'hiver 1988 et les municipalités à l'étude avaient en commun un traitement complet.

Une campagne visant à déterminer les teneurs des THM totaux dans les systèmes de distribution de 99 municipalités n'effectuant qu'une simple chloration a aussi été entreprise durant l'été 1987.

Quant aux types d'échantillons prélevés, aux méthodes d'échantillonnage et d'analyses utilisées, les mêmes procédures ont été retenues pour les différentes campagnes. Les échantillons d'eau provenant des réseaux de distribution ont été prélevés à 1,5 km des stations de purification dans des bouteilles contenant du thiosulfate de sodium. Les bouteilles de 60 ml étaient remplies à rebord et ensuite fermées hermétiquement. La méthode "purge and trap"/chromatographie en phase aqueuse est celle que le laboratoire du ministère de l'Environnement du Québec à Montréal utilise dans le dosage des THM. Cette méthode est reproduite en annexe de ce rapport.

Niveau d'Exposition

La concentration médiane calculée à partir des résultats des quatre campagnes été-hiver, pour les municipalités d'envergure est de 37 μg/L. La concentration moyenne de THM totaux correspondante est de 46 μg/L et les plages des concentrations détectées pour ces municipalités varient entre 1,5 et 182 μg/L.

Pour les petites municipalités, la concentration médiane des teneurs d'été est de 62 μg/L. Cette valeur se compare d'ailleurs assez bien à celle obtenue pour les grandes municipalités qui s'établit à 71 μg/L.

*Presented (unedited) in the language in which it was submitted.

QUÉBEC (continued)

Pour ces petites municipalités, les plages de concentrations varient de 4 à 421 μg/L. On note cependant pour ces dernières, l'absence fréquente de chlore résiduel libre (50% des cas) dans les réseaux. On peut ainsi penser à une sous-estimation du potentiel de ces dernières à former des THM.

Caractérisation des Modes de Traitement et Potentiel de Formation des THM

On compte au total, 1,114 réseaux de distribution municipaux d'eau potable au Québec. Six-cent-quarante-deux (642) de ces derniers possèdent un traitement, alors que quatre-cent-soixante-douze (472) ne procèdent à aucun traitement. De ces derniers toutefois, les 2/3 sont alimentés à partir de nappes d'eau souterraines.

Les réseaux de distribution d'eau traitée (642), alimentent plus de 90% de la population desservie par un système d'approvisionnement municipal et se distinguent en 3 catégories:

- les réseaux appliquant un traitement complet (nombre: 91);
- les réseaux appliquant un traitement quelconque avant la chloration (environ 66);
- les réseaux ne pratiquant qu'une simple chloration (près de 500).

Ainsi, on peut estimer qu'environ 14% des réseaux d'eau traitée subissent un traitement complet et près de 75% constituent des réseaux assurant une simple chloration, le reste regroupant les réseaux effectuant un traitement d'appoint. A la lumière de ces informations, on peut conclure que pour chacune des catégories de traitements étudiés (traitement complet et simple chloration), les études respectives ont considéré 20% des réseaux.

Lorsque les concentrations moyennes de chaque réseau sont considérées, on remarque pour les campagnes jumelées de juillet 1987 et d'hiver 1988, qu'aucune des 18 municipalités ne dépassaient une moyenne annuelle de 100 μg/L. Sept d'entre elles étaient supérieures à 50 μg/L. Aussi, peut-on conclure que pour les municipalités d'envergure, des valeurs supérieures à 100 μg/L demeurent exceptionnelles.

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QUÉBEC (continued)

Il demeure plus difficile, à partir des données disponibles, de dresser un tableau des teneurs moyennes dans les réseaux des petites municipalités. On note cependant que 3 d'entre elles présentent durant l'été, à au moins une occasion, des teneurs supérieures à 175 μg/L et laissent supposer dans ce cas, des dépassements supérieurs à 100 μg/L de moyenne annuelle.

Impact Économique

Malgré les données partielles disponibles, il est clair que la norme roposée de 50 μg/L fixée sur une moyenne annuelle, pourrait toucher près de 40% des grandes municipalités effectuant un traitement complet. Le Québec par ailleurs compte un nombre considérable de petits réseaux qui à la lumière des données pourraient être affectées de la même manière par cette norme.

La recherche d'une eau souterraine ou l'installation des équipements de filtration pour respecter une norme de turbidité de 1 UTN sont les premières étapes préconisées pour abaisser les THM. Il en coûterait ainsi de l'ordre de 360 millions de dollars répartis sur 310 municipalités et 160 réseaux privés. Malgré l'installation d'un système de traitement complet, 30% de ces usines devront ajouter un traitement d'appoint pour atteindre la norme de 50 μg/L. L'utilisation d'un système d'aération qui est peu coûteux pourrait être envisagée. Enfin, le polissage sur charbon actif pourrait être nécessaire pour 17% des petites municipalités qui en vertu des coûts et des difficultés d'opération seront contraintes à se limiter à la recherche des eaux souterraines ou plutôt attendre l'avènement d'autres technologies (membranes?)

Bref, advenant l'injection de fonds nécessaires, une norme moyenne de 50 μg/L de THM pourrait être respectée par environ 85% des exploitants au Québec.

Par ailleurs, le Québec ne s'est pas prononcé sur l'innocuité des sous-produits associés aux méthodes alternatives de désinfection et ne les considère pas actuellement lors des études techniques et économiques.

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SASKATCHEWAN

Sampling/Analytical Program

Communities with surface water supply sources are required to sample for trihalomethanes (THMs) in Saskatchewan. For the purposes of this report, we evaluated discrete samples of the treated water reservoirs and distribution systems of 152 communities treating surface water during the period March 1, 1985 to December 31, 1990 inclusive. The samples were collected in specially prepared 40 ml septum cap vials containing sodium thiosulphate to quench potential further THM formation.

During this period 2315 water samples were analyzed by the "Direct Aqueous Injection (DAI)" technique. Although a small amount of additional data is available by alternate analytical methodology such as purge & trap or gas spurge, this data was not used. It should be noted that 92% of the DAI data is from samples collected in the summer.

In order to assess provincial exposure with proposed THMs guidelines based on purge and trap analysis, DAI data has been adjusted to a "purge equivalent" value by a factor of 0.625.

Since the proposed guideline is based on an annual average of seasonal results we also "seasonally equated"[1] our data. Conversion factors for seasonal to annual average data were as follows:

-Spring (Apr. 1 - May 31) multiply by 0.9314
-Summer (Jun. 1 - Sept. 30) multiply by 0.9834
-Fall (Oct. 1 - Dec. 31) multiply by 0.8348
-Winter (Jan. 1 - Mar. 31) multiply by 1.4082

Provincial Exposure

Tables 1 and 2 show the numbers of communities with an annual average of seasonal THM results $\geq$ 50 μg/L and $\geq$ 100 μg/L by municipal type and population groups respectively.

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[1] Halomethane Study-Selected Saskatchewan Water Supplies Water Pollution Control Branch, Saskatchewan Department of the Environment (July 1978).

*Presented (unedited) in the language in which it was submitted.

TABLE 1 THMs BY MUNICIPAL TYPE

YEAR	# OF COMMUNITIES SAMPLED	THMs > = 50							THMs > = 100						
		A	B	C	D	E	F	TOTAL	A	B	C	D	E	F	TOTAL
1985	104	10	21	25	5	0	1	62	8	9	13	1	0	1	32
1986	106	14	14	23	4	0	0	55	4	8	12	2	0	0	26
1987	106	16	23	31	6	0	1	77	12	19	21	4	0	1	57
1988	110	16	29	29	6	0	1	81	12	16	15	5	0	0	48
1989	109	13	24	29	5	0	1	72	8	13	19	4	0	0	44
1990	101	9	24	29	7	1	2	72	9	14	19	5	0	1	48
*TOTALS	152	23	41	38	8	1	2	113	20	29	29	6	0	2	86

A=HAMLET; B=VILLAGE; C=TOWN; D=CITY; E=NORTHERN HAMLET; F=NORTHERN VILLAGE

TABLE 2 THMs BY COMMUNITY POPULATION

YEAR	# OF COMMUNITIES SAMPLED	THMs > = 50							THMs > = 100						
		A	B	C	D	E	F	TOTAL	A	B	C	D	E	F	TOTAL
1985	104	11	16	7	11	11	6	62	8	7	3	7	5	2	32
1986	106	14	13	4	10	9	5	55	7	6	1	4	5	3	26
1987	106	18	15	9	13	15	7	77	14	13	6	10	9	5	57
1988	110	18	21	9	10	16	7	81	13	13	4	6	7	5	48
1989	109	13	16	10	12	15	6	72	9	9	5	9	7	5	44
1990	101	13	16	9	11	15	8	72	9	11	5	9	8	6	48
*TOTALS	152	28	25	16	15	20	9	113	23	21	8	13	14	7	86

A=POPULATION < = 100; B=POPULATION > 100 AND < = 250; C=POPULATION > 250 AND < = 500
D=POPULATION > 500 AND < = 1000; E=POPULATION > 1000 AND < = 5000; F=POPULATION > 5000

SASKATCHEWAN (continued)

During the sampling period 113 of 152 communities had annual average THM results ≥ 50 μg/L. Eighty-six of these communities had annual average results ≥ 100 μg/L. Fourteen communities had "purge equivalent" THM results exceeding the existing guideline of 350 μg/L during the sampling period. Chloroform was the predominant THM observed.

Treatment Technology

In Saskatchewan, there are 537 municipal water treatment systems with 385 using ground water, 114 using surface water and 38 using a combination of ground and surface water sources. All communities treating surface water in Saskatchewan are required to disinfect their treated water supplies. Minimum surface water treatment in Saskatchewan can be chemically assisted filtration (direct filtration), slow rate filtration, or cartridge filtration. Conventional surface water treatment requires facilities for coagulation-flocculation, sedimentation, filtration and disinfection.

Table 3 shows the types of treatment processes utilized for the communities identified with THM problems in Table 1.

Provincial Impact

Optimization of direct filtration and conventional surface water treatment systems is practised in Saskatchewan. However, direct filtration and conventional treatment are not expected to consistently meet annual average THM guidelines of either ≥ 100 μg/L or ≥ 50 μg/L. Therefore, cost estimates are based on all communities identified in the provincial exposure assessment (113 communities ≥ 50 μg/L and 86 communities ≥ 100 μg/L).

For the purposes of cost estimating, Table 4 provides information concerning the unit costs of potential THM control technologies for communities which have surface water treatment capacities within specific ranges.

*Presented (unedited) in the language in which it was submitted.

TABLE 3 TREATMENT PROCESSES

	A	B	C
NUMBER OF PLANTS (THMs > = 50 μg/L)	21	20	72

	A	B	C
NUMBER OF PLANTS (THMs > = 100 μg/L)	14	15	57

A = FILTRATION AND DISINFECTION
B = COAGULATION, FILTRATION, AND DISINFECTION - SLOW RATE FILTER AND DISINFECTION - CARTRIDGE FILTRATION AND DISINFECTION
C = COAGULATION, FLOCCULATION, SEDIMENTATION, FILTRATION, AND DISINFECTION

TABLE 4 UNIT COSTS BY PLANT CAPACITY

	small	medium	large
CHLORAMINATION-LIQUID	$14,000	$ 22,000	$ 31,000
CHLORAMINATION-ANHYDROUS	$31,500	$ 60,000	$ 72,000
OZONE	$71,000	$100,000	$430,000
AIR STRIPPING	$28,000	$ 82,000	$300,000

small < 0.05 million gallons per day water treatment plants
medium > 0.05 & < 0.5 million gallons per day water treatment plants
large > 0.5 million gallons per day water treatment plants

SASKATCHEWAN (continued)

Table 5 provides total cost information for several potential THM control methods. The unit cost estimates have been derived from "The Cost Digest" USEPA EPA-600/8-84-010 dated October 1984. Unit costs for individual treatment technologies for water treatment plants which produce less than one million gallons per day have been extrapolated. Cost information has been converted to Canadian dollars and imperial gallons. For updating of cost information (the data presented are March, 1980), two approaches may be used: either an overall cost index, such as the Engineering News Record (ENR) index, or a number of specific indices.

The costs for air stripping do not include any allowance for volatile organic air pollution control and assume packed tower aeration with a 100 to 1 air to water ratio. The costs for chloramination assumes existing chlorine feed works. The costs for ozonation assume a 5 mg/L dosage rate and air as the oxygen source.

TABLE 5 **TOTAL COSTS**

TOTAL COSTS THMs > = 100

	small 54	medium 27	large 5	TOTALS
CHLORAMINATION-LIQUID	$ 756,000	$ 594,000	$ 155,000	$1,505,000
CHLORAMINATION-ANHYDROUS	$1,701,000	$1,620,000	$ 360,000	$3,681,000
OZONE	$3,834,000	$2,700,000	$2,150,000	$8,684,000
AIR STRIPPING	$1,512,000	$2,214,000	$1,500,000	$5,226,000

TOTAL COSTS THMs > = 50

	small 73	medium 33	large 7	TOTALS
CHLORAMINATION-LIQUID	$1,022,000	$ 726,000	$ 217,000	$ 1,965,000
CHLORAMINATION-ANHYDROUS	$2,299,500	$1,980,000	$ 504,000	$ 4,783,500
OZONE	$5,183,000	$3,300,000	$3,010,000	$11,493,000
AIR STRIPPING	$2,044,000	$2,706,000	$2,100,000	$ 6,850,000

small < 0.05 MGD plants
medium > 0.05 MGD & < 0.5 MGD plants
large > 0.5 MGD plants

May 1989

Part I

Derivation of Maximum Acceptable Concentrations and Aesthetic Objectives for Chemicals in Drinking Water

Part I

Derivation of Maximum Acceptable Concentrations and Aesthetic Objectives for Chemicals in Drinking Water

Introduction

The maximum acceptable concentrations (MACs) for chemical parameters in the *Guidelines for Canadian Drinking Water Quality* were derived based on careful consideration of available scientific data according to the principles outlined in this section.

Data on effects of exposure to chemical agents are obtained in toxicological studies in animal species and occasionally in epidemiological studies of human populations. Effects vary, depending upon the dosage, route of exposure (e.g., ingestion, inhalation or dermal), frequency or duration of exposure and the species, sex and age of the exposed population. Effects of exposure to chemicals are generally classified in the following broad categories: organ-specific, neurological/behavioural, reproductive, teratological and oncogenic/carcinogenic/mutagenic. Effects may be brief or prolonged, reversible or irreversible, immediate or delayed, single or multiple. The nature, number, severity, incidence and/or prevalence of specific effects in a population generally increase with increasing dose; this is commonly referred to as the dose–response relationship.

For some types of toxic effects that result from exposure to chemicals, it is believed that there is a dose (or threshold) below which adverse effects will not occur. For other types of toxic effects, it is assumed but not proven that there is some probability of harm at any level of exposure (i.e., no threshold). At present, the latter assumption is generally considered to be appropriate only for carcinogenesis. For some types of carcinogens (i.e., those that induce tumours by particular mechanisms, such as promotion), however, it is believed that there may be a threshold dose below which tumours will not occur.

There is uncertainty in the scientific database used in the derivation of guidelines for maximum exposure to chemical substances. Inadequate data on the level, frequency and duration of exposure, differences in sensitivity between species and among individuals in the same species, inadequate study design, potential for interactive effects and variations in statistical models for extrapolation of responses observed at high doses to those expected at low doses contribute to this uncertainty. Every effort has been made to take these uncertainties into account in the approaches for deriving maximum acceptable concentrations (MACs) for chemical parameters described in this section and the Supporting Documents. It should also be emphasized that fundamental to the approaches to derivation of guidelines outlined in this section is the need for application of sound scientific judgement on a case-by-case basis.

Approaches to Derivation of MACs

Different approaches were adopted for the derivation of guidelines for compounds considered to be carcinogenic and probably carcinogenic, compounds considered to be possibly carcinogenic and those considered to be probably not carcinogenic or for which data were inadequate for evaluation. It was necessary, therefore, to classify chemicals with respect to their potential carcinogenicity into various groups, as outlined in Appendix A, on the basis of rigorous examination of the quantity, quality and nature of the results of available toxicological and epidemiological studies. Chemicals classified as carcinogenic often also induce toxic effects other than malignant tumours; for these substances, the guideline was derived on the basis of the approach that led to the most stringent value. In most cases, this was the approach specified for carcinogenic chemicals.

Chemicals That Are Not Carcinogenic

For chemicals classified as "probably not carcinogenic to man" or for which data on carcinogenicity were "inadequate for evaluation" (Groups IV and V in Appendix A), the MAC was derived based on an acceptable daily intake (ADI)* for organ-specific, neurological/behavioural, reproductive or teratological effects. Where possible, the ADI was derived by division of the lowest no-observed-adverse-effect level (NOAEL) for a response considered to be biologically significant by an uncertainty factor. Ideally, the NOAEL was derived from a lifetime ingestion study or studies in the most sensitive subpopulation (e.g.,

* See Appendix B for definitions.

teratological studies); data from acute or short-term studies were not used in calculating ADIs. The uncertainty factor was derived on a case-by-case basis; in general, however, a factor of 1 to 10 times was used to account for each of the following elements of uncertainty: intraspecies variation, interspecies variation, nature and severity of effect, adequacy of study and lowest-observed-adverse-effect level (LOAEL) versus NOAEL. An additional factor of 1 to 5 times was incorporated where there was information that indicated a potential for interaction with other chemicals. If the chemical was an essential nutrient at low concentrations, the dietary requirement was also taken into consideration in derivation of the uncertainty factor.

Derivation of the MAC was generally based on an average daily intake of 1.5 L of drinking water by a 70-kg adult (Department of National Health and Welfare 1981). However, where appropriate, the MAC was derived based on intake in the most sensitive subpopulation (e.g., pregnant women, children). Human exposure from sources other than drinking water (e.g., air, food, consumer products) was taken into account by apportioning a percentage of the ADI to drinking water. Where possible, data concerning the proportion of total intake normally ingested in drinking water (based on mean levels in food, air and treated municipal water supplies) or intakes estimated on the basis of consideration of physical/chemical properties were used in the calculations. Where such information was unavailable, a value of 20% was used in the derivation of the MAC.

Contaminants present in drinking water may contribute to total intake not only by ingestion, but also by inhalation or dermal exposure to water during bathing and other household activities. For some compounds, intake by these routes has been estimated to be similar to that by ingestion. However, in most cases, available data were insufficient to enable estimation of exposure by inhalation and dermal absorption of contaminants present in drinking water. The 20% allocation of total daily intake to drinking water is believed to be generous, however, and should be sufficient to account for these additional routes of intake.

In some cases where the calculated total daily intake from all sources was less than 50% of the ADI, allocation to drinking water was based on consideration of additional factors, such as feasibility. In no case, however, could the calculated total daily intake from food, air and drinking water (containing levels at the MAC) exceed the ADI.

Maximum acceptable concentrations must be achievable by available treatment methods and measurable by existing analytical techniques. Where an MAC was less than levels considered to be reliably measurable or achievable, an "interim MAC" (IMAC) was established, and improvement in methods of quantitation and/or treatment was recommended.

Chemicals That Are Carcinogenic

As it is generally accepted that carcinogenesis is a non-threshold phenomenon, it is assumed that there is a probability of harm at any level of exposure to carcinogenic chemicals. Ideally, therefore, carcinogens should be absent from drinking water. However, the incremental risks associated with exposure to low levels of these chemicals in drinking water may be sufficiently small so as to be essentially negligible compared with other risks commonly encountered in society.

Quantitative risks associated with exposure to low levels of potential carcinogens are estimated by extrapolation (usually over many orders of magnitude) of the dose–response relationship observed at high doses in experimental studies (most often in animal species) to the low-dose range. There are a number of uncertainties involved in these mathematical extrapolations; the methods used are, however, based on conservative assumptions and probably tend to overestimate rather than underestimate the risks. The actual risks at low levels of exposure may, therefore, be lower than the estimated values by 1 to 2 orders of magnitude.

For chemicals classified as "carcinogenic to man" or "probably carcinogenic to man" (Groups I and II in Appendix A), lifetime cancer risks were estimated using the robust linear extrapolation model, applied to the tumour types considered to be most appropriate from a biological perspective. Wherever possible, information on pharmacokinetics, metabolism and mechanisms of carcinogenicity was incorporated into the model for risk estimation. To account for differences in metabolic rates between animals and man, a surface area to body weight correction was applied, except in those cases where it was not justified on the basis of available data on pharmacokinetics and metabolism.

For many carcinogenic compounds (substances classified in Groups I and II in Appendix A), available treatment technology is inadequate to completely eliminate exposure from drinking water. In addition, available analytical methods may be inadequate for reliable determination at extremely low levels. Therefore, MACs were set as close to zero as reasonably practicable, on the basis of consideration of the following factors:

- The MAC must be achievable by available water treatment methods at reasonable cost.
- Wherever possible, the upper 95% confidence limit for the lifetime cancer risk associated with the MAC was less than 10^{-5} to 10^{-6}, a range that is generally considered to be "essentially

negligible." In cases where intake from sources other than drinking water (e.g., food, air and consumer products) was significant, the upper 95% confidence limit for the lifetime cancer risk associated with the MAC was less than or equal to 10^{-6}.

- The MAC must also be reliably measurable by available analytical methods.

Where estimated lifetime cancer risks associated with the MAC were greater than those judged to be essentially negligible (i.e., 10^{-5} to 10^{-6}), an IMAC was established, and improvement in methods of quantitation and/or treatment was recommended.

Chemicals That Are Possibly Carcinogenic

For compounds that are "possibly carcinogenic to man" (Group III in Appendix A), the MAC was based upon an ADI determined as described in the section entitled "Chemicals That Are Not Carcinogenic"; however, an additional factor of 1 to 10 times was incorporated in the uncertainty factor to account for the limited evidence of carcinogenicity. In some cases where there were sufficient data (e.g., increased incidence of benign tumours at several sites in several species), a quantitative estimate of tumour incidence was considered in derivation of the MAC.

Pesticides

The approach to derivation of the MACs and IMACs for pesticides included in the Supporting Documentation differs somewhat from that for other chemical parameters. A number of pesticides considered to be "probably not carcinogenic to man" or for which data on carcinogenicity were "inadequate for evaluation" (Groups IV and V in Appendix A) have been considered by the Food Directorate, Health Protection Branch, Department of National Health and Welfare, to establish maximum tolerable residue levels in foods, as part of their registration under the Pest Control Products Act. These evaluations include an extensive assessment of data for establishment of either ADIs or, where there are data gaps or data of poor quality, negligible daily intakes (NDI), which incorporate a larger uncertainty factor. Wherever possible, these ADIs or NDIs established by the Food Directorate have been used in the derivation of MACs or IMACs, respectively, for the pesticides included in the Supporting Documentation, for the following reasons:

- to ensure consistency of approach in relation to the establishment of residue limits in foods.
- to take advantage of the very detailed scientific assessment already available in most cases.
- to ensure that all relevant data (including confidential data submitted under the Pest Control Products Act) are taken into consideration in derivation of MACs and IMACs.

The World Health Organization (WHO), in conjunction with the Food and Agriculture Organization (FAO), also conducts evaluations to derive ADIs or, where data are insufficient, provisional daily intakes, which incorporate a larger uncertainty factor, for pesticide residues in foods. For chemicals that fall into Groups IV and V in Appendix A ("probably not carcinogenic to man" or for which data on carcinogenicity are "inadequate for evaluation") and that have been evaluated by the WHO, MACs or IMACs were based upon FAO/WHO ADIs or provisional daily intakes, respectively.

Approach to Derivation of Aesthetic Objectives

In those cases where thresholds for organoleptic properties were less than the MAC, an "aesthetic objective" (AO) was derived, based on information on taste and odour thresholds reported in the literature.

Reference

Department of National Health and Welfare. Tap water consumption in Canada. 82-EHD-80, Environmental Health Directorate, Ottawa (1981).

Appendix A: Criteria for Classification of Carcinogenicity

Chemicals were classified into four main categories on the basis of the following criteria (modified from those of the International Agency for Research on Cancer):

Group I — Carcinogenic to Man

Group I — Data from adequate epidemiological studies indicate that there is a causal relationship between the agent and cancer in man (i.e., the observed association is unlikely to be due to chance, bias or confounding). Confidence in inferring a causal relationship is increased when the association is strong and observed in several studies, when there is a dose–response relationship or when a reduction in exposure is followed by a reduction in the incidence of cancer.

Group II — Probably Carcinogenic to Man

Group II — Data from epidemiological studies are inadequate to assess carcinogenicity either because there are few pertinent investigations or because chance, bias or confounding cannot be excluded as a possible explanation for the results. However, there is sufficient evidence of carcinogenicity in animal species (i.e., there is an increased incidence of malignant tumours in

multiple species or strains or in multiple experiments with different routes of exposure or dose levels, or the incidence, site or type of tumour at age of onset is unusual). Confidence in the sufficiency of the data from animal studies is increased when there is evidence of a dose–response relationship, supporting results from *in vitro* studies or limited carcinogenicity bioassays, evidence of structure–activity relationships or supporting data on mechanisms of toxicity.

Group III — Possibly Carcinogenic to Man

Group IIIA — Data from epidemiological studies indicate an association between exposure and human cancer, but alternative explanations such as chance, bias or confounding cannot be excluded.

Group IIIB — Data from epidemiological studies are inadequate to assess carcinogenicity. There is some evidence of increased tumour incidence in animals, but the data are limited because the studies involve a single species, strain or experiment, study design (i.e., dose levels, duration of exposure and follow-up, survival, number of animals) or reporting is inadequate, the neoplasms produced often occur spontaneously and have been difficult to classify as malignant by histological criteria alone (e.g., lung and liver tumours in mice), there is an increase in the incidence of benign tumours only, or it is believed on the basis of information on the mechanism of action that increased tumour incidence is observed only at very high doses or that it is species-dependent.

Group IV — Probably Not Carcinogenic to Man

Group IVA — There is no evidence of carcinogenicity in sufficiently powerful and well-designed epidemiological studies; there is no evidence of carcinogenicity in adequate studies in two animal species.

Group IVB — There is no evidence of carcinogenicity in sufficiently powerful and well-designed epidemiological studies; data in animal species are inadequate.

Group IVC — There are no adequate epidemiological data; there is no evidence of carcinogenicity in adequate animal studies in two different species.

Group V — Inadequate Data for Evaluation

Group VA — Data from epidemiological and/or animal studies are inadequate (i.e., because of major qualitative or quantitative limitations, the studies cannot be interpreted as showing either the presence or absence of carcinogenicity).

Group VB — There are no data available for evaluation.

Appendix B: Definitions

ADI — Acceptable daily intake: the amount that can be consumed from all sources each day by an adult, even for a lifetime, without any significant increased risk to health.

AO — Aesthetic objective: this applies to certain substances or characteristics of drinking water that can affect its acceptance by consumers or interfere with practices for supplying good water. For certain parameters, both aesthetic objectives and health-related guidelines (maximum acceptable concentrations) have been derived. Where only aesthetic objectives are specified, the values are below those considered to constitute a health hazard.

IMAC — Interim maximum acceptable concentration: in those instances where there were insufficient toxicological data to derive a maximum acceptable concentration (MAC) with reasonable certainty, interim values have been recommended, taking into account the available health-related data but employing a larger factor to compensate for the additional uncertainties involved. An interim value was also established for those substances for which estimated lifetime risks of cancer associated with the guideline (the lowest level that was practicably achievable) were greater than those deemed to be essentially negligible. Because of the nature of interim maximum acceptable concentrations, they will be reviewed periodically, as new toxicological data and developments in methods of quantitation and/or treatment become available.

LOAEL — Lowest-observed-adverse-effect level: the lowest dose in a toxicity study that results in an observed adverse effect (usually one dosage level above the NOAEL).

MAC — Maximum acceptable concentration: maximum acceptable concentrations have been established for certain substances that are known or suspected to cause adverse effects on health. They have been derived to safeguard health on the basis of lifelong consumption. To the extent possible, the use of drinking water for all usual domestic purposes, including personal hygiene, has been considered in the derivation of the guidelines. However, water of higher quality may be required for some special purposes, including renal dialysis.

Drinking water that continually contains substances at levels greater than the maximum acceptable concentrations will contribute significantly to consumers' exposure to these substances and may, in some instances, be capable of inducing deleterious

effects on health. However, short-term excursions above the maximum acceptable concentrations do not necessarily mean that the water constitutes an undue risk to health. The amount by which, and the period for which, the maximum acceptable concentration can be exceeded without posing a health risk must be assessed by taking into account the toxicity of the substance involved. When the maximum acceptable concentration for a contaminant is exceeded, however, the minimum action required is immediate resampling. If the maximum acceptable concentration continues to be exceeded, the authorities responsible for public health should be consulted concerning appropriate corrective action.

NDI — Negligible daily intake: when insufficient toxicological data are available to derive an acceptable daily intake (ADI) from all sources with reasonable certainty, a provisional value is recommended that takes into account the available health-related data.

NOAEL — No-observed-adverse-effect level: the highest dose in a toxicity study that does not result in any observed adverse effect (an adverse effect significantly alters the health of the target animal for a sustained period of time or reduces survival).