Arsenic in Drinking Water

Document for Public Comment

Prepared by the Federal-Provincial-Territorial Committee on Drinking Water

Comment Period Ends
May 17, 2005

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Public Comment Document

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Purpose of Consultation

The Federal-Provincial-Territorial Committee on Drinking Water has assessed the available information on arsenic with the intent of revising the current guideline for arsenic in drinking water. The purpose of this consultation is to solicit comments on the proposed guideline, on the approach used for its development, and on the potential economic costs of implementing it, as well as to determine the availability of additional exposure data.

The Committee has requested that this document be made available to the public and open for comment. Comments are appreciated, with accompanying justification, if required. Comments can be sent via E-mail to water_eau@hc-sc.gc.ca or by mail to the Committee on Drinking Water Secretariat, Water Quality and Health Bureau, 4th Floor, Sir Charles Tupper Bldg., A.L. 6604B, Ottawa, Ontario K1A 0K9. All comments must be received before **May 17, 2005.**

It should be noted that this supporting document on arsenic in drinking water will be revised following evaluation of comments received, and a maximum acceptable concentration (MAC) for arsenic in drinking water will be established. This document should be considered a draft for comment only.

November 2004

Arsenic

1.0 Proposed Guideline

The proposed maximum acceptable concentration (MAC) for arsenic in drinking water is 0.005 mg/L (5 µg/L) based on municipal-scale treatment achievability. At the residential scale, use of treatment devices[*] may not be able to achieve the proposed MAC, especially where influent levels are high. Certified devices are frequently designed to remove arsenic to well below this concentration, but certification to the standard only verifies that a final concentration of 0.01 mg/L or less is achieved.

2.0 Executive Summary

Arsenic is a natural element that is widely distributed throughout the Earth's crust. It is often found naturally in groundwater, through erosion and weathering of soils, minerals, and ores. Arsenic compounds are used commercially and industrially in the manufacture of a variety of products and may enter drinking water sources directly from industrial effluents and indirectly from atmospheric deposition.

Health Canada recently completed its review of the health risks associated with arsenic in drinking water. This review assesses all identified human health risks, taking into account new studies and approaches, as well as the limitations of available treatment technology. Based on this review, the proposed guideline for arsenic in drinking water is 0.005 mg/L.

During its April 2004 meeting, the Federal-Provincial-Territorial Committee on Drinking Water reviewed the proposed guideline for arsenic and gave approval for the guideline and the corresponding supporting document to undergo public consultations.

2.1 Health Effects

Arsenic is classified as a human carcinogen. As arsenic is a natural contaminant of groundwater, its health effects have been widely studied in humans, particularly in Taiwan. This is particularly significant because the toxic effects of arsenic vary significantly between species, making animal studies an unreliable basis on which to develop a guideline.

The maximum acceptable concentration (MAC) for arsenic in drinking water was established based on the incidence of internal (lung, bladder, liver, kidney) cancers in humans, through the calculation of a lifetime unit risk. The MAC for arsenic has been set at a level that is slightly higher than the level that would be considered to be associated with an "essentially negligible" risk, based on limitations of available treatment technology.

The health effects of arsenic in humans vary depending on the compound and form. Metallic arsenic is not absorbed from the stomach and does not have any adverse health effects. Although it was generally accepted that the inorganic forms of arsenic were responsible for its toxic and carcinogenic effects and that its organic forms were less toxic, recent evidence is now questioning this assumption.

2.2 Exposure

Arsenic can be found in both surface water and groundwater sources, with levels generally higher in groundwater. Most provinces and territories across Canada report some areas

[*] Treatment devices include point-of-use (POU) and point-of-entry (POE) devices.

where arsenic can be detected in drinking water supplies. Although levels are generally well below the proposed MAC, elevated arsenic concentrations have been found in areas with natural sources.

Drinking water is considered to be the major source of exposure to arsenic only in populations living near a source of arsenic (either a natural geological source or a site of contamination). For most Canadians, the primary source of exposure to arsenic is food, followed by drinking water, soil, and air.

2.3 Treatment

The establishment of a MAC in drinking water must take into consideration the ability to both measure the contaminant and remove it from drinking water supplies. Arsenic can be reliably measured at a concentration of 0.005 mg/L. The selection of an appropriate treatment process for a specific water supply will depend on the characteristics of the raw water supply and many other factors. It is important to determine what, if any, pretreatment is required.

Arsenic can be effectively treated in municipal-scale treatment facilities through a number of well-documented methods, which typically include both a pretreatment step and a final polishing step. Arsenic can be reduced to levels below the proposed MAC of 0.005 mg/L in both large and smaller municipal plants.

Arsenic can also be removed by residential-scale drinking water treatment devices, although certified treatment devices can ensure the reduction of arsenic only to a concentration of 0.01 mg/L. Certified devices are frequently designed to remove arsenic to well below this concentration, but certification to the standard verifies only that a final concentration of 0.01 mg/L or less is achieved.

Since the availability of treatment technology is a limiting factor in establishing a guideline for arsenic in drinking water, Health Canada and the Federal-Provincial-Territorial Committee on Drinking Water will continue to monitor emerging treatment technologies to revise and update the guideline and its supporting document as required.

3.0 Identity, Use, and Sources in the Environment

Arsenic is a metalloid with oxidation states of −3, 0, 3, and 5. It is widely distributed throughout the Earth's crust and is a major constituent of at least 245 mineral species. The most common source of arsenic in Canada is sulphide minerals. These minerals are typically composed of 0.02–0.5% arsenic; however, certain pyrite minerals may contain up to 5% arsenic (Hindmarsh and McCurdy, 1986; Pellerin, 2003).

Arsenicals are used commercially and industrially as alloying agents in the manufacture of transistors, lasers, and semi-conductors, as well as in the processing of glass, pigments, textiles, paper, metal adhesives, ceramics, wood preservatives, ammunition, and explosives. They are also used in the hide tanning process and, to a limited extent, as pesticides, feed additives, and pharmaceuticals, including veterinary drugs.

The principal sources of arsenic in ambient air are the burning of fossil fuels (especially coal), metal production, agricultural use, and waste incineration. Arsenic is introduced into water through the erosion and weathering of soil, minerals, and ores, from industrial effluents, and via atmospheric deposition (Hindmarsh and McCurdy, 1986; Hutton and Symon, 1986).

In surface water, arsenite (+3 valence) and arsenate (+5 valence) form insoluble salts with cations (usually iron) that are dissolved or suspended in the water. These particles generally settle out in sediments. This cleansing process occurs to a lesser extent in deep groundwater because of higher pH levels and lower iron concentrations (Hindmarsh and McCurdy, 1986).

Arsenic occurs in different forms (organic vs. inorganic) and valences depending upon the pH and oxidation potential of the water. In well-oxygenated surface waters, pentavalent

arsenic (arsenate) is generally the most common species present (Irgolic, 1982; Cui and Liu, 1988); under reducing conditions, such as those often found in deep lake sediments or groundwaters, the trivalent species (arsenite) is the predominant form (Lemmo *et al.*, 1983; Welch *et al.*, 1988).

4.0 Exposure

4.1 Water

Levels of arsenic are generally higher in groundwater sources than in surface water. Monitoring data for water supplies have been submitted by several Canadian provinces. Arsenic levels ranged from 0.1 to 26.0 µg/L in groundwater supplies in Prince Edward Island between 1986 and 2002; levels in 96% of samples were less than or equal to 5 µg/L, the average being approximately 1.5 µg/L (Prince Edward Island Department of Fisheries, Aquaculture and Environment, 2003).

In Quebec, arsenic levels ranged from 1.0 to 25.0 µg/L in municipal treated surface water in 523 communities for 1990–2002; levels in more than 99% of samples were less than and equal to 5 µg/L, the annual average being 1.6 µg/L. Levels ranged from 1.0 to 60 µg/L in municipal treated groundwater in 562 communities for the same period; approximately 95% of samples contained levels less than or equal to 5 µg/L, the annual average being 2.0 µg/L (Ministère de l'Environnement du Québec, 2003).

In Ontario, arsenic levels ranged from 0.1 to 18 µg/L in treated groundwater and surface water in 726 communities for 1997–2002. Levels in approximately 98% of samples were less than or equal to 5 µg/L, the annual average being less than or equal to 0.7 µg/L (Ontario Ministry of Environment and Energy, 2003). Ontario monitoring data submitted by private laboratories indicated that arsenic levels in treated and raw drinking water ranged from less than 2.5 to 68 µg/L for the period 1999–2002, the average value being less than 2.5 µg/L. The higher values came predominantly from wells (Ontario Ministry of Environment and Energy, 2003).

In Saskatchewan, arsenic levels ranged from 0.5 to 105.0 µg/L in municipal treated water supplies in 539 communities between 1976 and 2002; concentrations in approximately 87% of samples were less than or equal to 5 µg/L, the average being 3.0 µg/L (Saskatchewan Department of Environment and Resource Management, 2003). Arsenic levels ranged from 0.1 to 1000 µg/L in treated groundwater and surface water in 573 Alberta communities for 1980–2002. Approximately 96% of samples contained less than 5 µg/L, the annual average being 1.8 µg/L (Alberta Department of Environment, 2003).

Elevated arsenic concentrations have been reported in areas with natural sources. Levels exceeded 50 µg/L in 33–93% of wells in each of seven communities in Nova Scotia; concentrations were greater than 500 µg/L in 10% of the wells sampled (n = 94) (Méranger *et al.*, 1984). In Newfoundland, maximum arsenic levels ranged from 6 to 288 µg/L in public water supplies (54 wells) in 2002. Public schools (n = 16) with their own water supplies had levels ranging from 1 to 368 µg/L; approximately 69% of school wells had maximum levels below or equal to 5 µg/L (Newfoundland Department of Environment and Labour, 2003). In British Columbia, a maximum arsenic concentration of 580 µg/L was reported in groundwater samples taken on Bowen Island (Boyle *et al.*, 1998).

Some western U.S. states with volcanic rock and sulphidic mineral deposits have arsenic levels in groundwater exceeding 3 mg/L (ATSDR, 2000). Elevated levels of arsenic in drinking water from wells in the northern provinces of Argentina have been reported; in Médanos in Buenos Aires Province and La Francia in Córdoba, concentrations as high as 2 mg/L and 12 mg/L, respectively, have been reported. Levels exceeded 1 mg/L at several other locations (Grinspan and Biagini, 1985).

On the basis of results indicating that the concentration of arsenic in drinking water in areas without natural sources is usually less than 5 µg/L and assuming that the average daily intake of drinking water is 1.5 L, the mean daily intake of arsenic from this source (in the predominantly pentavalent inorganic form) for an adult will generally be less than 7.5 µg. A child (0.5–4.0 years) with an average daily intake of 0.7 L of drinking water would consume less than 3.5 µg.

4.2 Food

Food is generally considered the major source of arsenic exposure except in situations where a population is living near a point source (natural geological source or site of contamination). However, it is difficult to compare the intake of arsenic in food and drinking water because of the different forms (organic vs. inorganic) that can occur in these sources.

Arsenic is concentrated by many species of fish and shellfish and is used as a feed additive for poultry and livestock; fish and meat are therefore the main sources of dietary intake (78.9%, according to a U.S. survey) (Gartrell *et al.*, 1986). A 1997 British total diet study found that seafood contributed 94% of the total arsenic intake for the general population (U.K. MAFF, 1999). In Canada, arsenic levels ranging from 0.4 to 118 mg/kg have been reported in marine fish sold for human consumption, whereas concentrations in meat and poultry range up to 0.44 mg/kg (Department of National Health and Welfare, 1983). While organic arsenic compounds (e.g., arsenocholine and arsenobetaine) found in most seaweed and other marine foods have been determined to be relatively non-toxic (Sabbioni *et al.*, 1991), toxic inorganic forms have been found in hijiki seaweed (CFIA, 2001). Levels in vegetation are generally an order of magnitude lower than those in fish, whereas concentrations in shellfish are often far higher than those in fish (Subramanian, 1988). Exogenous sources of arsenic in the diet potentially include arsenic-containing fungicides used in fruit production. In North America, however, arsenic-containing pesticides are no longer used on food (ATSDR, 2000; PMRA, 2003).

Recent estimates of the mean daily intake of total arsenic in food for adults are as follows: 42 µg (range 22.5–78.7 µg) for adults 20–65+ years old in Canada (Dabeka *et al.*, 1993), 56 µg (range 27.5–92.1 µg) for adults 25–70+ years old in the United States (Tao and Bolger, 1998), 120 µg in the United Kingdom (U.K. MAFF, 1999), 150 µg in New Zealand (Vannoort *et al.*, 1995), 286 µg in Spain (Urieta *et al.*, 1996), and 182 µg in Japan (Mohri *et al.*, 1990).

In children aged 1–4 and 5–11 years, mean daily intakes of total arsenic in food from six Canadian cities have been reported to be 14.9 µg (range 11.4–18.1 µg) and 29.9 µg (range 25.5–39.7 µg), respectively (Dabeka *et al.*, 1993). Daily intakes of 2.15 µg, 23.4 µg, 20.3 µg, and 13.3 µg have been reported for children aged 6–11 months, 2 years, 6 years, and 10 years, respectively, in the United States (Tao and Bolger, 1998).

4.3 Air

Ambient levels of arsenic in air in 11 Canadian cities and one rural site for the period 1985–1990 ranged from <0.0005 to 0.017 µg/m^3 (24-hour average), the mean for cities being 0.001 µg/m^3 (Dann, 1990). Higher atmospheric concentrations are normally found near metal smelters. In Yellowknife in 1997, concentrations ranged from 0.002 to 0.063 µg/m^3, with an annual average of 0.005 µg/m^3 (Government of the Northwest Territories, 1998).

In the United States, average annual arsenic concentrations in air have been reported to be 0.4 ng/m^3 in rural areas remote from smelting activities, 3 ng/m^3 for all locations, and 30 ng/m^3 in areas within 80 km of non-ferrous smelters (Ball *et al.*, 1983). Concentrations of arsenic in indoor air in the presence of environmental tobacco smoke (ETS) ranged from <0.1 to 1

ng/m^3, while sites that were ETS-free had concentrations below 0.13 ng/m^3 (Landsberger and Wu, 1995).

Based on the Canadian (0.001 µg/m^3) and U.S. (0.003 µg/m^3) ambient air levels, intake of arsenic through inhalation (principally in the inorganic form) is likely to be negligible (<0.1 µg, assuming 16.2 m^3 of air inhaled per day) for adults compared with the amount ingested (mainly in the organic form). Intake of arsenic (inorganic) for a child (1–4 years) based on the same ambient levels and an inhalation rate of 5 m^3 air/day would be less than 0.05 µg (Health Canada, 1998).

4.4 Soil

Arsenic in soil (predominantly inorganic) originates from underlying materials that form soils, industrial wastes, or the use of arsenical pesticides. In Canada, average concentrations of arsenic in soil range from 4.8 to 13.6 mg/kg (Kabata-Pendias and Pendias, 1984). U.S. background levels are similar, ranging from 1 to 40 mg/kg, with a mean of approximately 5 mg/kg (ATSDR, 2000). Significantly higher concentrations in soil have been found near smelters (means of 50–100 mg/kg), near gold mining operations (means of 60–110 mg/kg), in arsenical-treated soils (means up to 54 mg/kg), and at wood preservation sites (means up to 6000 mg/kg) (Environment Canada and Health and Welfare Canada, 1993). Generally, exposure from soil is potentially significant only in those circumstances where residential neighbourhood areas have been built in contaminated sectors.

While exposure to arsenic via soil is unlikely to be a concern for older children and adults, hand-to-mouth behaviour and intentional ingestion may result in significant exposure for young children. In unexposed and exposed populations, young children (≤4 years) were estimated to be exposed to 0.02–0.05 and 0.01–1.9 µg/kg bw per day, respectively (Environment Canada and Health and Welfare Canada, 1993).

4.5 Relative Contribution of Drinking Water to Total Exposure

It is difficult to compare the intake of arsenic from food with that from drinking water, as the form, valence, and biological availability of arsenic in these two sources vary. For example, a major portion of the organic arsenic in fish is present as highly complexed forms that are biologically unavailable (e.g., arsenobetaine) (Vahter *et al.*, 1983; JECFA, 1988). The remainder is present largely as simple organic complexes, mainly trimethyl arsine, which are rapidly excreted from the body. Seafood contributes much of the daily arsenic intake, even where the consumption of fish is low (Hazell, 1985). On the basis of data on the organic and inorganic arsenic contents of various foodstuffs (Hazell, 1985; U.S. EPA, 1988), it can be estimated that approximately 25% of the intake of arsenic from food is inorganic and 75% is organic. Assuming that the average daily intake of arsenic from food is 42 µg, the daily intake of inorganic arsenic from food would be 10.5 µg. This contrasts with an intake of <7.5 µg of principally the pentavalent inorganic arsenic species in drinking water. Intake of inorganic arsenic for a child (1–4 years) based on an average daily intake of total arsenic from food of 14.9 µg would be approximately 3.7 µg, which is similar to the intake from drinking water for this age group (<3.5 µg).

Based on the above estimates for a typical population, the exposure media may be ranked in the following order of importance in terms of contributing to arsenic intake: food, drinking water, soil, and air. In a situation where a population is living near a point source (natural geological source or site of contamination), drinking water has been calculated to be the most important contributor to overall exposure (Environment Canada and Health and Welfare Canada, 1993).

5.0 Analytical Methods

The U.S. Environmental Protection Agency (EPA) has approved several analytical methods, based on spectroscopy, for the analysis of total arsenic in drinking water. Total arsenic is defined as the concentration of arsenic present in the dissolved and suspended fractions of a water sample. In these methodologies, the arsenic is oxidized and analysed without regard to its chemical form (inorganic or organic) or oxidation state (i.e., As(III) or As(V)).

Atomic absorption via gaseous hydride formation is considered to be the most suitable method for the determination of arsenic in water, with a detection limit of about 0.001 mg/L (1 µg/L); the practical quantitation limit (PQL) of this method, based on the capability of laboratories to measure arsenic within reasonable limits of precision and accuracy, is 0.005 mg/L (5 µg/L) (U.S. EPA, 1999).

Other methods, such as graphite furnace atomic absorption spectroscopy in combination with high-pressure liquid chromatography (detection limit 0.01 ng; Irgolic, 1982), selective ion monitoring with inductively coupled plasma mass spectroscopy (detection limit 0.1 µg/L; U.S. EPA, 1994), and stabilized temperature platform graphite furnace atomic absorption (detection limit 0.1 µg/L; U.S. EPA, 1994), may also be used, but they have limitations and may not be useful for routine monitoring.

6.0 Treatment Technology

In water in the pH range of 4–10, the predominant As(III) species are neutral in charge, while As(V) species are negatively charged. The neutral charge on As(III) makes its removal efficiency poor in comparison with that of As(V) (U.S. EPA, 2001a).

As(III) can be converted to As(V) using a pre-oxidation step. Chlorine, ferric chloride, potassium permanganate, ozone, and hydrogen peroxide are effective at oxidizing As(III) to As(V). However, pre-oxidation with chlorine may create undesirable concentrations of chlorinated disinfection by-products (U.S. EPA, 2000).

The selection of an appropriate treatment process for a specific water supply will depend on the characteristics of the raw water supply and many other factors. It is important to determine what, if any, pretreatment is required. Pretreatment may be necessary to remove competing ions such as iron, sulphate, and silicate, as well as total dissolved solids; to adjust the pH; and to oxidize As(III) to As(V). Pretreatment is critical for ensuring arsenic removal efficacy with any subsequent treatment technology. Most treatment technology is used in combination with pretreatment and a polishing step, which typically involves polishing the finished water with ion exchange to remove the resulting negative As(V) ion. In addition, contact time, system maintenance, and cost effectiveness are key considerations when selecting a treatment process for arsenic removal. An in-depth review of the various treatment technologies used to remove arsenic from drinking water is beyond the scope of this document. However, detailed information on the effectiveness and application of the various treatment technologies for arsenic removal is available in a review by Thirunavukkarasu and Viraraghavan (2003).

6.1 Municipal-scale Treatment Technologies

The most practical municipal-scale technologies for the removal of arsenic from drinking water include coagulation/filtration, lime softening, activated alumina, ion exchange, reverse osmosis, and manganese greensand filtration. The U.S. EPA has also identified electrodialysis reversal as a best available technology for arsenic removal. Removal efficiency can be very good (>90%) for some of these technologies; however, manganese greensand filtration and electrodialysis reversal usually achieve lower removal rates (U.S. EPA, 2001a). Recently, adsorption/filtration has also shown promise for arsenic removal.

Although it is difficult to achieve low levels of arsenic using coagulation/filtration alone, when coagulation/filtration is combined with pretreatment (oxidation to convert arsenic to its pentavalent form) and a polishing step (polishing the finished water with ion exchange), the process can reduce total arsenic levels in finished drinking water to concentrations as low as 0.003–0.005 mg/L (U.S. EPA, 2000).

Lime softening is widely used in large utilities and is effective at reducing total arsenic in drinking water to concentrations of 0.001–0.003 mg/L. However, lime softening is an expensive process and is not recommended unless there is also a need to reduce hardness in the raw water feed (U.S. EPA, 2000). The performance and consistency of lime softening can be improved by pretreating the raw water using oxidation and polishing the finished water with ion exchange.

Currently, the most common arsenic removal process for municipal-scale treatment uses activated alumina adsorption followed by microfiltration. Several studies have demonstrated that activated alumina is an effective treatment for the removal of arsenic (As(V)) from drinking water. Pilot plant studies of arsenic removal using activated alumina achieved effluent arsenic levels of <0.01 mg/L (Simms and Azizian, 1997). The U.S. EPA has identified activated alumina as a best available technology for arsenic removal, with a removal efficiency of 95% (U.S. EPA, 2001a). However, the chemical handling requirements may make this process too complex and potentially dangerous for smaller utilities (U.S. EPA, 2000), and therefore this treatment process is not commonly used for these smaller utilities.

The treatment processes described above are effective, but relatively expensive to build and maintain on a municipal scale, and they may not be appropriate for small water treatment utilities. These systems also create significant quantities of either sludge or brine, which must be disposed of appropriately, thus increasing the cost of these processes (NDWAC, 2001).

Ion exchange processes in combination with an oxidation pretreatment step have been shown to reduce total arsenic in finished drinking water to levels as low as 0.003 mg/L. Laboratory column studies using ion exchange resin achieved effluent concentrations as low as 0.002 mg/L where the influent had an arsenic concentration of 0.021 mg/L (Clifford *et al*., 1999). These systems are recommended for water supplies with low concentrations of total dissolved solids and sulphate (U.S. EPA, 2000).

Reverse osmosis systems, when combined with a pretreatment step, can remove up to 85% of total arsenic from drinking water. These systems are reliable but require large quantities of influent water to obtain the required volume of drinking water, as they reject a significant portion of the influent water as an arsenic-rich brine; as such, they may not be suitable for use in areas where water resources are scarce (U.S. EPA, 2000).

In manganese greensand filtration, the arsenic contained in the water passing through the filter is oxidized and then trapped in the filter. This technology does not achieve a high removal efficiency and is dependent on the presence of iron in the water to remove arsenic. This technology may be appropriate were the source water has a high iron level and requires only little arsenic removal (U.S. EPA,2000).

Adsorption/filtration appears to be a promising technology that is applicable to small water treatment utilities. Adsorption using media such as iron, aluminum, and titanium oxide is effective at removing arsenic. Fixed-bed treatment systems, such as adsorption and ion exchange, are becoming increasingly popular for arsenic removal in small water treatment systems because of their simplicity, ease of operation and handling, and regeneration capacity. Several studies that tested the removal of arsenic from drinking water under both laboratory- and pilot-scale conditions showed that adsorptive materials containing various iron oxides are capable of removing As(III) and As(V). More specifically, iron oxide-coated sand and granular ferric hydroxide can remove As(III) and As(V) present in the water to a concentration below 0.005 mg/L (Pierce and Moore, 1980, 1982; Fuller *et al*., 1993; Hsia *et al*., 1994; Wilkie and

Hering, 1996; Raven *et al*., 1998; Driehaus *et al*., 1998; Ramaswami *et al*., 2001; Thirunavukkarasu *et al*., 2001, 2003a,b).

6.2 Residential-scale Treatment Options

In cases where an individual household obtains its drinking water from a private well, a private residential drinking water treatment device (treatment device) may be an option for reducing arsenic concentrations in drinking water. Residential treatment devices are affordable and can remove arsenic from drinking water to concentrations below 0.010 mg/L. Periodic testing by an accredited laboratory should be conducted on both the water entering a treatment device and the water it produces to verify that the device is effective.

The most common types of treatment devices available for the removal of arsenic from drinking water in residential systems are reverse osmosis and steam distillation. Other types of systems based on alternative technologies such as adsorption are also becoming more common. Filtration systems may be installed at the faucet (point of use) or where water enters the home (point of entry).

Before a treatment device is installed, the well water should be tested to determine general water chemistry and to verify the type and concentration of arsenic in the water. Pretreatment with an oxidation step may be required to convert trivalent (dissolved) arsenic to pentavalent (filterable) arsenic and ensure good removal by the treatment device. Individuals should refer to the manufacturer's claims in its literature to obtain information on the amount of arsenic the treatment device will remove, as well as operational and maintenance requirements.

Residential reverse osmosis systems have been shown to effectively remove total arsenic from drinking water. The amount of arsenic removed depends on the type of membrane filter employed in the system. Reverse osmosis requires larger quantities of influent (incoming) water to obtain the required volume of drinking water, as reverse osmosis systems reject (waste) part of the influent water. A consumer may need to pretreat the influent water to reduce fouling and extend the service life of the membrane. The major advantage of using reverse osmosis systems is that they are widely available, affordable, and easy to service and can remove up to 98% of other dissolved minerals as well as fine colloidal and coarse suspended matter (U.S. EPA, 2000).

Distillation systems can remove virtually all arsenic in drinking water. These systems are more complex than reverse osmosis systems. Although distillation systems are usually installed in commercial applications, more systems are becoming available for residential applications. It should be noted that while there are no known harmful health effects associated with the long-term ingestion of drinking water from distillation or reverse osmosis systems, no specific studies have been conducted on the effects of ingestion of water from these systems. Since beneficial minerals such as calcium and magnesium are removed by both distillation and reverse osmosis processes, it is important to consume a reasonably well-balanced diet to offset the removal of these minerals.

Adsorption/filtration appears to be a promising technology that is applicable to residential-scale treatment. Adsorption using media such as iron, aluminum, and titanium oxide is effective at removing arsenic. Fixed-bed treatment systems, such as adsorption and ion exchange, are becoming increasingly popular for arsenic removal in small water treatment systems because of their simplicity, ease of operation and handling, and regeneration capacity.

Health Canada does not recommend specific brands of treatment devices, but it strongly recommends that consumers use devices that have been certified by an accredited certification body as meeting the appropriate NSF International (NSF)/American National Standards Institute (ANSI) drinking water treatment unit standards. These standards have been designed to safeguard drinking water by helping to ensure material safety and performance of products that come into contact with drinking water. Certification organizations provide assurance that a

product or service conforms to applicable standards. In Canada, a number of organizations have been accredited by the Standards Council of Canada to certify treatment devices as meeting the above-mentioned NSF/ANSI standards (SCC, 2003).

It must be noted, however, that the NSF/ANSI standards for arsenic removal currently test for removal to a concentration of 0.01 mg/L. Certified devices are frequently designed to remove arsenic to well below the 0.01 mg/L concentration, but certification to the standard verifies only that a final concentration of less than 0.01 mg/L is achieved. A qualified professional can design a system to meet residential needs and achieve arsenic concentrations below 0.005 mg/L. For example, a system designed with two or more filters in series will often result in removal of virtually all arsenic. As stated above, the selection of an appropriate treatment process for a specific water supply will depend on the characteristics of the raw water supply and many other factors. It is important to determine what, if any, pretreatment is required and to have the finished water tested by an accredited laboratory to ensure that any designed system is attaining the desired arsenic removal.

For a drinking water treatment device to be certified to NSF/ANSI Standards 53 (Drinking Water Treatment Units — Health Effects), 58 (Reverse Osmosis Drinking Water Treatment Systems), or 62 (Drinking Water Distillation Systems), the unit will have to be able to reduce the concentration of arsenic in water from 0.3 to 0.01 mg/L. Only units that can be certified as reducing the concentration of arsenic from 0.3 to 0.01 mg/L are appropriate for treating well water. Devices certified as reducing the concentration of arsenic from 0.05 to 0.01 mg/L are intended specifically for treating water previously treated by municipal facilities.

7.0 Kinetics and Metabolism

7.1 Essentiality

Although the results of available studies indicate that arsenic may be an essential element for several animal species (e.g., goats, minipigs, rats, chicks), there is no evidence that it is essential for humans. A Technical Panel on Arsenic convened by the U.S. EPA was "not aware of case reports describing an arsenic requirement for humans, nor of experimental or epidemiologic-type studies designed to determine whether arsenic is essential." After reviewing the available data, the Technical Panel concluded that "if arsenic is a required nutrient for humans, current environmental arsenic exposures are not known to produce human arsenic deficiency" (U.S. EPA, 1988).

7.2 Absorption, Distribution, Metabolism, and Elimination

Ingested elemental arsenic is poorly absorbed and largely eliminated unchanged. Arsenic oxides are readily absorbed (>80%) from the gastrointestinal tract (Fowler *et al.*, 1979) and, to a lesser extent, through the lungs and skin (Wickström, 1972). On the basis of faecal recovery experiments in human volunteers, soluble As(III) and As(V) and organic arsenic are well absorbed; As(III) tends to accumulate in tissues, but As(V) and organic arsenic are rapidly and almost completely eliminated via the kidneys (Bertolero *et al.*, 1987). Both organic and inorganic arsenic are not well absorbed by the skin. Dermal exposure is reported to be of minor importance compared with ingestion. The National Research Council (U.S. NRC, 1999) and the Agency for Toxic Substances and Disease Registry (ATSDR, 2000) reviewed the available information on dermal absorption of arsenic and indicated that systemic absorption of arsenic via the skin is sufficiently low that this route of exposure is unlikely to be of concern to health.

Following ingestion, inorganic arsenic appears rapidly in the circulation, where it binds primarily to haemoglobin (Axelson, 1980); within 24 hours, it is found mainly in the liver, kidneys, lungs, spleen, and skin (Wickström, 1972). Skin, bone, and muscle represent the major

storage organs. The accumulation of arsenic in skin is probably related to the abundance of proteins containing sulphydryl groups, with which arsenic readily reacts (Fowler *et al.*, 1979). In humans, inorganic arsenic does not appear to cross the blood–brain barrier; however, transplacental transfer of arsenic in both humans (Gibson and Gage, 1982) and mice (Hood *et al.*, 1987) has been reported.

Pathways for the conversion of one form of arsenic to another have been proposed (U.S. NRC, 2001). Methylation of inorganic arsenic is thought to occur following the reduction of pentavalent arsenic to trivalent arsenic. Methylation of this trivalent form of arsenic is then believed to result from the oxidative addition of a methyl group from S-adenosylmethionine by a methyl transferase. Sequential reduction and methylation of arsenic compounds result in the creation of pentavalent monomethylarsinic acid (MMA^V) and dimethylarsinic acid (DMA^V), as well as the trivalent monomethylarsinous acid (MMA^{III}) and dimethylarsinous acid (DMA^{III}) (U.S. NRC, 2001).

There appear to be two main processes, with different rates, for the elimination of ingested trivalent arsenic (As(III)) from the body (Lovell and Farmer, 1985). The first is the rapid urinary excretion of inorganic arsenic in both the trivalent and pentavalent forms (close to 90% of the total urinary arsenic over the first 12-hour period). The second involves the sequential methylation of As(III) in the liver to the organic forms MMA^{III}, DMA^{III}, MMA^V, and DMA^V (Buchet and Lauwerys, 1985; Lovell and Farmer, 1985). Excretion of the methylated compounds commences approximately 5 hours after ingestion but reaches its maximum level 2–3 days later. Less important routes of elimination of inorganic arsenic include skin, hair, nails, and sweat (ICRP, 1975; Kurttio *et al.*, 1999). The half-life of inorganic arsenic in humans is estimated to be between 2 and 40 days (Pomroy *et al.*, 1980).

The results of a study in which inorganic arsenic (125, 250, 500, or 1000 µg $NaAsO_2$) was administered orally once a day for 5 consecutive days to four volunteers indicate that the arsenic methylation capacity is progressively saturated when daily intake exceeds 0.5 mg (Buchet *et al*., 1981a); it does not, however, appear to be completely saturated even for daily doses as high as 1 mg. Studies with human volunteers indicate that most ingested organic arsenic is rapidly excreted unchanged (>80% of the dose within 4 days) (Buchet *et al.*, 1981b; Luten *et al.*, 1982; Tam *et al.*, 1982).

8.0 Health Effects

8.1 Effects in Humans

The acute toxicity of the various forms and valences of arsenic in humans is predominantly a function of their rate of removal from the body. Metallic arsenic (0 valence) is not absorbed from the stomach and as such does not have any adverse effect. Some arsenic compounds, such as the volatile arsenine (AsH_3), are not present in food or water. Additionally some organic arsenic compounds have little or no toxicity or are rapidly eliminated from the body in the urine. Lethal doses for the most common arsenic compounds (AsH_3, As_2O_3, As_2O_5, MMA^V, and DMA^V) in humans range from 1.5 mg/kg bw (As_2O_3) to 500 mg/kg bw (DMA^V) (Buchet and Lauwerys, 1982). AsH_3, As_2O_3, and As_2O_5 are gaseous forms of arsenic found in air, and MMA and DMA are organic forms of arsenic found in water.

Symptoms of acute arsenic intoxication associated with the ingestion of well water containing arsenic at 1.2 and 21.0 mg/L have been reported (Feinglass, 1973; Wagner *et al.*, 1979). Early clinical symptoms of acute arsenic intoxication include abdominal pain and vomiting, diarrhoea, pain to the extremities and muscles, and weakness with flushing of the skin. These symptoms are often followed by numbness and tingling of the extremities, muscular cramping, and the appearance of a papular erythematous rash 2 weeks later (Murphy *et al.*,

1981). A month later, symptoms may include burning paraesthesias of the extremities, palmoplantar hyperkeratosis, Mee's lines on fingernails, and progressive deterioration in motor and sensory responses (Fennell and Stacy, 1981; Murphy *et al.*, 1981; Wesbey and Kunis, 1981).

Signs of chronic arsenicalism, including pigmentation and development of keratoses, peripheral neuropathy, skin cancer, peripheral vascular disease, hypertensive heart disease, cancers of internal organs (bladder, kidney, liver, and lung), alterations in gastrointestinal function (non-cirrhotic hypertension), and an increased risk of mortality resulting from diabetes, have been observed in populations ingesting arsenic-contaminated drinking water in southwestern Taiwan (Chen *et al.*, 1985, 1992; Wu *et al.*, 1989), Bangladesh (Smith *et al.*, 2000), Chile (Borgono and Greiber, 1971; Zaldívar, 1980; Zaldívar and Ghai, 1980), India (Mandal *et al.*, 1998), the United States (Valentine *et al.*, 1982; U.S. NRC, 1999; U.S. EPA, 2001a), Mexico (Cebrian *et al.*, 1983), and Canada (Hindmarsh *et al.*, 1977). Dermal lesions, such as hyperpigmentation, warts, and hyperkeratosis of the palms and soles, are the most commonly observed symptoms in 70-kg adults after 5–15 years of exposure equivalent to 700 µg/day or within 6 months to 3 years at exposures equivalent to 2800 µg/day (U.S. EPA, 2001a).[**]

Numerous adverse effects, particularly among children, have been associated with the consumption of arsenic-contaminated water in Antofagasta, Chile (mean arsenic concentration 0.6 mg/L). Effects on the skin (leukomelanoderma, hyperkeratosis), respiratory system (chronic coryza, cough, bronchopulmonary diseases), cardiovascular system (myocardial infarction, peripheral vascular disorders such as ischaemia of the tongue, Raynaud's phenomenon, acrocyanosis), and digestive system (abdominal pain, chronic diarrhoea) were observed in children under 16 years of age (Zaldívar, 1980; Zaldívar and Ghai, 1980). The prevalence of these symptoms decreased after the installation of a water treatment plant in 1972 (mean arsenic concentration 0.08 mg/L); however, prevalence rates were still higher than those of the control population (Zaldívar and Ghai, 1980). Dermal lesions in young people ingesting drinking water containing high arsenic concentrations have also been reported elsewhere (Tseng *et al.*, 1968; Cebrian *et al.*, 1983).

The largest epidemiological study on arsenic to date was conducted in a limited area of southwestern Taiwan (an area well known for its high incidence of blackfoot disease). This data set has been analysed by numerous authors (e.g., Tseng, 1977; Chen *et al.* 1985, 1992; Wu *et al.* 1989; U.S. NRC, 1999, 2001) for assessing the health effects of arsenic through ingestion of arsenic-contaminated drinking water. Tseng (1977) divided a population of 40 421 into three groups based on the arsenic content of their well water (high ≥0.60 mg/L, medium 0.30–0.59 mg/L, and low 0.01–0.29 mg/L). There was a clear dose–response relationship between exposure to arsenic and the frequency of dermal lesions, "blackfoot disease" (a severe peripheral vascular disorder) (Yu *et al.*, 1984), and skin cancer. Despite certain methodological weaknesses in this early study, it is now widely accepted that exposure to high concentrations of arsenic is a cause of peripheral vascular disease. Blackfoot disease is now sometimes used as an indicator of exposure to arsenic (U.S. EPA, 2001b).

More epidemiological evidence for an association between the incidence of various cancers of the internal organs and the ingestion of arsenic-contaminated water comes from a study conducted in a limited area of southwest Taiwan. In this study, standardized mortality

[**] For comparison, the estimated intakes per day at various drinking water exposure levels (assuming consumption of 1.5 L of water per day) and a food contribution of 49 µg/day (total organic and inorganic arsenic) are as follows: at 5 µg/L = 56.5 µg/day ((1.5 L/day × 5 µg/L) + 49 µg/day); at 10 µg/L = 64 µg/day; at 100 µg/L = 199 µg/day; and at 500 µg/L = 799 µg/day.

ratios (SMRs) for cancers of the bladder, kidney, skin, lung, liver, and colon were significantly elevated in the area of arsenic contamination. The SMRs for bladder, kidney, skin, lung, and liver cancer also correlated well with the prevalence rate for blackfoot disease (Chen *et al.*, 1986). In an additional case–control study of 69 bladder, 76 lung, and 59 liver cancer mortality cases as well as 368 community controls matched for age and sex, the odds ratios of developing bladder, lung, and liver cancers for those who had used artesian well water for 40 or more years were 3.90, 3.39, and 2.67, respectively, compared with those who had never used artesian well water. Dose–response relationships were observed for all three cancer types by duration of exposure, and the odds ratios were not changed significantly when several other risk factors were taken into consideration in logistic regression analysis (Chen *et al.*, 1986).

In an ecological analysis in which cancer mortality was examined in relation to arsenic concentrations in drinking water in the villages of the blackfoot disease-endemic areas of southwestern Taiwan, Chen *et al.* (1985) found an association between high-arsenic artesian well water (ranging from 0.35 to 1.14 mg/L; median level 0.78 mg/L) and cancers of the bladder, kidney, lung, skin, liver, and colon. Both the SMR and cumulative mortality rate were significantly higher for cancers of the bladder, kidney, lung, skin, liver, and colon compared with the general population of southwestern Taiwan. The SMRs for cancers of the bladder, kidney, skin, lung, liver, and colon were 1100, 772, 534, 320, 170, and 160, respectively, for males and 2009, 1119, 652, 413, 229, and 168, respectively, for females. A dose–response relationship was observed between the SMRs of the cancers and blackfoot disease prevalence rate of the villages and townships in the endemic areas. An additional ecological analysis of the same southwestern Taiwanese population by Wu *et al.* (1989) also found significant dose–response relationships for age-adjusted rates of cancers of the bladder, kidney, skin, and lung in both sexes and cancers of the prostate and liver in men (the total number of cancers at each site were 181 cancers of the bladder in both sexes, 59 cancers of the kidney in both sexes, 36 cancers of the skin in both sexes, 9 cancers of the prostate in men, 123 cancers of the liver in men, and 268 lung cancers in both sexes) (Wu *et al.*, 1989). A study examining the ecological correlations between arsenic levels in well water and mortality from various malignant neoplasms in southwestern Taiwan demonstrated a significant association between the arsenic level in well water and cancers of the liver, nasal cavity, lung, skin, bladder, and kidney in both sexes and prostate cancer in men (Chen and Wang, 1990). A later reanalysis by Chen *et al.* (1992) on the same southwestern Taiwanese study population calculated cancer potency indices for liver, lung, bladder, and kidney. The study population was stratified into four groups according to the median arsenic level of well water in each village. There were 13 villages with median arsenic levels below 0.10 mg/L, eight villages with levels ranging from 0.10 to 0.29 mg/L, 15 villages with levels from 0.30 to 0.59 mg/L, and six villages with levels greater than or equal to 0.60 mg/L. The total numbers of cancer-related deaths observed during the study period were as follows: 140 male and 62 female liver cancer deaths, 169 male and 135 female lung cancer deaths, 97 male and 105 female bladder cancer deaths, and 30 male and 34 female kidney cancer deaths. Mortality rates were found to increase significantly with age for all cancers in both males and females. Significant dose–response relationships were observed between the ingested arsenic level and mortality from cancer of the liver, lung, bladder, and kidney in most age groups of both males and females.

Two more recent studies provide further support to the increased incidence of lung and bladder cancers associated with arsenic exposure. Both of these more recent studies differed from the southwestern Taiwan ones (Chen *et al.*, 1985; Wu *et al.*, 1989), in that they examined the risk factors for newly diagnosed cases of bladder cancer (Chiou *et al*., 2001) and lung cancer (Ferreccio *et al.*, 2000) rather than deaths. The study by Chiou *et al.* (2001) established a significant dose–response relationship between risk of urinary cancers and arsenic exposure after

adjustment for age, sex, and cigarette smoking. This work was, however, limited in terms of its size. Ferreccio *et al.* (2000) revealed a clear association between the odds ratios for lung cancer and concentrations of arsenic in drinking water. While this work further supports the association of cancer with arsenic in drinking water, it has been deemed limited because of control selection methods used (U.S. NRC, 2001).

In a study conducted in Mexico, the health status of the populations of two rural towns with average arsenic concentrations of 0.41 ± 0.114 mg/L ("exposed") and 0.005 ± 0.007 mg/L ("control") in their water supplies was examined (Cebrian *et al.*, 1983). The prevalence of non-specific symptoms, such as nausea, epigastric pain, colic abdominal pain, diarrhoea, headache, and oedema, was significantly higher in the "exposed" population; the relative risks for these various symptoms ranged from 1.9 to 4.8. The relative risk of developing cutaneous lesions ranged from 3.6 to 36. In this study, only 9.6% of the individuals with skin lesions were under 20 years of age. The prevalence of skin cancer (including the precancerous lesion papular keratosis and ulcerative lesions) in the "exposed" population in Mexico was 6.4% compared with 1.06% in the population with similar exposure in southwestern Taiwan (0.30–0.59 mg/L group) (Tseng, 1977). This variation could be due, in part, to the differences in the proportion of the various forms of arsenic salts ingested: 70% As(V) in the Mexican study compared with 89% As(V) in the southwestern Taiwanese study. The Mexican study suffered from methodological weaknesses; for example, the investigators were not blinded, and drinking water was assumed to be the only source of arsenic.

In a case–control study of 270 children with congenital heart disease and 665 healthy children, maternal consumption of drinking water containing detectable arsenic concentrations during pregnancy was associated with a threefold increase in the occurrence of coarction of the aorta. The prevalence odds ratio adjusted for all measured contaminants and source of drinking water was 3.4, with a 95% confidence interval of 1.3–8.9 (Zierler *et al.*, 1988). However, there was no adjustment for maternal age, socioeconomic status, or previous reproductive history. Exposure was determined by matching the results of available water analyses for the water supplies serving the mothers to their dates of conception. However, for 101 of the mothers residing in communities served by multiple water supplies, it was necessary to average contaminant concentrations from more than one source in the community; the mean interval from the date of analysis to date of conception for the entire study population was 227 days.

In a case–control study in Massachusetts of 286 women with spontaneous abortions and 1391 women with live births, elevated odds ratios for miscarriages were associated with exposure to arsenic in drinking water (Aschengrau *et al.*, 1989). The odds ratios for spontaneous abortion adjusted for maternal age, educational level, and history of prior spontaneous abortion for women exposed to arsenic in their drinking water at undetectable concentrations, 0.0008–0.0013 mg/L, and 0.0014–0.0019 mg/L were 1.0, 1.1, and 1.5, respectively. Exposure was determined by matching each woman to the results of a tap water sample taken in her city or town during pregnancy. However, the median interval from the date of matched metal analysis sample to the date of conception was 2.1 years, and it was reported that the variability of concentrations of metals in 20 Massachusetts towns and cities over the 7-year period between 1978 and 1985 was 10- to 100-fold. It would be desirable, however, to follow up these preliminary results in studies designed to more accurately assess exposure.

Although some effects have been observed in children and pregnant women, the U.S. NRC concluded that "there was insufficient scientific information to permit separate cancer risk estimates for potential subpopulations such as pregnant women, lactating women and children and that factors that influence sensitivity to or expression of arsenic-associated cancer and non-cancer effects need to be better characterized" (U.S. EPA, 2001a).

8.2 Effects in Experimental Animals and *In Vitro*

Arsenic presents unique problems for quantitative risk assessment because there is no test animal species for studying carcinogenicity. It appears that test animals do not respond to inorganic arsenic exposure in a way that makes them useful as a model for human cancer assessment. Their metabolism of inorganic arsenic is also quantitatively different than that by humans (U.S. EPA, 2001a).

The specific form or valence of arsenic that is responsible for teratogenesis in animals is not known, although there is evidence to suggest that it is arsenite (As(III)) rather than arsenate (As(V)) (Hanlon and Ferm, 1986b).

There were significant reductions in cardiac output and stroke volume in male Wistar rats and female New Zealand rabbits ingesting drinking water containing 50 µg As(III)/mL for 18 and 10 months, respectively. In contrast, there was no effect on cardiac function in rats following ingestion of the same concentration of As(V) for 18 months (Carmignani *et al.*, 1985).

Shirachi *et al.* (1986) investigated the potential of arsenic compounds — DMA (valence not specified), MMA (valence not specified), As(III), and As(V) — to act as promoters or initiators. Male Wistar rats were partially hepatectomized, injected intraperitoneally with a single dose of diethylnitrosamine (30 mg/kg bw), and, on day 7, administered the maximum tolerated doses (MTDs) of each of the arsenic compounds in drinking water for 7, 25, or 43 weeks (promotion protocol). The protocol for determination of initiation was similar but did not include diethylnitrosamine treatment. The initiation potential of arsenate and arsenite was also investigated in a study in which partially hepatectomized rats were exposed 18–24 hours later to 160 mg/L (As(III)) or 320 mg/L (As(V)) in drinking water for 3 days and then fed food pellets containing 0.05% phenobarbital starting 4 days later for 7 weeks. There were no significant increases in the incidence of tumours of the liver in any of the arsenic-exposed rats for any periods of treatment in either the initiation or promotion protocols. In the promotion protocol, however, there was a significant increase in the incidence of tumours of the kidney in the group exposed to the MTD of As(III) (160 mg/L) for 25 weeks (Shirachi *et al.*, 1986). Based on the results of their studies, the authors also concluded that the chronic toxicity of arsenic compounds in drinking water cannot be predicted from acute toxicity studies, because DMA was as "toxic" as arsenate and arsenite.

Other studies have shown carcinogenic effects in mice and rats (IPCS, 2001), although evidence of arsenic-induced cancer in animals is mostly negative (ATSDR, 2000). An extensive review of animal models of arsenic carcinogenicity is presented in Wang *et al.* (2002).

Arsenic has been known to induce chromosome breakage, chromosomal aberrations, and sister chromatid exchange in a linear, dose-dependent fashion in a variety of cultured cell types, including human cells (Jacobson-Kram and Montalbano, 1985; U.S. EPA, 1988). Most of the chromosomal aberrations are lethal events, so that the cells do not survive more than one or two generations (U.S. EPA, 1988). Trivalent arsenic is approximately an order of magnitude more potent than As(V) in this respect. The clastogenic effect of arsenic appears to be due to interference with DNA synthesis, as arsenic induces sister chromatid exchange and chromosomal aberrations only when present during DNA replication (Crossen, 1983). Arsenic has also been shown to block dividing cells in the S and G_2 phases (Petres *et al.*, 1977). Mass *et al*. (2001) demonstrated the genotoxicity of methylated trivalent arsenic metabolites *in vitro*. While the mechanism of arsenic genotoxicity remains unknown, mechanisms such as reactive oxygen species and the inhibition of DNA repair have been proposed (IPCS, 2001; WHO, 2003). Several possible modes of action for arsenic carcinogenesis, including chromosomal abnormalities, oxidative stress, altered DNA repair, altered DNA methylation patterns, altered growth factors, enhanced cell proliferation, promotion/progression, gene amplification, and suppression of p53, have been reviewed by Kitchin (2001).

In early studies, teratogenic effects of arsenic in chicks, golden hamsters, and mice were reported (Hood and Bishop, 1972; Zierler *et al*., 1988). Arsenate was found to be teratogenic in the offspring of pregnant hamsters following exposure on days 4–7 of gestation by minipump implantation (Ferm and Hanlon, 1985). The threshold blood level for teratogenesis was 4.3 µmol/kg (Hanlon and Ferm, 1986a). In studies with mice and hamsters, MMA^V and DMA^V have been considerably less teratogenic than As(III) or As(V). However, teratogenicity was not observed in mice or rabbits upon oral administration of arsenic acid at 48 mg/kg bw per day during gestation days 6–15 and at 0–3 mg/kg bw per day during gestation days 6–18 (Nemec *et al.*, 1998).

8.3 Relative Toxicity of Arsenic Compounds in Humans

While earlier studies reported organic forms of arsenic (MMA^V, DMA^V, MMA^{III}, and DMA^{III}) to be less toxic than their inorganic counterparts (i.e., As(III) and As(V)) (U.S. NRC, 1999), recent evidence suggests that this is not entirely the case. In humans, MMA^V and DMA^V, as well as MMA^{III} and DMA^{III}, result from the sequential reduction and methylation of inorganic arsenic by the liver (Buchet and Lauwerys, 1985; Lovell and Farmer, 1985). Inorganic arsenic that is not immediately removed from the body undergoes these reduction and methylation steps in the liver. Early tests measuring the toxicity of organic arsenic measured pentavalent MMA^V and DMA^V, but did not consider trivalent MMA^{III} and DMA^{III}, as it was believed that these trivalent forms were too transient to be found in urine (U.S. NRC, 2001). However, recent isolation of MMA^{III} in urine from humans has enabled testing that suggests that, contrary to previous belief, MMA^{III} is actually more toxic to hepatocytes than MMA^V and arsenite (As(III)) (Aposhian *et al.*, 2000; Petrick *et al.*, 2000; Styblo *et al*., 2000; U.S. NRC, 2001). Work on human hepatocytes performed by Petrick *et al.* (2000) has established a relative order of toxicity: MMA^{III} > arsenite (+3) > arsenate (+5) > MMA^V = DMA^V. In the past, it was widely accepted that the toxic and carcinogenic effects of arsenic resulted from the inorganic form and that methylation represented a mechanism of detoxification of arsenic. An important implication of these findings is that methylation of inorganic arsenic is not necessarily a detoxification process, as was once believed (U.S. NRC, 2001).

It should also be noted that the form of arsenic that is responsible for influencing tumour formation is still not known. Consequently, the contribution of different forms of arsenic to the overall response cannot be established (U.S. NRC, 2001).

9.0 Classification and Assessment

Arsenic is a documented human carcinogen; it has, therefore, been classified in Group 1 (carcinogenic to humans) as defined by Health Canada and the International Agency for Research on Cancer (IARC). Toxic effects other than cancer have also been observed in populations ingesting arsenic-contaminated water supplies; however, carcinogenicity is considered to be the critical effect for derivation of the guideline.

It is important to note that while animal studies have confirmed the carcinogenicity of arsenic, significant differences concerning the toxic effects of arsenic exist between species. Hence, human studies remain the most reliable sources to be used in establishing a maximum acceptable concentration (MAC).

While early studies on the southwestern Taiwanese population indicated an association between arsenic in drinking water and cancer of internal organs (Chen *et al.*, 1985; Wu *et al.*, 1989), this information on its own was not deemed to be sufficient for quantitative risk assessment during the development of the 1989 guideline for arsenic in drinking water. As a result, the 1989 guideline was based on the increased incidence of skin cancer observed in the southwestern Taiwanese population (Tseng *et al.*, 1968) and a model devised by the U.S. EPA,

which estimated lifetime skin cancer risks associated with the ingestion of arsenic in drinking water using a multistage model modified to take into account incidence stratified by age group. This model was quadratic as well as linear in dose and included an adjustment for the larger water consumption of southwestern Taiwanese compared with North American men. Based on this model, lifetime risks of skin cancer in the general population in Canada for ingestion of 1 µg/L of arsenic in drinking water were estimated to range from 1.3×10^{-5} (based on southwestern Taiwanese women) to 3.6×10^{-5} (based on southwestern Taiwanese men).

New data have become available that suggest that the risk of internal cancers due to ingestion of arsenic in drinking water is greater than previously believed (U.S. NRC, 1999). Chen *et al.* (1992) evaluated cancer potency indices in the liver, lung, bladder, and kidney for cancers induced by the ingestion of inorganic arsenic in drinking water. A comparison of observed number of deaths and mortality rate by age, sex, and arsenic level in drinking water for these various internal cancers indicated that lung and bladder cancer presented the greatest lifetime risks for development at an arsenic level of 10 µg/kg bw per day. Morales *et al.* (2000) calculated excess lifetime risk estimates in the same population for bladder, liver, and lung cancers resulting from exposure to arsenic in drinking water using several mathematical models (generalized linear model, multistage Weibull model, and several variations of these); results for risk estimates were sensitive to the choice of model used.

In addition, a review of the health assessment concerning the toxicity of arsenic in drinking water based on human data from southwestern Taiwan indicates a positive relationship between internal organ cancers (lung, bladder, liver, and kidney) and the ingestion of arsenic in drinking water. Similar conclusions were also reported by U.S. EPA (2001a), U.S. NRC (1999, 2001), and WHO (2003). It should also be noted that, although lacking in necessary data for risk quantification, other studies support the association of arsenic in drinking water with cancers of internal organs (lung and bladder) (Kurttio *et al.*, 1999; Lewis *et al.*, 1999; Ferreccio *et al.*, 2000; Chiou *et al.*, 2001).

The southwestern Taiwan ecological study, as reported by many authors, including Wu *et al.* (1989), Chen et al. (1992), and the U.S. NRC (1999), has been recommended for quantitative risk assessment (U.S. EPA, 2001a; U.S. NRC, 2001). This study population has been chosen because it presents sufficient duration of exposure to arsenic and follow-up, extensive pathology data, homogeny between lifestyles of the population, and a fair population size (U.S. NRC, 2001). A statistical analysis by Morales *et al.* (2000) fit nine Poisson-type models and one Weibull model to this data set in estimating the risk of cancer to the bladder, liver, and lung from exposure to arsenic in drinking water. The U.S. EPA (2001a) concluded that model 1 from Morales *et al.* (2000), which did not use a comparison population, was more reliable than those models utilizing a comparison population. Models with no comparison population estimate the arsenic dose–response curve only from the study population and are therefore more reliable and biologically plausible than those models using a comparison population, which often result in supralinear curves that lack a biological basis (U.S. EPA, 2001a). On the basis of a review of the available data, Health Canada (2003) concluded that the Poisson model recommended by the U.S. EPA and fit by Morales *et al.* (2000) is the most appropriate for estimating the cancer risks associated with the ingestion of arsenic in drinking water. In the quantitative risk assessment, Health Canada (2003) adopted assumptions similar to those of the U.S. EPA regarding the decision not to use a control population, the choice of preferred model, the choice of risk metric, and the use of a southwestern Taiwanese to Canadian conversion factor. The Health Canada (2003) model analysed data from Chen *et al.* (1992), U.S. NRC (1999), and Morales *et al*. (2000); these reports sourced their data from Chen *et al.* (1985) and Wu *et al.* (1989).

Overall, the risks associated with ingestion of 1 µg/L of arsenic in drinking water are estimated to range from 4.50×10^{-6} to 2.17×10^{-5}, with 95% upper bounds ranging from $6.84 \times$

10^{-6} to 2.47×10^{-5},*** based on a 1% increase in risk. The most sensitive endpoint for both males and females was lung cancer (Health Canada, 2003). The overall unit risk associated with the ingestion of arsenic in drinking water is reported as a range, given that lifetime exposure to arsenic results in more than one cancer endpoint in different individuals. The above unit risk range has the kidney cancer unit risk in men (4.50×10^{-6}) as its low end and the lung cancer unit risk in women (2.17×10^{-5}) as its high end. This range also includes the estimated risks for cancers of bladder, liver, and other internal organs.

Epidemiological data are often reported with the 95% upper-bound value. This value quantifies the variability in the unit risk due to the variability in the data from the study population. Sources of variability in these data may be, for example, individual differences in arsenic metabolism, drinking rates, or body weights. The 95% upper bound is often interpreted as a reasonable conservative upper-bound estimate of the unit risk. In other words, in repeated trials of the experiment, 95% of the time, the 95% upper-bound value will be above the true value of the unit risk.

Based on this unit risk calculation, an acceptable concentration of arsenic in drinking water can be established that would present an "essentially negligible" level of risk. This target concentration is calculated as 0.4 µg/L, based solely on health considerations.The upper 95% confidence interval for the lifetime cancer risk associated with this concentration in drinking water is 2.7×10^{-6} to 9.9×10^{-6}, which falls within the range considered to be "essentially negligible."

10.0 Rationale

Arsenic can be found in both surface water and groundwater sources, with levels generally higher in groundwater. Most provinces and territories across Canada report some areas where arsenic can be detected in drinking water supplies. Humans are exposed to many forms of arsenic that have different toxicities. The acute toxicity of the various arsenic compounds in humans is predominantly a function of their rate of removal from the body. Metallic arsenic (0 valence) is not absorbed from the stomach and as such does not have any adverse effect. Some organic arsenic compounds have little or no toxicity or are rapidly eliminated from the body in the urine. Inorganic arsenic compounds were generally considered to be responsible for its toxic and carcinogenic effects; however, inorganic arsenate and arsenite in water may be metabolized by the body to toxic organic forms of arsenic. There is no evidence that children or other groups such as pregnant women are at a greater risk of developing health effects from exposure to arsenic compared with the general population.

Levels of arsenic tend to be higher in groundwater than in surface water. Levels of arsenic naturally found in waters generally range between 0.001 and 0.002 mg/L, but arsenic may occur in much higher concentrations. Data collected indicate that the levels of arsenic in Canadian drinking water are generally less than 0.005 mg/L.

Since arsenic is classified in Group 1 (carcinogenic to humans), the MAC is derived based on the estimated lifetime cancer risk; consideration was also given to available practical treatment technology and the PQL.

A MAC of 0.005 mg/L (5 µg/L) for arsenic is proposed on the basis of the following considerations:

*** When calculating lifetime cancer risks associated with arsenic in drinking water, the widest possible range (i.e., from 4.50×10^{-6} to 2.47×10^{-5}) was used in order to be conservative.

- The concentration of arsenic in drinking water representing an "essentially negligible" risk is 0.4 µg/L. Levels of arsenic in drinking water should be as close as possible to this level.
- The MAC must be achievable at reasonable cost. Several advanced municipal-scale treatment processes can remove arsenic from drinking water to levels of 1–5 µg/L. Given their complexity and cost, these processes may not be practical for smaller communities. Alternative processes, such as adsorption and membrane systems, are suitable, with proper pretreatment, for reduction of arsenic to low concentrations (<3 µg/L) in small to mid-sized communities. At residential scale, drinking water treatment devices can be certified as reducing arsenic concentrations only to 10 µg/L.The most common types of residential-scale arsenic removal systems are reverse osmosis and steam distillation systems. The estimated lifetime risk associated with ingestion of water containing arsenic at 10 µg/L is 4.5×10^{-5} to 2.47×10^{-4}.
- The MAC must be measurable. The PQL, based on the ability of laboratories to measure arsenic within reasonable limits of precision and accuracy, is 5 µg/L.

The estimated lifetime cancer risk associated with the ingestion of drinking water containing arsenic at 5 µg/L is greater than the range that is considered generally to be "essentially negligible" (i.e., between 10^{-5} and 10^{-6}). Based on the incidence of internal (lung, bladder, liver, kidney) cancers in individuals in southwestern Taiwan, the estimated lifetime cancer risk associated with ingestion of water containing arsenic at 5 µg/L ranges from 2.3×10^{-5} to 1.2×10^{-4} (derived by multiplying the unit risk by the proposed MAC of 5 µg/L).

The MAC will be reviewed periodically in light of developments in treatment technology and additional data on health risks associated with exposure to arsenic in drinking water.

11.0 References

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Appendix I: Provincial/Territorial Cost Estimates

Prince Edward Island
Data on arsenic exposure in PEI drinking water are limited, especially for private wells; however, the existing data do not identify any municipal water supplies with arsenic concentrations above the proposed guideline value of 5 µg/L. I do note, however, that a number of systems have had arsenic detections not far below the proposed guideline value. It is assumed that observed arsenic concentrations are a reflection of local geological conditions and therefore not likely to change over time. However, given the low concentrations potentially of concern, it is likely that as more arsenic exposure data accumulate, some sites with concentrations slightly above the proposed guideline will be identified, particularly for private wells (i.e., data for existing muncipal supplies do not suggest a problem at this point). Cost implications for private wells are relatively minor (i.e., installation of point-of-use treatment) and roughly comparable to costs associated with more common problems with nitrate and other metals. Costs associated with potential exceedances for municipal supplies are difficult to quantify given the uncertainty in the probability of occurrence; however, they would be substantial, as current municipal water supply does not include any water treatment processes beyond disinfection.

Newfoundland and Labrador
Exposure
Information for Newfoundland and Labrador from the testing of public water supplies indicates 145 public water systems where arsenic has been detected at some level as low as 1 µg/L. Twelve systems are surface water systems, and the remainder are groundwater-based systems. Six (6) systems are no longer in use as of this date, and an additional eleven (11) will be removed by December 2004. The total serviced population affected by arsenic detection is 23 833, not counting those water supplies where corrective action has been completed. Of the 139 public water systems that are currently in use, and where arsenic has been detected, 35 water supplies have average arsenic concentrations over the proposed MAC of 5 µg/L. These are all groundwater-based supplies. The serviced population is estimated to be 3184. Of these, 22 water supplies have average arsenic concentrations over 10 µg/L. The serviced population for this subgroup is estimated to be 1533.

Treatment costs
Eleven (11) wells in two communities are already being replaced with a surface water system for a cost of $3.0 million. Additionally, it is estimated that the cost to replace the other wells where the average arsenic concentration exceeds 5 µg/L with surface water supplies will be in the order of $10.6 million. The cost to provide source treatment costs cannot be reliably estimated for small systems under 1000 people. The cost to provide point-of-use filters was also estimated where the arsenic concentration exceeded 10 µg/L. This cost would be $137,250 based on $450 per household. Where a new system will be provided, a point-of-use estimate was not included.

Arsenic has been found in private and semi-public water supplies as well. Because these water sources are not routinely tested, there is no way to estimate exposure or costs to reduce arsenic concentrations.

Nova Scotia

Reduction to 10 µg/L from 25 µg/L

Based on a review of the private well water database, it is estimated that approximately 10,000 households and small public water systems in Nova Scotia have arsenic concentrations that range from 10 µg/L to 25 µg/L. Assuming a cost of $1,000 to $1,200 per household/system to purchase and install a point-of-entry treatment device, the total estimated cost to private well owners and small public water systems in Nova Scotia is expected to be in the order of $10 to $12 million. This estimate is provisional as small public water systems may require larger expenditures. Other options that may be considered include switching to an alternate water supply which may have greater costs. With respect to municipal water supplies, a review of the limited historical data that is available indicates that one system may have difficulty meeting an arsenic guideline of 10 µg/L. Cost estimates to remediate this supply have not been prepared at this time.

Reduction to 5 µg/L from 25 µg/L

Based on a review of the private well water database, it is estimated that approximately 12,000 households and small public water systems in Nova Scotia have arsenic concentrations that range from 5 µg/L to 10 µg/L. Drinking water treatment devices that meet NSF/ANSI standards, however, are only certified to reduce the concentration of arsenic in water to 10 µg/L. Since these units may not be capable of reducing levels below 10 µg/L, no costs have been estimated for private well owners and small public water systems to meet the 5 µg/L using point-of-entry treatment device. Some private well owners and small public water systems may consider other treatment options or may choose to switch to an alternate source of water supply. Costs for these alternatives may be significant but have not been factored into the evaluation at this time.

With respect to municipal water supplies, a review of the limited historical data that is available indicates that six systems may have difficulty meeting an arsenic guideline of 5 µg/L. Cost estimates to remediate these supplies have not been prepared at this time.

New Brunswick

Reduction to 10 µg/L from 25 µg/L

A reduction to 10 µg/L would not have any impact on municipal water system operations in New Brunswick. There are 69 Crown-owned water systems (mainly schools) that would require remedial equipment and likely plumbing refitting at an estimated capital cost of $30,000/school (i.e., $2.3M in total). This cost does not include annual maintenance costs. For private well owners, it is estimated that 1200 to 1500 supplies would fall in the greater than 10 µg/L to less than the current 25 µg/L range. Based on $1200/household, the total estimated remedial cost to private homeowners is expected to fall in the range of $1.7M to $2.1M.

Reduction to 5 µg/L from 25 µg/L

A reduction to 5 µg/L may impact one municipal water system operation in New Brunswick. A cost estimate to remediate has not been estimated at this time. There are 174 Crown-owned water systems (mainly schools) that would require remedial equipment and likely plumbing refitting at an estimated capital cost of $30,000/school (i.e., $5.9 M in total). A significant impact will again be to private well owners, where an estimated 3120 to 3900 supplies would fall in the greater than 5 µg/L to less than the current 25 µg/L range. The total estimated remedial cost to private homeowners is expected to fall in the range of $4.3M to $5.4M.

Quebec
A reduction to 10 µg/L may impact some small municipal, private, campground, and school water systems.
Number of systems affected: 22
Estimated cost to meet 10 µg/L: $8.5 M.
A reduction to 5 µg/L may impact more small municipal, private, campground, and school water systems.
Number of systems affected: 60
Estimated cost to meet 5 µg/L: $21 M.
These estimates are provisional. We do not have substantial data for private wells to have a complete picture of the situation, but we already have some data indicating that arsenic is disseminated in some private wells in a few areas in Quebec.

Ontario
In Ontario, reducing the arsenic guideline from 25 µg/L to 5 µg/L will impact about 27 municipal groundwater water systems at an approximate cost of $30 million. Eleven municipal groundwater systems may be affected if the guideline was revised to 10 µg/L at an approximate cost of $11 million.

The impact to non-municipal systems using groundwater may be significant however at this time it is difficult to estimate the cost implications.

Manitoba
Arsenic concentrations are generally below the current Canadian interim maximum acceptable concentration (IMAC) guideline value of 0.025 mg/L and the proposed guideline value of .005 mg/L in groundwater from the major bedrock aquifers and most confined and unconfined sand/gravel aquifers in Manitoba.

Of 292 municipal systems monitored for (total) Arsenic, only 15 exceed the proposed guideline of 0.005 mg/L in the raw water. Of these 15, 6 have implemented treatment technologies that are capable of reducing concentrations to below the proposed guideline in the finished water. No cost estimates have been prepared related to the cost of implementing the treatment technologies required to bring the remaining 9 into compliance.

Concentrations above the guideline value are, with rare exceptions, found only in confined sand and gravel aquifers in south-central and south-western Manitoba where a number of private wells and some wells supplying groundwater to municipal systems have been found to produce groundwater having As concentrations exceeding the proposed drinking water guideline.

No detailed studies have yet been carried out in the province to examine the origins and geochemistry of arsenic in groundwater although the data currently available points toward elevated arsenic concentrations being primarily associated with sand/gravel aquifers developed in Cretaceous shale-rich environments.

Saskatchewan
Reducing the current IMAC to the new MAC for arsenic would have a significant impact on Saskatchewan communities.

Reduction to 10 µg/L from 25 µg/L

A reduction to 10 µg/L would have an impact on about 23 systems serving <1% of the total population in the province. A rough cost estimate based on the cost equations (linear regression model) was developed exclusively for arsenic treatment. Total treatment cost including everything except storage tanks would be about $4 M. If just filters needed to be replaced, then the cost would be about $2 M. For just changing filter media and chemical, an annual cost would be about $500 K.

Reduction to 5 µg/L from 25 µg/L
A reduction to 5 µg/L would have an impact on about 63 systems serving about 3% of the total population in the province. A rough cost estimate based on the cost equations (linear regression model) was developed exclusively for arsenic treatment. Total treatment cost including everything except storage tanks would be about $11 M. If just filters needed to be replaced, then the cost would be about $6 M. For just changing filter media and chemical, an annual cost would be about $1 M.

Alberta
Arsenic is generally not a problem in municipal systems, but it may be a problem with some private wells. By reducing the arsenic levels to 5 µg/L, three communities will be affected, and by reducing the levels to 10 µg/L, only one community will be affected. The capital cost impact at 5 µg/L on municipal systems would be approximately $3 million.

Cost impact on private systems is unknown; it will be significant, according to the Regional Health Units. The preliminary information I have is that a decrease in permissible levels from 25 to 5 µg/L would result in an increase from approximately 20% of the homes being in non-compliance to over 60%.

British Columbia
A reduction to 5 or 10 µg/L will have a moderate impact on the municipal water system. The total number of systems exceeding 5 or 10 µg/L is not available, although health authorities have been advised to review their records and identify those systems that may need to take action in response to the new guideline. Systems identified to date are relatively small and in several cases have already identified the need to treat their water in response to the presence of arsenic.

There are several areas in the province where arsenic-bearing rock formations are near the surface and private wells contain arsenic at levels above 10 µg/L. Public advisory material will require updating, and more home owners may choose to invest in point-of-use or point-of-entry treatment.

Yukon
Four of Yukon public drinking water systems (25%) would exceed a limit of 0.010 mg/L.

Seven of Yukon public drinking water systems (44%) would exceed a limit of 0.005 mg/L.

A dollar value has not yet been determined in terms of impact. I will pass on that information when it becomes available.

It is possible that other wells located in the same areas where public water systems exceed the proposed guideline for arsenic would also be in exceedance. However, there is no registration system for private wells in Yukon, so comprehensive data are not available.

Northwest Territories
There are no expected cost implications for the Northwest Territories if the guideline value for arsenic in drinking water is lowered to 0.005 mg/L. It is expected that all drinking water supplies in the NWT will comply with this new guideline value without additional treatment**.**

Nunavut
No information provided.