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Canadian Soil Quality Guidelines for

Arsenic: Environmental and Human Health

Supporting Document — Final Draft
December 1996

NOTICE

This final draft document provides the information supporting the derivation of environmental and human health soil quality guidelines for arsenic. Development of these soil quality guidelines was initiated through the National Contaminated Sites Remediation Program (NCSRP) which officially ended in March 1995. Given the need for national soil quality guidelines for contaminated sites management and many other applications, development was pursued under the direction of the CCME Soil Quality Guidelines Task Group after the end of the NCRSP.

This document is a working document that was released shortly after the publication of "A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines" (CCME 1996). The CCME recognizes that some refinements or changes to the Protocol may become necessary upon application and testing. If required, amendments to the Protocol will be made and the guidelines will be modified accordingly. For this reason guidelines are referred to in this document as CCME Recommended Guidelines. Readers who wish to comment or provide suggestions on the Protocol or on the guidelines presented in this document should send them to the following address:

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This document is a supporting technical document. It is available in English only. A French Abstract is given on page vii.

Ce document technique de soutien n'est disponible qu'en anglais avec un résumé en français présenté à la page vii.

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ABSTRACT

Canadian environmental quality guidelines, developed under the auspices of the Canadian Council of Ministers of the Environment (CCME), are numerical concentrations or narrative statements recommended to support and maintain designated resource uses. CCME Canadian soil quality guidelines can be used as the basis for consistent assessment and remediation of contaminants at sites in Canada.

This report was prepared by the Guidelines Division of the Science Policy and Environmental Quality Branch (Environment Canada), which acts as Technical Secretariat for the CCME Soil Quality Guidelines Task Group. The Guidelines were derived according to the procedures described in *A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines* (CCME 1996). Environment Canada was responsible for the derivation of the Soil Quality Guidelines for the protection of ecological receptors whereas Health Canada was responsible for the derivation of the Soil Quality Guidelines for the protection of human health.

Following the introduction, chapter 2 presents chemical and physical properties of arsenic and a review of the sources and emissions in Canada. Chapter 3 discusses arsenic's distribution and behavior in the environment while chapter 4 reports the toxicological effects of arsenic on microbial processes, plants, animals, and humans. These informations are used in chapters 5 and 6 to derive soil quality guidelines for arsenic to protect environmental receptors and human health in four types of land uses: agricultural, residential/parkland, commercial, and industrial.

The following soil quality guidelines are recommended by the CCME based on the available scientific literature. In the case of environmental soil quality guidelines (SQG_E) for agricultural and residential/parkland land uses, the final SQG_E is 19 $mg \cdot kg^{-1}$ soil and for commercial and industrial land uses, the final SQG_E is 26 $mg \cdot kg^{-1}$ soil. These environmental soil quality guidelines are optimized for soils within the pH range of 4 to 7.6 as the toxicological studies on which they are based were conducted within this pH range. For human populations, since the group of inorganic arsenic compounds as a whole has been classified as "carcinogenic to man", no specific human health soil quality guideline above background is recommended for inorganic arsenic. A risk-specific human health soil quality guideline of 12 $\mu g \cdot g^{-1}$ dry soil ($mg \cdot kg^{-1}$ dry soil) is proposed for residential/parkland and industrial land uses with an associated lifetime cancer risk of 10^{-6} above background.

RÉSUMÉ

Les recommandations canadiennes pour la qualité de l'environnement, élaborées sous les auspices du Conseil Canadien des Ministres de l'Environnement (CCME), sont des concentrations ou des énoncés décrivant les limites recommandées dans le but d'assurer le maintien et le développement durable d'utilisations désignées des ressources. Les recommandations canadiennes pour la qualité des sols proposées par le CCME peuvent être utilisées comme base pour l'uniformisation des processus d'évaluation et d'assainissement des terrains contaminés au Canada.

Le présent document a été préparé par la Division des Recommandations de la Direction de la Qualité de l'Environnement et de la Politique Scientifique (Environnement Canada), qui agit comme secrétaire technique pour le Groupe de Travail du CCME sur les Recommandation pour la qualité des sols.. Les Recommandations ont été élaborées selon les procédures décrites dans le *Protocole d'élaboration de recommandations pour la qualité des sols en fonction de l'environnement et de la santé humaine* (CCME 1996). Environnement Canada fut responsable de l'élaboration des recommandations pour la qualité des sols en vue de la protection des récepteurs écologiques alors que Santé Canada a procédé à l'élaboration des recommandations pour la qualité des sols en vue de la protection de la santé humaine.

Faisant suite à une brève introduction, le chapitre 2 présente les propriétés physiques et chimiques de l'arsenic de même qu'un survol des sources et des émissions au Canada. Le chapitre 3 discute du devenir et du comportement de cette substance dans l'environnement alors que le chapitre 4 rapporte ses effets toxicologique sur les processus microbiens, les plantes, les animaux et les humains. Ces informations sont utilisées aux chapitres 5 et 6 afin d'élaborer des recommandations pour la qualité des sols relatives à l'arsenic en vue de la protection de l'environnement et de la santé humaine dans le cadre de quatre types d'utilisations de terrains: agricole, résidentiel/parc, commercial et industriel.

Les recommandation pour la qualité des sols suivantes, proposées par le CCME, sont fondées sur les données scientifiques disponibles. Dans le cas des recommandations pour la qualité des sols en vue de la protection de l'environnement (RQS_E) relatives aux terrains à vocation agricole et résidentielle/parc, la RQS_E finale est de 19 $mg \cdot kg^{-1}$ de sol, alors que pour les terrains à vocation commerciale et industrielle, la RQS_E finale est de 26 $mg \cdot kg^{-1}$ de sol. Ces recommandations pour la qualité des sols en vue de la protection de l'environnement sont à leur optimum dans des sols avec pH entre 4 et 7.6 puisque les études toxicologiques utilisées pour leur élaboration ont été effectuées dans ces mêmes conditions de pH. Dans le cas de la protection de la santé humaine, puisque l'ensemble des composés inorganiques d'arsenic à été classé dans la catégorie des produits "cancérigènes pour les humains", aucune recommandation définitive allant au delà des concentrations de fond n'est proposée pour l'arsenic inorganic. Une recommandation pour la qualité des sols associée à un risque spécifique est proposée pour les terrains à vocations résidentielle/parc et industrielle. Cette recommandation est de 12 $\mu g \cdot g^{-1}$ de sol sec ($mg \cdot kg^{-1}$ de sol sec) et est associée à un risque additionnel de cancer au cours d'une vie de 10^{-6} au delà des risques de fond.

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Canadian Soil Quality Guidelines for Arsenic: Environmental and Human Health

1. INTRODUCTION

The Canadian environmental quality guidelines of the Canadian Council of Ministers of the Environment (CCME) are numerical limits for contaminants intended to maintain, improve, or protect environmental quality and human health. CCME Canadian soil quality guidelines can be used as the basis for consistent assessment and remediation of contaminants at sites in Canada along with CCME guidelines issued for the protection of water quality, sediment quality, and tissue quality. In response to the urgent need to begin remediation of high priority "orphan" contaminated sites, an interim set of criteria was adopted from values currently in use in various jurisdictions across Canada (CCME 1991). Many of the CCME remediation soil quality criteria do not have a complete supporting scientific rationale and are being updated based on current scientific information.

This report reviews the sources and emissions of arsenic, its distribution and behavior in the environment, and its toxicological effects on microbial processes, plants, animals, and humans. This information is used to derive guidelines for arsenic to protect human health and environmental receptors according to the processes outlined in A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines (CCME 1996) for agricultural, residential/parkland, commercial, and industrial land uses. An alternative protocol, described in section 6.2, was used to derive human health soil quality guidelines for residential and industrial land uses only.

The values derived herein are intended as general guidance. Site-specific conditions should be considered in the application of these values. The values may be applied differently in various jurisdictions, therefore, the reader should consult the appropriate jurisdiction before application of the values.

2. BACKGROUND INFORMATION

2.1 Physical and Chemical Properties

Arsenic is a metalloid of the group VA elements (N, P, As, Sb, Bi), which exhibits both metallic and nonmetallic properties. The atomic number and atomic weight of arsenic are 33 and 74.92 $g \cdot mole^{-1}$, respectively. Arsenic possesses three unpaired electrons in its outer shell, which allows it to have unusual electrical and spectroscopic properties (NRCC 1978). Chemically, arsenic closely resembles phosphorus (NRCC 1978). Arsenic can occur in four oxidation states as arsine (−3), arsenic metal (0), arsenite (+3), and arsenate (+5). Arsenic bonds covalently with most nonmetals and metals. It forms stable organic compounds in both its trivalent and pentavalent states. Some physical and chemical properties of arsenic and inorganic arsenic compounds are presented in table 1.

Arsenic, in various forms, occurs naturally in most environmental compartments. It is rarely found in its elemental form (Slooff et al. 1990) and occurs most commonly as sulphides and as complexes with iron, nickel, copper, and cobalt (Woolson 1975). The anthropogenic form of arsenic most frequently released to the environment is arsenic(III) oxide (Slooff et al. 1990).

2.2 Analytical Methods

A review of the analytical chemistry of arsenic is beyond the scope of this document but can be found in publications by Talmi and Feldman (1975), Irgolic et al. (1983), Korte and Fernando (1991), and most recently CCME (1993).

In soils, the fraction of the total arsenic that is of relevance is the "available" arsenic, and the amount that is rendered available is dependent on environmental conditions. Therefore, soils of differing physical and chemical characteristics with the same soil arsenic concentrations will exhibit varying levels of plant-available arsenic and phytotoxicity. Extraction of arsenic from soils has followed the use of separate media extraction or the fractionation approach common to soil phosphorus analysis. No one extractant will serve as a primary index of all plant available arsenic. The extractant selected should be specific for certain arsenic components in the soil.

One method of analysis for total arsenic in soil has been recommended by CCME (1993) in an effort to promote consistency within the National Contaminated Sites Remediation Program (NCSRP). U.S. EPA Method 6010 (inductively coupled plasma–atomic emission spectroscopy) prescribes a sequential sample preparation using nitric acid, hydrogen peroxide, and hydrochloric acid followed by analysis with an inductively coupled argon plasma emission spectrometer capable of background correction. The detection limit for the instrument is 53 $\mu g \cdot L^{-1}$, and the reproducibility of measurements, expressed as the standard deviation, is 23%. Following corrections for a 1-g soil sample, digested, with the final extract being diluted to 100 mL, a detection limit of 5.3 $mg \cdot kg^{-1}$ for arsenic would be calculated.

2.3 Production and Uses in Canada

Arsenic occurs as a minor constituent in complex ores that are mined primarily for their copper, lead, zinc, silver, and gold content. In Canada, gold ores are the main source of arsenic, whereas worldwide, copper ore is the predominant source. Arsenic is usually recovered from dusts and residues associated with the roasting of these ores. It is collected as impure arsenic trioxide and is either purified on site or sold directly to a refiner. Ninety-six percent of arsenic is utilized as arsenic trioxide or other arsenic compounds, and only 4% is utilized in the metallic state. Though four gold-ore roasters recovered arsenic in the 1970s, only two mines produced arsenic in 1992. Demand for arsenic has fallen sharply since the early 1980s because of concerns about environmental effects. Because of low prices, arsenic trioxide currently produced in Canada is being stockpiled for future sale (Ignatow et al. 1991).

At present in Canada, arsenic is used mainly in metallurgical applications (Environment Canada 1991) and in the manufacture of wood preservatives. For the period 1984–1988, Canadian sales of arsenic

(as arsenic pentoxide) for use in wood preservation fluctuated between about 650 and 1300 t per year (E. Brien 1992, Environment Canada, pers. com.). Arsenic compounds have also been used in Canada in herbicides (E. Brien 1992, Environment Canada, pers. com.), in pharmaceuticals, and in glass manufacturing (CCREM 1987). World production of arsenic trioxide was 56 000 t in 1990. This was an increase of approximately 19 000 t since the early 1980s (Ignatow et al. 1991; Law-West 1987). In contrast, the demand for arsenic in Canada has decreased over this period, partially because of concerns about its use in agriculture. Canadian production of arsenic trioxide in 1985 was approximately 8 400 t, whereas in 1986 it had decreased to 5 100 t (Law-West 1987).

In 1990, approximately 70% of the world's arsenic was used in wood preservatives, 22% in agricultural chemicals, 4% in glass manufacturing, 2% in nonferrous alloys, and 2% in other uses. Over 50% of the world's arsenic production is consumed by the United States (Ignatow et al. 1991).

2.3.1 Wood Treatment

Arsenical wood preservatives are used in order to prevent rotting or insect damage in building foundations, fence posts, submerged footings, and utility poles. The most common arsenic compounds used are chromated copper arsenate (CCA), ammoniacal copper arsenate (ACA), and fluor chrome arsenate phenol. In 1984, 5 000 t of 50% chromated copper arsenate concentrate (about 550 t of arsenic) were used in Canada for wood preservation (Konasewich and Henning 1988).

2.3.2 Agriculture

Arsenical agricultural chemicals are used as herbicides, plant desiccants, and defoliants to control grassy and broadleaf weeds. Since the 1970s, the inorganic arsenical herbicides such as lead arsenate and calcium arsenate have been replaced with the less phytotoxic organic arsenical herbicides. Monosodium methylarsonate, disodium methylarsonate, and dimethylarsinic acid are the most commonly used agricultural arsenical compounds (Adriano 1986; Ignatow et al. 1991; Wauchope and McDowell 1984).

2.3.3 Glass Manufacturing

Arsenic trioxide is used in the glass industry as a decolorizing agent. It is also used to remove tiny air bubbles in the glass (Ignatow et al. 1991).

2.3.4 Nonferrous Alloys and Other Uses

Arsenic metal is used as a minor alloying agent in certain copper- and lead-based alloys. Arsenic increases the corrosion resistance and tensile strength of copper used in industrial plant piping and auto radiators. Gallium arsenide and its alloys are employed as semiconductors, which are then used in such products as light-emitting diodes, microwave devices, solar cells, and photo-emissive surfaces (Ignatow et al. 1991).

2.4 Levels in the Canadian Environment

Base-metal and gold-production facilities, the use of arsenical pesticides, coal-fired power generation, and the disposal of domestic and industrial wastes are the principal anthropogenic sources of arsenic release into the Canadian environment. Most (80%) of the anthropogenic releases to the environment are ultimately released to soil (ATSDR 1993). Arsenic is present naturally in the aquatic and terrestrial environments from weathering and erosion of rock and soil. In areas of arsenic-enriched bedrock, background concentrations can be significantly elevated. Arsenic is released naturally into the atmosphere by volcanic eruptions and the escape of volatile methylarsines from soil (Chilvers and Peterson 1987).

2.4.1 Soil

Worldwide, there is a wide range in natural arsenic content in uncontaminated soils. Arsenic concentrations may vary greatly depending on the type of soil and the hydrology (Slooff et al. 1990). Walsh and Keeney (1975) reported arsenic concentrations in uncontaminated soils between 0.2 and 40 $\mu g \cdot g^{-1}$. However, Kabata-Pendias and Pendias (1984) have reported a range of <1–95 $\mu g \cdot g^{-1}$, with a mean arsenic concentration for world soils of 8.7 $\mu g \cdot g^{-1}$ (Kabata-Pendias and Pendias 1984; Adriano 1986). The average arsenic concentration, worldwide, in both soil and rocks, has been estimated at 3 $\mu g \cdot g^{-1}$ (Boyle and Jonasson 1973). Near arseniferous mineral deposits, natural soil concentrations of arsenic may average between 400 and 900 $mg \cdot kg^{-1}$, with some locations reaching concentrations as high as 8 000 $mg \cdot kg^{-1}$ (NRCC 1978). Arsenic is found primarily in its pentavalent form in soil (Amdur et al. 1991).

Mean concentrations of arsenic in several uncontaminated soil types in Canada were reported to range from 4.8 to 13.6 $\mu g \cdot g^{-1}$ dry weight (dw), based on a review of available data (Kabata-Pendias and Pendias 1992). Data from recent surveys undertaken by the Geological Survey of Canada demonstrate that the "natural background" concentrations of arsenic in surficial media such as glacial tills (the substrate on which most Canadian soils are developed) span over several orders of magnitude reflecting changes in the bedrock geology and effects of glacial erosion of bedrock debris (I. Kettles 1995, Natural Resources Canada, pers. com.). Concentrations of arsenic in glacial till samples from a number of Canadian sites range from 1 to 6 590 $\mu g \cdot g^{-1}$ dw (table 2). The concentrations of arsenic in surface soil found at various uncontaminated sites across Canada are summarized in table 3. It shows average urban and agricultural concentrations to be at or below 10 $\mu g \cdot g^{-1}$ dry soil with most of the values in the 4–6 $\mu g \cdot g^{-1}$ range. Background arsenic levels have been provisionally established for Ontario soils at 11 $\mu g \cdot g^{-1}$ for agricultural lands and 17 $\mu g \cdot g^{-1}$ for residential and parkland areas (OMEE 1994b). Background levels, as calculated by OMEE (1994b), consist of the value of the 98th percentile, which was obtained by adding two standard deviations to the mean (L. Pastorek 1995,Ontario Ministry of the Environment and Energy, pers. com.).

Higher concentrations were found near base-metal smelters (mean concentrations ranged from 50 to 110 mg $As \cdot kg^{-1}$, with a maximum of 2 000 $mg \cdot kg^{-1}$) (Brunswick Mining and Smelting Corporation 1992, pers. com.; Hertzman et al. 1991, Department of Health Care and Epidemiology, University of British Columbia, pers. com.; M. Murphy n.d., New Brunswick Department of the Environment,

pers. com.; H. Teindl 1992, Cominco Metals, pers. com.; Temple et al. 1977), and gold-mining/ roasting operations (mean concentrations of 60–110 mg As·kg^{-1} and maximum of >21 000 mg·kg^{-1}) (EBA Engineering Consultants Ltd. 1991; Gemmill 1977; Hutchinson et al. 1982; NRCC 1978; Plambeck and Smith 1976; Prokopuk 1976). These high levels may exist over wide areas as Plambeck and Smith (1976) observed that local background levels of 25 mg As·kg^{-1} in soils and lichens were reached only at distances of more than 80 km from the point source (a gold smelter near Yellowknife, Northwest Territories). Large amounts of arsenic have been reported in soil, sediment, and water in the vicinity of arsenic-bearing precious metal deposits near Cobalt, Ontario, and Halifax, Nova Scotia (Bottomley 1984; Boyle et al. 1969; Brooks et al. 1982). Highest arsenic concentrations (up to 75 000 mg·kg^{-1}; typically 3 000–4 000 mg·kg^{-1}) were found in tailings at base- and precious-metal mine sites in Ontario and Nova Scotia (Dale and Freedman 1982; Hawley 1980).

Arsenic residues can accumulate to very high levels in agricultural areas where arsenical pesticides or defoliants have been repeatedly used. Agricultural use of arsenicals can cause surface soil accumulation of 600 mg·kg^{-1} or more (Adriano 1986). Elevated arsenic levels have been reported in soils where arsenical pesticides (including wood-preservation compounds) have been used; for example, concentrations of arsenic of up to 290 mg·kg^{-1} (mean values of up to 54 mg·kg^{-1}) have been detected in soil from orchards in Ontario (Boyle and Jonasson 1973; Frank et al. 1976a). Concentrations of arsenic up to 10 860 mg·kg^{-1} (mean values of up to 6 000 mg·kg^{-1}) have been recorded at active wood-preservation facilities in Atlantic Canada (Bamwoya et al. 1991). A summary of the concentrations of arsenic in contaminated surface soils from various sites across Canada is shown in table 4. Analysis of whole-soil and soil pore water from a limited number of areas indicates that most (>90%) of the arsenic in soils is inorganic (Haswell et al. 1985; Masscheleyn et al. 1991; Woolson 1983).

2.4.2 Groundwater

Groundwater normally contains higher concentrations of arsenic than are found in associated surface waters, particularly when located near arseniferous mineral deposits (Boyle and Jonasson 1973). Available data indicate that most Canadian groundwaters contain <50 µg As·L^{-1} (Léger 1991; Michel 1990), however, concentrations of arsenic have been reported to range up to 9 100 µg·L^{-1} in groundwater from areas with a high content of arsenic in the bedrock, such as in regions of Ontario, Quebec, New Brunswick, and Nova Scotia (Bottomley 1984; Boyle et al. 1969; P. Deveau 1992, Brunswick Mining and Smelting Corporation, pers. com.; Grantham and Jones 1977; Lalonde et al. 1980; Méranger et al. 1984). Gold mining activities in Nova Scotia have been reported to contribute to high arsenic levels in local groundwaters (Bottomley 1984). High arsenic concentrations (up to 11 000 µg·L^{-1}) were also detected in groundwater in the vicinity of an abandoned arsenical wood preservative facility near Vancouver, British Columbia (Henning and Konasewich 1984). Recent studies (Korte 1991; Masscheleyn et al. 1991) in other areas suggest that arsenic in groundwater is present mainly as inorganic As(III) and As(V) species.

2.4.3 Sediments

Sediments generally have higher arsenic concentrations than their surrounding waters (NAS 1977). In Canada, background concentrations in relatively uncontaminated surface sediment are generally <20 $mg \cdot kg^{-1}$ dw; arsenic levels in deeper sediment are normally only a few milligrams per kilogram (Friske 1985; Johnson 1987). Arsenic accumulation (up to 65 $mg \cdot kg^{-1}$) has been reported in contaminated Halifax harbour sediments (Tay et al. 1991). Bamwoya et al.(1991) found 262 mg $As \cdot kg^{-1}$ in sediments downstream from an arsenical wood preservation facility near Elmsdale, Nova Scotia. Concentrations of arsenic are highest in sediments near base- and precious-metal mining and ore-processing operations. Average levels of 100–200 $mg \cdot kg^{-1}$ (maximum 650 $mg \cdot kg^{-1}$) were reported near base-metal mines and smelters in several provinces (Bailey 1988; Franzin 1984; Palmer et al. 1989). Near gold mines and abandoned precious-metal refineries, mean concentrations in sediments ranged from about 700 to 5 000 mg $As \cdot kg^{-1}$ (maximum 18 650 $mg \cdot kg^{-1}$) (Diamond 1990; K. Reimer and J.A.J. Bright 1992, Royal Roads Military College, pers. com.; Sutherland 1989; Trip and Skilton 1985). Limited data on the composition of pore waters from two areas in Canada (Diamond 1990; Reimer and Thompson 1988) suggest that most (≥85%) bioavailable arsenic in sediment is present as inorganic As(III) and As(V).

2.4.4 Surface Water

Arsenic levels in Canadian surface waters remote from point sources of contamination are typically <2 $\mu g \cdot L^{-1}$ (Bailey 1988; Belzile and Tessier 1990; Environment Canada 1988; Traversy et al. 1975), but higher concentrations occur in marine waters and in hot and cold springs (Waldbott 1973). High concentrations of arsenic in surface water are usually the result of anthropogenic contamination resulting from coal-burning power plants, nonferrous metal smelters, iron and steel plants, domestic wastewater effluents, and sewage sludge (Nriagu and Pacyna 1988). Elevated concentrations of arsenic have been reported in surface waters in the vicinity of gold-mining or ore-roasting operations. Mean levels of about 45 $\mu g \cdot L^{-1}$ (maxima about 140 $\mu g \cdot L^{-1}$) were found near abandoned gold mines in Nova Scotia (Mitchell Brook) and Ontario (Moira Lake) during the 1980s (Brooks et al. 1982; Diamond 1990). Highest arsenic levels were found in samples of water collected from several lakes near the gold mines and roasters at Yellowknife in the mid-1970s; for example, Keg and Kam Lakes contained from 700 to 1 500 $\mu g \cdot L^{-1}$ and from 1 500 to 5 500 $\mu g \cdot L^{1}$, respectively (CPHA 1977; Wagemann et al. 1978). Limited data from the early 1990s indicated that concentrations in these lakes had declined appreciably, to about 545 and 645 $\mu g \cdot L^{-1}$ in Keg Lake and Kam Lake, respectively (K.J. Reimer and J.A.J. Bright 1992, Royal Roads Military College, pers. com.). Most (≥80%) of the arsenic in contaminated waters, as well as in surface waters remote from point sources, is expected to be present as inorganic As(V) and to a lesser extent As(III) species (Cullen and Reimer 1989).

2.4.5 Drinking Water

Data on concentrations of arsenic in Canadian drinking water from a nationwide survey have not been identified. Based on data obtained in provincial monitoring programs, levels in Canadian drinking water from both surface water and groundwater supplies are generally <5 $\mu g \cdot L^{-1}$ (measured as total

arsenic), although concentrations in some groundwater supplies are greater (Environment Canada 1989a, 1989b, 1989c, 1989d; Manitoba Environment 1989; raw data from the Ontario Drinking Water Surveillance Program, Ontario Ministry of the Environment, 1989). Five percent of samples of well water from contaminated supplies in Nova Scotia contained >500 μg $As \cdot L^{-1}$ (Méranger et al. 1984).

2.4.6 Ambient Air

The two principal natural sources of atmospheric arsenic are biological methylation and volatilization following weathering processes. Worldwide, natural atmospheric emissions of arsenic are approximately 50% greater than anthropogenic emissions (NRCC 1978; Slooff et al. 1990). The mean atmospheric arsenic concentration is 1 $ng \cdot m^{-3}$ over land and ranges from 0.02 to 0.16 $ng \cdot m^{-3}$ over oceans (Walsh et al. 1979). Most of the arsenic in air is associated with fine suspended particulates of <5 μm in diameter.

In Canada, recent long-term studies undertaken by Environment Canada indicate low arsenic concentrations for inhalable particulates (<10 μm in diameter) in the ambient air of 15 urban areas and 4 rural sites. Between 1986 and 1993, 53% of 3 863 ambient air samples from across Canada contained arsenic in concentrations below the detection limit of 0.0005 $\mu g \cdot m^{-3}$; a mean concentration of 0.001 $\mu g \cdot m^{-3}$ was calculated incorporating all data points and substituting half the detection limit for values below detection (Dann 1994). NRCC (1978) reported that total arsenic concentrations ranged between 0.27 and 4.7 $ng \cdot m^{-3}$ in Canadian rural areas.

Higher arsenic concentrations were recorded in ambient air near mines and smelters. Between 1973 and 1975, the concentration of arsenic in suspended particulates near Yellowknife gold smelters ranged from <10 to 3 910 $ng \cdot m^{-3}$. Mean concentrations were between 60 and 90 $ng \cdot m^{-3}$ with arsenic concentrations near Giant Yellowknife mine (Northwest Territories), Con Mine (Northwest Territories), and Yellowknife (Northwest Territories) of 1 680 $ng \cdot m^{-3}$, 380 $ng \cdot m^{-3}$, and 391 $ng \cdot m^{-3}$, respectively (Gemmill 1977). In the 1980s and early 1990s, average annual concentrations of between 8.6 $ng \cdot m^{-3}$ and 300 $ng \cdot m^{-3}$ were measured at the Giant gold-ore roaster in Yellowknife (EC/HWC 1993). Atmospheric arsenic near such industrial sources is typically As_2O_3 in particulate form (Cullen and Reimer 1989).

2.4.7 Indoor Air and House Dust

There is a paucity of information on levels of arsenic in indoor environments such as air and house dust. Based on limited data and information, the levels of arsenic measured indoors appear to be comparable to those measured outdoors.

Bell et al.(1994) screened indoor air samples from 37 homes in the Windsor area for the presence of arsenic and other contaminants using Lo-Vol sampling methodology (low volume, 25 $L \cdot min^{-1}$). Filters were digested in weak (5%) nitric acid and analyzed using ICP-MS (inductively coupled plasma–mass spectrometry). The levels of airborne arsenic ranged from 0.3 to 4.3 $\mu g \cdot m^{-3}$ with a mean of 0.7 $\mu g \cdot m^{-3}$ and a median of 0.5 $\mu g \cdot m^{-3}$. Slightly higher levels of arsenic were measured

concurrently outdoors (residential and commercial areas of Windsor), with a mean of 1.3 $\mu g \cdot m^{-3}$ (N = 46 samples, range 0.4–3.3 $\mu g \cdot m^{-3}$).

In a recent survey performed for the Bureau of Chemical Hazards of Health Canada, house dust and yard soil samples from 50 Ottawa homes were analyzed for arsenic and other trace elements by ICP–MS after digestion with HF/HNO_3 (Kean 1995). Preliminary results indicate house dust and yard soil contain geometric mean concentrations of 4.76 $\mu g\ As \cdot g^{-1}$ dust (range 1.70–79.47) and 2.88 $\mu g\ As \cdot g^{-1}$ soil (range 1.70–9.90), respectively.

2.4.8 Commercial Food

Arsenic has been detected in most foodstuffs consumed by humans, however, the proportion of inorganic arsenic varies considerably. Much of the arsenic in fish, for example, is present in highly complexed forms that are not bioavailable, or as organic compounds that are rapidly excreted from the body. The percentage of total arsenic in various foods that is inorganic has been determined to range from 0% in saltwater fish to 75% in milk, dairy products, beef, and pork, based on limited data (Weiler 1987). Levels of total arsenic were recently determined in 112 food composites from six Canadian cities between 1985 and 1988 under the Total Diet Program (Dabeka et al. 1993). The highest mean concentrations of total arsenic in seven collections were found in fish (1 662 $ng \cdot g^{-1}$), meat and poultry (24.3 $ng \cdot g^{-1}$), bakery goods and cereals (24.5 $ng \cdot g^{-1}$), and fats and oils (19.0 $ng \cdot g^{-1}$). Lower mean concentrations of total arsenic were determined in an earlier survey of 10 food groups in a duplicate diet study in five Canadian cities (range 0.46 $ng \cdot g^{-1}$ in drinking water to 60.1 $ng \cdot g^{-1}$ in meat, fish, and poultry (Dabeka et al. 1987). Based on very limited data, concentrations of arsenic in produce grown in the vicinity of industrial sources may be somewhat higher than those reported in the duplicate diet study (Brunswick Mining and Smelting Corporation 1992, pers. com.; M. Murphy n.d., New Brunswick Department of the Environment, pers. com.; Noranda Mines 1992, pers com.; H. Teindl 1992, Cominco Metals, pers. com.).

2.4.9 Biota Used as Human Food

Levels of arsenic in the tissue of freshwater fish in areas remote from point sources of contamination generally range from <0.1 to 0.4 $mg \cdot kg^{-1}$ fresh weight (fw) (Moore and Ramamoorthy 1984). Concentrations in samples of fish in Lakes Erie and Ontario in the mid-1970s and in base-metal mining areas in Atlantic Canada in 1984 and 1985 were usually within this range (Bailey 1988; Traversy et al. 1975). Higher arsenic levels were reported in fish sampled near active and abandoned gold-mining operations, with maximum concentrations ranging from 2.36 to 4.77 $mg \cdot kg^{-1}$ fw (Azcue 1992; Dale and Freedman 1982; Gemmill 1977).

2.4.10 Human Tissues and Body Fluids

Mean levels of arsenic in human breast milk in India range from 0.3 to 0.6 $ng \cdot g^{-1}$ (Dang et al. 1982). Arsenic has been detected in human breast milk in Yugoslavia, with a mean concentration of total arsenic of 1.3 $\mu g \cdot kg^{-1}$ (range 0.2–3.8 $\mu g \cdot kg^{-1}$) (Kosta et al. 1983).

Urinary arsenic concentrations in "unexposed" populations are normally below 50 $\mu g \cdot L^{-1}$, based on a review of values considered "normal" in earlier reports (Landrigan 1981). Although in some earlier studies, control for external contamination of hair samples by arsenic was inadequate, concentrations in urine and hair are often greater in individuals living in the vicinity of point sources, such as smelters, than in comparison populations (Baker et al. 1977; Milham and Strong 1974; Polissar et al. 1990), and in persons whose diets frequently contain fish and seafood (Vahter 1986). Conversely, however, Jensen et al.(1991) reported that there was no significant difference in the concentration of arsenic in the urine of children and adults residing in a "chemically polluted area" (former sites of industries that used arsenic) of Denmark, compared to that of those living in an unpolluted area. Smokers are reported to have 50% higher mean arsenic levels in blood than nonsmokers (Kagey et al. 1977). Median arsenic levels in various other tissues of the general population and in "exposed" individuals ranged from 0.001 $\mu g \cdot g^{-1}$ in the heart to 3.0 $\mu g \cdot g^{-1}$ in nails, based on a summary of several reports (Fergusson 1990).

2.5 Existing Criteria and Guidelines for Arsenic in Various Media

Existing criteria, guidelines, or standards for arsenic in soil and groundwater from provincial and international agencies are summarized in table 5.

3. ENVIRONMENTAL FATE AND BEHAVIOR

As Woolson (1983) has noted, the ultimate sink for most environmental arsenic is ocean sediment. Because of its reactivity and mobility, however, arsenic can cycle extensively through both biotic and abiotic components of local aquatic and terrestrial systems, where it can undergo a variety of chemical and biochemical transformations, such as oxidation, reduction, methylation, and demethylation (Cullen and Reimer 1989). The following sections describe the major fate and behavior processes of arsenic in the terrestrial environment.

Many factors control the fate and behavior of arsenic in soil, and ultimately its bioavailability. It is generally accepted that only soluble arsenic is available for plant uptake. The solubility and speciation of arsenic in solution are primarily determined by pH and the reduction/oxidation potential (Eh). The solubility of arsenic may determine the amount of arsenic ultimately available within a system, while the speciation of arsenic determines the behavior and to a large extent the toxicity of arsenic.

3.1 Transformations

Generally, in oxidizing conditions or aerobic soils, inorganic arsenic is predominantly present as arsenate, AsO_4^{3-} with arsenic in the +5 oxidation state. In this environment, arsenic acid can dissociate into four different species depending upon the pH:

$$H_3AsO_4 \leftrightarrow H_2AsO_4^- \leftrightarrow HAsO_4^{2-} \leftrightarrow AsO_4^{3-}$$

Thermodynamically, $H_2AsO_4^-$ is the most stable species between pH 2 and pH 7, which overlaps with the normal soil pH range of 4–8. Above pH 7, $HAsO_4^{2-}$ is the most stable (O'Neil 1990).

Under reducing conditions, as in anaerobic, water-logged soils, arsenic is primarily present as arsenite, AsO_3^{3-}, with arsenic in the +3 oxidation state (O'Neill 1990). Arsenous acid dissociates into four pH-dependant species in the same manner as arsenic acid:

$$H_3AsO_3 \leftrightarrow H_2AsO_3^- \leftrightarrow HAsO_3^{2-} \leftrightarrow AsO_3^{3-}$$

H_3AsO_3 is thermodynamically the most stable species up to about pH 9, and is the predominate species within the normal soil pH range. Under strongly reduced conditions, elemental arsenic and arsine (AsH_3) can exist (Elkhatib et al. 1984a, 1984b).

Both redox potential (Eh) and pH can affect the speciation and solubility of arsenic. At higher soil redox levels (200 and 500 mV), arsenic solubility is low and most (65%–98%) of the arsenic in solution is present as As(V). With Eh values of 0 and −200 mV, As(III) becomes the major dissolved arsenic species and arsenic solubility generally increases; up to 40% of the total arsenic present in the soil becomes soluble. In alkaline, or reducing conditions, a substantial proportion of arsenic can be released into solution. Also, under moderately reducing conditions (0 and 100 mV), arsenic solubility can be controlled by the dissolution of iron oxyhydroxides (Masscheleyn et al. 1991). Further, a lower redox potential is required in alkaline soils for the reduction of arsenate to arsenite than in acid soils (Slòoff et al 1990).

A variety of microorganisms can methylate arsenic (Masscheleyn et al. 1991). Methylated arsenic oxyacids are found in various natural waters, soils, and sediments. Some of these methylated arsenic compounds include monomethylarsonic acid, $CH_3AsO(OH)_2$; dimethylarsinic acid, $(CH_3)_2AsO(OH)$; and trimethylarsenic oxide, $(CH_3)_3AsO$. Methylation to produce the volatile compounds, trimethylarsine [$(CH_3)_3As$] and dimethylarsine [$(CH_3)_2AsH$] has been observed (Masscheleyn et al. 1991).

Biomethylation depends upon both the microorganisms and the arsenic compounds present. Some microorganisms can fully methylate arsenic compounds over a wide range of pH conditions, whereas other microorganisms are more limited in the degree to which they can perform methylation (O'Neill 1990). Conditions that promote microbial activity, such as high organic matter content, warm temperatures, and adequate moisture, often enhance methylation of arsenicals (Woolson 1977).

Fungi, molds, and various strains of bacteria can also demethylate arsenicals. Demethylation of organic arsenicals by these microorganisms occurs through oxidative pathways in which methyl groups are cleaved and oxidized to CO_2 resulting in the production of inorganic arsenate (Bibhas 1975).

3.2 Adsorption

One of the most important processes influencing the behavior and bioavailability of arsenic in soil is its ability to sorb onto solids or particulates. Soil sorption controls the persistence, activity, and movement of arsenic species (Wauchope and McDowell 1984). Factors governing the sorption of arsenic in soil include pH and the amount of clay, iron, aluminum, calcium, and phosphorus present (Woolson et al. 1971).

The pH influences the distribution of arsenic species present in solution and activates the anion adsorption sites on clay minerals thus affecting adsorption. At low pH, hydroxyl groups exposed on the periphery of metal oxides, clay minerals, and amorphous silicate minerals are protonated and acquire a positive charge. These protonated (activated) sites are then available for interaction and adsorption of the arsenical anions present in solution (Sposito 1989).

Arsenic is strongly adsorbed by clay minerals such as kaolinite, limonite, montmorillonite, and vermiculite (Dickens and Hiltbold 1967; Slooff et al. 1990). Adsorption capacities for arsenate in a variety of soils were rated as follows: clay soils > calciferous soils > peat soils > acid woodland > podzol soils > sandy soils (Slooff et al. 1990). Arsenic can be strongly sorbed to electropositive hydroxides such as aluminum, iron, and calcium, which coat clay particles or react with cations in the soil solution (Jacobs et al. 1970b).

Some studies have reported that the insoluble complexes between iron(III) hydroxides and arsenic strongly influence arsenic soil adsorption (Jacobs et al. 1970b; Misra and Tiwari 1963), while other studies such as Johnson and Hiltbold (1969) found much of the particulate-bound arsenic in soils complexed with aluminum. There is evidence, however, that aluminum hydroxides act as the most active adsorbent initially, but iron hydroxides are more favoured over time as equilibrium is reached (Woolson 1973). Livesey and Huang (1981) reported that poorly ordered aluminum and iron compounds, and particularly aluminum components, are closely associated with the arsenic adsorption by soils.

3.3 Persistence/Movement

The extensive use of arsenic pesticides in early pest control programs has increased the residual content in certain soils. For example, arsenic residues of 330 $mg \cdot kg^{-1}$ have been reported in orchard soils with a history of lead arsenate ($PbHAsO_4$) application (Veneman et al. 1983). Another study has indicated that the entire application of lead arsenate to an orchard soil was still in the top 25 cm of the soil 15 years after the last application (Aten et al. 1980).

Arsenic residues in browse and herbaceous vegetation in Douglas fir, ponderosa pine, and western larch forests in which trees were treated with monosodium methyl arsonate (MSMA) for thinning were relatively low. Large quantities of arsenic did not move from the forest floor into the soil, indicating that arsenic residues were tightly bound in the foliage. Only small amounts of arsenic were detected in streams near forests treated with MSMA or cacodylic acid (CA) (or dimethylarsinic acid) (Norris et al. 1983).

Inorganic arsenicals used as soil sterilants, such as sodium arsenite, are persistent in the soil and may remain effective for periods as long as 5–8 years, especially in areas of low rainfall (White-Stevens 1971).

3.4 Leaching

Inorganic arsenic is more mobile than organic arsenic in soil, and arsenite is more mobile than arsenate (Xu et al. 1988). Thus, inorganic arsenic and arsenite might leach into surface waters and groundwaters (NRCC 1978). Despite the relative stability of soluble ionic species, concentrations of arsenicals in porewaters are usually low (O'Neill 1990). It seems that adsorption dominates the partitioning of arsenic to the particulate phase, hence leaching of arsenicals at greater soil depths is well correlated with the decreasing adsorptive capacities of the soil. Essentially, leaching occurs when arsenical application rates exceed the adsorption capacity of the soil. Leaching is pronounced in sandy soils and soils lacking in hydroxides of iron, aluminum, and calcium (Woolson et al. 1971).

3.5 Interactions with Other Elements

Soil solutions contain several different constituents capable of competing for adsorption sites (Roy et al. 1986), particularly phosphorus, selenium, and molybdate. Arsenic and phosphorus are chemically similar and exist as oxyanions, which may compete for fixation sites in soil and for uptake in plants (Asher and Reay 1979; Woolson 1973). Studies on interactions of arsenic with selenium and molybdenum have shown antagonistic action on arsenic uptake by plants. Arsenic, selenium, and molybdenum can compete as inorganic and organic ligands in the soil solution phases (Khattak et al. 1989).

4. BEHAVIOR AND EFFECTS IN BIOTA

4.1 Soil Microbial Processes

Studies on the effects of arsenic on microorganisms and microbial processes are summarized in table 6. The selection process is described in section 6.1.1. Studies reporting exposures to inorganic forms of arsenic have shown that arsenic(III) oxide will produce inhibitory effects on normal urease activity in selected soils, but that the degree of inhibition is dependent on soil type (Tabatabai 1977). Other studies have shown than As(III) is a more effective inhibitor of urease and amidase activity and nitrification than As(V) (Frankenberger and Tabatabai 1981; Liang and Tabatabai 1978; Tabatabai 1977). The opposite effect on acid and alkaline phosphatase activity is evident with As(V) causing greater inhibition of activity (Juma and Tabatabai 1977). Neither As(III) or As(V) were shown to strongly inhibit nitrogen mineralization (Liang and Tabatabai 1978).

4.2 Terrestrial Plants

Uptake of arsenic from soil by terrestrial plants is generally limited to the soluble fraction of arsenic within soil parameters. Measurements of total soil arsenic do not accurately reflect the quantity that

is available to plants since adsorbed arsenicals tend to be strongly bound and unavailable for plant uptake (NAS 1977). Lisk (1972) reported an average value of 0.12 for the ratio of arsenic in terrestrial plants to arsenic in soil. It has been proposed that the same mechanism for phosphorus uptake by plants is used in arsenical uptake (Asher and Reay 1979). The kinetics of arsenic uptake, and its subsequent translocation, can be influenced by plant species, the chemical form of arsenic, and temperature (NRCC 1978).

The degree of arsenic uptake from soil varies widely between plant species. It may also depend on the sensitivity of the plant to phosphorus concentrations in soil (Otte et al. 1990). The ability of a variety of crops to absorb and concentrate arsenic is listed in the following order: radishes > grass > lettuce > carrots > potato tubers > spring wheat (O'Neill 1990). In most cases the concentrations of arsenic in the edible portions of plants are quite low, even when grown on contaminated soil. In general, roots contain higher concentrations then stems, leaves, or fruit (Khattak et al. 1991; O'Neill 1990).

The proposed mode of entry from root uptake is from root to xylem to leaves and then to the phloem; for foliar uptake, the path is from leaves to phloem to roots and xylem (Crafts and Crisp 1971; Foy and Yamaguchi 1965). In a comparative study of absorption and transport of four arsenic herbicides, Sachs and Michael (1971) reported that CA and MSMA in beans *(Phaseolus vulgaris)* were transported about equally from the leaves to the terminal bud and expanding leaves, whereas negligible amounts of sodium arsenite and sodium arsenate were translocated. The inorganic compounds, however, caused more rapid contact injury to the treated leaves than either organic compound.

Terrestrial plants are limited in their ability to metabolize arsenate. In one study, 4-week-old pine seedlings were allowed to take up arsenate for 2 d. There was considerable reduction of arsenate to arsenite in the roots and shoots, but no methylation of inorganics to organoarsenicals occurred (Nissen and Benson 1982). Studies with 7-week-old corn, honey dew melons, peas, and tomatoes also showed the ability of plants to reduce arsenate to arsenite, but again no evidence of methylation was found. Methylation of arsenate did occur, however, in plants that were deficient in phosphorus and nitrogen. No arsenic-lipid compounds were found in leaves even when considerable methylation occurred, although some lipid soluble compounds were found in the roots and thought to be associated with microbial activity (Nissen and Benson 1982).

4.2.1 Bioaccumulation

Though plants grown in arsenic-treated soil may contain considerably greater concentrations of arsenic than plants grown in untreated soils, terrestrial plants rarely contain higher concentrations of arsenic than their substrate (Walsh and Keeney 1975). Grasses growing on contaminated spoil tips from old mines in southwestern England were reported to contain arsenic concentrations as high as 3 460 $mg \cdot kg^{-1}$, but still did not reach the 26 530 $mg \cdot kg^{-1}$ concentrations measured in the soil (Porter and Peterson 1977).

In contrast, the extended metabolic abilities of aquatic macrophytes allow considerable accumulation of arsenic from the water. Lunde (1973) reported arsenic bioconcentration factors for algae of 250–3000. Woolson (1975) reported that many species of marine plants and animals contain naturally high concentrations of arsenic, which is often in the organic form and substantially less toxic than other forms. Arsenic metabolism in aquatic plants has been described by Cooney (1981), Lunde (1973), Nissen and Benson (1982).

4.2.2 Toxicity

The concentrations of arsenic in soil that are toxic to plants span a broad range and overlap with concentrations reported as background and beneficial (Sheppard 1992). A summary of the consulted phytotoxicological data and the selected studies used to derive guidelines is presented in table 7.

The phytotoxic effects of arsenic are manifested in a sudden decrease in water mobility, loss of turgor, root plasmolysis, and discoloration, followed by necrosis. Sensitivity to arsenic appears to be related to an inability to prevent absorption or translocation of arsenic to sensitive sites (O'Neill 1990). Sheppard (1992) found that when all available phytotoxicological data were pooled, there was no significant difference between monocotyledons and dicotyledons in response to soil arsenic concentrations.

The action of arsenic-based pesticides is largely through foliar uptake at the time of application (O'Neill 1990). Continued application of these compounds, however, can lead to high residual levels of arsenic in the soil and continued phytotoxic effects long after application has ceased (Woolson 1983). Toxicity of soil arsenic to terrestrial plants is generally dependent on the concentration of available arsenic and not total arsenic (BIBRA 1990). This is influenced by the degree of adsorption and precipitation of arsenic in soil. The amount of arsenic available in the soil is determined by the organic matter content, the type and amount of clay present, the pH, and the presence of other minerals. In general, arsenic availability is highest in coarse-textured soil having little colloidal material and little ion exchange capacity and lowest in fine-textured soils high in clay, organic material, iron, calcium, and phosphate (NRCC 1978). In an extensive review of soil arsenic and phytotoxic effects, Sheppard (1992) found that inorganic arsenic is five times more toxic in sand and loam soils (geometric mean = 40 mg $As \cdot kg^{-1}$ dry soil) than in clay soils (geometric mean = 200 mg $As \cdot kg^{-1}$ dry soil). The availability of arsenic is also increased in wet (reduced) soils (Peterson et al 1981).

4.3 Terrestrial Invertebrates

A summary of the consulted studies on the effects of arsenic on terrestrial invertebrates and the selected studies used in guideline derivation is presented in table 8.

4.3.1 Bioaccumulation

In assessing the use of earthworms in bioassays, Honda et al. (1984) found that tissue concentrations of arsenic in *Pheretime hilgendorfi* were higher than those of the gut content, which indicates the

high bioaccumulation in the earthworm via uptake from food (mainly soil and humus). However, an experiment by Beyer and Cromartie (1987) reporting arsenic concentrations in earthworms from 20 different contaminated and uncontaminated sites indicated that arsenic does not bioaccumulate to any appreciable degree. The soil arsenic concentrations ranged from 0.77 to 3.5 $mg \cdot kg^{-1}$ dw at the uncontaminated sites and from 1.4 to 33 $mg \cdot kg^{-1}$ dw at the contaminated sites. Earthworms collected from the uncontaminated sites contained trace amounts to 1.5 $mg \cdot kg^{-1}$, while earthworms collected from contaminated sites contained from trace amounts to only 0.81 $mg \cdot kg^{-1}$.

4.4 Livestock and Wildlife

Terrestrial mammal populations may be exposed to various forms of arsenic through inhalation of aerosols or ingestion of particulate adsorbed arsenicals. Uptake of arsenic through ingestion of contaminated forage is not an important route of exposure because concentrations of arsenic in terrestrial plants are generally low (NRCC 1978; O'Neill 1990).

Direct ingestion of arsenic from soil can be a major source of dietary arsenic for grazing livestock. The amount of soil-bound arsenic ingested can account for 60%–75% of total arsenic intake in cattle (Thornton and Abrahams 1983). Only about 1% of this is actually absorbed, with the remainder being excreted directly. Slooff et al. (1990) reported that uptake of arsenic occurs maximally for oral doses and that absorption of arsenic from the gastrointestinal tract is usually rapid (O'Neill 1990).

Many of the studies involving the effects of arsenic on animals have been related to metabolism and excretion. Cows and dogs fed relatively large doses of arsenic rapidly excreted a large portion of the arsenic, showing little effect of accumulation (Lakso and Peoples 1975). Poultry typically fed 3-nitro-4-hydroxyphenylarsonic acid as a growth promoter were able to accumulate amounts in tissues under continuous feeding, but arsenic was rapidly excreted upon discontinued use of the feed additive (Walsh et al. 1977). Swine fed arsanilic acid showed accumulation of an arsenic compound in the liver, but when the dried liver was fed to rats, excretion rates for the compound were even higher than for inorganic arsenicals (NAS 1977).

Excretion rates of a variety of animals have been calculated. In general, smaller animals excrete arsenic at greater rates than larger species, although exceptions do occur (NRCC 1978). Ducoff et al. (1948) injected radio-labelled sodium arsenite into chickens and other animals. Results indicated that chickens were able to rapidly excrete the arsenic, with only 2% remaining in the tissues after 60 h. The slowest excretion rates were for rats, with more than 90% of the arsenic retained after 60 h. Overby and Frederickson (1965) determined a half-life of 36–38 h for arsanalic acid (*p*-aminobenzenearsonic acid) and of 60–63 h for arsenate in hens. In a review document, excretion by the kidneys was reported to account for 60%–90% of the administered arsenic (Slooff et al. 1990). As in plants, metabolism of arsenic by terrestrial animals seems limited. Cows and dogs fed sodium and potassium arsenate were reportedly able to methylate up to 50% of the total body burden (Carton 1976). Methylation of inorganic arsenic most likely occurs within the liver (Slooff et al. 1990).

4.4.1 Bioaccumulation

Arsenic becomes biologically available to waterfowl from soil leachate as a consequence of irrigation. It has been detected in the aquatic plant wigeongrass *(Ruppia maritima)* from waste irrigation water at 430 $mg \cdot kg^{-1}$ and is known to accumulate to high concentrations as sodium arsenate in plant roots and tubers. This exposure route leads to high concentrations of arsenic being consumed by the waterfowl that feed on aquatic plants (Whitworth et al. 1991).

Bird species nesting and feeding on or near arsenic-contaminated soils and wetlands are at high risk. Avian populations are particularly vulnerable near agricultural drainage waters contaminated with leached arsenic and in areas where arsenical herbicides are used. Toxic concentrations of metals have been noted in the diets of many wild and domesticated avian species (Fimreite 1971).

Highest concentrations of arsenic in livestock are generally found in the liver, kidneys, spleen and lungs (Slooff et al. 1990). Excretion of the compounds is rapid for most tissues except in skin, lungs, and hair. These tissues contain proteins with SH groups that can bind trivalent arsenic (Slooff et al. 1990). The relatively fast excretion rates and low exposure routes from plant tissues suggest that terrestrial animals would not bioaccumulate arsenic to a large degree. Sharama and Shupe (1977) have found no relationship between arsenic concentrations in soil and vegetation and concentrations in the livers of rock squirrels *(Spermophilus variegatus)* from different uncontaminated areas in the western United States.

There is no evidence for biomagnification of arsenic through the food chain in either aquatic or terrestrial systems (Brown 1983; Woolson 1975).

4.4.2 Toxicity

A summary of toxicological studies on the effects of arsenic on birds and mammals is presented in table 9. Symptoms of acute toxicity exposure include weakness, kidney disfunction, gastrointestinal disturbance (e.g., diarrhea), abortion, drastic reduction in milk production in cows, and effects on the nervous system (Hesse et al. 1990; NRCC 1978).

Symptoms of chronic exposure to organic forms of arsenic include lack of muscular coordination, muscle atrophy, morphological alteration of erythrocyte structure, demyelination of neurons, skin blemishes, and, in cattle, blindness and pathological changes to internal organs (Hesse et al. 1990; Luh et al. 1973; NRCC 1978). Chronic sublethal exposure to arsenic rarely results in death because of the rapid detoxification and elimination of the compound (Woolson 1975).

5. BEHAVIOR AND EFFECTS IN HUMANS AND MAMMALIAN SPECIES

The supporting documentation and assessment contained in this report were extracted and adapted from EC/HWC (1993), NHW/DOE (1993), Hughes et al.(1994), and HWC (1992).

5.1 Human Exposure Estimates

Estimated daily intakes (on a body weight basis) of inorganic arsenic from environmental media by various age groups in the Canadian population are presented in table 10. Ingestion in water and food represent the principal routes of inorganic arsenic intake for all age groups, followed by soil/dust in infants and children, and water and air for all age groups. Based on limited data on the relative proportion of inorganic arsenic in various foodstuffs, average daily intake from food ranges from <0.08 to 0.3 $\mu g \cdot kg^{-1}$ bw per day for adults and young children, respectively. Intake in soil is estimated to range from 0.003 $\mu g \cdot kg^{-1}$ bw per day in adults to 0.06 $\mu g \cdot kg^{-1}$ bw per day in young children. Based on the limited monitoring data describing arsenic concentrations in the drinking water of Canadians, average daily intake of arsenic (which in surface water supplies is predominantly in the pentavalent inorganic form) from drinking water, for all age groups, is usually <0.5 $\mu g \cdot kg^{-1}$ bw per day. However, intake of inorganic arsenic (both As(III) and As(V)) from some groundwater supplies used as drinking water may be higher. Average daily intake of arsenic from ambient air is estimated to range from 0.0003 to 0.0004 $\mu g \cdot kg^{-1}$ bw per day. Total daily exposure to inorganic arsenic from environmental sources ranges from 0.18 to 0.73 $\mu g \cdot kg^{-1}$ bw per day, with the greatest exposure being in infants (Table 10). Cigarette smoking may contribute an additional 0.01– to 0.04 $\mu g \cdot kg^{-1}$ bw per day.

5.2 Pharmacokinetics

Ingested elemental arsenic is poorly absorbed and largely eliminated, unchanged (HWC 1989). Arsenic oxides are readily absorbed (>80%) from the gastrointestinal tract (Fowler et al. 1979) and, to a lesser extent, through the lungs and skin (Wickstroem 1972). On the basis of fecal recovery experiments in human volunteers, the absorption of soluble As(III) and As(V) compounds is close to 95% (Hindmarsh and McCurdy 1986). Arsenic(III) tends to accumulate in the tissues, whereas As(V) and organic arsenic are well absorbed, but rapidly and almost completely eliminated via the kidneys (Bertolero et al. 1987).

Following ingestion, inorganic arsenic appears rapidly in the circulation, where it binds primarily to hemoglobin (Alexson 1980) ; within 24 h, it is found mainly in the liver, kidneys, lungs, spleen, and skin (Wickstroem 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 sulfhydryl 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.

There appear to be two main processes, with different rates, for the elimination of ingested trivalent arsenic from the body (Lovell and Farmer 1985). The first is rapid urinary excretion of nonmethylated arsenic in both the trivalent and pentavalent forms (close to 90% of the total urinary arsenic over the first 12-h period). The second involves detoxification by sequential methylation of As(III) in the liver to dimethylarsinic acid (DMAA) (Buchet and Lauwerys 1985; Lovell and Farmer 1985). Excretion of the methylated compounds commences approximately 5 h after ingestion, but

reaches its maximum level 2–3 d later. The half-life of inorganic arsenic in humans is estimated to be between 2 and 40 d (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 five 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 d) (Buchet et al. 1981b; Luten et al. 1982; Tam et al. 1982).

5.3 Essentiality

It has been postulated that arsenic might be an essential element in animals, as there is evidence that such essentiality is plausible, but there is no indication that arsenic is essential to humans (U.S. EPA 1988).

5.4 Toxic Effects

5.4.1 Acute and Short-term Toxicity

The acute toxicity of inorganic arsenic compounds increases with solubility in water. Soluble salts, such as sodium and potassium arsenates and arsenites, arsenic pentoxide, arsenic trichloride, and arsenic acid, are highly acutely toxic to mammals, with LD_{50}s in rats, mice, and rabbits ranging from 2.6 to approximately 40 mg $As \cdot kg^{-1}$ bw (BIBRA 1990). The reported LD_{50}s for the less-soluble arsenic trioxide and calcium arsenate, the virtually insoluble lead arsenate, and the insoluble magnesium arsenate, ranged from 2.2 to 230 mg $As \cdot kg^{-1}$ bw in rats, mice, rabbits, and dogs, while the acute toxicity of insoluble gallium arsenide and nickel subarsenide is much lower (LD_{50}s in the rat of more than 7.8 g $As \cdot kg^{-1}$ bw and 1 g $As \cdot kg^{-1}$ bw, respectively) (BIBRA 1990). The lowest reported no-observed-effect level (NOEL) following inhalation in limited available short-term and subchronic studies is 1.3 $\mu g \cdot m^{-3}$ arsenic trioxide, based on the observation of slower conditioned reflexes and histological changes in the brain, liver, and lungs of rats at the higher concentration (0.005 $mg \cdot m^{-3}$) (Rozenshtein 1970).

The acute toxicity of ingested inorganic arsenic in humans also increases with the solubility of the compound. The lethal dose in humans is estimated to be approximately 50–300 mg (0.8–5 $mg \cdot kg^{-1}$ bw) of arsenic trioxide, although severe effects have been reported following ingestion of as little as 20 mg, and persons have survived oral doses of approximately 10 g (BIBRA 1990).

5.4.2 Chronic and Long-term Toxicity

In studies with experimental animals, the main organs affected by repeated oral doses of arsenic compounds are the liver and kidneys, although effects have also been noted on the spleen, body weight, and various hematological and biochemical parameters.

The no-observed-adverse-effect level (NOAEL) for chronic ingestion of arsenic appears to be in the region of 1.5–3.5 $mg \cdot kg^{-1}$ bw per day, based on 2-year studies in which rats and dogs were administered arsenic in the diet (BIBRA 1990). However, toxic effects, such as reduced growth and litter size and histological changes in the liver, kidney, spleen, and skin, have occurred ≤ 1.5 mg $As \cdot kg^{-1}$ bw per day when arsenic is administered in the drinking water of rats or mice for up to 2 years (Byron et al. 1967; Schroeder and Balassa 1967; Schroeder and Mitchener 1971).

Chronic ingestion of arsenic in humans has been associated with adverse effects on the skin, including hyperkeratosis of the palms and soles and hyperpigmentation in several epidemiological studies. There has been an exposure–response relationship between concentrations of arsenic in drinking water supplies and the prevalence of noncancerous dermal lesions in studies in Taiwan and Chile (Borgono and Greiber 1972; Borgono et al. 1977; Borgono et al. 1980; Tseng 1977; Tseng et al. 1968). Similar dermatological effects have been reported in several studies in workers occupationally exposed to airborne arsenic (BIBRA 1990).

Ingestion of arsenic in drinking water has been associated with an increased prevalence of "blackfoot disease", a peripheral vascular disease leading to gangrene of the toes and feet, in a population of 40 421 residents in Taiwan whose well water contained up to 1820 $\mu g \cdot L^{-1}$ arsenic (Tseng 1977; Tseng et al. 1968). This effect has not been observed, however, in populations ingesting drinking water containing high concentrations of arsenic in other countries, and it has been suggested that other compounds in the water supply may play a role in the development of the disease (Chen et al. 1988; Lu 1990a, 1990b).

Mixed results have been obtained in numerous epidemiological studies designed to investigate the association between exposure to arsenic and mortality due to cardiovascular and cerebrovascular causes (BIBRA 1990; Wu et al. 1989). However, in most studies in which an increase in mortality due to these causes was reported (Lee and Fraumeni 1969; Lubin et al. 1981; Wall 1980; Welch et al. 1982), there was no relationship between excess mortality and average concentration of arsenic or duration of exposure.

5.4.3 Carcinogenicity

The results of early, limited carcinogenicity bioassays, in which experimental animals have been administered arsenic compounds by ingestion or inhalation, have been largely negative (U.S. EPA 1984). In some more recent studies, increases in the incidence of primarily pulmonary adenomas were observed in hamsters following weekly intratracheal instillations of calcium arsenate and arsenic trioxide for 15 weeks (Pershagen and Bjorklund 1985; Yamamoto et al. 1987), whereas in other similar studies, results for arsenic trioxide, gallium arsenide, and arsenic trisulphide were negative (Ohyama et al. 1988; Pershagen and Bjorklund 1985; Yamamoto et al. 1987). The results of initiation/promotion studies have provided some limited evidence that some arsenic compounds may act as tumor promoters in experimental animals (Shirachi et al. 1986, 1987). Arsenic appears to induce clastogenic damage rather than gene mutations in short-term in vitro and in vivo assays, and induces transformation in mammalian cells in vitro (BIBRA 1990; IARC 1987).

Excesses in mortality due to lung cancer have been associated with exposure to arsenic in numerous analytical epidemiological studies of workers in smelters and arsenical pesticide production facilities, with considerable evidence of an exposure–response relationship. Where it was possible to investigate the influence of possible confounders, such as concomitant exposure to sulfur dioxide or cigarette smoke, these factors have not explained this excess. In addition, in the populations for which exposure has been the most extensively characterized (i.e., three cohorts of workers employed at smelters in Washington, Montana, and Sweden), there was a clear exposure–response relationship between airborne arsenic levels and mortality due to lung cancer (Enterline et al. 1987; Higgins et al. 1986; Jarup et al. 1989). Although not as well studied, there is also evidence that cancers at sites other than the lung, including the stomach, colon, liver, and urinary system, may be associated with occupational exposure to arsenic (Gibb and Chen 1989).

Associations between the ingestion of arsenic in drinking water, or in the form of a medicinal, and skin cancer have also been observed in numerous case studies, case series, and epidemiological investigations in Taiwan (Chen et al. 1985; Tseng 1977; Tseng et al. 1968), Central and South America (Bergoglio 1964; Biagini et al. 1974; Cebrian et al. 1983; Zaldivar 1974, 1977), and England (Philipp et al. 1983).

The largest and most sensitive epidemiological study was conducted in Taiwan where a population of 40 421 was divided into three groups, based on the arsenic content of their well water (high ≥ 0.60 mg·L^{-1}, medium 0.30–0.59 mg·L^{-1}, and low 0.01–0.29 mg·L^{-1}) (Tseng 1977). There was a clear exposure–response relationship between the arsenic concentration of drinking water and the prevalence of skin cancer (Tseng 1977; Tseng et al. 1968). However, HWC (1989) found several methodological weaknesses and potential confounding factors that complicate the interpretation of the results of this investigation. For example, the investigators were not "blinded" as to whether persons being examined were from the arsenic-endemic area. The socioeconomic conditions in the study area are poor, and the population subsists on food somewhat low in protein and fat (Tseng 1977). Exposure to arsenic from sources other than drinking was not also examined, and there is some, albeit not well-documented, indication that these sources may have contributed significantly to the total exposure of the Taiwanese population (Heydorn 1970; Li et al. 1979).

There is also more recent evidence of an association between the ingestion of drinking water containing high concentrations of arsenic and increased mortality due to various cancers of internal organs, including the bladder, kidney, and lung, in exposed populations in Taiwan (Chen et al. 1986; Wu et al. 1989). Hepatic angiosarcoma (a rare tumour of the liver) and lung cancer have also been reported in patients ingesting arsenic medicinals (Falk et al. 1981; Lander et al. 1975; Popper et al. 1978; Regelson et al. 1968; Roat et al. 1982; Robson and Jelliffe 1963).

5.4.4 Immunotoxicity

Possible impairment of the immune system has been reported in an unconfirmed study of a small number of individuals drinking water containing 390 μg·L^{-1} of arsenic (Ostrosky-Wegman et al. 1991). Adverse effects on the immune system, including increased susceptibility to bacterial and viral infection, suppressed humoral response, and a reduction in the number of T lymphocytes, have been

reported in several studies in which mice were exposed to arsenic by inhalation or ingestion (BIBRA 1990).

5.4.5 Genotoxicity and Related Effects

Arsenic does not appear to be mutagenic in bacterial and mammalian assays, although it can 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; consequently, the damage caused by arsenic has no genetic consequences. 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. Arsenic has also been shown to block dividing cells in the S and G2 phases (Petres et al. 1977).

5.4.6 Neurotoxicity

Although slight neurological effects were reported in a small group of individuals in Nova Scotia whose well water contained more than 0.05 ppm (50 $\mu g \cdot L^{-1}$) arsenic (Hindmarsh et al. 1977), no adverse neurological effects were observed in two larger studies of populations exposed in some cases to higher concentrations (Kreiss et al. 1983; Southwick et al. 1983).

5.4.7 Reproductive Toxicity and Teratogenicity

Little recent information is available on the developmental and reproductive toxicity of arsenic. Fetotoxic effects have been observed at arsenic doses that are not maternally toxic (i.e., ≥ 8 $mg \cdot kg^{-1}$ bw per day) (Matsumoto et al. 1973), but teratogenic effects have been observed only at doses that were toxic to the dams. Available data on the reproductive toxicity of arsenic are limited largely to early studies, in which the lowest concentration reported to induce effects (decreased litter size and increased ratio of male to female offspring in a three generation study in mice) was 1.5 $mg \cdot kg^{-1}$ bw per day (Schroeder and Mitchener 1971).

There is no conclusive evidence that arsenic causes any adverse reproductive effects in humans, based on several very limited studies (Aschengrau et al. 1989; Beckman and Nordstrom 1982; Nordstrom et al. 1978a, 1978b; 1979a, 1979b).

In a case-control study of 270 children with congenital heart disease and 665 healthy children, maternal consumption during pregnancy of drinking water containing detectable arsenic concentrations 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 95% confidence limits of 1.3–8.9) (Zierler et al. 1988). However, there was no adjustment for maternal age, socioeconomic status, or previous reproductive history.

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 $mg \cdot L^{-1}$ and 0.0014–0.0019 $mg \cdot L^{-1}$ 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.

The following review on the teratogenicity of inorganic arsenic was extracted from HWC (1992). In early studies, teratogenic effects of arsenic in chicks, golden hamsters, and mice were reported (Hood and Bishop 1972; Zierler et al. 1988). In a more recent investigation (Ferm and Hanlon 1985), arsenate was teratogenic in the offspring of pregnant hamsters following exposure on days 4–7 of gestation by mini-pump implantation. The threshold blood level for teratogenesis was 4.3 $\mu mol \cdot kg^{-1}$ (Hanlon and Ferm 1986a). The specific form of arsenic that is responsible for teratogenesis is not known, although there is evidence to suggest that it is arsenite rather than arsenate (Hanlon and Ferm 1986b). In studies with mice and hamsters, MMAA (monomethylarsonic acid) and DMAA (dimethylarsinic acid) have been considerably less teratogenic than As(III) or As(V) (Lovell and Farmer 1985).

5.5 Classification and Evaluation

On the basis of documented carcinogenicity in human populations by more than one route of exposure (i.e., both inhalation and ingestion), HWC (1992) and EC/HWC (1993) have classified "the group of inorganic arsenic compounds as a whole" in Group I ("carcinogenic to man") of the classification scheme developed by the Bureau of Chemical Hazards for use in the derivation of the guidelines for Canadian drinking water quality (HWC 1989).

As 77% of Canadians live in cities (Statistics Canada 1993), an urban exposure scenario is the most common situation expected to arise. Given that 82% of these urban dwellers receive treated water supplies, mostly from surface water sources (Tate and Lacelle 1992), then the most likely route of exposure to contaminants in soil is expected to be direct soil ingestion (intentional [i.e., pica] or unintentional), for nonvolatile compounds. However, because no risk-specific doses have been estimated for direct soil ingestion, risk estimates from the ingestion of drinking water are used based on an adult daily exposure scenario.

Based on the increased incidence of skin cancer observed in the Taiwanese population (Tseng et al. 1968), the U.S. EPA has 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 (HWC 1992). On the basis of a review of available data, HWC (1992) has concluded that the EPA model was the most appropriate for estimation of the skin cancer risks associated with the

ingestion of arsenic in drinking water. The estimated doses corresponding to lifetime skin cancer risks of 10^{-4}, 10^{-5}, 10^{-6}, and 10^{-7} (based on Taiwanese men) were as follows (HWC 1992):

Lifetime incremental risk	Dose ($\mu g \cdot kg^{-1}$ bw per day)
10^{-4}	0.06
10^{-5}	0.006
10^{-6}	0.0006
10^{-7}	0.00006

However, HWC (1992) emphasized that these estimates probably represent a worst-case situation because of concomitant exposure to other compounds in the water in Taiwan and possible dietary deficiencies of the Taiwanese population compared with North American populations. Moreover, methylation (i.e., detoxification) of inorganic arsenic is progressively but not completely saturated when daily intake of humans exceeds approximately 500 µg, and this has not been taken into consideration in derivation of the quantitative estimates of risk (HWC 1992).

6. DERIVATION OF ENVIRONMENTAL AND HUMAN HEALTH SOIL QUALITY GUIDELINES

6.1 Environmental Soil Quality Guidelines

6.1.1 Introduction

Canadian soil quality guidelines are designed to protect four different land uses: agricultural, residential/parkland, commercial, and industrial. The Canadian soil quality guidelines for arsenic are based on the procedures described in A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines (CCME 1996).

All data selected for use in the following derivations have been screened for ecological relevance and are presented in tables 6, 7, 8, and 9. Studies consulted, but not used for guideline derivation are also presented in these tables. Studies were excluded from use for one or more of the following reasons: soil pH was not reported or was below 4 (which is outside pH range of most Canadian soils); no indication of soil texture was provided; inappropriate statistical analysis was used; test was not conducted using soil or artificial soil; test soil was amended with sewage sludge or a mixture of toxicants; or test did not use controls.

Lowest-observed-effect concentration (LOEC) and effect concentration (EC) data used in the following derivations were considered to be biologically significant in addition to statistically significant according to the study from which the data were taken.

According to Section 7.5.2.2 of the protocol (CCME 1996), the geometric mean should be used when multiple data are available for the same endpoint with the same species. For the arsenic data, the geometric mean has been applied to the Woolson (1973) study. Consequently, the number of data points for each species has been reduced to two: the no-observed-effect concentration (NOEC) and the lowest-observed-effect concentration (LOEC) at which the first adverse effects were observed.

6.1.2 Soil Quality Guidelines for Agriculture and Residential/Parkland Land Uses

6.1.2.1 Soil Quality Guidelines for Soil Contact

The derivation of the soil quality guideline for soil contact (SQG_{SC}) is based on the toxicological data for terrestrial plants and soil invertebrates, selected according to CCME (1996), presented in tables 7 and 8. Sufficient toxicological data are available to use the preferred weight of evidence method for guideline derivation.

Therefore the threshold effects concentration (TEC) was calculated as

$$TEC = NPER / UF$$

where,
TEC = threshold effects concentration ($mg \cdot kg^{-1}$ soil)
NPER = no potential effects range (25th percentile of distribution)
UF = uncertainty factor (if needed); an uncertainty factor was not applied because of the relatively large amount of data available.

Out of a total of 48 data points, the 25th percentile corresponds to the 12th datum point, which is 19 mg $As \cdot kg^{-1}$ soil from the Jacobs et al. (1970a) study on the growth reduction of snap beans.

Thus,
TEC = 19 mg $As \cdot kg^{-1}$ soil

Nutrient and Energy Cycling Check

Insufficient data on microbial processes were available to ascertain the effects of arsenic on nutrient and energy cycling at the level of the above calculated ECL; the only selected study is summarized in table 6. Therefore, the SQG_{SC} for agricultural and residential/parkland land uses is equivalent to the TEC of 19 $mg \cdot kg^{-1}$ soil.

6.1.2.2 Soil Quality Guideline for Soil and Food Ingestion

The soil quality guideline for soil and food ingestion (SQG_I) applies only to agricultural land use. Calculation of the SQG_I is based on the lowest-observed-adverse-effect level (LOAEL) taken from

the selected mammalian and avian toxicological data listed in table 9. The LOAEL, indicating the species most threatened, is 8 mg As·kg^{-1} bw·day^{-1}, an LD_{50} dose for rabbits (Sullivan 1969).

The LOAEL is used to calculate the daily threshold effects dose (DTED) according to the following equation:

$$DTED = \text{lowest } LOAEL / UF$$

where

DTED = daily threshold effects dose (mg·kg^{-1} bw per day)

LOAEL = lowest-observed-adverse-effect level (mg·kg^{-1} bw per day)

UF = uncertainty factor; an uncertainty factor of 2 was applied because the study was an acute lethal study.

DTED = 8 / 2 = 4 mg·kg^{-1} bw per day

An animal may be exposed to a contaminant by more than one route. Total exposure comes from a combination of contaminated food, direct soil ingestion, dermal contact, contaminated drinking water, and inhalation of air and dust. Exposure from all of these routes should not exceed the DTED. Assuming that drinking water, dermal contact, and inhalation account for 25% of the total exposure (CCME 1996), the remaining 75% of exposure is attributed to the ingestion of food and soil. It follows, then, that exposure from soil and food ingestion should not exceed 75% of the DTED.

$$\text{Exposure from direct soil ingestion} + \text{exposure from food ingestion} = 0.75 \cdot DTED$$

Exposure from Direct Soil Ingestion

To estimate the exposure of an animal from direct soil ingestion, the rate of soil ingestion must be calculated. The ingestion rate of soil and forage together is referred to as the dry matter intake rate (DMIR). To estimate the rate of soil ingested directly, the percentage of the DMIR attributed to soil ingestion must be isolated. In most soil-based exposure studies, the proportion of soil ingested (PSI) is reported with the DMIR. The animal's soil ingestion rate is calculated as a proportion of the DMIR according to the following equation:

$$SIR = DMIR \cdot PSI$$

where

SIR = the soil ingestion rate (kg dw soil·d^{-1})

DMIR = the geometric mean of available dry matter intake rates (kg·day^{-1}) (P. Warrington 1995, British Columbia Ministry of the Environment, Lands, and Parks, pers. com.)

PSI = geometric mean of available soil ingestion proportions reported with the DMIR; as no information is available on the PSI for the species used, a default value of 0.077 (McMurter 1993) was used for the above equation.

Thus,

SIR = $0.18 \cdot 0.077 = 0.014$ kg dw soil·d^{-1}

The SIR can then be combined with the bioavailability factor (BF), body weight (bw), and a concentration of the contaminant in the soil (SQG_I) to represent the exposure from soil ingestion. The soil concentration at this point is unknown, but it should not provide for greater than 75% of the DTED when combined with the exposure calculated for food ingestion.

$$\text{Exposure from soil ingestion} = SIR \cdot BF \cdot SQG_I / bw$$

where,

SIR = soil ingestion rate (kg dw soil·d^{-1})

BF = bioavailability factor; because of lack of specific information on the bioavailability of arsenic from ingested soil for livestock and terrestrial wildlife, a bioavailability factor (BF) of 1 is assumed (CCME 1996).

SQG_I = concentration of the contaminant in soil that will not result in >75% DTED (mg·kg^{-1} soil)

bw = mean body weight (kg); mean body weight of rabbits was determined to be 3 kg (P. Warrington 1995, British Columbia Ministry of the Environment, Lands, and Parks, pers. com.)

Exposure from Food Ingestion

Similar to the SIR, the food ingestion rate (FIR) for livestock and wildlife, is expressed as a portion of the DMIR. The FIR is the remaining portion of the DMIR minus the soil ingestion rate. The FIR is calculated as

$$FIR = DMIR - SIR$$

where,

FIR = food ingestion rate (kg dw food·d^{-1})

DMIR = geometric mean of dry matter intake rates (kg dw food·d^{-1}) (P. Warrington 1995, British Columbia Ministry of the Environment, Lands, and Parks, pers. com.)

SIR = soil ingestion rate (kg dw soil·d^{-1})

Thus,

FIR = $0.18 - 0.014 = 0.166$ kg dw food·d^{-1}

The FIR can then be combined with the bioconcentration factor (BCF), bw, and the SQG_I to express the exposure from food ingestion.

$$\text{Exposure from food ingestion} = FIR \cdot BCF \cdot SQG_I / bw$$

where,

FIR = food ingestion rate (kg dw food·d^{-1})

BCF = bioconcentration factor; calculated to be 0.059 (the geometric mean of soil to plant ratios taken from Jacobs et al. [1970a], Anastasia and Kender [1973], Rosehart and Lee [1973], Sheppard et al. [1985], and Wiersma et al. [1986])

SQG_I = concentration of the contaminant in soil that will not result in >75% DTED ($mg \cdot kg^{-1}$ soil)
bw = mean body weight (kg); the mean body weight of rabbits was determined to be 3 kg (P. Warrington 1995, British Columbia Ministry of the Environment, Lands, and Parks, per. com.)

Exposure from Direct Soil Ingestion and Food Ingestion

The equations for exposure from soil ingestion and exposure from food ingestion can be combined and rearranged to solve for the SQG_I.

$$[(SIR \cdot BF \cdot SQG_I) / bw] + [(FIR \cdot BCF \cdot SQG_I) / bw] = 0.75 \text{ DTED}$$

$SQG_I = (0.75 \cdot DTED \cdot bw) / (SIR \cdot BF + FIR \cdot BCF)$
$SQG_I = (0.75 \cdot 4 \cdot 3) / (0.014 \cdot 1 + 0.166 \cdot 0.059)$
$SQG_I = 380 \text{ mg} \cdot \text{kg}^{-1}\text{soil}$

6.1.3 Soil Quality Guidelines for Commercial and Industrial Land Uses

6.1.3.1 Soil Quality Guidelines for Soil Contact

The derivation of the soil quality guidelines for soil contact (SQG_{SC}) for commercial and industrial land uses is also based on toxicological data for terrestrial plants and soil invertebrates presented in tables 7 and 8. However, for commercial and industrial land uses only. the effects data are used and uncertainty factors are not applied. Sufficient toxicological data were available to use the preferred weight of evidence method for guideline derivation.

$$ECL = ERL$$

where,
ECL = effects concentration low ($mg \cdot kg^{-1}$)
ERL = effects range low (25th percentile of effects data distribution; $mg \cdot kg^{-1}$)

Out of a total of 29 data points, the 25th percentile corresponds to the 7th datum point of 26 $mg \cdot kg^{-1}$ soil from the Jacobs et al. (1970a) study on the growth reduction of snap beans.

ECL = 26 mg $As \cdot kg^{-1}$ soil

Nutrient and Energy Cycling Check

Insufficient data on microbial processes were available to ascertain the effects of arsenic on nutrient and energy cycling at the level of the above-calculated ECL; the only selected study is summarized in table 6. Therefore, the SQG_{SC} for commercial and industrial land uses is equivalent to the ECL of 26 mg $As \cdot kg^{-1}$ soil.

6.1.4 Derivation of the Final Environmental Soil Quality Guidelines

The following environmental soil quality guidelines (SQG_E) are optimized for soils within the pH range of 4–7.6. The toxicological studies upon which these guidelines are based were conducted within this pH range. A summary of all the derived soil quality guidelines is presented in table 11.

Agricultural Land Use

The lower value from the two procedures (SQG_{SC} and SQG_L) is selected as the final environmental soil quality guideline for agricultural land. The lower of the two procedures is the SQG_{SC}. Therefore, the final SQG_E is 19 $mg \cdot kg^{-1}$ soil.

Residential/Parkland Land Use

The SQG_{SC} of 19 $mg \cdot kg^{-1}$ soil is the final SQG_E for residential/parkland land use.

Commercial and Industrial Land Use

The SQG_{SC} of 26 $mg \cdot kg^{-1}$ soil is the final SQG_E for commercial and industrial land uses.

6.1.5 Data Gaps

There is a critical need for data on the effects of arsenic on nutrient and energy cycling by microorganisms. Although several studies exist that examine the effects on enzyme activity, there are insufficient data on nitrogen mineralization and carbon cycling to calculate the concentration of arsenic in soil that would be protective of microbes.

6.2 Human Health Soil Quality Guidelines

Human health soil quality guidelines are defined for residential/parkland and industrial land uses and for the protection of groundwater. The Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines (CCME 1996) can be used to establish guidelines for agricultural and commercial land uses, if desired. The following derivations have been based on inorganic forms of arsenic, as these forms cause the main toxicity in human receptors.

6.2.1 Human Health Soil Quality Guideline Protocol

Inorganic arsenic has been classified in the Group I category of substances (i.e., carcinogenic to man) (HWC 1992; EC/HWC 1993). Thus, it is considered as a nonthreshold toxicant (a substance for which there is considered to be some probability of harm from the critical effect at any level of exposure) requiring the development of soil quality guidelines that employ a critical risk-specific dose (RsD), based on lifetime incremental risks from soil ingestion (CCME 1996). For all land uses, the adult was chosen as the receptor when considering lifetime cancer risk (CCME 1996). The following derivation provides guideline values at estimated lifetime incremental risks from 10^{-4} to 10^{-7} which

are applicable to all land uses for which human health soil quality guidelines are derived (i.e., residential and industrial). The selection of acceptable levels of risk is a policy decision. The CCME Subcommittee on Environmental Quality Criteria for Contaminated Sites recommends the development of a soil guideline based on an incremental risk from soil exposure of 10^{-6} (CCME 1996). It should be noted that the 10^{-6} incremental risk above background falls within the range ($<10^{-5}$ to 10^{-6}) considered to be "essentially negligible" in the derivation of MACs (maximum acceptable concentration) for carcinogenic chemicals in drinking water (HWC 1989).

Absorption factors may be required where the critical toxicity study used in developing the cancer slope factor has used an absorbed dose rather than an administered dose or where the critical toxicity study has used a different medium in developing the cancer slope factor than that under investigation. Then soil ingestion rates are multiplied by corresponding absorption factors (AF), when these data are available.

6.2.2 Residential and Industrial Land Uses (Adult Receptor)

Using the above information, a guideline that applies to an adult lifetime cancer risk for urban residential/parkland (playground) and industrial soil exposure can be determined as follows:

$$IRRSQG_{HH} = [(RsD \times BW) / (AF_i \times SIR)] + BSC$$

where,

$IRSSQG_{HH}$	=	incremental risk specific human health soil quality guideline ($\mu g \cdot g^{-1}$);
RsD	=	risk specific doses ($\mu g \cdot kg^{-1}$ $bw \cdot d^{-1}$) corresponding to lifetime incremental risks above background (see section 4.5 for doses);
bw	=	body weight for adult = 70 kg (Health Canada 1994);
BSC	=	background soil concentration = 10.0 $\mu g \cdot g^{-1}$ (see section 2.4 and table 3);
AF_i	=	absorption factor from gut (medium specific)[1] = 1 by default;
SIR	=	soil ingestion rate = 0.02 $g \cdot d^{-1}$ (Health Canada 1994).

Thus, $IRSSQG_{HH}$ values between 220 and 10 $mg \cdot kg^{-1}$ can be derived using lifetime incremental risks above background from 10^{-4} to 10^{-7}, respectively. Using the incremental risk above background of 10^{-6} recommended by CCME (1996), the $IRSSQG_{HH}$ is 12 $mg \cdot kg^{-1}$, resulting in a total estimated risk (including background) of 5.7×10^{-6}.

The background concentration for inorganic arsenic, 10 $\mu g \cdot g^{-1}$, was the highest reported mean concentration in urban environments not directly impacted by point sources. The incremental risk-specific guidelines for residential/parkland and industrial land uses may be below soil background levels in some areas of Canada. Where local or regional background soil concentrations of arsenic differ markedly from the 10 $\mu g \cdot g^{-1}$, then site- or region-specific guidelines should be derived, incorporating the local/regional background soil concentrations.

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[1] This factor accounts for the attenuation of exposure resulting from the absorption of a contaminant to a specific environmental medium (e.g., soil). For practical purposes in this case, this factor is 1 by default, due to lack of data.

6.2.3 Protection of Groundwater Used as Raw Water for Drinking

No guideline for protection of groundwater used as a source of raw water for drinking was derived for inorganic arsenic because of constraints on the mathematical model when applied to inorganic compounds (CCME 1996).

6.2.4 Data Gaps

Data on the levels of arsenic in indoor air, dust, and consumer products were very limited or unavailable for this report. Research is needed to determine the levels of arsenic forms in these environmental compartments. In general, more recent data are needed to better estimate daily intakes of arsenic from various media.

Additional information on the occupational exposure of workers to arsenic and potential confounders (e.g., cigarette smoking) is needed to better estimate cancer potency associated with the inhalation of arsenic. Research is needed on the estimation of cancer risk from the exposure of arsenic through the ingestion of soil and/or dust as well as media other than drinking water. More recent data on the carcinogenicity of arsenic to experimental animals and humans is needed to replace much of the dated existing information.

6.2.5 Human Health Conclusion

As a human carcinogen, no specific soil guideline above background is recommended for inorganic arsenic. Risk-specific human health soil quality guidelines of 220, 31, 12, and 10 $\mu g \cdot g^{-1}$ dry soil ($mg \cdot kg^{-1}$ dry soil) for residential/parkland and industrial land uses with associated lifetime risks of 10^{-4}, 10^{-5}, 10^{-6}, and 10^{-7} above background, respectively, are proposed.

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TABLES

Table 1. Physical and Chemical Properties of Arsenic and Selected Inorganic Arsenic Compounds

Property	Arsenic	Arsenic acid	Arsenic pentoxide	Arsenic trioxide	Calcium arsenate	Gallium arsenide	Sodium arsenate	Sodium arsenite
Empirical formula	As	H_3AsO_4	$As_2O_5(As_4O_{10})$	$As_2O_3(As_4O_6)$	$Ca_3(AsO_4)_2$	GaAs	Na_2HAsO_4	$NaAsO_2$
Molecular weight	74.92	150.95	229.84	197.84	398.08	144.64	185.91	129.91
Valence	0	+5	+5	+3	+5	-3	+5	+3
CAS number	7440-38-2	7778-39-4	1303-28-2	1327-53-3	7778-44-1	1303-00-0	7778-43-0	7784-46-5
Common synonyms	Arsenic black, colloidal arsenic, gray arsenic	Orthoarsenic acid	Arsenic (v) oxide, arsenic acid anhydride, diarsenic pentoxide	Arsenic oxide, arsenious acid, arsenious oxide, white arsenic	Calcium orthoarsenate, arsenic acid, calcium salt	Gallium monoarsenide	Disodium arsenate, sodium biarsenate, arsenic acid, disodium salt	Arsenious acid, sodium salt, sodium metaarsenite
Color	Gray	White	White	White	Colorless	Dark gray	—	Gray-white
Physical state	Solid	Solid	Solid	Solid	Solid	Solid	Solid	Solid
Melting point	817°C at 28 atm	35.5°C	Decomposes at 315°C	312.3°C	1.455°C	1238°C	57°C	—
Boiling point	613°C sublimes	Loses H_2O at 160°C	—	465°C	—	—	—	—
Density ($g \cdot cm^{-3}$)	5.727	2.0–2.5	4.32	3.738	3.62	5.31	1.87	1.87
Vapor pressure	1 mm Hg at 372°C 40 mm Hg at 483°C, 100 mm Hg at 518°C	—	—	66.1 mm Hg at 312°C	—	—	—	—
Solubility in water	Insoluble; soluble in nitric acid	Soluble in 3020 $g \cdot L^{-1}$ at 12.5°C	Soluble in 1500 $g \cdot L^{-1}$ at 16°C, 658 $g \cdot L^{-1}$ at 20°C, 767 $g \cdot L^{-1}$ at 100°C; soluble in acid	Soluble in 37 $g \cdot L^{-1}$ at 20°C, 101 $g \cdot L^{-1}$ at 100°C; soluble in HCl	Soluble in 0.13 $g \cdot L^{-1}$ at 25°C; soluble in dilute acids	—	Soluble	Very soluble
Solubility in organic solvents	—	Soluble in alcohol	Soluble in alcohol	Soluble in glycerin; slightly soluble in alcohol	Insoluble	—	Slightly soluble in alcohol	Slightly soluble in alcohol

Source: Adapted from ATSDR 1993.

Table 2. Arsenic Concentrations in Canadian Glacial Till

Sampling site	Size fraction	Number of samples	Arsenic content (μg·g⁻¹ dw)			Sample preparation/ analytical method*	Reference
			Mean ± std. dev.	Range	Median		
Central Newfoundland	<0.002 mm	838	47.5 ± 98.2	1–2138	24	ICP–AES	Klassen 1994
Southeastern Cape Breton, Nova Scotia	<0.063 mm	324	—	2.5–128	<5	ICP–AES	McClenaghan et al. n.d.
South Miramichi	<0.002 mm <0.063 mm	873 873	34.2 17	0.5–664 1–310	—	—	Lamothe 1990
Central Miramichi	<0.002 mm <0.063 mm	1782 1782	38 15.2	1–2000 0.5–398			
North Miramichi	<0.002 mm <0.063 mm	1368 1368	46 23.7	1–640 3–201			
Southeastern Ontario and southwestern Quebec	<0.002 mm	2129	5 ± 18.7	1–430	2	AAS	Kettles and Shilts 1994
Northeastern Ontario, east of Timmins and north of Kirkland Lake	<0.063 mm	279	—	0.5–44	1	DCP–ES	McClenaghan 1990
Manitouwadge-Hornpayne region, Ontario	<0.002 mm	603	9.6 ± 9.9	1–61	6	ICP–AES Hot aqua-regia leach	Kettles 1994
Beardmore-Geraldton area, east of Lake Nipigon, Ontario	<0.063 mm	505	5.4 ± 7.9	3–85	3	ICP–AES Nitric aqua-regia partial leach	Thorleifson and Kristjansson 1990
Southeastern Manitoba, Bissett-English Brook area and Rice Lake-Greenstone Belt	<0.002 mm	208	53.7 ± 233.6	1–2704	14	ICP–AES Nitric aqua-regia partial leach	Henderson 1994
Cormorant Lake area, Saskatchewan/Manitoba	<0.002 mm	1591	—	1–6590	—	ICP–AES Nitric aqua-regia partial leach	MacMartin et al. 1994
Manson River/Fort Fraser area, British Columbia	<0.002 mm <0.063 mm <0.063 mm	402 401 400	—	<5–112 <5–73 1.9–69.8	9 5 13	ICP–AES Nitric aqua-regia partial leach INAA	Plouffe and Ballantyne 1993
Fish Lake area, British Columbia	<0.002 mm <0.063 mm <0.063 mm	78 108 108	—	0–38 0–7 0.5–31	9 7 <9	ICP–AES Nitric aqua-regia partial leach INAA	Plouffe and Ballantyne 1994

Sampling site	Size fraction	Number of samples	Arsenic content ($\mu g \cdot g^{-1}$ dw)			Sample preparation/ analytical method*	Reference
			Mean ± std. dev.	Range	Median		
Kaminak Lake/Turquetil Lake area, District of Keewatin, Northwest Territories	<0.063 mm	2264	—	2.4–20	<4	ICP–AES Nitric aqua-regia partial leach	Shilts and Wyatt 1989
Aylmer Lake, District of Mackenzie, Northwest Territories	<0.002 mm	193	54.5 ± 43.1	1–224	42	ICP–AES	Dredge et al. 1994
	<0.063 mm	191	11 ± 10.8	0.25–100	8.2	INAA	
Lac de Gras, District of Mackenzie, Northwest Territories	<0.002 mm	183	59.4 ± 34.2	1–154	54	ICP–AES	Ward et al. 1994
	<0.063 mm	178	3.8 ± 0.29	1.4–25	7.7	INAA	
Winter Lake, District of Mackenzie, Northwest Territories	<0.002 mm	120	36.4 ± 31.1	4–200	28	ICP–AES	Kerr et al. 1994
	<0.063 mm	115	4.4 ± 3.7	0.8–29	3.4	INAA	
South Melville Peninsula, Northwest Territories							Dredge 1995
1990	<0.002 mm	280	104 ± 14.7	2.5–207	8	ICP–AES	
1991	<0.002 mm	255	15.5±43.8	1–520	2	ICP–AES	

* Analytical methods:
AAS = atomic adsorption spectrometry
AES = atomic emission spectrometry
DCP = directly coupled plasma
ES = emission spectrometry
ICP = inductively coupled plasma
INAA = instrumental neutron activation analysis

Table 3. Background Concentrations of Arsenic in Canadian Surface Soils

Sampling site	Number of samples	Arsenic content ($\mu g \cdot g^{-1}$ dw)		Sample preparation/ analytical method*	Reference
		Mean	Range		
Ontario - uncontaminated agricultural soils	207	6.3	1.1–16.7	Chromogenic complex (arsine generation) spectrophotometry	Frank et al. 1976b
Ontario - uncontaminated orchards	—	—	1.1–8.6	—	NRCC 1978
Ontario - old urban parklands	60	5.9	<0.2–60.7	Hydride generation /flameless AAS	OMEE 1994a
Ontario - rural parklands	101	3.9	<0.2–30	Hydride generation/flameless AAS	OMEE 1994a
Ontario - Halton region agricultural soils (no sludge treatment)	252	—	5.1–6.0	Hydride generation/flameless AAS	Webber and Shamess 1987
Ontario - agricultural soils (no sludge treatment) from 10 locations	—†	4.2	1.1–7.2	Calorimetry	Webber et al. 1981
Ontario - Toronto, urban background	—	9.8	2.7–41	Hydride method/AAS	Temple et al. 1977
Ontario - southern, agricultural soils	26 sites	5.1	1.7–8.5	Arsine generation from HNO_3-H_2SO_4/ flameless AAS	Whitby et al. 1978
Ontario - rural and agricultural soils	—	—	1.3–13.5	—	OMAF 1989 unpub. data
Ontario - Windsor area, 1990 - urban - rural	 12 sites‡ 18 sites‡	 7.2 5.8	 4.1–16.7 4.8–7.2	 — —	Gizyn 1994
Ontario - Sudbury area	—	max: 4.4		—	McGovern and Balsillie 1975
Canada - literature review	—	—	4.8–13.6	—	Kabata-Pendias and Pendias 1992
Canada - near arseniferous mineral deposits	—	—	400–8000	—	NRCC 1978
Québec - Noranda background level in peat	—	1.9	—	—	Azzaria and Frechett 1987
British Columbia, Nelson - reference site	≈100	5	—	HNO_3/HF/$HClO_4$ digestion/ICP–AES	Lynch et al. 1980
British Columbia - Vancouver background level	—	3	—	—	Henning and Konasewich 1984
Manitoba, Saskatchewan, and Alberta surface soils	1273	6.6 (median: 6.6)	0.25–19	Instrumental neutron activity	R.G. Garrett 1995, Natural Resources Canada, pers. com.
Alberta - background concentration	78	—	0.40–7.1§	Graphite furnace AAS	Dudas and Pawluk 1980

Sampling site	Number of samples	Arsenic content ($\mu g \cdot g^{-1}$ dw)		Sample preparation/ analytical method*	Reference
		Mean	Range		
New Brunswick, 1983–84 survey	40 sites	8.9	2.3–34.9	—	MacMillan 1985
New Brunswick, 1992, residential garden soils					Pilgrim 1993
- East St John‖	18	9.0 ± 1.5	—	—	
- West St John‖	4	12.7 ± 2.4	—	—	
- Fredericton	2	10.7 ± 0.4	—	—	
Nova Scotia	—	—	Trace–7.9	—	NRCC 1978
Northwest Territories - Yellowknife area	—	—	10–25	—	Plambeck and Smith 1976, Prokopuk 1976

* Analytical methods:
AAS = atomic adsorption spectrometry
AES = atomic emission spectrometry
ICP = inductively coupled plasma

† Twenty cores per composite sample from four areas per location.

‡ Three replicates per site.

§ Most samples in the 4.0–6.5 $\mu g \cdot g^{-1}$ dw range.

‖ In addition to naturally high background levels of arsenic in soils, several potential point sources, such as the refinery and New Brunswick power generators, may be sources of arsenic in soils in St John. The weighted mean concentration for the two data sets is 9.7 $\mu g \cdot g^{-1}$ dry soil.

Table 4. Arsenic Concentrations in Contaminated Canadian Soils

Sampling site	Number of samples	Arsenic content ($\mu g \cdot g^{-1}$ dw)		Sample preparation/ analytical method*	Reference
		Mean	Range		
Northwest Territories - Yellowknife town site - near mines	—	Max.: 7600	1–524	XFS	CPHA 1977
British Columbia - vicinity of lead/zinc smelter in Trail	≈100	49	—	$HNO_3/HF/HClO_4$ digestion/ICP–AES	Lynch et al. 1980
British Columbia, Trail - vicinity of lead/zinc smelter - 1982 - 1989 (park samples)	 4 sites 41	 20 18	 8–43 <1–50	—	H. Teindl 1992, Cominco Metals, pers. com.
British Columbia, Trail - residential soils in the vicinity of lead/zinc smelter in 1989	122	48	33–352	UN–ICP	Hertzman et al. 1991, Department of Health Care and Epidemiology, University of British Columbia, pers. com.
Manitoba - near Flin Flon smelter	—	—	65–280	Cold-vapor/AES	Zoltai 1988
Ontario, Sudbury - within 26-km radius from a nickel/copper smelter	—	10	3–47	—	McGovern and Balsillie 1975
Québec - peat samples within 15 km of Noranda smelter	—	9	—	—	Azzaria and Frechette 1987
Québec, Duparquet - humus samples near site of old gold roasting plant - peat samples within a few kilomoetres of roasting site	 — —	 110 Max.: 1000	 — —	—	Azzaria and Frechette 1987
New Brunswick - near smelter	—	—	5–66.3	—	M. Murphy n.d., New Brunswick Department of the Environment, pers. com.
New Brunswick - garden soil within 1.9–15.07 kilometres of smelter	—	—	7.8–38.5	—	Brunswick Mining and Smelting Corporation 1992, pers. com.
British Columbia, Burnaby, near municipal incinerator (1987–1990) - residential gardens (2 sites) - dairy pasture - blueberry patch - produce farm using commercial fertilizers - cranberry patch - bog - woodland park	 48 24 24 24 24 18 18	 5.5 8.7 4.1 6.1 2.2 5.4 8.2	 2.8–8.8 6.2–15.2 1.0–14.5 4.3–10.6 0.4–4.4 2.3–3.9 4.5–6.8	Hydride generation/ICP–AAS	Soilcon 1990
British Columbia - site of former chromated copper arsenate wood preservation facility	—	>170	—	—	Henning and Konasewich 1984
Ontario - Toronto - near a secondary smelter - near power station with previous coal-burning activity - near a municipal incinerator - near a major expressway - near a sewage sludge incinerator	 — — — — —	 70, max.: 2000 25 16 — —	 6.8–114 3.0–49 3.0–24 4.2–20	Hydride method/AAS	Temple et al. 1977

Sampling site	Number of samples	Arsenic content ($\mu g \cdot g^{-1}$ dw)		Sample preparation/ analytical method*	Reference
		Mean	Range		
Ontario - Halton region sludge-treated agricultural soils	57	5.7	3.7–8.1	Nitric/sulfuric acid digestion + hydride generation Flame/flameless AAS	Webber and Shamess 1987
Ontario - sludge-treated agricultural soils from 10 locations	—†	4.6	1.5–6.3	Calorimetry	Webber et al. 1981
Ontario - agricultural soils where arsenical pesticides were used	89	26.5	1.7–91.6	Chromogenic complex spectrophotometry	Frank et al. 1976b
Ontario - agricultural soils where lead arsenate pesticide was used (surface soil: 0-15 cm)				—	Frank et al. 1976a
- apple orchards	—	54.2	3–126		
- sour cherry orchards	—	23	6–63		
- sweet cherry orchards	—	30	12–63		
- peach orchards	—	10	4–15		
- vineyards	—	8	4–12		
Southwestern Ontario - old apple orchards sprayed with arsenical pesticides	—	—	45–290	—	Boyle and Jonasson 1973
Nova Scotia - old apple orchards sprayed with arsenical pesticides	—	—	10–124	—	Boyle and Jonasson 1973
New Brunswick - wood preservation facilities	—	Max: 10 860		—	Bamwoya et al. 1991
Northwest Territories				—	Gemmill 1977
- Yellowknife City	—	—	1–600		
- near Yellowknife gold mine and smelters	—	>4000	—		
Northwest Territories - Yellowknife gold mine	—	Max.: 21 213	—	—	Plambeck and Smith 1976, Prokopuk 1976

* Analytical methods:
AAS = atomic adsorption sepctrometry
AES = atomic emission spectometry
ICP = inductively coupled plasma
UN = ultrasonic nebulizer
XFS = X-ray fluorescence spectrography

† Twenty cores per composite sample from four areas per location.

Table 5. Existing Guidelines/Criteria for Arsenic in Soil from Various Jurisdictions

Jurisdiction	Category	Guideline/criterium		Reference
		Soil ($mg \cdot kg^{-1}$)	Groundwater ($\mu g \cdot L^{-1}$)	
Canada (national)	Interim assessment criteria	5	5	CCME 1991
	Interim remediation criteria			
	- agriculture	20	—	
	- residential/parkland	30	—	
	- commercial/industrial	50	—	
	Interim maximum acceptable concentration for drinking water	—	25	
British Columbia	A	5	5	BC MOE 1989
	B	30	50 (DW*)	
	C	50	50 (DS†)	
Alberta	Tier 1	10	—	Alberta Environment 1990
Ontario	Surface soil remediation criteria‡			OMEE 1994b
	Potable groundwater situation:			
	- agricultural land	20	25	
	- residential/parkland	20	25	
	- commercial/industrial	20	25	
	Nonpotable groundwater situation:			
	- agricultural land	20	480	
	- residential/parkland	20	480	
	- commercial/industrial	20	480	
	Subsurface remediation criteria§			
	Potable groundwater situation:			
	- residential/parkland	40	—	
	- commercial/industrial	—	—	
	Nonpotable groundwater situation:			
	- residential/parkland	40	—	
	- commercial/industrial	—	—	
Quebec	A	10	5	MENVIQ 1988
	B	20	50	
	C	30	100	
Australia	Interim investigation threshold level	20	—	ANZEC 1992
Germany	Trigger level:			Schuldt 1990
	- agriculture	50	—	
	- residential	100	—	
The Netherlands	Target value	29	10	RIVM 1994
	Intervention value	55	600	
New Jersey	Interim soil action level	20	—	NJDEP 1990
United Kingdom	Threshold trigger values:			UK DOE 1990
	- domestic gardens	10	—	
	- parklands	40	—	
World Health Organization	Provisional drinking water guideline	—	10	WHO 1993

*DW: remediation criterion for groundwater intended for human consumption.
†DS: remediation criterion for water-based discharges.
‡Surface soil criteria apply only where soil pH is 5.0–9.0.
§Subsurface soil criteria apply only where pH is 5.0–11.0.

Table 6. Toxicological Studies on the Effects of Arsenic on Soil Microorganisms and Microbial Processes

Organism	Effect	Endpoint*	Effective concentration ($mg \cdot kg^{-1}$)	Arsenic compound	Exposure period	Soil pH	Test substrate	Extraction method	Reference
Selected studies									
Native soil microflora	Inhibition of N mineralization 40% 51% Inhibition of nitrification	 EC EC NOEC	 50 300 300	Na_2HAsO_4	8–9 years	6	A horizon of sandy cambisol; 9% clay, 12% silt, 79% sand, 1.2% organic content; CEC 10.3 meq/100 g	Nominal	Wilke 1989
Consulted studies									
Native soil microflora/N mineralization	2% inhibition 3% inhibition 5% inhibition 7% inhibition 4% inhibition 4% inhibition 1% inhibition 7% inhibition	EC EC EC EC EC EC EC EC	375 375 375 375 375 375 375 375	$NaAsO_2$ Na_2HAsO_4 $NaAsO_2$ Na_2HAsO_4 $NaAsO_2$ Na_2HAsO_4 $NaAsO_2$ Na_2HAsO_4	20 d 20 d 20 d 20 d	5.8 6.6 7.8 7.4	4.4% organic content, 23% clay, 39% silt 5.0% organic content, 45% clay, 54% silt 3.7% organic content, 30% clay, 44% silt 9.3% organic content, 34% clay, 50% silt	Nominal	Liang and Tabatabai 1977
Native soil microflora/ nitrification	98% inhibition 71% inhibition 88% inhibition 27% inhibition 64% inhibition 14% inhibition	EC EC EC EC EC EC	375 375 375 375 375 375	$NaAsO_2$ Na_2HAsO_4 $NaAsO_2$ Na_2HAsO_4 $NaAsO_2$ Na_2HAsO_4	10 d 10 d 10 d	5.8 7.8 7.4	4.4% organic content, 23% clay, 39% silt 3.7% organic content, 30% clay, 44% silt 9.3% organic content, 34% clay, 50% silt	Nominal	Liang and Tabatabai 1978

Organism	Effect	Endpoint*	Effective concentration ($mg \cdot kg^{-1}$)	Arsenic compound	Exposure period	Soil pH	Test substrate	Extraction method	Reference
Urease activity	98% inhibition	EC No effect	375 375	$NaAsO_2$ Na_2HAsO_4	30 min	5.1	1.51% organic content, 17% clay, 1% sand	Nominal	Tabatabai 1977
	9% inhibition	EC No effect	375 375	$NaAsO_2$ Na_2HAsO_4	30 min	6.1	3.32% organic content, 30% clay. 25% sand		
	27% inhibition	EC No effect	375 375	$NaAsO_2$ Na_2HAsO_4	30 min	5.8	2.6% organic content, 23% clay, 38% sand		
	24% inhibition 7% inhibition	EC EC No effect	375 37.5 375	$NaAsO_2$ $NaAsO_2$ Na_2HAsO_4	30 min	7.8	3.7% organic content, 30% clay, 26% sand		
	44% inhibition	EC No effect	375 375	$NaAsO_2$ Na_2HAsO_4	30 min	6.8	4.4% organic content, 42% clay, 3% sand		
	18% inhibition 14% inhibition	EC EC No effect	375 37.5 375	$NaAsO_2$ $NaAsO_2$ Na_2HAsO_4	30 min	7.4	5.45% organic content, 34% clay, 16% sand		
Phosphatase enzyme activity	20% inhibition	EC	749	$NaAsO_2$	3 h	6.3 4.4	13% organic matter, 29% clay 93% organic matter, <1% clay	Nominal	Tyler 1981
Acid phosphatase activity	1% reduction 39% reduction	EC EC	1873 1873	$NaAsO_2$ Na_2HAsO_4	30 min	7.8	3.7% organic content, 30% clay	Nominal	Juma and Tabatabai 1977
	16% reduction 62% reduction	EC EC	1873 1873	$NaAsO_2$ Na_2HAsO_4	30 min	7.4	5.5% organic content, 34% clay		
	9% reduction 4% reduction 80% reduction	EC EC EC	1873 187.3 1873	$NaAsO_2$ Na_2HAsO_4	30 min	5.8	2.5% organic content, 23% clay		
Alkaline phosphatase activity	35% reduction 75% reduction	EC EC	1873 1873	$NaAsO_2$ Na_2HAsO_4	30 min	7.8	3.7% organic content, 30% clay	Nominal	Juma and Tabatabai 1977
	19% reduction 57% reduction	EC EC	1873 1873	$NaAsO_2$ Na_2HAsO_4	30 min	7.4	5.5% organic content, 34% clay		
Native soil microflora/ population	None	No effect	439 $kg \cdot ha^{-1}$ per year for 4 years	$Ca(AsO_4)_2$	Applied annually for 4 years	6.6	Flanagan silt loam; 6% organic matter	Nominal	Cole and Turgeon 1978

Organism	Effect	Endpoint*	Effective concentration ($mg \cdot kg^{-1}$)	Arsenic compound	Exposure period	Soil pH	Test substrate	Extraction method	Reference
Amidase activity	99% inhibition 2% inhibition	EC EC	375 375	$NaAsO_2$ Na_2HAsO_4	30 min	5.6	28% clay, 3% sand, 2.63% organic content	Nominal	Frankenberger and Tabatabai 1981
	98% inhibition 1% inhibition	EC EC	375 375	$NaAsO_2$ Na_2HAsO_4	30 min	7.6	30% clay, 31% sand, 3.24% organic content		
	97% inhibition 3% inhibition	EC EC	375 375	$NaAsO_2$ Na_2HAsO_4	30 min	7	34% clay, 21% sand, 4.7 % organic content		
Dehydrogenase activity	35% inhibition	EC	150	Na_2HAsO_4	24 h	NR†	1.3% organic content	Nominal	Rogers and Li 1985
	63% inhibition	EC	150	Na_2HAsO_4	24 h	NR	1.3% organic content; soil enriched with 1% alfalfa		
Citrobacter frundii, *Aeromonas* sp., *Klebsiella* sp. isolated from soil	Population reduction	LC_{50} LC_{50} LC_{50}	27 000 $g \cdot mL^{-1}$ 1 300 $g \cdot mL^{-1}$ 270 $g \cdot mL^{-1}$	MSMA‡ Na_2HAsO_4 $NaAsO_2$	24 h	NR	Nutrient broth	Nominal	Anderson and Abdelghani 1980
		LC_{50}	60 000 220 000	MSMA MSMA	48 h	NR			

Note: All studies, except Anderson and Abdelghani (1980) are single-dose studies.
*The EC endpoint represents the effects concentration as calculated by the CCME from the data presented by the authors.
†Not reported.
‡Monosodium methyl arsonate.

Table 7. Toxicological Studies on the Effects of Arsenic on Terrestrial Plants

Organism	Effect	Endpoint*	Effective concentration ($mg \cdot kg^{-1}$)	Arsenic compound	Exposure period	Soil pH	Test substrate	Extraction method	Reference
Selected studies									
Radish *(Raphanus sativa)*	Reduced seedling emergence	NOEC EC_{25} EC_{44} EC_{50}	11 (std. dev. 2) 12 (std. dev. 3) 13 (std. dev. 4) 14 (std. dev. 3)	KH_2AsO_4	72 h	4–4.2	Artificial soil; 69–75% sand, 8–9% silt, 16–22% clay, 2.4–3.7% organic content, 2.8–3.3% moisture	HNO_3, HCl, and H_2O_2 digestion analyzed with ICP (#6010A)	Environment Canada 1995b
Lettuce *(Lactuca sativa)*	Reduced seedling emergence	NOEC EC_{25} EC_{30} EC_{50}	20 (std. dev. 3) 32 (std. dev. 4) 36 (std. dev. 8) 46 (std. dev. 6)	KH_2AsO_4	120 h	4–4.3	Artificial soil; 67–75% sand, 8–10% silt, 16–23% clay, 2.7–3.7% organic content, 2.5–3.3% moisture	HNO_3, HCl, and H_2O_2 digestion analyzed with ICP (#6010A)	Environment Canada 1995b
Green beans *(Phaseolus vulagris)*	42% total plant yield reduction	EC	10	Na_2HAsO_4	Grown to maturity	6.2	Loamy sand; 10.5% clay, 0.90% organic matter	Nominal	Woolson 1973
Lima beans *(Phaseolus linensis)*	99% total plant yield reduction	NOEC EC	10 50	Na_2HAsO_4	Grown to maturity	6.2	Loamy sand; 10.5% clay, 0.90% organic matter	Nominal	Woolson 1973
Spinach *(Spinacia oleracea)*	41% total plant yield reduction	EC	10	Na_2HAsO_4	Grown to maturity	6.2	Loamy sand; 10.5% clay, 0.90% organic matter	Nominal	Woolson 1973
Radish *(Raphanus sativis)*	23% total plant yield reduction	EC	10	Na_2HAsO_4	Grown to maturity	6.2	Loamy sand; 10.5% clay, 0.90% organic matter	Nominal	Woolson 1973
Tomato *(Lycopersicon esculentum)*	42% total plant yield reduction	NOEC EC	10 50	Na_2HAsO_4	Grown to maturity	6.2	Loamy sand; 10.5% clay, 0.90% organic matter	Nominal	Woolson 1973
Cabbage *(Brassica oleracea)*	68% total plant yield reduction	NOEC EC	50 100	Na_2HAsO_4	Grown to maturity	6.2	Loamy sand; 10.5% clay, 0.90% organic matter	Nominal	Woolson 1973
Green beans *(Phaseolus vulagris)*	29% total plant yield reduction	NOEC EC	10 50	Na_2HAsO_4	Grown to maturity	5.5	Silty clay; 30% clay, 2.5% organic matter	Nominal	Woolson 1973
Lima beans *(Phaseolus linensis)*	16% total plant yield reduction	NOEC EC	50 100	Na_2HAsO_4	Grown to maturity	5.5	Silty clay; 30% clay, 2.5% organic matter	Nominal	Woolson 1973
Spinach *(Spinacia oleracea)*	22% total plant yield reduction	NOEC EC	10 50	Na_2HAsO_4	Grown to maturity	5.5	Silty clay; 30% clay, 2.5% organic matter	Nominal	Woolson 1973
Radish *(Raphanus sativis)*	25% total plant yield reduction	NOEC EC	10 50	Na_2HAsO_4	Grown to maturity	5.5	Silty clay; 30% clay, 2.5% organic matter	Nominal	Woolson 1973

Organism	Effect	Endpoint*	Effective concentration ($mg \cdot kg^{-1}$)	Arsenic compound	Exposure period	Soil pH	Test substrate	Extraction method	Reference
Tomato *(Lycopersicon esculentum)*	77% total plant yield reduction	NOEC EC	100 500	Na_2HAsO_4	Grown to maturity	5.5	Silty clay; 30% clay, 2.5% organic matter	Nominal	Woolson 1973
Cabbage *(Brassica oleracea)*	23% total plant yield reduction	NOEC EC	100 500	Na_2HAsO_4	Grown to maturity	5.5	Silty clay; 30% clay, 2.5% organic matter	Nominal	Woolson 1973
Green beans *(Phaseolus vulagris)*	22% total plant yield reduction	EC	10	Na_2HAsO_4	Grown to maturity	4.4	Clay loam; 24.4% clay, 0.99% organic matter	Nominal	Woolson 1973
Lima beans *(Phaseolus linensis)*	19% total plant yield reduction	EC	10	Na_2HAsO_4	Grown to maturity	4.4	Clay loam; 24.4% clay, 0.99% organic matter	Nominal	Woolson 1973
Spinach *(Spinacia oleracea)*	33% total plant yield reduction	EC	10	Na_2HAsO_4	Grown to maturity	4.4	Clay loam; 24.4% clay, 0.99% organic matter	Nominal	Woolson 1973
Radish *(Raphanus sativis)*	17% total plant yield reduction	EC	10	Na_2HAsO_4	Grown to maturity	4.4	Clay loam; 24.4% clay, 0.99% organic matter	Nominal	Woolson 1973
Tomato *(Lycopersicon esculentum)*	97% total plant yield reduction	NOEC EC	100 500	Na_2HAsO_4	Grown to maturity	4.4	Clay loam; 24.4% clay, 0.99% organic matter	Nominal	Woolson 1973
Cabbage *(Brassica oleracea)*	26% total plant yield reduction	EC	10	Na_2HAsO_4	Grown to maturity	4.4	Clay loam; 24.4% clay, 0.99% organic matter	Nominal	Woolson 1973
Snap beans *(Phaseolus vulgaris)*	54% reduction of fresh weight of marketable portions	NOEC EC	19 26	$NaAsO_2$	1 growing season	5.5	Plainfield sand; 4% silt, 7% clay, 0.7% organic content	$H_2SO_4/HClO_4$ digestion	Jacobs et al. 1970a
Peas *(Pisium sativum)*	54% reduction of fresh weight of marketable portions	NOEC EC	26 63	$NaAsO_2$	1 growing season	5.5	Plainfield sand; 4% silt, 7% clay, 0.7% organic content	$H_2SO_4/HClO_4$ digestion	Jacobs et al. 1970a
Corn *(Zea mays)*	54% reduction of fresh weight of marketable portions	NOEC EC	26 63	$NaAsO_2$	1 growing season	5.5	Plainfield sand; 4% silt, 7% clay, 0.7% organic content	$H_2SO_4/HClO_4$ digestion	Jacobs et al. 1970a
Potato *(Solanum dulce)*	76% reduction of fresh weight of marketable portions	NOEC EC	73 250	$NaAsO_2$	1 growing season	5.5	Plainfield sand; 4% silt, 7% clay, 0.7% organic content	$H_2SO_4/HClO_4$ digestion	Jacobs et al. 1970a
Corn *(Zea mays)*	Growth reduction measured in fresh weight	EC_{50} EC_{50} EC_{50} EC_{50}	325 69 42 77	$Fe(H_2AsO_4)_3$ $Al(H_2AsO_4)_3$ NaH_2AsO_4 $Ca(H_2AsO_4)_2$	4 weeks 4 weeks 4 weeks 4 weeks	6.2 6.2 6.2 6.2	Lakeland loamy sand; 10.5% clay, 0.90% organic matter	$H_2SO_4/HClO_4$ digestion	Woolson et al. 1971

Organism	Effect	Endpoint*	Effective concentration ($mg \cdot kg^{-1}$)	Arsenic compound	Exposure period	Soil pH	Test substrate	Extraction method	Reference
Lowbush blueberry *(Vaccinium angustifolium)*	22% reduction in total linear growth 30% reduction in fresh weight of leaves	NOEC EC NOEC EC	43.8 69.5 43.8 69.5	As_2O_3	17 weeks	4.6	Loamy sand	NR†	Anastasia and Kender 1973
Corn *(Zea mays)*	90% reduction in dry weight yield	NOEC EC	100 1000	Na_2HAsO_4	4 weeks	5.5	Silty clay loam; 30.0% clay, 2.5% organic matter	Nominal	Woolson et al 1973
Oats *(Avena sativa)*	10% reduction in dry weight yield	NOEC EC	10 100	Na_2HAsO_4	4 weeks	5.5	Silty clay loam; 30.0% clay, 2.5% organic matter	Nominal	Woolson et al 1973
Corn *(Zea mays)*	91% reduction in dry weight yield	NOEC EC	10 100	Na_2HAsO_4	4 weeks	6.2	Loamy sand; 10.5% clay, 0.90% organic matter	Nominal	Woolson et al 1973
Oats *(Avena sativa)*	78% reduction in dry weight yield	NOEC EC	10 100	Na_2HAsO_4	4 weeks	6.2	Loamy sand; 10.5% clay, 0.90% organic matter	Nominal	Woolson et al 1973
Oats *(Avena sativa)*	Straw yield 41% reduction	NOEC EC	20 40	Na_2HAsO_4	21 d	6.7	Sandy loam; 1.2% organic matter	Nominal	Kulich 1984
Consulted studies									
Cotton *(Gossypium hirsutum)*	52% growth reduction	NOEC EC	18.8 $mg \cdot kg^{-1}$ 37 $mg \cdot kg^{-1}$	As_2O_3	6 weeks	NR	Sandy loam	Nominal	Deuel and Swoboda 1972
Soybean *(Glycine max)*	45% growth reduction	NOEC EC	<18.8 $mg \cdot kg^{-1}$ 18.8 $mg \cdot kg^{-1}$	As_2O_3	6 weeks	NR	Sandy loam	Nominal	Deuel and Swoboda 1972
Cotton *(Gossypium hirsutum)*	40% growth reduction	NOEC EC	150 $mg \cdot kg^{-1}$ 187 $mg \cdot kg^{-1}$	As_2O_3	6 weeks	NR	Clay	Nominal	Deuel and Swoboda 1972
Soybean *(Glycine max)*	40% growth reduction	NOEC EC	75.5 $mg \cdot kg^{-1}$ 112 $mg \cdot kg^{-1}$	As_2O_3	6 weeks	NR	Clay	Nominal	Deuel and Swoboda 1972
Clover *(Trifolium pratenze)*	Growth reduction	NOEC	5 $mg \cdot kg^{-1}$	Na_2HAsO_4	21 d	6.7	Sandy loam; 12% organic matter	Nominal	Kulich 1987
Barley *(Hordeum vulgare)*	Growth reduction	NOEC	40 $mg \cdot kg^{-1}$	Na_2HAsO_4	21 d	6.7	Sandy loam; 12% organic matter	Nominal	Kulich 1987

Organism	Effect	Endpoint*	Effective concentration ($mg \cdot kg^{-1}$)	Arsenic compound	Exposure period	Soil pH	Test substrate	Extraction method	Reference
Horse bean *(Vicia fabae)*	4% growth reduction	EC	20 $mg \cdot kg^{-1}$	Na_2HAsO_4	21 d	6.7	Sandy loam; 12% organic matter	Nominal	Kulich 1987
Sunflower *(Helianthus annus)*	20% growth reduction	EC	20 $mg \cdot kg^{-1}$	Na_2HAsO_4	21 d	6.7	Sandy loam; 12% organic matter	Nominal	Kulich 1987
Oats *(Aveena sativa)*	Straw yield Grain yield 41% reduction in straw yield 13% reduction in grain yield	NOEC NOEC EC EC	20 $mg \cdot kg^{-1}$ <20 $mg \cdot kg^{-1}$ 40 $mg \cdot kg^{-1}$ 20 $mg \cdot kg^{-1}$	Na_2HAsO_4	21 d	6.7	Sandy loam; 12% organic matter	Nominal	Kulich 1984
Alfalfa *(Medicago sative)*	Growth reduction Growth reduction	NOEC EC	80 $mg \cdot kg^{-1}$ >80 $mg \cdot kg^{-1}$	Na_2HAsO_4	21 d	6.7	Sandy loam; 12% organic matter	Nominal	Kulich 1984
Scots pine seedlings *(Pinus sylvestris)*	Mortality	LC_{90}	>200 $mg \cdot kg^{-1}$	Na_2HAsO_4	50 d	NR	Clay loam	Nominal	Sheppard et al. 1985
Urtica dioica	Growth reduction	LOEC	1 $mg \cdot kg^{-1}$	Cacodylate	60 d	NR	80% garden soil, 20% sand	Nominal	Otte et al. 1990
Phragmites australis	Growth reduction	NOEC	30 $mg \cdot kg^{-1}$	$PbHAsO_4$	60 d	NR	80% garden soil, 20% sand	Nominal	Otte et al. 1990
Creeping bentgrass *(Agrostis palustris)*	Growth	NOEC	272 $kg \cdot ha^{-1}$ applied over 4 years 184 $mg \cdot kg^{-1}$ applied over 4 years‡	$Ca_3(AsO_4)_2$	4 years	6.4	"Purr-wick" green; 94.5% sand, 1.0% clay, 2.4% organic matter	Nominal	Callahan and Shepard 1991
Silene vulgaris	Reduction in seed germination	NOEC	20 $mg \cdot L^{-1}$ (highest application)	Na_2HAsO_4	10 d	NR	Filter paper	Nominal	Paliouris and Hutchinson 1991
Onion bulbs *(Allium cepa)*	Nonreversible root growth inhibition	LOEC	2 $mg \cdot L^{-1}$	Arsenate	4 d	NR	Solution	Nominal	Pepper et al. 1988
Barley *(Hordeum vulgare)*	Reduced yield at five-leaf stage	LOEC	4 $mg \cdot L^{-1}$	Na_2HAsO_4	14 d	NR	Solution	Nominal	Davis et al. 1978
Lettuce *(Lactuca sativa)*	Reduction in root length	EC_{50} EC_{50}	65 $mg \cdot L^{-1}$ 54 $mg \cdot L^{-1}$	MSMA§ MSMA	115 h 5 d	NR NR	Filter paper Solution	Nominal	Ratsch and Johndro 1986

Organism	Effect	Endpoint*	Effective concentration ($mg \cdot kg^{-1}$)	Arsenic compound	Exposure period	Soil pH	Test substrate	Extraction method	Reference
Radish *(Raphanus sativa)*	Reduction in root elongation	LOEC 35% EC_{25} EC_{50}	6 $mg \cdot L^{-1}$ 4 $mg \cdot L^{-1}$ 12 $mg \cdot L^{-1}$	KH_2AsO_4	72 h	NR	Filter paper	Nominal	Environment Canada 1995b
Lettuce *(Lactuca sativa)*	Reduction in root elongation	LOEC 52% EC_{25} EC_{50}	1.3 $mg \cdot L^{-1}$ 0.6 $mg \cdot L^{-1}$ 1.3 $mg \cdot L^{-1}$	KH_2AsO_4	120 h	NR	Filter paper	Nominal	Environment Canada 1995b

*The EC endpoint represents the effects concentration as calculated by the CCME from the data presented by the authors.
†Not reported.
‡Conversion factor used: 1 $kg \cdot ha^{-1}$ = 0.677 $mg \cdot kg^{-1}$ = 0.67 ppm (Denneman and van Gestel 1990).
§Monosodium methyl arsonate.

Table 8. Toxicological Studies on the Effects of Arsenic on Terrestrial Invertebrates

Organism	Effect	Endpoint*	Effective concentration	Arsenic compound	Exposure period	pH	Test substrate	Extraction method	Reference
Selected studies									
Earthworm *(Eisenia foetida)*	Mortality	NOEC LC_{50}	50 mg·kg^{-1} 100 mg·kg^{-1}	KH_2AsO_4	8 weeks	7.6	1:1 mixture of peaty marshland soil and horse manure	Nominal	Fisher and Koszorus 1992
Earthworm *(Eisenia foetida)*	Mortality	NOEC LC_{25} LC_{42} LC_{50}	83 mg·kg^{-1} (std .dev. 36) 142 mg·kg^{-1} (std. dev. 11) 163 mg·kg^{-1} (std. dev. 70) 213 mg·kg^{-1} (std. dev. 27)	KH_2AsO_4	14 d	4–4.3	Artificial soil; 67–75% sand, 8–12% silt, 16–21% clay, 2.5–3.3% moisture, 2.7–4.1% organic content	HNO_3, HCl and H_2O_2 digestion analyzed with ICP (#6010A)	Environment Canada 1995b
Consulted studies									
Grasshopper *(Conocephalus fasciatus)*	50% mortality	LD_{50}	3.1 mg·L^{-1}	As_2O_3	—	NR†	Raised in cages	Nominal	Watson et al. 1976
	50% mortality	LD_{50}	4.5 mg·L^{-1}	As_2O_3	—				
	50% mortality	LD_{50}	12.1 mg·L^{-1}	Cacodylic acid	—				
	50% mortality	LD_{50}	1.3 mg·L^{-1}	Phytar 569^R (12.7% elemental arsenic)	7 d (oral in solution)				
	50% mortality	LD_{50}	1.2 mg·L^{-1}	As_2O_3	—				
	50% mortality	LD_{50}	1.5 mg·L^{-1}	As_2O_3	—				
	50% mortality	LD_{50}	2.6 mg·L^{-1}	Dimethylarsinic acid	—				
	50% mortality	LD_{50}	0.4 mg·L^{-1}	Phytar 569^R (12.7% elemental arsenic)	14 d (oral in solution)				
Spruce budworm (Western) *(Choristonereura occidentalis)*	10% mortality	LC_{10}	2550 mg·L^{-1}	As_2O_3	3 d (oral in liquefied diet)	NR	Sterile petri dish	Nominal	Robertson and McLean 1985

Organism	Effect	Endpoint*	Effective concentration	Arsenic compound	Exposure period	pH	Test substrate	Extraction method	Reference
Earthworms *(Eisenia foetida andrei)*	5 % mortality 50% mortality	LC_5 LC_{50}	92 $mg \cdot kg^{-1}$ 107 $mg \cdot kg^{-1}$	$NaAsO_2$	1 week	NR	Artisol (a mixture of glass balls and silica)	Nominal	Gal et al. 1988
	5% mortality 50% mortality	LC_5 LC_{50}	98 $mg \cdot kg^{-1}$ 102 $mg \cdot kg^{-1}$	$NaAsO_2$	2 weeks				
	5% mortality 50% mortality	LC_5 LC_{50}	94 $mg \cdot kg^{-1}$ 110 $mg \cdot kg^{-1}$	$NaAsO_2$	3 weeks				
	5% mortality 50% mortality	LC_5 LC_{50}	87 $mg \cdot kg^{-1}$ 103 $mg \cdot kg^{-1}$	$NaAsO_2$	4 weeks				
Earthworm *(Eisenia foetida)*	~1/3 reduction in worm mass	EC	50 $mg \cdot kg^{-1}$	KH_2AsO_4	8 weeks	7.6	1:1 mixture of peaty marshland soil and horse manure	Nominal	Fisher and Koszorus 1992
	~2/3 reduction in cocoon production	EC	50 $mg \cdot kg^{-1}$						
Honey bees *(Apis mellifera)*	100% mortality (accidental)	LD	5–13 $mg \cdot kg^{-1}$ fw larvae 21–31 $mg \cdot kg^{-1}$ fw adults	Arsenite	NR (spray dusting)	NR	NR	NR	Jenkins 1980
Beetles	100% mortality	LD	100–1000 $mg \cdot kg^{-1}$	Cacodylic acid	NR (dietary)	NR	NR	NR	Hood 1985

*The EC endpoint represents the effects concentration as calculated by the CCME from the data presented by the authors.
†Not reported.

Table 9. Toxicological Studies on the Effects of Arsenic on Mammals and Birds

Species	Effect	Endpoint	Effective concentration	Arsenic compound	Exposure route	Reference
Selected studies						
California quail	Mortality	LD_{50}	47.6 $mg \cdot kg^{-1}$ bw per day	$NaAsO_2$	Oral	Hudson et al. 1984
Mallard duck	Mortality	LD_{50}	323 $mg \cdot kg^{-1}$ bw per day	$NaAsO_2$	Oral	Hudson et al. 1984
Ring-necked pheasant	Mortality	LD_{50}	386 $mg \cdot kg^{-1}$ bw per day	$NaAsO_2$	Oral	Hudson et al. 1984
Rabbit	Mortality	Death	23 $mg \cdot kg^{-1}$ bw per day	$Ca_3(AsO_4)_2$	Oral	Chappellier and Raucourt 1936
White-tailed deer	Minimal lethal dose	LOEC	34 $mg \cdot kg^{-1}$ bw per day	$NaAsO_2$	Oral	Boyce and Verme 1954
Rabbit	Mortality	LD_{50}	8 $mg \cdot kg^{-1}$ bw per day	As_2O_5/H_3AsO_4	Oral	Sullivan 1969
Consulted studies						
Horse *(Equus caballus)*	Mortality	Death	Daily doses of 2–6 $mg \cdot kg^{-1}$ bw for 14 weeks	$NaAsO_2$	Oral	NRCC 1978
Cat *(Felis domesticus)*	NR*	NR	Chronic toxicity at 1.5 $mg \cdot kg^{-1}$ bw	Inorganic arsenate or arsenite	Oral	Pershagen and Vahter 1979
Cattle	Mortality	Death	Single oral dose of 15–45 $mg \cdot kg^{-1}$ bw	$As_2O_3(As_4O_6)$	Oral	NRCC 1978
Horse	NR	Toxic dose No-effect dose Toxic dose	50 $mg \cdot kg^{-1}$ bw single oral dose 0.24–0.72 $g \cdot d^{-1}$ for 2 years 1.0–3.0 $g \cdot d^{-1}$ for 14 weeks	$As_2O_3(As_4O_6)$ $As_2O_3(As_4O_6)$ Na_2HAsO_4	Oral	Puls 1988
Pigs	NR	Toxic dose Toxic dose	50 $mg \cdot kg^{-1}$ bw single oral dose 0.05–0.15 g single dose	$As_2O_3(As_4O_6)$ $NaAsO_2$	Oral	Puls 1988
Sheep	NR	Toxic dose Toxic dose	11 $mg \cdot kg^{-1}$ bw single oral dose 100 $mg \cdot kg^{-1}$ bw single oral dose	$NaAsO_2$ $As_2O_3(As_4O_6)$	Oral	Puls 1988

*Not reported.

Table 10. Estimated Average Daily Intake of Arsenic by the Canadian General Population

Medium	Estimated daily intake ($\mu g \cdot kg^{-1}$ bw per day)				
	Infants (0–6 months)*	Preschoolers (7 months–4 years)†	School-age children (5–11 years)‡	Teenagers (12–19 years)§	Adults (20–70 years)‖
Water#	0.5	0.3	0.2	0.1	0.1
Food**	0.2	0.3	0.2	0.1	0.08
Air††	0.0003	0.0004	0.0004	0.0004	0.0003
Soil/Dirt‡‡	0.029	0.062	0.007	0.004	0.003
Tobacco smoking§§	[—]	[—]	[—]	[0.01–0.04]	[0.01–0.03]
Total	0.729	0.662	0.407	0.204	0.183

Source: Adapted from Hughes et al. 1994.

* Assumed to weigh 7 kg, breathe 2 m^3 of air per day, drink 0.75 L of water per day (Health Canada 1994), and ingest 20 mg of soil per day (Angus Environmental Limited 1991).

† Assumed to weigh 13 kg, breathe 5 m^3 of air per day, drink 0.8 L of water per day (Health Canada 1994), and ingest 80 mg of soil per day (Angus Environmental Limited 1991).

‡ Assumed to weigh 27 kg, breathe 12 m^3 of air per day, drink 0.9 L of water per day (Health Canada 1994), and ingest 20 mg of soil per day (Angus Environmental Limited 1991).

§ Assumed to weigh 57 kg, breathe 21 m^3 of air per day, drink 1.1 L of water per day, and ingest 20 mg of soil per day (Health Canada 1994).

‖ Assumed to weigh 70 kg, breathe 23 m^3 of air per day, drink 1.5 L of water per day, and ingest 20 mg of soil per day (Health Canada 1994).

\# Based on a mean concentration of 5 $\mu g \cdot L^{-1}$ measured as total arsenic; levels in most Canadian surface drinking-water supplies are considerably less than this value, although concentrations in groundwater often exceed 5 $\mu g \cdot L^{-1}$ (Environment Canada 1989a, 1989b, 1989c, 1989d; OMOE 1989; Manitoba Environment 1989).

** Based on mean concentrations of total arsenic detected in various food composites in a Total Diet Study conducted in six Canadian cities (mean concentrations ranged from 0.4 $ng \cdot g^{-1}$ in applesauce to 3048 $ng \cdot g^{-1}$ in marine fish) (Dabeka et al. 1993), food consumption patterns data for various age groups in Canada (Health Canada 1994), and limited information on the percentage of the total arsenic in various food groups which is inorganic (37%) (EC/HWC 1993). Insufficient data were identified to estimate intake of arsenic by infants in breast milk.

†† Based on the mean airborne arsenic concentration of 0.001 $\mu g \cdot m^{-3}$ associated with PM_{10} in most Canadian cities surveyed by Environment Canada (Dann 1994). Since data on levels of arsenic in indoor air are limited, it is assumed that levels in indoor air are comparable to those measured in ambient air.

‡‡ Based on the upper value of mean arsenic levels, 10.0 $\mu g \cdot g^{-1}$, determined at various uncontaminated urban sites (see Table 3). It is assumed that all of the arsenic present in soils is inorganic. Since data on levels of arsenic in housedust are limited, it is assumed that levels of arsenic in dust are comparable to those measured in soils.

§§ Based on estimated arsenic content of mainstream cigarette smoke ranging from 40 to 120 ng per cigarette (U.S. DHHS 1989) and 20 cigarettes smoked per day.

Table 11. Summary of Environmental and Human Health Soil Quality Guidelines for Arsenic

Guideline	Land use		
	Agriculture ($mg \cdot kg^{-1}$)	Residential/ parkland ($mg \cdot kg^{-1}$)	Commercial and industrial ($mg \cdot kg^{-1}$)
TEC or ECL			
20th percentile	17	17	17
25th percentile	19	19	26
30th percentile	20	20	36
35th percentile	26	26	37
NECC	Insufficient data	Insufficient data	Insufficient data
SQG_{SC}*	19	19	26
SQG_{I}	380	Not applicable	Not applicable
SQG_{E}	19	19	26
SQG_{HH}	12	12	12
Interim remediation criteria†	20	30	50

* As per the CCME (1996) protocol, the SQG_{SC} for agricultural and residential/parkland land uses corresponds to the 25th percentile of the effects and no effects data distribution when using the weight of evidence method. The SQG_{SC} for commercial and industrial land uses corresponds to the 25th percentile of the effects data distribution. The other percentiles are presented for comparison purposes only.

†CCME 1991.

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CANADIAN SOIL QUALITY GUIDELINES FOR ARSENIC: ENVIRONMENTAL