Contaminant Profiles

Introduction

Contaminant Profiles describes in greater detail many of the contaminants mentioned in chapters 1–10. While by no means exhaustive, it includes profiles of a wide variety of contaminants identified by scientists, health professionals and members of the general public as being "of concern." These contaminant profiles are arranged in alphabetical order.

For the purpose of the profiles, and for the document as a whole, the term "contaminants" refers to a substance that is either foreign to a natural system, or is present at unnatural concentrations. In the environment, contaminants are unwanted substances that have entered the air, food, water, or soil. These include chemicals, living organisms (e.g., bacteria, viruses, or protozoa), and radionuclides.

For each contaminant the following information is discussed:

- origins and uses;
- persistence and movement in the environment;
- how humans are exposed;
- how to reduce exposure;
- human health considerations; and
- standards and guidelines.

At the end of each contaminant description, references are listed for further reading. Some profiles also contain a "thermometer" that summarizes the health effects observed in the scientific literature at various levels of exposure to the contaminant.

Contaminants

Acetone
Aldrin and Dieldrin
Aluminum
Ammonium Phosphates
Arsenic
Asbestos and Fibrous Materials
BTEX: Benzene, Toluene, Ethyl benzene and Xylene/Gasoline
Cadmium
Campylobacter spp.
Carbon Monoxide (CO)
Chlorofluorocarbons (CFCs)
Chlorophenols/PCP
Clostridium botulinum
Clostridium perfringens
Cryptosporidium parvum and *Giardia lamblia*
DDT and related compounds: DDE, TDE, DDA, methoxychlor
Dieldrin: see Aldrin and Dieldrin
Dioxins (polychlorinated dibenzodioxins) and Furans (polychlorinated dibenzofurans)
Escherichia coli (E. coli)
Fluoride
Furans: see Dioxins
Hexachlorobenzene (HCB)
Lead
Mercury
Mirex
Moulds
Nitrites, Nitrates and Nitrosamines
Nitrogen Oxides (NO_x)
Ozone: see Smog and Ground-Level Ozone
Particulate Matter
Perchloroethylene (Perc)/PCE/Tetrachloroethylene
Phthalates
Polychlorinated Biphenyls (PCBs)
Polycyclic Aromatic Hydrocarbons (PAHs): Benzo[a]pyrene and related compounds
Radon
Salmonella spp.
Smog and Ground-Level Ozone
Sodium Chloride—Road Salt
Staphylococcus aureus

Sulphur Dioxide (SO_2)
Toxaphene
Trihalomethanes (THMs)
Tritium
Vibrio vulnificus
Vinyl Chloride and Polyvinyl Chloride
2,4-Dichlorophenoxyacetic acid (2,4-D)

Tables and Figures

DDT and related compounds: DDE, TDE, DDA, methoxychlor

Dioxins (polychlorinated dibenzodioxins) and Furans (polychlorinated dibenzofurans)

Fluoride

Hexachlorobenzene (HCB)

Lead

Mercury

Mirex

Nitrites, Nitrates and Nitrosamines

Nitrogen Oxides (NO_X)

Particulate Matter

Perchloroethylene (Perc)/PCE/Tetrachloroethylene

Phthalates

Table 1: TDIs for phthalates

Figure 1: Human health effects from breathing di(2-ethylhexyl)phthalate
Figure 2: Human health effects from ingesting di(2-ethylhexyl)phthalate

Polychlorinated Biphenyls (PCBs)

Table 1: Guideline, PTDI and IMAC values for PCBs
Table 2: Health Protection Branch "guideline values" for blood, 1986

Figure 1: Human health effects from breathing PCBs
Figure 2: Human health effects from ingesting PCBs
Figure 3: Human health effects from skin contact with PCBs

Polycyclic Aromatic Hydrocarbons (PAHs): Benzo[a]pyrene and related compounds

Table 1: MAC and guideline values for B[a]P

Figure 1: Human health effects from ingesting benzo[a]pyrene

Radon

Table 1: Human health effects from breathing radon
Table 2: MAC and other values for uranium, radium, cesium, iodine and strontium

Smog and Ground-Level Ozone

Table 1: National Ambient Air Quality Objectives for ground-level ozone

Sodium Chloride — Road Salt

Table 1: Guideline values for sodium and chloride

Sulphur Dioxide (SO_2)

Table 1: National Ambient Air Quality Objectives for sulphur dioxide

Toxaphene

Table 1: Human health effects from eating or drinking toxaphene
Table 2: PTDI value for toxaphene

Trihalomethanes (THMs)

Table 1: IMAC, MAC and other values for trihalomethanes in drinking water

Figure 1: Human health effects from breathing chloroform
Figure 2: Human health effects from ingesting chloroform
Figure 3: Human health effects from skin contact with chloroform

Tritium

Table 1: MAC and guideline values for tritium in drinking water

Vinyl Chloride and Polyvinyl Chloride

2,4-Dichlorophenoxyacetic acid (2,4-D)

Acetone

Origins and Uses

Acetone, also referred to as dimethyl ketone, 2-propanone, and beta-ketopropane, is a colourless liquid with a distinct smell and taste. While it occurs naturally in the environment in plants, trees, volcanic gases, forest fires, and as a product of the breakdown of body fat, industrial processes contribute a larger proportion to the environment. Acetone is used commercially to make plastic, fibres, drugs and other chemicals. It is also present in nail polish removers, synthetic glues, tobacco smoke, vehicle exhaust and landfill sites.

Persistence and Movement in the Environment

About 97 percent of the acetone released during manufacture or use goes into the air where roughly half breaks down due to sunlight or other chemicals within about three weeks. Acetone can move back and forth between air, water, and soil through rain, snow and evaporation. Acetone is broken down by micro-organisms in water and soil, although the time needed for this breakdown varies. Acetone does not bind to soil nor does it build up in animals. It can move into the groundwater from spills and landfill sites.

Exposure

The pathways of exposure to acetone include inhalation, ingestion, and dermal exposure. Most of the background exposure is due to breathing in small amounts in the air. However, higher levels can be found in contaminated air in certain occupational settings or during the use of products containing acetone such as household chemicals, nail polish, and some paints. Higher levels and higher exposure can also be incurred by breathing in second-hand tobacco smoke.

Reducing Exposure

Warning signs of exposure to moderate levels of acetone are the smell, respiratory irritation and burning eyes. If these symptoms occur, the individual should remove her/himself from the source of exposure. Consumer products containing acetone should only be used in a well ventilated area and care should be taken in handling the products; follow all package directions. Acetone is highly flammable. Some acetone-containing products can be abused, such as glue sniffing, which can be harmful to health.

Human Health Considerations

Once exposure has occurred, acetone gets into the blood and is carried to all body organs. Its effects depend on the amount, the pathway, and the duration of exposure. The liver can break acetone down into harmless chemicals when it is present in small amounts. Breathing moderate to high levels of acetone for short periods of time can cause nose, throat, lung, and eye irritation; headaches; light-headedness, confusion; increased pulse rate; effects on blood; nausea; vomiting; unconsciousness and possibly coma; and shortening of the menstrual cycle in women. Ingesting very high levels of acetone can result in unconsciousness and damage to the skin in the mouth. Dermal contact can result in irritation and damage to the skin.

The effects of long-term exposure are known mostly from animal studies, which have found kidney, liver, and nerve damage, increased birth defects, and lowered ability to reproduce in males. It is not known whether the same effects occur in people.

References

United States Department of Health and Human Services, Agency for Toxic Substances and Disease Registry. *Acetone*. Atlanta, Georgia, September 1995.

Aldrin and Dieldrin

Origin and Uses

Aldrin and dieldrin are chemical pesticides first produced in 1948. The manufacture of dieldrin continued until 1987 and aldrin until 1990. In addition to being manufactured, dieldrin is also a by-product of the metabolic conversion of aldrin. Both chemicals were used worldwide as broad-spectrum insecticides to protect crops such as cotton, corn, and citrus products, and for the extermination of termites. In Canada, aldrin and dieldrin were used as pesticides. Dieldrin was also used in tropical areas for control of insects capable of transmitting diseases such as malaria. In the tropics it was sprayed on the inside walls and ceilings of homes. In temperate areas, it was generally applied as a spot treatment on cracks and crevices in homes. In Canada, the permitted uses of aldrin and dieldrin were cancelled in 1990.

Persistence and Movement in the Environment

Aldrin is converted to dieldrin in the environment and in biological systems. Aldrin is not environmentally persistent, and is rarely present in aquatic or terrestrial organisms. Following application of aldrin there is a short period of volatilization, followed by a longer period of degradation to dieldrin. Aldrin adheres so strongly to soil particles that little leaching or contamination of groundwater occurs, except in sandy soils. Loss of aldrin from the soil appears to be mainly by volatilization, though atmospheric levels are generally low.

Dieldrin is a very stable compound. Most dieldrin in the environment is found in soil, plants or animal fat. Dieldrin bioaccumulates in organisms and is biomagnified through the terrestrial and aquatic food chains. It is available in soil for uptake by worms and also by plants (where it is stored mostly in the roots) that are then consumed by fish, animals and people. Dieldrin can travel large distances on dust particles. In air, dieldrin may be changed to a more persistent form, photodieldrin, through a chemical reaction promoted by sunlight.

Exposure

Aldrin and dieldrin can be absorbed via the oral, dermal and inhalation routes. Exposure to aldrin is limited due to its rapid conversion to dieldrin. Exposure to dieldrin is more common and occurs primarily through the ingestion of food. Water and air are not considered to be significant routes of exposure for the general population. Dieldrin is primarily stored in fat and is excreted in feces (via the bile), urine and breast milk.

The average dietary intake of aldrin and dieldrin in Canadian adults is estimated to be 0.02 µg/kg•bw/day. This is well below the World Health Organization (WHO) tolerable daily intake of 0.1 µg/kg•bw/day. Dieldrin is found mainly in dairy and meat products, fish, oils and fats, potatoes, and other root vegetables. Tissues and eggs of birds and predatory species at the top of the food chain have the highest levels. Fish consumption may contribute to exposure, although residues in most fish are less than 200 µg/kg (ppb), which is well below the WHO guideline. Providing that fish consumption advisories are followed, dieldrin levels in the Great Lakes basin are unlikely to be a risk to human health.

Dieldrin concentrations in human adipose tissue in Ontario residents appear to be similar to those in residents from other parts of Canada. Canadian studies indicate mean adipose tissue levels of 0.04-0.05 mg/kg fat (maximum 0.21) from 1976 to 1980. Values for Ontario in 1992 indicate that dieldrin is excreted in milk (0.37 µg/kg whole milk) (Newsome 1995). Dieldrin is present in the fetus and newborn infants due to transplacental exposure. Levels can be higher in fetal blood than in the mother's blood and higher in the placenta than in the uterus (Polishuk et al. 1977).

Aldrin and dieldrin levels in the environment decreased in the 1970s, but have since levelled off. Dieldrin was not detected in any drinking water samples analysed by the Ministry of the Environment and Energy as part of the 1987 Ontario Drinking Water Surveillance Program. Dieldrin was found in rainfall at three points in Canada in 1984 (mean concentrations): 0.78 ng/L (over Lake Superior), 0.27 ng/L (New Brunswick), 0.38 ng/L (over northern Saskatchewan). People living in the vicinity of hazardous waste sites containing organochlorine pesticides may be at an increased risk of dieldrin exposure, as it may enter the environment through leakage from the sites. Levels of aldrin and dieldrin have also been detected in indoor air several years after these pesticides were used in homes.

Reducing Exposure

Exposure to dieldrin from eating sport fish can be reduced by following guidelines in the *Guide to Eating Ontario Sport Fish*. (See Chapter 8. "Food Quality.")

Human Health Considerations

Exposure to single doses of 8–30 mg/kg•bw of aldrin in humans results in adverse effects including vomiting, epigastric pain, drowsiness, headache, sweating, rise in blood pressure, dizziness, and convulsions; a single dose of 25.6 mg/kg•bw results in elevated blood urea nitrogen, gross hematuria, albuminuria, inability of the kidney to concentrate the urine; a single dose of 120 mg/kg•bw causes tachycardia, elevated blood pressure and convulsions. Reported cases of direct exposure to dieldrin suggest that its toxicity is perhaps slightly less than that of aldrin.

Table 1

MAC, MRL, TDI VALUES FOR ALDRIN AND DIELDRIN

Agency	Focus	Level	Comments
Health Canada	Drinking water	0.7 µg/L	MAC
Health Canada	Meat, meat by-products and fat of cattle, goats, hogs, poultry and sheep	0.2 µg/g (ppm) (calculated on fat content)	MRL
	Dairy products	0.1 µg/g (ppm) (calculated on fat content)	MRL
OMOEE	Drinking water	0.7 µg/L	MAC
FAO/WHO	All sources	0.1 µg/kg•bw/day	TDI
WHO	Drinking water	0.03 µg/L	guideline value

MAC: Maximum acceptable concentration
MRL: Maximum residue limit
OMOEE: Ontario Ministry of Environment and Energy
FAO/WHO: Food and Agriculture Organization/World Health Organization
TDI: Tolerable daily intake

WHO Human Blood Guideline[1]

WHO human blood guideline = 100 µg/L (aldrin + dieldrin)

1. WHO Technical Report Series 667. Recommended health-based limits in occupational exposure to pesticides. Geneva: World Health Organization, 1982. Cited in: *Human Toxicology of Pesticides* by F.P. Kaloyanova and M.A. El Batawi. CRC Press Inc. Florida 1991, p. 91.

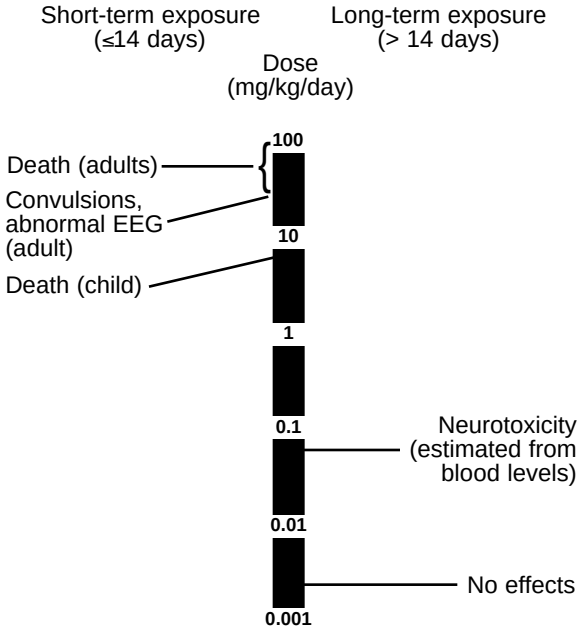
Long-term ingestion of small amounts of dieldrin by male volunteers did not result in any neurological disturbances nor any other health effects or clinical chemistry changes. During long-term low-level exposure to aldrin or dieldrin, an equilibrium is reached such that intake equals excretion and blood levels remain nearly constant.

Occupational exposure studies have produced conflicting results regarding increases in or increased frequency of pneumonia and other pulmonary diseases, serum cholesterol, hypertension, serum hepatic enzymes (SGOT, SGPT), sister chromatid exchanges, contact dermatitis (dermal exposure), immunohemolytic anemia, abnormal EEG patterns. The studies are limited by small sample size and/or possible exposure to other chemicals. It is also not certain how the subjects were exposed, i.e., oral, inhalation or dermal.

One case report indicates that ingestion of fish containing high levels of dieldrin resulted in immunohemolytic anemia. Another study indicates that aldrin levels in blood and placental tissues of women who had premature labour or spontaneous abortions were significantly higher than in women with normal deliveries. Interpretation of this study is difficult because six other organo-chlorine pesticides were also elevated and confounding factors (smoking, alcohol consumption) were not included in the analyses. Studies in humans have not addressed whether adverse reproductive or developmental effects could occur as a result of exposure to aldrin or dieldrin.

Figure 1

HUMAN HEALTH EFFECTS FROM INGESTING ALDRIN OR DIELDRIN



Adapted from: U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR). *Public Health Statements – What you need to know about toxic substances commonly found at Superfund hazardous waste sites.* Atlanta, Georgia.

The International Agency for Research on Cancer (IARC) has concluded that evidence for carcinogenicity of aldrin and dieldrin is inadequate, and has placed aldrin and dieldrin in Class 3 (non-classifiable).

References

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United States. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry. *Toxicological Profile for Aldrin/Dieldrin*. Atlanta, Georgia, April 1993.

World Health Organization. *Guidelines for drinking-water quality*. 2nd ed. Vol. 2. Geneva, 1996.

Aluminum

Origins and Uses

Aluminum is the third most common element in the Earth's crust and makes up more than 8 percent of it by weight. It does not occur as a free metal in nature and is always found combined with other elements in the Earth such as minerals and rocks. Aluminum is found in igneous and sedimentary rock, and in soils.

Aluminum is used in water treatment in the form of salts, for example aluminum sulphate (alum) or poly-aluminum chloride. It is added as a coagulant to assist in the removal of pathogenic micro-organisms, organic compounds and particulate matter from raw surface water. The amount of aluminum remaining in the treated water (residual) depends on the aluminum levels in the source water, the amount of aluminum used, water acidity and temperature, fluoridation of the water, frequency of addition of the aluminum, and the efficiency of the filtration process. Aluminum compounds are also used in many drugs (e.g., antacids and ASA) and consumer products (e.g., cooking utensils, food packaging material, antiperspirants, and cosmetics).

Aluminum occurs naturally in many foods. Certain aluminum compounds used as food additives may contribute additional aluminum to certain foods including dairy products (processed cheese and yogurt), grain products, desserts and beverages. The addition of aluminum to processed foods is not controlled by the government. It is the manufacturer's responsibility to decide on the most appropriate amount of aluminum, according to Good Manufacturing Practice.

Persistence and Movement in the Environment

Most of the aluminum in soil minerals is tightly bound and weathers slowly. Levels in soil, groundwater and in surface water are low compared with the concentrations found in the surrounding rocks. Weathering and leaching rates of aluminum can be elevated by acidification. Acidic environments cause the dissolved aluminum content of the surrounding waters to increase resulting in a rise in the mobility of aluminum. Aluminum becomes bioavailable to plant roots under acidic or very alkaline conditions. Aluminum can also be found in the air bound to dust particles.

Exposure

Humans are exposed to aluminum in the air, water and food because of its natural occurrence in the environment. The average adult takes in 9 to 14 milligrams of aluminum each day from all exposure routes. Average intakes for children are estimated to be between 2 and 6 mg/day. The major exposure route (90 percent) for individuals not using aluminum-containing medications is from food. While levels of aluminum in foods may increase when aluminum cookware, utensils, and wrapping are used for food preparation and storage (particularly acidic foods such as tomatoes, cabbage, rhubarb and sauerkraut), studies to date have shown that contamination from this source contributes only a small portion of the total daily aluminum exposure. Health Canada surveys have found higher levels of aluminum in infant formulae than in human or cow's milk although intakes are still significantly lower than the Joint Food and Agriculture Organization/World Health Organization Expert Committee on Food Additives and Contaminants (JECFA) tolerable intake guideline (see table below).

Exposure to aluminum from drinking water is usually less than 3 percent of the total daily exposure from food and drugs. Based on an intake of 1.5 L/day, the aluminum exposure for a Canadian adult ranges from 0.006–0.8 mg/day. For Ontario residents, it is estimated that the mean intake of aluminum is 0.16 mg/day. Actual intake may vary widely across the country depending on the natural presence of aluminum in drinking water. Levels in unpolluted air are generally low and result in intakes of less than 0.002 mg/day. Ambient aluminum levels in industrial areas may be much higher resulting in intakes of up to 0.124 mg/day.

Very little of the aluminum taken in is retained in the body. The human body's main defence against aluminum in food is that it does not allow most of the ingested aluminum to pass through the intestinal wall. This is because aluminum in food is in a bound form. However, aluminum in drinking water can be absorbed to some extent because aluminum in water following treatment seems to be largely in a "free" (unbound) form. Nonetheless, the amount of aluminum absorbed from drinking water is generally very small.

Reducing Exposure

In the past, controlling levels of aluminum in dialysis fluids and medications in dialysis patients significantly reduced the incidence of dialysis encephalopathy (a progressive form of dementia characterized by speech and behavioural changes, tremors, convulsions, and psychosis). However, there has been a recent change to aluminum-free water dialysis (< µ10 g/L aluminum in water).

In order to reduce exposure to aluminum, avoid:

- using cosmetics such as anti-perspirants, antacids, buffered aspirins and other medications containing aluminum compounds.
- cooking and preserving acidic foods such as tomatoes and coffee in aluminum pots and pans.
- using food preservatives, such as baking powder, containing aluminum powder.

Human Health Considerations

Aluminum has been considered to be relatively non-toxic in healthy individuals, who can tolerate oral daily doses of as much as 7.2 grams of aluminum without any apparent harmful effects. Aluminum is included in the U.S. Food and Drug Administration's list of substances generally regarded as safe for consumption. However, there is now abundant evidence that aluminum may cause adverse effects on the nervous system. Abnormal disposition of aluminum in nerve tissues is thought to be associated with severe diseases of the nervous system, such as Parkinson's disease, amyotrophic lateral sclerosis (Lou Gehrig's disease) and

Table 1

PTDI AND OPERATIONAL GUIDELINE VALUES FOR ALUMINUM

Agency	Focus	Level	Comments
Health Canada	Drinking water	There is no health-based guideline at present [1]	
OMOEE	Drinking water	0.10 mg/L	Operational guideline
JECFA	All sources	1.0 mg/kg•bw/day	PTDI
WHO	Drinking water	There is no health based guideline, however, a concentration of 0.2 mg/L in drinking water provides a compromise between the practical use of aluminum salts in water treatment and discoloration of distributed water.	

PTDI: Provisional tolerable daily intake
JECFA: Joint Food and Agriculture Organization/World Health Organization Expert Committee on Food Additives and Contaminants
OMOEE: Ontario Ministry of Environment and Energy
WHO: World Health Organization

1. The drinking water guideline for aluminum has been proposed to the Federal Provincial Subcommittee on Drinking Water. The subcommittee is evaluating and considering the guideline.

Alzheimer's disease. However, these associations are not completely understood and are still under study. The effects in humans exposed to low levels of aluminum over a long period are not known, but earlier onset or progression of a wide range of diseases of the nervous system is a distinct possibility. With respect to Alzheimer's disease, the epidemiological research needs to be confirmed by analytical studies.

Kidney dialysis patients exposed to high levels of aluminum in dialysis fluids and medications have developed a condition called dialysis encephalopathy. Intake of large amounts of aluminum can also cause brittle or soft bones, anemia, glucose intolerance and cardiac arrest in humans.

The available data from animal studies and epidemiological studies in humans do not indicate that aluminum is a potential carcinogen.

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Canada. Health Canada. *Guidelines for Canadian Drinking Water Quality — Sixth Edition*. Prepared by the Federal-Provincial Subcommittee on Drinking Water of the Federal-Provincial Committee on Environmental and Occupational Health. Ottawa: Canada Communication Group, 1996.

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University of Toronto and McMaster University. "Health Effects of Aluminum: A Critical Review with Emphasis on Aluminum in Drinking Water." For Ontario Ministry of Health. June 4, 1993.

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United States. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry. *Aluminum*. Atlanta, Georgia, September 1995.

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World Health Organization. *Guidelines for drinking-water quality*. 2nd ed. Vol. 2. Geneva, 1996.

Ammonium Phosphates

Origin and Uses

Ammonium phosphates are two chemical compounds composed of nitrogen, hydrogen, oxygen and phosphorus. They are monoammonium phosphate ($(NH_4)H_2PO_4$) and diammonium phosphate ($(NH_4)_2HPO_4$), which are white granular or powdered solids, and are either odourless or have only faint odours. Fires may release toxic and irritating fumes containing ammonia, oxides of nitrogen and phosphorus.

Ammonium phosphate production and use is widespread in Canada. Monoammonium phosphate is mainly used in specialty fertilizers, as food ingredient, in paints, as an agent in dye baths, powder fire extinguishers and for flame proofing fabrics, wood and papers. Diammonium phosphate is used in fertilizers, as a food ingredient, in flame retardants and pharmaceuticals. Phosphorus is an important nutrient in freshwater lakes as it plays a role in determining the extent of plant growth.

Persistence and Movement in the Environment

All forms of ammonium phosphate dissolve rapidly in water, and may alter species population balances in aquatic environments. Phosphorus and nitrogen are nutrients that are readily absorbed by micro-organisms, stimulating the growth of algal blooms. These blooms block sunlight to submerged plants and can produce toxins capable of killing aquatic animals and plants. For example, Lake Erie was severely affected by eutrophication because of heavy nutrient loadings from municipal and industrial sources, in combination with its shallow, warm waters. Restrictions have been successful in reducing phosphate loadings to the Great Lakes to 25 percent of 1972 levels. Ammonium phosphates do not bioaccumulate in organisms, and do not biomagnify up the food chain.

When ammonium phosphates are exposed to soil (e.g., an accidental spill) penetration is unlikely to occur if the soil is dry and clean-up of the ammonium phosphate occurs before precipitation (rain, snow). Ammonium phosphates are non-volatile, therefore they do not disperse in air.

Exposure

Phosphates primarily enter waterways from fertilizer run-off (from agricultural, dairy, beef and other animal production), human and animal wastes, and detergents. While other chemicals such as nitrates and potassium can cause problems with nutrient loading, it has been estimated that 30 to 70 percent of phosphate loading comes from the phosphates added to detergents. Most detergents contain polyphosphates used to bolster their cleaning action.

Reducing Exposure

Use of detergents that are phosphate-free reduces nutrient loading. Agricultural practices that minimize phosphorus levels in run-off can be adopted. Conservation tillage, crop rotation and grass waterways are only a few examples of the practices that should be part of future cropping systems.

Human Health Considerations

Monoammonium phosphate dust is irritating to the eyes, nose and throat. If inhaled, it causes coughing or difficult breathing. Diammonium phosphate vapours also cause coughing, difficult breathing and lung irritation. Contact with solid forms of ammonium phosphate may cause mild irritation to the skin and the eyes.

Standards for Phosphates

Since 1972, the content in all effluent from large municipal waste treatment plants has been limited to 1 mg/L. Sewage treatment plants have been upgraded (to tertiary treatment) to remove phosphates and regulations were enacted restricting phosphate content of detergents to 5 percent by weight. Detergents with less than 1 percent phosphate can be termed "phosphate free." Dish washing liquids are exempt and still contain up to 45 percent phosphate.

References

Canada. Environment Canada, Environmental Protection Programs Directorate. *Ammonium Phosphates: Environmental and Technical Information for Problem Spills (Enviro-TIPS)*. Ottawa: Supply and Services Canada, March 1984.

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United States Department of Health and Human Services, Agency for Toxic Substances and Disease Registry. *Ammonia—Public Health Statements: What you need to know about toxic substances commonly found at Superfund hazardous waste sites*. Atlanta, Georgia.

Arsenic

Origin and Uses

Natural sources of inorganic arsenic are from weathering and erosion of rock and soil. The principal sources resulting from human activity are gold- and base-metal processing. Other sources resulting from human activity include coal-fired power generation, and the disposal of domestic and industrial wastes. Arsenic is used mainly in metallurgical applications, and in wood preservatives. Prior to 1975, arsenical pesticides were used in fruit and vegetable production.

Persistence and Movement in the Environment

When arsenic is combined with elements other than carbon, such as oxygen, chlorine and sulphur, it is called inorganic arsenic. Organic arsenic is formed when arsenic combines with carbon and hydrogen. Organic forms are generally believed to be less harmful to humans than the inorganic forms.

Due to its mobility and reactivity, arsenic moves through aquatic and terrestrial systems where it can undergo chemical and biochemical transformations. Arsenic bioaccumulates in aquatic organisms (including algae, molluscs, crustaceans and fish) in a form that is not as toxic as inorganic arsenic and which is rapidly excreted from the body. Arsenic does not greatly biomagnify through aquatic or terrestrial food chains.

Exposure

Exposure to inorganic arsenic (inhalation/ingestion) occurs through food, drinking water, soil, and ambient air, with food being the major source of intake. Please see Table 1 on estimated daily intakes for more information. The largest contribution to arsenic exposure from food is from shellfish, meat, poultry, grain and dairy products. Exposure to arsenic may be elevated in populations residing in the vicinity of industrial or geological sources, causing drinking water levels to be naturally high. This is the case for Nova Scotia drinking water. Estimates of exposure for such populations, based on data from different areas of Canada, i.e., a "worst-case scenario," are presented in Table 1.

Reducing Exposure

Washing fruits and vegetables will help to remove arsenic (in dirt) on the exterior of produce. Well water containing excessive levels of arsenic should be avoided. Well water can be tested if suspect. Reducing the amount of tobacco smoked will decrease exposure to arsenic.

Human Health Considerations

Non-cancerous effects on the skin have been observed in occupationally exposed populations or in those drinking water containing high concentrations of arsenic. Such effects have generally only been observed at levels associated with a significantly increased risk of cancer. Symptoms of arsenic exposure include nausea, diarrhea, peripheral neuropathy, anemia, vascular lesions, hyperkeratinizations, hyperpigmentation and, at high doses, death.

Table 1

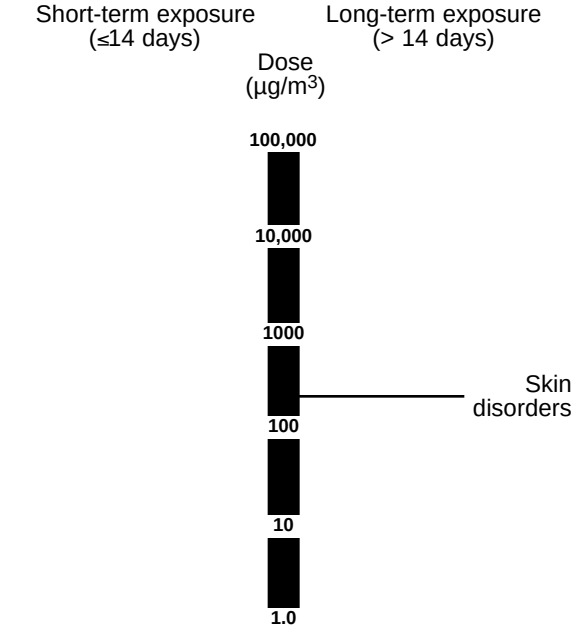
Estimated average daily intake of inorganic arsenic by adults (20–70 years) in the general population and in adults living near point sources in Canada

Medium	General adult population (µg/kg•bw/day)	Adult population living near point sources (µg/kg•bw/day)
Water	0.1	< 0.1–11
Food	0.08	0.08
Air	0.0003	0.003–0.07
Soil/Dirt	0.001–0.004	0.0009–0.1
Total ingested	0.2	< 0.2–11
Total inhaled	0.0003	0.003–0.07
Tobacco smoking	0.01–0.03	

Adapted from: Hughes, K., Meek, M.E. and Burnett, R. 1994. Inorganic arsenic: evaluation of risks to health from environmental exposure in Canada. Environ Carcino & Ecotox Revs. C12(2):145–159.

Figure 1

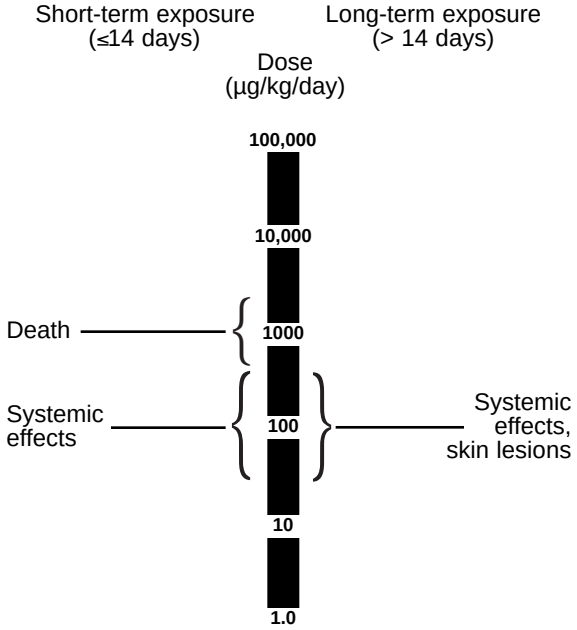
Human health effects from breathing inorganic arsenic



Adapted from: U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR). *Public Health Statements – What you need to know about toxic substances commonly found at Superfund hazardous waste sites.* Atlanta, Georgia.

Figure 2

Human health effects from ingesting inorganic arsenic



Adapted from: U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR). *Public Health Statements – What you need to know about toxic substances commonly found at Superfund hazardous waste sites.* Atlanta, Georgia.

Table 2

IMAC, PTDI AND TOLERANCE VALUES FOR INORGANIC ARSENIC

Agency	Focus	Level	Comments
Health Canada	Drinking water	0.025 mg/L	IMAC[1]
Health Canada	Fruit juices, cider, wine; in beverages as consumed and water in sealed containers other than mineral water	0.1 mg/L (ppm)	tolerance
Health Canada	Edible bone meal	1.0 mg/kg (ppm)	tolerance
Health Canada	Fish protein as defined in B.21.027	3.5 mg/kg (ppm)	tolerance
OMOEE	Drinking water	0.025 mg/L	IMAC
JECFA	All sources	2.0 µg/kg•bw/day as inorganic arsenic	PTDI
WHO	Drinking water	0.01 mg/L	guideline value[2]

IMAC: Interim maximum acceptable concentration
PTDI: Provisional tolerable daily intake
OMOEE: Ontario Ministry of Environment and Energy
JECFA: Joint Food and Agriculture Organization/World Health Organization Expert Committee on Food Additives and Contaminants
WHO: World Health Organization

1. The IMAC will be reviewed periodically in light of developments in treatment technology and additional data on health risks associated with exposure to arsenic in drinking water.
2. Associated with excess skin cancer risk of 6×10^{-4}

Inorganic arsenic compounds are human carcinogens. Arsenic has been associated with an increase in mortality due to lung cancer in workers exposed via inhalation in the occupational environment. Ingestion of high levels of inorganic arsenic in drinking water has been associated with an increased prevalence of skin cancer and an increased incidence of cancer of various internal organs, including the bladder, kidney, liver and lung.

References

Canada. Department of Health and Welfare. *The Food and Drugs Act and Regulations*. Ottawa: Supply and Services Canada, 1981.

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United States. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry. *Toxicological Profile for Arsenic*. Atlanta, Georgia, April 1993.

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Asbestos and Fibrous Materials

Origin and Uses

Asbestos is the commercial name for a family of minerals that separate into dust-like fibres when crushed. They are present naturally in rock formations worldwide. Of the six common types, chrysotile is the major type used in Canada.

Asbestos is a very good insulator against heat, cold, electricity and noise. It is also resistant to fire and many corrosive metals. It has been used for fireproofing (sprayed onto steel beams and overhead surfaces in commercial buildings, apartments and houses), as heat insulation (pipes, furnace boilers, heating and ventilation ducts), and as noise insulation (ceilings and walls). Both the spray application of asbestos and its use as pipe and boiler insulation on heating systems were stopped in 1973. Some of its current uses include insulation in brake linings, clutch facings and automatic transmissions in automobiles.

Fibrous glass (pink insulation) and mineral wool are examples of fibrous materials small enough to be inhaled.

Persistence and Movement in the Environment

Asbestos and fibrous materials tend to be inert until they are installed, removed or otherwise disturbed. It is non-biodegradable and environmentally cumulative. Asbestos does not evaporate, dissolve, burn, or undergo significant reactions with other chemicals. It does not evaporate into the air or dissolve in water. Small fibres may be transported long distances in the air or water. Asbestos fibres do not generally build up in plants or animals.

Exposure

Asbestos exposure is primarily through inhalation, while ingestion and dermal absorption are minor exposure routes. The size and shape of asbestos fibres affect the ability of the lungs to effectively remove them.

Exposure to asbestos fibres (through inhalation) is greatest in the occupational environment (mining, milling, industrial use of asbestos, and construction). Asbestos levels in ambient air may be elevated near asbestos-emitting industries, but in general concentrations are some thousand-fold less than those in occupational settings. Intake of asbestos from air has been suggested to be < 0.6 µg/day. In a Canadian survey, the fibre concentrations in raw water ranged from 6.4×10^6 – 190×10^6 chrysotile fibres/L. Water treatment further reduces asbestos levels in drinking water. Asbestos intake from water has been estimated at 1.88 ng (2 L/day of water containing 1.9×10^6 fibres/L). See the guidelines section for further explanation of asbestos in water. Nearly all ingested asbestos fibres are excreted in the feces.

Personal exposures to asbestos are determined by the fibre concentrations in the immediate vicinity of a person — measured in fibres per cubic centimetre of air (f/cm^3). In other words, risk is determined by exposures to airborne fibres rather than the presence of asbestos-containing materials in the building. This is an important concept because more exposure can be generated by attempts to remove asbestos than by preventing its release into the air by methods such as enclosure or encapsulation. For general occupants of a building asbestos is not a public health hazard **except**:

a) if they are within the vicinity or range of air circulation of maintenance work disturbing asbestos; or

b) significant quantities of asbestos-containing insulation have fallen onto building surfaces and are being disturbed.

It is wise to periodically check materials containing mineral fibres for signs of deterioration. Avoid skin contact with fibrous glass and mineral wool during renovations, ensure good ventilation and disturb as little as possible. Advice should be sought before removing and disturbing materials thought to contain asbestos.

Reducing Exposure

In a home, if material that might contain asbestos has become exposed, it should be assessed by a professional. If it has fallen on the floor, it should not be dusted, vacuumed, or swept. It can be wiped up with wet rags or sponges and contained in a dust-proof plastic bag for disposal. Professional guidance or services should be sought on repair or removal of asbestos-containing materials. Home owners should not undertake such activities themselves.

Substitute asbestos materials have been developed for private in-house applications. They are called "man-made mineral fibres" (MMMFs). There are however, health implications associated with exposure to these substitute materials. Asbestos is still used in industrial settings.

Human Health Considerations

No deaths due to acute exposure to asbestos have been reported. Diseases associated with high occupational exposure (through inhalation) are asbestosis (fibrosis of the lung), mesothelioma (cancer of the pleural sac lining the lung or the peritoneal sac lining the abdomen), and cancer of the lung. Asbestos-related diseases generally develop 10 to 30 years after exposure. Two studies have investigated asbestos exposure in individuals living in cities for a number of years. Over half of the subjects had some asbestos fibres in their lung tissues; none had evidence of resulting disease.

Extensive studies have revealed that ingested asbestos causes little or no risk of noncarcinogenic injury. Dermal exposure has been associated with the formation of small warts or corns, mostly on the hands.

Table 1

HUMAN HEALTH EFFECTS FROM BREATHING ASBESTOS

Short-term exposure (less than or equal to 14 days)		
Levels in air	**Length of exposure**	**Description of effects**
		The health effects resulting from short-term exposure of humans to air containing specific levels of asbestos are not known
Long-term exposure (greater than 14 days)		
Levels in air (f/m^3)	**Length of exposure**	**Description of effects***
1 000 000	20 years	Lung injury in some people
3 400 000	20 years	Death from asbestosis in some people

* These effects are listed at the lowest level at which they were first observed. They may also be seen at higher levels.

Adapted from: U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR). *Public Health Statements – What you need to know about toxic substances commonly found at Superfund hazardous waste sites.* Atlanta, Georgia.

Table 2

DRINKING WATER VALUES FOR ASBESTOS

Agency	Focus	Level	Comments
U.S. EPA	Drinking water	7 million fibres/L	see below
Health Canada	Drinking water		see below
WHO	Drinking water		see below

The U.S. Environmental Protection Agency's proposed (no regulation exists) maximum level for asbestos in drinking water is 7 million fibres (larger than 10 microns in length) per litre.

The WHO and Health Canada have not established a guideline for asbestos in drinking water, as it is believed that asbestos ingestion at the concentrations found in drinking water do not constitute a health hazard. Exposure to high concentrations of asbestos in drinking water is unlikely, as standard water treatment processes effectively remove asbestos fibres from drinking water supplies.

References

Canada. Health Canada. Guidelines for *Canadian Drinking Water Quality — Sixth Edition*. Prepared by the Federal-Provincial Subcommittee on Drinking Water of the Federal-Provincial Committee on Environmental and Occupational Health. Ottawa: Canada Communication Group, 1996.

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BTEX — Benzene, Toluene, Ethyl benzene and Xylene/Gasoline

BTEX is a name given to the composite of benzene, toluene, ethyl benzene and xylene that are found together in gasoline. Toluene, ethyl benzene and the xylenes (there are three possible isomers) all belong to a group of organic compounds known as alkyl benzenes. Despite the fact that human exposure is frequently to all four of these compounds, the properties and health effects of each individual contaminant are discussed separately. There is currently little information regarding any synergistic or interactive effects of these contaminants.

Benzene

Origins and Uses

As a pure chemical, benzene is a clear, colourless liquid. Benzene occurs naturally in the environment in low concentrations. It is a product of volcanoes and forest fires, and is present in many plants and animals. Benzene is also a major industrial chemical made from coal and oil. It is used to make other chemicals as well as some types of plastics, detergents and pesticides, and makes up about 1 to 2 percent of gasoline. The major source of release of benzene into the environment is through gasoline and other petroleum fuels that together account for 76 percent of the total atmospheric releases.

Persistence and Movement in the Environment

Benzene can be found in air, soil and water from both natural and industrial sources. Benzene does not persist in water or soil because it biodegrades and volatilizes rapidly to the atmosphere, where it undergoes rapid photo-oxidation.

Exposure

In general, people are exposed to benzene from breathing air contaminated by benzene, frequently via tobacco smoke, automobile exhaust and gasoline vapour emissions. More than 99 percent of the total personal exposure is through air. Water, food and beverages have been found to have little to no benzene. Total daily intake from all sources (ambient air, drinking water, food, automobile-related activities, household products) for Canadian adults in the general population is estimated to be 2.4 µg/kg•bw/day. For the same age group, the estimated intakes from ambient air, drinking water, food, automobile related activities and household products are 1.3, 0.02, 0.02, 0.7 and 0.4 µg/kg•bw/day, respectively.

Other environmental sources of benzene include gasoline (filling) stations, underground storage tanks that leak, chemical spills, waste water from industries using benzene, groundwater next to landfills containing benzene, and food products containing benzene naturally. Certain industries also release benzene into the surrounding air. No effect on personal exposure was detected in people living close to major fixed sources of benzene (oil refineries, storage tanks, chemical plants).

For smokers, about 90 percent of their benzene exposure is from mainstream cigarette smoke. Smokers have an average benzene body burden about six to 10 times that of non-smokers. The sources of exposure for non-smokers are mostly automobile exhaust or gasoline vapour emissions. This includes exposure due to outdoor air, indoor exposures and activities such as driving. A smaller percentage of exposure is also due to environmental tobacco smoke, and major point sources such as petrochemical plants or refineries. For Canadian adults in the general population, cigarette smoke may contribute an additional 26 µg/kg•bw/day to the daily intake of benzene, while passive smoke may contribute 0.9 µg/kg•bw/day.

Human Health Considerations

Benzene is harmful to human health, most notably to tissues forming blood cells. The way in which benzene affects health depends on how much the individual is exposed to, and for how long. Acute exposure to high levels affects primarily the central nervous system, resulting in drowsiness, dizziness and headaches. Symptoms usually disappear after exposure stops. Death may occur in humans (and animals) after brief oral or inhalation exposures to high levels. Long-term exposure may affect normal blood production, possibly resulting in severe anemia and internal bleeding. Benzene is considered a "non-threshold toxicant" — a substance for which there is believed to be some chance of adverse effects at any level of exposure.

Human evidence and animal studies have clearly shown benzene to be carcinogenic. Associations between leukemia and benzene exposure in occupationally exposed populations have been observed in many epidemiological and case studies.

Guidelines

The Health Canada Drinking Water Quality Guideline is 5 μg/L (maximum acceptable concentration).

References

Canada. Environment Canada and Health Canada, *Canadian Environmental Protection Act. Benzene, Priority Substances List Assessment Report.* Ottawa: Supply and Services Canada, 1993.

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Wallace, L. 1996. Environmental exposure to benzene: an update. Environ Health Perspect. 104(Suppl 6):1129–1136.

Toluene

Origins and Uses

Toluene is a clear, colourless liquid with a sweet pungent odour. The principal end-use for toluene in Canada is in the production of benzene. It is also used as a solvent in paints and varnishes, pesticide formulations, printing inks, adhesives and sealants, cleaning agents, and for chemical extractions. Toluene is a natural component of petroleum and makes up 8.3 percent of gasoline (most of which is burned during normal engine operation).

Persistence and Movement in the Environment

Toluene does not persist in water or soil because it biodegrades and volatilizes rapidly to the atmosphere where it undergoes rapid photo-oxidation. In humans, toluene is rapidly metabolized and is excreted primarily in the urine.

Exposure

Toluene enters the environment primarily through atmospheric releases such that the most significant route of exposure for the general population is inhalation. Major sources include solvents and gasoline engines which together account for 80 percent of atmospheric releases. For smokers, cigarette smoke is by far the greatest source of exposure. Toluene can also be released into the soil from spills and in leachate from contaminated landfill sites, or from spills and discharge of contaminated effluents.

Reducing Exposure

The easiest way to reduce exposure to toluene is to avoid tobacco smoke and gasoline, paint or solvent fumes.

Human Health Considerations

Studies of occupational groups exposed to toluene in their work environment have shown impairment of the central nervous system, respiratory effects and irritation of the mucous membranes. Toluene can also be toxic to the developing embryo and fetus. There is no clear evidence for teratogenic activity in laboratory animals and humans. The available epidemiological data are inadequate to properly assess the carcinogenicity of toluene in humans. Toluene has been classified as possibly carcinogenic to man by the International Agency for Research on Cancer (IARC).

Table 1

HUMAN HEALTH EFFECTS FROM BREATHING TOLUENE

	Short-term exposure (less than or equal to 14 days)	
Levels in air (ppm)	**Duration of exposure**	**Description of effects***
4		Minimal risk level
100	8 hours	Moderate fatigue
300	8 hours	Severe fatigue, headache
600	8 hours	Extreme fatigue, confusion, dizziness, intoxicated behaviour
	Long-term exposure (greater than 14 days)	
Levels in air (ppm)	**Duration of exposure**	**Description of effects***
1		Minimal risk level (based on animal studies)
200	1—10 years	Headache, dizziness, loss of coordination
300	1—10 years	Impaired memory and thinking ability

* These effects are listed at the lowest level at which they were first observed. They may also be seen at higher levels.

Adapted from: U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR). *Public Health Statements – What you need to know about toxic*

Clinical studies of human volunteers after single exposures as well as after repeated exposures have shown decreases in neurological function with elevated levels of exposure to toluene (375 mg/m^3). These levels of exposure are far in excess of those experienced by the general population.

The estimated total average daily intakes of toluene for various age groups in the Canadian population range from 1.6–21.6 µg/kg•bw/day. They are 50 to 670 times less than the tolerable daily intake derived on the basis of bioassays in animals species, and also 60 to 780 times less than that calculated from data from the clinical studies of human volunteers.

Table 2

GUIDELINES FOR TOLUENE

Agency	Focus	Level	Comments
Health Canada	Drinking water	≤ 24 µg/L	aesthetic objective
WHO	Drinking water	700 µg/L	(allocating 10 percent of the TDI to drinking water)

WHO: World Health Organization

References

Canada. Environment Canada and Health Canada, Canadian Environmental Protection Act. *Toluene, Priority Substances List Assessment Report*. Ottawa: Supply and Services Canada, 1992.

———.Health Canada. *Guidelines for Canadian Drinking Water Quality — Sixth Edition*. Prepared by the Federal-Provincial Subcommittee on Drinking Water of the Federal-Provincial Committee on Environmental and Occupational Health. Ottawa: Canada Communication Group, 1996.

United States. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry. *Toluene — Public Health Statements: What you need to know about toxic substances commonly found at Superfund hazardous waste sites.* Atlanta, Georgia.

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Ethyl benzene

Origins and Uses

The primary sources of ethyl benzene in the environment are the petroleum industry and the use of petroleum products.

Persistence and Movement in the Environment

In general, more than 96 percent of ethyl benzene in the environment is present in air. It is also found in trace amounts in surface water, groundwater and drinking water as well as in certain foods.

Human Health Considerations

In humans, ethyl benzene is almost completely converted to soluble metabolites, which are excreted in urine.

There is very little information on the health effects of exposure to ethyl benzene, especially in regard to reproduction, long-term toxicity, carcinogenicity or genotoxicity. The acute oral toxicity is low.

Table 3

GUIDELINES FOR ETHYL BENZENE

Agency	Focus	Level	Comments
Health Canada	Drinking water	2.4 µg/L	aesthetic objective
WHO	Drinking water	300 µg/L	10 percent allocation of TDI to drinking water.

WHO: World Health Organization
TDI: Tolerable daily intake

References

World Health Organization. *Guidelines for drinking-water quality*. 2nd ed. Vol. 2. Geneva, 1996.

Xylene

Origins and Uses

Xylenes are clear, colourless, volatile liquids with a strong, aromatic odour. Most xylenes are produced from the catalytic reformation of petroleum and as by-products of the cracking of crude and heavy oil. Minor amounts are produced from coal-derived coke oven light oil.

The major end-use for xylenes in Canada is as an octane enhancer in gasoline, and as solvents in products including paints, varnishes and other coatings, pesticide formulations, printing inks, dyes, adhesives and sealants, cleaning agents, degreasing agents, paint removers, and for chemical extractions. It is also used as feed stock for the plastics industry.

Total xylene emissions into the atmosphere are expected to decline in the future, mainly because of the planned reduction of emissions of volatile organic compounds (VOCs) from light-duty vehicles, and the efforts to reduce VOC emissions from a variety of other sources in order to reduce ground-level ozone (see the Contaminant Profile for Smog and Ground-Level Ozone).

Persistence and Movement in the Environment

Because most of the xylene in the environment is present in the air, the atmosphere plays an important role in the distribution and fate of xylene. Once xylenes are released into the air, they photo-oxidize relatively quickly to compounds that are then further degraded. Xylenes do not persist in water or soil although there have been reports of cases where xylenes leached through soils to groundwater. The movement through soils is slowed in the presence of organic matter and in soils with high moisture content. Xylenes are degraded by micro-organisms in soil, groundwater, surface water, and sediments.

Xylenes do not persist in either water, air or soil. In humans, xylenes are almost completely metabolized and excreted in urine.

Exposure

Xylenes are released into the air primarily from their use as solvents and from transportation sources. Releases into soil and water are generally through spills and leakage from petroleum and other chemical products. Releases have resulted in the presence of measurable quantities of xylenes in air, water, and soil in Canada. The highest concentrations of xylenes in groundwater in Canada have been recorded near waste disposal sites, including beneath landfill sites, near deep injection wells formerly used for the disposal of liquid industrial waste, and near an active industrial chemical waste disposal lagoon.

About 300 registered pest-control products in Canada contain xylene. Because many of these products are applied to crop foliage or directly to the soil, much of the xylenes in these formulations can be expected to reach the soil surface.

While the data on food as a source of exposure is inadequate, it is likely that intake from foods is negligible compared with intake from the air. Exposure to xylenes is higher in smokers.

Reducing Exposure

Exposure to xylenes can be reduced by a decrease in smoking, or exposure to tobacco-smoke, and a decreased exposure to solvent and paint use.

Table 4

HUMAN HEALTH EFFECTS FROM BREATHING XYLENE

	Short-term exposure (less than or equal to 14 days)	
Levels in air (ppm)	**Length of exposure**	**Description of effects***
100	1 day	Eye, nose, and throat irritation
299	70 minutes	Delayed response to a visual stimulus and impaired memory during exercise
	Long-term exposure (greater than 14 days)	
Levels in air (ppm)	**Length of exposure**	**Description of effects***
		The health effects resulting from long-term exposure of humans to air containing specific levels of xylene are not known

* These effects are listed at the lowest level at which they were first observed. They may also be seen at higher levels.

Adapted from: U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR). *Public Health Statements – What you need to know about toxic substances commonly found at Superfund hazardous waste sites.* Atlanta, Georgia.

Human Health Considerations

The available epidemiological evidence is limited to a few studies of small groups of workers who were exposed to thinners and solvents containing not only xylenes but also high percentages of benzene or toluene, and other aromatic and non-aromatic compounds. Due to the limited power of the studies and the mixed exposures causing the observed effects, the epidemiological data are inadequate to properly assess the health risks from xylenes in humans.

While the human data are considered insufficient for development of a tolerable daily intake (TDI), a TDI of 0.144 mg/kg•bw/day has been developed on the basis of animal studies. The estimated total average daily intake of xylenes from various sources for different age groups in the Canadian population ranges from 3.2–9.5 g/kg•bw/day; this is 15 to 45 times less than the TDI.

According to a 1993 *Canadian Environmental Protection Act* (CEPA) assessment, xylenes are not entering the Canadian environment in quantities or under conditions that may constitute a danger to the environment on which human life depends, or to human health. There is currently no convincing evidence for teratogenicity or carcinogenicity. Based on the information available from animal studies, xylenes have been classified as probably not carcinogenic to humans under CEPA.

Table 5

GUIDELINES FOR XYLENE

Agency	Focus	Level	Comments
Health Canada	All sources	xylene (mixed isomers) 1.5 mg/kg•bw/day	TDI
Health Canada	Drinking water	xylenes (total) ≤ 300 µg/L	aesthetic objective
WHO	Drinking water	500 µg/L	allocating 10 percent of the TDI to drinking water

WHO: World Health Organization
TDI: Tolerable daily intake

References

Canada. Environment Canada and Health Canada, *Canadian Environmental Protection Act. Xylenes, Priority Substances List Assessment Report.* Ottawa: Supply and Services Canada, 1993.

———. Health Canada. *Guidelines for Canadian Drinking Water Quality — Sixth Edition*. Prepared by the Federal-Provincial Subcommittee on Drinking Water of the Federal-Provincial Committee on Environmental and Occupational Health. Ottawa: Canada Communication Group, 1996.

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World Health Organization. *Guidelines for drinking-water quality*. 2nd ed. Vol. 2. Geneva, 1996.

Cadmium

Origin and Uses

Cadmium's major natural sources are weathering and erosion of rock and soil (transport by rivers to the world's oceans), forest fires and volcanic activity (atmospheric release). Cadmium is produced commercially as a by-product of zinc refining. In Canada, industrial wastes (base metal smelting and refining operations) account for the majority of cadmium releases to air and water. Globally, cadmium has four main applications: nickel-cadmium batteries, coatings and pigments, stabilizers in plastics and synthetic products, and as alloys. For example, cadmium may be a constituent of solders used in fitting water heaters or incorporated into stabilizers in (black polyethylene) pipes.

Persistence and Movement in the Environment

Cadmium is most often found as inorganic cadmium, in combination with chloride (cadmium chloride), oxygen (cadmium oxide) or sulphur (cadmium sulphate, cadmium sulphide). High soil acidity favours cadmium availability to plants and water. With respect to biota, cadmium accumulates in freshwater and marine organisms. Because it cannot break down and is not easily expelled from the body, levels of cadmium tend to increase with age. Although biota may accumulate cadmium, most evidence suggests that little or no biomagnification occurs.

Exposure

For nonsmokers of the general population, the principal exposure to cadmium is through the ingestion of food. Human absorption studies suggest that < 5 percent of ingested cadmium is absorbed. Levels in food may be elevated where cadmium exists in increased levels in the soil due to atmospheric deposition, fertilizer use, or sludge application. Kidney and liver of terrestrial wildlife (e.g., moose, caribou, bear) and marine mammals (e.g., dolphins, whales) contain high enough levels of cadmium that consumption advisories have been issued. Low levels of cadmium are found in grains, cereals, potatoes and leafy vegetables. For the general adult population, estimated intake of cadmium through food is 0.21 µg/kg•bw/day.

Tobacco smoke is a significant source of exposure (2.0 µg Cd/Canadian cigarette). About 10 percent of the cadmium in a cigarette is transferred to mainstream smoke and becomes available for inhalation (0.187 µg Cd/cigarette). Cadmium in cigarette smoke is both respirable and absorbed (estimated at 40 percent). A smoker has a higher respiratory intake of cadmium (≥ 2.0 µg/day for ≥ 10 cigarette/day) than does a non-smoker (<0.1 µg/day).

Cadmium concentrations in ambient air reflect proximity to urban and industrial areas, with Canadian values comparable to those recorded worldwide. In non-occupational settings, inhalation of cadmium in air is not a significant source of exposure. For the general adult population, the estimated intake of cadmium through air is 0.00033–0.0013 µg/kg•bw/day.

Intake from drinking water and water-based beverages is small compared with dietary intake. Cadmium may be present in some water supplies as a result of its use in pipes (see Origin and Uses section above). For the general adult population, the estimated intake of cadmium through drinking water is 0.01–0.09 µg/L. Mean concentrations for Canadian rivers, lakes and near shore surface water are 0.01–0.12 µg/L for areas away from cadmium mineralization or contamination. Dermal absorption is not a significant route of cadmium exposure.

Cadmium is present in many gold and silver solders used in making jewellery, as well as in the dust produced from grinding or engraving cadmium-plated surfaces. Cadmium is also present in some paint pigments, especially certain artists' colours.

Reducing Exposure

Reduce exposure to tobacco smoke (especially active smoking). Follow any recommendations provided by provincial, territorial or federal governments regarding consumption of wildlife offal (internal organs) and other foods. When working with cadmium containing metals, make sure that the work area is well ventilated. Avoid smoking, eating and drinking in these areas. Use latex gloves when working with cadmium based paints and do not put paint brushes in the mouth.

Human Health Considerations

Most cadmium is stored in the liver and kidneys. After absorption, cadmium is transported in the blood to the liver where it is bound to a protein called metallothionein (Mt). After accumulation of cadmium-Mt in the liver, it is slowly released into the blood and subsequently reabsorbed by the kidney. Mt is present in most organs but is in highest concentrations in the liver, particularly following recent cadmium exposure, and in the kidney. Cadmium bound to Mt within tissues is thought to be nontoxic; however when the levels of cadmium exceed the critical concentration, it becomes toxic.

The main long-term effects of high level exposure to cadmium are kidney disease, chronic obstructive lung disease (limited to occupational exposure), and bone disease (osteomalacia, osteoporosis, spontaneous fractures and severe pain). A disease called itai-itai was first noticed in Japan. It has been associated with consumption of foods with elevated levels of cadmium over the long-term (compared with reference areas) and is characterized by bone disease. This disease is believed to have resulted from a combination of nutritional deficiencies and cadmium-induced nephropathy.

Cadmium's carcinogenic effects have been demonstrated in animal studies (lung tumours in rats inhaling cadmium compounds). Epidemiologic studies of occupationally exposed workers suggest a possible association between cadmium inhalation and lung and prostatic cancer. The latter association is weaker than it appeared in the 1980s and has not been confirmed in subsequent follow-up studies. There is some debate as to whether smoking, or exposure to other chemicals has contributed to these findings. The International Agency for Research on Cancer (IARC) has classified cadmium as probably carcinogenic to humans.

Chronic exposure to high levels of cadmium has been reported to cause mild anemia, anosmia (loss of smell), yellowing of teeth, and occasionally, liver damage. No conclusive evidence indicates that cadmium alone causes hypertension. People with low calcium, protein, or iron reserves absorb cadmium more efficiently, and thus may be at increased risk of developing toxic effects associated with cadmium, as discussed earlier.

Table 1

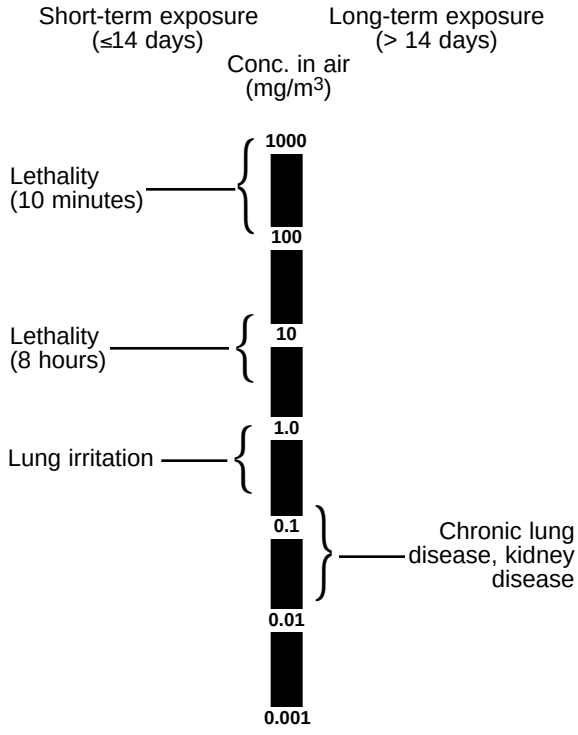
MAC AND PTWI VALUES FOR CADMIUM

Agency	Focus	Level	Comments
Health Canada	Drinking water	0.005 mg/L	MAC
OMOEE	Drinking water	0.005 mg/L	MAC
WHO/FAO	Drinking water	0.003 mg/L	Guideline value
WHO/FAO	Air	< 20 ng/m^3 urban centres < 5 ng/m^3 rural centres	
WHO/FAO		400–500 g/week/adult	PTWI
JECFA	All sources (for a non-smoking person)	7.0 µg/kg•bw/week	PTWI

MAC: Maximum acceptable concentration
OMOEE: Ontario Ministry of Environment and Energy
PTWI: Provisional tolerable weekly intake
JECFA: Joint Food and Agriculture Organization/World Health Organization Expert Committee on Food Additives and Contaminants
FAO/WHO: Food and Agriculture Organization/World Health Organization

Figure 1

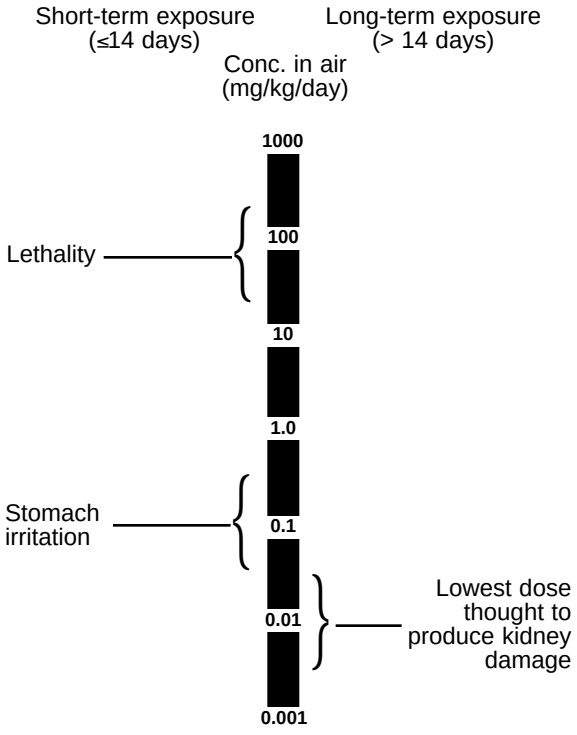
HUMAN HEALTH EFFECTS FROM BREATHING CADMIUM



Adapted from: U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR). *Public Health Statements – What you need to know about toxic substances commonly found at Superfund hazardous waste sites.* Atlanta, Georgia.

Figure 2

HUMAN HEALTH EFFECTS FROM INGESTING CADMIUM



Adapted from: U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR). *Public Health Statements – What you need to know about toxic substances commonly found at Superfund hazardous waste sites.* Atlanta, Georgia.

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Campylobacter spp.

Origin

Campylobacter are bacteria that are found worldwide in a range of animals, including humans. *Campylobacter jejuni* and *Campylobacter coli* are the most common of the 10 or so species.

Persistence and Movement in the Environment

Animals are the primary reservoir of *Campylobacter*. They are found on raw foods of animal origin, especially poultry (but rarely beef), raw clams and shellfish, and also in human and animal excreta. Birds are a well-documented carrier of *Campylobacter*; it is released in their droppings. Infection may be passed from animal to person, from person to animal and from person to person.

Water is an important reservoir of *Campylobacter*, especially in the summer and fall when levels of *Campylobacter* in water are highest. In addition, water serves as a means of contact through which *Campylobacter* may reach people and domestic animals. Heavy rainfall and the subsequent run-off from adjacent farmland were found to contribute to increased counts of *Campylobacter* in the river system. *Campylobacter jejuni* has been tested for in drinking water, river water, and sewage; results indicate that its survival is restricted to a few days.

Exposure

The illness caused by *Campylobacter* may be transmitted through the ingestion of food or water containing the organism. The primary food sources of human infection are of animal origin, especially raw (unpasteurized) milk and chickens (feces, and both fresh and frozen birds). The organisms are excreted in the feces of healthy domestic animals. Studies with chickens, turkeys and cattle have shown that as much as 50 to 100 percent of a flock or herd of these animals excrete *C. jejuni.* Domestic pets (e.g., cats, dogs), farm animals and the handling of raw meat have been sources of infection in humans. The incidence of infection appears to greatly increase during the summer months. *Campylobacter* may also be sexually transmitted.

Campylobacter has been associated with samples collected adjacent to, or downstream from, sewage works. A *campylobacter* enteritis outbreak has been linked to the community raw water system (Taylor et al. 1983).

Reducing Exposure

To prevent outbreaks of *Campylobacter*:

- Consume only pasteurized milk.
- Properly cook all foods of animal origin.
- Avoid cross-contamination of food. This can be done by keeping raw and cooked foods separated by using separate surfaces and equipment. These should be thoroughly cleaned before and after use. Different personnel should handle raw and cooked foods, and should observe strict personal hygiene in addition to washing their hands before and after handling foods, especially raw meat and poultry.

Human Health Considerations

Campylobacter jejuni appears to be by far the most important species from the standpoint of human diseases. The infection caused by *Campylobacter* is called campylobacteriosis, also known as campylobacter enteritis or gastroenteritis. For food-borne infection, the incubation period is usually two to five days (ranges from one to 11 days). The symptoms are abdominal pain, severe diarrhea (for up to three days; sometimes with blood in the stool), fever and flu-like symptoms. Nausea and vomiting are uncommon. Untreated individuals may excrete the organism for up to several months. Very young children are at high risk to campylobacteriosis.

Guidelines and Other Values

The Guidelines for Canadian Recreational Water Quality do not specify a limit for *Campylobacter jejuni* in recreational waters. Sampling should be carried out when there is evidence (epidemiological or other) of its presence in the water or to assess the hazards of excessive utilization of the water with possible person-to-person transfer of pathogens. In ready-to-eat foods, *Campylobacter* is unacceptable at any concentration.

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Carbon Monoxide (CO)

Origin and Uses

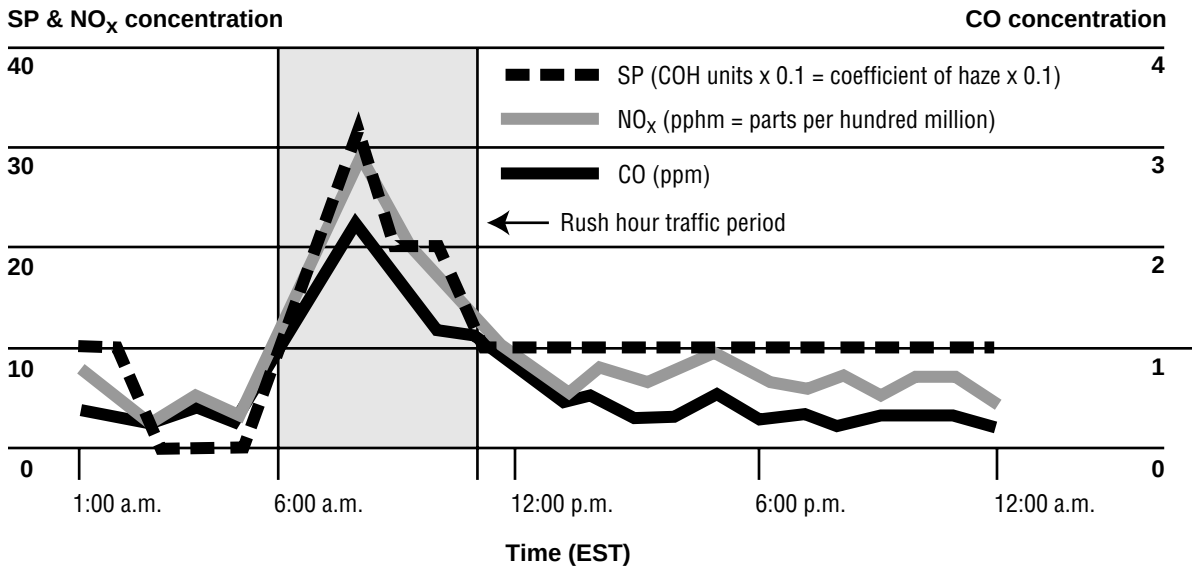
Carbon monoxide (CO) is a colourless, odourless gas that arises from incomplete combustion. It is produced primarily through natural sources: volcanic, marsh and natural gases, the world's oceans, fires, and electrical storms. Human activity accounts for about 10 percent of total worldwide CO production. The major manufactured source of CO in Canada is from fuel combustion in vehicles and stationary engines. Other important sources of CO emissions are industrial, such as burning fuel to generate electricity or heat; sources in and around the home such as fires, wood or gas stoves, and kerosene lamps; and waste disposal. Indoors, tobacco smoke is the most significant anthropogenic source of CO.

Persistence and Movement in the Environment

Carbon monoxide is non-persistent and dissipates with air currents. Carbon monoxide reaches a peak concentration during the morning rush hour and then levels off. This is due largely to the wind patterns. Light wind conditions in the morning limit vertical mixing and dispersion, resulting in a build-up of CO. The winds generally increase in speed in the afternoon, allowing for more mixing, resulting in lower CO concentrations.

Figure 1

DAILY VARIATION OF VEHICLE RELATED POLLUTANTS IN DOWNTOWN TORONTO



Note: Bay/Grosvenor monitoring site, April 22, 1992
Source: Ontario Ministry of Environment and Energy. *Air Quality in Ontario — A Review of the State of Air Quality.* 1992.

Exposure

Carbon monoxide in the body is either endogenously produced through the breakdown of hemoglobin and other heme-containing pigments, or it is inhaled. Carbon monoxide has a much higher affinity (about 250 times) for hemoglobin than oxygen does. When CO binds to hemoglobin, carboxyhemoglobin is formed. Human body burdens of carboxyhemoglobin depend on many factors other than the concentration of carbon monoxide in the air that is inhaled. These include time of exposure, pulmonary ventilation, and blood volume.

Large variations in CO concentrations exist in any place at any time. The exposure of individuals to CO is also quite variable. It depends on the type of activity in which a person is engaged, the duration of the activity, its location (e.g., indoors, outdoors, at a shopping mall, in a vehicle, at work or school, in a parking garage), and the proximity to CO sources. High levels of carbon monoxide in the home can be caused by the "back draughting" of a furnace or fireplace — when too little ventilation causes gases from the flue or chimney to be drawn back into the home. Other indoor exposures can be caused by running vehicles in an attached garage, smoking and the use of unvented kerosene heaters.

Reducing Exposure

Precautions should be taken to ensure that fireplaces are drawing well and furnaces have enough air supply. Ensure that kerosene heaters used in cottages and homes are well vented and that they are not used in tents or trailers. Reduced vehicle use, especially in garages attached to the home, will decrease carbon monoxide exposure.

Human Health Considerations

The toxic effects of carbon monoxide are due to its preferential combination with the heme component of red blood cells to form carboxyhemoglobin, which reduces the capacity of the red blood cells to carry oxygen to the tissues. The tissues with the greatest oxygen demand — heart, brain and exercising skeletal muscle — are the most sensitive to the effects of CO.

The long-term health effects of exposure to low levels have not been well documented. High levels, usually indoors can result in headache, drowsiness, cardiac arrhythmias, and in sufficient levels — coma and death. Misdiagnosis of carbon monoxide exposure is not uncommon as some of these symptoms resemble those of a flu-like illness (headache, dizziness).

Effects have been observed in healthy adults at carboxyhemoglobin concentrations of 5 percent (of available active hemoglobin). These effects include a decrease in maximum aerobic capacity; impaired work capacity; reductions in vigilance, visual perception, manual dexterity and performance of complex sensory-motor tasks. Although epidemiological studies have found associations between CO exposure and low birth weight, slowed postnatal development and incidences of sudden infant death syndrome (SIDS), the data were not sufficient to establish causal relationships.

Individuals should not be exposed to CO that would result in carboxyhemoglobin levels of 5 percent for any but transient periods. Individuals that are especially susceptible to CO should not be subjected to concentrations giving carboxyhemoglobin levels exceeding 2.5 percent.

Chronic exposure studies of animals (rats and mice) have revealed physiological and behavioural changes including impairment in time discrimination and consistent trace metal loss. More work is required in this area before any firm conclusions can be drawn.

Table 1

NATIONAL AMBIENT AIR QUALITY OBJECTIVES FOR CARBON MONOXIDE

Objective	Duration	
	1-hour	8-hour
Maximum Acceptable Concentration	35 mg/m^3 (31 ppm)	15 mg/m^3 (13 ppm)

High levels of CO are considered most harmful to people with severe anemia, chronic lung disease (bronchitis, emphysema), arteriosclerosis of the coronary and peripheral vessels, and chronic angina. Other possible risk groups include pregnant women, fetuses, newborn infants, people living at high altitudes, and people with cardiovascular or respiratory disease — especially the elderly and young children. Smokers may be a special risk group, because they have higher levels of carbon monoxide from smoking.

In Canada, objectives have been set for carbon monoxide in air under the National Ambient Air Quality Objectives.

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Chlorofluorocarbons (CFCs)

Origin and Uses

Chlorofluorocarbons are either fully halogenated (CFC-11, CFC-12, CFC-13, CFC-112, CFC-112a, CFC-113, CFC-113a, CFC-114, CFC-114a, CFC-115) or partially halogenated (HCFC-141b, HCFC-142b, HCFC-132b, HCFC-133a, HCFC-123, HCFC-124). In fully halogenated CFCs, all of the hydrogen atoms have been substituted with fluorine and chlorine atoms. In contrast, partially halogenated CFCs only have part of their hydrogen atoms substituted.

Both kinds of compounds are not known to occur naturally in the environment. CFC-11, CFC-12, and CFC-113 are also potentially ozone depleting. Worldwide uses include refrigerants, solvents, foam-blowing agents and insulator in foam production, aerosol propellants, sterilants, and as intermediates for plastics. Most CFCs releases to the environment occur during the disposal of waste refrigerant-containing equipment, and not during manufacture, storage or handling. HCFC release is not significant in comparison with that of CFCs.

Persistence and Movement in the Environment

Almost all CFCs released into the environment eventually accumulate in the atmosphere. They are stable compounds that do not degrade at ground level. They have exceedingly long atmospheric lifetimes relative to most other atmospheric pollutants. The current best estimates for the average residence times in the atmosphere are 65, 110, 400, 90, 180 and 380 years for CFC-11, CFC-12, CFC-13, CFC-113, CFC-114 and CFC-115 respectively. Atmospheric lifetimes for HCFCs are 8, 19, 4, 5, 2, and 7 years for HCFC-141b, HCFC-142b, HCFC-132b, HCHC-133a, HCHF-123 and HCFC-124, respectively. Depletion of stratospheric ozone (from 11 to 29 km above the Earth's surface) by CFCs increases penetration of ultraviolet-B (UV-B) radiation to the Earth's surface and influences changes in the vertical distribution of stratospheric ozone and water vapour that contribute to global warming effects and altered climatic conditions. The global-warming potentials for HCFCs are expected to be lower than those of CFCs, and should not contribute significantly to global warming.

CFCs tend to accumulate in lipid-rich tissues of organisms, including people. CFC-11 has been detected in various organs of fish and molluscs, although its bioaccumulation potential is thought to be low.

Exposure

People are exposed to CFCs primarily through inhalation, but also through ingestion and dermal contact. CFCs are eliminated from the body almost exclusively through the respiratory tract via exhalation, regardless of the route of exposure. Contributions from local sources of emission account for higher CFC concentrations in urban/suburban air compared with rural/remote areas. There is no significant variation of CFCs with altitude up to 6 km above the Earth's surface.

CFCs are present in saltwater and freshwater, and in snow and rain water in Alaska, Lake Ontario and in the Niagara River. They have not been detected in drinking water. The presence of CFCs in processed food has not been documented.

Reducing Exposure[1]

People can reduce their exposure to CFCs by purchasing ozone-friendly products. For example, choose Canadian-made aerosol products as they do not contain CFCs. All new cars now have CFC-free air conditioning systems. In older cars, individuals should make sure their car's air conditioning systems are properly maintained, and that CFC coolants are captured and recycled during servicing.

New refrigerators are now available that do not contain CFCs. Individuals can help reduce CFC emissions by ensuring that refrigerators and freezers that do contain CFCs are carefully serviced and that companies recover and recycle the CFC coolants rather than just replace them. Some municipalities have programs that remove CFCs from refrigerators before disposal.

Human Health Considerations

There is currently no evidence that human health effects are likely to occur at CFC concentrations found in the general environment or even at higher levels sometimes observed in urban areas. Controlled studies of volunteers exposed to CFCs have been conducted. Health effects from these studies include significant acute reduction of ventilatory lung capacity, bradycardia and increased variability in heart rate and atrioventricular block; and difficulty in mental concentration. For short- term exposures of up to 1000 ppm, no adverse health effects are expected.

Elevated UVB radiation is expected to result in increased incidences of non-melanoma skin cancers and of cataracts. It is estimated that non-melanoma skin cancer rates will increase by 5 percent for every 1 percent depletion of ozone. Other potential health effects from increased UVB radiation include a higher incidence of some infectious diseases, as a result of effects on the immune system and of cutaneous melanomas. See Chapter 3 "Contaminants" for more information on ultraviolet radiation. The available information indicates that CFCs have little or no mutagenic or carcinogenic potential.

No data are available on the human health effects of HCFCs.

Table 1

REGULATIONS FOR OZONE DEPLETING SUBSTANCES

Agency	Comments
Government of Canada	As a result of Canada's ozone protection programs and Canada's obligations under the *Montreal Protocol on Substances that Deplete the Ozone Layer*, as of January 1, 1996, Canada has prohibited the production and import of newly produced CFCs and other ozone depleting substances (ODS) except for certain essential uses such as CFCs for metered dose inhalers for the treatment of asthma and chronic obstructive pulmonary disease. Environment Canada regulates the production, use, import and export of ODS and certain products containing ODS under the *Canadian Environmental Protection Act.*
Provincial governments	The provinces are responsible for implementing programs for recovery and recycling of ozone-depleting substances. Nine provinces have regulations on the recovery and recycling of ODS. The Yukon and Newfoundland are developing regulations. Environment Canada chairs a federal-provincial working group that provides support for implementing the recovery, recycling and reclamation of CFCs.

1 The information in the Reducing Exposure section has been adapted from: *Chemical Control Bulletin: An Update on Environmental Issues. Replenishing the Ozone Layer.* Environment Canada, from the Commercial Chemicals Website. December 1995.

References

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Chlorophenols/PCP

Origins and Uses

Chlorophenols are organic compounds that are made up of a phenol molecule and one or more atoms of chlorine. Of the 19 possible congeners, the ones with greatest biological activities are those with the greatest chlorination (e.g., pentachlorophenol and tetrachlorophenol).

The chlorophenols most often used in Canada are pentachlorophenol (PCP) and tetrachlorophenol (T_4CP). These are used solely as wood preservatives (pressure-treating timber for telephone poles). The use of chlorophenols in sapstain control, for in situ groundline treatment of existing utility poles, and in most specialty applications (paints, stains, wood joinery products, industrial water treatment products, oil field biocides and material preservatives) were terminated on December 31, 1990. The lower chlorinated phenols were used in Canada for the manufacture of higher chlorinated phenols and phenoxy herbicides. These compounds are no longer produced in Canada.

At the elevated temperatures used in the manufacture of chlorophenols, various toxic by-products (impurities) may be formed. Impurities in chlorophenols include chlorodibenzo-p-dioxins and chlorinated diphenyl ethers, dibenzofurans, phenoxyphenols and dihydroxy biphenyls (see Contaminant Profile for Dioxins and Furans). Some of the health effects noted with chlorophenols are in fact associated with exposure to these impurities rather than with exposure to the chlorophenols themselves.

Persistence and Movement in the Environment

Chlorophenols do not occur naturally and are released into the environment from either intentional disposal or accidental release, or as a result of historical widespread uses. Because of their wide historical application, chlorophenols have been detected throughout the environment. Chlorophenols are generally broken down in air, land and water through physicochemical, photochemical, and microbiological action.

Based on figures from the early 1990s, the most important source of chlorophenol release (over 70 percent) is from treated products. Of this amount, roughly two thirds comes from industrial sources (leachate from treated products, leaked drilling fluids, and spillage from in-service wood treatment). These chlorophenols would be in the form of PCP, T_4CP, or their sodium salts. The other major chlorophenol releases (22 percent) occur from industries that use chlorophenols, as emissions from the incineration of wood waste at sawmills, leachate from landfills, liquid wastes from pulp mills, or discharge/spills of untreated wastes at wood preservation sites. These would be in the form of sodium tetrachlorophenol (NaT_4CP) and sodium pentachlorophenol (NaPCP). About 5 percent of releases occur at chlorophenol manufacturing sites, in waters containing chlorophenols from incomplete reactions or recoveries, as by-products, or as a result of accidents.

Municipal chlorination of drinking water and chlorination in industrial processes can also produce, as by-products mono-, di- and trichlorophenols in the parts per billion range.

Exposure

People can be exposed to low levels of tetra- and pentachlorophenols in indoor and outdoor air, food, and drinking water.

After consuming contaminated food or water, chlorophenols can enter the lungs and the digestive tract. Therefore ingestion is the most significant pathway through which chlorophenols enter the body. The primary pathway for excretion of chlorophenols is in the urine. Small amounts of chlorophenols tend to bioaccumulate in the body tissues, particularly the liver, kidney, blood and body fat.

Human Health Considerations

The overall health hazard associated with non-occupational exposure to chlorophenols is extremely low. A conservative estimate of daily intake for the Canadian adult population from all sources is 0.18 µg/kg•bw/day for a 70 kg adult. This level is more than 10 000 times less than the no observed effect level (NOEL) reported in laboratory animal studies and is not thought to pose a significant hazard to human health. It is important to note that most commercial chlorophenol products contain impurities as well as products of degradation and condensation. While present in very small quantities, some of these compounds are extremely toxic.

Because chlorophenols consist of several compounds with different biological activities, the effects on human health of these compounds are not the same. This discussion will focus on exposure to PCP as it is one of the most industrially important as well as one of the most biologically active chlorophenols.

Impurities in commercial PCP may cause many harmful effects. Direct contact with PCP can irritate the skin, eyes, and mouth, particularly when it is a hot vapour.

Table 1

HUMAN HEALTH EFFECTS FROM BREATHING PCP

	Short-term exposure (less than or equal to 14 days)	
Levels in air (ppm)	**Duration of exposure**	**Description of effects***
0.09	unknown	Irritation to eyes and nose
14	unknown	Immediately dangerous to life and health
	Long-term exposure (greater than 14 days)	
Levels in air (ppm)	**Duration of exposure**	**Description of effects***
		Effects of long-term exposure of humans to air containing PCP are unknown

* These effects are listed at the lowest level at which they were first observed. They may also be seen at higher levels.

Adapted from: U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR). *Public Health Statements – What you need to know about toxic substances commonly found at Superfund hazardous waste sites.* Atlanta, Georgia.

Table 2

HUMAN HEALTH EFFECTS FROM EATING OR DRINKING PCP

	Short-term exposure (less than or equal to 14 days)	
Levels in food (ppm)	**Duration of exposure**	**Description of effects***
0.1		Estimated minimal risk level (based on animal studies)
Levels in water (ppm)		
1.6	unknown	Odour threshold
	Long-term exposure (greater than 14 days)	
Levels in food (ppm)	**Duration of exposure**	**Description of effects***
0.09		Estimated minimal risk level (based on animal studies)
Levels in water (ppm)		
		The health effects of long-term exposure of humans by drinking water containing PCP are not known

* These effects are listed at the lowest level at which they were first observed. They may also be seen at higher levels.

Adapted from: U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR). *Public Health Statements – What you need to know about toxic substances commonly found at Superfund hazardous waste sites.* Atlanta, Georgia.

Table 3

GUIDELINES FOR CHLOROPHENOLS

Agency	Focus	Level
Health Canada	Drinking water	2,4-dichlorophenol: 900 µg/L (MAC) ≤ 0.3 µg/L (AO) 2,4,6-trichlorophenol: 5 µg/L (MAC) ≤ 2 µg/L (AO) 2,3,4,6-tretrachlorophenol: 100 µg/L (MAC) ≤ 1 µg/L (AO) pentachlorophenol: 60 µg/L (MAC) ≤ 30 µg/L (AO)
WHO	Drinking water	2-chlorophenol: no health-based guideline has been derived 2,4-dichlorophenol: no health-based guideline has been derived 2,4,6-trichlorophenol: 2 µg/L (this is the lowest reported taste threshold; if water containing this chlorophenol is free from taste, it is unlikely to present an undue health risk)

MAC: Maximum acceptable concentration
AO: Aesthetic objective
WHO: World Health Organization

Studies of workers exposed to high levels of chlorophenols and from misuse of products have demonstrated that short term (lasting 14 days or less) exposure to high levels can cause harmful effects in the liver, kidneys, skin, blood, lungs, nervous system, gastro-intestinal tract and can cause death. Similar effects have been observed in laboratory animals exposed for short periods to high levels of pentachlorophenol.

Long-term (lasting one year or longer) exposure to low-levels (such as those exposures which occur in the workplace) can cause damage to the liver, kidney, blood and nervous system. The major organs and systems affected by long-term exposure to low-levels of chlorophenols in animals include the liver, kidneys, nervous system, and the immune system. The effects are worsened when the level of exposure increases.

It is not known whether pentachlorophenol causes birth defects in humans. Animal studies have found a decrease in the number of offspring born to animals that were exposed to PCP while pregnant. Harmful effects on the developing fetus have been seen in animals exposed to PCP at exposure levels high enough to cause noticeable sickness in the mothers.

Exposure to large amounts of PCP have also been associated with increased cancer risk in laboratory animals. The International Agency for Research on Cancer has classified PCP as possibly carcinogenic to humans.

Table 4 shows the estimated PCP intake for the Canadian general population by age class via air, drinking water, soil/household dust, and foods.

Table 4

ESTIMATED PCP INTAKE (NG/KG•BW/DAY)

Environmental Media	Infants	Toddlers	Children	Adults	Amortized over lifetime
Air	11.1	12.8	14.3	9.5	10.8
Drinking Water	0.1	0.2	0.1	0.1	0.1
Soil/Dust	1.6	2.9	0.2	0.1	0.3
Food	99.3	105.3	49.9	27.7	38.0
Total PCP Intake	**112.1**	**121.2**	**64.5**	**37.4**	**49.2**

Source: S. Coad and Newhook, R.C. 1992. PCP Exposure for the Canadian General Population: A Multimedia Analysis. Journal of Exposure Analysis and Environmental Epidemiology. 2(4): 391–413.

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Clostridium botulinum

Origin and Uses

Clostridium botulinum produce exotoxins. Strains of *C. botulinum* are separated into seven types, A through G, based on the serological specificity of the neurotoxin produced. Human botulism, including food borne, wound and infant botulism, is associated with types A, B, E, and, very rarely, F. Types C and D cause botulism in animals.

C. botulinum are usually present in the environment as spores, rather than vegetative cells. The spores are commonly present in soils and sediments worldwide, but their numbers and types vary, depending on the location.

Persistence and Movement in the Environment

C. botulinum spores are extremely persistent in the environment; they are known to remain viable for decades. Spores can be transported by wind, rain, dust, and insects. Any soil crop is likely to be contaminated, as are fish from contaminated waters.

Exposure

Food-borne botulism is caused by the ingestion of foods containing preformed neurotoxins. Infant botulism occurs in infants who ingest spores that then germinate and produce toxin in the intestine. Wound botulism is caused by contamination of a wound with spores that germinate and produce toxin *in situ* (i.e., at the site of the wound).

Most foods spoil before becoming toxic due to *C. botulinum*. However, foods that undergo processes such as canning, fermentation and smoking to extend their shelf life may become hazardous. In Canada, most botulism outbreaks have occurred in northern Aboriginal communities. The foods involved were mainly native dishes: raw and parboiled meats from sea mammals, fermented meats such as muktuk (meat, blubber and skin of the beluga or white whale), and fermented salmon eggs. In the United States, most of implicated foods are home-preserved vegetables.

The environment appears to be the most common source of spores causing infant botulism. Honey has been implicated in a few cases, and is the only food that has been associated with infant botulism. It is recommended by Health Canada that honey not be fed to infants less than one year of age.

A properly filtered and disinfected water supply should be free of *C. botulinum*.

Reducing Exposure

Spores of *C. botulinum* are extremely resistant to heat. To be destroyed, spores must be exposed to 80°C for 10 minutes or boiled for 5–15 minutes. Commercial canning processes ensure that all spores are destroyed. When canning products at home, approved heat processes should be used; swollen or spoiled cans should not be used; and food from suspect cans should never be tasted.

For foods that do not undergo heat processing, proper hygienic practices are essential. Cleaning and eviscerating procedures of fish flesh will minimize the level of contamination. Great care should be taken in the chill storage of fish or fish products. Meat and fish curing brines should be controlled according to their acidity, salinity and temperature. The minimum temperature for growth of *C. botulinum* is 3.3°C.

Human Health Considerations

Botulism is the most severe type of food poisoning. The fatality rate due to food-borne botulism is approximately 15 percent. The fatality rate due to infant botulism is extremely low, <1 percent. Botulism is classified in four categories: classical food-borne botulism; wound botulism; infant botulism; and an undetermined classification of botulism for those cases involving individuals older than 12 months in which no food or wound source is implicated.

For food-borne intoxication of *C. botulinum*, the incubation period is usually 12 to 36 hours (ranges from two hours to eight days). Early symptoms include nausea, constipation, dizziness and severe dryness of the mouth and throat. This is followed by cranial nerve paralysis causing blurred vision and impaired speech. Later symptoms are difficulty in swallowing, progressive weakness, and respiratory failure that can lead to death by asphyxia.

With respect to infant botulism, the ingestion of spores by the infant during the first weeks of life is probably a prerequisite for the development of the disease. The first symptom is constipation, although it is usually mild and often overlooked. Patients who are ultimately hospitalized usually develop lethargy and mild weakness with feeding difficulties, pooled oral secretions and an altered cry. The baby eventually becomes floppy, loses head control, and may go on to develop other symptoms including cranial nerve deficits and generalized muscular weakness. Respiratory insufficiency necessitating therapy also may occur, as in other forms of botulism.

Guidelines

Since water is not a significant source of *C. botulinum*, no standards or guidelines have been proposed.

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Lennette, E.H., Balows, A., Hausler Jr., W.J., Shadomy, H.J. eds. *Manual of Clinical Microbiology*, 4th ed. Washington D.C.: American Society for Microbiology, 1985.

Clostridium perfringens

Origin and Uses

There are five types of *Clostridium perfringens* bacteria based on the production of toxins (A, B, C, D and E). The strains that are responsible for food poisoning belong primarily to type A and some type C. Spores of *C. perfringens* are widespread, and are found in freshwater and marine sediments, soil, dust, air, water, sewage, human and animal feces (usually at low numbers) and on many food products.

Persistence and Movement in the Environment

C. perfringens is a member of the normal intestinal microbial population but is usually not found on food. When ingested food contains high levels of vegetative cells, the cells multiply and produce spores in the intestine. During sporulation (the liberation of spores), enterotoxins are released. Spores of *C. perfringens* survive initial cooking; the spores then germinate, and vegetative cells proliferate during slow cooling or reheating.

Exposure

In food-borne disease, the food vehicle is almost always improperly cooked meat (e.g., beef, turkey, chicken or pork) or meat product (e.g., gravy) in which inadequate heat was used to destroy the *C. perfringens*. The food may have been cooled slowly after cooking or reheated to a moderate temperature.

Reducing Exposure

Preventing outbreaks of *C. perfringens* involves stopping the growth of the bacteria or the germination (production) of spores. This can be done by cooking foods and serving them soon after. Food should be held at temperatures of 60°C (80°C for roast joints) or refrigerated in small quantities to speed cooling.

Human Health Considerations

Food-borne intoxication from *C. perfringens* has an incubation period of 12 to18 hours (ranges from 8 to 22 hours). The health effects are diarrhea, abdominal pain and nausea. Vomiting rarely occurs. The illness is usually mild (most patients recover within two to three days after onset), but can be severe in the elderly, ill or debilitated people.

C. perfringens also produces a disease of the small bowel known as enteritis necroticans, which can occur sporadically or in an epidemic form. The syndrome, associated with the consumption of uncooked pork at pig feasts, has been called Darmbrand in Germany and pig-bel in Papua, New Guinea. In this condition, seen mostly in children, there is evidence that *C. perfringens*, which is either part of the normal intestinal microbial population or is ingested with contaminated meat, proliferates in the small intestine and produces toxin. This toxin production probably leads to focal paralysis, inflammation, hemorrhage, and tissue death of the intestine. Likely causal factors are overeating; poor nutrition; a diet rich in trypsin inhibitors (e.g., semi-cooked sweet potatoes often eaten at pig feasts).

C. perfringens is encountered in a wide variety of clinical settings, ranging from simple contamination of a wound to traumatic or nontraumatic myonecrosis, gangrenous inflammation of the gallbladder, post-abortion infection with blood poisoning, and intravascular hemolysis (release of the hemoglobin), necrotizing pneumonia, and empyema (accumulation of pus in a body cavity).

Although exogenous infections (including food-borne illness, gas gangrene associated with traumatic wounds, and tetanus) are well known, endogenous infections are much more common. Special circumstances are required for the development of infection with the clostridia. Common predisposing factors include trauma, operative procedures, vascular stasis, obstruction, treatment with immunosuppressive agents, chemotherapeutic agents as the treatment of malignancy, prior treatment with anti-microbial agents, and underlying illness such as leukemia, carcinoma, or diabetes mellitus. Since clostridia are naturally present in the human intestinal microbial population, their presence does not necessarily imply association with an illness.

Guidelines

For *C. perfringens*, current guidelines set a maximum acceptable concentration in dry mixes at 10 colony forming units/gram.

References

Jackson, M.H., Morris, G.P., Smith, P.G. and Crawford J.F. *Environmental Health Reference Book*. London: Butterworths, 1989.

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Cryptosporidium parvum and *Giardia lamblia*

Origins and Uses

Cryptosporidium parvum and *Giardia lamblia* are microscopic parasites that infect the intestinal tract causing gastro-intestinal illness. Cattle appear to be the primary source of *Cryptosporidium*, although they have also been found in humans and other animals. The primary sources of *Giardia* are the feces from humans, beavers, muskrats and dogs.

Persistence and Movement in the Environment

Both parasites produce cysts/oocysts at certain stages in their life cycles that are very resistant to harsh environmental conditions. *Giardia* produce cysts and *Cryptosporidium* produce oocysts. The cysts/oocysts persist in the environment for lengthy periods of time. Once ingested, the cysts/oocysts germinate, reproduce and cause illness if present in sufficient quantities. After feeding, the parasites form new cysts/oocysts, which are then passed in the feces.

Exposure

The most common source of exposure to both *Giardia* and *Cryptosporidium* is the consumption of water containing cysts/oocysts. They can be found in untreated, or inadequately treated, water supplies. Drinking water sources can be contaminated when feces containing the parasites are deposited or flushed into water. *Giardia* can also be spread by food or person-to-person contact. Day-care centres are prime sites for *Giardia* outbreaks where the cysts are spread among children and staff. Infected individuals can then spread the parasites to their parents and siblings. Other sources of exposure to both parasites include direct exposure to feces of infected humans and animals, eating contaminated food and accidental ingestion of contaminated recreational water.

Low levels of both parasites, especially *Giardia*, were detected in a national survey of drinking water conducted by Health Canada. Only a small fraction of the parasites appeared to be viable. However, outbreaks linked to drinking water have been reported in several provinces (see Chapter 3. "Contaminants"). There have also been reports of *Giardia* and *Cryptosporidium* being spread in swimming pools.

Reducing Exposure

Treatment of municipal drinking water, including filtration and disinfection, can reduce the risk of giardiasis and cryptosporidiosis. The parasites can also be killed by boiling the contaminated water. It is also beneficial to protect the raw water supply. Improper treatment may result in water that contains sufficient numbers of parasites to cause illness.

When using water supplies that have not been treated or which may have been inadequately treated (e.g., when camping, or when travelling in countries where the water supply is suspect), water should be boiled for at least one minute before being used for drinking, food preparation, or dental hygiene. This will destroy *Giardia* and *Cryptosporidium* as well as other disease-causing micro-organisms that may be present. Water filters with an absolute pore size of 1 µm can also remove parasites. It should be noted that bottled water may not be a suitable alternative to boiled tap water as bottled water is not routinely monitored for *Giardia* and *Cryptosporidium*.

People with compromised immune systems, such as those with AIDS, cancer, or transplant patients receiving immunosuppressive drugs, may consider taking extra precautions to ensure the safety of the water used for drinking, food preparation, or dental hygiene. Anti-parasitic drugs are available to fight *Giardia* and are particularly helpful for people with compromised immune systems in whom the illness could otherwise develop into a persistent state. There are currently no approved drugs to fight cryptosporidiosis although some are now being tested.

Human Health Considerations

Giardia causes an intestinal illness called giardiasis, commonly referred to as "beaver fever." The symptoms include diarrhea, abdominal cramps, gas, malaise and weight loss. Vomiting, chills, headache and fever may also occur. The onset of symptoms is generally within six to 16 days of the initial contact and can continue for up to a month. *Cryptosporidium* causes a similar illness called cryptosporidiosis characterized by watery diarrhea, abdominal cramps, nausea and headaches. Symptoms occur within two to 25 days of infection and can last from one to two weeks or as long as a month. Studies of human volunteers have shown that ingestion of only a few cysts will cause illness.

Both parasites, *Cryptosporidium* in particular, pose a more serious threat for people with compromised immune systems. The symptoms may be more severe in these individuals and can be life threatening.

Guideline

Health Canada is developing a guideline for *Giardia* and *Cryptosporidium* as part of the *Guidelines for Canadian Drinking Water Quality*.

References

Canada. Health Canada. *Guidelines for Canadian Drinking Water Quality — Sixth Edition*. Prepared by the Federal-Provincial Subcommittee on Drinking Water of the Federal-Provincial Committee on Environmental and Occupational Health. Ottawa: Canada Communication Group, 1996.

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DDT and related compounds: DDE, TDE, DDA, methoxychlor

Origin and Uses

DDT (dichlorodiphenyltrichloroethane) is a chemical of anthropogenic origin first used in 1939 for the control of insects capable of transmitting malaria, typhus, and other insect vectors of diseases during World War II. Commercial production of DDT for agricultural use and vector control began in 1945, its use peaking in 1963. In Canada, the use of DDT was restricted in 1974 and stopped in 1989. In the United States, DDT can no longer be used except in cases of public health emergency. There are still major producers of DDT in India and some Central and South American countries, where DDT is used in malaria control. The World Health Organization (WHO) has concluded that DDT should not be used if an alternative insecticide is available.

DDE is the primary metabolic product of DDT, with TDE (also called DDD) and DDA being less prominent metabolites. TDE was used as a pesticide, and one form (o,p'-DDD) has been used medically to treat cancer of the adrenal gland. Use of TDE as a pesticide has been banned. DDE is an impurity in and a breakdown product of DDT, but it is not commercially produced or used. Methoxychlor, a compound closely related to DDT, was used as a pesticide.

Persistence and Movement in the Environment

DDT, DDE, and TDE are distributed worldwide. Studies show prolonged persistence in soils (particularly DDE) indicating that they are not easily displaced from their site of application. However, they can be transported in air, ocean currents, and in biota. In soil DDT can be taken up by worms and by plants, and in water by aquatic organisms (shellfish and fish). Both DDT and DDE bioaccumulate in organisms and biomagnify in the food chain. In humans, serum DDT concentrations correlate strongly with age. DDE is more fat soluble and less toxic than DDT and is not easily biodegraded. As a result, it is mostly DDE and not DDT that bioaccumulates in the fat tissue of animals and humans. Measurable levels of DDE persist in human tissues because of its long half-life (approximately six to eight years). Methoxychlor has a lower acute toxicity and a shorter biological half-life than does DDT.

DDT levels in water, biota, and human serum and breast milk have dropped considerably in the years following its restriction. Recently, however, these concentrations have levelled off. This may be due to atmospheric transfer from countries still using DDT.

Exposure

Food is the primary route of exposure. Foods that may contain DDT include: meat, fish, and poultry animal fats; dairy products; and root and leafy vegetables. Dietary intake of total DDT by the general population in Canada is estimated at 0.02–0.04 µg/kg•bw/day. Recent analysis of human fat tissue from Great Lakes municipalities showed average concentrations of approximately 3 mg/kg, similar to residents in other parts of Canada. Current levels in human breast milk are low and appear to have reached a baseline.

Municipal drinking water and air are not likely routes of exposure. Intake through inhalation is negligible in comparison with dietary sources. DDT, DDE and DDD do not enter through the skin very easily, thus dermal exposure is not a significant route of entry.

Once inside the body, DDT is broken down into its metabolites, which are stored in fatty tissue or are excreted (primarily in urine, but also in breast milk, and feces). Stored amounts leave the body very slowly.

Reducing Exposure to DDT and its metabolites

The Guide to Eating Ontario Sport Fish should be consulted and advisories should be followed when consuming sport fish.

Human Health Considerations

Target organs for DDT, DDE and DDD toxicity include the nervous system, the reproductive system, and the liver.

Acute exposure: Dermal or respiratory overexposure to DDT have caused irritation of the nose, throat and eyes. Ingestion of >16–20 mg/kg•bw produces hyperesthesia of the mouth and lower part of the face, paresthesia, dizziness, confusion, headache, fatigue and delayed vomiting. The onset of DDT poisoning is characterized by mild effects that progress only gradually but continuously and lead to convulsions only in severe poisonings. People who ingested high doses of DDT, 571 mg/kg (ppm) in food, experienced excitability, tremors, and seizures. These effects ceased after exposure was discontinued.

Table 1

HUMAN HEALTH EFFECTS FROM EATING OR DRINKING DDT, DDE OR DDD

	Short-term exposure (less than or equal to 14 days)	
Levels in food (ppm)	**Duration of exposure**	**Description of effects**
214	Single dose	Headache, nausea
357	Single dose	Headache, nausea, vomiting
560	Single dose	Heart, increased rate
571	Single dose	Vomiting, convulsions
0.0178	Single dose	Minimal risk level
Levels in water (ppm)		
		The health effects resulting from short-term human exposure to water containing specific levels of DDT, DDE or DDD are not known
	Long-term exposure (greater than 14 days)	
Levels in food (ppm)	**Duration of exposure**	**Description of effects**
22	18 months	No effects
0.0125	60 days	Minimal risk level
Levels in water (ppm)		
		The health effects resulting from long-term human exposure to water containing specific levels of DDT, DDE or DDD are not known

Adapted from: U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR). *Public Health Statements – What you need to know about toxic substances commonly found at Superfund hazardous waste sites.* Atlanta, Georgia.

Occupational exposure: Long-term occupational exposure has resulted in depression of visiomotor functions, elevation of liver enzymes, depression of serum bilirubin.

Environmental exposure: Studies involving only low-level environmental exposure document higher organochlorine blood levels (DDT, PCBs, lindane, dieldrin) in women undergoing spontaneous abortions or premature delivery compared with those mothers having full-term births. Because these studies involve elevated levels of several organochlorines, it is not possible to implicate DDT alone.

Recent data indicate that current blood and adipose tissue concentrations of DDT are well below levels associated with clinical manifestations. Suppression of reflexes in neonates appear to be associated with levels of DDE in breast milk exceeding 4 mg/kg (fat basis), however, it has not been substantiated if DDE is the causative factor. Elevated levels of DDE in human breast milk (3.0 mg/kg, fat basis) have been correlated with

a shortening of breast-feeding duration; inhibition of lactation was hypothesized. Blood concentrations have shown a dependence on alcohol consumption and serum triglyceride levels, while duration of breast-feeding and parity influence milk concentrations.

DDD used in the treatment of adrenal hyperplasia (40 or 100 mg/kg/day for several weeks) often produces general lassitude, anorexia, nausea, vomiting, diarrhea and/or dermatitis. The symptoms disappear soon after administration of the drug is stopped or the dosage is reduced.

An association between exposure to DDT and the development of cancer has not been found in studies of occupational exposure to DDT. Based on animal studies (oral exposure), the U.S. EPA has concluded that DDT, DDE and DDD are probable human carcinogens.

Table 2

MAC, MRL AND ADI VALUES FOR DDT AND ITS METABOLITES

Agency	Focus	Level	Comments
Health Canada	Drinking water	0.03 mg/L (DDT and metabolites) 0.9 mg/L (for methoxychlor)	MAC MAC
Health Canada	Fish flesh	5.0 µg/g (ppm) (DDT)	MRL
Health Canada	Dairy products, meat and meat by-products and fat of cattle, hogs, poultry and sheep	1.0 µg/g (DDT) (calculated on fat content)	MRL
Health Canada	Eggs, vegetables	0.5 µg/g (ppm) (DDT)	MRL
OMOEE	Fish flesh	> 5.0 µg/g (ppm) (DDT and metabolites) Restricted Consumption and no consumption for women of child-bearing age and children (<15 years)	
OMOEE	Drinking water	0.03 mg/L (DDT and metabolites) 0.9 mg/L (methoxychlor)	MAC MAC
WHO	All sources	20 µg/kg•bw/day (p,p'-DDE)	ADI
WHO	All sources	20 µg/kg•bw/day (p,p'-DDD)	ADI
WHO	All sources	20 µg/kg•bw/day (p,p'-DDT)	ADI
WHO	Drinking water	2 µg/L (DDT and metabolites) 20 µg/L (methoxychlor)	guideline value guideline value

MAC: Maximum acceptable concentration
ADI: Acceptable daily intake
MRL: Maximum residue limit
OMOEE: Ontario Ministry of Environment and Energy
WHO: World Health Organization

WHO Human Blood Guideline[1]
WHO human blood guideline = 200 µg/L (total DDT)

1. Source: WHO Technical Report Series 667. Recommended health-based limits in occupational exposure to pesticides. Geneva: World Health Organization. 1982. Cited in: *Human Toxicology of Pesticides* by F.P. Kaloyanova and M.A. ElBatawi. CRC Press Inc. Florida 1991.

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Dioxins (polychlorinated dibenzodioxins) and Furans (polychlorinated dibenzofurans)

Origin and Uses

Dioxins and furans are two chemical families closely related by their structural formulae. There are 75 dioxins and 135 furans. The most toxic, and the most studied of these chemicals is 2,3,7,8-tetrachloro-dibenzodioxin (2,3,7,8-TCDD or TCDD).

Although produced through natural sources (volcanoes, forest fires), the majority of dioxins are by-products of certain chemicals (e.g., pentachlorophenol used in wood preservatives, the herbicide 2,4-D) or chemical processes (e.g., chlorine bleaching used in pulp and paper mills) or combustions (e.g., incineration). Other sources of small amounts of dioxins and furans are wood burning stoves and fireplaces, and barbecuing; cigarette smoke also contains dioxins. Furans are a trace contaminant of PCBs. Although the use and storage of these compounds is now strictly controlled, fires and spills involving PCBs remain a source of furan contamination.

In Canada, the sale and use of pesticides containing TCDD is not permitted and the content of other dioxins in pesticides has been regulated. The Government of Canada, in conjunction with the provinces, has established codes of practice to reduce contamination by the wood preservation and protection industries.

Persistence and Movement in the Environment

Dioxins and furans are highly persistent compounds. They can be transported in the atmosphere over long distances, thus traces can be found throughout the environment. Dioxins and furans are fat soluble and bioaccumulate in most animals, and are present in the natural and human food chains. Bioaccumulation occurs mostly in the fat and fatty organs. Small amounts may also be found in skin, muscle and other organs.

Exposure

Researchers have developed a method for expressing the toxicity of different dioxins and furans on a common basis. International Toxicity Equivalency Factors (TEQs) are assigned to individual dioxins and furans on the basis of how toxic they are in comparison with the toxicity of 2,3,7,8-tetrachloro-dibenzodioxin. This contaminant has been assigned the value of 1.0. For example, 2,3,7,8-TCDF is one tenth as toxic as 2,3,7,8-TCDD, and has a toxic equivalent of 0.1.

All Canadians have detectable concentrations in their body fat. The average daily Canadian intake of dioxins and furans over a lifetime is estimated to be 2.0–4.2 pg of TCDD toxic equivalents/kg•bw/day. Smoking has not been included in the above estimate of general population exposure. Nonetheless, smoking may contribute significantly to dioxin and furan exposure (see calculation below). Virtually all intake (94 to 96 percent) in non-smoking adults comes from food, especially fish, meat and dairy products. The remaining exposure comes from air and other routes (soil, water, consumer products). Dioxin concentrations in water and on vegetation are generally below present detection limits. The amount of dioxins detected in paper-based products (disposable diapers, tissue) is considered insignificant.

In general, absorption is greater when dioxins and furans are ingested or inhaled than through dermal exposure. Persons eating highly contaminated fish in quantities well in excess of the general population may approach or exceed the guideline for intake. Elevated levels in some fish and shellfish near pulp and paper mills that use chlorine bleaching have necessitated closure of the affected fisheries in order to protect human health.

Although dioxins and furans have been found in breast milk, current levels are not considered to pose a risk to infants. Studies suggest tobacco smoking as a pathway of dioxin exposure. The potential exposure of a person who smokes 20 cigarettes/day is 0.5 pg TCDD toxic equivalents/kg•bw/day. This is based on the assumption that a 70 kg person inhales 1 litre of smoke per cigarette containing dioxins at the concentration of 1.8 ng toxic equivalents/m^3, and that inhalation is 100 percent (Environment Canada and Health Canada 1990). It should be noted that this estimate is preliminary in that it does not take several factors into account (see Environment Canada and Health Canada 1990).

Dioxins and furans are eliminated predominantly through the feces. Elimination in breast milk is also substantial, with amounts being similar to or higher than those in feces.

High levels of exposure occur in occupational settings through industrial processes (mainly pesticide production) and hazardous waste accidents (PCB transformer fire clean-ups; Seveso, Italy; Vietnam; Yusho, Japan; and Yu-Cheng, Taiwan).

Reducing Exposure

Fish and seafood consumed should be within the Canadian contaminant guidelines for dioxins and dibenzofurans.

Human Health Considerations

Dioxins and furans are believed to produce similar health effects. The most consistent overt effect of exposure (through inhalation, ingestion, or dermal contact) in humans is chloracne. Other effects that have been observed include fluctuations in serum levels of liver enzymes; pulmonary deficiency; sensory changes such as numbness, nausea, headaches, loss of hearing, sleep disturbances, tiredness; sexual dysfunction (decreased libido); depression; and loss of appetite. Soft-tissue sarcomas, lymphomas, peripheral neuropathy, birth defects, and reproductive effects have been reported in humans exposed to high levels of dioxins and furans, but are not confirmed. Other dermal effects include hyperpigmentation, hirsutism, increased skin fragility, and vesicular eruptions on exposed areas of the skin; these effects may be due to exposure to several chemicals, and not only to dioxins and furans.

Animal studies indicate that TCDD acts as a cancer promoter, but not as an initiator. Induction of cancer is by a non-genetic mechanism. A conclusive link between cancer in humans and dioxins and furans has not been established. The International Agency for Research on Cancer (IARC) considers dioxins and furans to be possibly carcinogenic to humans.

Long-term exposure to dioxins and furans has not been conclusively shown to affect the health of fish, wildlife or domestic animals. Long-term exposure of test animals can affect reproduction, cause birth defects, damage the liver and suppress the immune system. There may be a link between exposure to dioxins and furans and the loss of reproductive capability in fish-eating birds in the Great Lakes basin.

Table 1

TDI, TOLERABLE EXPOSURE AND IMAC VALUES FOR TCDD

Agency	Focus	Level	Comments
Health Canada	Fish/seafood	15 ng/kg (ppt) TCDD toxic equivalents	[1]
Health Canada/WHO	All sources	10 pg/kg bw/day TCDD equivalents	TDI
Canada & Ontario	All sources	10 pg TCDD toxic equivalents/kg•bw/day	tolerable exposure
OMOEE	Drinking Water	15 ppq (pg/L) TEQ[2]	IMAC
OMOEE	Air	30 ppt TCDD	

TDI: Tolerable daily Intake
IMAC: Interim maximum acceptable concentration
WHO: World Health Organization
OMOEE: Ontario Ministry of Environment and Energy

1. calculated by applying TEQs to all 17 2,3,7,8-chlorine substituted PCDD/PCDF congeners
2. total toxic equivalents when compared with 2,3,7,8-TCDD

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World Health Organization, International Programme on Chemical Safety (IPCS), *Environmental Health Criteria 88: Polychlorinated dibenzo-para-dioxins and dibenzo furans*. Geneva, 1989.

Escherichia coli (E. coli)

Origin

Enterobacteriaceae is a family of bacteria widely distributed on plants, in soil and in the intestines of humans and animals. Although they are a major component of the organisms found in the human intestine, they are relatively uncommon in other body sites. *E. coli* is a species of the *Enterobacteriaceae* family. It is normally a harmless inhabitant of the intestine of warm-blooded mammals (including humans), however, certain strains are pathogenic to humans and animals. Its presence in soil and water, as a result of human and animal fecal contamination, is used as a measure of fecal pollution.

Persistence and Movement in the Environment

E. coli is routinely used as an index of the presence of enteric pathogenic bacteria. It is unable to grow in temperate aquatic environments but survives as long or longer than most enteric pathogenic bacteria.

Exposure

Exposure to humans occurs through the ingestion of contaminated drinking water or food. Human or animal wastes and raw foods of animal origin are the main sources of contamination. Properly treated drinking water should be free of *E. coli* and other bacterial pathogens. Cooked foods are readily infected through contact with contaminated hands, surfaces or equipment. Outbreaks have been associated with the consumption of under-cooked ground beef, unpasteurized dairy products, apple cider and salads containing raw vegetables. Infants in nurseries or day-care centres are particularly at risk of an *E. coli* outbreak from fecal-oral transmission directly or through contaminated food.

Reducing Exposure

Preventing outbreaks of *E. coli* involves avoiding the contamination and growth of the organisms in food and water. The following steps can reduce exposure:

- Prevent cross-contamination by washing hands and all equipment before and after handling food, especially raw meat and poultry; keep such foods separate from cooked foods.
- Avoid using contaminated water to wash fruit and vegetables.
- Ensure that food is properly cooked, and is stored at temperatures < 4 °C or > 60 °C.

Human Health Considerations

For food-borne infections the incubation period is usually 12 to 24 hours (range of 10 to 72 hours). The effects are abdominal pain, fever, diarrhea and sometimes vomiting. Severity of the symptoms will vary depending on the type and number of *E. coli* cells ingested, and the host's age and physical condition. Children under one year of age, the elderly and those who are immuno-compromised are particularly at risk.

E. coli is associated with at least four types of human enteric (intestinal) diseases: enteropathogenic (produces a disease in the intestinal tract), enterotoxigenic (toxin produced by bacteria living in the intestine), enteroinvasive, and hemorrhagic.

- Enteropathogenic *E. coli* strains cause diarrhea, mostly in infants.
- Enterotoxigenic *E. coli* strains typically cause profuse, watery diarrhea, and they are often implicated in cases of traveller's diarrhea.
- Enteroinvasive strains of *E. coli* cause a dysentery-like illness, characterized by severe cramping abdominal pain and diarrhea with blood and mucus.
- Hemorrhagic colitis is a more recently recognized enteric infection due to specific *E. coli* strains. These strains cause a severe diarrhea, characterized by grossly bloody stools. This disease may progress to haemolytic uraemic syndrome.

Guidelines and other values for *E. coli*

The Guidelines for Canadian Recreational Water Quality set a maximum limit of 2000 *E. coli*/L, as the geometric mean of at least five samples taken during a period not to exceed 30 days. The Health Canada MAC (maximum acceptable concentration) for coliforms in drinking water is zero organisms detectable per 100 mL. Because coliforms are not uniformly distributed in water and are subject to considerable variation in enumeration, drinking water that fulfils the following conditions is considered to be in compliance with the coliform maximum acceptable concentration (MAC):

1. No sample should contain more than 10 total coliform organisms per 100 mL, none of which should be fecal coliforms (as indicated by *E. coli*).
2. No consecutive sample from the same site should show the presence of coliform organisms.
3. For community drinking water distribution systems:
   a. no more than one sample from a set of samples taken from the community on a given day should show the presence of coliform organisms; and
   b. not more than 10 percent of the samples based on a minimum of 10 samples should show the presence of coliform organisms.

Current regulations prohibit the presence of *E. coli* in fish and protein meal, and set the maximum acceptable concentration in cheese at 100 *E. coli* per gram from pasturized milk and 500 *E. coli* per gram from unpasteurized milk. Current guidelines set the maximum acceptable concentrations of *E. coli* per gram in pasta at 500, frozen cream pies at 100, heat treated fermented sausage at 50, raw fermented sausage at 250, deboned poultry at 100, dry mixes at 100, and soybean products at 100.

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Fluoride

Origin and Uses

Fluoride is a natural chemical that forms salts known as fluorides. Certain fluorides occur naturally in rock and soil and are released into the environment by the erosion of minerals and by volcanic activity. Canadian data on releases from natural sources is not available, however, estimates of global releases of hydrogen fluoride from volcanoes range from 60–6000 kilotonnes/year and the estimated annual release from marine aerosols is 20 kilotonnes. Approximately 23 500 tonnes of fluoride are released into the Canadian environment each year from human sources not including the fluoride added to drinking water. Hydrogen fluoride, calcium fluoride, sodium fluoride, and sulphur hexafluoride are the compounds released to the Canadian environment in the largest amounts. Of the total inorganic fluorides reported to enter the Canadian environment from anthropogenic sources, approximately 23 percent is released to the air, 58 percent to water, and 19 percent to land.

In Canada, fluorides are used mainly in the production of phosphate fertilizers, chemical production and aluminum smelting. Other anthropogenic sources include glass, brick and ceramics manufacturing; the combustion of coal; pesticide manufacture; glue and adhesive production; and petroleum. The two most common therapeutic uses of inorganic fluoride are in the prevention of tooth decay and for the treatment of osteoporosis. It is estimated that 38 percent of the Canadian population is served by fluoridated public drinking water, as it significantly reduces tooth decay. Inorganic fluoride is also added to products such as toothpaste, tooth powders, mouthwash and vitamin supplements.

Persistence and Movement in the Environment

It has been concluded by Environment Canada and Health Canada (1993) that inorganic fluorides are entering the environment in quantities or under conditions that may be harmful to the environment. There is insufficient information to conclude whether sulphur hexafluoride is entering the environment in quantities or under conditions that may constitute a danger to the environment on which human life depends.

Gaseous inorganic fluorides are primarily released into the atmosphere, whereas particulate fluorides are released into the aquatic and terrestrial environments. Once released into the atmosphere, fluorides are carried by wind and rain to water, soil and food sources. Inorganic fluorides have been measured in ambient air, freshwater (including groundwater), seawater, aquatic sediments and soil throughout Canada, as a result of both natural and anthropogenic sources. Some aquatic and terrestrial species bioaccumulate inorganic fluorides. Uptake and absorption by these species appears to be greater from water than from food. Limited evidence indicates that biomagnification of inorganic fluoride does not occur in aquatic or terrestrial food chains.

Exposure

Drinking water, drinks and juices, toothpaste and food are the main sources of fluoride exposure for most Canadians. Fluorides can be found in most types of bottled water. All vegetation and virtually all foods contain at least trace amounts of fluorides. In natural water, the fluoride concentration varies widely, as it is dependent on the water's sources and surrounding geological formations. Surface waters usually contain fluoride at concentrations below 1 mg/L.

Healthy adults absorb about 90 percent of the fluorides they ingest via the gastro-intestinal tract. Approximately 50 to 75 percent of ingested fluoride is excreted in the urine within 24 hours of exposure (Spencer 1981 and 1970; Ekstrand 1977 as cited in Health Canada 1993). Fluoride is excreted from the body

primarily in the urine, with smaller amounts in breast milk, perspiration and feces. Compared with adults, children and adolescents have a lower rate of fluoride clearance (Spak 1985 cited in Health Canada 1993). This is partly due to the greater incorporation of absorbed fluoride into developing bone. Approximately 99 percent of the fluorides retained by the body are stored in the skeleton and the teeth.

In 1986, approximately 62 percent of the population of Canada received "non-fluoridated" drinking water and 38 percent was supplied with "fluoridated" drinking water. Non-fluoridated refers to drinking water to which inorganic fluoride has not been intentionally added for the prevention of tooth decay, while fluoridated water has been intentionally treated. Based on the results of studies conducted between 1984 and 1989, the mean concentration of fluoride in non-fluoridated drinking water supplies in Canada ranges from < 0.05–0.21 mg/L. The mean concentration of fluoride in fluoridated drinking water supplies in Canada ranges from 0.73–1.25 mg/L. Fluoride concentrations in bottled water obtained from across Canada ranged from < 0.05–5.85 mg/L (Dabeka 1992 cited in Health Canada 1993).

The following exposure data have been taken from Environment Canada and Health Canada (1993). The estimated total daily intake of inorganic fluoride by the general population of Canada through ingestion ranges from 32.8–160.4 µg/kg•bw/day. This estimate includes exposure from air, food, soil, household products (including dentifrice, see below) and fluoridated drinking water. A similar calculation using non-fluoridated drinking water ranges from 17.2–96.4 µg/kg•bw/day. The available data suggest that for individuals consuming non-fluoridated drinking water, the greatest source of exposure to inorganic fluoride occurs through the ingestion of food. For individuals consuming fluoridated drinking water, the greatest contribution to the total intake of inorganic fluoride comes from the water itself, as well as from food. The exception to these two previous statements concerns seven-month to four-year old children, for whom dentifrice can potentially be the greatest source of fluoride intake. Although it is generally recognized that water consumption is higher for people residing in very hot climates, available Canadian survey data show that drinking water consumption does not vary significantly from season to season. Exposure through air (0.01 µg/kg•bw/day) represents a minor contribution to the daily intake for the general Canadian population.

Dental products that contain fluoride have been identified as sources of inorganic fluoride for children and adolescents. In toothpaste, inorganic fluoride concentrations range from 1000–1500 µg/g (ppm); topical mouthwashes marketed for daily home use contain between 250 and 500 mg/L (ppm) inorganic fluoride, while mouthwash products intended for weekly or biweekly use contain up to 1000 mg/L (ppm) fluoride. The estimated intake of dentifrice is 0.26–0.78 g/day for children 7 months to 4 years, 0.22–0.54 g/day for children 5 to 11 years, 0.14 g/day for adolescents 12 to 19 years, and 0.08 g/day for adults 20+ years.

Some foods such as seafood (especially those types in which the bones are consumed), bone products (bone meal and gelatin), canned fish, meat, wine and tea contain high concentrations of fluoride. Foods cooked in water with added fluorides may have increased fluoride levels. For example, the total average daily intake of inorganic fluoride through ingestion by exclusively breast-fed infants was estimated to range from 0.5–2.6 µg/kg•bw/day, whereas the intake in exclusively bottle-fed infants was estimated to range from 13.6–93 µg/kg•bw/day. The estimated daily intake of inorganic fluoride by the general Canadian population from food ranges from 13.6–91.5 µg/kg•bw/day.

Populations exposed to relatively high concentrations of fluoride include workers in fluoride processing industries and people living near industrial sources (e.g., aluminum smelters and brick and phosphate plants). Fluoride intake from food may be elevated in populations residing near these sources. However, there is insufficient data available to estimate the intake of fluoride from this source. Consequently, their intake of fluoride (from food) is assumed to be similar to that for the general population.

Reducing Exposure

There are several things that can be done to reduce the occurrence of dental fluorosis. Fluoride mouthwashes or mouth rinses should never be given to children under six years of age as they may swallow the liquid after use. If a mouthwash contains fluoride, it will say so on the label. Children should not use more than a

pea-sized amount of toothpaste on their toothbrush and should not swallow toothpaste. Children under six years of age should be supervised while brushing. Fluoride supplements should not be used if the drinking water supply is already fluoridated or if the naturally occurring fluoride in the water supply is already at optimal levels.

Human Health Considerations

It has been concluded by Environment Canada and Health Canada (1993) that inorganic fluorides are not entering the environment in quantities or under conditions that may constitute a danger to human life or health.

Dental Health Effects

Numerous studies have demonstrated the association between the consumption of fluoridated drinking water and the reduction of dental cavities (U.S. DHHS 1991). In most fluoridated Canadian communities, the concentration of fluoride in drinking water is maintained at an optimum concentration of 1.0 mg/L to reduce tooth decay (Droste 1987). At optimum concentrations, fluoride helps to remineralize cavities when they first start forming and fluoride also increases the resistance of tooth enamel to acids that cause tooth decay (Thylstrup 1990; Beltran 1988; Groeneveld 1990). However, regular exposure to slightly elevated amounts of fluorides during the period of tooth formation from birth to approximately six years of age, may be associated with increased incidences of mild dental fluorosis. This is characterized by white areas, and occasionally brown stains, on the teeth (Health Canada 1995). The current maximum acceptable concentration in Canadian drinking water (1.5 mg/L) was developed to prevent excessive incidences of moderate to severe dental fluorosis (Health Canada 1995 and 1996). Although incidences of mild dental fluorosis may occur in communities with drinking water concentrations < 1.5 mg/L fluoride, there is no evidence of any adverse health effects associated with mild dental fluorosis (Health Canada 1995).

Other Health Effects

An increased incidence of skeletal fractures in some osteoporotic patients receiving sodium fluoride has been reported at doses equivalent to 260 µg/kg•bw/day or more. However, the incidences of hip fractures were low in these studies, they generally involved small numbers of elderly patients with osteoporosis, who were also receiving other dietary supplements (i.e., calcium, vitamin D_2) and individual exposures to fluoride (i.e., intakes from other sources) were not measured. Elevated rates of hip fracture have been observed in studies of areas with drinking water containing elevated levels of fluoride, or where the proportion of the population consuming fluoridated drinking water was greater than that consuming non-fluoridated drinking water. However, these observations should be interpreted with caution because of limitations of the epidemiological studies from which they were derived (Environment Canada and Health Canada 1993).

Long-term chronic exposure to high levels of fluoride (as might occur through occupational exposure) has been linked to skeletal fluorosis. The more severe forms of skeletal fluorosis can be associated with sporadic pain and stiffness of the joints, chronic joint pain, osteosclerosis, and calcification of ligaments. In cases where doses are known, they are generally 15–20 mg fluoride/day for over 20 years. The most severe form of the condition, crippling skeletal fluorosis, is characterized by complete rigidity of the spine, often accompanied by a curvature of the spine.

The increased susceptibility of the elderly, and people with cardiovascular disease and impaired renal function to the toxicity of fluoride may be due, at least in part, to decreased renal clearance. Dietary deficiencies in calcium and protein, and increased fluid intake related to strenuous manual labour may increase susceptibility to the toxic effects of fluoride (Health Canada 1993). However, these factors may be more relevant to underdeveloped areas of the world rather than North America.

The acute ingestion of large amounts of fluoride may cause stomach aches, nausea, vomiting, gastric pain, diarrhea, drowsiness, coma, convulsions, cardiac arrest and death. The cardiac and neurological effects observed in severe cases may be due to fluoride-induced hypocalcemia and/or hyperkalemia, as well as the inhibition of cellular enzymes (Health Canada 1993). There is no conclusive evidence that the consumption of water containing the recommended levels of fluoride can produce heart disease, thyroid gland dysfunction, hearing problems, vision impairment or result in an increased incidence of Down's Syndrome.

There is no conclusive evidence that fluoridated drinking water is carcinogenic to humans or to support claims of allergic reactions to fluoridated water. In virtually all of the epidemiologic studies reported to date, there has been no association between the consumption of fluoridated drinking water and increased incidence of, or mortality due to cancer (Environment Canada and Health Canada 1993). Although the design of these studies prevents them from providing conclusive proof for or against an association, several reviewers have noted that their consistency in not showing an association can impart a degree of confidence in their findings (U.S. DHHS 1991).

Table 1

MAC AND TOLERANCE VALUES FOR FLUORIDE

Agency	Focus	Level	Comments
Health Canada	Drinking water	1.5 mg/L (It is recommended that fluoride concentrations be adjusted to 1.0 mg/L, which is the optimum level for the control of dental caries. Where the annual mean daily max temp is < 10°C, 1.2 mg/L should be maintained.)	MAC. This guideline is being reviewed for possible revision owing to new data.
Health Canada	All sources	200 µg/kg•bw/day (inorganic fluoride)	TDI; based on skeletal effects; dental effects not considered.
Health Canada	Edible bone meal	650 µg/g (ppm)	tolerance
Health Canada	Fish protein (Food and Drugs Act)	150 µg/g (ppm)	tolerance
OMOEE	Drinking water	*	
WHO	Drinking water	1.5 mg/L (Climatic conditions, volume of water consumed and intake from other sources should be considered when setting national standards.)	guideline value

MAC: Maximum acceptable concentration
OMOEE: Ontario Ministry of Environment and Energy
TDI: Tolerable daily intake
WHO: World Health Organization

* Where fluoridation of drinking water is practised, it is recommended that the concentration be adjusted to 1.0 ± 0.2 mg/L, the optimum level for control of dental caries. Communities in Northern Ontario, where the annual mean daily max temperature is less than 10%C may wish to consider adjusting the fluoride concentration to 1.2 ± 0.2 mg/L. Adverse effects of fluoride in drinking water above 1.5 mg/L and below 2.4 mg/L are cosmetic in nature (dental mottling in a small proportion of the population). Levels above 1.5 mg/L should be reported to the local medical officer of health.

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Hexachlorobenzene (HCB)

Origin and Uses

Hexachlorobenzene (HCB) was manufactured primarily for use as a fungicide. It has not been used as a commercial chemical in Canada since 1972. Small quantities of HCB have been imported into Canada during the 1980s. Currently the principal sources of HCB in the Canadian environment are the by-products from the manufacture and use of industrial chemicals and pesticides; the emissions from incinerators containing HCB-containing wastes; and long-range transport from other countries.

Persistence and Movement in the Environment

HCB is ubiquitous in the Canadian environment, primarily because it is mobile and does not easily biodegrade. HCB breaks down slowly in air and water, producing chlorinated phenols and other chlorinated benzenes. It adsorbs strongly onto organic components of soil and sediment, allowing ingestion and subsequent bioaccumulation. Biomagnification of HCB occurs in the food chain. HCB tends to accumulate in fatty tissues of organisms.

Exposure

Food is the major exposure pathway (> 98 percent) for HCB. Foods with a high lipid content have a higher HCB concentration, primarily dairy products (milk, butter, ice cream) and to a lesser extent, fresh meat, eggs and peanuts/peanut butter. When eaten or ingested in fat or oil (through foods), a greater concentration of HCB is biologically available and is taken up by the body than when HCB is consumed in drinking water. This is because HCB is not very water soluble.

Reports of HCB levels in milk, blood and adipose tissue indicate that current exposure to HCB of the general Canadian population is low. Estimated intakes of HCB for the Canadian general adult population, in ng/kg•bw/day, are 0.05 in air, 0.002 in drinking water, 0.0002 in soil, and 2.7 in food. Mean intakes of HCB from all sources are estimated to range from 45 ng/kg•bw/day for breast-fed infants to 2.8 ng/kg•bw/day for adults. Assuming a 70 year lifespan, the daily intake of HCB for the general population averaged over a lifetime is estimated to be 6.2 ng/kg•bw/day.

Groups likely to be exposed to higher HCB levels include anglers consuming salmonids from Lake Ontario and people living in the Arctic who consume large quantities of wildlife species known to accumulate relatively high body burdens of lipophilic contaminants (e.g., certain marine mammals). Mean consumption of Lake Ontario salmonids was 14.24 g/person/day. The mean concentration of HCB in fish muscle consumed is estimated to be 12.2 ng/g wet weight, thus intake via fish would be 2.5 ng/kg•bw/day (for a 70 kg adult). In combination with the 2.8 ng/kg•bw/day (above), the total calculated intake for people who consume salmonid species from Lake Ontario is estimated to be 5.3 ng/kg•bw/day, almost twice the level for adults of the general population. Based on an assumed body weight of 62 kg for residents of an isolated island community on the east coast of Baffin Island, the mean intake for adult consumers of Inuit food is calculated to be 92 ng/kg•bw/day. Roughly half of this intake is consumed in blubber, with significant contribution from meat, mattak (skin) and fat.

Table 1

HUMAN HEALTH EFFECTS FROM EATING OR DRINKING HCB

	Short-term exposure (less than or equal to 14 days)	
Levels in food	**Length of exposure**	**Description of effects**
		The health effects resulting from short-term human exposure to food containing specific levels of HCB are not known
Levels in water		
		The health effects resulting from short-term human exposure to water containing specific levels of HCB are not known
	Long-term exposure (greater than 14 days)	
Levels in food (ppm)	**Duration of exposure**	**Description of effects**
0.17	15 weeks	Minimal risk level (based on animal studies)
0.029	130 weeks	Minimal risk level (based on animal studies)
Levels in water		
		The health effects resulting from long-term human exposure to water containing specific levels of HCB are not known

Adapted from: U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR). *Public Health Statements – What you need to know about toxic substances commonly found at Superfund hazardous waste sites.* Atlanta, Georgia.

Although HCB has been detected in surface water and sediments, it is rarely detected in drinking water. Of the 189 surface water samples collected from the Great Lakes (1980-1986), the concentration in only one was found to exceed 1.0 ng/L (Hamilton Harbour at 4.0 ng/L). In industrial and municipal waste water, HCB has been detected at levels ranging from < 1–11.6 ng/L (various Canadian locations) to 28–2800 ng/L (Sarnia industrial complex). Levels in ambient air at Canadian sites averaged 0.15 ng/m^3, contributing a maximum of 1 to 2 percent of the total estimated intake.

Leachate from landfill sites may be a major source of HCB in the environment. Workers involved in the production of chlorinated chemicals and pesticides may be exposed to HCB (through inhalation) during synthesis or waste-disposal. Industrial water discharges serve as point sources of HCB and contribute to the contamination of suspended solids and sediments.

Reducing Exposure

Some Canadian communities may want to exercise some caution in the quantity of fish, sea mammals, or marine-bird eggs that they may be consuming. Follow any consumption recommendations provided by provincial, territorial or federal governments.

Human Health Considerations

HCB food poisoning occurred in Turkey in the late 1950s. Human health effects from this event included dermatological symptoms (porphyria cutanea tarda), liver toxicity (abnormal levels of porphyrin precursors), hyperpigmentation, hypertrichosis, enlarged thyroid, decreased uroporphyrin synthase levels, arthritis and small distinctive hands (due to osteoporosis in the phalangeal, carpal and metacarpal bones). Subfebrile temperature, anorexia and weight loss, weakness, and marked wasting of skeletal muscles were also common findings. In breast-fed infants, pink sores were noted, and exposure was fatal in children under two to three

years of age (mostly from cardio-respiratory failure, and convulsions in some cases). Two years after the onset of illness, affected children were significantly shorter and weighted less than their age peers in the region. The precise doses that produced these effects were not determined. It was estimated that extended exposure to such levels produced 10 percent mortality among the population exposed.

Although overt health effects seem not to be associated with HCB blood levels in the 2–40 µg/L range, there is some evidence (although weak) that marginal perturbations of porphyrin metabolism may occur. Such minor subclinical effects are not expected in association with the blood levels (< 2µg/L) seen in the general population. The International Agency for Research on Cancer has classified HCB as a possible human carcinogen, based on sufficient evidence in animals, but inadequate evidence of carcinogenicity in humans.

Table 2

TDI AND MAC VALUES FOR HCB

Agency	Focus	Level	Comments
Health Canada (CEPA)	All sources	0.050 µg/kg•bw/day	TDI
WHO	Drinking water	0.1 µg/L	guideline value *
WHO	Cow's milk	20 ng/g	MAC
U.S. FDA	Fish Action Level	0.3 µg/g (ppm)	

MAC: Maximum acceptable concentration
TDI: Tolerable daily intake
WHO: World Health Organization
U.S. FDA: United States Food and Drug Administration

* based on an evaluation of the production of liver tumours in female rats and applying the linearized multi-stage model to calculate an excess lifetime cancer risk of 10^{-6}

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Lead

Origin and Uses

Although lead occurs naturally, its dispersion throughout the environment is mostly due to human activities. Lead-acid battery waste is the most common source of lead, although most batteries are now recycled. Other uses include gasoline, the production of ammunition, metal products, paints, ceramic glazing, mining, smelting, refining of lead ores and the secondary smelting of lead containing materials (these are also sources for other metal emissions). The use of leaded fuels was the primary source of lead in the atmosphere, however, concentrations in the air have declined significantly since the introduction of unleaded gasoline in 1975. Use of lead in fuels was banned after 1990, with a few minor exemptions. In 1976, the *Hazardous Products Act* regulated lead levels for paint used in interiors. Although the lead content of exterior paint is not regulated, a voluntary agreement with Canadian paint manufacturers ensures that no lead will be intentionally added to exterior paint.

Persistence and Movement in the Environment

Lead is persistent in the environment. It is transferred continuously between air, water and soil by natural processes such as weathering, run-off, precipitation, dry deposition of dust and stream/river flow. It is removed from air by rain and by particles falling to the ground or into surface water. Soil and sediments appear to be important sinks for lead. Movement of lead from soil particles into underground water or drinking water is unlikely unless the water is acidic or soft. Many plants take up lead from the soil and it is present on plant surfaces as a result of atmospheric deposition. Plants and animals bioaccumulate lead, but biomagnification has not been detected. In general, the highest lead concentrations are found in aquatic and terrestrial organisms that live near anthropogenic sources.

Exposure

People are all exposed to lead through the air, dirt, household dust, food, drinking water and various consumer products. However, exposure to lead has decreased significantly since the early 1970s due primarily to the phase-out of leaded gasoline and the overall reduction of lead in the manufacture of cans and food packaging. The U.S. Food and Drug Administration (FDA) has recently amended its food additive regulations to prohibit the use of lead solder to manufacture cans for packaging foods. This recommendation is based on the available toxicological and exposure data that indicate that the use of lead solder to manufacture cans for packaging food may be injurious to health, particularly that of the fetus, infants, and children. The deadline for compliance for all affected products was June 27, 1996.

Food is likely the greatest source of lead exposure, with an estimated daily exposure of 1.1 micrograms per kilogram body weight (µg/kg•bw) for children (aged one to four years) and 0.75 µg/kg•bw for adults. Intake from air is in the range of 2–10 µg/day. An Ontario study found the median concentration of lead in drinking water to be 4.8 µg/L (Graham 1988). Using this median concentration and a daily intake of 1.5 L for an adult and 0.6 L for a child, the average daily consumption of lead from drinking water is 7.2 µg for an adult and 2.9 µg for a child. Soil surrounding housing may be high in lead levels due to paint chalking off the exterior, nearby point sources, or a home's proximity to high traffic areas.

Food can become contaminated from several sources: airborne lead deposited directly onto crops, and seepage into the soil and absorption by the plants. Illegal whiskey, made using stills containing lead soldered parts may contain lead; this is not thought to be a problem in Canada. Consumption of produce from family gardens, intake via smoking and wine are additional sources of exposure to lead. Contamination of fish by alkyl lead occurred in the St. Lawrence River as a result of discharges from manufacturing plants. Lead levels have declined in the St. Lawrence River since these plants were shut down in 1985.

Lead is present in raw water as the result of atmospheric deposition in surface water bodies. Very little lead is usually found in drinking water, although this amount can increase in communities with acidic water supplies. Lead present in drinking water is a result of leaching from water supply pipes, in solder used in plumbing, and in some plumbing fixtures (e.g., brass). The use of lead in solder supplied for plumbing purposes is now restricted in some provinces (e.g., Ontario). As a result, lead levels in drinking water increase with the length of time the water is left standing in the plumbing system.

Houses built before 1960 were likely painted with a lead-based paint. Exterior paint containing lead will carry a warning label; this paint should not be used on the inside of a building. People living in houses built after 1980 need not be concerned about lead levels in interior paints.

People living in areas surrounding hazardous waste sites may be exposed to lead through the ingestion of contaminated water or soils, or by inhalation of lead particles in the air. Wastes such as old lead paint-covered debris (i.e., from renovations), some ceramics, and plastics containing lead contribute to the contamination of landfill sites.

Children are at greatest risk for experiencing lead-induced health effects, particularly in urbanized low-income districts. Soils and household dust are significant sources of lead exposure for small children. The other important pathways are ingestion of chips from lead-painted sources, inhalation of airborne lead, intake of drinking water from lead-soldered plumbing, and medications (cold remedies). Health effects in children generally occur at lower blood lead levels than in adults. The pulmonary absorption of lead is estimated to be 48 to 64 percent in adults and > 70 percent in children. Gastro-intestinal absorption is approximately 10 percent for adults and up to 50 percent for children. Placental transfer of lead occurs in humans as early as the 12th week of gestation, and uptake of lead by the fetus continues throughout development. Exposure is also increased for young children because they swallow more lead through normal mouthing activity and pica. Pica is abnormal eating behaviour in which materials are ingested that are not fit for consumption. These include soil, clay, ashes, paint chips, and plaster.

It has recently been discovered that inexpensive one inch (2.5 cm) horizontal PVC (plastic) mini-blinds made in China, Taiwan, Indonesia, Hong Kong and Mexico contain lead. It has been estimated that exposure to lead from these mini-blinds could result in a daily lead intake by children that is greater than the World Health Organization's tolerable level.

Occupational exposure to lead is under provincial jurisdiction and a requirement for monitoring covers those in the construction industry, particularly renovators, smelter or foundry workers, and even artisans who work with stained glass. Home hobby supplies (e.g., paint pigments, stained glass products) can also be a source of lead exposure. Lead can still be found in ceramic glazes on pottery manufactured in some foreign countries (e.g., Mexico) and in some types of crystal. Even properly applied glazes may contain lead that can disintegrate after severe scouring or when exposed to acidic solutions. Decorative, lead-glazed pottery purchased outside Canada has been identified as a potentially serious source of lead exposure to individuals unaware of the need for its non-food use.

Once lead enters the body, it travels to the liver, kidneys, lungs, brain, spleen, muscle and heart, and particularly accumulates in the bones and teeth. Approximately 90 percent or more of the lead in adults is stored in the bones and teeth, while in children only about 70 percent is stored in the bones. The remainder is stored in the soft tissues and blood. In times of stress, immobilization or diseases of the skeleton such as osteoporosis, the body can mobilize bone lead stores, thereby increasing the level of lead in the blood. Lead is excreted from the body in the urine and feces.

Reducing Exposure

If lead-based paints are chipping or cracking, removal of paints and wallpaper should follow appropriate measures that will contain dust and debris. Advice has been provided in two pamphlets (HWC 1993; HPB 1991). Dry-sanding, heat guns and blowlamps **should not** be used to remove lead-based paint. Chemical strippers that consist of a paste applied with a brush (application strippers) are probably the safest way to remove lead-based paint. However, care must be taken when using chemical strippers, as they contain potentially harmful substances. The following precautions should be taken when using them: children and pregnant women should limit their exposure; remove as much of the furnishing from the work area as possible, or cover completely with plastic sheeting; cover doorway and vents with plastic sheeting and tape in order to isolate the work area from the rest of the house; make sure the work space is properly ventilated (place an electric fan between the entrance to the room and an open window); wear protective masks and clothing; work for short periods at a time, taking a break outside; never eat, drink or smoke while removing paint; clean the work area thoroughly at the end of each day. Lead-containing paint scrapings should not be discarded with the garbage. Contact either your local municipality or the local office of the provincial Ministry of the Environment to find out how to dispose of old paint strippings. If professionals have been hired to do the work, ensure that the above is followed.

If the painted surfaces are not cracking, it is best that they be sealed with vinyl wallpaper, wallboard or panelling. In areas that are out of children's reach, surfaces may be covered by non-leaded paint.

It is recommended that ceramic dishes or crystal not be used to store acidic food or drink. For example, vinegary food or drink should never be stored in pottery. Surfaces should be kept clean, especially where food is prepared. Brandy or sherry that has been stored for several years in crystal decanters should not be consumed as the lead used in the making of the glass may leach out into the liquid; the liquor should be discarded. The storage of brandy or sherry for short periods of time (a few months) should not be of concern.

Because lead is a component of many plumbing systems, first-drawn water may contain higher concentrations of lead than are found in running water after flushing. In order to minimize exposure to lead in drinking water, it is recommended that only the cold water supply be used, after an appropriate period of flushing to rid the system of standing water, for drinking water, beverage preparation, cooking and especially in the preparation of baby formula. The recommended flushing time is as follows: if the water has been standing more than five hours, run the tap vigorously for at least one minute so that the water runs cold (or as cold as the water will get). For most municipalities, only water that has been standing unused for more than five hours needs to be flushed. Boiling the water will not reduce lead levels.

Individuals with children aged six years or less should remove inexpensive mini-blinds made in China, Taiwan, Indonesia, Hong Kong, and Mexico from their homes. PVC mini-blinds containing lead should also be removed from other locations frequented by children of this age group (e.g., child-care facilities, schools).

Human Health Considerations

The effects of lead are the same regardless of whether it enters the body through inhalation or ingestion. Susceptibility to lead toxicity is influenced by dietary levels of calcium, iron, phosphorus, vitamins A and D, dietary protein and alcohol.

The fetus, infants, and children up to six years of age are the most susceptible to the adverse health effects of lead. They absorb lead from the gastro-intestinal tract more readily that adults do and their tissues are in a stage of rapid development. Pregnant women are also a sub-population of special concern as a result of the potential for lead exposure to the developing fetus. There is no placental barrier to the transfer of lead to the fetus. Researchers believe that the last trimester of pregnancy may be the most critical time for the adverse effects of lead exposure to occur.

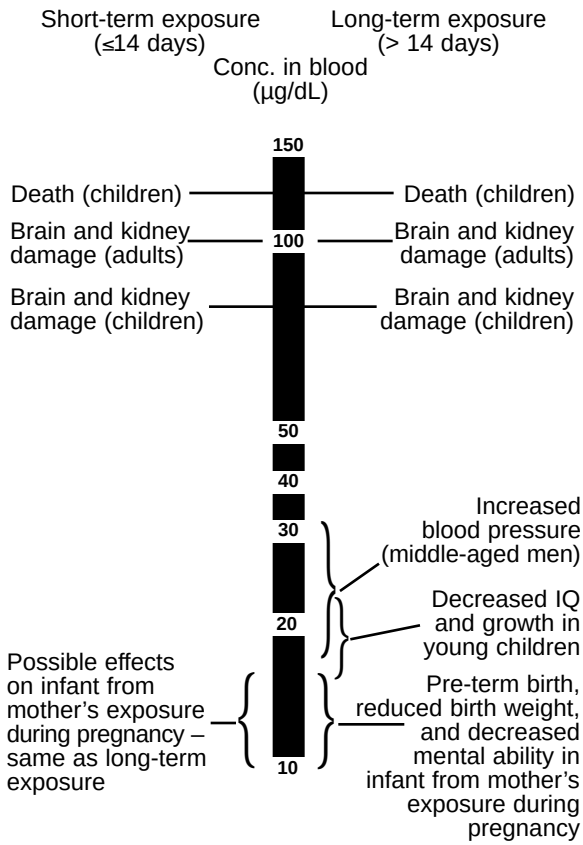
The central and peripheral nervous systems are the principal targets of lead toxicity. Childhood lead poisoning is one of the most common pediatric health problems in North America. Studies indicate that exposure to even low levels of lead prior to birth, or during infancy and early childhood have been associated with

reduced birth weight, impairment to intellectual development (decrease in IQ), behavioural disturbances, decreased childhood size, speech and hearing impairment and effects on peripheral nerve function. The first signs of lead poisoning in children are often subtle neurobehavioural problems that adversely affect classroom behaviour and social interaction. The blood level considered to pose negligible risk has decreased steadily over the years and evidence suggests possible adverse health effects on children at blood levels of only 10 µg/dL (decilitre). The Federal-Provincial Committee on Environmental and Occupational Health has recommended a series of intervention strategies for different blood levels at the individual and community levels (see tables 2 to 4).

Early symptoms of acute exposure to high levels of lead include metallic taste, dullness, irritability, poor attention span, headache, muscular tremor, memory loss, and hallucinations. These symptoms worsen to delirium, convulsions, paralysis, coma and even death. Subsequent to high levels of exposure the following have been noted: damage to the brain and kidneys in adults or children; increased incidences of spontaneous abortion, miscarriages and stillbirths; damage to the male endocrine (disruption of luteinizing hormone secretion); and the male reproductive system (decreased sperm production). Lead interferes with a hormonal form of vitamin D, which affects multiple processes in the body, including cell maturation and skeletal growth.

Figure 1

HUMAN HEALTH EFFECTS FROM INGESTING AND BREATHING LEAD



Adapted from: U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR). *Public Health Statements – What you need to know about toxic substances commonly found at Superfund hazardous waste sites.* Atlanta, Georgia.

In adults, health effects from chronic exposure to lead include extensor muscle weakness, anorexia, paresthesias in lower limbs, weakness of upper limbs, poor performance on cognitive and visual-motor coordination, impaired verbal reasoning ability, anemia, abdominal pain and constipation, fatigue, sleeplessness. Lead affects heme biosynthesis by affecting the activities of several enzymes of the heme biosynthetic pathway; the clinical symptom is anaemia. Chronic exposure (mostly due to occupational exposure) has resulted in an increased number of deaths due to cerebrovascular disease. Conflicting data exist concerning an association between blood lead levels and hypertension, chronic nephropathy and effects on thyroid function. For hypertension, the evidence is most convincing in adult men aged 40-59 years of age and for systolic rather than diastolic pressure. Recent evidence of raised blood lead levels in older people, as a result of mobilization of lead from their bone, has particular significance in the light of this link to hypertension. In the case of chronic nephropathy, blood lead levels measured at the time of renal function testing may not fully reflect the exposure history that contributed to its development in lead workers. Lead-induced chronic renal insufficiency may result in gout. Data on immunological effects in occupationally exposed people are inconsistent. They indicate that while an association may exist between the cellular component of the immune system and lead exposure, the humoral component is relatively unaffected.

Lead is classified as possibly carcinogenic to humans (inadequate data in humans, limited evidence in animals) by the International Agency for Research on Cancer.

Table 1

MAC, TOLERANCE, PTWI AND ADI VALUES FOR LEAD

Agency	Focus	Level	Comments
Health Canada	Drinking water	0.01 mg/L	MAC
Health Canada	Fruit juices, cider, wine; in beverages as consumed and water in sealed containers other than mineral water.	0.2 mg/L (ppm)	tolerance
Health Canada	Edible bone meal	10 µg/g (ppm)	tolerance
Health Canada	Evaporated, condensed milk, concentrated infant formula	0.15 µg/g or mg/L (ppm)	tolerance
Health Canada	Ready to serve infant formula	0.08 mg/L (ppm)	tolerance
Health Canada	Fish protein	0.5 µg/g (ppm)	
Health Canada	Tomato paste and sauce	1.5 µg/g (ppm)	tolerance
Health Canada	Whole tomatoes	0.5 µg/g (ppm)	tolerance
OMOEE	Drinking water	0.01 mg/L *	MAC
OMOEE	Fish	< 1.0 µg/g (ppm); unrestricted consumption	
WHO	All sources (total lead)	25 µg/kg•bw/week (children)	PTWI. (This is equivalent to an ADI of 3.5 µg/kg•bw/day.)
WHO	Air	0.5 – 1 µg/m^3 (annual)	
WHO	All sources	3.5 µg/kg•bw/day	ADI
WHO	Drinking water	0.01 mg/L (it is recognized that not all water will meet the guideline value immediately; meanwhile, all other recommended measures to reduce the total exposure to lead should be implemented)	guideline value
JECFA	All sources	3.57 µg/kg•bw/day (adults and children)	PTDI

MAC: Maximum acceptable concentration
ADI: Acceptable daily intake
PTDI: Provisional tolerable daily intake
OMOEE: Ontario Ministry of Environment and Energy
PTWI: Provisional tolerable weekly intake
JECFA:Joint Food and Agriculture Organization/World Health Organization Expert Committee on Food Additives and Contaminants
Health Canada: *Food and Drugs Act* and Regulations.

* The objective applies to water at the point of consumption. Since lead is a component in some plumbing systems, first flush water may contain higher concentrations of lead than water that has been flushed. Faucets, therefore, should be thoroughly flushed before water is taken for consumption.

Table 2

Blood lead intervention levels (individual children or adults)

Blood level	Intervention
< 10 µg/dL (0.5 µmol/L)	None
> 10 µg/dL – 14 µg/dL[1] (0.5 – 0.75 µmol/L)	Identify possible exposure source. Where significant number of children are in this category, consideration of community intervention.
> 15 µg/dL[1] (0.75 µmol/L)	Review exposure history to identify source of lead exposure. Exposure reduction counselling by medical personnel or public health authority and advice on hygienic and nutritional measures.
20 – 44 µg/dL[2] (1.0 – 2.2 µmol/L)	Case management medical assessment. Identify and eliminate environmental lead problem.
45 – 69 µg/dL[2] (2.2 – 3.4 µmol/L)	Urgent[3] medical and environmental assessment and possible hospitalization of children.
70 and greater[2] (3.4 µmol/L)	Urgent[3] medical assessment and possible hospitalization of children.

1. Retest in 3–6 months in all cases.
2. Retest immediately.
3. Urgency depends on age, blood lead level and clinical symptoms as determined by a knowledgeable authority

Note: The levels and intervention strategies in this table are considered appropriate for environmental exposures. Alternative strategies may be considered for workplace exposures.

Source: *Update of Evidence for Low-level Health Effects of Lead and Blood Lead Intervention Levels and Strategies* by the Federal-Provincial Committee on Environmental and Occupational Health for the Environmental Health Directorate, Health Canada, September 1994, 59pp.

Table 3

Blood lead intervention levels (community)

Blood lead µg/dL (µmol/L)	Intervention
Mean blood lead levels in children exceed the mean plus 3 standard deviations from the mean general population [1] or when % of children with blood lead above 0.5 µmol/L (10 µg/dL) is double that of the general population[2]	Consider community program to identify/reduce source of exposure and provide information to the community about methods to reduce individual exposures.

1. The most current data, which may be used as a reference population, are derived from 1992 Ontario Blood Lead Surveys (OBLS) of blood lead in children:
   OBLS control (no point source, 3.48 ± 2.22 µg/dL urban, downtown Toronto)
   OBLS (remote community, Moosonee) 2.71 ± 2.39 µg/dL
   OBLS (remote native community, 3.40 ± 1.86 µg/dL, Moose Factory)
   OBLS (remote, combined) 3.10 ± 2.10 µg/dL
2. In 1990 indications were that for the general population, approximately 5 percent of children exceeded a mean lead level of 0.5 µmol/L (10 µg/dL). As values may still be declining, a decision on the appropriate value for the population mean blood lead would rest with each jurisdiction.

Source: *Update of Evidence for Low-level Health Effects of Lead and Blood Lead Intervention Levels and Strategies* by the Federal-Provincial Committee on Environmental and Occupational Health for the Environmental Health Directorate, Health Canada, September 1994, 59pp.

Table 4

EXPOSURE TO LEAD FOR CHILDREN AND ADULTS FROM SEVERAL SOURCES OF EXPOSURE

Amount	Population Group	Source of Exposure
1.1 µg/kg•bw/day	children	food
0.75 µg/kg•bw/day	adults	food
20–100 µg/day		air
2.9 g	children	drinking water*
7.2 g	adults	drinking water*

* Average levels of lead in drinking water are 4.8 µg/L. Intakes are based on the assumption that daily intakes for adults are about 1.5 L and 0.6 L for a child.

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Mercury

Origin and Uses

Mercury exists in three forms: in its elemental form (Hg^0), as inorganic salts (e.g., mercuric chloride - Hg^{2+}) and as organic (alkyl) mercury (e.g., methyl mercury - meHg).

Mercury is present in air, water and soil from both natural sources and as a result of human activity. Atmospheric mercury is primarily a result of global off-gassing of mercury from soils and surface waters, the burning of fossil fuels, and the disposal of solid wastes (batteries, electrical switches) in landfills. All soils in Canada contain mercury at levels averaging about 0.06 µg/g (0.08 µg/g in Ontario). Higher natural background contamination is associated with rock and mineral deposits in the Canadian Shield and in the Rocky Mountains. Weathering of naturally occurring mercury in rocks and industrial effluent are the major sources of water contamination. Water sources near mercury deposits have been found to contain up to 80 µg mercury/L, compared with 0.1 µg/L in low natural mercury areas.

Anthropogenic sources in Canada include historical industrial activity (old mercury, gold, and silver mines and their tailings); releases from sulphide ore (copper, lead, nickel) smelting activities, coal-burning plants, and the burning of other fossil fuels; chlor-alkali plants; historical use of mercury as a slimicide in the pulp and paper industry; and the historical agricultural use of alkyl mercury fungicides. Registration for the treatment of cereal grains with alkyl mercury fungicides was discontinued in Canada in 1973. The amount of mercury released into the environment has decreased steadily since the early 1970s due to regulations and changes in industrial processes. Mercury has not been mined in Canada since 1975.

There are many additional uses of mercury including dental amalgam; thermometers, electrical switches, and batteries; and paint preservatives. Although used in the past, mercury is no longer used in medicine (diuretics, antiseptics, and skin preparations). Prior to 1991, 30 percent of interior latex paint contained mercury compounds as a preservative. This was discontinued in January 1991 for interior latex paints but exterior paints may still contain mercury. Mercury salts are also used in skin-lightening creams and as antiseptic creams and ointments.

Persistence and Movement in the Environment

Mercury can enter the atmosphere as elemental mercury, as volitilized organic mercury components, as inorganic mercury, or associated with particulate matter. Elemental mercury released to the atmosphere can be transported long distances before being removed by wet or dry deposition. Naturally occurring mercury in soils and water and most industrial discharges are inorganic and not easily absorbed by animals and plants (spinach is an exception). The dominant process controlling the distribution of mercury compounds in the environment appears to be the sorption of non-volatile forms to soil and sediment particulates.

Inorganic mercury is methylated to methyl mercury by microbes in soils, and in river and lake sediments. Methyl mercury is more bioavailable and active in living systems. Flooding causes the biochemical mobilization of mercury to change due to the presence of decomposing submerged plants (a food source for microbes) that results in an increase in mercury methylation by microbes. While the source of the mercury is natural, the observed levels are a result of anthropogenic changes to the ecosystem.

Reduction of inorganic mercury to elemental mercury in aqueous systems can also occur. Methyl mercury bioaccumulates in fish and other aquatic organisms, and is biomagnified up the food chain. Thus methyl mercury is highest in concentration in species at the top of the food chain such as pike, walleye, and bass in freshwater; tuna, swordfish, and shark in seawater. Virtually all of the total mercury detected in fish is in the form of methyl mercury.

Exposure

Of the total amount of mercury taken in, referred to as the delivered dose, the amount actually absorbed depends on both the route of exposure, and the form of mercury the individual is exposed to. Inhaled as a vapour, elemental mercury (Hg^0) is almost completely absorbed (about 80 percent) and diffuses rapidly across the placental and blood-brain barriers. When ingested, elemental mercury is poorly absorbed from the gastro-intestinal tract (about 0.01 percent) and on average, less than 10 percent of an ingested mercury salt (Hg^{2+}) is absorbed by adults. However, absorption of mercury salt by infants and toddlers is assumed to be 100 percent. Dermal absorption of elemental mercury and ionic mercury salts can also cause toxicity. Organic mercury (methyl mercury) is readily absorbed regardless of the exposure route. Feces and urine are the principal routes of elimination for mercury.

The estimated intake for an adult Canadian of all forms of mercury via all routes is 7.7 µg/day (0.11 µg/kg•bw/day). This is equivalent to an absorbed dose of 5.3 µg/day. Of these total amounts, fish consumption contributes about 27 percent of the intake and 40 percent of the absorbed dose. Mercury in fish is in the methylated form. Exposure to mercury from dental amalgam, present as elemental mercury, accounts for a greater proportion of total mercury exposure than fish consumption, accounting for roughly 37 percent of the intake and 42 percent of the absorbed dose. Mercury from foods other than fish (present as Hg^{2+}) represents about 23 percent of the intake and only 3.4 percent of the absorbed dose.

While dental amalgam has been shown to be a significant source of mercury, the current weight of evidence suggests that amalgam does not release sufficient quantities of mercury to cause illness. There is currently no evidence to indicate a need to have amalgam fillings replaced unless there is a particular sensitivity to dental amalgam. Although alternatives are available, there is currently no direct filling material available that can fully replace amalgam, and the potential toxicity of the alternatives has not been fully investigated.

A sub-population at greater risk for methyl mercury exposure are the recreational anglers who may be heavily exposed through their fish-rich diet. One estimate puts exposure of recreational anglers at 13 µg total mercury per day from all sources, with roughly half in the form of meHg from fish. Fish caught in the Great Lakes and Lake St. Clair are below Health Canada's 0.5 µg/g advisory. However, in the St Lawrence River and northern communities some species have reached and exceeded this guideline. Aboriginal populations may also be at higher risk for mercury exposure via their diet, which is likely to contain high levels of mercury contamination in both methylated and inorganic forms. Sources of dietary exposure include whale and seal muscle, liver and kidney of sea mammals, reindeer and caribou, fish-eating birds or the eggs of fish-eating birds. It has been estimated that Amerindian adults may be exposed to 105 µg meHg/day while Inuit adults may be exposed to 257 µg Hg^{2+}/day and 92 µg meHg/day.

Mercury in Ontario drinking water was below the detection limit (0.02 µg/L) in all but eight of 1355 samples taken. Only a small portion of this was in the more toxic organic form (of methyl mercury), thus drinking water contributes very little to overall exposure. Ambient air is also unlikely to be a major contributor. Urban air levels in and around Toronto in 1981 were 10 ng Hg/m^3. However, exposure to elemental mercury via inhalation in indoor air may be substantial.

A major source of mercury in the indoor environment is through mercury containing paints. Mercury use in indoor paints was discontinued as of January 1991 although retail supplies were not recalled after the ban. It is possible that some people are still using mercury-containing paints, especially if partly used cans were saved for repainting. Painted surfaces can continue to emit mercury vapours (primarily elemental mercury) for several years. Other sources of mercury in the home include breakage of thermometers, barometers, and regulators.

Reducing Exposure

Proper ventilation should be provided when mercury-containing paints are used. These paints should not be used indoors. For populations consuming high levels of fish and seafood, advisory guidelines should be followed. If a mercury thermometer breaks and spills in the house, **do not** try to vacuum it up. Call your local health official for advice because even a single broken thermometer can lead to toxic effects and hospitalization.

While the use of amalgam for dental restoration continues to be supported by the Canadian Dental Association, there are some sub-populations at particular risk for mercury exposure who may want to consider alternatives to amalgam for dental restoration. These include individuals who are immunologically compromised, individuals who suffer from neurological conditions, people with impaired kidney function, and those who have a particular sensitivity to mercury (about 3 percent of the population). Other individuals who may wish to consider alternatives to amalgam include people with occupational exposure to heavy metals, people with greater than average exposure from the diet, and women who are pregnant or breast feeding.

There is currently insufficient evidence to justify the removal of amalgam for the general population for health reasons.

Human Health Considerations

The two major responses to mercury poisoning are neurological and renal disturbances. Neurological effects are characteristic of poisoning by methyl and ethyl mercuric salts, while renal effects are characteristic of inorganic mercurial poisoning.

An important consideration from the standpoint of human health is that infants and children may have an increased exposure to mercury due to natural mouthing activities. They also absorb more of the mercury salts they consume as compared with adults.

Inorganic mercury salts

Acute inhalation effects include inflammation of the oral mucous membranes and gastro-intestinal complaints. Chronic inhalation leads to tremors of the head and extremities, failure of muscular coordination, slowness of movement (physical and mental responses) and disturbances in the peripheral nervous system. Proteinuria can occur without apparent renal failure. Acute ingestion has resulted in corrosion of mucosa (gastric pain, vomiting and bloody diarrhea), death of the tissue of the intestinal mucous membranes leading to circulatory collapse and death, and renal failure. Chronic ingestion produces symptoms of ptyalism (excessive secretions of saliva), gingivitis (inflammation of the gums), black lines of mercuric salts on the teeth, inflammation of the kidney. An association has been suggested between dermal exposure of skin creams containing mercury and an accumulation of acid in the kidney in children, and between kidney disease and mercury-containing skin-lightening creams. Other dermal effects include topical lesions and inflammation of the skin. Contact with eyes causes development of ulcers of the conjunctiva (membrane that lines the eyelids) and the cornea.

Table 1

HUMAN HEALTH EFFECTS FROM BREATHING ELEMENTAL MERCURY

Short-term exposure (less than or equal to 14 days)		
Levels in air (ppm)	**Length of exposure**	**Description of effects***
0.13	3 hours	Chest pains, shortness of breath, cough
5.4	8 hours	Persistent irritability, lack of ambition, lack of sexual desire
Long-term exposure (greater than 14 days)		
Levels in air (ppm)	**Length of exposure**	**Description of effects***
0.0032	15 years	Shakiness.
0.000032		Estimated minimal risk level (based on effects seen in humans)

* These effects are listed at the lowest level at which they were first observed. They may also be seen at higher levels.

Adapted from: U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR). *Public Health Statements – What you need to know about toxic substances commonly found at Superfund hazardous waste sites.* Atlanta, Georgia.

Elemental mercury

Acute inhalation effects are similar to those for inorganic mercury salts, and also include bronchitis and bronchopneumonia, inflammation of the lungs, tremors and increased excitability. Death from severe pulmonary damage or pulmonary edema (the presence of large amounts of fluid) can occur. Chronic inhalation of mercury results in micromercurialism (weight loss, fatigue, anorexia, gastro-intestinal complaints), followed by tremor, gingivitis and increased excitability. Other symptoms include ptyalism, thyroid enlargement, unstable pulse, and excessive sensibility to stimulation. An association with toxicity to the kidneys has been indicated. Proteinuria and enzymuria (the presence of protein and enzymes, respectively, in the urine) have been observed. Ingested liquid mercury has not been shown to be toxic in people (doses of 100–500 g), due to poor adsorption.

Organic mercury

Effects from acute and chronic exposure through ingestion are known primarily from large populations affected by the ingestion of methyl mercury contaminated fish in Minimata Bay (1956) and Niigata (1965), and through the ingestion of methyl mercury contaminated seed consumed as bread in Iraq (1971-72). In Canada, some Aboriginal communities consuming large amounts of methyl mercury contaminated fish and wildlife have reported symptoms which suggested mild poisoning. The health effects are known as Minimata Disease. Exposure results in progressive nervous system effects. The initial symptoms are numbness and tingling of the lips, tongue and distal extremities. These are followed by loss of motor coordination (including walking), tremor, loss of fine movement, muscular rigidity, spasticity and seizures. Other symptoms that may occur are constriction of the visual fields, hearing loss, nervous exhaustion caused by abnormal fatiguability, imperfect articulation in speech, coma and death; erythema (abnormal redness) and desquamation (shedding of the skin), and other skin rashes; behavioural changes, fits of laughter and intellectual impairment.

Prenatal exposure to organic mercury affects the development and maturation of the central nervous system and results in psychomotor retardation. Such exposure can occur at levels at which no maternal effects are noted (maternal hair mercury levels > 10–20 µg/g). Delays in walking and speech of over 12 months may occur (maternal hair mercury levels 50 µg/g). Retarded walking may occur at maternal hair levels of 6–10 µg/g, with a threshold at 5 µg/g. Neurobehavioural dysfunction may occur at maternal hair levels of > 6 µg/g. Seizure and features resembling cerebral palsy often arise. Mild neurologic and developmental delays have been reported in infants exposed to methyl mercury through breast milk.

Chronic exposure to inorganic or organic mercury

Acrodynia is a rare disorder that occurs in a small number of children (four months to four years) even though many are exposed. It has been associated mostly with mercury salts found in calomel or topical medications, but also from phenylmercury fungicides used in diaper cleaning, and mercury compounds in interior latex paints. Acrodynia is characterized by leg cramps; a generalized rash followed later by skin ulceration; tachycardia (excessive rapidity in the action of the heart); intermittent low grade fever, chills, profuse perspiration, dilated enlarged sweat glands; erythema, swelling, and desquamation of the hands, feet, cheeks, and nose; weakness of the pelvic and pectoral girdles; marked personality change; impaired reflexes, tremor, lower-extremity nerve dysfunction; hair loss, photophobia, and hypotonia (abnormally diminished tone).

Organic mercury is mutagenic in animal experiments. Mercury compounds have not been demonstrated to be carcinogenic in humans or animals.

Table 2

MAC, TOLERANCE, PTWI AND TDI VALUES FOR MERCURY

Agency	Focus	Level	Comments
Health Canada	Drinking water	1.0 µg/L	MAC
Health Canada	Edible portion of fish	0.5 µg/g (ppm) (total Hg)	guideline
Health Canada	Shark and swordfish exempt from guideline above	consumption of these species should not exceed 1 meal/wk	guideline
OMOEE	Fish flesh	<0.5 mg/kg: unrestricted consumption, adults 1.5 mg/kg: restricted consumption, adults <0.5 mg/kg: restricted consumption, children under 15 and women of childbearing age	guideline
OMOEE	Drinking water	1.0 µg/L	MAC
WHO	All sources	0.47 µg/kg•bw/day: methyl mercury	TDI
WHO	All sources	0.71 µg/kg•bw/day: total mercury (methyl + inorganic mercury)	TDI
WHO	Drinking water	0.001 mg/L (total mercury)	guideline value
JECFA	All sources	3.3 µg/kg•bw/week (methyl mercury)	PTWI
JECFA	All sources	5.0 µg/kg•bw/week (total mercury)	PTWI

MAC: Maximum acceptable concentration
TDI: Tolerable daily intake
OMOEE: Ontario Ministry of Environment and Energy
PTWI: Provisional tolerable weekly intake
JECFA: Joint Food and Agriculture Organization/World Health Organization Expert Committee on Food Additives and Contaminants
WHO: World Health Organization

Table 3

HEALTH CANADA GUIDELINES FOR TOTAL MERCURY

	Whole blood (µg/L or ppb)	Hair (µg/g or ppm)
Normal acceptable range	20	6
Increasing risk	20 – 100	6 – 30
At risk	> 100	> 30

Source: *Methyl mercury in Canada.* Vol II. Ottawa: National Health and Welfare, p.21.

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Mirex

Origin and Uses

Mirex does not occur naturally in the environment. It was first manufactured in 1946, primarily for use as an insecticide to control fire ants. Mirex was also used as a stable fire retardant additive in thermoplastics and resins, and as an additive in paper, paint, rubber, electrical adhesive and textile applications. It was sold under the trade names Dechlorane and Ferriamicide. Although use of mirex in Canadian agriculture was never permitted, it has been imported into Canada for other uses, and has been used extensively in the southeastern United States. Approximately 75 percent of the mirex used in the United States was for non-agricultural applications.

All uses of mirex have been banned in Canada since 1978, and its use as an insecticide was banned in the United States in the same year. Patents for mirex use exist in several countries including Belgium, France, Germany, Japan, the Netherlands, and the United Kingdom but little use information is available.

Persistence and Movement in the Environment

Mirex is extremely stable and highly persistent in the environment. Mirex is not soluble in water and is essentially non-volatile, thus it is not usually present in water or air. Slow partial photodegradation does occur, and mirex is degraded to photomirex (8 monohydromirex), a compound containing one less chlorine atom. Mirex is very resistant to degradation by soil bacteria, and is only dechlorinated to photomirex by anaerobic microbial action. Mirex is highly persistent in sediment and soil for extended periods of time, where it is bioaccumulated by biota. Mirex is biomagnified in aquatic and terrestrial food chains, including humans.

Exposure

In humans, mirex is stored mainly in fat tissue, where it is not broken down. Mirex that is not stored is excreted primarily in the feces, and a very small amount in the urine.

The primary route of exposure for mirex is through food, mostly the consumption of contaminated fish. The highest concentrations were found in fish from Lake Ontario, the St. Lawrence River, and the southeastern United States. It is unlikely that current levels in Great Lakes fish are a risk to human health, if fish consumption advisories are followed. For example, in 1988 a person who consumed Lake Ontario fish (114 g fish/meal/week) containing a concentration of 0.10 mg/kg of mirex would ingest 5.7 µg of mirex/person/week (or 0.022 µg/kg•bw/day). This is below the Canadian provisional tolerable daily intake (PTDI) of 0.07 µg/kg•bw/day. Mirex levels in breast milk are above average for communities consuming high amounts of fish, marine-bird eggs, or sea mammals (Dewailly et al. 1991; Davies and Mes 1987).

Releases from waste disposal sites continue to add mirex to the environment. Thus, populations living near hazardous waste sites storing mirex might be exposed through dermal contact (although it is not known if mirex can enter the body through dermal contact) or through ingestion of contaminated soil or indigenous wildlife. Higher levels of mirex in human adipose tissue have been correlated with areas of mirex usage, manufacture, or disposal at waste sites in the southeastern United States (Burse et al. 1989; Kutz et al. 1974), New York (Bush et al. 1983), and Ontario (Williams et al. 1988).

Mirex is rarely found in drinking water and air. It was detected in only five out of 1147 samples taken of Ontario drinking water in 1987, the highest concentration was 5 ng/L. Mirex was measured at very low levels, ranging from 4–1000 ppq in ambient air samples taken from southern Ontario.

Reducing Exposure

Contaminated fish, marine-bird eggs, or sea mammals may present a risk when consumed at levels in excess of advisory recommendations. Follow fish consumption advisories.

Human Health Considerations

Data on the human health effects of mirex are not available. For this reason, the primary organs affected by mirex in experimental animals have been included. They are the liver, kidneys, eyes, and thyroid.

Acute and intermediate duration: diarrhea (due to hemorrhagic intestines); increase in hematocrit; hepatic effects (adaptive and toxic effects); dermal/ocular effects (hair loss, production of cataracts in very young, mild epidermal proliferation; in mice); toxic effects to the thyroid; adrenal gland hypertrophies and releases increased levels of corticosterone; decreases in serum glucose levels; decreases in body weight or body weight gain greater than 10 percent; abnormal behaviour (lethargy, weakness, hyper-excitability, tremors, convulsions); reproductive and developmental effects in female and male rats.

Chronic exposure: renal effects; decreases in body weight or body weight gain greater than 10 percent, non-precancerous lesions of the liver; cancer (an increased incidence of hepatocellular adenomas have been noted, but only in animals having hepatotoxicity). In 1992, it was concluded that only weak evidence exists for the hepatocarcinogenicity of mirex (Sauer 1992).

The International Agency for Research on Cancer (IARC) has classified mirex as possibly carcinogenic to humans, based on sufficient evidence in animals, but inadequate evidence of carcinogenicity in humans.

Table 1

PTDI AND OTHER VALUES FOR MIREX

Agency	Focus	Level	Comments
Health Canada	Drinking water	no guideline	
Health Canada	Fish	0.1 µg/g	
Health Canada	All sources	0.07 µg/kg•bw/day	PTDI
OMOEE	Fish flesh	> 0.1 µg/g consumed occasionally < 0.1 µg/g unrestricted consumption (excluding women of childbearing age and children)	

PTDI: Provisional tolerable daily intake
OMOEE: Ontario Ministry of Environment and Energy

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Moulds

Origins

Moulds are part of the fungal kingdom, which includes approximately 50 000 species. Moulds are ubiquitous in indoor and outdoor environments. They produce particles (spores and residual matter) and gases (mouldy or musty odours). High moisture levels in the home will encourage the growth of moulds.

Exposure

Moulds grow in moist, damp places. A musty odour, black, white or multi-colour discolourations, dampness, and moisture condensation indicate the presence of moulds.

Inhalation of fungal spores in the air is the most common route of exposure. Ingestion of contaminated foods, such as peanuts, or dermal exposure with contaminated surfaces or particles in the air are additional exposure routes.

Prime places within a home for mould growth are in basements and cold cellars, on wet windows, in carpets, kitchen and bathroom sinks, floor drains, and closets. Attics, humidifier trays (as well as dehumidifiers, air conditioners, refrigerators and laundry equipment), filters (for the furnace, air conditioner, heat recovery ventilator), potted plants, house dust, and pet dander are all additional sources of exposure to mould.

Reducing Exposure

Reducing exposure rests on removing the substrate on which the mould is growing, and on moisture control. This can include cleaning affected areas; decontaminating heating, ventilation, air-conditioning systems; removing contaminated materials; repairing or replacing damaged material or structures; and modifying the environmental conditions in the affected area. For example, reducing water leaks and condensation will generally reduce growth. Fungicides have limited, if any value in these situations.

If mould is evident on surfaces such as windows or tile, or if the basement is damp and musty, dry out these areas by opening a window slightly, and by running an exhaust fan. During the summer use a dehumidifier or an air conditioner.

Heating, ventilating, and air conditioning systems should be properly cleaned and maintained. Room or furnace humidifiers should be thoroughly cleaned and disinfected regularly. A chlorine bleach diluted one part bleach to two parts water, should be used for cleaning, and humidifiers should be kept dry when they are not in use.

Household surfaces should be wiped regularly and kept free of dust. Vacuum regularly to eliminate dust and small insects or mites. Heating ducts should be cleaned regularly.

Human Health Considerations

Moulds in the home or other indoor environments, can cause many health problems. These include allergies and other immune-mediated illnesses, and non-specific respiratory and flu-like symptoms, ranging from acute infections to chronic illness. Allergic reactions may be immediate (develop within minutes of exposure) or delayed (four to eight hours later). Both of these have been observed in an asthmatic response to an allergen. Chest tightness, cough and sometimes wheeze occurs. Systemic infections result from initially inhaling the responsible organism. They begin primarily as lung infections, but the fungus can migrate to other organs including the heart, brain and kidneys.

The evidence for clinical effects of exposure to indoor air moulds is clear for conditions such as asthma, hypersensitivity pneumonitis, fungal infections, organic dust toxic syndrome, and aflatoxin-related cancers. Factors influencing the severity of the illness include the concentration, shape and size of the organism, and the production of toxins and volatiles by the organism.

Certain individuals are more susceptible to air moulds. They include infants, asthmatics, individuals with pre-existing respiratory conditions, and individuals with compromised immune systems.

It is not possible to definitively conclude that moulds are responsible for the wide variety of reported symptoms. This is because characterization of human exposure to moulds in residential settings is difficult with current methodologies, methods for measuring the production of mycotoxins in home settings are inexact, and the symptoms and reported health effects are difficult to validate objectively.

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Nitrites, Nitrates and Nitrosamines

Origin and Uses

Nitrates are naturally occurring inorganic ions, which are part of the nitrogen cycle, and are commonly found in groundwater and surface waters. The nitrate ion (NO_3^-) is the most stable form of nitrogen in oxygenated environments, thus all nitrogen-containing molecules can act as sources of nitrates. Under acidic conditions, nitrites (NO_2^-) are formed naturally from nitrates, and nitrites in turn may combine with amines or amides to form N-Nitroso compounds (nitrosamines).

Nitrates used in fertilizers are a major source of contamination of shallow groundwater aquifers that provide drinking water. Other sources of nitrate contamination are from septic system leaching, especially in wells less than 30 metres deep; industrial and municipal wastes; internal combustion engine emissions; sodium nitrite as an anti-corrosive agent in cooling fluids; ammonium nitrate in cold packs; and nitrous gases in arc welding.

Nitrites have been used for centuries as antimicrobial preservatives, especially to prevent botulism, in cured meat products (e.g., sausages and smoked meats).

Certain medications contain nitrates or nitrites. These include topical silver nitrate used in burn therapy, antimalarials, nitroglycerine, antidiarrheals, diuretics, antidotes for cyanide and hydrogen sulfide poisoning, and vasodilators used in coronary artery disease therapy.

Persistence and Movement in the Environment

Wastes containing organic nitrogen are decomposed in soil or water by microbial action forming ammonia, which is then oxidized to nitrite and nitrate. Because nitrite is easily oxidized to nitrate, it is primarily nitrate that is found in groundwater and surface waters. Nitrate-containing compounds in the soil are generally soluble and readily migrate with groundwater. Nitrosamines have a short lifespan in ambient air.

Exposure

Food is the most significant source of nitrates for adults, but water may play a more important role for infants. The nitrosamines most commonly reported in foods are dimethylnitrosamine, diethylnitrosamine, nitrosoproline and nitrosopyrrolidine. Vegetables (cauliflower, spinach, collard greens, broccoli, and root vegetables) account for more than 70 percent of the nitrates ingested in the human diet. The remainder of nitrate in a typical diet comes from drinking water (21 percent) and meat and meat products (6 percent). Nitrate levels in Canadian municipal wells are generally less than 5 mg/L. Nitrate levels in well water are often higher than those in surface water supplies. For bottle-fed infants (< 4 months old), the major source of nitrate exposure is drinking water from rural domestic wells used to dilute formula. Nitrites are produced naturally in the saliva, and it is estimated that 75 percent of a person's intake of these substances originates from this source. Nitrosamines can also be found in some alcoholic beverages and in tobacco products.

It has been estimated that the daily intake of nitrates for an adult Canadian is 51 mg (44.3 mg from food and 6.8 mg from drinking water containing nitrate at a concentration of 4.5 mg/L) (Choi 1985). At drinking water concentrations greater than 30 mg/L (consumption of 1.5 L water/day), the total daily intake is estimated to be 99 mg (44.3 mg from food and 45 mg from drinking water). For bottle-fed infants, the daily intake is estimated to be 2.7 mg (consumption of 0.6 L/day at a concentration of 4.5 mg/L).

Nitrates are metabolized to inorganic nitrites in the liver, which are then excreted in the urine (60 to 70 percent), and saliva (25 percent, which is potentially reabsorbed). Nitrates can be absorbed from the gut, and under acidic conditions (e.g., in the presence of stomach acid) nitrates can be converted to nitrite.

Reducing Exposure

Ensure that drinking water concentrations follow the recommended guidelines, especially that used for infants. To avert infant exposure, new mothers whose drinking water is from a private well should consider using an alternative source of water for preparing infant formula if there is any history of nitrate contamination of wells in the area or if any nitrate is detected with testing of well water, even if below guideline levels.

Table 1

MAC AND TARGET LEVEL VALUES FOR NITRITES, NITRATES AND NITROSAMINES

Agency	Focus	Level	Comments
Health Canada	Food	regulated on an input basis for specific foods, as listed in Table XI of the *Food and Drugs Act* and Regulations[1]	
Health Canada	Drinking water	45.0 mg/L for nitrate that is equivalent to 10.0 mg/L nitrate as nitrogen.	MAC
Health Canada	Drinking water	Where nitrate and nitrite are determined separately, nitrite should not exceed 3.2 mg/L	
Health Canada	Barley malt and malt beverages	1 µg/L (ppb)	target level
OMOEE	Drinking water	10.0 mg/L (nitrate as nitrogen)[2] 1.0 mg/L (nitrite as nitrogen)[2] 10.0 mg/L (nitrate and nitrite as nitrogen)[2] 9 ng/L (dimethylnitrosamine)	MAC MAC MAC MAC
WHO	Drinking water	The sum of the ratios of the concentrations of each to its respective guideline value should not exceed 1. 50 mg/L (nitrate as NO_3^-) 3 mg/L (nitrite as NO_2^-)	guideline value

MAC: Maximum acceptable concentration
OMOEE: Ontario Ministry of Environment and Energy
WHO: World Health Organization

1. The *Food and Drugs Act* and Regulations, Departmental Consolidation of the *Food and Drugs Act* and of the Food and Drug Regulations, with Amendments to December 15, 1993. Issued by National Health and Welfare.
2. Where nitrate and nitrite are present, the total of the two should not exceed 10 mg/L. Samples can be tested for nitrates at many private labs usually at a cost of $15 to $30 per sample. Tap water should be run for five minutes before taking the sample and the sample should be kept out of direct sunlight in a cool place.

Human Health Considerations

Nitrite is toxic because it can combine with hemoglobin to form methemoglobin, which is incapable of carrying oxygen. Methemoglobinemia is the most important adverse health effect caused by excessive nitrate or nitrite exposure. Pregnant women may be more sensitive to the induction of clinical methemoglobinemia by nitrites or nitrates at or near the 30th week of pregnancy. Infants less than three months of age are more susceptible to methemoglobinemia than older infants. Some methemoglobin-inducing agents can also cause Heinz body hemolytic anemia or sulfhemoglobinemia. Severe methemoglobinemia can result in hypotension, shock, cardiac arrhythmias and metabolic acidosis. A chocolate-brown cyanosis is one of the hallmarks of methemoglobinemia.

Elevated nitrate contamination is linked to increased occurrence of methemoglobinaemia ("blue baby") syndrome among infants less than four months of age. There is little evidence that breast-fed infants develop methemoglobinemia from exposure to nitrates ingested by the nursing mother.

Nitrates can react with amines and amides to form nitrosamines, which have been reported to cause cancer in animals. Conclusive evidence that nitrate or nitrite ingestion causes carcinogenic or teratogenic effects has not been found in humans. Therefore, the International Agency for Research on Cancer (IARC) has classified nitrate/nitrite as possibly carcinogenic to humans, although the weight of evidence is weak.

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Nitrogen Oxides (NO_x)

Origin and Uses

Nitrogen oxides (NO_x) are a group of seven compounds composed of nitrogen and oxygen. The most important of these are nitric oxide (NO), nitrogen dioxide (NO_2) and nitrous oxide (N_2O). Nitric oxide and nitrogen dioxide are air pollutants. Nitrous oxide is not generally considered to be an air pollutant, but it is an ozone depleting gas, and also contributes significantly to visibility impairment in the form of plumes and hazes.

Most of the nitrogen oxides in our environment come from natural rather than anthropogenic sources. Natural sources of NO_x are lightning, and biological and non-biological processes in soil; they also descend from the stratosphere. The major sources of NO_2 from human activity are transportation, stationary source fuel combustion, various industrial processes, solid waste disposal and forest fires. Important indoor sources of NO_x are gas stoves, unvented space heaters, kerosene heaters, wood stoves, and tobacco products. In Canada, about 94 percent of NO_x results from vehicles, industry and home heating. While catalytic converters have helped decrease individual vehicular emissions, the increased numbers of vehicles has contributed to a substantial increase in NO_x from 1970 to 1985.

Persistence and Movement in the Environment

Transport of reactive NO_x occurs in regional air masses where they can be transported for many hundreds of kilometres. NO and NO_2 are minimally soluble in water. About one third of the emissions of NO_x in the United States are estimated to be removed by wet deposition. Canadian data are not available.

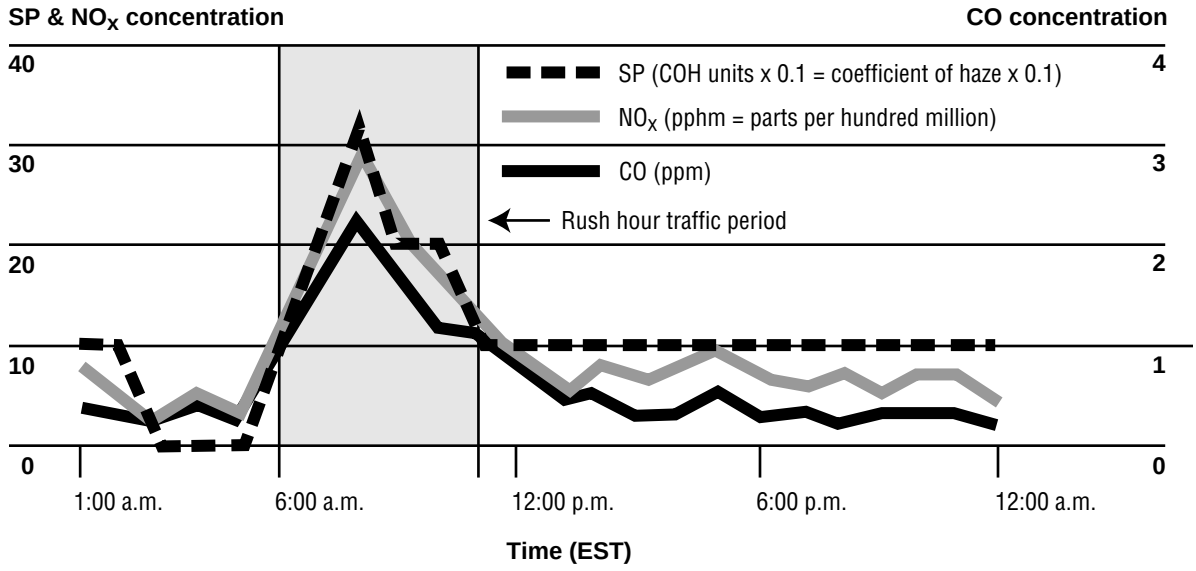
Exposure

On average, the highest NO_2 concentrations for urban and rural sites occur in the late afternoon and evening hours (17:00 to 22:00 hours). The pattern of NO_2 concentration in urban areas is generally characterized by two daily peaks, which are related to motor vehicle traffic patterns in the morning and afternoon. For areas with low vehicular traffic, there is little variation in NO_2 levels throughout the day unless there is atmospheric transport of NO_2 into that region. For daily variation of vehicle related NO_x in downtown Toronto, see Figure 1: Daily variation of vehicle related pollutants in downtown Toronto.

In addition to inhalation of outdoor air, people can be exposed to nitrogen dioxide in their homes. Homes with gas appliances often have indoor NO_2 levels exceeding those encountered outdoors. Gas appliances (gas range/oven, water heater) and unvented kerosene and gas space heaters are an important source of indoor exposure. Average indoor concentrations in bedrooms range from 20 to 120 $\mu g/m^3$ (0.010–0.064 ppm) in some homes with gas ranges. Homes with gas ranges with pilot lights have higher NO_2 levels than homes with gas ranges without pilot lights.

Figure 1

DAILY VARIATION OF VEHICLE RELATED POLLUTANTS IN DOWNTOWN TORONTO



Note: Bay/Grosvenor monitoring site, April 22, 1992
Source: Ontario Ministry of Environment and Energy. *Air Quality in Ontario — A Review of the State of Air Quality*. 1992.

Reducing Exposure

Particularly on hot, humid summer days exposure can be reduced by decreasing emissions of air pollutants and by changing outdoor activities. This can be done by using public transportation instead of cars and avoiding the use of other gasoline-powered vehicles or equipment (motorbikes, motorboats, gas lawnmowers). Exercise increases the total uptake of NO_2 in the respiratory tract. Avoid outdoor aerobic exercise during afternoon and early evening when levels are their highest or choose indoor activities during these times.

Gas stoves and kerosene heaters in the home and in tents should have adequate ventilation.

Human Health Considerations

Nitric oxide (NO) and nitrogen dioxide (NO_2) are the most important of the NO_x in terms of potential adverse health effects. They can exist in the atmosphere in significant concentrations and are quite chemically reactive. It has not yet been shown that concentrations in ambient air of NO_x other than NO_2 have any significant biological activity (NO_2 has a greater impact on human health than does NO). A large fraction of inhaled NO_2 is removed in the respiratory tract. Many factors will influence the amount of NO_2 that is absorbed, for example, more NO_2 will be absorbed in the upper respiratory tract during nasal breathing than during oral breathing. Little is known about NO absorption in the respiratory tract.

People with asthma and chronic obstructive pulmonary disease (emphysema and chronic bronchitis), children and the elderly may be at increased risk to the health effects of NO_2. There have been both positive and negative findings at various levels of NO_2 exposure. Results are inconsistent for effects on lung function, both

in healthy individuals and in those with respiratory diseases. The lowest concentration that results in observed effects on airway responsivity is 376–940 µg/m³ (0.2–0.5 ppm) in exercising asthmatics and minimum levels of 2822–3763 µg/m³ (1.5–2 ppm) in healthy individuals. Studies involving two hour exposures to NO_2 (1881, 4703, 9407, and 14110 µg/m³; 1, 2.5, 5 and 7.5 ppm) indicate a change in total respiratory resistance occurring at 4703 µg/m³ (2.5 ppm), although the effects are quite small. Some studies suggest that an increased rate of respiratory illness in young children is associated with gas stoves in their homes (compared with electric stoves) and with outdoor exposure.

Animal studies have shown that exposure to about 940 µg/m³ (500 ppb) nitrogen dioxide for four hours reduces host resistance to bacterial and viral infection. Alterations in pulmonary function and in morphological and biochemical parameters also occur.

Vitamin C and E are antioxidants and may protect against effects induced by NO_2 exposure. In some studies, people who did not receive Vitamin C prior to NO_2 exposure showed an increased airway responsiveness. Fatal pulmonary edema and bronchopneumonia have been reported at extremely high concentrations. Exposure to nitrogen dioxide also causes visual and olfactory disturbances of uncertain medical significance at levels between 188 and 376 µg/m³ (100 and 200 ppb) for a few minutes.

Table 1

National Ambient Air Quality Objectives for nitrogen oxides

Objective	Duration	
	1-hour	Annual
Maximum Acceptable Concentration	400 µg/m³ (210 ppb)	100 µg/m³ (50 ppb)

In Canada, objectives have been set for nitrogen dioxide (NO_2) in air under the *National Ambient Air Quality Objectives*.

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Particulate Matter

Origin

Particulate matter consists of a variety of minute solid or liquid particles that remain suspended in the air and can be inhaled into the respiratory system. The terms particulate matter, particulates, particles and aerosols are used interchangeably. Particulate matter can be found in both indoor and outdoor air. Common types of particulate matter include dust, fumes, smoke, mist, fog, smog, and haze.

These particles vary in size, shape, and chemical composition and are a vehicle for a variety of substances because many compounds adsorb onto their surfaces. Particles can range in size from 0.005 to 100 microns (µm) where one micron is equal to one millionth of a metre (or 1/1000 of a millimetre). Particles larger than about 10 µm are large enough to settle to a nearby surface after being emitted from a source. Anything smaller can remain suspended in the air for long periods and constitutes a "suspended particulate matter." Particles in the atmosphere have a distinctive bimodal distribution in terms of size and composition. The fine particle fraction (less than 2.5 µm) is usually acidic and the coarse fraction (2.5–10 µm) is basic, due to the different origin of the two size fractions. The fine fraction is made up mainly of particles such as sulphates that are formed from gases while the coarse fraction is formed from the break up of larger particles originating from wind blown dust.

Particles originate both from natural sources and anthropogenic sources. While there have always been particles in the air from natural sources, in more recent times industrial activity has added to the measured levels of particulate matter. The principal sources of particulate matter released into the air, as a result of human activity, are:

- industrial processes;
- fuel combustion;
- transportation; and
- solid wastes.

Industrial operations such as power stations, smelters, mills, refineries, factories, solid waste incinerators and construction activities can release very fine particles containing a wide range of compounds into the air. Agriculture, landfills, and exhaust from motor vehicles, aircraft and marine vessels are also sources of particulate matter. Particulates can contain such compounds as acid mist, arsenic, beryllium, cadmium, lime, mercury, and silica.

Natural sources of particles released into the air are:

- volcanoes;
- wind erosion of soil and rock;
- forest fires; and
- plants.

The particles released from natural sources include dust, ash, smoke, and pollens from plants.

The principal sources of particulate matter in indoor air are:

- cooking, especially when using wood, coal, oil or gas as fuel;
- cleaning activities such as vacuuming and dusting;
- heating, ventilation and air conditioning systems;
- use of consumer products such as spray disinfectants, cleaners and repellents;
- unvented clothes dryers;
- tobacco smoke; and
- particles carried into the dwelling from outdoor air.

Persistence and Movement in the Environment

Particulate matter is dispersed in the air and can be carried long distances by the wind. Particles eventually settle out of the air and are deposited on surfaces such as soil, water and plants. Some contaminants (such as metals) that are adsorbed onto the surface of particles can be taken up by plants through the soil or directly by the plants or may enter the food chain via other pathways. In itself, a particle may not be persistent, but the origin and composition of the contaminants adsorbed on its surface will determine its persistence.

Exposure

Inhalation of particulate matter is the route of exposure most important to human health. Overall, exposure is dependent on the amount of particulate matter in the air, the length of exposure and breathing rate or the amount of air inhaled. Breathing rate itself is related to age, health status and level of physical activity. For example, breathing rate can increase more than tenfold during exercise.

People generally spend more than 80 percent of their time indoors and indoor levels of air particles can be higher than the outdoor levels. People living near sources such as heavily travelled roadways, paved and unpaved roads, incinerators, petroleum refineries, metal foundries, farms, quarries, large construction sites or surface mines are often exposed to relatively high levels of dust and other particulate matter. Particulate matter accounts for about 10 percent of the material emitted into the air as a result of human activity. However, individual localities may vary widely from this figure. For example, downwind from a steel mill, particulate matter would account for a greater percentage of the total pollution than suggested here.

Reducing Exposure

Filters on furnaces and air conditioning systems should be properly installed, cleaned and/or replaced regularly. The use of aerosol products indoors should be avoided. Proper ventilation of clothes dryers should also be ensured. Distilled or deionized water should be used in ultrasonic humidifiers to minimize particle formation. Tobacco smoke should be eliminated in buildings.

Human Health Considerations

From the perspective of human health, the particle size may be the most important characteristic; the smaller the particle, the greater the chance for penetration into the lungs. Particulate matter smaller than about 10 microns, known as PM_{10}, can enter deep into the airways where there is greatest potential for health effects. The chemical composition of the particle will also determine its effect on health. For example, particles containing sulphates or nitrates are acidic and affect lung function differently than chemically neutral particles.

There seem to be three ways that particulate matter is harmful:

- some of the particles themselves may be toxic or have toxic elements adsorbed onto the particle;
- too many particles within a short time can overwhelm the ability of the lung to clear itself of unwanted substances; and
- the particles may interact with other pollutants in the air and act synergistically, that is, the effect of two or more contaminants acting together may be greater than the sum of effects attributable to each contaminant.

The principal organ affected by particulates seems to be the lung. Particulates deposited in the respiratory system appear to affect pulmonary function and to aggravate existing heart and lung disease. The presence of particles may also impede the natural ability of the respiratory system to clear itself of foreign matter through

mucociliary action and may affect other body defence mechanisms. A recent study has shown a strong association between premature mortality due to respiratory disease and airborne particulates (among other air pollutants).

As an example of the health effects associated with particulate matter, the health effects resulting from exposure to wood smoke are presented in the box below.

Health Effects from Exposure to Particulates in Wood Smoke

- Decreased level of lung function in children
- Increased respiratory symptoms in children and adults
- Increased functional limitations as reflected by school absenteeism and restricted activity days
- Increased physician and emergency visits for asthma
- Increased hospitalization for respiratory conditions
- Increased mortality on days after those with high ambient particulate levels

Source: Greater Vancouver Regional District Air Quality Management Plan: Working Paper — Fine Particulate and Visibility. Greater Vancouver Regional District, April 13, 1994.

Inhalation of particulates may also increase the risk of developing respiratory tract cancers because of mutagenic and carcinogenic chemical compounds either in the particulates or adsorbed onto their surfaces. Particles containing polycyclic aromatic hydrocarbons (PAHs), nitrosamines and nitroaromatics have been linked to respiratory cancer in some epidemiological studies. Please see the Contaminant Profiles of these compounds for more information.

The elderly, those with chronic pulmonary or cardiovascular disease, and the very young appear to be the most vulnerable segments of the population to particulate air pollution. Asthmatics, smokers and people with the flu or bronchitis may also be at particular risk when particulate levels are high.

In Canada, objectives have been set for total suspended particulate matter (TSP) in air under the *National Ambient Air Quality Objectives*.

Ambient air quality objectives for PM_{10} and $PM_{2.5}$ will be recommended in 1997 following completion of the scientific review, risk and benefit analysis. The Province of British Columbia has established an interim 24-hour PM_{10} standard of 50 µg/m^3. Particulate matter in air is measured using samplers with pumps drawing measured volumes of air through filters that trap the particles. The filters are then weighed to obtain the mass of the filtered particles. Different filters are used to measure the mass of particles of different sizes. The particulate matter concentrations are usually expressed in micrograms per cubic metre (µg/m^3).

Table 1

National Ambient Air Quality Objectives for particulate matter

Objective	Duration	
	24-hour	Annual
Maximum Acceptable Concentration	120 µg/m^3	70 µg/m^3

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Perchloroethylene (Perc)/PCE/Tetrachloroethylene

Origin and Uses

Perchloroethylene, also referred to as perc, PCE, 1,1,2,2-tetrachloroethylene, tetrachloroethene, ethylene tetrachloride, carbon dichloride, carbon bichloride and perchloroethylene, is a nonflammable, nonviscous liquid. There are no known natural sources of perchloroethylene; entry into the environment results solely from anthropogenic sources. Perc is released into the environment during its production and use. Releases have also occurred from discharge of industrial and municipal liquid effluents and in leachate from some landfill sites.

While it is no longer produced in Canada, perc is imported into the country for use in the dry-cleaning industry and in the production of chlorofluorocarbons (CFCs). Perc is also used in smaller quantities in the finishing and processing of textiles, the manufacture of paint removers and printing inks, in the formulation of adhesives and specialized cleaning fluids, and for aerosols and dye carriers. Perchloroethylene can be present in certain household products including automobile cleaners, suede protectors, paint removers and strippers, water repellents, silicone lubricants, belt lubricants and dressings, specialized aerosol cleaners, ignition wire dryers, fabric finishers, spot removers, adhesives and wood cleaners (see Chapter 10. "Home Environments" for more information on household products).

Persistence and Movement in the Environment

The majority of perc in Canadian commerce is expected to enter the environment, primarily through the atmosphere. This is because perc is volatile and its uses are dispersive and do not result in its transformation or destruction. Perc can also be discharged into aquatic systems where it remains in solution, forms coalesced droplets on the bottom, or volatilizes into the atmosphere. In surface water, volatilization of perc is considered the dominant fate. Levels of perc in groundwater can be considerably higher than in surface water, because the rates of volatilization and biodegradation are greatly reduced. In anaerobic (low oxygen) groundwater, perc may degrade to more toxic compounds, including vinyl chloride (see Vinyl Chloride Contaminant Profile.)

Perc can adsorb onto soils although the amount depends on the partition coefficient, the organic carbon content of the soil, the type of release, and the concentration of perc in the liquid phase. Perc is expected to be mobile in most soils and can penetrate to depths where groundwater can be contaminated.

Exposure

Perchloroethylene is widespread in the environment and has been detected in trace amounts in outdoor and indoor air, surface and groundwater, drinking water, sediment, and biota, in various regions of Canada. The highest environmental levels are found in the commercial dry-cleaning and metal-degreasing industries. Emissions can sometimes lead to high concentrations in groundwater. In a Health Canada study, which investigated perc levels in groundwater, 113 of 836 sites tested contained perc. This suggests that 13.5 percent of all water supplies in Canada are tainted with perc.

Signs of perc use in dry-cleaning practices include clothes that come back from the dry cleaner smelling of a pungently sweet chemical, the chemicals can be smelled when entering the store, a one-hour service is available, headaches are experienced after wearing newly dry-cleaned clothing, and skin irritation or sensitivity is noticed after wearing clothes that have been recently dry cleaned.

From Table 2, it is evident that the time spent indoors makes the greatest contribution to the overall exposure (approximately 80 percent), while the ingestion of drinking water generally makes a minor contribution (1 percent). However, when perc is present in higher concentrations in drinking water, it can contribute more to total intake through ingestion of water, and indirectly through dermal and inhalation exposure. The use of household products containing this substance, as well as residual perc present on recently dry-cleaned clothes are likely to be responsible for the greater levels observed in indoor air compared with general ambient levels.

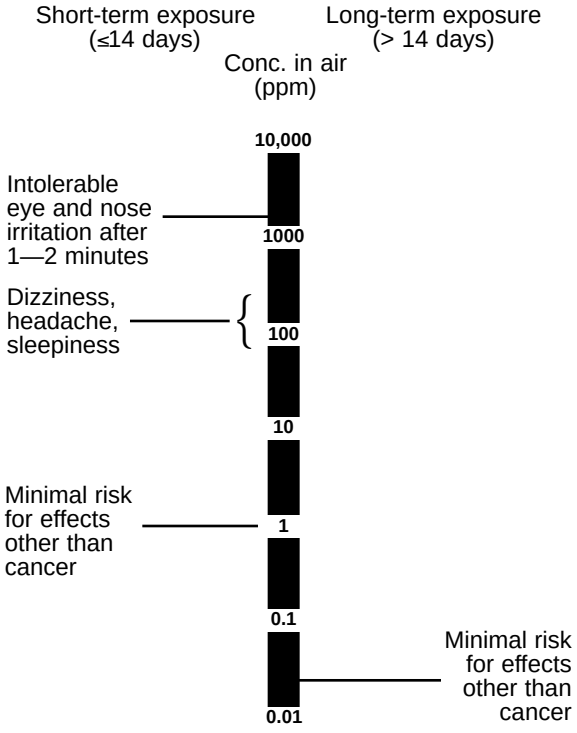
Perchloroethylene has been shown to be readily and rapidly absorbed by inhalation and ingestion in humans and in laboratory animals. Absorption through the skin is slow and limited. The estimated average daily intakes of perchloroethylene for the general population in Canada are 13- to 28-fold lower than the TDI of 34 µg/kg•bw/day, derived from inhalation studies in laboratory animals. Even though it is widespread in the environment, perc does not appear to be at levels that pose a danger to the health of most Canadians.

Reducing Exposure

Water-based cleaning is an alternative to solvent-based cleaning. Machines can be adjusted for exact temperatures, degree of agitation and length of drying cycles. While the potential effects from the detergents used in this process have not been fully assessed, many people believe this to be a safer and effective alternative to dry-cleaning. Individuals concerned with the effects of perc residues on their clothing after dry-cleaning may want to consider wet-cleaning if it is available in their community.

Figure 1

HUMAN HEALTH EFFECTS FROM BREATHING TETRACHLOROETHYLENE



Adapted from: U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR). *Public Health Statements – What you need to know about toxic substances commonly found at Superfund hazardous waste sites.* Atlanta, Georgia.

Other preventive measures that can be taken by sensitive individuals include limiting the amount of "dry clean only" clothing purchased and hanging newly dry-cleaned clothes outside or in a separate room for a few days to ensure the perc is removed.

Products containing perchloroethylene should be handled with care and all safety and precautionary instructions on the label should be followed closely.

Human Health Considerations

Exposure to 200 ppm or more of perc for prolonged periods of time can induce headaches, dizziness, nausea, and eye and skin irritation. At higher exposures, these reactions can intensify and unconsciousness or even death can occur in extreme cases. High levels have also been correlated with damage to the liver, the central nervous system and even death. Perc is also moderately toxic if ingested. As the concentration of perc increases, the time until onset of effects shortens, and the severity of the effects increases. Levels inside most dry cleaners are generally no more than 30 ppm, which is below the level at which acute effects can be observed.

Neurobehavioural changes have also been correlated with long-term exposure. These include deficits in visual/spatial function and cognitive flexibility, changes in mood, and clinical and preclinical effects on frontal lobe and limbic functions.

Study results of effects on reproduction and development are somewhat conflicting. An increased risk of spontaneous abortion has been noted in some but not all studies. Occupational exposure has not been associated with an increased risk of birth defects or significant alterations in the quality of sperm. There have been reports of an association between idiopathic infertility in females and exposure to dry-cleaning chemicals, although exposure was likely to many chemicals rather than to perc alone.

There have been numerous studies that have examined the potential for increased risk of certain cancers and exposure to perc. Cancers that have been studied include liver, pancreatic, cervical, bladder, kidney, lung and respiratory system, genital, esophageal as well as lymphosarcoma, multiple myeloma and non-Hodgkin's lymphoma. There is little evidence to support an association between perc and any specific type of cancer. As such, the available information to assess human carcinogenicity has been determined as being inadequate. Based on the animal evidence, perchloroethylene has been classified by the *Canadian Environmental Protection Act* (CEPA) as possibly carcinogenic. However, the International Agency for Research on Cancer (IARC) recently re-classified perc as probably carcinogenic to humans.

Table 1

GUIDELINES FOR TETRACHLOROETHYLENE

Agency	Focus	Level	Comments
Health Canada	Drinking water	0.03 mg/L	MAC
WHO	Drinking water	0.04 mg/L	guideline value

MAC: Maximum acceptable concentration
WHO: World Health Organization

Table 2

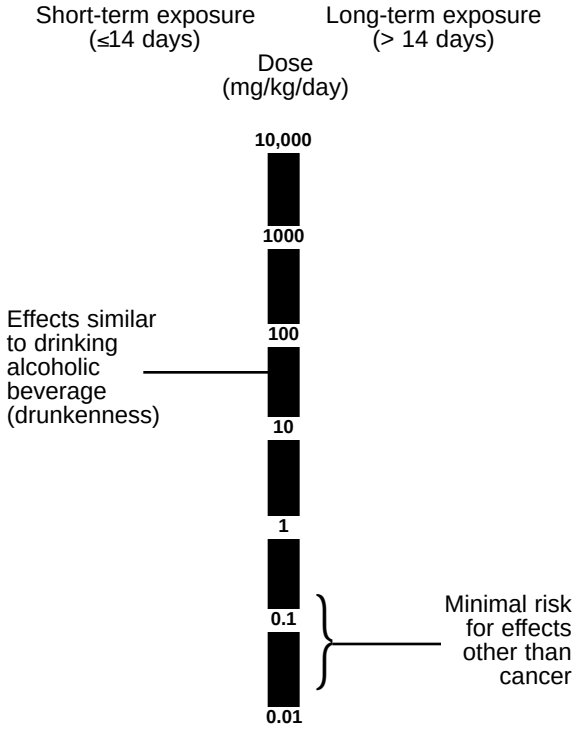
ESTIMATED DAILY INTAKE OF TETRACHLOROETHYLENE (G/KG•BW/DAY) BY THE CANADIAN POPULATION

Route of Exposure	0–6 mo.	7 mo. – 4 yr.	5 – 11 yr.	12 – 19 yr.	20+ yr.
Ambient Air	0.01–0.24	0.01–0.32	0.01–0.37	0.01–0.31	0.01–0.27
Indoor Air	1.21	1.63	1.88	1.56	1.40
Total Air	1.22–1.45	1.64–1.95	1.89–2.25	1.57–1.87	1.41–1.67
Drinking Water	–	0.006–0.06	0.003–0.03	0.002–0.02	0.002–0.02
Food	–	0.65	0.39	0.20	0.12
Total Intake	1.22–1.45	2.30–2.66	2.28–2.67	1.77–2.09	1.53–1.81

Source: CEPA Assessment Report, 1993.

Figure 2

HUMAN HEALTH EFFECTS FROM INGESTING TETRACHLOROETHYLENE



Adapted from: U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR). *Public Health Statements – What you need to know about toxic substances commonly found at Superfund hazardous waste sites.* Atlanta, Georgia.

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Phthalates

Origin and Uses

Phthalates are a group of chemical compounds, three of which will be included in this section: bis(2-ethylhexyl) phthalate, also known as di(2-ethylhexyl) or DEHP; di-*n*-octyle phthalate (D*n*OP); and dibutyl phthalate. Although it is not certain if these chemicals occur naturally, it is unlikely that natural sources present a significant contribution in comparison to anthropogenic sources. Release of phthalates directly to the atmosphere, primarily as a result of their manufacture and their industrial uses, is believed to be the most important mode of entry to the environment. Small amounts are also released through the incomplete combustion of plastic materials.

Phthalates are used as plasticizers in such products as gloves, flooring and flexible sheets. DEHP is the most important phthalate plasticizer used in Canada, accounting for well over 50 percent of total use of phthalate plasticizers. Additional uses of dibutyl phthalate include adhesives, coatings (including lacquers), cosmetics (perfume solvent and fixative), suspension agents for solids in aerosols, lubricants for aerosol valves, antifoamers, and skin emollients. At present there are two DEHP manufacturing facilities in Canada, and it has also been imported from the United States. DnOP and dibutyl phthalate are not produced in Canada, but are imported for use.

Persistence and Movement in the Environment

Phthalates are rapidly removed from the atmosphere by photo-oxidation. They are present in low concentrations in the atmosphere, and are not expected to contribute significantly to the formation of ground-level ozone, global warming, or to the depletion of stratospheric ozone.

There is not sufficient information to conclude whether DEHP is entering or may enter the environment in a quantity or concentration or under conditions that are having a harmful effect on the environment. More than 50 percent of DEHP in the atmosphere occurs in the vapour phase. It is removed from the atmosphere through washout by precipitation and dry deposition. DEHP bioaccumulates in aquatic organisms, but biomagnification through the aquatic food chain does not likely occur. Uptake by plants is very low. No information on bioaccumulation in wild animals has been identified.

It has been concluded that D*n*OP and dibutyl phthalate are not entering the environment in a quantity or concentration or under conditions that are having a harmful effect on the environment. D*n*OP is not persistent in air or surface water, but it may accumulate in sediment under anaerobic conditions. No measured bioconcentration factors have been identified. Dibutyl phthalate is not expected to be persistent in air and water, but may be more persistent in sediments and soil. It is biodegradable in surface waters. Dibutyl phthalate has been detected in aquatic organisms and in the egg yolks of the double-crested cormorant. It is uncertain if it is bioaccumulated in aquatic organisms. No information is available on the bioaccumulation of dibutyl phthalate in wild mammals.

Exposure

DEHP

The major exposure pathway for the general population is from the ingestion of food. Exposure by inhalation of indoor air, and the ingestion of drinking water are considerably less than that from food. In addition, intake in ambient air and soil are estimated to be relatively small. Dermal absorption is poor. Once absorbed in the body, phthalates are widely distributed, with no apparent accumulation.

For adults, the estimated daily intake of DEHP for the general population in Canada is 5.78–5.82 µg/kg•bw/day. Infants and toddlers may have a higher total intake as a result of exposure from children's products containing DEHP (pacifiers, nipples, flexible toys). The total average intake, including the estimated intake from children's products, is 8.9–20.6 µg/kg•bw/day for infants (aged 0–0.5 years) and 18.9–23.1 µg/kg•bw/day for children (0.5–4 years). It should be noted that these estimates are based on very limited data. In addition, the intake of DEHP from food is likely to be underestimated as concentrations of zero were assumed for foodstuffs in which DEHP was not detected. Therefore, average daily intakes of DEHP for some age groups of the general population in Canada may approach or slightly exceed the Tolerable daily intake (see Table 1).

DnOP and dibutyl phthalate

There is insufficient information to conclude whether D*n*OP is entering the environment in quantities considered harmful to human health. It has been concluded by the *Canadian Environmental Protection Act* (CEPA) that dibutyl phthalate is not entering the environment in a quantity considered harmful to the environment or human health. On occasion D*n*OP has been detected in industrial effluents and in sewage sludge in Canada. There is very limited information on surface water and sediment concentrations of D*n*OP. Levels in surface waters of D*n*OP and dibutyl phthalate were well below the chronic effects threshold set for the most sensitive aquatic species. Data on D*n*OP levels in air, rain, soil or biota are not available. Exposure of the general population in Canada to D*n*OP cannot be estimated as adequate data are not available. D*n*OP has not been detected in food and drinking water samples.

Dibutyl phthalate has been detected in air, surface water and groundwater, sediment, biota, sewage sludge and waste effluents. In order of their relative importance, the principal media of exposure to dibutyl phthalate are food, indoor air, drinking water, soil and ambient air. The estimated daily intake of dibutyl phthalate for the general population in Canada ranges from 1.9–5.0 µg/kg•bw/day. Due to current limitations in the available data, it is not possible to estimate intakes on the basis of concentrations in all media. Instead, rather less representative ranges have been used for soil and indoor air. Dibutyl phthalate has been detected in drinking water at concentrations of 1.0 µg/L (the detection limit).

Reducing Exposure

There are no known steps for reducing phthalate exposure. Although food is the primary exposure pathway to phthalates, the estimated daily intakes are based on limited data.

Human Health Considerations

DEHP

It has been concluded by CEPA that DEHP may enter the Canadian environment in a quantity that may constitute a danger to human health. Data on blood, nervous system and pulmonary function from studies of chronically exposed populations through occupation are limited and inconclusive. Effects in humans of DEHP on carcinogenicity, reproductive or developmental effects have not been identified. These effects have been reported in laboratory animals.

DnOP

There are insufficient data to conclude whether D*n*OP is entering the environment in a quantity that constitutes a danger to human life or health. The human health effects of D*n*OP are not known; the data are inadequate to estimate exposure of the general population. Occupational exposures include a case report of irritation of the eye and upper respiratory track and poorly documented studies indicating neurological and reproductive effects. Poorly documented clinical studies indicate skin irritation and sensitization on dermal contact. Available data are inadequate to assess the carcinogenicity of D*n*OP in experimental animals.

Dibutyl phthalate

It has been concluded by CEPA that dibutyl phthalate is not entering the environment in a quantity that constitutes a danger to human life or health. Studies on occupational exposure to dibutyl phthalate have been conducted but are not considered adequate as a basis for assessment of neurotoxic and reproductive effects. Dibutyl phthalate is unclassifiable with respect to its carcinogenicity to humans.

The Guidelines for Canadian Drinking Water Quality (1996) do not give a numerical guideline for phthalic acid esters (PAE) as they are not likely to occur in drinking water at levels that present a health risk. The available data for D*n*OP are considered inadequate to calculate a tolerable daily intake (TDI).

Table 1

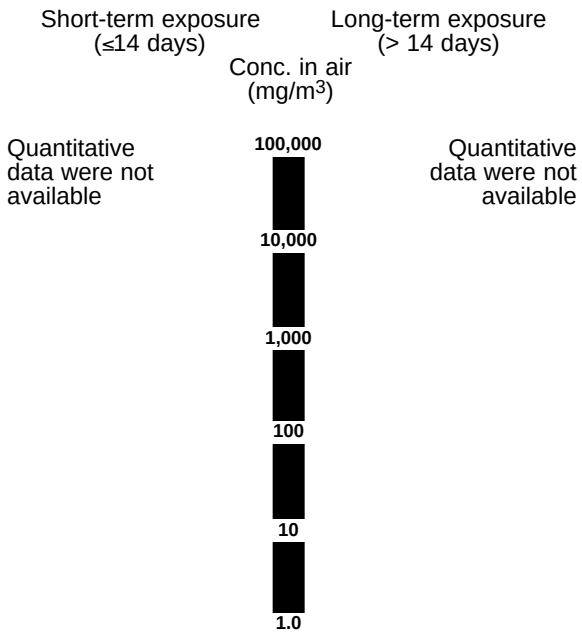
TDIs FOR PHTHALATES

Agency	Focus	Level	Comments
Health Canada	All sources	DEHP: 44 µg/kg•bw/day	TDI
Health Canada	All sources	dibutyl phthalate: 63 µg/kg•bw/day	TDI

TDI: Tolerable daily intake

Figure 1

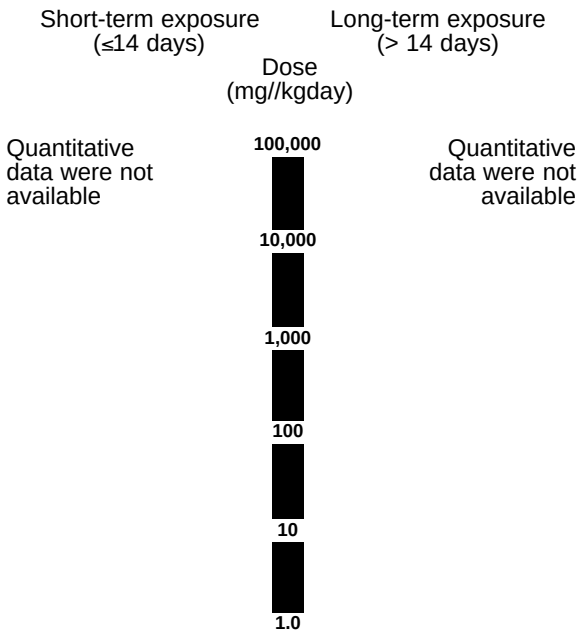
HUMAN HEALTH EFFECTS FROM BREATHING DI(2-ETHYLHEXYL)PHTHALATE



Adapted from: U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR). *Public Health Statements – What you need to know about toxic substances commonly found at Superfund hazardous waste sites.* Atlanta, Georgia.

Figure 2

HUMAN HEALTH EFFECTS FROM INGESTING DI(2-ETHYLHEXYL)PHTHALATE



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Polychlorinated Biphenyls (PCBs)

Origin and Uses

Polychlorinated biphenyls (PCBs) were first used in the 1930s for industrial purposes. They do not occur naturally. PCBs are a family of 209 compounds (referred to as congeners) with similar structures. The most common PCB trade name is Aroclor (produced by Monsanto Chemicals). Numbered compounds of Aroclor (i.e., Aroclor 1260) designate the PCB mix by percentage of chlorination. The first two numbers represent the type of molecule (12=chlorinated biphenyl) and the last two numbers give the weight percentage of chlorine (i.e., the 1260 chlorinated biphenyl mixture has an average chlorine content of 60 percent).

PCBs are chemically stable, unreactive and have high electrical resistivity. They have been used in capacitors and transformers, hydraulic fluids, adhesives, plasticizers, heat transfer fluids, wax extenders, lubricants, cutting oils and flame retardants.

Concerns over the environmental effects of PCBs led to a North American ban in 1977 on their manufacture, importation and most non-electrical uses and also to restrictions on their use in existing electrical and mechanical equipment. Despite these actions, PCBs are still in use today from application previous to their restriction. PCBs are released to the environment from improper disposal practices and accidental releases. Properly done, high-temperature incineration destroys PCBs at an efficiency of 99.99 percent without producing toxic by-products. It is this method that the Government of Canada has chosen to eliminate PCBs. In Ontario, PCBs are considered a registerable waste and their storage, transportation and disposal is restricted.

Persistence and Movement in the Environment

Depending on their degree and pattern of chlorination, PCBs biodegrade at different rates. They are fat soluble and bioaccumulate in the fatty tissue of aquatic and terrestrial organisms. They are biomagnified through the food chain, becoming available for human consumption. PCBs are transported through water and soil, and occasionally through the air when waste materials containing PCBs are burned.

Exposure

Human exposure to PCBs is primarily through the ingestion of food, mostly from contaminated fish. Depending on the age of the fish and the lake of origin, concentrations in the range of 0.8-5 mg/kg wet weight are observed (whole lake trout from Lake Ontario, 1988). As a result of restrictions on its use and manufacture, levels of PCBs have declined 10-fold in many fish species in the Great Lakes since the 1970s. Traces of PCBs have been found in milk, meat, and eggs. The estimated daily intake of PCBs from the diet for the average Canadian is less than 1 microgram or 0.017 µg/kg•bw/day for a 60 kg person.

PCB levels in human milk (0.5–1.5 mg/kg fat basis), serum (2–5 µg/L, mean values) and adipose tissues (0.4 µg/g) in Canadians are comparable to those found in other developed countries. Levels in people living in the Great Lakes basin are about the same as in residents elsewhere in Canada. Mothers consuming large amounts of contaminated fish, seafood, marine mammals and sea-bird eggs (e.g., Aboriginal peoples) may have significantly higher PCB levels in their blood and breast milk. Studies of the levels of chlorinated hydrocarbon residues (including PCBs) in human fat tissue in Canada show a significant downward trend from 1969 to 1985. This trend began in 1977 when PCB use was restricted.

PCBs are rarely detected in Ontario drinking water supplies. A 1986 study by the Ontario Ministry of the Environment found no PCBs in drinking water (detection level of 20 ng/L). PCB exposure through inhalation of air is not a significant exposure route.

Reducing Exposure

Fish advisory guidelines and any recommendations provided by the provincial, territorial or federal health departments should be followed. Health professionals advise that the known benefits of breast feeding outweigh the potential risk that may be associated with PCBs in human milk.

Human Health Considerations

All PCB congeners are not equally toxic. The actual effect depends on the dose and type of PCB. It is unlikely that PCBs initiate cancer, but that they act as promoters once growth has begun. The International Agency for Research on Cancer (IARC) has classified PCBs as probably carcinogenic to humans. There is limited evidence for carcinogenicity in humans, but sufficient evidence in animals to link long-term high level exposure to highly chlorinated commercial PCB mixtures and an increased incidence of cancer, particularly liver cancer.

There have been two major incidents of consumption of rice oil contaminated with a mixture of PCBs and polychlorinated dibenzofurans in Japan and Taiwan. The human health effects observed from these two events included changes to the skin (chloracne, pigmentation), the eye (hypersecretion, abnormal pigmentation, conjectiva, narrowed field of vision), the mucosal surfaces (irritative effects), the liver (fatty changes, abnormal liver function), and the peripheral nervous system (peripheral neuropathy). General malaise, fatigue, irritation of mucous membranes, respiratory problems, and lowered resistance to infections have been reported. In both these cases the primary contributor was thought to be the dibenzofuran.

Effects on behavioural functions and responses in neonates and cognitive deficits in infants are suspected for prenatal exposures corresponding to serum levels < 15 µg/L in fish-eating mothers. The latter are the only effects that have been associated with PCB exposure through fish consumption. In chronic low-dose environmental exposure by ingestion, an increase in blood pressure has been reported. However it is not possible to conclude that PCBs are the causal agent since other factors were not taken into consideration (e.g., exposure to other chemicals). There is no evidence that PCB levels found in breast milk have caused adverse effects in nursing infants.

Table 1

GUIDELINE, PTDI AND IMAC VALUES FOR PCBS

Agency	Focus	Level	Comments
Health Canada	Meat, beef, fat basis, Dairy products, fat basis	0.2 µg/g	guideline
Health Canada	Whole egg, less shell	0.1 µg/g	guideline
Health Canada	Poultry, fat basis	0.5 µg/g	guideline
Health Canada	Fish, edible portion	2.0 µg/g	guideline
Health Canada	All sources	1.0 µg/kg•bw/day	PTDI
OMOEE	Drinking water	3 µg/L	IMAC
OMOEE	Fish flesh	< 2 mg/kg: edible portion, unrestricted consumption > 2 mg/kg: edible portion, occasional consumption (adults), no consumption (children, pregnant women)	
U.S. FDA	Fish Action Level	2 µg/g	

IMAC: Interim maximum acceptable concentration
PTDI: Provisional tolerable daily intake
OMOEE: Ontario Ministry of Environment and Energy
U.S. FDA: United States Food and Drug Administration

Table 2

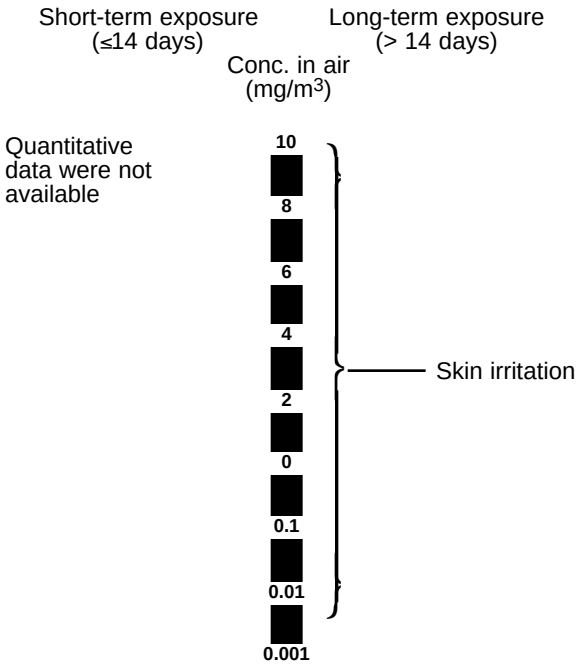
HEALTH PROTECTION BRANCH "GUIDANCE VALUES" FOR BLOOD, 1986.

	Women of reproductive age (µg/L)	Men and post-menopausal women (µg/L)
Tolerable	< 5	< 20
Concern	5–100	20 – < 100
Action	100	100

Source: Health Protection Branch, Foods Directorate memorandum, 1986.

Figure 1

HUMAN HEALTH EFFECTS FROM BREATHING PCBs



Adapted from: U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR). *Public Health Statements – What you need to know about toxic substances commonly found at Superfund hazardous waste sites.* Atlanta, Georgia.

Figure 2

HUMAN HEALTH EFFECTS FROM INGESTING PCBs

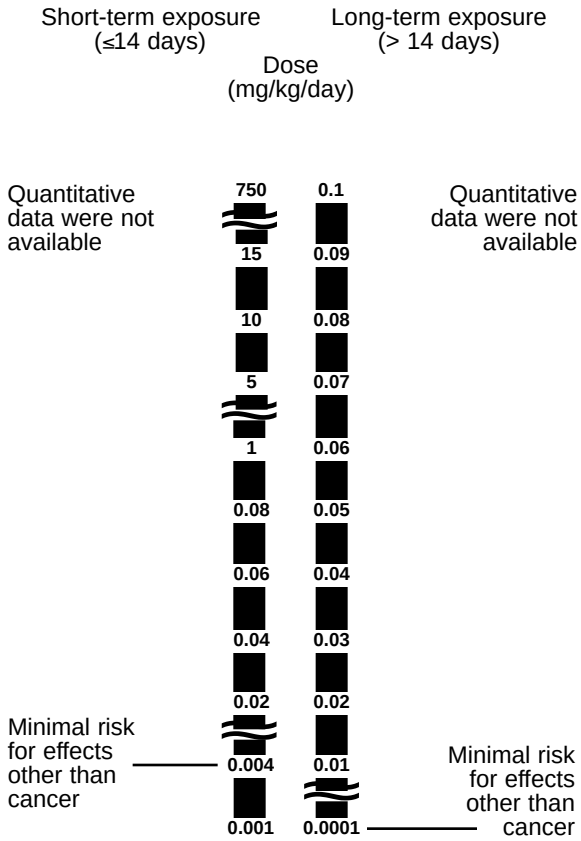


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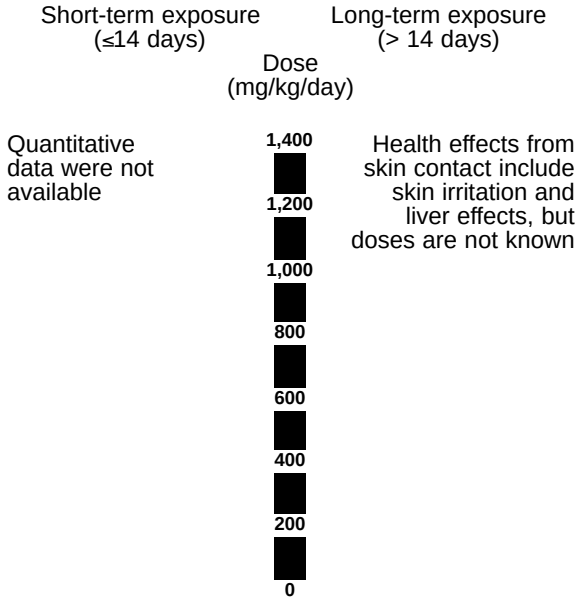
Figure 3

HUMAN HEALTH EFFECTS FROM SKIN CONTACT WITH PCBs



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Polycyclic Aromatic Hydrocarbons (PAHs): Benzo[a]pyrene and related compounds

Origin and Uses

PAHs are organic compounds, and are also known as polynuclear aromatics (PNAs). Common PAHs include benzo[a]pyrene (B[a]P), benzo[e]pyrene, benzo[a]anthracene, chrysene, pyrene, benzo[k]fluoranthene, benzo[g,h,i]perylene, coronene, dibenz[a,h]anthracene, and dibenz[a,h]acridine. These chemicals often occur together in the environment, and many have similar toxicological effects and environmental fate.

B[a]P is manufactured in very small amounts for commercial use. Natural sources of PAHs include forest fires, volcanoes and fossil fuels. PAHs are formed from fossil fuels through pyrolysis, a process in which material is chemically changed at a high temperature, while the oxygen supply is restricted. Sources from human activity include the incomplete combustion of fossil fuels, organic matter and garbage; the industrial production of many petroleum products, such as creosote, asphalt; cigarette smoke; and vehicle exhaust. These sources release greater volumes of PAH than do the natural sources. Wood burning in residential homes is the largest anthropogenic source of PAHs.

Persistence and Movement in the Environment

PAHs are ubiquitous in the environment and they may be transported over great distances in the air. PAHs are bioaccumulated in aquatic organisms. Bioconcentration factors of 100 to 2000 have been found in fish and crustaceans. These aquatic organisms are exposed to PAHs through the ingestion of water, sediment, and food. Biomagnification has not been reported, as organisms (with the exception of mussels and other invertebrates) can rapidly eliminate PAHs. Some terrestrial plants can take up PAHs through their roots (from the soil) or leaves (from the atmosphere).

Exposure

The greatest sources of exposure to PAHs for the general population are inhalation of tobacco smoke (active or passive), wood smoke, and contaminated air, and the ingestion of PAHs through food. The most significant influence on indoor PAH levels is environmental tobacco smoke. Drinking water and accidental ingestion of soil are minor sources of exposure. It is estimated that drinking water accounts for a negligible amount of the total B[a]P ingested (0.1 to 0.3 percent), while food accounts for 99 percent. The total daily potential exposure of carcinogenic PAHs for adult males is estimated to be 3 µg (median), and may be as high as 15 µg. For smokers, exposure levels may be twice as high. For nonsmokers, food is the main source of B[a]P exposure (90 percent of total), with air a less important source (5 percent of total).

Populations living near waste sites containing PAHs or living near industries involved in PAH production, may be exposed through contact with the air, water, and soil.

PAH concentration in food depends on both the method of preparation and the origin of the food. Barbecuing foods increases their PAH concentration. Smoked and cooked fish and meats are higher in PAHs than uncooked products. Consumption of Great Lakes fish is not expected to contribute a significant amount to PAH intake, unless the fish are smoked (Environment Canada 1991). Other foods containing PAHs include roasted peanuts and coffee, and refined vegetable oil. Leafy vegetables and crops such as wheat, rye, and lentils may contain small amounts of PAHs from atmospheric deposition; the contribution to the total daily exposure is negligible in most cases.

Dermal exposure to PAHs may occur in certain occupations (e.g., the use of coal-tar shampoos in hairdressing) and as a result of swimming or surfing in severely contaminated water. Otherwise, the extent of exposure via dermal absorption is small.

Reducing Exposure

The following steps may be taken to reduce PAH intake in foods:

- reduce excessive consumption of foods high in PAH concentration (smoked foods made with liquid smoke have almost no PAHs);
- the lower the fat content of foods, the lower the PAH concentration;
- cook with as low a heat as possible; and
- cook with the heat source above (i.e., not BBQ). There are fewer PAHs in oven cooked and fried foods. PAHs result when fat drips on coals and is pyrolyzed, releasing PAHs in the smoke.

Given the sources of PAH exposure, reduction may also be achieved through a decreased inhalation of tobacco smoke, and properly manufactured, installed and maintained wood stoves (to ensure more complete combustion).

Human Health Considerations

Benzo[a]pyrene is a well known carcinogen in animals. It is classified by the International Agency for Research on Cancer (IARC) as possibly carcinogenic to humans (adequate data does not exist). Lung cancer following inhalation and skin cancer following dermal exposure have been reported in occupationally exposed workers. Additional effects reported from occupational exposure to PAHs have been observed from exposure to fuels and other petroleum products (e.g., truck drivers and mechanics) and tars (e.g., coke oven workers and roofers). Effects include chronic bronchitis, dermatitis, cutaneous photosensitization, and pilosebaceous reactions. Animal studies have shown that PAHs are metabolized primarily in the liver and kidney, and are excreted in bile, feces and urine.

Table 1

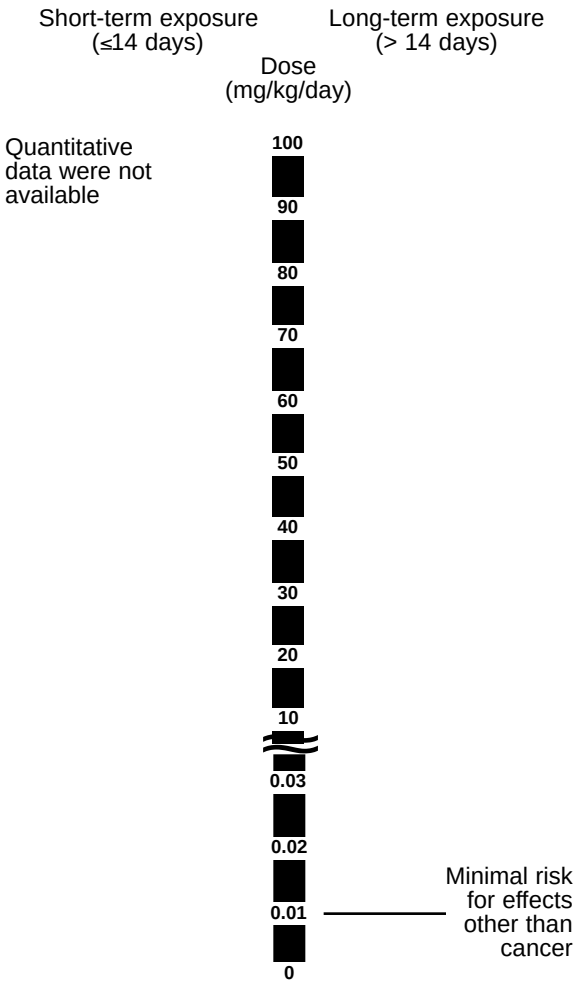
MAC AND GUIDELINE VALUES FOR B[A]P

Agency	Focus	Level	Comments
Health Canada	Drinking water	0.01 µg/L	MAC. B[a]P is the only PAH for which there is sufficient toxicological evidence to allow the setting of a guideline.
OMOEE	Drinking water	0.01 µg/L	MAC
WHO	Drinking water	0.7 µg/L	Guideline value; this concentration corresponds to an excess lifetime cancer risk of 10^{-5} for stomach tumours.

MAC: Maximum acceptable concentration
OMOEE: Ontario Ministry of Environment and Energy
WHO: World Health Organization

Figure 1

HUMAN HEALTH EFFECTS FROM INGESTING BENZO[A]PYRENE



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Radon

Origin and Uses

Radon-222 is a colourless, odourless, chemically inert gas that is naturally radioactive. It is present in the environment as a result of the radioactive decay of radium-226, an element commonly found in rock and soil, and occurring naturally from the decay of uranium-238. Radon itself decays to produce daughters or progeny that are emitters of energetic alpha particles. Elevated concentrations of radon in air can be found where soils and rocks contain high levels of uranium, granite, shale or phosphate. It may also be found in soils contaminated with certain types of industrial waste, such as the by-products of uranium or phosphate mining. The tailings of uranium processing (mine and mill wastes) contain radium-226 and will continue to emit radon over thousands of years. In the past, radon has been produced commercially for use in radiation therapy, and by research laboratories and universities for experimental studies.

Movement in the Environment

When formed, radon can remain below the soil surface, it can enter groundwater, or it can move to the soil surface and enter the atmosphere. Radon is rarely found in surface water, as it is rapidly released to the air when water reaches surface levels. Because it is inert, radon does not bioaccumulate in plants, aquatic organisms or animals. Uranium-238 has a very long half-life, thus radium-226 and radon will continue to exist indefinitely at about the same levels as they do at present.

When radon gas enters the atmosphere, it is dispersed in the air, and concentrations outdoors are low. When the gas enters a dwelling, however, the concentration can build up because it is not dispersed.

Exposure

Radon decays with a half-life of 3.8 days. Because it is chemically inert, most radon that is inhaled is then exhaled. However, the immediate decay products of radon-222 are radionuclides with short half-lives (less than 30 minutes) that attach themselves to dust particles in air. Exposure to radon occurs primarily through inhalation in air. When inhaled, the radionuclides are retained in the lung and respiratory tract. The principal dose to the lung, and the major portion of the total background radiation, comes from the breakdown of these short-lived radionuclides, rather than the decay of radon itself. Factors that influence the radiation dose received by the lungs include the amount and characteristics of inhaled air (for example, the amount of dust), the person's breathing pattern, and the architecture and biological characteristics of the lungs.

In open air, radon gas is so diluted that it is not considered a health hazard. The average level of radon in outdoor air is about 10 Bq/m^3. However in confined spaces, such as basements, radon gas and its progeny can accumulate, causing an indoor air problem. Levels of radon are not considered a serious problem in most Canadian homes. Studies indicate that less than 0.1 percent of all homes in Canada could have levels of radon sufficiently high to warrant remedial action. Indoor radon levels typically range from about 30–100 Bq/m^3, with an average concentration of about 45 Bq/m^3. Average levels of indoor radon in Canadian cities range from 5 Bq/m^3 in Vancouver to 57 Bq/m^3 in Winnipeg.

Radon gas can enter a building by diffusion from soil and building materials, but pressure-driven flow is a more important mechanism. During much of the year, the air pressure inside the home is lower than in the soil surrounding the foundation as a result of exhaust fans and by rising warm air created by fireplaces, clothes dryers, and furnaces. This difference in pressure draws air and other gases in the soil, including radon, into the home.

Radon gas can move through small spaces in the soil and rock on which a house is built. Entry into a home occurs through dirt floors, cracks in concrete walls and floors, sumps, joints, basement drains, under the furnace base, and jack posts if the base is buried in the floor. Concrete-block walls are particularly porous to radon. New ventilation standards and construction guidelines should reduce radon exposure. Local geography alone is an inadequate predictor of concentrations, due to other determinants of indoor radon levels.

Intake of radon from drinking water through both ingestion and inhalation is generally less than that from ambient and indoor air. Radon gas and its progeny can be inhaled when released from water used in the home for showers or dishwashers, however, this source generally contributes only 1 to 2 percent to the mean indoor concentration. Exposure is more likely through ingestion of drinking water from private wells or small communal water systems. Large municipal treatment plants provide better aeration of water to release the gas. In general, higher levels can be expected in areas where rock containing elevated concentrations of the uranium or radium is in contact with water.

Cigarette smoking can also be a direct source of long-lived radon decay products. Cigarette smoke is a small contribution to the total radon dose. Radon can deposit its radioactive decay products on growing tobacco leaves. Polonium-210, a radon decay product, has been found in inhaled cigarette smoke and in lung tissue at autopsy, but is a small contribution to the total radon dose.

Reducing Exposure

Increasing ventilation will decrease the exposure to radon in homes. This can be done through renovations to existing basement floors (especially Earth floors), sub-floor ventilation, or sealing cracks and openings. Radon levels cannot be predicted; they must be measured.

There is no regulation in Canada that governs what is deemed to be an acceptable level of radon in a home. Health Canada has adopted the following guideline for radon in Canadian homes, as established through the Federal/Provincial Committee on Environmental and Occupational Health (CEOH):

It is recommended that remedial measures be taken where the level of radon in a home is found to exceed 800 Bq/m^3 *as the annual average concentration in the normal living area. Because there is some risk at any level of radon exposure, homeowners may wish to reduce levels of radon as low as practicable.*

Human Health Considerations

Radon causes no symptoms of irritation or discomfort and there are no early signs of exposure. Human skin is thick enough not to be affected at the concentrations found in air, but bronchial and lung tissue is not. If a person breathes radon decay products, there is some risk that the alpha radiation may damage lung tissue and cause cancer. Observations of miners over the last few centuries and rigorous international studies over the last few decades have shown a relationship between radon exposure and an increased risk of lung cancer. It is generally many years before cancer develops. More recent studies in Canada have failed to establish such a link at radon levels normally found in homes. Two recent studies in Finland and the United States have confirmed these findings, whereas a study in Sweden has shown a small increase in risk.

The risk of lung cancer due to radon exposure is thought to be second only to that of smoking; however, the actual risk from smoking is very much greater than that from radon exposure. Exposure to both radon and tobacco smoke may further increase the risk of lung cancer. Comparison of cancer rates in smoking and non-smoking uranium miners indicate that smoking promotes earlier development of lung cancers. **Not smoking is the most effective way to reduce the risk of lung cancer.** The health effects of radon in drinking water are not known.

Table 1

HUMAN HEALTH EFFECTS FROM BREATHING RADON

Short-term exposure (less than or equal to 14 days)		
Levels in air (ppm)	**Length of exposure**	**Description of effects**
		The health effects resulting from short-term exposure of humans to air containing specific levels of radon are not known
Long-term exposure (greater than 14 days)		
Levels in air (pCi/L)	**Length of exposure**	**Description of effects***
100	Occupational (10 years)	Severe lung damage

* These effects are listed at the lowest level at which they were first observed. They may also be seen at higher levels.

Adapted from: U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR). *Public Health Statements – What you need to know about toxic substances commonly found at Superfund hazardous waste sites.* Atlanta, Georgia.

Table 2

MAC AND OTHER VALUES FOR URANIUM, RADIUM, CESIUM, IODINE AND STRONTIUM

Agency	Focus	Level	Comments
Health Canada	Residential areas average	annual uranium level should not exceed 800 *Bq*/m^3	
Health Canada	Drinking water	10 *Bq*/L (cesium-137)	MAC
		6 *Bq*/L (iodine-131)	MAC
		0.6 *Bq*/L (radium-226)	MAC
		5 *Bq*/L (strontium-90)	MAC
		0.10 mg/L (uranium)[1]	MAC
		radon[2]	MAC
OMOEE	Drinking water	50 *Bq*/L (cesium-137)	MAC
		10 *Bq*/L (iodine-131)	MAC
		1 *Bq*/L (radium-226)	MAC
		10 *Bq*/L (strontium-90)	MAC
		0.10 *Bq*/L (uranium)	MAC
WHO	Drinking water	10 *Bq*/L (cesium-137)	guideline value
		6 *Bq*/L (iodine-131)	guideline value
		1 *Bq*/L (radium-226)	guideline value
		5 *Bq*/L (strontium-90)	guideline value

MAC: Maximum acceptable concentration
OMOEE: Ontario Ministry of Environment and Energy
WHO: World Health Organization

1. The guideline for uranium is being reviewed for possible revision owing to new data.
2. The Health Canada *Guidelines for Drinking Water Quality* (1996) state "that there is no need to establish a maximum allowable concentration (MAC) for radon in drinking water. However, anyone whose indoor air radon concentrations exceeds acceptable levels (800 *Bq*/m^3 as an annual average concentration in the normal living area) should investigate the possibility that their groundwater also contains high levels of radon. Individuals who attempt to remove radon from their water supply using point-of-use devices containing activated carbon should be cautioned regarding the difficulties of disposing of the used radioactive carbon."

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Salmonella spp.

Origin

There are approximately 2000 known serotypes of *Salmonella* bacteria, although only about 50 are responsible for causing the majority of illness in man and animals. All salmonellae species are pathogenic to humans; varying, however, in their infectious dose. *S. typhimurium* is the most frequently isolated and reported serotype from both humans and animals in many countries.

Over 96 percent of the human form of salmonellosis (the illness caused by infection of salmonellae) comes from animals consumed by humans. Salmonellae are primarily found in the intestinal tract of mammals (including humans, and domestic and wild animals), birds, reptiles and occasionally insects.

In the developed parts of the world, the salmonellae carrier rate is rarely higher than 0.3 percent, with food animals being the primary carriers. Salmonellae occur worldwide; their distribution has been facilitated by the shipment of animal products and feeds.

Persistence and Movement in the Environment

Salmonellae can survive drying and freezing for long periods. Infection is easily spread in animals on the farm, during transport to the market and in the slaughter house.

Salmonellosis is a disease that may be naturally transmitted between man and animals. Salmonellae excreted in the feces can directly contaminate foods and water and can also be transmitted by insects and other animals to food, water and other sources. If these polluted sources are consumed by man or other animals, the organisms will again be excreted in the feces, thus continuing the cycle of infection.

Exposure

Ingestion of contaminated foods (and water) and abuse of time/temperature factors are the most common contributing factors to human salmonellosis. The organisms are passed in the feces of an infected individual and then ingested indirectly by another person through the medium of food. It is necessary to ingest viable *Salmonella* bacteria, thus salmonellosis is designated as a food infection. Foods that carry salmonellae are mostly of animal (especially poultry) origin. These include fresh meats and poultry, eggs and foods made with eggs (ice cream, custard-filled pies), milk and milk products (fresh milk, fermented milks and cheese).

Poor personal hygiene and sanitation in people handling food is often cited as a factor in the transmission of *Salmonella*.

Reducing Exposure

To avoid exposure to salmonellae, food should not come into contact with sources carrying the organism (e.g., humans exhibiting clinical symptoms, animals) and ingredients (e.g., eggs — fresh infected eggs or uncooked eggs). People handling food must maintain appropriate personal hygiene, although it is recognized that food handlers are usually the victim of, rather than the source of a food-borne outbreak.

The growth of salmonellae in foods can easily be prevented. Heat from cooking and pasteurization (for milk and egg products) will destroy salmonellae in food. Foods should not be held between 4 °C and 60 °C, except during short periods of food preparation; during heating and cooling these temperatures should be traversed as quickly as possible.

Human Health Considerations

There are three distinct clinical types of disease syndromes. Two of them are the enteric fevers, typhoid and paratyphoid fever, caused by *S. typhi* and *S. paratyphi*, respectively. These diseases produce fever and affect the intestines; typhoid may produce constipation rather than diarrhea. Symptoms for both diseases may include anorexia, an enlarged spleen and rose coloured spots on the body. In most developed countries, enteric fever is uncommon but can occur sporadically.

The third type of disease syndrome is gastroenteritis (salmonellosis). For food-borne infections the incubation period is usually from 12-36 hours (range 6-72 hours). The effects are diarrhea, abdominal pain, vomiting and fever. If further infection occurs, the illness may persist for weeks or months and it may result in death. Children and older persons may be especially affected.

Salmonellae are one of the major causes of bacterial gastroenteritis in developed countries. Many cases of salmonellae are mild, even unapparent and do not require medical care. There are an estimated 600 000 cases of salmonellae poisonings each year in Canada (Health and Welfare Canada 1992). Most are unreported as the symptoms resemble the common flu.

Guidelines and other Values for *Salmonella*

The *Guidelines for Canadian Recreational Water Quality* do not set a limit for *Salmonella* concentration in recreational waters. It is recommended that it be used as a parameter to assist in interpreting the results of sanitary and microbiological surveys. In ready-to-eat foods, *Salmonella* is unacceptable at any concentration.

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Smog and Ground-Level Ozone

Origin and Uses

The ozone found in the atmosphere results primarily from chemical reactions between nitrogen oxides and volatile organic compounds (VOCs) in the presence of sunlight. Normally in unpolluted air, nitric oxide combines with ozone, creating more oxygen and nitrogen dioxide. However in polluted air, nitric oxide combines with VOCs. This creates a buildup of ozone at ground level. VOCs and nitric oxides (NO_x) are produced through natural processes and by incomplete fuel combustion from industries, homes and vehicles (see Contaminant Profile for Nitrogen Oxides). Increases in anthropogenic emissions of the pollutants that contribute to ozone formation have resulted in an increase in smog levels in recent years. Elevated concentrations of ground-level ozone may be found in both urban and rural areas as a result of long range atmospheric transport.

Ozone mixes with other pollutants to form a brownish-yellow haze or "smog." Although the term originally meant the presence of smoke and fog, it is now used to describe the "chemical soup" that occurs as haze over urban, suburban and rural areas. Ground-level ozone often occurs at higher levels in some rural areas of southern Ontario compared with urban areas. Smog can also contain particulates, which act as a transport medium for other compounds such as acids, metals, benzene, and dioxins. Summer smog is primarily composed of oxidants (ozone), whereas winter smog is primarily particulates, often sulphates.

Persistence and Movement in the Environment

Light winds and stagnant air masses influence the degree of smog in an area. Under these conditions locally produced pollutants are not dispersed.

Exposure

In the stratosphere, ozone acts as a protective layer filtering out harmful UV radiation. Ground-level ozone, however, is hazardous to human health when inhaled. Exposure to smog and ground-level ozone occurs through inhalation. Those who work or exercise outdoors are at greatest risk because of the increased volume of air inhaled during these activities. Individuals who may be more susceptible to the health effects of ground-level ozone include the very young and old, and people with pre-existing cardiac disease or respiratory problems.

Indoor concentrations of ozone are almost always substantially lower than those outdoors due to efficient scavenging by indoor surfaces and the lack of indoor sources. The only common indoor sources are copying machines and electrostatic air cleaners. Since most people spend more than 80 percent of their time indoors, their ozone exposures are much lower than estimates based on outdoor concentrations.

Diurnal patterns are strongly influenced by atmospheric conditions and by location. At night, ozone concentrations are generally lower than daytime values. Rural areas with little urban influence have smooth diurnal cycles with minimum ozone concentrations between 06:00-08:00 hours and maximum concentrations between 14:00-16:00 hours. Annual averages in rural locations are greater than in areas under considerable urban influence. Areas under urban influence have a more pronounced cycle in which minimum ozone concentrations occur immediately after sunrise (06:00 hours) and maximum concentrations occur during mid-afternoon (15:00 hours).

In Canada, high concentrations of ozone occur in the Windsor–Quebec corridor, the Lower Fraser Valley and regions of the southern Maritimes. The region most frequently and seriously affected by elevated ozone concentrations is southwestern Ontario, where the guideline of 160 µg/m^3 (0.08 ppm) can be exceeded on up to 25 percent of summer days. Rural sites in southwestern Ontario have greater frequencies of high ozone concentrations than do urban sites in Toronto, Montreal or Vancouver. In urban areas, vehicles emit NO_x which can actually remove ozone from the air, whereas sources of NO_x are more limited in rural areas.

Reducing Exposure

Particularly on hot, humid summer days exposure to ground-level ozone can be decreased by reducing emissions of air pollutants and by changing outdoor activities. Outdoor aerobic exercise should be avoided during the afternoon and early evening when ozone levels are at their highest. In the long term, ground-level ozone can be reduced by using public transportation instead of cars and avoiding the use of other gasoline-powered vehicles and equipment (motorbikes, motorboats, gas lawnmowers).

Human Health Considerations

The main concern with respect to the health effects of smog is the damage it can cause to affect the respiratory system. Because smog is a mixture, the possible health effects can vary. In some cases, the health effects of one pollutant may be intensified when combined with another. A person may also react more strongly to a specific pollutant if exposed to another pollutant first (i.e., by being "sensitized").

Health effects associated with ground-level ozone

Short-term exposure (one to two hours in a controlled laboratory) can irritate the eyes, nose and throat, and can produce respiratory problems such as coughing and painful deep breathing. Similar health effects have been associated with outdoor activity.

The effects of low-level, long-term exposure to ground-level ozone are not as well known but are currently under investigation. Research indicates that ozone exposure appears to decrease the ability of the lungs to ward off disease. For example, ground-level ozone increases the susceptibility of asthmatics to common allergens. As well, people with respiratory problems may suffer more symptoms during periods of high ozone levels. The number of daily hospital admissions for respiratory problems increases significantly when ozone levels are high as well as when sulphate levels are high (see *Contaminant Profile* for Sulphur Dioxide). Five percent of Ontario hospital admissions from May to August are associated with high ozone levels.

The results of animal and population-based studies indicate that there may be an accelerated aging of the lung associated with living in communities with persistently elevated ambient ozone. Associations have been found between premature mortality due to respiratory disease and cardiovascular deaths and several pollutants including ground-level ozone. Two to four percent of excess respiratory deaths are accounted for by pollution. However, because it is not possible to accurately assign an exposure level in the human population studies, such data is interpreted very cautiously.

In chronic animal exposure studies, a small percentage of inhaled ozone was shown to penetrate deep into the lungs damaging some of the alveoli (the individual air sacs where oxygen and carbon dioxide are exchanged). After years of exposure, small lesions in the lungs of experimental animals were shown to result in connective tissue changes (e.g., scar tissue formation deep in the lung).

Other components of smog and their health effects

Although other components of smog occur in small quantities compared with ozone, they affect peoples health nonetheless. These other components include nitrogen oxides (NO_x), sulphur oxides and sulphates, volatile organic compounds (VOCs), peroxyacetyl nitrate (PAN), particulate matter and carbon monoxide. The health effects of these pollutants, with the exception of PAN, are discussed individually in this document. PAN, which is derived from motor vehicle exhaust and aldehydes (e.g., formaldehyde), a class of volatile organic compound, are linked with the eye irritation that is commonly associated with smog.

Table 1

NATIONAL AMBIENT AIR QUALITY OBJECTIVES FOR GROUND-LEVEL OZONE

Objective	Duration	
	1-hour	Annual
Maximum Acceptable Concentration	160 µg/m^3 (82 ppb)	30 µg/m^3 (15 ppb)

In Canada, objectives have been set for ground level ozone in air under the *National Ambient Air Quality Objectives*.

In Ontario an Air Quality Advisory is triggered as a forecast to an episode of smog. An episode is defined when **levels** are forecast to exceed 80 ppb (160 µg/m^3) ozone for a 24-hour period or greater than 120 ppb (235 µg/m^3) for any one-hour period.

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Sodium Chloride — Road Salt

Origin and Uses

Sodium chloride (NaCl) is a common road deicer (deicing salt is almost 95 percent pure sodium chloride) that has been used since the 1940s. Any chemical deicers used on roads in the winter will eventually end up in the environment at detectable levels. At a cost of $93.6 million, 5.1 million tons of salt were poured onto Canadian highways in 1992 to provide safer driving conditions during the winter months.

Effective chemical alternatives include calcium magnesium acetate (CMA) and sodium formate (NaFo). CMA provides safe winter driving without compromising the roadside environment, water supplies or highway structures. It requires an application rate (by weight) of 1.6 times that of salt, is somewhat slower to take effect and is more expensive (approximately 33 times) to produce. American and Canadian cities have incorporated CMA into their snow and ice control strategies. NaFo has not been tested as extensively as CMA. It is produced as a by-product of other chemical processes, and is more costly (approximately 13 times) than NaCl. Although NaFo releases sodium into the environment, it does not release chloride. NaFo is similar in effectiveness (endurance of effect, temperature range and speed) to NaCl and does not corrode steel.

Persistence and Movement in the Environment

Sodium and chloride move through the soil at different rates. Chloride is highly mobile and is quickly transported through the soil to water supplies. Sodium tends to remain in the soil. Where sodium chloride is the most common salt added to the environment, chloride concentrations are a reliable indicator of the sodium present. There is a seasonal surge of salt pollution that occurs through the winter, intensifies in the spring thaw and gradually decreases through the summer. Deicing salt is implicated in the high concentrations of chlorides, sodium and hardness in well water supplies.

Much of the salt applied to roads enters the soil, reaches groundwater aquifers, and may then seep into surface waters. With respect to surface waters, large or flowing bodies of water (rivers and streams in particular) are not significantly affected by road salt pollution as dilution takes place quickly. Some smaller lakes may show significant effects from salt pollution. The concentration of salts in surface waters, roadside soils and vegetation is proportional to the use of road salts. Sodium and chloride levels decrease with distance from the highway, with highest values occurring closest to the highway. The greater the number of years of salting, the higher the salt concentrations and the greater the area of contamination.

Aquatic biota are affected by salt-related changes: osmotic stress and the release of heavy metals from lake sediments. Saline run-off from highways affects roadside soil (alters pH, decreases soil fertility) and vegetation (dehydration, salt injuries). Terrestrial animals (wildlife and domestic animals) are quite tolerant to salt and increased concentrations in ground and surface waters are not usually a problem. The most serious consequence is the traffic hazard created by salt-hungry animals seeking roadside salt accumulations. Salt also results in corrosion damage to vehicles, bridges and roads.

Exposure

The average daily intake of sodium for an adult is primarily from food (5600 mg/day). The major human health concern with salt pollution of groundwater is the increased sodium levels in drinking water supplies. This, however, has not presented a problem, as sodium and chloride concentrations in drinking water are not considered to be a significant source of exposure. Less than 1 percent of daily intake of sodium comes from water.

Reducing Exposure

A decrease in the consumption of foods high in sodium will significantly reduce exposure. Although chloride concentrations are below the recommended guideline, high concentrations can make drinking water less palatable. Thus, deicing salts should be kept away from water supplies through proper storage and application.

Human Health Considerations

Sodium in drinking water is not a health concern for most people but may be an issue for someone with heart disease, or on a sodium controlled diet. Studies have shown that reducing salt intake will lower blood pressure in people with hypertension, but it cannot be conclusively inferred that increased sodium intake will cause hypertension. Evidence indicates (but does not conclude) that reduction of salt in the diet to below 2 g/day results in the prevention of essential hypertension and its disappearance as a major public health problem.

The symptoms of exposure in this paragraph are cited in Enviro-TIPS (1984). Slight nose irritation and sneezing will occur only when large amounts of fine dust (of sodium chloride salts) are inhaled. Upon ingestion of sodium chloride salts, nausea and vomiting and hypertension have been observed; a gain in body water and hypertoxicity (NRCC 1980); and in infants, excessive amounts cause coma and convulsions which may be persistent due to vascular injury (Dresibach 1980). When concentrated solutions of sodium chloride salts remain in contact with skin, irritation, inflammation and small ulcerations have been observed. The following have been reported from eye contact: mechanical irritation, watering of the eyes, inflammation of conjunctiva.

Table 1

GUIDELINE VALUES FOR SODIUM AND CHLORIDE

Agency	Focus	Level	Comments
Health Canada	Drinking water	≤ 250 mg/L chloride ≤ 200 mg/L sodium	AO * AO *
WHO	Drinking water	250 mg/L chloride 200 mg/L sodium	These are not health-based guideline values; they may affect the taste of drinking water above the specified value.

AO: Aesthetic objective
WHO: World Health Organization

* It is recommended that sodium be included in routine monitoring programs, as levels may be of interest to physicians who wish to prescribe sodium-restricted diets for their patients. Snow disposal sites are not strictly controlled except for disposal directly into water courses, which must be approved by the Ontario Ministry of Environment and Energy.

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Staphylococcus aureus

Origin

The bacteria *Staphylococcus aureus* live in the human nose, throat and skin. They are transient residents of the skin, and live permanently in the sweat glands and in the intestinal tract. *S. aureus* also live in inflamed lesions and skin infections (e.g., boils, pimples, styes).

Persistence and Movement in the Environment

Staphylococcus is not considered to be a natural inhabitant of environmental waters and is usually unable to grow there. It is, however, resistant to many environmental influences and can survive for relatively long periods of time. For example, *S. aureus* can withstand freezing and drying for long periods.

S. aureus leave the nose and mouth in nasal secretions, and during coughing, spitting and sneezing. The presence of staphylococci in recreational waters is considered to be mainly due to discharges from the mouth, nose, and throat of swimmers, as well as from their skin surface. *S. aureus* are also transmitted during actions in which the fingers touch the lips or nose (e.g., smoking, biting fingernails) and during eating as utensils enter the mouth.

Exposure

Sources of exposure include air, dust, cloth materials (e.g., clothing, bedding, handkerchiefs), floors, water, sewage and insects. *S. aureus* usually enter food from a human source, either by the hands or by cross contamination from other foods, utensils, surfaces or equipment previously contaminated by humans. A properly disinfected water supply should be free of *S. aureus*.

S. aureus usually grows in cooked foods such as meat (especially ham), poultry and sea foods that are held at improper temperatures. They are also in prepared foods such as custards, trifles, and cream and milk products. Outbreaks of staphylococcal food poisoning have occurred due to raw milk (from cows and goats), cream and occasionally cheese.

The numbers of *S. aureus* in contaminated food will increase if this food is kept between 4°C and 60°C for extended periods. Such temperature conditions may be found in steam tables in cafeterias and restaurants. Pasteurization, ultra heat treatment and normal cooking do not destroy the *S. aureus* enterotoxin. It is relatively stable to heat, surviving a temperature of 100°C for 30 minutes. *S. aureus* is not normally present in natural waters.

Reducing Exposure

Good hygienic practice will prevent staphylococcal food poisoning. Use ingredients free of staphylococci (e.g., pasteurized milk rather than raw milk) and keep people with staphylococcal infections (e.g., colds, boils, styes) away from food. Foods should be cooled rapidly and adequately refrigerated.

Human Health Considerations

Food poisoning is a common illness. *S. aureus* produce toxins while they are growing and multiplying in food. These toxins are released into the food which, if ingested, can irritate the stomach very quickly. The incubation period for food-borne intoxication is usually two to four hours (range of one to seven hours). The symptoms include severe vomiting, abdominal pain, diarrhea, sweating, headache, prostration and occasionally severe dehydration leading to collapse. The disease is rarely fatal.

Skin infections caused by *S. aureus* are the most common human staphylococcal infections. These include cellulitis, pustules, boils, carbuncles, impetigo, post-operative wound infections of various sites, and infected cuts and scratches. In contaminated recreational waters *S. aureus* can be responsible for ear and skin infections. There does not appear to be a significant relationship between bather illness and concentration of *S. aureus* in the water.

Serious illnesses such as bacteremia, endocarditis, meningitis, pneumonia, and osteomyelitis continue to be seen as both community- and hospital-acquired infections. In the late 1950s and early 1960s, *S. aureus* caused considerable morbidity and mortality in hospitalized patients. The advent and use of penicillin in the intervening years have provided successful therapy of serious *S. aureus* infections.

Toxic shock syndrome has been attributed to infection or colonization with *S. aureus* . Most of the patients have been young, menstruating women using certain types of highly absorbent tampons during menses, however, the toxin(s) associated with the syndrome may also be present at sites other than the genital in non-menstruating women and in men.

Guidelines and other values for *Staphylococcus aureus*

The *Guidelines for Canadian Recreational Water Quality* (Health and Welfare Canada 1992) have not specified a limit for *S. aureus*. Sampling should be carried out when there is evidence (epidemiological or other) of its presence in the water or to assess the hazards of excessive utilization of the water with possible person-to-person transfer of pathogens.

Current food regulations set the maximum acceptable concentration of *S. aureus* in cheese from pasteurized milk at 100 per gram and from unpasteurized milk at 1000 per gram. Current guidelines set maximum acceptable concentrations of *S. aureus* per gram in pasta at 500, frozen cream pies at 100, heat treated fermented sausage at 50, raw fermented sausage at 250, deboned poultry at 100, dry mixes at 100, and soybean products at 100.

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Sulphur Dioxide (SO_2)

Origin and Uses

Sulphur dioxide is a highly soluble gas. Its major sources are industrial, particularly the burning of fossil fuels. Metal smelters, petroleum refining, pulp and paper mills, electrical utilities, incineration and fuel combustion all contribute to SO_2 in the air. Occasionally sulphur containing fuels are burned in the home in unvented appliances. Sources of indoor SO_2 concentrations are poorly vented kerosene space heaters as well as outdoor sources. Sulphur dioxide is produced in nature by volcanoes.

Persistence and Movement in the Environment

SO_2 emissions persist in the atmosphere for days, combining with water molecules to produce sulphuric acid, an acidic aerosol present in acid rain and urban smog. When discharged from tall stacks, most of the SO_2 is carried away by wind currents and is gradually converted into SO_3. The SO_3 rapidly combines with water vapour to produce very fine droplet aerosols of H_2SO_4 (sulphuric acid). The sulphuric acid is then gradually neutralized by ammonia in the atmosphere, to ammonium sulphate, a nearly neutral salt. Rates of ammonia neutralization vary widely, depending on emission rates from their sources. Rates of neutralization are high over cities and agricultural areas, lower over forests and virtually nil over deep water bodies.

Exposure

SO_2, which is highly soluble, is efficiently captured in the upper respiratory tract (nose and mouth cavities, sinuses, trachea) during inhalation. Virtually none of the gas penetrates deep in the lungs during normal, quiescent breathing. However during vigorous physical activity, breathing rates increase so that there is less time for SO_2 to be captured in the upper respiratory tract. In addition, breathing patterns shift from predominantly nasal (i.e., through the nose) to oronasal (i.e., through both the mouth and nose) which is less efficient at capturing SO_2. Consequently, during exercise SO_2 can penetrate deeper into the lungs where it has the most potential for health effects.

Reducing Exposure

Reduce outdoor exercise when high levels are reported. When using indoor heating appliances, low sulphur fuels should be used, and the availability of proper ventilation should be ensured.

Human Health Considerations

In general, levels of sulphur dioxide in ambient air will not produce adverse effects in healthy individuals. Asthmatics are particularly susceptible to the respiratory effects of SO_2. They experience bronchoconstriction from acute exposure to SO_2 at levels 650–1300 $\mu g/m^3$ (0.25–0.50 ppm) and higher while exercising. Exposure concentrations of an order of magnitude higher (greater than ambient levels) are needed to produce similar responses in healthy people.

Other health effects from exposure to SO_2 and related acid aerosols are eye irritation, shortness of breath, and reduction of lung function. Effects are more severe or frequent in persons with underlying lung disease. Significant associations have been found between the number of daily admissions to acute care hospitals in

Ontario for respiratory problems and sulphate levels. While ozone accounted for 5 percent of these hospital admissions (see Contaminant Profile for Smog and Ground-Level Ozone), sulphates accounted for an additional 1 percent of admissions. Results were consistent for all age groups although infants were found to be most affected while the elderly were least affected. The way in which sulphates affect cardio-respiratory health is not understood.

In Canada, objectives have been set for sulphur dioxide levels in air under the *National Ambient Air Quality Objectives*.

Table 1

NATIONAL AMBIENT AIR QUALITY OBJECTIVES FOR SULPHUR DIOXIDE

Objective	Duration		
	1-hour	24-hour	Annual
Maximum Acceptable Concentration	900 µg/m^3 (340 ppb)	300 µg/m^3 (110 ppb)	60 µg/m^3 (20 ppb)

References

Canada. Health Canada, Great Lakes Health Effects Program. "Outdoor Air and Your Health. A summary of research related to the health effects of outdoor air pollution in the Great Lakes basin." March 1996.

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Toxaphene

Origin and Uses

Toxaphene is not a naturally occurring compound. It is a contact insecticide consisting of a complex mixture of over 670 chemicals. It was widely used on cotton crops, cereals, grains, fruits, nuts, oil seeds and vegetables; as a piscicide in fish eradication programs; and on livestock and poultry. Toxaphene use began in 1949. Since 1984, toxaphene has been banned from use in Canada; all maximum residue limits in foods were revoked. Other countries continue to use toxaphene, for example it is used as an insecticide on banana and pineapple crops in Puerto Rico and the Virgin Islands.

Persistence and Movement in the Environment

Toxaphene will continue to exist in the environment for a long time due to its persistent nature. In soil, toxaphene will vaporize to air or stick to soil particles and does not dissolve easily in water. In surface water, toxaphene will vaporize to air or settle in the sediments. Toxaphene can be transported over long distances atmospherically. Toxaphene is an important fish contaminant in the Yukon and the Northwest Territories even though it has never been used in northern Canada. This is a result of long-range transport through the atmosphere to the Canadian North from countries where toxaphene is used.

Toxaphene is bioaccumulated by aquatic organisms and fish-eating birds, and is biomagnified in the food chain. Due to metabolic processes, toxaphene is not bioaccumulated to the same degree as other chlorinated compounds, such as DDT and PCBs.

Exposure

Farmers and applicators who used toxaphene in the past to control insects on livestock and in emergency applications were at risk to high concentrations. People, particularly children living near hazardous waste sites contaminated with toxaphene, may be exposed through ingestion of contaminated water or soil.

Risk to human health is unlikely at current detectable levels. Toxaphene is rarely found in drinking water samples and has been detected in only 0.6 percent of food samples from Canadian cities. The largest route of exposure is through the ingestion of contaminated fish. In the late 1970s, toxaphene levels in some Great Lakes fish ranged from 1.0–10.7 mg/kg with carnivorous fish having the highest concentrations. Levels have since decreased to 1 mg/kg or below. If 114 g of fish containing 1 mg/kg of toxaphene was consumed once per week, then 0.23 µg toxaphene/kg•bw/day would be ingested. This is approximately equal to the PTDI (see Table 2).

Ingestion of toxaphene is more toxic than through dermal exposure. Almost all ingested toxaphene is excreted through the urine and feces within a few weeks. There are no data on toxaphene residues in human tissues, although it has been detected at a concentration of 0.1 mg/kg (milk fat basis) in breast milk samples collected in Uppsala, Sweden.

Reducing Exposure

Populations consuming high levels of fish and seafood should follow any consumption recommendations provided by provincial, territorial or federal governments.

Human Health Considerations

Acute exposure in people from breathing, eating or drinking has been reported to cause damage to the lungs (reversible allergic bronchopneumonia), nervous system (salivation, hyperexcitability, behavioural changes, muscle spasms, convulsive seizures), liver (increase in enzymes), kidneys (elevated blood uric acid and occult blood in the urine) and can result in death. Limited data suggest that chronic low-level exposure may also result in damage to the liver, kidneys, lungs, and nervous system.

There is no conclusive evidence available to link toxaphene exposure with cancer in humans. The International Agency for Research on Cancer (IARC) has classified toxaphene as possibly carcinogenic to humans. Based on animal studies (thyroid and hepatic carcinomas), toxaphene has been classified as a probable human carcinogen by the U.S. EPA.

Table 1

HUMAN HEALTH EFFECTS FROM EATING OR DRINKING TOXAPHENE

Short-term exposure (less than or equal to 14 days)		
Levels in food (ppm)	**Length of exposure**	**Description of effects**
0.18		Minimal risk level (based on animal studies)
Levels in water (ppm)		
		The health effects resulting from short-term exposure of humans to water containing specific levels of toxaphene are not known
Long-term exposure (greater than 14 days)		
Levels in food (ppm)	**Length of exposure**	**Description of effects***
0.002		Minimal risk level (based on animal studies)
Levels in water (ppm)		
		The health effects resulting from long-term exposure of humans to water containing specific levels of toxaphene are not known

* These effects are listed at the lowest level at which they were first observed. They may also be seen at higher levels.

Adapted from: U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR). *Public Health Statements – What you need to know about toxic substances commonly found at Superfund hazardous waste sites.* Atlanta, Georgia.

Table 2

PTDI VALUE FOR TOXAPHENE

Agency	Focus	Level	Comments
Health Canada	All sources	0.2 µg/kg•bw/day	PTDI

PTDI: Provisional tolerable daily intake

The *Guidelines for Canadian Drinking Water Quality* (1996) do not give a numerical guideline for toxaphene.

References

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Trihalomethanes (THMs)

Origin and Uses

Trihalomethanes (THMs) are primarily a by-product from the reaction between chlorine or bromine and naturally occurring organic substances (e.g., plant parts, humus) in raw water. The reaction occurs in water supplies during the disinfection process when organic matter is chlorinated to inactivate bacteria, viruses and some parasites. THMs are released into the environment from industrial sources, as well as from their indirect production during water chlorination.

The most common THMs include chloroform, bromodichloromethane, dibromochloromethane and bromoform. THMs are used as an indicator of the various disinfection by-products of chlorination, therefore they are most commonly found in municipally treated water supplies.

Levels of THMs in municipally treated water depend on several factors: where the water came from (rivers have higher organic content than lakes), the water temperature, the pH, bromide ion concentration, the method used to screen out suspended matter, the season (there tends to be less organic matter in the water sources during the winter and therefore lower levels of chlorination by-products), chlorination dose, and other characteristics of the treatment process.

Chloroform is the THM detected most frequently and at highest concentrations in municipally treated water. Chloroform is the only THM produced commercially in Canada. It is used in the production of refrigerants, plastics and agrochemicals, in the production of aerosol propellants, and as a solvent and degreasing agent. In the past, minor uses of chloroform included cough medicines, cosmetics and fumigants. Chloroform is no longer used in these products. Bromoform is used in the synthesis of pharmaceuticals, as a solvent and in the aircraft and shipbuilding industries. Bromodichloromethane is used as a solvent and in the synthesis of other chemicals. Chlorodibromomethane is an intermediate in the manufacture of refrigerants, pesticides, propellents and other organic chemicals.

Persistence and Movement in the Environment

Volatilization and evaporation into the air is the major removal process of the volatile THMs (such as chloroform) during water chlorination. However, other less volatile THMs may not be removed from water this way. Once in the atmosphere, chloroform may be oxidized. The volatilization half-life, or the amount of time it takes for one half of the THMs in water to evaporate, has been estimated at 3.5 hours for THMs in a shallow river and 44 hours for THMs in a pond. The half-life for THMs in the atmosphere is approximately 80 days. This volatilization process does not occur in water in a distribution system.

Chloroform is slightly lipophilic (soluble in fat) and has been detected in aquatic organisms. Tissue residue levels are not significantly influenced by a fish's diet, but rather by the chemical concentration in the water. Biomagnification does not occur due to the establishment of equilibrium between the fish and water concentration of the chemical.

Exposure

Most THM intake comes from chlorinated drinking water and beverages produced with treated water. The remainder comes from air and food. THMs are the most common disinfection by-products in municipal drinking water and are normally present at some level. For people who consume chlorinated drinking water, this will be their main source of THM exposure. Because of its volatility, there is potential for exposure in the home to airborne chloroform released from tap water (e.g., showering, swimming pools). There is also some potential for dermal absorption through bathing.

The amount of chloroform formed depends on the type and amount of organic matter in the water. Because groundwater usually has a lower total organic carbon concentration than surface waters, the THM concentration of chlorinated groundwater is also lower than that of chlorinated surface water. High-level point sources of chloroform in groundwater supplies are industrial effluents and accidental spills. In *A National Survey of Chlorinated Disinfection By-Products in Canadian Drinking Water*, Health Canada reports that the majority of treatment facilities had relatively low total THM levels (<50 µg/L) during both the summer and winter, and a small number of facilities had relatively high total THM values (>100 µg/L). There are seasonal variations in THM drinking water concentrations, in which concentrations are usually higher in the summer than in the winter for all treatment processes.

Reducing Exposure

Alternative disinfectants such as ozone, chlorine dioxide and chloramines are now in use. These alternatives vary in their cost, efficiency and by-products (which have not all been examined for health effects). Recent research has shown that THM levels in drinking water can be reduced by adjusting the chlorine dose, improving filtration practices in order to remove as much of the organic material in the water as possible, and adding chlorine after filtration (rather than before).

Consumers can also remove the volatile THMs from their drinking water supply at home by aerating it in a blender, boiling it or storing it in the refrigerator for 24 hours. Water treatment devices containing activated carbon also remove some THMs. Manufacturers instructions should be followed to avoid risks of bacterial contamination.

Human Health Considerations

Chloroform and bromodichloromethane have been classified by the International Agency for Research on Cancer (IARC) as possibly carcinogenic to humans due to inadequate evidence in humans, but sufficient data in animals. According to IARC, chlorodibromomethane and bromoform are not classifiable as to their carcinogenicity to humans. Studies with experimental animals indicate that at levels of chloroform similar to

Table 1

IMAC, MAC AND OTHER VALUES FOR TRIHALOMETHANES IN DRINKING WATER

Agency	Focus	Level	Comments
Health Canada	Drinking water	0.1 mg/L (chloroform)	IMAC[1]
OMOEE	Drinking water	0.1 mg/L	MAC
WHO	Drinking water	100 µg/L bromoform[2] 100 µg/L dibromochloromethane 60 µg/L bromodichloromethane 200 µg/L chloroform	guideline value guideline value guideline value; for bromodichloromethane and chloroform, this value is associated with an excess lifetime cancer risk of 10^{-5}.

IMAC: Interim maximum acceptable concentration
MAC: Maximum acceptable concentration
OMOEE: Ontario Ministry of Environment and Energy
WHO: World Health Organization

1. This has been designated as an interim MAC until such time as the risks from other disinfection by-products are ascertained.
2. The sum of the ratio of the concentration of each to its respective guideline value should not exceed 1.

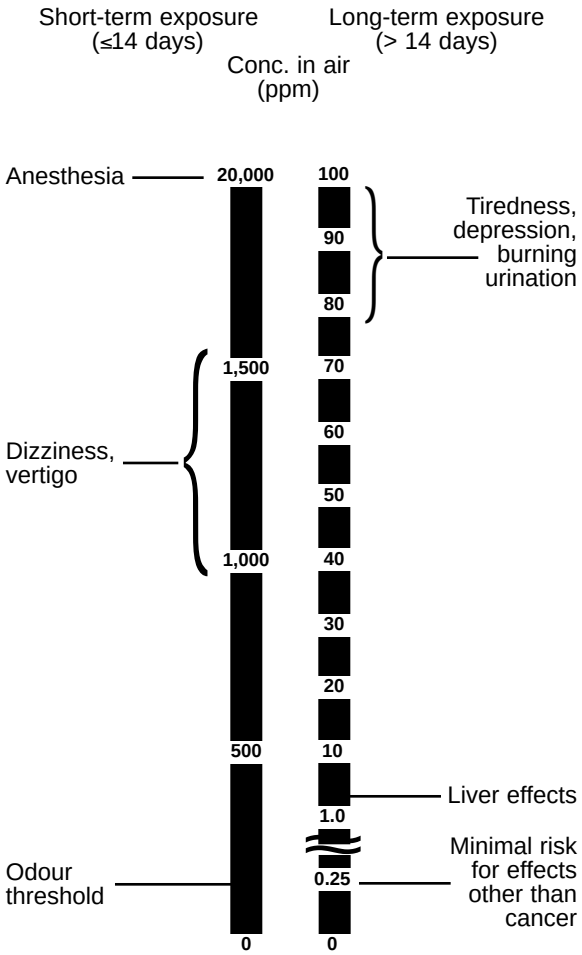
those present in drinking water, reproductive and developmental effects are very unlikely. THMs accumulate most in tissues with a high adipose content: adipose tissue > brain > kidney > blood.

Results of a recent Heath Canada and Ontario Cancer Treatment and Research Foundation (OCTRF) study support the overall weight of evidence from other published scientific studies that there is an association between the use of chlorinated water and bladder cancer risk, and a somewhat weaker association with colon cancer risk. Unlike some other studies, this study did not find an association between the use of chlorinated water and rectal cancer. The study is one of the largest and most convincing to date, involving nearly 5000 residents of Ontario.

Risks were highest for those people with the longest duration of exposure (35 years of exposure or more) and the highest concentrations of THMs. People using treated water from sources with low chlorination by-products (i.e., groundwater) or those who have used water with elevated levels of chlorination by-products for shorter periods of time (less than 35 years) did not appear to be at increased risk.

Figure 1

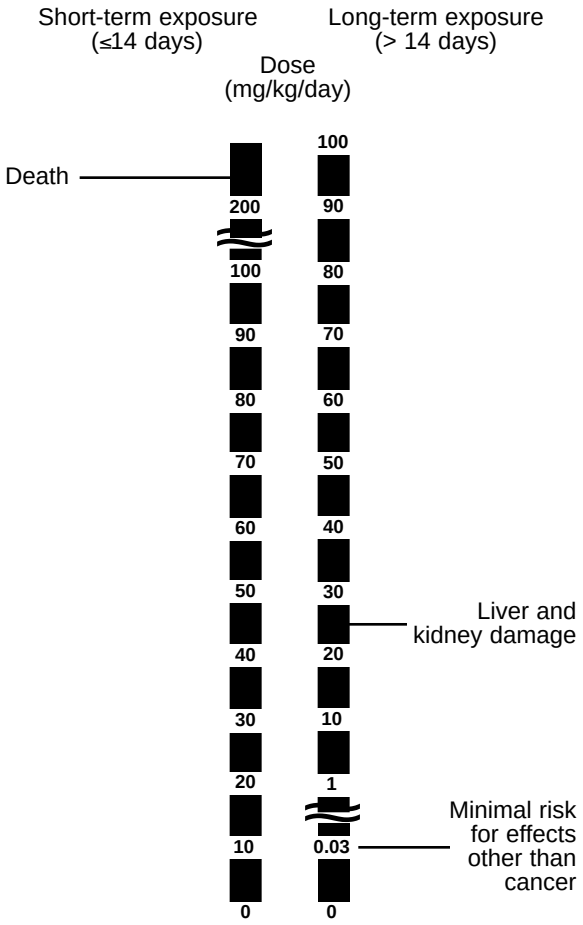
HUMAN HEALTH EFFECTS FROM BREATHING CHLOROFORM



Adapted from: U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR). *Public Health Statements – What you need to know about toxic substances commonly found at Superfund hazardous waste sites.* Atlanta, Georgia.

Figure 2

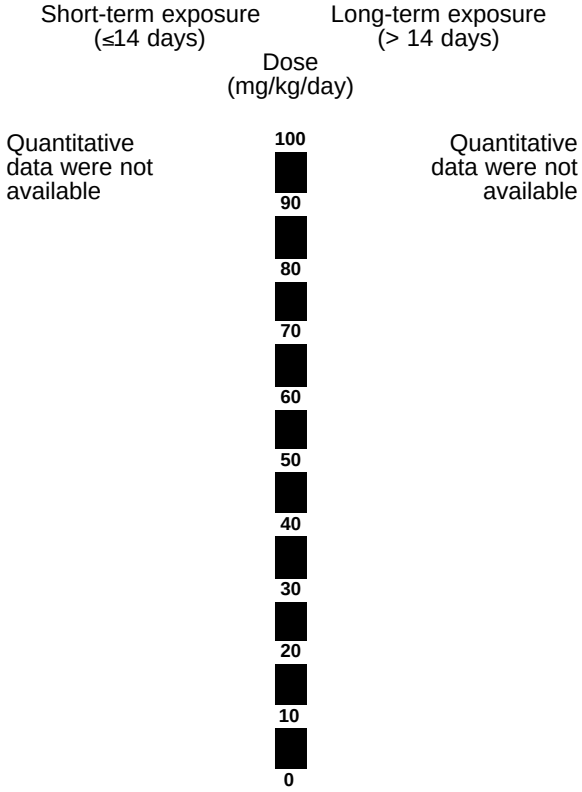
HUMAN HEALTH EFFECTS FROM INGESTING CHLOROFORM



Adapted from: U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR). *Public Health Statements – What you need to know about toxic substances commonly found at Superfund hazardous waste sites.* Atlanta, Georgia.

Figure 3

HUMAN HEALTH EFFECTS FROM SKIN CONTACT WITH CHLOROFORM



Adapted from: U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR). *Public Health Statements – What you need to know about toxic substances commonly found at Superfund hazardous waste sites.* Atlanta, Georgia.

The results of the study suggest that the lifetime probability that an individual living in Ontario will develop bladder or colon cancer due to the use of chlorinated water is quite low. For example, about 1.34 percent of men who are exposed to chlorinated surface water for 35 years or more will develop bladder cancer before the age of 70, compared with about 1 percent of men not exposed to chlorinated surface water. For women, approximately 0.37 percent of those exposed to chlorinated surface water will develop bladder cancer before the age of 70, compared with 0.27 percent of women who are not exposed. It is important to note that 50 percent of bladder cancers are associated with smoking.

Studies have also looked at the possible association between exposure to THMs in municipally treated water and other cancers including cancers of the esophagus, pancreas, urinary tract, stomach, breast, lung, and brain. However the associations in these studies have not been consistent and confounding factors (exposure, population mobility) were not taken into account, thereby limiting the usefulness of the data in assessing cause-effect relationships.

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Tritium

Tritium (^3H) is one minor source of ionizing radiation, and everyone is exposed to ionizing radiation. A natural background of radiation, 2-3 m*Sv*/year (*Sv* = sievert; the unit used to measure a radioactive dose), occurs from cosmogonic sources, radionuclides in soil, food, water, air, and building materials. Medical procedures such as x-rays and radiation therapy can increase an individual's exposure. The OMOEE has set a tritium drinking water standard of 7000 *Bq*/L. Continuous exposure at this level adds 0.1 m*Sv* to a person's total exposure. It is the total exposure to ionizing radiation that must be controlled, not just one radionuclides that contributes only a small fraction of the total dose. It is not appropriate to develop a guideline for one radionuclides in isolation from all the others.

Origin and Uses

Tritium is a naturally occurring radionuclide. It is a heavy hydrogen atom that has captured a neutron, and releases low energy in the form of a beta (ß) particle. The decay formula is:

$^3H \rightarrow {}^3He + ß^-$.

A natural source of tritium comes from the interaction of cosmic rays with nitrogen, oxygen and argon in the upper atmosphere. Natural sources of tritium account for < 1 percent of tritium exposure. Almost all of the remaining 99 percent is due to fall-out from thermonuclear weapons testing. CANDU (Canadian Deuterium Uranium) nuclear reactors are also a source. Tritium is released from nuclear reactors, both operationally and accidentally, in the form of tritiated water. Low levels of tritium occur in air in the vicinity of CANDU reactors.

Tritium is used commercially as a light source (in flares, emergency lights, exit signs and luminous dials), and in medical research. It is an essential fuel for nuclear fusion, and is also used in the production of some types of fusion weapons.

Persistence and Movement in the Environment

Tritium produced in the upper atmosphere is converted into tritiated water and enters the global water cycle. Airborne releases of tritium contaminate ambient air and can settle on surface waters and agricultural lands. Tritium has a radiation half-life of 12.3 years. This is the time for one half of a quantity of tritium to decay into non-radioactive helium. In the environment, tritium can bond with organic molecules and these can in turn be incorporated into living organisms, where the radioactive energy released by it may cause cell damage and genetic damage.

Exposure

Tritium is most commonly found in the form of tritiated water (^3H_2O), including in air and food. Exposure to tritiated water from drinking water supplies can occur through ingestion, inhalation or dermal absorption (swimming, showering). Tritiated water is not distinguishable from normal water by way of taste, smell or texture.

Across Canada, average tritium concentrations in surface waters are 5–10 *Bq*/L, primarily due to residual fall-out from atmospheric weapons tests conducted prior to 1963. During 1982 to 1984, average levels in the Great Lakes ranged from 7–10 *Bq*/L. In 1990, an average tritium concentration across Ontario was 6 *Bq*/L. Tritium levels in Ontario drinking water are generally very low and rarely exceed 20 *Bq*/L. Levels are higher near all Ontario nuclear reactors, ranging from 10–47 *Bq*/L, in 1995. However, higher levels have been recorded. For example, tritium spills have resulted in short-term increases of several days in duration. A spill at the Pickering Nuclear Generating Station in August 1992 resulted in a peak concentration at the nearest water supply of 1300 *Bq*/L.

Inhalation occurs through exposure to an atmosphere contaminated with tritiated water vapour. The human body absorbs most of the tritium that is inhaled and about half of the tritium that comes into contact with the skin. Ingested tritiated water is completely absorbed from the gastro-intestinal tract and is then rapidly distributed throughout the body by the blood. Only rarely does tritiated water concentrate in particular tissues.

A biological half-life is the amount of time the body requires to excrete one half of the tritium absorbed. The majority of the tritium is removed from the body with a biological half-life ranging from 2.4 to 18 days, representing the turnover of body water. The remainder is removed with a half-life of one month to one year, representing the turnover of tritium incorporated in organic compounds. Tritium is excreted with water, in breath and in urine.

Ingestion of contaminated foods (fruits, vegetables or food animal products) that are grown near nuclear generating stations may be a source of tritium exposure. A statistically elevated concentration of tritium in air, water and vegetation close to Ontario Hydro nuclear power facilities can be attributed to station operation. These levels are very low, so that the doses received by the portion of the population that is most exposed are < 1 percent of the public dose limits. In addition, no more than half of this calculated dose is due to tritium and only 5 to 30 percent of the tritium dose is due to tritium in drinking water.

Reducing Exposure

Ontario Hydro's Tritium Removal Facility, which has been in operation since 1990, removes tritium that could otherwise be released to the environment during operations. Located at the Darlington CANDU reactor, this facility extracts tritium from the moderator systems of Hydro's various reactors. Despite these measures, tritium releases to the environment still do occur.

Human Health Considerations

Internal injury from beta particles occurs when tritium is inhaled or ingested. Because the beta particle from tritium decay has a range of only 0.7 µm, tritium must be located very close to the cell to cause biological injury. Tritium incorporated into DNA is a potential health concern. Beta particles from tritium present no hazard when exposure occurs externally, from outside of the body. They are stopped by 6 mm of air or 5 µm of water, and cannot penetrate the outermost layer of skin.

Three separate epidemiological studies have been conducted in populations living near Ontario Hydro facilities to address childhood leukemia (Clarke et al. 1989; Clarke et al. 1991), birth defects (Johnson and Rouleau. 1991) and leukemia among children of reactor workers, including those exposed to significant doses from tritium (McLaughlin et al. 1993). In addition, the Ontario Ministry of Health maintains an ongoing surveillance of health statistics and the Ontario Cancer Registry maintains a registry of all cancer cases occurring in Ontario. Although studies and ongoing surveillance have found increases in Down's Syndrome and elevated childhood leukemia, there is no evidence to suggest these rates are correlated with tritium or other ß emitters, and radiation exposure in general.

Guidelines for drinking water for all radionuclides, including tritium, apply whenever radioactive substances are present in the water. When more than one radioactive substance is present, the maximum acceptable level is set so that the dose from all these substances in the water does not exceed 0.1 m*Sv*. The Health Canada guideline for drinking water of 7000 *Bq*/L (MAC) is derived by converting sieverts into becquerels using an appropriate conversion factor for tritium. The MAC of 7000 *Bq*/L is based on the dose for an adult, as a result of chronic daily exposure, of 0.1 m*Sv* in a year.

Table 1

MAC AND GUIDELINE VALUES FOR TRITIUM IN DRINKING WATER

Agency	Focus	Level	Comments
Health Canada	Drinking water	7000 *Bq*/L	MAC
OMOEE	Drinking water	7000 *Bq*/L	
WHO	Drinking water	7800 *Bq*/L	guideline value

MAC: Maximum acceptable concentration
OMOEE: Ontario Ministry of Environment and Energy
WHO: World Health Organization

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Vibrio vulnificus

Origins and Uses

Vibrio vulnificus is a naturally occurring marine bacterium that normally occurs in oysters, clams and mussels. It is the leading cause of death in the southern United States in persons eating raw or undercooked seafood harvested from the Gulf of Mexico. While there have been no reported cases of *Vibrio vulnificus* infection due to eating raw or undercooked molluscan shellfish in Canada, Canadians are still at risk from eating shellfish from the Gulf of Mexico.

Persistence and Movement in the Environment

The contamination of shellfish by the *Vibrio vulnificus* bacterium occurs during feeding. Shellfish are "filter-feeders," which means that they strain and eat particulate matter — including *Vibrio vulnificus* — from water. If contaminated shellfish are eaten raw or undercooked, there is a health risk to consumers. *Vibrio vulnificus* is not the result of sewage or other water pollution.

Exposure

Most incidences of *Vibrio vulnificus* occur in the states bordering on the Gulf of Mexico. Eighty-five percent of reported cases were associated with oysters that were consumed in restaurants, while the remaining 15 percent were shellfish purchased from retail and wholesale markets. Infections generally occur between March and December with a peak between May and October.

Reducing exposure

Proper cooking is the only effective method to kill *Vibrio vulnificus*. Refrigeration after harvest, during transportation and retail sale limits the number of bacteria, but does not completely eliminate them. Cooked seafood should be stored above 60°C (140°F) or below 4°C (39.2°F).

The following precautions should be taken when shopping for, storing, and cooking shellfish.

Shopping: When buying shellfish, keep in mind that molluscs in the shell should always be alive when they are bought. Shells will be tightly closed or will close when tapped lightly or put on ice. Seafood should only be bought from reputable dealers.

Storing: Molluscs should be stored in the refrigerator in containers covered loosely, with a clean, damp cloth. Live molluscan shellfish should not be stored in airtight containers or in water. Shellfish should be cooked as soon as possible — no later than two days after purchase. Raw and cooked seafood should be kept separate. Cooked seafood should not be put back in the same container used for raw seafood.

Cooking: Most bacteria are only destroyed by cooking food to a minimum internal temperature of 60°C (140°F) for five to six minutes. When cooking shellfish in the shell, shells should open during boiling. After shells open, boiling should continue for three to five more minutes. When steaming shellfish, cooking should take four to nine minutes from the start of steaming. Clams, oysters or mussels whose shells do not open during cooking should be discarded. If too many shellfish are cooked in the same pot, it is possible that the ones in the middle will not be thoroughly cooked. Shucked shellfish meat should be boiled or simmered for at least three minutes, fried in oil for at least 10 minutes at 160°C (375°F), or baked for at least 10 minutes at 190°C (450°F).

Human Health Considerations

Symptoms due to *V. vulnificus* infections usually appear within 18 hours or less. Septicemia, a severe form of blood infection, can occur from eating contaminated food or from wound infection. Symptoms include fever, chills, nausea, vomiting, and diarrhea. A sharp drop in blood pressure also usually occurs and the majority of patients develop painful skin lesions.

Ninety percent of cases of *V. vulnificus* septicemia are associated with eating raw oysters. The quantity of shellfish required to result in infection is not known. Wound infections occur when the bacteria enter directly through a wound after exposure to seawater, after handling shellfish, or during marine activities. The infection is characterized by swelling, redness, and intense pain around the site of injury. The infection can also spread into the bloodstream resulting in septicemia. Gastroenteritis, an infection of the intestinal tract resulting from eating contaminated food, is a much less common result of infection by this bacterium. Patients with gastroenteritis usually do not have underlying disease and rarely die.

Certain individuals are at higher risk of contracting the infection than others. Susceptible persons include those with pre-existing diseases such as liver disease (cirrhosis, haemochromatosis, chronic alcohol abuse); diabetes mellitus; immune disorders associated with AIDS, cancer and steroid or immunosuppressant therapy, and stomach and intestinal disorders. Even with medical treatment, persons with liver disease have about a 60 percent chance of dying if they contract septicemia.

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Vinyl Chloride and Polyvinyl Chloride

Origins and Sources

Vinyl chloride is a colourless gas with a mild, sweet odour. It does not occur naturally in the environment and is generally used to make polyvinyl chloride (PVC). PVC is in turn used to make many plastics and vinyl products including pipes, wire and cable coatings, housewares, and automotive parts. Smaller amounts of PVC are also used as cooling gas and in the manufacture of other compounds.

Persistence and Movement in the Environment

Vinyl chloride is released mainly into the air and discharged into waste water from plastics industries; primarily from vinyl chloride and PVC manufacturers. Vinyl chloride is also formed from trichloroethylene and perchloroethylene (see Contaminant Profile on Perchloroethylene) in groundwater. Most of the vinyl chloride entering the environment ends up in the air where it gradually breaks down into less harmful substances.

Exposure

Humans are exposed to vinyl chloride from both environmental and occupational sources. The highest potential exposure groups in non-occupational settings are people living in communities close to vinyl chloride plants, and people living near waste disposal sites. The most likely exposure pathway is breathing air containing vinyl chloride. Vinyl chloride can also enter the body through eating food or drinking water containing vinyl chloride. The skin is not likely an important pathway of exposure.

The air inside new cars may also contain vinyl chloride levels that are higher than expected because it may seep into air from plastic parts. Levels decrease when windows are opened or the air conditioning is turned on.

Vinyl chloride can be found in drinking water as a consequence of wastes released into rivers and lakes from factories, and from seepage into groundwater in storage areas. Small amounts can enter drinking water from contact with PVC pipes manufactured with incompletely polymerized vinyl chloride monomer. Excessive leaching of vinyl chloride from PVC water pipes is prevented by the existing product standards. These specify the quality of PVC water pipes to limit the amount of free vinyl chloride monomer present.

Reducing Exposure

Health Canada recently issued a warning against the use of certain inexpensive PVC blinds in homes or other establishments frequented by children six years of age and under. The adverse health effects were, however, not due to exposure to polyvinyl chloride but rather to the fact that the blinds were found to contain lead. No increased risk was associated with exposure to the PVC itself (see Contaminant Profile on Lead for more details).

Human Health Considerations

Short-term exposure to high levels of vinyl chloride in air has been known to cause dizziness, stumbling, lack of muscle coordination, headache, unconsciousness, and even death. Chronic inhalation exposure to low levels of vinyl chloride can cause degenerative bone changes, circulatory disturbances in the extremities, dermatitis

and damage to the lungs, spleen and liver. To date, epidemiological studies are not sufficient for quantitatively estimating cancer risks associated with exposure to low levels of vinyl chloride in outside air, drinking water, or food.

Vinyl chloride is mutagenic *in vitro* and *in vivo*. It is a known human and animal carcinogen. Exposure to vinyl chloride has been associated with angiosarcoma cancer) of the liver. Other toxicological studies suggest that vinyl chloride is also associated with hepatocellular carcinoma, brain tumours, lung tumours, and malignancies of the lymphatic and haematopoietic tissues.

Table 1

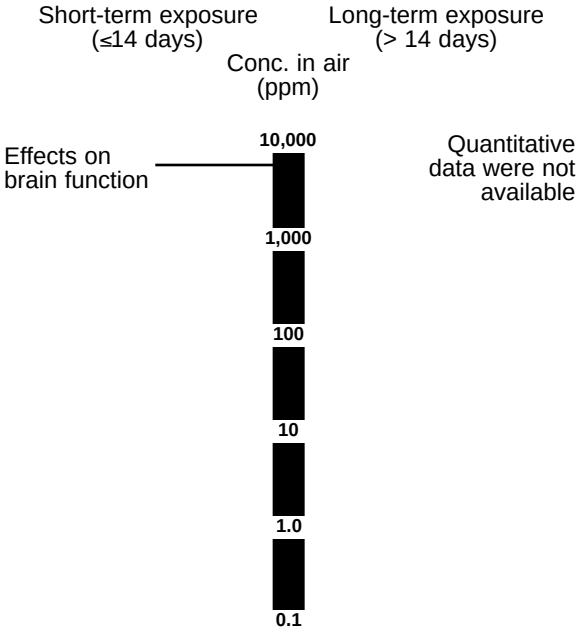
GUIDELINES FOR VINYL CHLORIDE IN DRINKING WATER

Agency	Focus	Level	Comments
Health Canada	Drinking water	0.002 mg/L	MAC
WHO	Drinking water	0.005 mg/L	Guideline value; this value is associated with an excess cancer risk of 10^{-5}.

WHO: World Health Organization
MAC: Maximum acceptable concentration

Figure 1

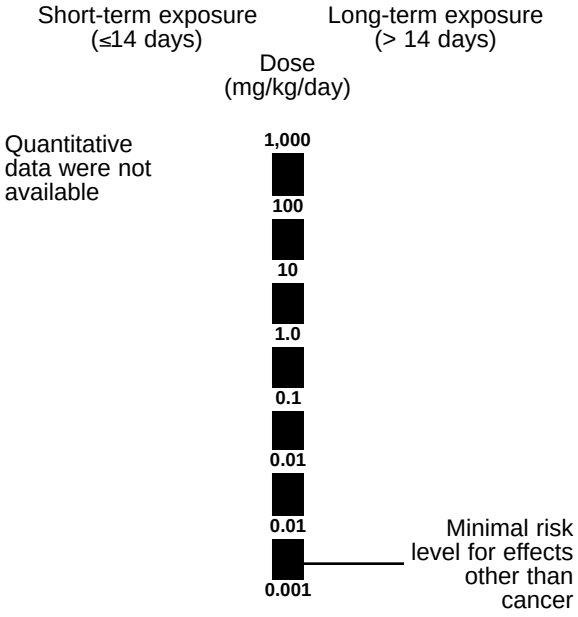
HUMAN HEALTH EFFECTS FROM BREATHING VINYL CHLORIDE



Adapted from: U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR). *Public Health Statements – What you need to know about toxic substances commonly found at Superfund hazardous waste sites.* Atlanta, Georgia.

Figure 2

HUMAN HEALTH EFFECTS FROM INGESTING VINYL CHLORIDE



Adapted from: U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR). *Public Health Statements – What you need to know about toxic substances commonly found at Superfund hazardous waste sites.* Atlanta, Georgia.

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2,4-Dichlorophenoxyacetic acid (2,4-D)

Origins and Uses

2,4-dichlorophenoxyacetic acid (2,4-D) has been used in Canada and globally for almost 50 years. It is a popular choice of herbicide because of its low cost and effectiveness on broad-leaf plants. 2,4-D controls weeds in a wide variety of settings including farms, pastures, rangelands, roadsides, industrial sites, forestry operations, parks, golf courses and home lawns. It is also used to control aquatic weeds. In recent years, the use of 2,4-D in parks, recreation, and other areas frequently used by the public has been reduced in some countries because of increasing concern about possible toxic effects.

The 2,4-D formulations used today are mainly the water soluble forms, i.e., amine salts. These are formulated with solvents, carriers, or surfactants and are marketed in the form of dusts, granules, emulsions, oils, water solutions, gels and water soluble packets in a wide range of concentrations. 2,4-D acid and its salts are less toxic to aquatic organisms than are the esters.

Persistence and Movement in the Environment

2,4-D is applied to foliage of existing weeds, and the primary mode of uptake is foliar (through leaves). 2,4-D does not persist in soil because of its rapid degradation. 2,4-D undergoes microbial breakdown in warm, moist soil. Its rate of breakdown depends on temperature, moisture, organic matter and other soil characteristics that affect microbial activity. The resultant average persistence of phytotoxicity in soil at recommended rates is one to four weeks in warm, moist soil. 2,4-D degrades fairly rapidly in aerobic soils with high organic contents and at high pH and temperature. In most organisms, including humans, 2,4-D is rapidly excreted and is not retained by most organisms.

Exposure

2,4-D may enter the environment through its production and disposal and from industrial effluents. Improper disposal of unused 2,4-D in agriculture and washing of equipment may result in localized land pollution and also pollution of water supplies through direct contamination or leaching from soil. 2,4-D has been detected in samples of raw and treated drinking water from municipal and private supplies across Canada at levels below 30 µg/L.

Reducing Exposure

All chemicals, including 2,4-D, should be used with caution. The best way to ensure good health is to closely follow label instructions for the safe use of pesticides (see Chapter 10. "Home Environments"). A Label Improvement Program has recently been implemented by the Pest Management Regulatory Agency for 2,4-D. Specific emphasis was placed on operator and public exposure.

People who consider themselves sensitive to chemicals should avoid exposure to 2,4-D as much as possible. Accidental spread of the herbicide to other vegetation should be avoided.

Human Health Effects

In the 1970s and early 1980s, some epidemiological studies reported an association between soft-tissue sarcomas (STS) and multiple lymphomas, including Hodgkin's disease and non-Hodgkin's lymphoma (NHL), and the use of chlorophenoxy herbicides by agricultural or forestry workers. Since that time, further work has provided inconsistent results regarding the causal association between farming, chlorophenoxy herbicide use and NHL. The association between 2,4-D and STS (commonly attributed to dioxins) is weak and inconsistent.

Laboratory experiments with 2,4-D (and chemical compounds similar to it) have not provided conclusive evidence for their carcinogenicity, although a slight dose-related increase in brain tumours was noted in male rats. A case-control study concluded that pet dogs may be at risk of developing cancer from exposure to 2,4-D. The total weight of evidence currently available does not support a conclusion that any of the phenoxy herbicides (including 2,4-D) present a carcinogenic hazard to people.

Surveys of heavily exposed workers and studies of people living where 2,4-D and related herbicides were heavily used did not show clear evidence linking the chemicals to adverse reproductive health effects (e.g., decreased fertility, miscarriages, stillbirths, and birth defects). Impacts on the reproductive system have only been associated with very high doses which would not be encountered by typical 2,4-D users.

Human exposure studies were performed by Harris et al. (1992) and Harris and Solomon (1992) to assess home owner and bystander exposure to 2,4-D during application of liquid or granular formulations to residential turf. They concluded that exposure was highest for home owners applying liquid formulations who did not receive instructions on use and wore their everyday clothing. Exposures were directly related to spills of liquid concentrate on the bare hands or forearms, or excessive contact with the dilute liquid. 2,4-D was not detected in the urine of bystanders potentially exposed during application to domestic turf. Another study used biological monitoring to assess exposure to adults following one hour of controlled activities (walking, sitting, laying on turf sprayed 1 or 24 hours previously). They concluded that individuals (wearing long pants, t-shirt, socks and covered footwear) exposed to turf sprayed either 1 or 24 hours previously had no detectable exposure to 2,4-D.

Poisonings from accidental exposure to, or intentional ingestion of, large amounts of the chemical have occurred. These cases often result in full recovery although a few fatal cases were reported from suicidal consumption of 2,4-D in the 1960s.

Table 1

GUIDELINES FOR 2,4-D IN DRINKING WATER

Agency	Focus	Level	Comments
Health Canada	Drinking water	0.1 mg/L	IMAC
WHO	Drinking water	30 µg/L	Guideline value

WHO: World Health Organization
IMAC: Maximum acceptable concentration

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