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Benzene

Guideline

The maximum acceptable concentration (MAC) for benzene in drinking water is 0.005 mg/L (5 μg/L).

Identity, Use and Sources in the Environment

Benzene, the simplest homologue of the aromatic hydrocarbons, is a planar, cyclic molecule with six carbon atoms arranged in a regular hexagon. It is a volatile, colourless liquid with a characteristic odour; it is soluble in water at 1000 mg/L and miscible with many organic solvents.[1] Its vapour pressure is 13 kPa at 25°C.

Approximately 700 kilotonnes of benzene are produced in Canada annually for use mainly in the manufacture of other organic chemicals.[1] Benzene is present in gasoline at concentrations of approximately 1 to 2%, and vehicular emissions constitute the main source of benzene in the environment. Benzene is introduced into water from industrial effluents and atmospheric pollution.

Exposure

Only limited data are available on the levels of benzene in Canadian drinking water supplies. Benzene was detected in 50 to 60% of potable water samples taken at 30 treatment facilities across Canada. Mean concentrations ranged from 1 to 3 μg/L, and a maximum value of 48 μg/L was recorded in one instance.[2] The total amount of benzene that would be ingested daily by drinking 1.5 L of water containing 2 μg/L benzene is 3 μg. Because of its volatility, there is also potential for exposure in the home to airborne benzene released from tap water.

Few data are available on the benzene content of food; however, based on a rough estimate of dietary intake in the United States[3] (250 μg/d), food is probably the main source of ingested benzene. Concentrations of benzene in air sampled in Toronto between October 1, 1984, and October 1, 1985, averaged 10.2 $\mu g/m^3$ and ranged up to 38.2 $\mu g/m^3$ (105 samples). Concentrations in Montreal air during the same time period were higher, averaging 24.3 $\mu g/m^3$ and ranging up to 91.4 $\mu g/m^3$ (49 samples).[4]

Analytical Methods and Treatment Technology

Benzene is detected by a purge and trap gas chromatographic procedure. The U.S. Environmental Protection Agency determined that the practical quantitation limit (PQL) for benzene in water (based on the ability of laboratories to measure benzene within reasonable limits of precision and accuracy) is 5 μg/L.[5] This conclusion is supported by work carried out by the Department of National Health and Welfare.[6,7]

Available data indicate that benzene concentrations are not reduced significantly during conventional drinking water treatment processes.[1] However, removal of volatile organic compounds by packed tower aeration and granular activated carbon adsorption has been estimated to be 90 to 99% effective,[5] and concentrations of benzene below 1 μg/L are commonly achieved using these methods.

Health Effects

Benzene is rapidly and efficiently (30 to 50%) absorbed following inhalation. Quantitative data are not available on the absorption of benzene from the gastrointestinal tract following ingestion, but benzene is known to be poorly absorbed through the skin. Up to one-half of inhaled benzene is exhaled unchanged, whereas the remainder is excreted principally in the urine as metabolites (e.g., the ester sulphates and glucuronides of phenol, catechol, quinol and hydroxyquinol). Because of its lipophilicity, benzene accumulates primarily in adipose tissue. It also accumulates in bone marrow and central nervous system tissue; intermediate levels are found in the liver, and low levels are present in the spleen and blood.[1]

There is a fairly extensive database available on the health effects associated with the inhalation of benzene by humans. Acute exposure to high levels affects primarily the central nervous system, causing dizziness,

giddiness, nausea and vomiting, headache and drowsiness and, at concentrations in the order of 20 000 ppm (65 000 mg/m^3), narcosis, coma and, sometimes, death. The early symptoms of acute toxicity are rapidly reversible and are not observed at lower levels, no matter how long the exposure period.[8]

At lower concentrations, benzene is toxic to the haematopoietic system, causing a continuum of haematological changes, ranging from a mild decrease in platelet count to aplastic anaemia, a rapidly fatal disease.[1,8] In addition, benzene causes acute myeloblastic leukaemia, acute myelomonocytic leukaemia and erythroleukaemia. Secondary clinical symptoms associated with chronic exposure to benzene include increased susceptibility to infection: benzene may impair immune responses. It is difficult to draw firm conclusions concerning no-effect levels for the various haematological abnormalities because of the paucity of exposure data in available epidemiological studies, as well as difficulty in separating out various effects in a continuum of related symptoms; however, changes in platelet counts in workers have been associated with exposure to concentrations as low as 10 to 20 ppm (32 to 65 mg/m^3).[9–11] Increased incidence of leukaemia has been observed in populations occupationally exposed to less than 10 ppm (<32 mg/m^3) for periods as short as one year,[12–15] and chromosomal aberrations have been demonstrated at workplace concentrations averaging 2 ppm (6 mg/m^3).[16]

Haematological abnormalities similar to those observed in humans, including cytopenia and aplastic anaemia, have been observed in animal species exposed to benzene. Although most of the data have been obtained in inhalation studies, an investigation of the effects of long-term ingestion of benzene in rats and mice was recently conducted under the auspices of the National Toxicology Program (NTP).[17] Haematological effects in F344/N rats and B6C3F$_1$ mice (50 of each sex) ingesting benzene at a concentration of 0 to 200 mg/kg bw per day by gavage in corn oil, five days per week for 103 weeks, included lymphoid depletion of the splenic follicles (rats) and thymus (male rats), bone marrow haematopoietic hyperplasia (mice), lymphocytopenia and associated leucocytopenia (rats and mice). Several of these effects occurred at the lowest exposure level (25 mg/kg bw per day).

Results from animal species have only recently confirmed the carcinogenicity of benzene observed in occupationally exposed humans, following both inhalation[18,19] and ingestion.[17,20,21] In studies conducted by Maltoni and Scarnato, a significant increase in the incidence of Zymbal gland tumours, mammary carcinomas and leukaemia in Sprague-Dawley rats was observed following administration of benzene at concentrations of both 50 and 250 mg/kg bw per day by gavage in olive oil, four to five days per week for 52 weeks.[21] In addition, increased incidence of squamous cell carcinomas of the oral cavity, nasal carcinomas and liver angiosarcomas was observed in a continuing study in which 500 mg/kg bw per day was administered by gavage in corn oil to the same strain, four to five days per week for up to 92 weeks. In the NTP studies described above, the incidence of primary neoplasms of the Zymbal gland, oral cavity and skin (males only) was increased in rats.[17] In mice, the incidence of tumours of the Zymbal gland, preputial gland (male), ovary and mammary gland (females), harderian gland and lung and lymphoid tissue was increased.

Benzene has not been found to be mutagenic in bacterial assays;[1,8] however, *in vivo* it has caused chromosomal aberrations in a variety of species. It also gave positive results in the micronucleus test in mice. Available data from teratological studies indicate that benzene is not teratogenic at dose levels that are not maternally toxic.[1]

Classification and Assessment

Benzene is a documented human carcinogen; it has, therefore, been classified in Group I (carcinogenic to man). Epidemiological data are insufficient to serve as a basis for quantitative estimation of cancer risks associated with exposure to low levels of benzene; the relevant studies do not contain adequate information on degree of exposure or size of the population at risk. In addition, workers in benzene-related occupations typically are exposed to other chemicals as well. Cancer risks have, therefore, been estimated on the basis of the results of the recently completed NTP carcinogenesis bioassay in F344/N rats and B6C3F$_1$ mice (gavage).[17] Incorporating a surface area correction and using the robust linear extrapolation model for each of the significantly increased tumour types, one can calculate that unit lifetime risks associated with the ingestion of 1 μg/L benzene in drinking water range from 6.1×10^{-7} (based on leukaemia and lymphomas in female mice) to 6.7×10^{-6} (based on oral cavity squamous cell carcinomas in male rats).* The estimated ranges of concentrations in drinking water corresponding to lifetime risks of 10^{-5}, 10^{-6} and 10^{-7} for these same tumour types based on the model described above are as follows:

Lifetime risk	Concentrations in drinking water (μg/L)
10^{-5}	1.5 – 16
10^{-6}	0.15 – 1.6
10^{-7}	0.015 – 0.16

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

Rationale

Because benzene is classified as a carcinogen in Group I (carcinogenic to man), the maximum acceptable concentration (MAC) is based on consideration of available practicable treatment technology and estimated lifetime cancer risk. Because the MAC must also be measurable by available analytical methods, the PQL is also taken into consideration in its derivation.

An MAC of 0.005 mg/L (5 µg/L) for benzene was established, therefore, on the basis of the following considerations:

(1) The estimated unit lifetime risks associated with the ingestion of benzene in drinking water range from 6.1×10^{-7} (based on leukaemia and lymphomas in female mice) to 6.7×10^{-6} (based on oral squamous cell carcinomas in male rats). Therefore, the estimated lifetime risk associated with the ingestion of drinking water containing 5 µg/L benzene (i.e., 3.1×10^{-6} to 3.4×10^{-5}) is within a range that is considered to be "essentially negligible."

(2) Available data indicate that benzene concentrations are not reduced significantly during conventional drinking water treatment processes. However, concentrations of benzene below 1 µg/L can be achieved by packed tower aeration and granular activated carbon adsorption.

(3) The PQL (based on the ability of laboratories to measure benzene within reasonable limits of precision and accuracy) is 5 µg/L.

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