Arsenic

(endorsed 2011)

GUIDELINE

Based on human health considerations, the concentration of arsenic in drinking water should not exceed 0.01 mg/L.

GENERAL DESCRIPTION

Arsenic is a naturally occurring element which can be introduced into water through the dissolution of minerals and ores (where it exists mainly in the sulfide form), or from industrial effluent, atmospheric deposition (through the burning of fossil fuels and waste incineration), drainage from old gold mines, or the use of some types of sheep dip. Natural sources can make a significant contribution to the arsenic concentration in drinking water. Arsenate (i.e. pentavalent (As(V)) is generally the most common form in well oxygenated surface waters and drinking water, but under reducing conditions, such as those found in deep lake sediments or groundwaters, the trivalent form (As(III), arsenite) predominates.

Arsenic compounds have commercial and industrial uses as alloying agents in the manufacture of transistors, lasers and semiconductors, and in the processing of glass, pigments, textiles, paper, metal adhesives, ceramics, wood preservatives, ammunition and explosives. They are also used in the hide-tanning process, and to a limited extent as feed additives, pesticides and pharmaceuticals. Although inorganic forms of arsenic are the most common, organic arsenic compounds are also used.

In surface- and groundwater not affected by arsenic mineral deposits or pollution, the concentration of arsenic is generally less than 0.005 mg/L

Food may be an important source of arsenic intake. The average Australian adult dietary intake of arsenic is approximately 0.04 mg per day. Arsenic is concentrated by many species of fish and shellfish, and is present in poultry and livestock. Concentrations in vegetables are usually an order of magnitude less than those found in fish and meat. It is difficult to make direct comparisons between the arsenic intake from food and water because the form of arsenic and biological availability differ markedly. For example, a major portion of the arsenic in fish is present as highly complexed forms that are biologically unavailable, or as simple organic compounds (arsenobetaine and arsenocholine) that are essentially nontoxic.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In Australian drinking water supplies, typical concentrations of arsenic range from <0.001 mg/L to 0.03 mg/L.

TREATMENT OF DRINKING WATER

Arsenic can be removed from drinking water using conventional coagulation processes. It is desirable to convert trivalent arsenic to the pentavalent form before treatment by oxidation using chlorine or potassium permanganate. Lime softening can also be effective for removal from hard waters, but the efficiency is dependent on pH and valence state. The World Health Organization (WHO) (2003) states: "removal of arsenic to concentrations below 10 µg/litre is difficult in many circumstances," and Health Canada (2006) indicates that devices designed to remove arsenic from drinking water are certified to perform to 0.01 mg/L (10 µg/L) or less.

MEASUREMENT

The arsenic concentration in drinking water can be determined by hydride generation followed by atomic absorption spectroscopy. The limit of determination is approximately 0.001 mg/L. Alternatively, graphite furnace atomic absorption spectroscopy can be used, with a limit of determination of approximately 0.005 mg/L (APHA Method 3500-As Part B 1992).

HEALTH CONSIDERATIONS

The health considerations apply mainly to inorganic arsenic compounds, as they are more likely than the organic compounds to be present in drinking water supplies.

Soluble arsenic salts are readily absorbed by the gastrointestinal tract. After absorption, inorganic arsenic binds to haemoglobin, and is deposited in the liver, kidney, lungs, spleen, and skin. Inorganic arsenic does not appear to cross the blood-brain barrier but can cross the placenta. Very little ingested arsenic is excreted in faeces, but approximately 45-85% appears in the urine within 1 to 3 days.

Extensive reviews and summaries of the human and animal toxicity data for arsenic are available (IPCS 2001, WHO 2003, IARC 2004, Health Canada 2006, ATSDR 2007). Consumption of elevated levels of arsenic through drinking-water is causally related to the development of cancer at several sites, particularly skin, bladder kidney and lung. Cancer is considered to be the most sensitive toxicity end-point for setting a drinking water guideline for arsenic, however the mechanisms or modes of action by which arsenic causes cancer are yet to be definitively elucidated (WHO 2003).

Inorganic arsenic undergoes sequential reduction and methylation reactions leading to the formation of monomethylarsinic acid (MMA) and dimethylarsinic acid (DMA). This metabolism is regarded as bioactivating arsenic to more toxic forms. *In vitro* and *in vivo* chromosomal and DNA damage by arsenics are dose-dependent, with arsenite more potent than arsenate. Both MMA and DMA are directly genotoxic and are many times more potent than arsenite at inducing DNA damage (ATSDR 2007). Sub-populations that may be at greater risk from arsenic toxicity are those with gene variants of arsenic metabolism that lead to a higher percentage of MMA in the urine (Chen 2009, Smith and Steinmaus 2009).

The International Agency for Research on Cancer has concluded there is sufficient evidence in humans that arsenic in drinking-water causes cancers of the urinary bladder, lung and skin and has classified arsenic in drinking-water as carcinogenic to humans (Group 1) (IARC 2004).

DERIVATION OF GUIDELINE

The European Union (1998), WHO (2004), Health Canada (2008), USEPA (2008), and New Zealand (MoH NZ 2008) have drinking water guidelines for arsenic of 0.01 mg/L. Most of these were based on assessments conducted between 2003 and 2006; the previous Australian DWG was based on a 1988 evaluation. These overseas guidelines were based on considerations of the lowest concentration that is reasonably and economically achievable with water treatment technologies, measurability of arsenic at low concentrations, and lack of observed effects in humans at such low concentrations.

Epidemiological studies show that elevated cancer risks and other adverse health effects are not demonstrable at arsenic concentrations around 0.01 mg/L (e.g. Mazumder *et al.* 1998, Baastrap *et al.* 2008, Celik *et al.* 2008, Chen *et al.* 2009, Smith and Steinmaus 2009). On the other hand, there are many recent studies demonstrating a range of adverse health effects at higher concentrations (>0.05 or 0.1 mg/L) (e.g. Mazumder 2008, Majumdar *et al.* 2009, Mazumder *et al.* 2009, Smith and Steinmaus 2009). These health effects include skin, lung and bladder cancer; skin pigmentation and keratosis; diseases of the lung, liver, peripheral- and cardio-vascular systems; peripheral neuropathy; and diabetes. The majority of affected populations are from Indian, Bangladesh or Asian rural areas. Health Canada (2006) noted that recent epidemiological studies conducted in the United States had not found a clear association between cancer

NOTE: Important general information is contained in PART II, Chapter 6

risks and arsenic in drinking water at levels greater than 0.01 mg/L and below 0.05 mg/L. The study by Baastrap *et al.* (2008) is also informative as it was conducted in populations of similar socio-economic and nutritional status to those in Australia. This was a prospective Danish cohort of 57,053 people that was followed from 1970 to 2003; arsenic drinking water concentrations were up to 0.0253 mg/L (mean 0.0012 mg/L, 99th percentile 0.0057 mg/L), and no association with lung, bladder, liver, kidney, prostate, colorectum, or skin melanoma cancers was found.

After considering the above, a drinking water guideline of 0.01 mg/L was adopted for arsenic.

REFERENCES

APHA Method 3500-As Part B (1992). Arsenic: Atomic Absorption Spectrophotometric method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

ATSDR (Agency for Toxic Substances and Disease Registry) (2007). Toxicological Profile for Arsenic. ATSDR. PB2008-100002, August 2007.

Baastrup R, Sørensen M, Balstrøm T, Frederiksen K, Larsen CL, Tjønneland A, Overvad K, Raaschou-Nielsen O (2008). Arsenic in drinking-water and risk of cancer in Denmark. *Environmental Health Perspectives,* 116 (2):231-237.

Celik I, Gallicchio L, Boyd K, Lam TK, Matanoski G, Tao X, Shiels M, Hammond E, Chen L, Robinson KA, Caulfield LE, Herman JG, Guallar E, Alberg AJ (2008). Arsenic in drinking water and lung cancer: A systematic review. *Environmental Research,* 108(1):48-55.

Chen Y, Parvez F, Gamble M, Islam T, Ahmed A, Argos M, Graziano JH, Ahsan H (2009). Arsenic exposure at low-to-moderate levels and skin lesions, arsenic metabolism, neurological functions, and biomarkers for respiratory and cardiovascular diseases: Review of recent findings from the Health Effects of Arsenic Longitudinal Study (HEALS) in Bangladesh. *Toxicology and Applied Pharmacology.* In Press, Corrected Proof.

European Union (1998). Council Directive 98/83/EC on the quality of water intended for human consumption. *Official Journal of the European Communities.* L330.

Health Canada (2006). Guidelines for Canadian Drinking Water Quality: Guideline Technical Document for Arsenic. Health Canada, Ottawa, Ontario.

Health Canada (2008). Guidelines for Canadian Drinking Water Quality Summary Table, p. 14. Federal-Provincial-Territorial Committee on Drinking Water, Federal-Provincial-Territorial Committee on Health and the Environment.

IARC (International Agency for Research on Cancer) (2004). Arsenic in Drinking Water. IARC.

IPCS (International Programme on Chemical Safety) (2001). Environmental Health Criteria 224: Arsenic and arsenic compounds. World Health Organization, IPCS, Geneva, Switzerland.

Majumdar KK, Mazumder DNG, Ghose N, Ghose A, Lahiri S (2009). Systemic manifestations in chronic arsenic toxicity in absence of skin lesions in West Bengal. *Indian Journal of Medical Research,* 129(1):75-82.

Mazumder DNG, Haque R, Ghosh N, De BK, Santra A, Chakraborty D, Smith AH (1998). Arsenic levels in drinking water and the prevalence of skin lesions in West Bengal, India. *International Journal of Epidemiology,* 27(5):871-877.

Mazumder DNG (2008). Chronic arsenic toxicity and human health. *Indian Journal of Medical Research,* 128:436-447.

NOTE: Important general information is contained in PART II, Chapter 6

Mazumder DNG, Majumdar KK, Santra SC, Kol H, Vicheth C (2009). Occurrence of arsenicosis in a rural village of Cambodia. *Journal of Environmental Science and Health, Part A*, 44(5):480 - 487.

MoH NZ (Ministry of Health New Zealand) (2008). *New Zealand Ministry of Health, Drinking-water standards for New Zealand* 2005 (Revised 2008), p. 175. Ministry of Health, Wellington.

Smith AH, Steinmaus CM (2009). Health effects of arsenic and chromium in drinking water: recent human findings. *Annual Review of Public Health*, 30:107-122.

USEPA (Unites States Environmental Protection Agency) (2008). National Primary Drinking Water Standards. Available Online: http://www.epa.gov/safewater/consumer/pdf/mcl.pdf

WHO (World Health Organization) (2003). Arsenic in drinking-water: Background document for development of WHO Guidelines for Drinking-water Quality, WHO.

WHO (World Health Organization) (2004). Guidelines for Drinking-water Quality. First addendum to 3rd edition. Volume 1. Recommendations. WHO.

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