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Benzo[a]pyrene

Guideline

The maximum acceptable concentration (MAC) for benzo[a]pyrene in drinking water is 0.00001 mg/L (0.01 µg/L).

Identity, Use and Sources in the Environment

Benzo[a]pyrene, BaP, is a member of a class of compounds, known as the polycyclic aromatic hydrocarbons (PAHs), in which the molecular structure includes two or more fused aromatic rings and adjacent rings share two or more carbon atoms. Benzo[a]pyrene is considered here because it is the only PAH for which there is sufficient toxicological evidence to allow the setting of a guideline. It is a solid, melting at 179°C; it is freely soluble in aromatic hydrocarbon solvents but has only a limited solubility in water, which ranges from 0.2 to 6.1 µg/L.[1,2]

Benzo[a]pyrene is not manufactured and has no industrial uses. It is ubiquitously distributed throughout the environment as a consequence of its formation during the combustion of organic matter. There is no quantitative relation between measured BaP and concentrations of any other PAH; however, if BaP is found, other PAHs are probably also present. The principal natural sources of BaP are forest fires and volcanoes during eruptions.[3] Anthropogenic sources include the combustion of fossil fuels, coke oven emissions and vehicle exhausts.[4,5] Based on an inventory of Canadian emissions,[2] it has been estimated that 19 000 to 22 000 kg are emitted annually from anthropogenic sources (i.e., industrial processes, fossil fuel combustion and transportation). Direct atmospheric input appears to be the major source of BaP in surface waters. The annual deposition rate of BaP in the Great Lakes, for example, is proportional to their surface area and ranges from 9.1 to 9.8 ng/cm^2.[6] Exfoliation from asphaltic or bituminous liners in water distribution systems may also contribute to the BaP content of drinking water.

Exposure

Data on the amounts of BaP in drinking water are sparse. In a recent survey of seven water treatment plants in the Niagara area, BaP was not detected using methodology with a detection limit of 1 µg/L.[7] Based on a review of the literature, it has been reported that concentrations of BaP can range from 0 to 1000 ng/L in tap water,[8] but a typical level in U.S. drinking water is 0.55 ng/L.[9] In the absence of any pertinent data, it is not possible to assess individual exposure to BaP in tap water from routes other than the ingestion of drinking water. In view of its physicochemical properties, inhalation exposure to BaP from drinking water sources would appear to be limited. No data are available on the rate of percutaneous absorption of BaP from aqueous solution.

The principal sources of BaP in foods are the absorption and deposition of particulates during processing (smoked foods, leafy vegetables), the pyrolysis of fats and the incomplete combustion of charcoal. Typical concentrations (µg/kg) of BaP in food products are: broiled sausage, 0.17 to 0.63; charcoal-broiled meat, 2.60 to 11.20; spinach, 7.40; crude coconut oil, 43.7; and tea, 3.90 to 21.3.[10]

In ambient air, BaP (as well as PAHs in general) is normally associated with fine particulates.[8] In a recent review, it was reported that atmospheric concentrations of BaP in summer in urban centres average 3.6 ng/m^3 (standard deviation 4.0).[11] In winter, concentrations are higher (mean 7.1 ng/m^3; standard deviation 5.1), probably because of the contribution from industrial and domestic heating, which uses fossil fuels.

Significant amounts of BaP are inhaled in tobacco smoke. The amount of BaP per cigarette is between 18 and 50 ng, and the total PAHs can amount to as much as 248 ng.[3]

Drinking water is estimated to account for only 0.1 to 0.3% of the total BaP ingested. Air is estimated to contribute about 0.9% of the total exposure, and the greatest source is foods, which contribute 99%.[12]

Analytical Methods and Treatment Technology

Benzo[a]pyrene and other PAHs can be analysed using gas chromatography in conjunction with mass spectrometry. The practical quantitation limit (PQL) (based on the ability of laboratories to measure BaP within reasonable limits of precision and accuracy) is 0.01 µg/L.[13]

The conventional water treatment processes of alum coagulation, settling and sand filtration are capable of reducing the BaP concentration of surface waters to less than 0.001 µg/L, even if the influent concentration is high.[1]

Health Effects

There is good evidence to suggest that the physicochemical properties of BaP allow its rapid uptake and transport through systemic circulation. The principal sites of uptake are the gastrointestinal tract, following the ingestion of food, and the lungs, following the inhalation of contaminated aerosols and particulate matter. Distribution to the well-perfused organs and tissues occurs within minutes.[14] Excretion is via the urine as water-soluble metabolites, although some enterohepatic recycling (biliary excretion followed by reabsorption into the systemic circulation) also occurs. Benzo[a]pyrene crosses the placenta and is readily distributed to the developing foetus.[15] The lipophilic nature of BaP favours its distribution and storage in fatty tissues, including mammary fat and bone marrow.

Metabolism of BaP takes place in two steps. The initial step, or Phase I metabolism, involves its interaction with mixed-function oxidases and the formation of diol-epoxides, including trans-9,10-epoxy-7,8-dihydrodiol BaP. This diol-epoxide is considered to be the ultimate carcinogen. It is noteworthy that BaP (as well as other PAHs) is a potent inducer of mixed-function oxidases and may thus potentiate its own toxicity. Phase II metabolism involves the conjugation of the metabolic intermediates with glutathione, sulphates and mercapturic or glucuronic acids. These conjugates are much more water-soluble than the parent compound, which allows their rapid excretion via the kidney. Although the liver is considered to be the primary site of metabolism, significant metabolism can occur in tissues of the lung, gastrointestinal tract, placenta and skin. The endothelial tissues are known to be particularly active.[16]

No human health effects have been unequivocally associated with exposure to BaP *per se*. Acute, subchronic and chronic studies in animals to examine effects other than carcinogenesis are not plentiful because of the prevailing concern about the role of the compound as a proximal carcinogen. Doses at least an order of magnitude greater than those that result in neoplastic lesions are required to induce other effects. The only LD_{50} datum available, 250 mg/kg bw, is for mice exposed by the intraperitoneal route.[8] Other effects induced in animals following generally acute exposure to high concentrations of BaP include inflammation of the skin, hyperplasia, hyperkeratosis, pneumonitis, modifications of the lymph nodes, ulceration, reduction in growth and fertility rates and the induction of immunosuppressive effects.[9]

Benzo[a]pyrene is carcinogenic in a variety of species and by a number of routes. Tumours have been produced in mice, rats, hamsters, guinea pigs, rabbits, ducks and monkeys following intragastric, subcutaneous, dermal or intratracheal administration of BaP. The induced primary tumours may be at the site of administration or remote from it. The preferred target sites appear to be proliferating tissues of the intestinal epithelia, bone marrow, lymphoid organs and testes, which interact with the active metabolite of BaP in their S-phase of the mitotic cycle.[9]

The only carcinogenesis bioassay in which BaP has been administered orally to animals is that of Neal and Rigdon.[17] This study is, therefore, the most appropriate for assessing the risk associated with the ingestion of BaP in drinking water. In this investigation, CFW strain mice were given BaP in laboratory chow diet at concentrations of 0.001, 0.01, 0.02, 0.03, 0.04, 0.045, 0.05, 0.10 and 0.25 mg/g of food, for approximately 110 days. Stomach tumours — mostly squamous cell papillomas with some carcinomas — were found, and their frequency was significant relative to controls and dose-related.

Benzo[a]pyrene is mutagenic when S-9 activated liver enzymes and TA1538 *Salmonella* are used in an Ames test.[18] Further, the diol-epoxide metabolites of BaP are considerably more mutagenic than the parent compound. Forward mutations are also induced by BaP in cultured mammalian cells, and induction of sister chromatid exchanges in Chinese hamsters, after intraperitoneal administration, has also been observed.[19] Teratogenic effects of BaP are questionable,[9] although transplacental carcinogenic action is known to occur after the subcutaneous administration of large doses.[20]

Classification and Assessment

The evidence for the carcinogenicity of BaP is sufficient to classify it in Group II — probably carcinogenic to man (sufficient evidence in animals; inadequate or no data in man), and cancer risks have been estimated on the basis of the feeding study of Neal and Rigdon using CFW strain mice.[17] Incorporating a surface area correction and using the robust linear extrapolation model for the significant increase in stomach tumours (squamous cell papillomas and some

carcinomas), the estimated lifetime risk associated with the ingestion of 1 µg/L BaP in drinking water is 5×10^{-5}.* The estimated concentrations in drinking water corresponding to lifetime risks of 10^{-5}, 10^{-6} and 10^{-7} for these tumour types based on the model described above are 0.2, 0.02 and 0.002 µg/L, respectively.

Rationale

Because BaP is classified in Group II (probably carcinogenic to man), the MAC is derived based on consideration of available practicable treatment technology and estimated lifetime cancer risks. Because the MAC must also be measurable by available analytical methods, the PQL is also taken into consideration in its derivation.

An MAC of 0.01 µg/L was established, therefore, on the basis of the following considerations:

(1) The estimated unit lifetime risk associated with the ingestion of 1 µg/L BaP in drinking water is 5×10^{-5}, based on increased incidence of stomach tumours (squamous cell papillomas and some carcinomas). Therefore, the estimated lifetime risk associated with the ingestion of drinking water containing 0.01 µg/L BaP (i.e., 5×10^{-7}) is within a range that is considered to be "essentially negligible."

(2) The conventional water treatment processes of alum coagulation, settling and sand filtration are capable of reducing BaP concentrations to less than 0.001 µg/L.

(3) The PQL (based on the ability of laboratories to measure BaP within reasonable limits of precision and accuracy) is 0.01 µg/L.

References

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