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Dimethoate

Guideline

The interim maximum acceptable concentration (IMAC) for dimethoate in drinking water is 0.02 mg/L (20 µg/L).

Identity, Use and Sources in the Environment

Dimethoate is an organophosphorus insecticide and acaricide used for the control of houseflies, as well as a wide range of insects and mites on a variety of fruit, vegetable, field and forestry crops. More than 100 000 kg are used annually in Canada.[1]

Dimethoate has a vapour pressure of 1.1×10^{-3} Pa at 25°C; its solubility in water at 21°C is 25 g/L.[2] Reported log octanol–water partition coefficients are 0.78 and 0.79.[3]

Dimethoate released to the environment does not adsorb onto the soil and is subject to considerable leaching. It is also lost from the soil through evaporation and biodegradation. The half-life of dimethoate in soil ranges from four to 16 days.[4] It is relatively stable in aqueous media at pH 2 to 7.[5] Reported half-lives for dimethoate in raw river water range from 18 hours to eight weeks.[4] Dimethoate is degraded in the environment to another more toxic pesticide, omethoate; the proportion of omethoate in the total residue reaches about 50% after five weeks.[6]

Exposure

Dimethoate was not detected in 98 samples from municipal and private drinking water supplies in Nova Scotia, Quebec, Metropolitan Toronto and Manitoba surveyed from 1971 to 1986 (reported detection limits 0.2 and 0.6 µg/L).[7] It was detected at trace levels in a private well in Nova Scotia (detection limit 0.01 µg/L).[8] It was not detected in a survey of surface water at 38 sites in the Prairies from 1973 to 1974 (detection limit not reported),[7] nor was it found in 11 agricultural watersheds in southern Ontario from 1975 to 1977 (detection limit 0.5 µg/L)[9] or in three Ontario river basins from 1981 to 1985 (detection limit 0.5 µg/L).[10]

The theoretical maximum daily intake of dimethoate from food is 0.46 mg/d, based on the residue tolerance limits set by the Food Directorate of the Department of National Health and Welfare.[11] Based on market basket surveys in the United States, the actual average daily intake of dimethoate by adults has been estimated to be 0.008 µg/kg bw.[12] Dimethoate was found in 358 of 6391 U.S. domestic food samples analysed; 96% of the samples had levels at or below 2 ppm.[13]

Analytical Methods and Treatment Technology

The concentration of dimethoate in water may be determined by extracting into dichloromethane, drying the extract and redissolving it in hexane, and analysing by gas–liquid chromatography with flame photometric detection (phosphorus mode; detection limit 0.5 µg/L).[9,10]

No information on the effectiveness of current treatment technologies in removing dimethoate from drinking water was identified.

Health Effects

Dimethoate is readily absorbed from the gastro-intestinal tract; 76 to 100% of an orally administered dose (quantity unspecified) was excreted in the urine of human volunteers within 24 hours.[14] In rats, about 60% of an administered dose (quantity unspecified) was excreted in the urine and expired air in 24 hours.[15] The principal metabolite of dimethoate is the thiocarboxy derivative.[16] Based on comparisons of the rate of degradation in liver specimens from humans and other species and acute toxicity data in animals, the oral LD_{50} for humans is predicted to be about 30 mg/kg.[16]

Dimethoate is a cholinesterase inhibitor. Thirty-six male and female human volunteers were administered oral doses of dimethoate of 5, 15, 30, 45 and 60 mg/d for periods of 14 to 57 days. There were no significant effects on cholinesterase levels in 12 persons ingesting 5 mg/d for 28 days or in nine persons ingesting 15 mg/d for 39 days. There was a decrease in whole blood

cholinesterase activity by day 20 in the eight volunteers receiving 30 mg/d, which persisted until the end of the study. This depression occurred earlier and to a somewhat greater degree in the higher dose groups. The no-observed-adverse-effect level (NOAEL) was considered to be 15 mg/d, or 0.2 mg/kg bw per day.[17] In other studies in humans, no inhibition of cholinesterase activity was observed in two adults ingesting 9 and 18 mg/d for 21 days or in 20 adults ingesting 2.5 mg/d for four weeks.[14]

The NOAEL for erythrocyte and plasma cholinesterase inhibition of dimethoate administered daily in the diet of dogs was 10 and 50 ppm, respectively, or 0.2 and 1.0 mg/kg bw per day.[18] In rats, the reported NOAEL for cholinesterase inhibition has been found to range from 1 ppm (about 0.05 mg/kg bw per day) to 32 ppm dimethoate in the diet.[2]

On the basis of the results of an early study, the U.S. National Cancer Institute (NCI) concluded that dimethoate was not carcinogenic in Osborne-Mendel rats and B6C3F$_1$ mice.[19] However, recent histological examination and re-evaluation of the NCI and other studies indicate that dimethoate is oncogenic in two strains of rats and probably in mice. Benign and malignant neoplasms of the liver, endocrine organs and lymphatic system were observed as well as other toxic effects (atrophy of the testes, chronic renal disease and parathyroid hyperplasia).[20] Owing to inadequacies of the available studies, the International Agency for Research on Cancer (IARC) was unable to classify dimethoate with regard to its potential carcinogenicity.[21]

Dimethoate was mutagenic in a number of *in vivo* and *in vitro* short-term tests.[6] In mice drinking water containing 60 ppm (9.5 to 10.5 mg/kg bw per day) dimethoate, there were adverse reproductive effects on mating success, survival of pups and growth of surviving pups; however, no teratogenic effects were observed.[22] Effects on foetal development were observed in cats and rats exposed to 12 mg/kg bw per day but not at 3 or 6 mg/kg bw per day in either species.[23,24]

Rationale

In 1984, the Food and Agriculture Organization (FAO) and the World Health Organization (WHO)[6] withdrew their estimate of an acceptable daily intake (ADI) for dimethoate because of the incomplete data base. Until further experimental studies are completed, a temporary ADI has been established by WHO[6] as follows:

$$\text{ADI} = \frac{0.2 \text{ mg/kg bw per day}}{100} = 0.002 \text{ mg/kg bw per day}$$

where:

- 0.2 mg/kg bw per day is considered to be the NOAEL from studies in human volunteers[17]
- 100 is the uncertainty factor.

Based on the above temporary ADI, the interim maximum acceptable concentration (IMAC) for dimethoate in drinking water is derived as follows:

$$\text{IMAC} = \frac{0.002 \text{ mg/kg bw per day} \times 70 \text{ kg bw} \times 0.20}{1.5 \text{ L/d}} \approx 0.02 \text{ mg/L}$$

where:

- 0.002 mg/kg bw per day is the temporary ADI established by the FAO/WHO
- 70 kg bw is the average body weight of an adult
- 0.20 is the proportion of daily intake of dimethoate allocated to drinking water
- 1.5 L/d is the average daily consumption of drinking water by an adult.

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