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Parathion

Guideline

The maximum acceptable concentration (MAC) for parathion in drinking water is 0.05 mg/L (50 µg/L).

Identity, Use and Sources in the Environment

Parathion ($C_{10}H_{14}NO_5PS$) is an organophosphorus insecticide and acaricide used to control soil-dwelling pests and a wide range of insects and mites on a variety of agricultural crops. Between 10 000 and 50 000 kg are used annually in Canada.[1]

The vapour pressure of parathion is 5.0×10^{-3} Pa at 20°C,[2] and its solubility in water is 24 mg/L at 25°C.[3] Reported log octanol–water partition coefficients range from 3.40 to 3.93.[4]

*P*arathion released to the environment will adsorb strongly onto the top layer of soil and is not likely to leach significantly. It is degraded by photolysis on the surface and by both biological and soil-catalysed hydrolysis below the surface layer. Parathion disappears from surface water in about a week. Products of degradation include p-nitrophenol, diethylthiophosphoric acid and paraoxon.[5]

Exposure

Parathion was not detected in 248 samples of municipal and private drinking water supplies from Prince Edward Island, Quebec, Ontario, Saskatchewan and Manitoba analysed from 1971 to 1986 (detection limits ranged from 0.001 to 0.2 µg/L).6 It was not found in 949 samples from stream waters of 11 southern Ontario agricultural watersheds from 1975 to 1977 (detection limit 0.1 µg/L),7 nor was it detected in 446 samples from three Ontario river basins from 1981 to 1985 (detection limit 0.1 µg/L).8

The theoretical maximum daily intake of parathion from food is 0.4 mg/d, based on the residue tolerance limits set by the Food Directorate of the Department of National Health and Welfare.9 Based on U.S. market basket surveys, the actual average daily intake has been estimated to be 0.166 µg/d.10 Detectable concentrations of parathion and its oxygen analogue were found in only 50 of 6391 U.S. domestic food samples; in 86% of the samples, levels were 0.5 ppm or less.[11]

Analytical Methods and Treatment Technology

The concentration of parathion in water may be determined by extracting into dichloromethane, drying the extract and redissolving it in hexane, and analysing by gas–liquid chromatography (phosphorus mode; detection limit 0.1 µg/L).[7,8]

Little information was identified on the effectiveness of current treatment technologies in removing parathion from drinking water. Coagulation does not appear to be effective.[12]

Health Effects

Parathion is readily absorbed from the gastrointestinal tract and through the skin. It is converted to the oxygen analogue, paraoxon, which is the active form. Parathion and paraoxon are metabolized and excreted as p-nitrophenol and ethyl and diethyl esters of phosphoric and/or thiophosphoric acid.[13]

Parathion is a cholinesterase inhibitor. Human volunteers (four per group) were administered oral doses of 0, 0.6 (increased to 4.8 after four weeks), 1.2, 2.4 or 7.2 mg/d for 25 to 70 days. In the highest dose group, whole blood, red cell and plasma cholinesterase levels had decreased to 67%, 84% and 63% of control levels, respectively, within six weeks. No effects on cholinesterase levels were reported in the group administered 0.6 to 4.8 mg/d, which the authors considered to be equivalent to 0.05 mg/kg bw per day.[14] Prison volunteers (five per dose group) were administered parathion in capsules at levels of 3.0, 4.5, 6.0 or 7.5 mg/d (or 0.043, 0.064, 0.086 and 0.11 mg/kg bw per day, respectively) for up to 30 days. There was an average decrease in plasma cholinesterase of 28% of the control level by day 16 in the highest dose group; the maximum depression in any individual in this group was to 50% of the pre-test level after 16 days of treatment. There was a slight depression of plasma cholinesterase

(extent not reported) in those ingesting 0.086 mg/kg bw per day of parathion. Doses of 0.043 and 0.064 mg/kg bw per day had no effect on cholinesterase activity.[15] Based on reports of incidental poisonings, children appear to be more susceptible to the toxic effects of parathion than adults.[16]

Groups of 20 female rats (strain unspecified) were fed doses of 0.02, 0.04 or 0.06 mg/kg bw per day of parathion for 84 days. There were no effects on cholinesterase activity in animals exposed to the lowest dose and only minimal effects on plasma cholinesterase activity at the two higher dose levels. The no-effect level was considered to be 0.02 mg/kg bw per day.[14] Dogs were fed diets containing 1, 2 or 5 ppm parathion (or 0.021, 0.047 and 0.117 mg/kg bw per day, respectively) for 24 weeks. A minimal but significant reduction in plasma cholinesterase was reported in animals ingesting 1 ppm; at the higher doses, plasma cholinesterase was reduced by 60 to 70%.[17]

The International Agency for Research on Cancer (IARC)[18] reviewed carcinogenicity bioassays conducted by the U.S. National Cancer Institute (NCI) and other researchers. A dose-related increase in the incidence of adrenal cortical adenomas and carcinomas was reported in male and female Osborne-Mendel rats by the NCI. However, IARC considered the results of this study to be inconclusive because the significance of the occurrence of adrenal cortical adenomas in aged rats is not well understood and because the number of carcinomas was low. As the NCI study was limited and additional studies in rats (strain unspecified) and a study in $B6C3F_1$ mice were considered to be inadequate, IARC concluded that there is inadequate evidence to evaluate the carcinogenicity of parathion in animals.

Chromosomal abnormalities were detected in *in vivo* studies on male guinea pigs injected inter-testicularly with parathion, indicating an inhibition of cell division at metaphase.[19] In studies reviewed by IARC, parathion was not mutagenic in a wide range of micro-organisms with or without rat liver microsomal preparations; it did not induce unscheduled DNA synthesis in cultured mammalian cells, recessive lethal mutations in *Drosophila melanogaster* or dominant lethal mutations in mice.[18]

Parathion caused a reduction of growth in foetal rats and mice and produced toxic effects, including a decrease in pseudocholinesterase and plasma renin activities and an increase in resorptions and post-natal mortality, following exposure of the dams during gestation; however, no malformations of developing embryos have been reported.[18]

Rationale

The acceptable daily intake (ADI) of parathion has been derived by the Food and Agriculture Organization (FAO) and the World Health Organization (WHO)[20] as follows:

$$\text{ADI} = \frac{0.05 \text{ mg/kg bw per day}}{10} = 0.005 \text{ mg/kg bw per day}$$

where:
- 0.05 mg/kg bw per day is considered to be the no-observed-adverse-effect level (NOAEL) derived from studies in rats, dogs and human volunteers
- 10 is the uncertainty factor.

Based on the above ADI, the maximum acceptable concentration (MAC) for parathion in drinking water is derived as follows:

$$\text{MAC} = \frac{0.005 \text{ mg/kg bw per day} \times 70 \text{ kg bw} \times 0.20}{1.5 \text{ L/d}} \approx 0.05 \text{ mg/L}$$

where:
- 0.005 mg/kg bw per day is the 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 parathion allocated to drinking water
- 1.5 L/d is the average daily consumption of drinking water by an adult.

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