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Carbaryl

Guideline

The maximum acceptable concentration (MAC) for carbaryl in drinking water is 0.09 mg/L (90 μg/L).

Identity, Use and Sources in the Environment

Carbaryl ($C_{12}H_{11}NO_2$) is a carbamate insecticide used for the control of a variety of pests on fruit, vegetables, cotton and other crops. Between 100 000 and 200 000 kg are used annually in Canada.[1] The solubility of carbaryl in water is 40 mg/L at 30°C; its vapour pressure at 26°C is less than 0.7 Pa.[2] The log octanol–water partition coefficient is reported to range from 2.31 to 2.86;[3] therefore, carbaryl is not likely to bioaccumulate significantly.

The degradation of carbaryl in water is influenced by temperature, light and pH.[4] It is rapidly hydrolysed to 1-naphthol in neutral and alkaline waters; half-lives at 20°C of 10.5 days, 1.8 days and 2.5 hours are reported at pH 7, 8 and 9, respectively. Hydrolysis is much slower in acidic waters; half-lives of 1500 days at pH 5 and 27°C and 406 days at pH 6 and 25°C have been reported.[5] Carbaryl is degraded in soil, undergoing photolysis, hydrolysis and microbial action. It is moderately mobile in the soil and may leach to groundwater.[5]

Exposure

Carbaryl was not detected in 199 samples of treated municipal and private water supplies from all 10 provinces in 1985. It was detected only once (at 7.3 mg/L) in 307 samples from all provinces except Ontario in 1986, although it was not detected when the site was resampled (detection limit 1.0 μg/L).[6] Carbaryl was not detected in 314 samples from three Ontario river basins during 1981 to 1985 (detection limit 1.0 μg/L), although total annual use of the pesticide in the regions was nearly 4000 kg.[7]

Based on the residue tolerance limits set by the Food Directorate of the Department of National Health and Welfare,[8] the theoretical maximum daily intake of carbaryl from food is 4.1 mg/d. Actual intake of carbaryl is likely to be much lower; average daily intake for an adult male is estimated to be 1.37 μg.[9] Carbaryl was detected in only 21 of 6391 U.S. domestic food samples surveyed from 1981 to 1986 (detection limit not reported), 76% of which had levels at or below 2.0 ppm.[10]

Analytical Methods and Treatment Technology

The concentration of carbaryl in water may be determined by extraction of the pesticide into dichloromethane, hydrolysis, derivatization and separation by gas–liquid chromatography with electron capture detection (detection limit 1.0 μg/L).[7]

No information was found on the effectiveness of current treatment technologies in removing carbaryl from drinking water supplies.

Health Effects

Carbaryl is very readily absorbed by the gastrointestinal tract and is rapidly metabolized. An oral dose of carbaryl (quantity unspecified) was 53% and 82% absorbed in rats in 20 minutes and one hour, respectively.[11] Metabolites of carbaryl are eliminated in the urine, exhaled breath, faeces and bile. Urinary metabolites identified in two human volunteers administered single doses of 2 mg/kg bw carbaryl included 1-naphthol and its glucuronide and sulphate conjugates, 4-hydroxycarbaryl and a minor amount of 1-naphthyl methylimido carbamate-O-glucuronide. Recovery of 26% and 28% of the administered dose in the urine was reported within four days.[12] Carbaryl crosses the placental barrier in rats and mice.[2]

The acute toxicity of carbaryl is due to its ability to inhibit cholinesterase activity. However, because carbaryl is rapidly metabolized, its acute oral toxicity is low, and toxic effects are rapidly reversible.

Groups of five and six human volunteers ingested doses of 0, 0.06 or 0.12 mg/kg bw per day for six months. No adverse effects were observed in the group receiving 0.06 mg/kg bw per day. In the higher dose group, there was an increase in the ratio of the

concentration in urine of amino acid nitrogen to that of creatinine, indicative of a decrease in the ability of the proximal convoluted tubule of the kidney to reabsorb amino acids. This effect appeared to be reversible, as the ratio returned to normal after treatment was discontinued. The authors concluded that repeated oral doses of 0.12 mg/kg bw per day were "unsafe."[13]

No relationship was found between exposure to a variety of compounds (including carbaryl) used in a forest spraying program to control spruce budworm and the incidence of Reye's syndrome in children in an epidemiological study in New Brunswick.[14]

There were no adverse effects in rats consuming food containing 200 mg/kg carbaryl (equivalent to a dose of 10 mg/kg bw per day) for two years. At 400 mg/kg diet, cloudy swelling of the kidney tubules and the central hepatic cords was noted.[15] Diffuse cloudy swelling of the kidney tubules was observed in dogs fed 7.2 mg/kg bw per day for five days per week for one year. No adverse effects were noted at 1.8 mg/kg bw per day.[16]

Groups of 18 male and 18 female (C57BL/6xC3H/Anf)F_1 mice and 18 male and 18 female (C57BL/6xAKR)F_1 mice were fed commercial carbaryl at a concentration equivalent to a dose of 4.64 mg/kg bw per day from seven days of age for four weeks, after which time they were given carbaryl at 14 mg/kg diet up to 78 additional weeks. There was no significant increase in the incidence of tumours of any type in any group.[11] No significant increase in tumours was reported in groups of 20 male and 20 female CF-N rats administered doses of carbaryl of 0, 2, 4, 8 or 16 mg/kg bw per day in the diet for up to 736 days.[15]

Carbaryl was not mutagenic in *in vivo* tests in mice or rats, nor did it increase the percentage of abnormal sperm or decrease the sperm count in humans. Chromosomal damage has been observed in *in vivo* tests in human, rat and hamster cells at doses producing cell death. Negative results were obtained in a number of bacterial assay systems.[12]

Carbaryl produced developmental effects in several studies in rats, mice, gerbils, hamsters, rabbits, pigs and dogs, but only at dose levels that were maternally toxic. Teratogenic effects, such as abdominal-thoracic fissures with varying degrees of brachygnathia, ecaudate pups, failure of skeletal formation and superfluous phalanges, have been observed in two studies in beagle dogs at doses of 5.0 mg/kg bw per day and greater.[17,18] However, it should be noted that maternal toxicity was not described in these studies, although cholinesterase inhibition would be expected at this level.[12]

Carbaryl does not produce any delayed neurotoxic effects or cause myelin degeneration.[12]

Rationale

The acceptable daily intake (ADI) of carbaryl in drinking water has been established by the Food and Agriculture Organization (FAO) and the World Health Organization (WHO)[19] as 0.01 mg/kg bw per day, based on the no-observed-adverse-effect levels of 0.06 mg/kg bw per day in human volunteers[13] and 10 mg/kg bw per day in rats.[15]

The maximum acceptable concentration (MAC) of carbaryl in drinking water may be derived as follows:

$$\text{MAC} = \frac{0.01 \text{ mg/kg bw per day} \times 70 \text{ kg bw} \times 0.20}{1.5 \text{ L/d}} \approx 0.09 \text{ mg/L}$$

where:

- 0.01 mg/kg bw per day is the ADI established by the FAO/WHO[19]
- 70 kg bw is the average body weight of an adult
- 0.20 is the proportion of daily intake of carbaryl allocated to drinking water
- 1.5 L/d is the average daily consumption of drinking water by an adult.

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