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Diclofop-methyl

Guideline

The maximum acceptable concentration (MAC) for diclofop-methyl in drinking water is 0.009 mg/L (9 μg/L).

Identity, Use and Sources in the Environment

Diclofop-methyl is a chlorophenoxy derivative used in large quantities (over 1 million kilograms in Canada in 1986) for the control of annual grasses in grain and vegetable crops.[1]

Diclofop-methyl is relatively soluble in water (50 mg/L at 22°C) and common organic solvents. It has a very low vapour pressure of 3.4×10^{-8} kPa at 20°C. It is stable at normal pH but is hydrolysed at strong acid or alkaline pH.[2]

The U.S. Environmental Protection Agency does not consider diclofop-methyl to be a groundwater contaminant; it was therefore not included in the U.S. EPA survey on potential groundwater contaminants.[3] Diclofop-methyl ranked very low with respect to potential for groundwater contamination in a similar Agriculture Canada survey.[4]

Exposure

Diclofop-methyl was not detected in 78 municipal drinking water supplies in Manitoba and Alberta.[5] It was detected in 1% of private farm wells in southern Ontario.[6] It has occasionally been detected in surface waters, with a maximum concentration of 4 μg/L recorded in the LaSalle River, Manitoba.[5]

Based on Canadian residue limits for diclofop-methyl[7] and on Canadian consumption patterns,[8] the theoretical maximum dietary exposure to diclofop-methyl for an adult Canadian would be 0.0455 mg/d, or 0.00065 mg/kg bw per day. This intake is greater than actual intake, as it assumes that every crop for which diclofop-methyl is registered is treated, and that residues are present in all crops at the maximum residue level of 0.1 μg/g. No actual residue levels in foods are available, as diclofop-methyl was not included in total diet residue surveys in either Canada or the United States.

Analytical Methods and Treatment Technology

Diclofop-methyl is analysed by gas chromatography followed by electron capture detection; the detection limit is about 0.1 μg/L,[9] and the quantitation limit can therefore be expected to be about 0.5 μg/L.

No information has been found on the effectiveness of current treatment technologies in removing diclofop-methyl from drinking water.

Health Effects

Diclofop-methyl is fairly readily absorbed through the gastrointestinal tract. In rats, 59% of a single oral dose (dose unknown) was excreted after 24 hours. After seven days, 90% was excreted — 70% in faeces and 20% in urine. In the faeces, 20% of the radioactivity from three repeated oral daily doses was excreted as unchanged parent compound.[10] The compound is metabolized to one minor and two major metabolites,[10] with elimination of hydroxylated metabolites as conjugated compounds.[2]

The acute toxicity of diclofop-methyl is relatively low.[2] Its principal toxic action is on the liver. A no-observed-adverse-effect level (NOAEL) of 8 μg/g in the diet or 0.2 mg/kg bw per day was established in a 15-month study on dogs, with increased liver weights, increased enzyme levels and centrilobular fatty deposits noted at higher doses (0.63 and 2 mg/kg bw per day).[10,11] In a two-year study in rats in which the compound was administered at doses of 0, 2, 6.3 or 20 μg/g in the diet (about 0. 0.1, 0.3, and 1 mg/kg bw per day), some liver enzyme changes (elevations in serum glutamic acid phosphatase and alkaline phosphatase) were noted at 20 μg/g food (equivalent to 1 mg/kg bw per day). Elevation in relative and absolute weights of the ovaries was noted at 2 or 6.3 μg/g food (about 0.1 and 0.3 mg/kg bw per day, respectively).[10] In a two-year mouse feeding/oncogenicity study in which diclofop-methyl was administered at doses of 0, 2, 6.3 or 20 μg/g in the diet (about 0, 0.1, 0.3 or 1 mg/kg bw per day), a NOAEL of 2 μg/g in the diet or approximately

0.1 mg/kg bw per day was established for systemic effects, primarily elevated liver enzyme levels (serum glutamic acid phosphatase and alkaline phosphatase) and liver pathology.[10]

There was no evidence of tumorigenicity in the two-year rat study.[10] In the two-year mouse study, a statistically significant increase in hepatocellular benign tumours was noted in male mice, along with some malignant tumours at the highest dose — 20 µg/g in the diet or approximately 1 mg/kg bw per day.[10,11] These were considered to be in the category of limited evidence for tumorigenicity;[10] no metastases were observed, and tumours were in one sex of a species noted for production of hepatocellular tumours of this type in control as well as experimental animals.

Diclofop-methyl was non-genotoxic in a battery of short-term tests, including Ames mutagenicity tests, an *in vivo* micronucleus test in bone marrow cells of mice orally dosed at 0, 10, 32 or 100 mg/kg bw per day, a gene conversion study in yeast, a dominant lethal test in mice at doses of 0, 10, 32 or 100 mg/kg bw per day and an unscheduled DNA synthesis study in rat hepatocytes *in vitro*.[10,11]

Diclofop-methyl had no effect on reproduction at any dose up to 5 mg/kg bw per day in a three-generation rat reproduction study. The NOAEL for systemic maternal toxicity (liver weight increases and abnormal pathology) was 30 µg/g in the diet, or 1.5 mg/kg bw per day.[10] No teratological effects were noted in studies on rats and rabbits at doses up to 100 µg/g in the diet, or 5 mg/kg bw per day in the rat and 3 mg/kg bw per day in the rabbit.[3,10] However, in the rat study, there were indications of embryotoxicity at all dose levels, including the lowest dose of 10 µg/g in the diet or 0.5 mg/kg bw per day.[10]

Rationale

Based on evaluations by the Food Directorate of the Department of National Health and Welfare,[11] an acceptable daily intake (ADI) for diclofop-methyl is derived as follows:

$$\text{ADI} = \frac{0.1 \text{ mg/kg bw per day}}{100} = 0.001 \text{ mg/kg bw per day}$$

where:

- 0.1 mg/kg bw per day is the NOAEL for liver enlargement and liver enzyme changes in two-year feeding studies on rats and mice[10,11]
- 100 is the uncertainty factor assigned by the Food Directorate of the Department of National Health and Welfare (×10 for interspecific variability; ×10 for intraspecific variability).

The maximum acceptable concentration (MAC) for diclofop-methyl in drinking water is derived from the ADI as follows:

$$\text{MAC} = \frac{0.001 \text{ mg/kg bw per day} \times 70 \text{ kg} \times 0.20}{1.5 \text{ L/d}} \approx 0.009 \text{ mg/L}$$

where:

- 0.001 mg/kg bw per day is the ADI, as derived above
- 70 kg is the average body weight of an adult
- 0.20 is the proportion of daily intake of diclofop-methyl allocated to drinking water (the theoretical maximum food intake is 65% of the ADI)
- 1.5 L/d is the average daily consumption of drinking water for an adult.

References

1. Environment Canada/Agriculture Canada. Pesticide Registrant Survey, 1987 report. Commercial Chemicals Branch, Conservation and Protection, Environment Canada, Ottawa (1987).

2. The Royal Society of Chemistry. The agrochemicals handbook. Nottingham (1983).

3. U.S. Environmental Protection Agency. EPA draft final list of recommendations for chemicals in the National Survey for Pesticides in Groundwater. Chem. Regul. Rep., 9(34): 988 (1985).

4. Agriculture Canada. Pesticide priority scheme for water monitoring program. Unpublished report, Pesticides Directorate, Agriculture Canada (1986).

5. Hiebsch, S. The occurrence of thirty-five pesticides in Canadian drinking water and surface water. Unpublished report prepared for the Environmental Health Directorate, Department of National Health and Welfare, January (1988).

6. Frank, R., Clegg, S., Ripley, B. and Braun, H. Investigations of pesticide contamination in rural wells, 1979–84, Ontario, Canada. Arch. Environ. Contam. Toxicol., 16: 19 (1987).

7. Department of National Health and Welfare. National pesticide residue limits in foods. Chemical Evaluation Division, Food Directorate, Ottawa (1986).

8. Department of National Health and Welfare. Food consumption patterns report. Nutrition Canada, Bureau of Nutritional Sciences (1977).

9. U.S. Environmental Protection Agency. Pesticide tolerances for diclofop-methyl. Fed. Regist., 51(102): 1975 (1986).

10. Department of National Health and Welfare. Unpublished studies; summary of evaluations by the Food Directorate (1988).

11. Department of National Health and Welfare. Memorandum from D. Clegg, Food Directorate, to P. Toft, Environmental Health Directorate, August 6 (1986).