February 1986
(edited March 1989)

Diazinon

Guideline

The maximum acceptable concentration (MAC) for diazinon in drinking water is 0.02 mg/L (20 μg/L).

Identity, Use and Sources in the Environment

Diazinon ($C_{12}H_{21}N_2O_3PS$) is an organophosphorus insecticide used for the control of soil and household insect pests, as well as sucking and chewing insects and mites on a wide variety of crops. It is also used as an animal ectoparasiticide. Less than 100 000 kg are used annually in Canada.[1]

The vapour pressure of diazinon is 1.9×10^{-2} Pa at 20°C, and its solubility is 40 mg/L at 20°C.[2] Diazinon has a log octanol–water partition coefficient of 3.02.[3]

Diazinon is not very persistent in soil, owing to rapid chemical and biological degradation.[4] The half-life of diazinon in soil ranges from 21 to 80 days, depending upon soil type, activity of microorganisms, water content and the concentration of the pesticide.[5] It is not considered to be a significant leacher.[6] Diazinon is hydrolysed slowly by water to the less toxic 2-isopropyl-6-methyl-4-hydroxypyrimidine;[5] the half-life of diazinon is 185 days in neutral waters (pH 7.4), 0.5 days at pH 3.14 and 6 days at pH 10.9.[4]

Exposure

In surveys of municipal and private water supplies conducted from 1971 to 1986 across Canada, diazinon was detected in only two (private wells) of 620 samples analysed (detection limits 0.001 to 0.5 μg/L).[7] It was detected only once (0.21 μg/L) in 446 samples from three Ontario river basins analysed from 1981 to 1985, although over 1000 kg had been used in these areas in 1983 (detection limit 0.05 μg/L).[8]

Based on the residue tolerance limits set by the Food Directorate of the Department of National Health and Welfare,[9] the theoretical maximum daily intake of diazinon in the diet is 0.33 mg/d. Based on market basket surveys in five regions of Canada during 1976 to 1978, the actual daily intake of diazinon has been estimated to be 0.001 μg/kg bw per day.[10] In the United States, the average daily intake was estimated to be 0.0088 μg/kg bw per day, based on total diet studies.[11] Diazinon was detected in only 54 of 6391 U.S. domestic food samples surveyed from 1981 to 1986, at or below concentrations of 1.0 ppm (detection limit not reported).[12]

Analytical Methods and Treatment Technology

The diazinon content of water may be determined by extracting into dichloromethane, drying and redissolving the extract in hexane and analysing by gas/liquid chromatography with flame photometric detection, phosphorus mode (detection limit 0.05 μg/L).[8,13]

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

Health Effects

Diazinon is readily absorbed from the gastrointestinal tract and is rapidly metabolized within a few hours.[5] Diazinon is metabolized principally by four enzyme systems: mixed-function oxidases, hydrolases or phosphatases, glutathione-dependent transferases and non-specific esterases.[4] Based on *in vivo* studies in animals, the metabolites that can inhibit cholinesterase include diazoxon, hydroxydiazinon (with the isopropyl secondary carbon hydroxylated), isohydroxydiazinon (with the ring methyl group hydroxylated) and a propylenediazinon metabolite.[4,14] However, the majority of administered diazinon appears in the urine as products of cleavage of the phosphorus ester linkage; these cleavage products are not considered to be toxicologically active.[4,5] In rats, 50% of an oral dose of diazinon is excreted within 12 hours,[14] and 95 to 98% is eliminated within seven days, mainly in the urine.[5] Less than 1% of an orally administered dose of 0.02 mg/kg bw was present in tissues of the rat one day after cessation of treatment; most was found in the gastrointestinal tract.[5]

Diazinon is a cholinesterase inhibitor, the dose–response relationship for which appears to depend upon the formulation.[14] A dose of 0.05 mg/kg bw per day administered (route unspecified) to three human volunteers for 28 days reduced plasma cholinesterase by 35 to 40%. Three replicate tests involving human volunteers administered 0.02 to 0.03 mg/kg bw per day for 32 to 43 days (route unspecified) reduced plasma cholinesterase activity by 0, 15 to 20 and 14%, respectively;[14,15] more than 20% inhibition is considered to be biologically significant.[16] No effects on red blood cell cholinesterase activity were observed. The no-observed-adverse-effect level (NOAEL) was considered to be 0.02 mg/kg bw per day.[14,15]

In studies in which dogs were fed diets containing diazinon at dose levels of 0.02, 0.04 or 0.08 mg/kg bw per day for 31 days, the NOAEL with respect to plasma cholinesterase inhibition was considered to be 0.02 mg/kg bw per day.[17]

The U.S. National Cancer Institute evaluated the carcinogenic potential of diazinon in Fischer F344 rats and B6C3F_1 mice. Rats were fed food containing 400 to 800 ppm diazinon for 103 weeks, and mice were given diets containing 100 to 200 ppm diazinon for the same period. It was concluded that diazinon was not carcinogenic to males or females of either test species under the conditions of the bioassay.[18]

Diazinon was not mutagenic in a study involving five microbial assay systems.[19] It did not induce sister chromatid exchanges in Chinese hamster cell line V79.[20]

Teratogenic effects on the spinal columns of chick embryos were observed when diazinon was injected into yolk sacs at the rate of 1 mg per egg before incubation and after four days of incubation.[21] Diazinon was not teratogenic to rabbits receiving oral doses of 7 or 30 mg/kg bw during organogenesis, or in hamsters ingesting 0.125 to 0.25 mg/kg bw during organogenesis.[22] No dose-related abnormalities were observed in rats whose dams had been administered doses of diazinon that increased maternal mortality (e.g., 95 mg/kg bw on day 9) by gavage during gestation; however, reduced foetal development, as indicated by reduced weight of litters and mild hydronephrosis, was observed.[23] In studies submitted to the U.S. Environmental Protection Agency in which diazinon was orally administered to New Zealand white rabbits on days 6 to 18 of gestation, NOAELs of 7 mg/kg bw for reproductive effects (abortions, death of foetuses) and 25 mg/kg bw for maternal effects (increased mortality) were reported. No effects on foetal development were observed at any of the dose levels tested.[24]

Rationale

The acceptable daily intake (ADI) for diazinon has been derived by the Food and Agriculture Organization (FAO) and the World Health Organization (WHO)[25] as follows:

$$\text{ADI} = \frac{0.02 \text{ mg/kg bw per day}}{10} = 0.002 \text{ mg/kg bw per day}$$

where:

- 0.02 mg/kg bw per day is the NOAEL for cholinesterase inhibition derived from studies in the dog[17] and in human volunteers[14,15]
- 10 is the uncertainty factor.

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

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

where:

- 0.002 mg/kg bw per day is the ADI established by the FAO/WHO
- 70 kg is the average body weight of an adult
- 0.20 is the proportion of daily intake of diazinon arbitrarily allocated to drinking water (actual intake from water appears to be much less)
- 1.5 L/d is the average daily consumption of drinking water for an adult.

References

1. Environment Canada/Agriculture Canada. Pesticide Registrant Survey, 1986 report. Commercial Chemicals Branch, Conservation and Protection, Environment Canada, Ottawa (1987).

2. Agriculture Canada. Guide to the chemicals used in crop protection. 7th edition. Publication No. 1093 (1982).

3. Suntio, L.R., Shiu, W.Y., Mackay, D., Seiber, J.N. and Glotfelty, D. Critical review of Henry's Law constants for pesticides. Rev. Environ. Contam. Toxicol., 103: 1 (1988).

4. National Academy of Sciences. Drinking water and health. Vol. 1. U.S. National Research Council, Washington, DC (1977).

5. FAO/WHO. 1970 evaluation of some pesticide residues in food. WHO Food Additive Series No. 42, World Health Organization, Geneva (1971).

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

7. Hiebsch, S.C. 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).

8. Frank, R. and Logan, L. Pesticide and industrial chemical residues at the mouth of the Grand, Saugeen and Thames rivers, Ontario, Canada, 1981–85. Arch. Environ. Contam. Toxicol., 17: 741 (1988).

9. Department of National Health and Welfare. National pesticide residue limits in foods. Food Directorate, Ottawa (1986).

10. McLeod, H.A., Smith, D.C. and Bluman, N. Pesticide residues in the total diet in Canada, V: 1976 to 1978. J. Food Saf., 2: 141 (1980).

11. Gunderson, E.L. FDA Total Diet Study, April 1982 – April 1984, dietary intakes of pesticides, selected elements, and other chemicals. J. Assoc. Off. Anal. Chem., 71(6): 1200 (1988).

12. Hundley, H.K., Cairns, T., Luke, M.A. and Masumoto, H.T. Pesticide residue findings by the Luke method in domestic and imported foods and animal feeds for fiscal years 1982–1986. J. Assoc. Off. Anal. Chem., 71(5): 875 (1988).

13. Braun, H.E. and Frank, R. Organochlorine and organophosphorus insecticides: their use in eleven agricultural watersheds and their loss to stream waters in Southern Ontario, Canada, 1975–1977. Sci. Total Environ., 15: 169 (1980).

14. Hayes, W.J., Jr. Pesticides studied in man. Williams and Wilkins, Baltimore, MD (1982).

15. FAO/WHO. Evaluation of some pesticide residues in food. FAO.PL:CP/15, WHO/Food Add./67.32, Food and Agriculture Organization of the United Nations, Geneva (1967), cited in reference 4.

16. National Academy of Sciences. Drinking water and health. Vol. 3. U.S. National Research Council, Washington, DC (1980).

17. FAO/WHO. Evaluation of the toxicity of pesticide residues in food. FAO Meeting Report No. PL: 1965/10/1; WHO/Food Add./27.65, Food and Agriculture Organization of the United Nations, Rome (1965), cited in reference 4.

18. National Cancer Institute. Bioassay of diazinon for possible carcinogenicity. Carcinogenicity Testing Program. DHEW Publication No. NIH 79-1392. MRID 00073372. National Institutes of Health, Bethesda, MD (1979).

19. Poole, D.C., Simmon, V.F. and Newell, G.W. In vitro mutagenic activity of fourteen pesticides. Toxicol. Appl. Pharmacol., 41: 196 (1977).

20. Chen, H.H., Hsueh, J.L., Sirianni, S.R. and Huang, C.C. Induction of sister-chromatid exchanges and cell cycle delay in cultured mammalian cells treated with eight organophosphorus pesticides. Mutat. Res., 88: 307 (1981).

21. Khera, K.S. and Bedok, S. Effects of thiol phosphates on notochordal and vertebral morphogenesis in chick and duck embryos. Food Cosmet. Toxicol., 5: 359 (1967), cited in reference 14.

22. Robens, J.F. Teratologic studies of carbaryl, diazinon, Norea, disulfiram and thiram in small laboratory animals. Toxicol. Appl. Pharmacol., 15: 152 (1969), cited in reference 14.

23. Dobbins, P.K. Organic phosphate insecticides as teratogens in the rat. J. Fla. Med. Assoc., 54: 542 (1967), cited in reference 14.

24. U.S. Environmental Protection Agency. Health advisory — Diazinon. Office of Drinking Water (1987).

25. FAO/WHO. Pesticide residues in food — 1979. Evaluations. Data and recommendations of the Joint Meeting on Pesticide Residues, Geneva, December 3–12, 1979. FAO Plant Production and Protection Paper 20 (Suppl.), Food and Agriculture Organization of the United Nations, Rome (1980).