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Diuron

Guideline

The maximum acceptable concentration (MAC) for diuron in drinking water is 0.15 mg/L (150 μg/L).

Identity, Use and Sources in the Environment

Diuron is a substituted urea-based herbicide used in Canada in 1986 in moderately low quantities (in the range 10 000 to 50 000 kg).[1] It is employed principally for the control of vegetation in non-crop areas, including irrigation and drainage ditches.[1]

Diuron is a non-ionic compound with moderate water solubility of 22 to 42 mg/L at 20°C. Its hydrolysis rate is negligible at neutral pH but increases under strongly acidic or alkaline conditions.[2] It is stable to oxidation and breakdown, persisting in soils for a full season or longer.[3] It has a log octanol–water partition coefficient of 2.6, which is considered low to moderate. It is adsorbed to soils to some degree, with a moderate soil–water partition coefficient of 485.[4] The U.S. Environmental Protection Agency has ranked diuron fairly high — as a Priority B chemical — with respect to potential for groundwater contamination,[5] and it also rates highly in Agriculture Canada's ranking of potential leaching agents.[6]

Exposure

Diuron has not often been included in Canadian water monitoring surveys; it has been detected once in a survey of 15 private wells in Ontario.[7] In the United States, diuron was detected in 0.03% of more than 900 groundwater samples. It was found in the low parts per billion range (2 to 3 μg/L) in California wells as a result of agricultural practices.[8]

The theoretical maximum dietary intake of diuron for an adult Canadian would be about 0.48 mg/d, or 0.007 mg/kg bw per day, assuming that every crop for which it is registered contained it at the maximum residue level (MRL).[9] However, its use pattern indicates that it is seldom used on crops, especially wheat and potatoes, which contribute 70% of the theoretical daily intake.[10] No actual residue levels in foods are available, as diuron was not included in total diet surveys in either Canada or the United States.

Analytical Methods and Treatment Technology

Diuron may be monitored in water by extraction into hexane, hydrolysis to the aniline derivative, quantification by gas/liquid chromatography and Hall conductivity detection. The detection limit using this method is 0.1 μg/L.[11]

Granular activated carbon and powdered activated carbon are effective in removing up to 90% of diuron from drinking water.[12]

Health Effects

Diuron is absorbed from the gastrointestinal and respiratory systems. In humans, it is metabolized within hours by hydroxylation and N-dealkylation, then excreted via the urine.[13] In rats and dogs, one-sixth to one-half the total eliminated was found in the faeces.[14] Following ingestion of diuron for nine months to two years by rats and dogs, little storage was observed in any tissue; the highest levels were seen in liver and kidney.[14]

Diuron is of low acute toxicity. Juveniles and animals on protein-deficient diets are more susceptible than adults to the toxic effects of diuron, based on LD_{50} results.[13] Ingestion by a woman of a single dose of diuron at 38 mg/kg bw had no apparent effect.[13] In animals, the principal toxic effects of chronic ingestion of diuron are weight loss and abnormalities in blood, liver and spleen.[13]

Two chronic feeding studies have been conducted with diuron, which was administered in the diet for two years to groups of two male and three female beagle dogs at dose levels corresponding to 0, 0.625, 3.125, 6.25 or 31.25 mg/kg bw per day, and to 35 rats of each sex at dose levels corresponding to 0, 1.25, 6.25, 12.5 or 125 mg/kg bw per day.[8,13,14] At 125 ppm (3.125 mg/kg bw in dogs and 6.25 mg/kg bw in rats), traces of abnormal blood pigments were observed in a few animals but were not statistically significant. At

250 ppm and above (6.25 mg/kg bw in dogs and 12.5 mg/kg bw in rats), haematological alterations, weight loss, haemosiderosis of the liver and erythroid hyperplasia were observed. The no-observed-adverse-effect level (NOAEL) was 125 ppm, or 3.125 mg/kg bw in dogs and 6.25 mg/kg bw in rats. Although there was no evidence of carcinogenicity in these experiments, no firm conclusions can be drawn because the studies had methodological flaws.

Diuron was not mutagenic in most microbial tests with or without metabolic activation.[15] One positive result was reported, in *Salmonella typhimurium*, with metabolic activation.[16] Diuron was negative in two *in vitro* mammalian test systems, for forward gene mutations in Chinese hamster ovary cells and for unscheduled DNA synthesis in rat hepatocytes.[17] However, clastogenic effects were observed in an *in vivo* test on rats.[17]

Reproduction was not affected in a three-generation rat study with dietary levels equivalent to 6 mg/kg bw per day; however, this dose was slightly foetotoxic, causing reduction of body weights in the F_2 and F_3 litters.[8,13] Diuron was not teratogentic but was foetotoxic in rats receiving 250 mg/kg bw per day, with lowered foetal weights and minor rib and bone anomalies observed. The same effects were observed at 125 mg/kg bw per day but were not statistically significant.[18] The lowest-observed-adverse-effect level (LOAEL) was therefore 125 mg/kg bw per day.

Rationale

Based on evaluations by the Food Directorate of the Department of National Health and Welfare,[19] the acceptable daily intake (ADI) for diuron is derived as follows:

$$\text{ADI} = \frac{3.125 \text{ mg/kg bw per day}}{200} \approx 0.0156 \text{ mg/kg bw per day}$$

where:

- 3.125 mg/kg bw per day is the NOAEL observed in a two-year dog study[14] that was evaluated by the Food Directorate and considered to be the most suitable study on which to base an ADI. Body weight loss, increased liver weight, erythroid hyperplasia and decreased haematological values were observed at higher doses[14,19]
- 200 is the uncertainty factor assigned by the Food Directorate.

The maximum acceptable concentration (MAC) is derived from the ADI as follows:

$$\text{MAC} = \frac{0.0156 \text{ mg/kg bw per day} \times 70 \text{ kg} \times 0.20}{1.5 \text{ L/d}} \approx 0.15 \text{ mg/L}$$

where:

- 0.0156 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 total daily intake of diuron assigned to drinking water (theoretical maximum intake from food is 45% of the ADI)
- 1.5 L/d is the average daily consumption of drinking water for an adult.

References

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