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Diquat

Guideline

The maximum acceptable concentration (MAC) for diquat (measured as the cation) in drinking water is 0.07 mg/L (70 µg/L).

Identity, Use and Sources in the Environment

Diquat ($C_{12}H_{12}N_2$) is a bipyridyl herbicide generally marketed as a dibromide salt or a dichloride monohydrate. It is used as a pre-harvest desiccant for various seed crops, for potato haulm destruction and for the control of aquatic weeds. Less than 100 000 kg are used annually in Canada; 96% of this amount is applied as a harvest aid.[1] Diquat is non-volatile and has no measurable vapour pressure.[2] It is soluble in water at 700 g/L at 20°C.[3]

The double positive charge on the diquat cation causes it to be adsorbed tightly to the negatively charged clay minerals present in the soil.[4] As a result, diquat remains in the upper layers of soil for a long time[5] and is unlikely to leach to groundwater. Diquat applied to aquatic systems as a weed control agent disappears from the water in one to four weeks through adsorption to plants and sediments.[5] Photodegradation also contributes to the loss of diquat from water.[6]

Exposure

No information on the concentrations of diquat in Canadian drinking water or surface waters was identified.

The theoretical maximum daily intake of diquat in food is 0.03 mg/d, based on the residue tolerance limits set by the Food Directorate of the Department of National Health and Welfare.[7] The maximum residue limit for all crops for which diquat use is registered is 0.1 mg/kg. Few data are available on the actual levels of diquat found in foodstuffs. Supervised trials in Canada in which diquat was applied to wheat and oats at the rates of 0.28 and 0.56 kg diquat ion per hectare resulted in mean residues of 0.05 (range <0.02 to 0.08) and 0.07 (range <0.02 to 0.12) mg/kg, respectively, in the desiccated wheat, and 0.33 (range 0.14 to 0.51) and 0.80 (range 0.14 to 1.4) mg/kg, respectively, in the desiccated oats.[6]

Analytical Methods and Treatment Technology

Diquat in water may be determined by passing a large sample (approximately 500 mL) directly through a cation-exchange column, followed by washing and elution steps and spectroscopic measurement (detection limit 0.003 ppm).[8]

Under alkaline conditions, chlorination removes diquat from water by oxidation; the rate of removal increases with a rise in pH. However, at pH 7 to 8, a relatively long contact time (two hours or more) is necessary to achieve reasonable removal. Chlorine dioxide acts extremely rapidly (within a few minutes) to oxidize diquat.[9] The use of clay adsorbents (e.g., bentonite) or activated carbon, followed by chemical coagulation, also appears to be effective for the rapid removal of diquat from water.[10]

Health Effects

Diquat is poorly absorbed from the gastrointestinal tract following ingestion. Most of a single oral dose of 45 mg/kg bw per day of radioactively labelled diquat was eliminated by rats in four days; 6% of the dose was detected in the urine and 89% in the faeces, most of it unchanged. The metabolite diquat monopyridone, detected mainly in the faeces, accounted for 5% of the dose, whereas the minor urinary metabolite diquat dipyridone accounted for approximately 0.1% of the dose.[11] Toxicity is believed to be due to the parent compound, rather than to its metabolites.[12] Diquat was not found to accumulate in the tissues of rats fed 25 ppm in the diet for eight weeks.[13]

A single oral dose of 6 to 12 g of diquat ion has been reported to be fatal to humans.[5] Diquat poisoning in humans may cause damage to the lining of the gastrointestinal tract, brain, liver, kidneys and lungs.[11]

Chronic ingestion of diquat has induced the formation of cataracts in rats and dogs, an effect that has never been reported in humans following exposure. Groups of 35 male and 35 female Wistar rats were fed diets containing 0, 15, 25 or 75 ppm diquat ion for up to two years. Although there was no significant effect on body weight, food consumption or mortality in any of the treated groups, a significant increase in cataract formation occurred in the group exposed to the highest dose (75 ppm) when compared with the control group. No significant difference in the number of cataracts was observed in rats consuming 15 and 25 ppm diquat ion compared with the controls, but cataracts appeared somewhat earlier in the group exposed to 25 ppm diquat ion than in the controls. The dietary no-effect level in terms of cataract formation was considered to be 15 ppm diquat ion, equivalent to 0.75 mg/kg bw.[11]

Groups of dogs (three males and three females of unspecified strain) were fed diets containing 0, 16, 32, 68, 200 or 600 ppm diquat dichloride for up to four years. Animals consuming diets containing 200 and 600 ppm diquat dichloride developed bilateral lens opacities at 10 and 15 months, respectively; no effects on growth, tumour formation, food consumption, blood chemistry or gross and microscopic pathology were observed. There was no effect on cataract formation in animals consuming diets containing 68 ppm throughout the four-year study period. The no-observed- adverse-effect level (NOAEL) was considered to be 68 ppm diquat dichloride,[14] corresponding to a diquat ion concentration of 1.22 mg/kg bw.[11]

No carcinogenic or tumorigenic potential was reported for diquat in the long-term feeding studies in rats and dogs. Diquat increased unscheduled DNA synthesis in *in vitro* human cells both with and without metabolic activation by liver microsomes,[15] but it was not mutagenic in dominant lethal tests in mice.[16,17] Following a single intraperitoneal injection of 7 mg/kg bw on days 6 to 14 of gestation, diquat was reported to induce a high incidence of retarded growth of sternum and auditory ossicles in rat foetuses, as well as a marked reduction in the weight of the embryos. Following a dose of 14 mg/kg bw, most pregnancies were interrupted, and effects were more pronounced in rats that reached term.[18] However, no teratogenic effects were observed in studies on rats, mice and rabbits.[19] Diquat appears to have strong embryotoxic action at high doses.[19]

Rationale

The acceptable daily intake (ADI) for diquat (measured as the cation) has been derived by the Food and Agriculture Organization (FAO) and the World Health Organization (WHO)[11] as follows:

$$\text{ADI} = \frac{0.75 \text{ mg/kg bw per day}}{100} \approx 0.008 \text{ mg/kg bw per day}$$

where:

- 0.75 mg/kg bw per day is considered to be the NOAEL in terms of cataract formation in rats[11]
- 100 is the uncertainty factor.

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

$$\text{MAC} = \frac{0.008 \text{ mg/kg bw per day} \times 70 \text{ kg} \times 0.20}{1.5 \text{ L/d}} \approx 0.07 \text{ mg/L}$$

where:

- 0.008 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 diquat allocated to drinking water
- 1.5 L/d is the average daily consumption of drinking water for an adult.

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