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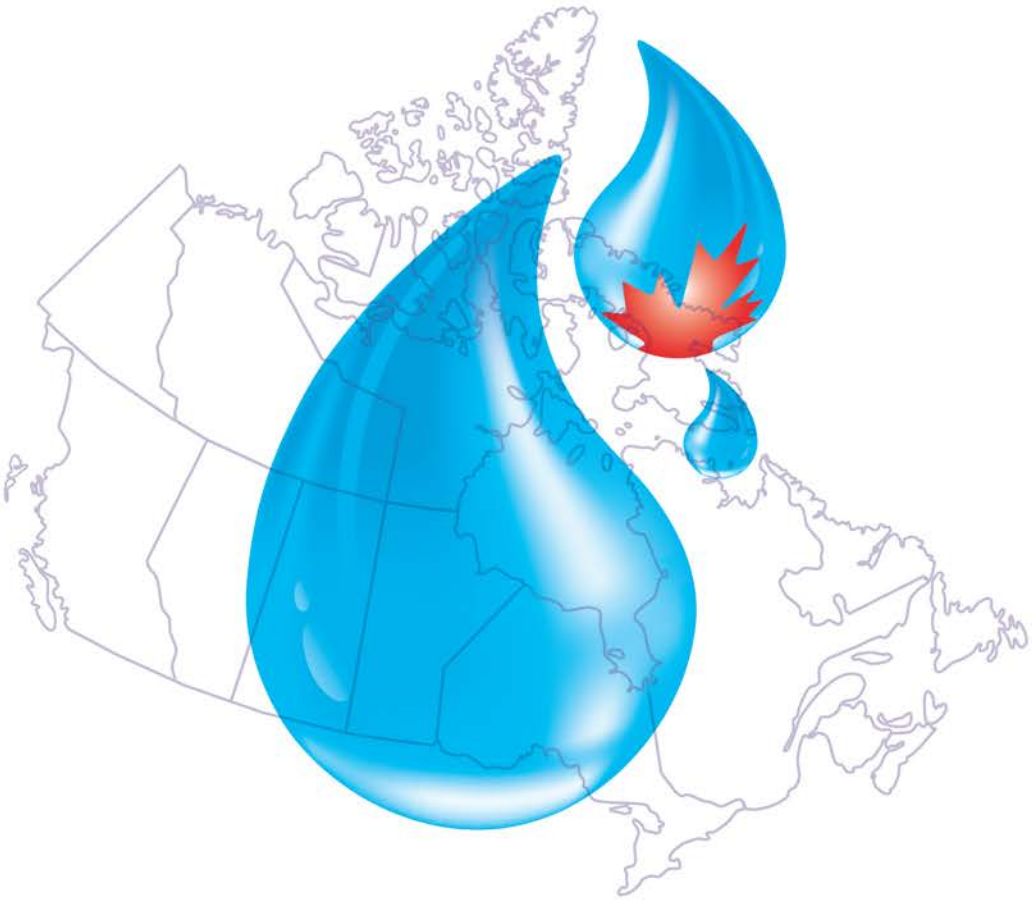
Guidelines for Canadian Drinking Water Quality

Guideline Technical Document

Paraquat



Canada

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Paraquat

Guideline

The maximum acceptable concentration (MAC) for paraquat as the dichloride in drinking water is 0.01 mg/L (10 μg/L), or 0.007 mg/L (7 μg/L) expressed as the paraquat ion

Identity, Use and Sources in the Environment

Paraquat ($C_{12}H_{14}N_2$) is a bipyridyl contact herbicide most commonly marketed as paraquat dichloride. It is used to control aquatic weeds and weeds in seed crops and orchards, as a crop desiccant and defoliant on cotton and potato vines, and as a harvest aid for soybeans. Less than 50 000 kg are used annually in Canada.[1]

Paraquat has no measurable vapour pressure and is very soluble in water[2] (approximately 700 g/L at 20°C).[3] Its log octanol–water partition coefficient is 2.44.[2]

Paraquat applied to plant surfaces as the dichloride will undergo photochemical degradation to 4-carboxyl-1-methylpyridylium dichloride and methylamine hydrochloride.[4] Paraquat may be microbially degraded before it is rapidly and completely adsorbed onto the clay particles in the top layer of the soil, because of the positively charged paraquat ion.[4] Once inactivated by the soil, paraquat disappears only very slowly and is unlikely to leach to groundwater.[5] Paraquat applied to aquatic systems disappears from the water rapidly, within six to 14 days, because of adsorption onto sediments and plants and uptake by plants.[4]

Exposure

No information was identified regarding levels of paraquat in Canadian surface or drinking water. As it is rapidly and completely bound to clay particles in the soil, paraquat is unlikely to contaminate drinking water supplies as a result of its application to crops. However, paraquat may remain in water for several days following its use for aquatic weed control.[4]

The theoretical maximum daily intake of paraquat in food is 0.04 mg/d, based on the residue tolerance limits set by the Food Directorate of the Department of National Health and Welfare.[6] Little information is available on actual levels found in foods. Field trials indicate that paraquat applied to crops at the normal rate for weed control results in no significant concentrations in the produce. However, application of the herbicide as a pre-harvest aid may result in detectable concentrations in crops; for example, following application of 0.5 to 2 kg/ha, levels ranged up to 0.5 ppm in onions and 0.13 ppm in potatoes. Paraquat does not accumulate in tissues of animals exposed to low doses of treated feed over long periods of time.[5]

Analytical Methods and Treatment Technology

The concentration of paraquat in water may be determined by passing the sample through a cation exchange column, followed by washing, elution and spectrophotometric measurement. Other analytical methods include thin-layer chromatography, hydrogenation or pyrolysis, followed by gas chromatographic separation and detection by flame ionization.[5] A bioassay, in which the effect of paraquat on the rate of chlorosis in lesser duckweed is measured, is also used.[4,5]

Techniques effective in removing paraquat from water include adsorption by charcoal (37.3 and 93.2 mg of paraquat per gram of charcoal at initial concentrations of 0.373 and 37.3 mg/L), ion exchange (66 and 70%) and modified peat (95 to 99%).[2] Chlorine dioxide has been found to oxidize paraquat concentrations of 15 and 30 mg/L within minutes above pH 8.[7] The use of bentonite, a clay adsorbent, for 10 minutes, followed by a 15-minute coagulation period, resulted in 90% removal of paraquat present at 1 mg/L.[8]

Health Effects

Paraquat is rapidly but incompletely absorbed from the gastrointestinal tract. An oral dose of paraquat dichloride (quantity unspecified) was largely excreted in the faeces of rats (93 to 96%); 30% of the dose was present in the faeces as unspecified metabolic products, possibly from microbial degradation in the gut.[9] Paraquat is distributed via the bloodstream to practically all organs and tissues and accumulates in the lungs and,

to some extent, in the kidneys.[4] Serum values of orally administered ^{14}C-paraquat (dose unspecified) peaked in 30 to 60 minutes in rats, guinea pigs and monkeys,[10] and in 75 minutes in dogs.[11]

The minimum lethal dose of paraquat in humans is approximately 35 mg/kg bw.[4] Acute poisoning may cause respiratory distress and effects on the nervous system and kidneys. Death is usually due to progressive pulmonary fibrosis and epithelial proliferation in the lungs;[12] renal failure may also occur.[13]

Groups of dogs (two to four males and females per group) were fed diets containing paraquat dichloride at concentrations of 0, 10, 50, 125 or 250 ppm for 26 to 27 months. Toxic effects reported in the two highest dose groups included decreased intake of food and weights of body, spleen and testes, respiratory distress, increased ratios of the weights of liver, heart, thyroid and adrenal gland to body weight, and growths and microscopic changes in the lungs. The no-observed-adverse-effect level (NOAEL) for paraquat dichloride was considered to be 50 mg/kg diet, or 1.25 mg/kg bw per day (equivalent to 0.91 mg/kg bw per day for the paraquat ion).[14] In a two-year study in which rats (strain unspecified) were administered paraquat dichloride in the diet, the NOAEL was considered to be 30 ppm as the dichloride, or 1.5 mg/kg bw per day, based on degenerative effects on the kidney at 100 ppm.[15]

A three-generation reproductive study was conducted on rats (strain unspecified) exposed to levels of paraquat ion of 100 mg/kg in the diet. No adverse effects were observed in terms of fertility, fecundity, neonatal morbidity or mortality, gonadotoxicity, structural or functional lesions, or pulmonary function of treated offspring.[4] However, a study in mice indicated that paraquat produced a significant reduction of pregnancy rates in females mated to males with sperm that had been treated in the post-meiotic late spermatid stage.[16] Paraquat does not produce any significant teratogenic or embryotoxic effects, likely because of low transplacental transfer.[5]

Results of an 80-week study in mice designed to investigate the carcinogenic potential of levels of paraquat of 25, 50 and 75 mg/kg per day in the diet indicated that paraquat was not tumorigenic in mice.[4] No signs of tumorigenicity were observed in long-term studies in rats reviewed by the International Programme on Chemical Safety.[4] In bacterial test systems for mutagenicity, results have been weakly positive or negative.[4] Paraquat was not mutagenic in human leukocytes or in *in vivo* cytogenic tests on mouse bone marrow[17] or in dominant lethal tests in mice.[16,18]

Rationale

The Food and Agriculture Organization (FAO) and the World Health Organization (WHO)[13] have established a temporary acceptable daily intake (ADI) for paraquat dichloride of 0.001 mg/kg bw per day (equivalent to 0.0007 mg/kg bw per day as paraquat ion), based on toxicological data from studies in rats[15] and dogs[14] that have not been validated.

Based on the temporary ADI established by the FAO/WHO, the maximum acceptable concentration (MAC) for paraquat dichloride in drinking water is derived as follows:

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

$$\approx 0.01 \text{ mg/L (or } 0.007 \text{ mg/L expressed as paraquat ion)}$$

where:

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

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