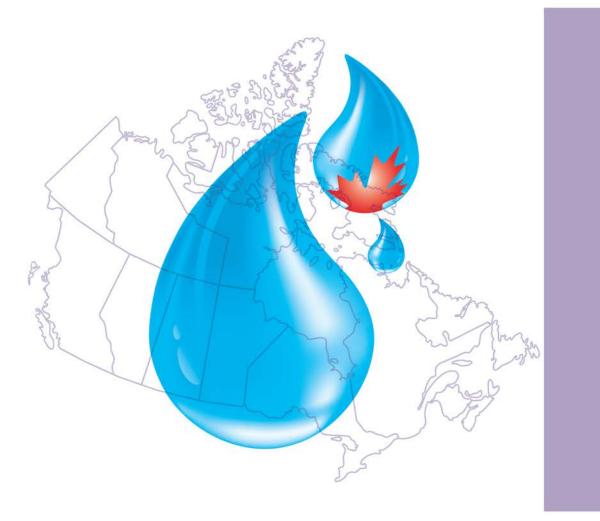# Guidelines for Canadian Drinking Water Quality

Guideline Technical Document

Simazine

Canada

Simazine

Guideline

*The maximum acceptable concentration (MAC)
for simazine in drinking water is 0.01 mg/L (10 µg/L).*

Identity, Use and Sources in the Environment

Simazine ($C_7H_{12}ClN_5$) is a triazine soil sterilant and pre-emergence herbicide used in the control of broadleaf and grassy weeds for a wide variety of crops. It is also used in aquatic weed control. Between 100 000 and 500 000 kg are used annually in Canada.[1] The solubility of simazine in water at 20°C is 3.5 mg/L; its vapour pressure is 8.1×10^{-7} Pa at 20°C.[2] The log octanol–water partition coefficient of simazine is reported to be 1.94;[3] it is therefore not likely to bioaccumulate to a significant degree in human or animal tissue.

Simazine applied to soil will remain primarily in the upper 5 cm.[4] The movement of simazine through the soil layers is pH-dependent; it is more soluble at lower pH, whereas it binds more to the clay or organic matter in soil at higher pH.[5] Simazine is less likely to leach than other triazine herbicides;[6] the extent of leaching decreases with an increase in organic matter and clay content.[7] Microbial degradation may contribute significantly to the removal of simazine from soil.[4] Biological processes are also principally responsible for the removal of simazine from water. Its persistence in aquatic environments is dependent upon many factors, including the amount of algae and weeds present; the average half-life of simazine in ponds is reported to be about 30 days.[4]

Exposure

Simazine was detected in nine of 440 surface water samples (mean detectable concentration 0.6 µg/L) from three Ontario river basins surveyed from 1981 to 1985 (detection limit 0.2 µg/L); a total of only 800 kg had been used in these areas in 1983.[8] Simazine was detected in 55 of 1199 samples of municipal and private drinking water supplies in Nova Scotia (1986), Quebec (1986), Ontario (1979 to 1986), Manitoba (1986) and Alberta (1978 to 1986) (detection limits ranged from 0.025 to 1.0 µg/L). The maximum concentration reported was 23 µg/L, obtained from a private well in Ontario.[9]

The theoretical maximum daily intake of simazine from food is 0.02 mg/d, based on the residue tolerance limits set by the Food Directorate of the Department of National Health and Welfare.[10] No information on the actual intake of simazine was found. Standard residue trials conducted in several countries revealed that residues in harvested crops were usually less than 0.04 mg/kg. Exceptions were asparagus, which was found to contain 0.13 mg/kg simazine, and maize forage, which contained 0.35 mg/kg, seven days after application of 4.4 kg active ingredient per hectare. Residues in beef, mutton and milk were less than 0.1 mg/kg (the Canadian negligible residue limit),[10] following diets containing 100 ppm simazine.[7]

Analytical Methods and Treatment Technology

Simazine in water may be determined by extraction with chloroform, separation by gas/liquid chromatography and detection by electrolytic conduction, nitrogen mode (detection limit 0.2 µg/L).[8] An alternative method involves extraction into dichloromethane, gas chromatographic capillary column separation and nitrogen phosphorus detection (estimated detection limit 0.1 to 2 µg/L).[11]

Conventional treatment processes are reported to be relatively ineffective in removing simazine from drinking water supplies.[11] A 44% reduction in simazine concentration has been reported following treatment of water with an initial simazine concentration of 480 ng/L by powdered activated carbon adsorption in St. Hyacinthe, Quebec.[12] Powdered activated carbon adsorption has also been reported to result in 43 to 100% removal of simazine from water, whereas granular activated carbon adsorption has resulted in 35 to 89% removal (initial concentrations not reported).[11] Other treatment methods investigated for efficiency in simazine removal (initial concentrations not reported)

are ion-exchange resins (81 to 95%), chlorine oxidation (17 to 74%, depending on dose), chlorine dioxide oxidation (8 to 27%), ozonation (92% in spiked, distilled water), oxidation (19 to 42% in spiked, distilled water with hydrogen peroxide, 1 to 25% in spiked river water) and potassium permanganate oxidation (up to 26% in spiked, distilled water).[11]

Health Effects

Little information is available on the extent of absorption of ingested simazine. Four days following administration by stomach tube of 0.8 mg radioactively labelled simazine in 1 mL arachis (peanut) oil to Carworth Farm E strain rats, 91.9 (males) to 94.5% (females) was recovered; 40.6% was eliminated via the urine, 47.2% was recovered in the faeces, and 3% of the dose remained in the animal tissues. N-dealkylation and mercapturic acid formation were the major routes of metabolism,[13,14] with major urinary metabolites being N-acetyl-S-[4-amino-6-(1-methyl-1-cyanoethylamino)-s-triazinyl-2]-L-cysteine and 2-chloro-4-amino-6-(1-methyl-1-cyanoethylamino)-s-triazine.[13]

No reports on the toxicity of simazine ingested by humans were identified, although skin contact has caused dermatitis.[14] Simazine is considered to be only slightly acutely toxic to mammals.[7]

In a two-year dietary study in Charles River rats (30 per sex per dose), animals were administered 0, 1, 10 or 100 ppm simazine in the feed. No signs of toxicity were observed at any dose level, and no differences were noted between treated and control animals upon gross pathological and histopathological examination. The no-observed-adverse-effect level (NOAEL) was considered to be above 100 ppm,[15] which is equivalent to 5 mg/kg bw per day.[11] This study is very limited for the derivation of a NOAEL because of the very high mortality from unreported causes (50 to 82% for various groups).[7]

Dogs (two males and two females per dose group, strain unspecified) were fed diets containing 0, 15, 150 or 1500 ppm for up to two years, with two animals per group being sacrificed after 52 weeks for detailed pathological examination. Dogs receiving 1500 ppm had slightly lower body weights; marginally higher levels of serum alkaline phosphatase and serum glutamic–oxaloacetic transaminase were also noted sporadically in these animals after 84 or 104 weeks of treatment. A slight hyperplasia of the thyroid gland was also observed in the highest dose group. No other compound-related differences were noted based on urinalysis, gross autopsy or histopathological examination. The NOAEL for dogs was considered to be 150 ppm, which was reported to be equivalent to 5 mg/kg bw per day.[7]

Simazine was not tumorigenic in a limited 18-month study in which hybrid mice were administered doses of 215 mg/kg bw by gavage from ages 7 to 18 days, followed by a daily diet containing 215 mg/kg bw per day.[16] Another study in mice (Swiss-CD-1 strain), in which there were no significant increases in tumour incidence in 60 animals per sex per dose exposed to simazine (doses of 0, 15, 1000 and 3000 ppm, route unspecified) for 21 months, was judged inadequate for assessment of carcinogenicity because of the loss by autolysis of a large number of animal tissues.[7]

Simazine was not mutagenic in several microbial assay systems.[11] It induced lethal mutations in the sex-linked recessive lethal test in *Drosophila melanogaster*;[17] an elevation in X-linked lethals was induced by injection of simazine into male *D. melanogaster*.[18] Both positive[19] and negative[20] results have been reported for the induction of unscheduled DNA synthesis in human lung fibroblasts. Based on results from an additional six *in vivo* and *in vitro* assays, it was concluded that neither simazine nor its metabolites showed any mutagenic potential.[7]

In three studies in rats, no teratogenic effects were noted at levels below those that produced maternal toxicity. Pregnant rats received doses of simazine of up to 600 mg/kg bw per day by intubation on days 6 to 15 of gestation. Both maternal toxicity and foetotoxicity were observed at dose levels of 100 mg/kg bw per day and above. Observed adverse effects included an increased incidence of aborted foetuses and embryonic resorptions, along with hypoplastic lungs, reduced pup weight and vitality and increased incidence of skeletal variations, such as non-ossification of the sternebrae.[7] Simazine was not teratogenic at maternally non-toxic doses in rabbits exposed to 5, 75 or 200 mg/kg bw per day during gestation.[7]

No adverse reproductive effects were observed in a three-generation study in albino Charles River rats receiving 50 or 100 ppm simazine in the diet. The NOAEL for this study was considered to be 100 ppm, or 5 mg/kg bw per day.[7]

Rationale

The Food Directorate of the Department of National Health and Welfare has derived a negligible daily intake (NDI) for simazine as follows:

$$NDI = \frac{5 \text{ mg/kg bw per day}}{4000} \approx 0.0013 \text{ mg/kg bw per day}$$

where:

- 5 mg/kg bw per day is the NOAEL from a two-year study in dogs[7]

- 4000 is the uncertainty factor.

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

$$MAC = \frac{0.0013 \text{ mg/kg bw per day} \times 70 \text{ kg} \times 0.20}{1.5 \text{ L/d}} \approx 0.01 \text{ mg/L}$$

where:

- 0.0013 mg/kg bw per day is the NDI derived by the Food Directorate

- 70 kg is the average body weight of an adult

- 0.20 is the proportion of daily intake of simazine allocated to drinking water

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

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