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Canadian Water Quality Guidelines for Simazine

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SCIENTIFIC SERIES NO. 187

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WATER QUALITY BRANCH
OTTAWA, ONTARIO, 1991

(Disponible en français sur demande)

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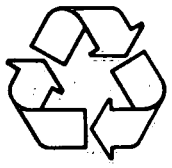

Printed on paper that contains recovered waste

Published by authority of
the Minister of the Environment

Cat. No. En 36-502/187E
ISBN 0-662-18992-2

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Abstract

A literature review was conducted on the uses, fate, and effects of simazine on raw water for drinking water supply, freshwater aquatic life, agricultural uses, recreational water quality and aesthetics, and industrial water supplies. The information is summarized in this publication. From it, water quality guidelines for the protection of specific water uses are recommended.

Résumé

On a étudié la documentation relative aux utilisations, au devenir et aux effets du simazine sur l'eau naturelle utilisée comme eau potable non traitée, sur les organismes aquatiques en eau douce, sur l'utilisation de l'eau en agriculture, sur la qualité de l'eau pour les loisirs et l'esthétique, ainsi que sur l'eau utilisée à des fins industrielles. Ces renseignements, résumés dans notre étude, nous permettent de recommander des seuils de concentration de la simazine pour la protection des diverses utilisations de l'eau.

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SOURCES, OCCURRENCE, AND CHARACTERISTICS

Uses and Production

Simazine is the common name for the chemical 6-chloro-N^2,N^4-diethyl-1,3,5-triazine-2,4-diamine (IUPAC). It has the Chemical Abstracts Service (CAS) name 2-chloro-4,6-bis(ethylamino)-1,3,5-triazine and CAS Registry Number 122-34-9. Simazine is a selective triazine herbicide used for the control of annual broadleaf and grass weeds in numerous crop and non-crop applications. It is a member of the triazine group of pesticides (Knüsli, 1970; Smith *et al.*, 1982) and was first registered in Canada in 1963 (Agriculture Canada, 1989).

Uses of simazine in Canadian agriculture include weed control in corn, established asparagus, bird's foot trefoil, raspberries, loganberries, blackberries, highbush blueberries, alfalfa, apples and pears established 1 year or more, grapes, woody ornamentals, nursery and Christmas tree plantations, and pasture and rangeland (Agriculture Canada, 1989; Ontario Ministry of Agriculture and Food, 1989). In Nova Scotia, simazine is registered in forestry as a conifer release herbicide, and in forestry nurseries as a pre-emergent herbicide, using ground-spray equipment (P. Neily, 1990, Nova Scotia Department of Lands and Forests, pers. com.). A list of the weeds controlled by simazine can be found in Table 1. In the United States, 36% of simazine marketed is applied to corn, 25% to citrus (oranges, lemons, and grapefruit) and deciduous fruit and nut crops, 20% to non-crop industrial areas, and 19% to aquatic sites (U.S. EPA, 1984).

Non-crop uses for simazine include non-selective weed control in industrial areas, at airports, and along shelterbelts and rights-of-way, and aquatic weed control in ditches, farm ponds, recirculating water cooling towers, fish hatcheries, aquaria, fountains, and swimming pools (Ghassemi *et al.*, 1981; Worthing and Walker 1987; U.S. EPA, 1987; Agriculture Canada, 1989). A common name for the formulation of simazine added to ponds in the United States is Aquazine®, an 80% active ingredient (ai) wettable powder.

Simazine is generally formulated as a wettable powder containing varying percentages of the technical-grade active ingredient. Canadian-registered compounds with simazine as the sole ingredient include Simadex Simazine 80W, a wettable powder made up of 80% simazine; Simadex Simazine, a suspension containing 500 $g \cdot L^{-1}$ simazine (both from the Nor-Am Chemical Company); Simmaprim 80W and Princep 80W, 80% wettable powders from Ciba-Geigy Canada; and Princep Nine-T and Simmaprim Nine-T, 90% soluble granules from Ciba-Geigy Canada. Simazine can also be added to tank mixtures with other herbicides, such as atrazine, amitrole, diuron, monuron, and paraquat (Agriculture Canada, 1989) (Table 2).

During agricultural applications, simazine can be incorporated into the soil as a pre-plant treatment or applied pre-emergence to the soil surface; methods of

Table 1. Weeds Sensitive to Simazine

Annual Broadleaf Weeds	
	pigweed
	lady's-thumb
	lamb's-quarters
	purslane
	ragweed
	volunteer clover
	wild buckwheat
	smartweed
	plantain and groundsel
Annual Grasses	
	barnyard grass
	crab grass
	wild oats
	yellow foxtail
	most perennial species starting from seed
Other Weeds	
	"pond scum"
	filamentous algae, such as *Chara* and *Nitella*
	most emerged and submerged aquatic vegetation

Source: Ontario Ministry of Agriculture and Food, 1989.

Table 2. Simazine Tank Mixtures Registered in Canada

Mixture	Components
Amazine	Amitrole+simazine
X-All Liquid	Amitrole+simazine
Ekko	Atrazine+simazine
Mag-X-String	Atrazine+diuron+simazine
C-I-L Vegetation Killer	Amitrole+simazine
Aqua-Biotics Algae Destroyer	Atrazine+dichlone+monuron +simazine
Algimycin GLB-X Algicide	Atrazine+monuron+simazine
Algimycin 400	POD*+simazine
Terraklene	Paraquat+simazine
Mag-X-cide	Atrazine+diuron+simazine
Later's Calcide	Amitrole+simazine
Swimfree Non-metallic Algaecide	POD+simazine
Lal Ultra	POD+simazine
Western Purity Biochemical Algymicide 444 (Algyzine)	POD+simazine

Source = Agriculture Canada 1989

*POD = poly(oxyethylene(dimethyliminio)ethylene(dimethyliminio) ethylene dichloride).

application are broadcast or band (Ontario Ministry of Agriculture and Food, 1989). Agricultural application rates are usually 2–4 kg•ha^{-1}, but non-crop vegetation control rates may be as high as 20 kg•ha^{-1}. For aquatic weed control, simazine is applied to yield water concentrations of 0.5–2.5 mg•L^{-1} on a water-volume basis (Smith *et al.*, 1982; Jenkins and Buikema, 1990).

In Ontario, in 1978, 8260 kg of simazine were used on field crops, fruits, vegetables, and roadsides (Roller, 1979). In 1983, 3000 kg were used (McGee, 1984). In 1988, 7860 kg of simazine were used on field crops, fruits, and vegetables (Moxley, 1989). This indicates a fairly constant use of the herbicide in the province over 10 years. In Nova Scotia, a survey of major pesticide retailers indicated that nearly 2000 kg of the active ingredient in simazine were sold in 1986 (D.R. Briggins, 1990, Nova Scotia Department of the Environment, pers. com.). Sales of simazine in Alberta have been reported to average about 3 t per year over the years 1981–86 (H.P. Sims, 1990, Alberta Environment, pers. com.). In 1986, 62 t of formulated simazine and 1615 t of technical-grade simazine were imported into Canada (Statistics Canada, 1987). In 1987, 219 t of formulated simazine and 1684 t of technical grade simazine were imported (Statistics Canada, 1988).

Physical and Chemical Characteristics

Simazine is known as an s-triazine because of the symmetrical orientation of the nitrogen atoms on the triazine ring. Its structural formula is shown in Figure 1. The U.S. EPA (1984) reregistration guideline for simazine and Worthing and Walker (1987) list its American trade names.

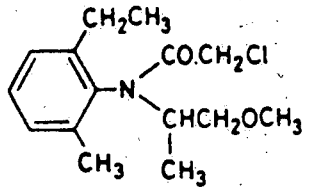

Figure 1. Structural formula for simazine.

The physical and chemical properties of simazine are summarized in Table 3. Simazine is a solid at room temperature and standard pressure and has a low vapour pressure (8.1 x 10^{-7} Pa at 20°C) and octanol/water partition coefficient (log K_{ow} = 1.9). Reported aqueous solubilities for simazine at 20°C range from 3.5 to 5.0 mg•L^{-1}. Glotfelty *et al.* (1984) calculated an adsorption constant (K_d) of 12.5 and an organic carbon/water partition coefficient (K_{oc}) of 284 on a sediment that had an organic matter content of 4.4%. (The unitless K_d was calculated from initial and equilibrium herbicide concentrations in a simazine–sediment solution determined by liquid scintillation counting of ring-labelled simazine after 24 h of agitation on a mechanical shaker. The K_{oc} was calculated as the K_d divided by the percent organic matter content of the sediment, then multiplied by 100.) Reported soil adsorption constants (K_d = amount of herbicide adsorbed in mg•kg^{-1} divided by the solution concentration in mg•L^{-1}) ranged from 1.0 to over 21 and were related to soil organic matter content, cation exchange capacity, and clay content (Talbert and Fletchall, 1965).

Table 3. Physical and Chemical Properties of Simazine

Property	Value
Chemical formula	$C_7H_{12}ClN_5$[1]
Molecular weight	201.7
Elemental analysis	C, 41.68%; H, 5.95%; N, 17.58%; O, 34.71%
Physical state	Colourless powder[1]
Specific gravity	1.302 g•cm^{-3} at 20°C[1]
Melting point	225°C–227°C (with decomposition)
Boiling point	Not determined
Vapour pressure	8.1 x 10^{-7} Pa at 20°C[1] (6.07 x 10^{-9} mmHg)
Henry's law constant	0.00034 Pa•m^3•mol^{-1}[2]
Aqueous solubility	5 mg•L^{-1} at 20°C[1]
	2 mg•L^{-1} at 0°C[3]
	3.5 mg•L^{-1} at 20°C[3],[4],[5]
	84 mg•L^{-1} at 85°C[3]
Solvent solubility	
- chloroform	0.09 g•L^{-1} at 20°C[1]
- diethyl ether	0.3 g•L^{-1} at 20°C[1]
- light petroleum	0.002 g•L^{-1} at 20°C[1]
- methanol	0.04 g•L^{-1} at 20°C[1]
Log K_{ow} (estimated)	2.18
	1.9
K_{oc}	0.140 m^3•kg^{-1}[7]
pK_a	1.65[6]
	1.7[1]

* High mobilization in soil (McCall *et al.*, 1981).
[1] Worthing and Walker, 1987
[2] Suntio *et al.*, 1988
[3] Weed Science Society of America, 1983
[4] Marchini *et al.*, 1988
[5] U.S. EPA, 1987
[6] Technical Database Services, 1986
[7] Jury *et al.*, 1987
[8] Dubach, 1970

Mode of Action

The triazine herbicides, including simazine, are potent inhibitors of the Hill reaction of photosynthesis (Esser *et al.*, 1975; Bryfogle and McDiffett, 1979). The triazines interfere with the complex of biochemical reactions involving phytokinin and indolylacetic acid, which affects protein synthesis (Knüsli, 1970). The interference of the Hill reaction by simazine was studied by Moreland *et al.* (1959) using chloroplasts isolated from barley plants. Simazine inhibited the photochemical activity of the isolated chloroplasts, as evidenced by a decrease in the reduction of ferricyanide. Other postulated mechanisms for the herbicidal activity of simazine include plant growth regulation and effects on nitrogen and nucleic acid metabolism (U.S. Department of Agriculture, 1984). Leblová and Rovenská (1987) found that simazine inhibited alcohol dehydrogenase in germinating peanut seeds through a competition with nicotinamide adenine dinucleotide (NAD) for the enzyme binding site.

The means of resistance to simazine is the ability of certain plants, such as corn, to convert the compound to the herbicidally inactive 6-hydroxy analogue (Worthing and Walker, 1987). There are three major degradative pathways for simazine in plants, animals, and soil: hydrolysis of the substituent group at C_2, stepwise dealkylation at the C_4 and C_6 sites, and the splitting of the triazine ring (Knüsli, 1970). Simazine is quickly metabolized by tolerant plant species to hydroxysimazine and amino acid conjugates. Hydroxysimazine can be further degraded by dealkylation and hydrolysis of amino groups. The accumulation of unaltered simazine in sensitive plant species causes chlorosis and death (Funderburk and Davis, 1963; Bryfogle and McDiffett, 1979; U.S. EPA, 1988).

Methods of Analysis

Current analytical methods for simazine include gas–liquid chromatography (GLC) and high-performance liquid chromatography (Worthing and Walker, 1987). Various detectors for GLC have been used, including alkali flame ionization detector (AFID), nitrogen–phosphorus detector (NPD), Coulson conductivity detector, flame ionization detector (FID), electron capture detector (ECD), and photoionization detector. Mass spectrometry (MS) and liquid chromatography (LC) have been used, and combined LC/ MS methods have been developed. Lee and Stokker (1986) used GLC with NPD to achieve a detection limit of 0.025 µg•L^{-1} for simazine spiked into water samples. Richards *et al.* (1987) used dual-column capillary GLC with NPD in their study of pesticides in rainwater; the detection limit was 0.25 µg•L^{-1}. Bagnati *et al.* (1988) used GLC with selected-ion recording MS to detect s-triazine residues in groundwater at concentrations as low as 0.03 µg•L^{-1}. Detection limits of about 5–10 ng•L^{-1} using GLC/MS with an NPD detector have been achieved for simazine in water (Víden *et al.*, 1987). A GC equipped with an ECD or N–P thermionic selective detector was used by Pionke *et al.* (1988) to achieve a minimum detection limit of 3 ng•L^{-1} for simazine in water. Finally, Albanis *et al.* (1986) used GC with ECD and FID to achieve a minimum detection limit of 0.2 ng•L^{-1} for simazine in water. Selim *et al.* (1989) recently used a bioassay with the aquatic flowering

plant Eurasian watermilfoil (*Myriophyllum spicatum*) to detect a concentration of simazine in water of 20.2 $\mu g \cdot L^{-1}$; they listed simplicity and inexpensiveness as the advantages of the method.

Entry into the Environment

Translocation of simazine from agricultural applications to surface waters may result from spraying directly into watercourses, from vapour drift and precipitation, and from surface runoff and groundwater intrusions from treated lands. As simazine is registered for use as an aquatic herbicide, it is also added to watercourses and ditchbanks.

Richards *et al.* (1987) detected simazine in rainwater collected at four stations in the north-central United States. Simazine was detected in 8–23 of the 30 rainwater samples collected at each station, at concentrations ranging from below 0.1 to 0.5 $\mu g \cdot L^{-1}$. Simazine has also been found in fog, with concentrations at sites in Maryland and California ranging from 0.045 to 1.2 $\mu g \cdot L^{-1}$ in the fog water and below 0.2 $\mu g \cdot L^{-1}$ in the "interstitial air" of the fog (Glotfelty *et al.*, 1987).

Accidents and spills of simazine have been reported to contaminate surface waters (Frank *et al.*, 1982). These include mixing herbicides or cleaning equipment close to watercourses, spills into water, and seepage from discarded containers. Frank *et al.* (1982) estimated that of the total 2-year loss of simazine from 11 watersheds in southern Ontario, 43% was due to storm runoff and snowmelt events, 56% to "spills," spray drift, and direct application to streams, and 1% to "baseflow" from internal soil drainage. The mean loss was calculated to be 26 $mg \cdot ha^{-1}$ for 1975–76, and 8 $mg \cdot ha^{-1}$ for 1976–77. The ratio of application to loss in the first year was calculated to be 0.0007.

Reported levels of simazine in agricultural runoff are summarized in Table 4. Triplett *et al.* (1978), for instance, examined residues from eight conventional and 14 no-tillage watersheds over 3 years in Ohio. The watersheds, planted to corn, were small (0.4–3.5 ha) and sloped (8%–22%). The highest simazine concentration in runoff, 1200 $\mu g \cdot L^{-1}$, occurred during the first runoff event (22 d post-treatment) from a conventionally tilled watershed. Herbicide losses and runoff from the no-till watersheds were lower. This reduction in runoff was attributed to increased infiltration and resistance to overland flow by the mulch cover. The maximum annual loss from any watershed was 5.4% (0.123 $kg \cdot ha^{-1}$) of the initial application.

Glotfelty *et al.* (1984) studied the movement of simazine from cornfields to the Wye River estuary in Maryland. The total amount of herbicide reaching the water depended upon the quantity applied and the timing of runoff with respect to the date of application. In a year when runoff occurred 2 weeks after application, about 0.3% of the herbicide moved to the estuary. The concentration of simazine peaked near 300 $\mu g \cdot L^{-1}$ in the first runoff, but this level declined rapidly, and runoff losses effectively ceased (detection limit 0.01 $\mu g \cdot L^{-1}$) after about 6 weeks.

Glenn and Angle (1987) conducted a 5-year study in the coastal piedmont region of Maryland on a loam soil that was planted to corn and cultivated using conventional and no-tillage techniques. Two small watersheds with 6%–7% slopes were treated with simazine at 2.2 kg $ai \cdot ha^{-1}$. The maximum concentrations of simazine in runoff were 456 $\mu g \cdot L^{-1}$ from the conventionally tilled field and 210 $\mu g \cdot L^{-1}$ from the no-till field 2 weeks after herbicide application. These concentrations decreased to 4 and 0.6 $\mu g \cdot L^{-1}$, respectively, after 18 weeks. The total runoff loss of simazine from the conventionally tilled and non-tilled fields was 0.52% and 0.36%, respectively, of that applied.

Simazine may also enter the aquatic environment as a result of ditchbank applications for weed control. Anderson *et al.* (1978) studied concentrations of simazine residues in irrigation water after simazine applications of 2.25–7.43 $kg \cdot ha^{-1}$ to sections of the banks of flowing and dry irrigation canals in California, Colorado, and Washington. Residue concentrations found in flowing canal water immediately after application to one bank of the canal did not exceed 60 $\mu g \cdot L^{-1}$. In the first-flowing water collected 4–6 months after application to one bank of dry canals, the maximum reported simazine concentration was about 250 $\mu g \cdot L^{-1}$ (from an application of 4.5 $kg \cdot ha^{-1}$). This concentration decreased rapidly, however, to less than 5 $\mu g \cdot L^{-1}$ in the fourth volume of water passing through a sampling point located at the downstream end of the treated section.

In a similar study undertaken in Saskatchewan by Smith *et al.* (1975), simazine was applied to irrigation ditches in the fall of 1970 at a rate of 22.4 $kg \cdot ha^{-1}$.

Table 4. Simazine Residues in Runoff From Agricultural Land

Plot description (soil type/crop)	Formulation (% ai)	Application rate ($kg \cdot ha^{-1}$)	Method of Application	Residues in runoff ($mg \cdot L^{-1}$)	Time post-treatment	Reference
Chesapeake Bay, 1984 20 ha, 0%–2% slope, silt loam soil, corn	wettable powder	1.68	—	~0.300 Effectively 0.0	2 weeks 6 weeks	Glotfelty *et al.*, 1984
Coshocton, Ohio, non-glaciated soils, conventional tillage, corn	—	4.48	—	1.2	22d, first runoff event	Triplett *et al.*, 1978
	—	2.24	—	0.65	22 d first runoff event	
Coshocton, Ohio, non-glaciated soils, no tillage, corn	—	2.24	—	0.27	10 d first runoff event	Triplett *et al.*, 1978
	—	1.68	—	0.42	4 d, first runoff event	
Howard County, Maryland, 0.37 ha, 6% slope, coarse loam, pH 5.9, 2.7% OM, conventional tillage, corn	100%	2.2	Pre-emergence	*1979* 0.456 0.004 *1981* 0.002 *1982* 0.001	 2 weeks 16 weeks 4 weeks 1 week	Glenn and Angle, 1987
Howard County, Maryland, 0.26 ha, 7% slope, coarse loam, pH 5.9, 2.7% OM, no tillage, corn	100%	2.2	Pre-emergence	*1979* 0.210 0.0006 *1981, 1982* 0.0	 2 weeks 16 weeks 4 and 1 weeks, respectively	Glenn and Angle, 1987

Om = organic matter

Simazine concentrations in water immediately after the first filling of the ditches in June 1971 were approximately 700 $\mu g \cdot L^{-1}$. The first irrigation waters collected during the same month contained simazine at a concentration of about 150 $\mu g \cdot L^{-1}$. The second irrigation waters, collected in September 1971 (12 months after application), contained about 70 $\mu g \cdot L^{-1}$. Concentrations steadily decreased, and no detectable simazine residues were recovered in waters collected during the tenth filling of the irrigation ditches in September 1973.

Wind erosion is another potential mechanism by which simazine may be translocated to non-target areas. Gaynor and MacTavish (1981) reported that 8 d after application of granular simazine at 4.4 $kg \cdot ha^{-1}$ to field plots in southwestern Ontario, an early spring windstorm removed 43% of the applied herbicide. Simazine was deposited 2.5 m downwind at concentrations ranging from less than 0.1 to 0.2 $kg \cdot ha^{-1}$. These concentrations were in the range of phytotoxicity for susceptible crops.

Airborne simazine has also been found in dust at pig-fattening farms in Russia (Raszyk, 1986). A mean simazine concentration of 0.060 ± 0.020 $mg \cdot kg^{-1}$ was found on deposited dust particles, but no further details were provided.

Concentrations in Water, Sediment, and Biota

Concentrations of simazine in water, sediment, and biota are summarized in Appendix A. Reported surface water concentrations range from below

detection limits to a maximum of 1300 $\mu g \cdot L^{-1}$, found in an American surface water sample (U.S. EPA, 1987).

Frank *et al.* (1979) reported simazine residues in water samples taken at the mouths of Canadian streams flowing into the Great Lakes from southern Ontario. In July 1977, simazine was detected in 26 of the 92 streams sampled (one sample per stream; detection limit 0.02 $\mu g \cdot L^{-1}$), with a mean concentration of 0.2 $\mu g \cdot L^{-1}$. The highest reported concentration in the water of any stream was 6 $\mu g \cdot L^{-1}$. The results of the stream water analyses on a watershed basis are presented in Table 5.

Table 5. Concentrations of Simazine Residues in Canadian Streams Flowing into the Great Lakes

Location	No. of streams (samples)	Simazine concentration ($\mu g \cdot L^{-1}$) Range	Mean
Lake Ontario	39	ND–6.0	0.3
Lake Erie/Niagara River	23	ND–0.6	<0.1
Lake St. Clair and St. Clair River/ Detroit River	11	ND–0.9	0.1
Lake Huron	19	ND–2.7	0.1

ND = not detected

No simazine was found (detection limit of 0.05 $\mu g \cdot L^{-1}$) in 45 suspended solids samples collected from the mouths of 12 Ontario streams from 1974 to 1976 (Frank *et al.*, 1979). Similarly, no simazine was detected on 30 suspended solids and stream bed sediment samples collected at the mouths of the Grand and Saugeen rivers, Ontario, during 1976–77. Simazine was found at a mean concentration of 1.2 $ng \cdot L^{-1}$ in the water at the mouth of the Grand River and at 0.3 $ng \cdot L^{-1}$ in the water at the mouth of the Saugeen River (Frank, 1981).

Roberts *et al.* (1979) reported finding simazine in 132 of 320 water samples (37%) collected from the Hillman Creek watershed in southwestern Ontario during 1973–75. Concentrations ranged from less than 0.02 to 3.6 $\mu g \cdot L^{-1}$, with a highest reported monthly mean of 0.3 $\mu g \cdot L^{-1}$. No simazine residues were found in 33 whole fish samples of three fish species at the same location.

Frank and Logan (1988) studied pesticide loading in three watersheds in agricultural areas in southwestern Ontario. They collected 440 river-mouth water samples between January 1981 and December 1985. Simazine was not detected in water samples collected during the pretreatment period of January–April (detection limit <0.02 $\mu g \cdot L^{-1}$). During 1 typical year of the study (1983), 260 kg of simazine were applied to the 679 000-ha area of the Grand River basin. Simazine was not detected in any of the 95 river-mouth water samples collected from this basin over the 5 years of the study. Also during 1983, 10 kg of simazine were applied to the 399 840 ha of the Saugeen River basin; one of 143 river-mouth water samples collected contained simazine, (at a concentration of 0.1 $\mu g \cdot L^{-1}$). During the same year, 520 kg of simazine were applied to the 684 000-ha area of the Thames River basin. Of 202 river-mouth water samples collected over 5 years, simazine was detected in eight samples at a mean concentration of 1.1 $\mu g \cdot L^{-1}$.

Frank *et al.* (1987a, 1987b) investigated pesticide contamination of farm wells in southern Ontario from 1979 to 1984. Simazine was detected in 4 of 112 wells where contamination from surface runoff or spray drift was suspected (maximum concentration 6.0 $\mu g \cdot L^{-1}$, detection limit 0.1 $\mu g \cdot L^{-1}$), and in 6 of 48 wells where contamination as a result of spills was suspected. In the latter wells, a maximum concentration of 2070 $\mu g \cdot L^{-1}$ was measured 1 d after a herbicide tank mixture had been back-siphoned directly into a 7-m dug well (Frank *et al.*, 1987b). In the 1984 farm well water survey (Frank *et al.* 1987a), simazine was not detected in any of the wells surveyed (detection limit 0.1 $\mu g \cdot L^{-1}$); however, the pesticide was used on only one of the 91 farms included in the survey. During follow-up studies, 179 wells were sampled over the years 1986–1987. Simazine was used on four farms in both years, but no residues of the herbicide were detected in any of the wells (Frank *et al.*, 1990a). Frank (1986) summarized the well surveys by reporting that 15 of 596 farm wells in Ontario that were suspected of pesticide contamination and were sampled between 1969 and 1984 were found to contain simazine. The main causes of the contamination were storm runoff, spray drift, and spills. During these studies, between 1971 and 1985, water samples from 211 rural ponds in Ontario were also analyzed for pesticide residues (Frank *et al.*, 1990b). Simazine was found in 10 of the ponds; eight instances of contamination resulted from surface water runoff into the ponds (mean concentration 1.0 $\mu g \cdot L^{-1}$, range 0.1–3.0 $\mu g \cdot L^{-1}$), and two instances occurred because of spills (mean concentration 1470 $\mu g \cdot L^{-1}$, range 246–2694 $\mu g \cdot L^{-1}$).

Ripley *et al.* (1986) sampled well water at 291 farms in Ontario in 1985. Six wells had simazine levels

above 1.0 $\mu g \cdot L^{-1}$. The authors cited the incidence of spills, careless handling of pesticides, and the faulty or poor construction of the wells as the cause of much of the contamination.

A recent survey of 145 farm wells for pesticide contamination in Nova Scotia revealed five wells with simazine concentrations ranging from 0.22 to 3.4 $\mu g \cdot L^{-1}$ (detection limit 0.02 $\mu g \cdot L^{-1}$). Several traces of desethyl simazine were also detected (D.R. Briggins, 1990, Nova Scotia Department of the Environment, pers. com.).

In other provinces, no simazine was detected in 77 water samples from Quebec, New Brunswick, and Alberta (Bailey, 1985; O'Neill and Bailey, 1987; NAQUADAT, 1989; AEC, 1989). Detection limits ranged from 0.05 to 1.0 $\mu g \cdot L^{-1}$. No simazine was found in 54 New Brunswick sediment samples (Bailey, 1985; O'Neill and Bailey, 1987). In Quebec, concentrations of 0.4, 0.3, 0.2, and 0.2 $\mu g \cdot L^{-1}$, respectively, were measured for the municipalities of Saint-Hyacinthe, Bécancour, Nicolet, and Sorel (I.-Giroux, 1989, Ministère de l'environnement du Québec, pers. com.).

Simazine has been found as a frequent contaminant of water in Europe. In central Europe, 82 of 118 water samples collected from nine streams during 1976–77 contained simazine residues (Hormann *et al.*, 1978). However, no sample contained more than 10 $\mu g \cdot L^{-1}$ of simazine. Simazine was found very frequently in the Po River in Italy (Galassi *et al.*, 1989), and concentrations ranging from below 0.01 to 0.06 $\mu g \cdot L^{-1}$ were discovered in samples taken from raw water sources and finished (treated) drinking water sources. Also in Italy, Bagnati *et al.* (1988) found simazine in wells (maximum concentration 0.2 $\mu g \cdot L^{-1}$). Albanis *et al.* (1986) reported simazine concentrations in surface water ranging from less than 0.2 $ng \cdot L^{-1}$ (the detection limit) to 80.2 $ng \cdot L^{-1}$ in an agricultural basin located in northwestern Greece.

Monitoring studies have reported simazine in the surface waters of 22 states in the United States (U.S. EPA, 1987). Simazine was found in a total of 877 of 5067 (17.3%) surface water samples and in 229 of 2282 (10.0%) groundwater samples. The maximum concentration found in the surface waters was 1300 $\mu g \cdot L^{-1}$, and in the groundwater, 800 $\mu g \cdot L^{-1}$. In a study by Monsanto reported by Wnuk et al. (1987), simazine was detected in one of 130 untreated surface water samples in Iowa (detection limit 0.27 $\mu g \cdot L^{-1}$) and in none of the 140 samples of treated water collected (detection limit 0.2 $\mu g \cdot L^{-1}$). Howell and Ries (1972) found no simazine in a stream running through an agricultural area with "heavy use" of simazine in central Michigan (no further details were provided).

Pionke *et al.* (1986) tested water from 18 wells and two springs in agricultural areas of Pennsylvania. Simazine was found in three wells with a concentration range of 0.049–0.170 $\mu g \cdot L^{-1}$. Fishel and Lietman (1986) also sampled groundwater in Pennsylvania and detected a maximum concentration of 3.4 $\mu g \cdot L^{-1}$ during the summer. In 1985, Pionke *et al.* (1988) found simazine in groundwater collected by 3 of 20 wells and piezometers sunk into a 740-ha watershed in Pennsylvania. In 1986, six of the wells were found to be contaminated, whereas five more had simazine in trace concentrations (detection limit 0.003 $\mu g \cdot L^{-1}$). Measureable concentrations in the 2 years ranged from 0.01 to 0.170 $\mu g \cdot L^{-1}$. Well contamination occurred in spite of the fact that simazine was applied in proximity to one well only. Cohen *et al.* (1984) reported that simazine was found in 6 of 166 wells sampled in California, at concentrations between 0.5 and 3.5 $\mu g L^{-1}$. More recently, Cohen *et al.* (1986) reported that simazine had been found in the groundwater of three states (California, Pennsylvania, and Maryland) at concentrations typically ranging between 0.2 and 3.0 $\mu g \cdot L^{-1}$.

Environmental Fate, Persistence, and Degradation

Soil

The fate of a herbicide in soil depends on a number of interrelated factors, including the type and rate of application, the physical and chemical properties of the herbicide and the soil, the climatic conditions, the amount of leaching, microbial activity, chemical decomposition, volatilization, photodegradation, and plant uptake and metabolism (Ivey and Andrews, 1965; Reed and Holt, 1982). Laboratory studies have indicated that soil degradation of simazine results from both chemical and biochemical processes (Jordan *et al.*, 1970; Esser *et al.*, 1975; Smith, 1985). Non-biological detoxification of simazine in soil can occur through photodecomposition, phototransformation, volatilization, and hydroxylation and dealkylation reactions (Jordan *et al.*, 1970).

Microbial degradation may be the dominant pathway of simazine degradation in soil (Weed Science Society of America, 1983). Kaufman and Kearney (1970) listed the numerous soil microorganisms

capable of degrading the herbicide. Studies with the soil microbe *Aspergillus fumigatus* showed that the organism degraded simazine through dealkylation or deamination reactions, or both, of the side chains, without the production of hydroxysimazine (Kaufman *et al.*, 1965). The authors concluded that cleavage of the triazine ring was unlikely during their experiments.

Laboratory experiments have shown that photodecomposition of simazine will occur (Jordan *et al.*, 1970). Comes and Timmons (1965) studied the photodecomposition of soil-applied simazine exposed to sunlight. Simazine loss was 25% during the first 25 d of the spring. Soil temperatures were high, however (65°C–82°C in the summer), and volatilization as well as photodecomposition may have contributed to the loss. Although the relevance of photodecomposition to the loss of simazine applied to the soil remains unclear (Jordan *et al.*, 1970), the Weed Science Society of America (1983) considered the loss of simazine by photodecomposition under normal climatic conditions to be insignificant.

Studies with radioactively labelled simazine have shown that simazine will volatilize from metal planchets, and from sand and clay substrates, at elevated temperatures (approximately 72°C) (Davis *et al.*, 1959). Jordan *et al.* (1965) noted a rapid loss of simazine from metal planchets at 43°C in the dark. Foy (1964) reported a 35% loss of simazine in 24 h at 25°C from nickel planchets. Kearney *et al.* (1964) noted that volatilization proceeded more slowly from soil than from metal planchets. They reported a 10% loss of simazine from soils maintained at 35°C for 72 h and a half-life of 2 months at 71°C–74°C. The low vapour pressure of simazine (8.1×10^{-7} Pa at 20°C) may be the reason that the Weed Science Society of America (1983) concluded that loss of simazine from soil by volatilization is inconsequential.

Non-biological hydroxylation of simazine may also occur (Jordan *et al.*, 1970). Soil treatments with the microbial inhibitor sodium azide at 200 $mg \cdot kg^{-1}$ did not prevent the accumulation of hydroxysimazine, indicating that hydrolysis was occurring in the absence of microbial activity (Harris, 1967). No data were presented, however, on the amount of microbial inhibition occurring as a result of the soil treatments. It has been concluded that the amount of organic matter in the soil, because of its catalytic properties, enhances the non-biological hydroxylation of simazine (Esser *et al.*, 1975).

The factors that influence the leaching of simazine through soil include its adsorption relationships with soil colloids, the physical properties of the soil, the amount of water passing through the soil, climatic conditions, and the aqueous solubility of the herbicide (Hall and Hartwig, 1990). Simazine, as a weakly basic herbicide, is protonated to a cation in water or soil solutions; the protonated herbicide can then be adsorbed on soil colloids (Kalouskovà, 1986). These adsorption processes are pH-dependent, with adsorption occurring most readily at pH 1.7, which is equal to the pK_a of the herbicide (Worthing and Walker, 1987). Adsorption decreases with increasing pH as the amount of protonated herbicide decreases. Kalouskovà (1986) found that the interaction between simazine and humic acids involved the formation of ionic bonds, hydrogen bonds, and physical adsorption by van der Waals forces.

Simazine exhibits very complex leaching behaviour in soil (Day *et al.*, 1968; Hance, 1984) but generally has limited mobility (Roadhouse and Birk, 1961; Helling, 1970; Smith *et al.*, 1975; Reed and Holt, 1982; Jensen and Kimball, 1982; Hall and Hartwig, 1990). (As mentioned above, its presence in wells and groundwater can often be explained by spills or mishandling.) Simazine readily adsorbs on muck and soils of high organic matter and clay content and has little tendency to leach either vertically or horizontally in these soils (Nearpass, 1966; Scott and Lutz, 1971; Jensen and Kimball, 1982). Conversely, it has a relatively weak potential for adsorption in sandy soils and will more readily leach in these soils than other herbicides with higher water solubilities (e.g., prometryne) (Caro, 1976).

The behaviour of simazine in soil may also influence its phytotoxic properties. Upchurch *et al.* (1966) measured, at 17 field locations in the coastal plain of North Carolina, 14 characteristics of the soil, climate, and biotic factors that might influence the phytotoxicity of simazine. These were correlated with plant phytotoxic responses obtained after normal and above-normal simazine applications. There was a strong negative correlation between phytotoxicity and soil organic matter content, which would indicate a decrease in available simazine due to adsorption.

In a study by Day *et al.* (1968), the phytotoxicity of soil slurries of 100 g soil, 100 g water, and 400 µg simazine was measured using 65 different California soils. Simazine phytotoxicity was related to the organic

matter content and cation exchange capacity of the soil and the amount of simazine available in solution. The amount of simazine in solution was positively related to the sand content and negatively related to the clay content and cation exchange capacity. Simazine phytotoxicity was more closely related to the organic matter content of the soil than to any of the other soil properties. The authors stated that no simple model explains the relationship between soil and the biological activity of simazine. Their data would suggest, however, that over 80% of the variability in the phytotoxicity of simazine in 65 soils could be explained by incorporating only three parameters in the regression equation: percent organic matter, cation exchange capacity, and amount of simazine in solution. Earlier reports have also mentioned that adsorption of simazine onto soil colloids accounts for a significant loss of phytotoxicity; the adsorption itself has been correlated with a number of soil parameters, but soil organic matter content appears to be the most important factor (Burnside *et al.*, 1963).

Helling (1970) provided a summary of simazine mobility in soil. He concluded from an extensive literature review that simazine was immobile in soil. Dawson *et al.* (1968) applied simazine at 3.36 kg•ha^{-1} to a silt loam soil for 6 consecutive years and then analyzed persistence using an oat bioassay. One year after the last simazine application, 75% of the applied simazine (2.52 kg•ha^{-1}) was found remaining in the 0- to 5-cm soil layer; no simazine was encountered in the 20- to 30-cm soil layer. Similarly, Roadhouse and Birk (1961) found negligible movement of simazine below the 10-cm soil layer 1–2 years after field applications of 0.56 kg•ha^{-1} to 22.4 kg•ha^{-1}. Most of the simazine remained in the upper 2.54 cm.

Soil properties influence the mobility of simazine. Helling (1970) reported a study in which simazine movement through four Swiss soils was compared. The soils were held in 17.5-cm leaching columns through which 20 cm of water were passed. In a soil with 27%–30% organic matter, no leaching occurred, whereas leaching was limited to 7 cm in a soil with 24% clay. Moderate leaching (to 12 cm) occurred in a soil with 4.6%–4.9% organic matter and 11% clay, and extensive movement (to 17.5 cm) occurred in a sand soil. Similarly, Hogue *et al.* (1981) compared the mobility of simazine through soil columns containing two orchard soils from the Okanagan Valley of British Columbia. The two soils were a sandy loam with 70.1% sand, 4.8% clay, and 12.4% organic matter, and a loam soil with 39.5% sand, 22.4% clay, and 3.8% organic matter. Simazine was more mobile in the sandy loam soil.

Ivey and Andrews (1965) studied simazine movement in four soil types using laboratory leaching columns. After application of simazine at rates equivalent to 2.24 kg•ha^{-1}, measured amounts of water (to depths of 7.6, 22.9, and 45.7 cm) were applied to the surface of the soil columns. After the leaching runs were completed, the phytotoxicity at different soil depths was evaluated using an oat bioassay. Simazine was leached the farthest in columns filled with a fine sandy loam soil, followed by a silt loam soil. The least leaching occurred in loam and clay loam soils. The latter two soils are high in clay, organic matter, and cation exchange capacity. No correlation could be made between pH and the amount of leaching of the herbicide.

Field studies concerning simazine mobility in soil have also been conducted. In a Hagerstown silty clay loam soil in Pennsylvania, Hall *et al.* (1989) bored horizontal channels 1.2 m under conventionally tilled (CT) and no-tillage (NT) cornfields and installed plastic gutters to collect water percolating to this depth after rainfall events. A pre-emergence simazine application of 1.7 kg ai•ha^{-1} was made to the soil surface in May. The mean concentration of simazine in NT percolates was higher (3.0 µg•L^{-1}) than in CT percolates (2.8 µg•L^{-1}). The maximum concentration of simazine in NT percolates was considerably higher (21.5 µg•L^{-1}) than in CT percolates (3.8 µg•L^{-1}). The percentage of applied herbicide leaching in 1985 was less than 0.1% for CT and 0.66% for NT.

Simazine residues were detected at all soil depths (i.e., to 1.2 m) in both tillage systems. The maximum simazine concentration (122 µg•L^{-1}) was recorded from the CT system during 1985. The loss for 1985 was 1.56% for CT and approximately 3.2% for NT. The maximum runoff loss of simazine was 0.6% (0.01 kg•ha^{-1}) under CT. The authors concluded that the yearly differences were related to the number of leaching events and their proximity to the herbicide application date.

Hance (1984) reported the results of studies in which the leaching columns were cylinders of soil held in drainpipes driven into field plots. There was little movement of simazine over 16 months. The experiment was also conducted in the laboratory where, with continuous leaching using 20.6 cm of water, the herbicide was more mobile; 7.5% of the applied herbicide

emerged from the bottom of a 30-cm column. However, as nearly 40% of the simazine was retained in the top 1 cm, solubility, as well as adsorption, was apparently affecting the behaviour of the herbicide in the leaching column. Because the compound leached in the laboratory column but not in the field, the author concluded that laboratory leaching column studies may give a misleading indication of actual field behaviour.

Simazine still remained in the top 8 cm of a silty clay loam and two loam soils in Nebraska 4 months after application (Burnside *et al.*, 1963). Twelve months after treatment, oats were injured on all soils at all application rates (2.8, 5.6, and 11.2 $kg \cdot ha^{-1}$). Yet there was considerable loss of simazine after 16 months from the 0- to 8-cm soil depth, with an average of only 0.63 $\mu g \cdot g^{-1}$ in this layer 16 months after the 11.2 $kg \cdot ha^{-1}$ application. These data support the conclusion of Dawson *et al.* (1968) that injury to plants may occur if a sensitive crop is rotated onto a field to which simazine was applied the previous year.

Other simazine persistence studies are summarized in Table 6 and Appendix B. In two sandy loam orchard soils in southern Ontario, seven to nine annual applications of 4.5 $kg \cdot ha^{-1}$ resulted in little accumulation of simazine; the annual loss was over 95%. However, the herbicide and its metabolite hydroxysimazine (a non-phytotoxic degradation product) persisted for up to 40 months in the soils (Khan and Marriage, 1979). Hydroxysimazine degraded more slowly than the parent compound and residue levels of the metabolite were 40 times those of the parent simazine 28–48 months after the final application. No dealkylated metabolites of simazine were found.

Simazine degradation has been studied under field conditions in British Columbia, Alberta, Saskatchewan, Ontario, and Nova Scotia (Smith 1982, 1985). Persistence of simazine was greater in the western provinces, with carry-over after 52 weeks of over 20% compared with about 10% in the eastern provinces. The author did not speculate on the reasons for this difference but mentioned earlier (Smith and Hayden, 1976) that the climatic conditions of western Canada—long, cold winters and hot, dry summers—may result in carry-over of herbicide residues in these areas.

A 3-year study of the persistence of herbicides at three locations in Saskatchewan was conducted by Smith and Hayden (1976). The plots were sampled 5 months after a May application of simazine. Residues were detected at the end of the growing season, and minimal degradation occurred during the winter months. Minimal leaching occurred, as most of the residues were recovered from the 0- to 5-cm soil layer. The 5-month loss of simazine amounted to 65.7% of the herbicide applied, whereas the loss after 17 months was approximately 78.3%. In a southern Ontario field study, an average of 44% of the applied simazine (0.56–22.4 $kg \cdot ha^{-1}$) remained 47 d after a May application, and 8.4% remained 1 year after application (Roadhouse and Birk, 1961).

In Poland, residues of simazine toxic to oats did not persist beyond one growing season in an uncropped light loamy sand soil treated with 1.5 $kg \cdot ha^{-1}$ (Zurawski and Ploszynski, 1968). However, at application rates of 5 and 10 $kg \cdot ha^{-1}$, simazine phytotoxicity persisted until the following summer (13 months). When applied at 25 kg $ai \cdot ha^{-1}$, phytotoxic residues persisted for up to 26 months. The cultivation of maize on the test plots led to a more rapid dissipation of the simazine. Half-life values based on the residues remaining in the top 20 cm of soil after application of 1.5–25 $kg \cdot ha^{-1}$ averaged 4–4.5 months for uncropped plots and 3–4 months for plots cultivated with maize.

In Sweden, Torstensson (1974) found that a 2 $kg \cdot ha^{-1}$ application of simazine left residues (concentrations not given) in a silty clay soil (43% silt, 42% clay) at the end of the sampling period 99 d later. Allott (1969) found 80% simazine degradation in 11–22.5 weeks in 1966 and 1967, respectively, in the 0- to 5-cm soil horizon of a sandy (66.8%) soil in Northern Ireland.

Zimdahl *et al.* (1970) investigated degradation of simazine in laboratory studies conducted with loam soils at temperatures of 13.2°C and 31.2°C. The results revealed a reaction with first-order kinetics and with no apparent lag period. Degradation proceeded more quickly at the higher temperature (half-life approximately 2 months) than at the lower temperature (half-life 5 months).

The persistence of simazine in soil has been studied extensively since the early 1980s as a result of an international collaborative effort initiated by the European Weed Research Society (EWRS). The results from these experiments were summarized by Walker *et al.* (1983) and Chen *et al.* (1983) (see Table 6). The laboratory studies consisted of incu-

Table 6. Soil Properties and Half-lives of Simazine at Different Locations

Location of site	Organic carbon (%)	Clay (%)	Sand (%)	Silt (%)	pH	Field capacity (%, w/w)	Field half-life (d)	Laboratory half-life at 20°C and 90% field capacity
Regina, Saskatchewan	4.00	69	5	26	7.7	40.0	101	114
Alberta	1.26	32	41	27	7.8	24.9	88	125
Harrow, Ontario (I)	0.52	5	88	7	5.2	14.0	63	62
Harrow, Ontario (II)	1.50	8	78	14	5.6	23.0	63	71
Summerland, British Columbia	0.71	5	79	16	7.5	10.0	38	42
Uppsala, Sweden	3.60	42	28	30	6.5	28.7	88	102
Braunschweig, Federal Republic of Germany	0.99	12	49	39	6.5	23.9	54	58
Wageningen, Holland	2.38	3	89	8	5.6	18.3	51	50
Maarn, Holland	1.40	3	93	4	5.6	8.0	21	21
Warwick, England	1.30	20	75	5	6.6	17.0	46	50
Harpenden, England	1.75	35	31	34	7.5	28.2	37	46
Oxford, England	2.10	15	66	19	5.8	18.0	31	34
Maidstone, England	1.74	10	55	35	7.5	23.7	<14	-
Firenze, Italy	0.98	14	59	27	6.7	23.0	39	39
Taichung, Taiwan (winter season)	0.83	31	42	27	5.2	30.3	24	55
Taichung, Taiwan (summer season)	0.83	31	42	27	5.2	30.3	18	55
Taipei, Taiwan	1.04	21	32	47	4.3	27.5	14	39
Bogor, Indonesia	1.40	26	57	17	4.6	9.6	<14	-
Horotiu, New Zealand	9.40	15	58	18	5.4	42.8	<14	-

Source: From the European Weed Research Society collaborative studies. Adapted from Chen *et al.* (1983) and Walker *et al.* (1983).
* No laboratory data available.

bating three soil samples, and field investigations consisted of spray treatments of fallow plots with application rates of 2–4 kg•ha^{-1}. Laboratory experiments revealed that in most cases degradation followed first-order kinetics. Significant correlations between laboratory half-lives and soil organic carbon content, clay content, and pH were found, but the improvement over a simple correlation with clay content alone was small. The survey also found that changes in temperature from 10°C to 30°C resulted in two- to five-fold increases in degradation rates. The effect of soil moisture content on degradation rates was more variable. In some soils, the rate of degradation was reduced considerably when dry; half-lives at 0% field capacity were twice as long as those at 90% field capacity. The variability in moisture dependency of simazine degradation rates was attributed to the differences between soils in the relative importance of degradation in the adsorbed and solution phases. The EWRS study produced field half-life estimates for simazine ranging from less than 14 d to approximately 100 d (see Table 6).

The U.S. EPA (1987) concluded that under aerobic soil conditions, simazine loss depends mainly on soil moisture and temperature. In sandy loam soil, half-lives can range from 36 to 234 d. In loamy sand and silt loam soils incubated at 25°C–30°C for 48 weeks, the half-lives were 114 and 179 d, respectively. Under anaerobic conditions, ^{14}C-simazine had a half-life of 56–84 d in a loamy sand soil, and about 30–139 d in sandy loam and silt loam soils. The U.S. EPA (1988) stated that the average half-life of simazine

under anaerobic soil conditions is longer than 12 weeks, whereas the half-life under aerobic soil conditions is 8–12 weeks.

The various mechanisms by which simazine can degrade in soil/sediment and water are summarized in Table 7.

Water and Sediment

There is little information on the fate of simazine in water and sediments. Bioaccumulation of the compound is negligible (see below), and volatilization to the atmosphere would not be a major fate process (low Henry's law constant of 0.00034 $Pa \cdot m^3 \cdot mol^{-1}$; Suntio *et al.*, 1988). The major paths of dissipation of simazine in water under field conditions are slow microbial degradation and, possibly, a sensitized photochemical degradation to N-dealkylated compounds combined with sorption to sediments and aquatic plants (Muir, 1990).

Non-biological degradation of simazine can occur, but there is only a slow hydrolysis of the compound at 70°C in neutral solution; the rate of hydrolysis increases outside of the neutral pH range (Worthing and Walker, 1987). Burkhard and Guth (1981) calculated hydrolysis half-life of 70 d in a buffer solution of pH 5 at 25°C; the hydrolysis product was 2-hydroxysimazine. At pH 7 and 9, the hydrolysis half-life estimates exceeded 200 d at this temperature. Simazine can be relatively persistent in aquatic systems, particularly shallow, well-mixed lakes and ponds (Jenkins and Buikema, 1990). Schwartz *et al.* (1981) applied simazine at a concentration of 0.45 $mg \cdot L^{-1}$ to a 4.1-m-deep lake in Arizona. Two years later, the herbicide was still present in the water of the lake, at a concentration of 0.14 $mg \cdot L^{-1}$. Simazine residues may persist up to 3 years in flooded soil, and dissipation in pond and lake water is variable, with a half-life ranging from 50 to 700 d (U.S. EPA, 1987). The U.S. EPA (1988) later reported that the average half-life of simazine in ponds is 30 d. This value apparently depends on many factors, including the level of algae and weed infestation in the pond.

Tucker and Boyd (1981) investigated the relationship between pond sediments and simazine loss from pond water and sediment solutions in 250-mL flasks. After 32 d, more than 75% of an initial 3 $mg \cdot L^{-1}$ simazine concentration was lost from the water–sediment solution. In flasks where pond water alone (no sediment) was tested, a maximum of only 20% of the simazine was lost. The greater loss in the flasks with sediment was attributed to increased adsorption on the sediment and greater microbial activity in the sediment. Simazine half-life estimates for the pond water in the flasks ranged from 7.8 to 72.5 d and were dependent on organic matter content and pH. Organic matter content of the sediment was positively correlated with the rate of simazine loss from the overlying water ($p \leq 0.01$), whereas sediment pH was negatively correlated with the rate of loss ($p \leq 0.05$). The authors concluded that sediment is the major sink for simazine applied to ponds. The presence of simazine metabolites was not measured during these studies.

From field pond studies, Mauck *et al.* (1976) reported that simazine degraded much more rapidly when adsorbed to sediment. Five ponds near Columbia, Missouri, were drained, refilled, and stocked with 250 subadult bluegills (*Lepomis macrochirus*). The ponds were treated with estimated concentrations of simazine in the water of 0.1, 0.3, 1.0, and 3.0 $mg \cdot L^{-1}$, with the fifth pond being reserved for a control. Samples of sediment, water, benthic invertebrates, and fish were obtained at irregular intervals over a 2-year period. After the 3.0 $mg \cdot L^{-1}$ treatment, residues were still detectable in the water and sediment 346 d after the first application (maximum concentration of 0.14 $mg \cdot L^{-1}$ in the water and 0.32 $\mu g \cdot g^{-1}$ in the sediment) and 456 d after the second application (maximum concentrations of 0.50 $mg \cdot L^{-1}$ in the water and 0.16 $\mu g \cdot g^{-1}$ in the sediment). The maximum sediment concentration was 11.0 $\mu g \cdot g^{-1}$, which occurred in the high-treatment-level pond 15 d after treatment. The time taken for 50% dissipation of simazine from initial concentrations in water of 450–870 $\mu g \cdot L^{-1}$ ranged from 155 d for the low concentration to about 246 d for the high concentration. The time taken for 50% dissipation from sediment for an initial concentration of 8200 $\mu g \cdot kg^{-1}$ was 7–10 d and from an 11 000 $\mu g \cdot kg^{-1}$ initial concentration, 15–25 d.

RATIONALE

Raw Water for Drinking Water Supply

Guideline

The Guidelines for Canadian Drinking Water Quality (Health and Welfare Canada, 1989a) specify an interim maximum acceptable concentration (IMAC)

Table 7. Summary of Simazine Degradation in Soil/Sediment and Water

Pathway	In soil/sediment	In water
Photolysis	- insignificant[1][2] - little degradation with near UV or sunlight[3][4]	- insignificant[5]
Oxidation	- no data	- no data
Aerobic metabolism	- dominant degradation pathway[4] - depends on moisture and temperature[6] - dissipation in sediment (adsorption or metabolism) depends on organic matter content and pH[7] - major metabolite = hydroxysimazine[8] - pathways: dealkylation, hydrolysis, ring cleavage[9]	- proceeds slowly in absence of sediments[10]
Anaerobic metabolism	- no data	- no data
Hydrolysis	- major non-biological pathway forms hydroxysimazine[9]	- relatively resistant to hydrolysis[5][10] - no hydrolysis in stable aqueous solution over 28 d[6] - $t_{1/2}$ = 96 d (pH 5)[11]
Volatilization	- insignificant[1][12][13][14] - $t_{1/2}$ = 2 months from metal at 72.5°C[15]	- insignificant[14] - not a major path of loss, as predicted volatilization $t_{1/2}$ (two-film theory) > 1000 d[10]
Mobility	- slightly to very mobile depending on soil texture[6] - little leaching in soil[2][4][5][14] - low concentrations in runoff[16][17][18]	
Adsorption/desorption	- depends on soil organic matter content, cation exchange capacity, and clay content[6] K_d = 1.0 for sandy loam 7.9 for a silty loam >21 for peat and peat moss[4]	
Persistence	$t_{1/2}$ = 8–12 weeks (aerobic soil conditions) >12 weeks (anaerobic soil conditions)[1] 36–234 d (sandy loam soil) 25.5 weeks (silt loam soil) 16.3 weeks (loamy sand soil)[6]	$t_{1/2}$ = 12–456 d (field dissipation depending on application rate[10] 30 d in ponds[1] >32 d in ponds without sediment 8–72 d in presence of sediment mostly due to sorption[7] 5 d (with pond sediment as major sink)[19]

[1] U.S. EPA, 1988.
[2] Weed Science Society of America, 1983.
[3] Jordan *et al.*, 1985.
[4] Talbert and Fletchall, 1964.
[5] Ghassemi *et al.*, 1981.
[6] U.S. EPA, 1987.
[7] Tucker and Boyd, 1981.
[8] Harris, 1967.
[9] U.S. Department of Agriculture, 1984.
[10] Muir, 1990.
[11] Burkhard and Guth, 1981.
[12] Comes and Timmons, 1965.
[13] Foy, 1964.
[14] Burnside *et al.*, 1961.
[15] Davis *et al.*, 1959.
[16] Glenn and Angle, 1987.
[17] Glotfelty *et al.*, 1984.
[18] Triplett *et al.*, 1978.
[19] Hawxby and Mehta, 1979.

for simazine in drinking water of 10 $\mu g \cdot L^{-1}$ as recommended by the Federal–Provincial Subcommittee on Drinking Water of the Federal–Provincial Advisory Committee on Environmental and Occupational Health. This was based on a negligible daily intake (NDI) over the lifetime of a 70-kg individual consuming 1.5 L of water per day. The NDI of 0.0013 $mg \cdot kg^{-1}$ body weight (b.w.) was based on a no-observed-adverse-effect level (NOAEL) of 5 $mg \cdot kg^{-1} \cdot d^{-1}$ from a 2-year dog feeding study during which simazine caused reduced body weights, increased concentrations of several liver enzymes, and slight thyroid hyperplasia (Health and Welfare Canada, unpubl. data).

Summary of Existing Guidelines

In the U.S. EPA Health Advisory for simazine (U.S. EPA, 1987), the 1-d and 10-d health advisories for a 10-kg child were 50 $\mu g \cdot L^{-1}$. Longer-term (7-year) health advisories were 50 $\mu g \cdot L^{-1}$ for children and 175 $\mu g \cdot L^{-1}$ for a 70-kg adult. The lifetime health advisory was 35 $\mu g \cdot L^{-1}$ in drinking water (U.S. EPA, 1987). An allowable daily intake (ADI) of 0.005 $mg \cdot kg^{-1} \cdot d^{-1}$ from a 2-year dog study resulted in the development of a drinking water guideline IMAC of 17 $\mu g \cdot L^{-1}$ by the World Health Organization (WHO, 1988). The WHO may lower this IMAC after review of more recent toxicity studies.

Concentrations in Drinking Water Supply

The Ontario Ministry of the Environment (OMOE, 1987a, 1987b) surveyed municipal waterworks and private wells in 1985 and 1986 for the presence of simazine. At eight municipal waterworks in 1985, 121 samples of raw water and 111 samples of treated water were analyzed. Only one raw water sample contained simazine (concentration <0.3 $\mu g \cdot L^{-1}$, including D-ethyl simazine). In 351 private wells sampled in 1985, simazine (including D-ethyl simazine) was detected in 12 wells (maximum concentration 23 $\mu g \cdot L^{-1}$). The authors emphasized that the 351 wells sampled were not randomly selected but were shallow wells in sandy soils in agricultural areas where contamination had been found previously. In 1986, 37 domestic wells and 5 municipal groundwater supply wells in areas of intense corn and soybean production in southern Ontario were sampled (OMOE, 1987b). No simazine was detected in the groundwater (detection limit 0.1 $\mu g \cdot L^{-1}$). Twenty-five different municipal waterworks supplied by surface water sources were also sampled in 1986. Simazine and D-ethyl simazine were detected in 11 of 422 raw surface water samples collected at nine waterworks. Reported levels ranged from less than 0.06 to 0.150 $\mu g \cdot L^{-1}$. Of the 150 treated water samples analyzed, only one sample contained simazine (concentration <0.06 $\mu g \cdot L^{-1}$) (OMOE 1987b).

The Ministère de l'environnement du Québec sampled drinking water supplies in 18 municipalities (representing 50% of the population served by surface water sources) during February and July of 1986 (Anonymous, 1987). Raw and treated water samples were analyzed. The sampling programs detected simazine, but concentrations were below Health and Welfare Canada's (1989a) standard of 10 $\mu g \cdot L^{-1}$ (actual concentrations and detection limits were not provided).

Water Treatment

Miltner *et al.* (1988) reported that conventional water treatment operations were ineffective in removing simazine from water. Baker (1985), who found simazine in tap water at Bowling Green, Fremont, and Tiffin, Ohio, in concentrations similar to those found in river water (actual concentrations not provided), noted that a granular activated carbon filter at the treatment plant at Fremont removed considerable amounts of the herbicide. The U.S. EPA (1987) indicated that treatment operations using high doses of granular activated charcoal (GAC) removed simazine from water. Galassi *et al.* (1989), however, noted that simazine was not removed by water treatment operations in Italy, even though activated charcoal beds were employed. Moreover, the WHO (1988) stated that during water treatment operations using GAC, simazine in the presence of intermediary nitrite might give rise to N-nitroso compounds, which could be carcinogenic. Finally, the Ontario Ministry of the Environment (OMOE, 1987b) emphasized that the doses of powdered activated charcoal (PAC) used for taste and odour control were not effective in removing the high concentrations of pesticides that occurred in rainfall runoff from fields. They recommended increasing the PAC dose to 40–50 $mg \cdot L^{-1}$ before, during, and immediately following rainfall events to reduce pesticide levels in treated water.

Freshwater Aquatic Life

Accumulation and Elimination in Aquatic Biota

Persistence studies of simazine in water and aquatic organisms are summarized in Table 8. The available information indicates that simazine does not bioaccumulate, nor is it biomagnified, in the food web.

Table 8. Simazine Persistence in Water and Aquatic Organisms

Medium	Application rate ($mg \cdot L^{-1}$)	Concentration ($mg \cdot L^{-1}$)	Time after treatment (d)	Half-life (d) (first order)	Comments	Reference
Surface water (Alabama, fish ponds)	1.50	1.50 1.22 1.00 0.97 0.67 0.28 0.07	0 4 8 16 32 64 128	18.5	Average concentrations from 3 ponds; residue levels approximate, because interpolated from graph	Tucker and Boyd, 1978b
Surface water (Missouri, 4 fish ponds)	0.1–3.0	—	—	46–174	Two-year study; half-life range as calculated by Reinert and Rodgers (1987)	Mauck *et al.*, 1976
Pond water	3 (initial concentration)	0.6–0.7 2.5–2.9	32 d with sediment present 32 d without sediment present	7.8–72.5 in water (range for 16 sediment types present)	Laboratory investigation; residues determined in water phase only; temperature = 25°C ± 2°C; organic matter and pH of sediment related to half-life; dissipation due mainly to sorption	Tucker and Boyd, 1981
Pond water (artificial pond in a greenhouse)	2	—	—	5	Temperature 15°C–25°C; time for 90% degradation was 10 d; sediment the major sink for the compound (20% at 16 d vs. 8% in algae at 16 d)	Mehta and Hawxby, 1979
Estuarine water (Wye River estuary, Chesapeake Bay)	—	—	—	~30	Corn cultivation equals 26% of watershed; losses attributed more to surface-catalyzed hydrolysis than effective transport	Glotfelty *et al.*, 1984
Fish (freshwater) (fathead minnows, *Pimephales promelas*)	—	—	—	<3	No residues detected after 3 d in uncontaminated water	Mayer and Sanders, 1977
Fish (freshwater) (green sunfish) *Lepomis cyanellus* Raf.)	—	No residue detected in fish (whole body)	7, exposure to untreated water	—	Depuration half-life <7 d; original residue in fish 0.95 and 2.29 $\mu g \cdot g^{-1}$	Rodgers, 1970

The bioaccumulation potential is low, as evidenced by bioconcentration factors of less than 100 (Appendix C), and simazine concentrations in the tissues of fish rarely exceed the concentration in the water to which they are exposed. Although simazine may have a half-life of 50–700 d in water (U.S. Department of Agriculture, 1984), the depuration half-life in fish is short (<7 d following exposure if the organism is transferred to uncontaminated water) (Rodgers, 1970; Mayer and Sanders, 1977; Niimi, 1987), indicating that it is rapidly excreted or metabolized. Roberts *et al.* (1979) found no simazine residues in whole fish homogenate of brown bullheads (*Ictalurus nebulosus*), gizzard shad (*Dorosoma cepedianum*), and black crappie (*Pomoxis nigromaculatis*) collected in the Hillman Creek watershed of Ontario in 1974, where simazine was detected in the water at concentrations ranging from trace (<0.1 $\mu g \cdot L^{-1}$) to 3.6 $\mu g \cdot L^{-1}$.

In a brief report on the effects of simazine on non-target aquatic organisms, Mayer and Sanders (1977) mentioned that simazine was accumulated by fathead minnows (*Pimephales promelas*) up to 55 times the concentration in their exposure water. No simazine residues were found in the fish after they had been in uncontaminated water for 3 d. No further details were provided. In a study by Mauck *et al.* (1976), residual simazine concentrations in *L. macrochirus* generally did not exceed the concentration in the water to which they were exposed. Some bioaccumulation was evident in benthic invertebrate samples (e.g., mayflies, *Hexagenia* sp.) for the first 3 months, and a bioconcentration factor of approximately 90 could be calculated 8 d after herbicide application. The concentration in the invertebrates declined markedly after this time.

In a laboratory microcosm study, the partitioning behaviour and fate of ^{14}C-labelled simazine applied as a foliar spray in a terrestrial chamber were studied (Gile *et al.*, 1980). The chamber consisted of a synthetic soil medium, Douglas fir and red alder seedlings, rye grass, numerous invertebrates, and a vole (*Microtus canicaudus*). Approximately 80% of the applied ^{14}C was recovered 26 d after application. The remainder may have been lost to the air as $^{14}CO_2$, which was not detectable by the chamber's filtering apparatus. The ^{14}C detected in the various media and organisms as a percentage of herbicide applied was animals, 0.8%; plants, 43%; soil, 35%; and groundwater, 0%. Concentrations in the animals after 26 d were snail, 1.97 $\mu g \cdot g^{-1}$ (fresh weight); snail feces, 9.7 $\mu g \cdot g^{-1}$; pillbugs (*Armadillarium* spp. and *Porcellia* spp.), 0.66 $\mu g \cdot g^{-1}$; mealworm (*Tenebrio molitar*) larvae, 1.04 $\mu g \cdot g^{-1}$; and whole body of vole, 0.59 $\mu g \cdot g^{-1}$.

Acute Toxicity to Aquatic Organisms

Discussions of the aquatic toxicity of simazine usually take into account the phytotoxic mechanism of this compound through the inhibition of photosynthesis. Because of this mode of action, much of the published material on the toxicity of simazine deals with its effects on aquatic macrophytes and algae. The following discussions of simazine toxicity are directed primarily towards non-target organisms; for additional information on the efficacy of simazine as an aquatic herbicide, the reader is directed to Mauck (1974).

Appendix D provides summaries of a number of acute toxicity tests with simazine and a wide variety of aquatic organisms.

Fish and Amphibians

Simazine has a low toxicity to fish (Weed Science Society of America, 1983) (Appendix D). The U.S. Department of Agriculture (1984) concluded that the compound should not affect fish at concentrations below its water solubility. Published median lethal concentrations vary widely depending on the species, water chemistry, and herbicide formulation. Alabaster (1969) reported a 24-h median lethal concentration (TL_m) for rainbow trout (*Salmo gairdneri*) of 95 $mg \cdot L^{-1}$ for a wettable powder formulation. The 48-h TL_m was 85 $mg \cdot L^{-1}$. Hashimoto and Nishiuchi (1981) published 48-h TL_m values for technical simazine for carp (*Cyprinus carpio*), (>40 $mg \cdot L^{-1}$), goldfish (*Carassius auratus*) (>40 $mg \cdot L^{-1}$), and the medaka (*Oryzias latipes*) (>10 $mg \cdot L^{-1}$). The 48-h TL_m using formulated simazine (formulation not reported) for the pond loach (*Misgurnus anguillicaudatus*) was also above 40 $mg \cdot L^{-1}$. Dodson and Mayfield (1979) observed no mortality of *S. gairdneri* in a solution of Princep 80W®, a wettable powder formulation containing 80% simazine, at 200 mg $ai \cdot L^{-1}$. No mortality in coho salmon (*Oncorhynchus kisutch*) smolts was reported at 2.5 $mg \cdot L^{-1}$ (Bouck and Johnson, 1979), but Snow (1963) reported pumpkinseed (*Lepomis gibbosus*) mortality at 2.0 $mg \cdot L^{-1}$ simazine. Simazine at 120 $mg \cdot L^{-1}$ caused a 70% mortality in 4 h in the same species according to a study cited by Rao and Dad (1979). Wellborn (1969) reported that simazine was toxic to striped bass (*Roccus saxatilis*), with a 96-h LC_{50} of 0.25 $mg \cdot L^{-1}$. A concentration of 1.5 $mg \cdot L^{-1}$ in pond water reduced *L. macrochirus* biomass (U.S. Department of Agriculture, 1984).

Results of fish toxicity investigations may also be influenced by the experimental technique. For example, rapid depletion of simazine in static aquarium water was found by Prowse (1960). Simazine at 120 $mg \cdot L^{-1}$ killed 70% of *mouthbrooder (Tilapia* sp.) fingerlings in 4 h, but after 12 h the water in the tanks was no longer toxic. In another study, Dodson and Mayfield (1979) examined the effects of simazine on the rheotropic response of year-old *S. gairdneri* held in circular tanks with a striped background, which could be rotated to simulate a current of 20 $cm \cdot s^{-1}$. No change in behaviour occurred when the fish were exposed to up to 12.5 $mg \cdot L^{-1}$ of simazine for 24 h. The addition of Tween 80®, a wetting agent, to the simazine, however, resulted in decreased swimming speed and a greater frequency of no response to the simulated current.

Although simazine is generally not considered to be acutely toxic to fish, an excessive kill of vegetation can lead to dissolved oxygen depletion and stress and mortality. Two reports of trout mortality following simazine treatments were investigated by Norton and Ellis (1977). Toxicological tests on the fish suggested that the deaths were not the result of direct poisoning by the herbicide, but that oxygen depletion following the death of aquatic plants, or the rapid kill of large numbers of toxin-releasing algae, may have been the cause.

While conducting 48-h LC_{50} tests, Fitzmayer *et al.* (1982b) observed that both 3-d-old and 7-d-old striped bass (*Morone saxatilis*) larvae became inactive at simazine concentrations of 10–100 $mg \cdot L^{-1}$. The 48-h LC_{50} for 3-d-old fish was 17 $mg \cdot L^{-1}$, and for the 7-d-old fish, above 100 $mg \cdot L^{-1}$. About 60% of the exposed 7-d-old larvae eventually developed a scoliotic curvature of the vertebral column. Inferences from this study are difficult to make, however, because of the high mortalities reported in the controls, which in some cases approached 30%. In these experiments, there was no significant difference between tests run at water hardness levels of 120 $mg \cdot L^{-1}$ (as $CaCO_3$) and 220 $mg \cdot L^{-1}$.

The lowest reported 96-h LC_{50} for a fish species exposed to simazine, 0.25 $mg \cdot L^{-1}$, was reported for *R. saxatilis* by Wellborn (1969) (Appendix D). This result, however, has not been confirmed by other researchers who have examined this species and found LC_{50} values an order of magnitude higher (Cook and Smith, 1976; McCann, 1980; Fitzmayer *et al.*, 1982b). McCann (1980) postulated that the large differences in reported LC_{50} estimates may be the result of additives in some pesticide formulations or differences in the handling techniques used for these sensitive fish. This hypothesis is supported by the work of Dodson and Mayfield (1979), mentioned above.

Marchini *et al.* (1988) reported data concerning the toxicity of simazine to various unnamed fish species: for nine different fish species, the 48-h EC_{50}s ranged from 5.2 to 350 $mg \cdot L^{-1}$; for eight species of fish, the 96-h EC_{50}s ranged from 2.8 to 100 $mg \cdot L^{-1}$. A single amphibian toxicity value was found in the literature; Hashimoto and Nishiuchi (1981) reported a 48-h TL_m of greater than 100 $mg \cdot L^{-1}$ formulated simazine for *Bufo bufo japonicus* tadpole.

Invertebrates

Snow (1963) reported that production measurements of ponds treated with 0.5, 1.0, and 2.0 $mg \cdot L^{-1}$ simazine revealed that the herbicide was not toxic to zooplankton and other animal life constituting the diet of fish being cultured in the ponds. Sanders (1970) found that *Daphnia magna* and seed shrimp (*Cypridopsis vidua*) were immobilized after 48 h of exposure to simazine concentrations of 1.0 and 3.2 $mg \cdot L^{-1}$, respectively. However, scud (*Gammarus fasciatus*), sowbugs (*Asellus brevicaudus*), glass shrimp (*Palaemonetes kadiakensis*), and crayfish (*Orconectes nais*) were not affected by a single exposure to 100 $mg \cdot L^{-1}$ of simazine added to aquaria or beakers. For *D. magna* and *C. vidua*, immobilization was used as the measured response; for all other invertebrates, observations on survival were made at 24-, 48-, and 96-h intervals. Gilderhus (1969) reported that bottom faunal communities in control ponds and ponds treated with 1.0 $mg \cdot L^{-1}$ simazine were nearly identical, suggesting that the treatment had no effect on benthic organisms. Laboratory tests on bottom organisms gave an acute LD_{50} of 28 $mg \cdot L^{-1}$ (Walker, 1964).

Marchini *et al.* (1988) tested the acute toxicity of simazine to *D. magna* using 24-h and 48-h immobilization tests. The daphnids were less than 24 h old. The 24- and 48-h EC_{50}s were greater than 3.5 $mg \cdot L^{-1}$.

Kosanke *et al.* (1988) examined the effect of simazine on the ontogenesis of freshwater snail (*Lymnaea stagnalis*) embryos. Egg masses containing 50–100 eggs were removed from the aquaria and kept in vials containing the herbicide (static test). Live and dead embryos and hatched snails were counted every day

for 20 d. Simazine at 2.02 and 0.202 mg•L^{-1} killed all snail embryos (1477 eggs in total) during the first 9 d of the experiment. Only 4.5% of embryos died in the control batches of eggs. Even with 0.0202 mg•L^{-1} simazine, all (762) eggs were killed, but with a lag in mortality that was indicative of a toxicologically weaker action. In other tests with molluscs, Hashimoto and Nishiuchi (1981) published a 48-h TL_m value of greater than 100 mg•L^{-1} for the snails *Indoplanorbis exustus, Semisulcospira libertina*, and *Physa acuta*.

The U.S. Department of Agriculture's (1984) review of simazine lists LC_{50}s of 1.9 (96 h) to 50 (48 h) mg•L^{-1} for stonefly larvae (*Pteronarcys* sp.) (Hashimoto and Nishiuchi [1981] published a 48-h TL_m of >40 mg•L^{-1} formulated simazine for the mayfly larvae *Cloeon dipterum*) and a 24-h LC_{50} of 1.0 mg•L^{-1} for the freshwater copepod *Heliodiaptomus viduus*. Walker (1962, 1964) reported a population reduction of 50% or more in aquatic worms, leeches, and snails after simazine applications of 0.5–10 mg•L^{-1}. A 96-h LC_{50} of 28 mg•L^{-1} was reported for aquatic worms (species not given).

While conducting 48-h LC_{50} tests with *Daphnia pulex*, Fitzmayer *et al.* (1982b) noted that the daphnids became sedentary at simazine concentrations of 1–50 mg•L^{-1}. For both of the freshwater cladocerans *D. pulex* and *Moina macrocopa*, Hashimoto and Nishiuchi (1981) published 3-h TL_ms of greater than 40 mg•L^{-1} technical simazine.

Algae and Macrophytes

In 1966 and 1967, 17 farm ponds in Ontario were treated with simazine at concentrations of 0.5, 1.0, and 2.0 mg•L^{-1} (concentrations added to the water) to control submerged macrophytes and algae (Wile, 1967). At 1 mg•L^{-1}, simazine was effective in controlling several species of submerged vascular plants and filamentous algae in ponds having little or no water exchange. *Chara* sp. (a filamentous alga) was controlled at 2 mg•L^{-1} in ponds with some water exchange. Filamentous algae were controlled at 0.5 mg•L^{-1} in a pond with little water exchange. The degree of water exchange in the ponds also affected persistence and hence the overall effectiveness of the applications.

The effects of simazine on the photosynthetic pigments of green algae were investigated by Paromenskaya and Lyalin (1968). Three species of algae (*Chlorella vulgaris, Ankistrodesmus braunii*, and *Chlorosarcina* sp., the latter resistant to simazine) were grown in 50 mg•L^{-1} simazine for 17 d. On days 7 and 17, there was practically no growth of *C. vulgaris* and *A. braunii*. Pronounced changes in photosynthetic pigments had occurred by the 17th day of incubation, when 27%–86% of the amount of pigment found in the controls was measured in the sensitive species.

In laboratory experiments using unfiltered, nitrate-enriched river water from an agricultural watershed in central Michigan, Howell and Ries (1972) found that simazine at very low levels (10^{-8} M) decreased the dry weight of inorganic material in algal culture flasks as well as the chlorophyll-*a* content.

Turbak *et al.* (1986) tested the toxicity of simazine to the unicellular green alga *Selenastrum capricornutum* using the U.S. Environmental Protection Agency's (EPA) 21-d bottle test. They found that water chemistry may play an important role in the determination of toxicity thresholds for algae. A simazine concentration of 0.614 µg•L^{-1} decreased algal biomass to 50% of the control value when the alga was grown on an assay medium. However, when the cells were grown in a nutrient-enriched stream water sample, simazine concentrations from 0.01 to 10 µg•L^{-1} did not produce an equivalent inhibition.

The effects of simazine on the photosynthetic organelles of the blue-green alga *Anacystis nidulans* were studied by Mehta and Hawxby (1979). A 10-d flask culture treated with simazine at a concentration of 2.017 mg•L^{-1} was incubated at 25°C–28°C for up to 10 d. Aliquots of the culture were removed for electron microscopy. The thylakoids (the photosynthetic lamellae) developed granularity after 12 h of treatment. The polyhedral bodies, which are vital for cell activities, disintegrated. Growth was completely halted eventually, and death of the cells was indicated by empty and distorted thylakoids and depletion of RNA.

In a similar study, Markova *et al.* (1985) exposed *Salmonella typhimurium* in a broth culture to 50 mg•L^{-1} simazine and examined the exposed cells for ultrastructural changes. They found changes in the cell wall and cytoplasmic membrane, although no destruction or lysis was observed and cell integrity was preserved. The cytoplasm lost its regular, finely grained structure as a result of a collapse of ribosomes.

The influence of simazine on vascular plant photosynthesis, as measured by the inhibition of oxygen evolution, was investigated by Sutton *et al.* (1968). Simazine concentrations of 0.12, 0.50, and 1.0 mg•L^{-1}

were added to nutrient cultures of duckweed (*Lemna minor*), *Elodea canadensis*, and parrotfeather (*Myriophyllum brasiliense*). The minimum dissolved oxygen concentrations occurred after exposure to the highest simazine concentration, and were approximately 50% of control for *L. minor*, 60% of control for *E. canadensis*, and 10% for the submersed form of *M. brasiliense*.

Tucker *et al.* (1983) treated ponds having heavy growths of *Chara vulgaris* with 1.3 mg•L^{-1} simazine. In addition to killing the *Chara*, the simazine also completely eliminated the abundant blue-green algae communities in these ponds: blue-green algae species were not found in samples from these ponds during the remainder of the study (up to 52 d following treatment).

The impact of simazine on periphyton communities in *in situ* 300-L enclosures of marsh water in Manitoba was investigated by Goldsborough and Robinson (1983). Colonization and growth on acrylic substrata by periphyton were monitored by measuring the carbon assimilation rate and chlorophyll-*a* accumulation. At 0.1 mg•L^{-1}, no change in either parameter was observed relative to untreated enclosures. At 1.0 and 5.0 mg•L^{-1}, increasing inhibition (to approximately 95%) was observed. Recovery of the communities began within 1 week after treatment. Periphyton productivity was correlated with water chemistry, light availability, date, and herbicide treatment, suggesting that the herbicidal effects are not the result of the herbicide alone but the result of a complex interaction of several parameters.

Using these same enclosures and simazine concentrations, Goldsborough and Robinson (1986) also observed the community structure of the algal communities colonizing the acrylic rods. Herbicide concentrations in water were near the added level during the first 2 weeks of sampling, with the exception of the 5.0 mg•L^{-1} treatment, in which the herbicide concentration did not exceed 3.06 mg•L^{-1} (probably determined by the maximum water solubility limit of the compound). When the enclosures were naturally flooded and refilled with fresh marsh water, simazine was not detectable (detection limit 0.01 mg•L^{-1}) in the enclosures treated with the lower simazine concentrations but was recorded at a maximum concentration of 0.48 µg•L^{-1} in the 5.0 mg•L^{-1} treatment enclosure. The mean periphyton "biovolume" (mean cell volume multiplied by cell density for each taxon) over the 6-week duration of the experiment was not significantly different from control for the 0.1 mg•L^{-1} treatment. With the 1.0 and 5.0 mg•L^{-1} treatments, biovolume was inhibited in the preflood period by 94% and 98%, respectively. The authors stated that this would suggest a community biovolume LC_{50} of between 0.1 and 1.0 mg•L^{-1} for simazine. Simazine treatment also appeared to alter community structure. The dominant filamentous green alga *Stigeoclonium* sp. occurred only rarely in the 1.0 and 5.0 mg•L^{-1} enclosures, whereas diatoms assumed overwhelming dominance.

Simazine concentrations above 0.4 mg•L^{-1} delayed algal blooms in laboratory flasks for at least 2 months (Bryfogle and McDiffett, 1979). At 0.15 mg•L^{-1}, however, the major effect of the herbicide was overcome by the second day of the experiment. In this experiment, there were changes in community structure with the addition of herbicide above 0.05 mg•L^{-1}; these changes included a reduction in diversity and a change in the dominant species.

Sublethal Reactions and Chronic Toxicity in Aquatic Organisms

Fish

Mayer and Sanders (1977) investigated the effects of continuous simazine exposure on growth, reproduction, and survival of *P. promelas* using a flow-through dilution apparatus. *Pimephales promelas* egg hatch and fry growth were reduced (amount of reduction not reported) during continuous exposure to 1.7 mg•L^{-1}.

Reduced growth of channel catfish (*Ictalurus punctatus*) and *L. macrochirus* in simazine-treated ponds was reported by Tucker and Boyd (1978a, 1978b). They found that a simazine application of 13.4 kg ai•ha^{-1} to the bottom of catfish ponds (0.04–0.06 ha) before flooding resulted in an extended period of low dissolved oxygen concentrations, a 19% reduction in *I. punctatus* yield, and reduced feed conversion by fish when compared with control ponds. The authors stated that the cause of decreased catfish growth may be partly due to exposure to prolonged periods of lowered dissolved oxygen concentration caused by the simazine application.

Invertebrates

Fitzmayer *et al.* (1982a) evaluated the effect of simazine on *D. pulex* moulting and growth. At 4 mg•L^{-1}, 65% of the daphnids were dead by day 25.

With 20 mg•L^{-1}, all daphnids were dead by day 15. Reproductive maturity was delayed by about one moult cycle at 4 mg•L^{-1} simazine, which amounted to about 3 d at 20°C. The number of broods produced at 4 mg•L^{-1} (56) was significantly less than the number produced by controls (104).

No-observed-effect levels (NOELs) of 4 and 1000 mg•L^{-1} for *Daphnia* and the mud crab, respectively, were reported by the U.S. Department of Agriculture (1984). Concentrations of 0.01–1.5 mg•L^{-1} (ai) of the simazine formulation Tafazine® caused a very slight decline in the rate of asexual budding in an Indian colonial freshwater bryozoan (*Plumatella casmiana*); the percent germination of statoblasts in the control samples was 80% and 70% with the two simazine concentrations (Rao and Dad, 1979).

Mayer and Sanders (1977) also investigated the effect of continuous simazine exposure on *Daphnia* reproduction and midge emergence using their flow-through dilution apparatus. Simazine concentrations of 0.25 to 3.0 mg•L^{-1} had no adverse effect on *Daphnia* reproduction. At 0.66 and 2.2 mg•L^{-1} exposures, midge emergence was temporarily delayed.

Whitley (1966) found that an 80% wettable powder of simazine applied at 1.0 mg•L^{-1} did not adversely affect the zooplankton within a pond; although zooplankton populations declined slightly, the decline was attributed to a reduction in the phytoplankton crop on which they grazed. Jenkins and Buikema (1990) studied the effects of simazine on a variety of plankton species in 4-L microcosms suspended for 21 d 25 cm below the surface of a lake in Virginia. The experiment was conducted in December, as tests in warmer-weather months revealed that periphyton growth on the outside of the microcosms interfered with light penetration. Few of the species were affected by simazine concentrations of 0.1–1.0 mg•L^{-1}. Groups tested included phytoplankton, bacteria, and zooplankton. The only species showing a significant inhibition ($0.01 < p < 0.05$) of mean cell densities at 0.5 mg•L^{-1} after 21 d was the phytoplankton *Glenodinium*. The only other negative response occurred with the phytoplankton *Trachelomonas* sp., which showed a significant inhibition ($p = 0.01$) at 1.0 mg•L^{-1} after 21 d. Diatom species showed a significant increase in mean cell densities after 21 d at 1.0 mg•L^{-1}. Bacteria and zooplankton (copepods, ciliates, and rotifers were enumerated) cell densities were not affected by the simazine treatment. The authors concluded that levels of simazine as high as 1.0 mg•L^{-1} may not have a deleterious impact on winter zooplankton communities; although phytoplankton may be adversely affected, the lack of dependence of winter zooplankton communities on autotrophic organisms may prevent adverse effects on the winter food web.

Algal inhibition as a result of simazine exposure was reported to be dependent on light intensity (O'Neal and Lembi, 1983). Chlorophyll concentrations in cultures of filamentous algal species (*Pithophora oedogonia, Cladophora glomerata,* and *Spirogyra jurgensii*) exposed to 1.01 mg•L^{-1} of simazine were consistently less than the control but continued to increase over a 45-d period at a light intensity of 100 µE•m^{-2}•s^{-1}. Chlorophyll content decreased markedly when light intensity was 400 µE•m^{-2}•s^{-1}. *Spirogyra* sp. was the most sensitive species tested, with a 50% inhibition of photosynthesis occurring at a simazine concentration of 0.2 mg•L^{-1}.

Summary of Existing Guidelines

The Environmental Studies Board (1973) of the U.S. EPA proposed a water quality guideline for simazine of 10.0 µg•L^{-1} for the protection of freshwater aquatic life (Environment Canada, 1979). The provinces of Ontario (in 1978) and Manitoba (in 1979) also proposed maximum concentration limits for simazine of 10.0 µg•L^{-1} for the protection of aquatic life and wildlife (CCREM, 1985). A survey of the existing water quality guidelines in place in Canada in 1985 indicated that both Saskatchewan and Alberta had recommended "multi-purpose water quality objectives" to ensure that the pesticide concentration in receiving waters did not exceed 1% of the lowest 48-h LC_{50} for the most sensitive species (CCREM, 1985). The province of Quebec has used the value of 10 µg•L^{-1}, published by the U.S. EPA, for the protection of aquatic life (Ministère de l'environnement du Québec, 1989, unpublished draft document).

Guideline

From individual species tests, the most simazine-sensitive North American species appears to be the unicellular green alga *S. capricornutum* (Turbak *et al.*, 1986). When the green alga was tested using an artificial assay medium, a 21-d EC_{50} of 0.614 µg•L^{-1} was reported. This was approximately two orders of magnitude lower than concentrations affecting other aquatic plants (Appendix D). The low value was not

surprising, as the bioassay used was developed for its sensitivity to herbicide contamination of water, and the formulation of simazine used (Princep® 4G) is registered for algae control (Agriculture Canada, 1989). Moreover, the sublethal effect level was determined using a long-term assay and artificial growth medium; when a nutrient-enriched water sample was used, a 50% inhibition of growth did not occur with 0.01–10 $\mu g \cdot L^{-1}$ simazine.

Pond treatment studies have shown that much higher concentrations of simazine do not result in adverse impacts on non-target organisms. Goldsborough and Robinson (1986), for instance, found a periphyton community LC_{50} above 100 $\mu g \cdot L^{-1}$ in Manitoba ponds. They also found that recovery of the colonies commenced 1 week after treatment with 1.0 and 5.0 $mg \cdot L^{-1}$. Similarly, Bryfogle and McDiffett (1979) found that at 150 $\mu g \cdot L^{-1}$, the effects of the herbicide on algal growth were overcome by the second day of the experiment. Jenkins and Buikema (1990) found that few plankton species were affected by 1.0 $mg \cdot L^{-1}$ even in static microcosms; they concluded that levels of simazine as high as 1.0 $mg \cdot L^{-1}$ may not have a deleterious impact on a winter plankton food web.

The above information indicates that aquatic phytoplankton are the most sensitive organisms to the toxic effects of simazine. Simazine would therefore exert its most deleterious effects on this component of the aquatic food web. After extensive studies of *in situ* enclosures, Goldsborough and Robinson (1986) arrived at a minimum periphyton community LC_{50} of 100 $\mu g \cdot L^{-1}$. Their data indicated rapid recovery of the organisms within the enclosures even after treatment with 5.0 $mg \cdot L^{-1}$ and, because simazine was detected at only 0.48 $\mu g \cdot L^{-1}$ after one flooding of the 5.0 $mg \cdot L^{-1}$ treatment enclosure, a short persistence of the compound in the marsh they studied. Therefore, even though simazine may exert adverse effects on the organisms that form the basis of the aquatic food web, these effects are transient and do not translate to adverse effects on the organisms that depend on the plankton community for food. For a freshwater aquatic life water quality guideline, the minimum community LC_{50} value of 100 $\mu g \cdot L^{-1}$ is lowered by a safety factor of one order of magnitude (to account for possible longer-term effects of simazine) (CCREM, 1987), resulting in a guideline of 10 $\mu g \cdot L^{-1}$. Concentrations of simazine found in Canadian surface waters are below this level.

Agricultural Uses

Livestock Watering

Acute Toxicity to Livestock and Related Biota

Data on the acute and chronic toxicity of simazine to mammals and birds are summarized in Table 9. Available data indicate that this compound exhibits low toxicity via oral, dermal, and inhalation routes of exposure. Gaines and Linder (1986) fed simazine to rats older than 90 d and to weanlings 4–6 weeks old and taped granular simazine to their skin. The oral LD_{50}s were 972 and 23biom67 $mg \cdot kg^{-1}$ for the adults and weanlings, respectively. The dermal LD_{50} for the adults was above 2500 $mg \cdot kg^{-1}$. The acute oral toxicity (LD_{50}) as a result of a single oral dose of simazine, deter-mined for rats, mice, and rabbits, was above 5000 $mg \cdot kg^{-1}$. For chickens and pigeons, no mortality was observed at this concentration (U.S. Department of Agriculture, 1984).

Table 9. Acute and Chronic Toxicity of Simazine to Mammalian and Avian Species

Species	Route	Toxicity parameter
	ACUTE	
Rat	Oral	LD_{50} = >5000 $mg \cdot kg^{-1}$ b.w.[1]
Rat	Dermal	LD_{50} = >3100 $mg \cdot kg^{-1}$ b.w.[1]
Mouse	Oral	LD_{50} = >5000 $mg \cdot kg^{-1}$ b.w.[2]
Rat, mouse	Inhalation	No mortalities, 4-h exposure to >2.0 $mg \cdot L^{-1}$[4]
Vole	Oral	LD_{50} = >2000 $mg \cdot kg^{-1}$ b.w.[3]
Rabbit	Oral	LD_{50} = >8000 $mg \cdot kg^{-1}$ b.w.[2]
Sheep	Oral	NOEL (5 weeks) = 25 $mg \cdot kg^{-1}$ b.w.$\cdot d^{-1}$[3]
Quail	Oral	LC_{50} = >3270 $mg \cdot kg^{-1}$ b.w.[3]
Bobwhite, ring-necked pheasant, mallard	Oral	LC_{50} = >5000 $mg \cdot kg^{-1}$ b.w.[3]
	CHRONIC	
Rat	Oral	NOEL (2 years) = 100 $mg \cdot kg^{-1}$ diet (7 $mg \cdot kg^{-1}$ b.w.$\cdot d^{-1}$)[1]
Dog	Oral	NOEL (2 years) = 150 $mg \cdot kg^{-1}$ diet (5 $mg \cdot kg^{-1}$ b.w.$\cdot d^{-1}$)[1]

[1] Worthing and Walker, 1987.
[2] U.S. EPA, 1987.
[3] U.S. Department of Agriculture, 1984.
[4] Weed Science Society of America, 1983.

There is little information on the acute effects of simazine on wildlife species. The U.S. EPA (1988) stated that simazine is "not very toxic" to birds. For prairie voles and grey-tailed voles, the reported LD_{50} from a single oral dose was between 2010 and 3980 $mg \cdot kg^{-1}$ (U.S. Department of Agriculture, 1984). Five-day feeding tests with bobwhite quail (*Colinus virginianus*), ring-necked pheasant (*Phasianus colchicus*), and mallards (*Anas platyhynchos*) revealed LC_{50}s above 5000 $mg \cdot kg^{-1}$ for simazine and above 10 000 $mg \cdot kg^{-1}$ for simazine 80W. For Japanese quail (*Coturnix coturnix japonica*), the LC_{50} was above 3720 $mg \cdot kg^{-1}$. No mortality was observed in the birds at these concentrations (Hill and Camardese, 1986). For rabbits, a single dermal application produced an LD_{50} above 10 000 $mg \cdot kg^{-1}$; repeated applications for 21 d produced an LD_{50} of 2000 $mg \cdot kg^{-1}$. Simazine also exhibits low inhalation toxicity: rats exposed for 1 h to 1.8–4.9 $\mu g \cdot g^{-1}$ simazine absorbed to dust were not affected (U.S. Department of Agriculture, 1984).

Ruminants appear to be more susceptible to simazine poisoning than are laboratory animals. A single oral dose of 500 $mg \cdot kg^{-1}$ b.w. was lethal to sheep (Hapke, 1968). Palmer and Radeleff (1972) later showed that repeated but smaller doses of simazine were also fatal to sheep: 50 $mg \cdot kg^{-1}$ was fatal after 31 doses, 100 $mg \cdot kg^{-1}$ was fatal after 14 doses, and 400 $mg \cdot kg^{-1}$ was fatal after nine doses. A short-term NOEL for sheep was 25 $mg \cdot kg^{-1} \cdot d^{-1}$ for 10 d (U.S. Department of Agriculture, 1984). Chickens receiving 50 $mg \cdot kg^{-1} \cdot d^{-1}$ in the diet over 10 d lost weight, but there was no effect on reproduction with dietary levels of 2.0 and 20 $mg \cdot kg^{-1} \cdot d^{-1}$ (U.S. Department of Agriculture, 1984). A feed concentration of 20–50 $mg \cdot kg^{-1} \cdot d^{-1}$ for 6–10 d caused a 5%– 21% weight loss in cattle, whereas a dose of 100 $mg \cdot kg^{-1} \cdot d^{-1}$ for 7 d caused noticeable morbidity. The short-term NOEL for a 10-d feeding study in cattle was 10 $mg \cdot kg^{-1} \cdot d^{-1}$ (U.S. Department of Agriculture, 1984).

Egyed and Shlosberg (1977) documented two cases of poisoning causing the death of 30 sheep and 2 horses. In both cases, the animals were grazing on weeds during or soon after application of simazine. Simazine was detected in the rumen contents and liver of several sheep and in the stomach contents of the horses. The simazine concentration in the sprayed weeds was not measured, but a dose of 18.7 $mg \cdot kg^{-1}$ sheep body weight was calculated based on average feed intake and rate of herbicide application. This dose is much lower than toxic doses reported in the literature, which was attributed to increased susceptibility of the ewes during lactation, possible breed differences, or underestimation of the actual dose.

Chronic Toxicity

The National Academy of Sciences (NAS, 1977) concluded that simazine appears to have low chronic toxicity to birds and mammals. The WHO (1988) reported chronic toxicity data from a 2-year feeding study with dogs, which produced a NOEL of 5 $mg \cdot kg^{-1}$ b.w.$\cdot d^{-1}$. The U.S. EPA (1984) reported that a 21-d dermal toxicity NOEL was above 1000 $mg \cdot kg^{-1}$ for rabbits. A NOEL in rats after a 2-year feeding study was 100 $mg \cdot kg^{-1}$ in the diet. The lowest-observed-adverse-effect level (LOAEL) reported by the U.S. EPA (1987) was 1.4 $mg \cdot kg^{-1} \cdot d^{-1}$ for a study concerned with the histological changes in the organs of sheep following exposures to simazine for up to 22 weeks.

Fink (1975) found that the reproductive capability of *A. platyrhynchos* was not impaired when the ducks were fed 2–20 $mg \cdot kg^{-1}$ simazine from prior to the onset of egg laying through the normal egg production cycle. Parameters examined included eggs laid, egg-shell cracks and thinning, viable embryos, live 3-week embryos, normal hatchlings, and 14-d survival rate.

Carcinogenicity and Teratogenicity

The U.S. Department of Agriculture (1984) stated that simazine was non-carcinogenic in mice fed 603 $mg \cdot kg^{-1}$ in the diet for 18 months. Garrett *et al.* (1986) noted some genotoxic activity of simazine in a screening survey for the genetic activity of pesticides. Anderson *et al.* (1972) found that simazine was not mutagenic to histidine-requiring mutants of *S. typhimurium*, nor was there any evidence of point mutations. Emnova *et al.* (1987) found that simazine had no mutagenic properties in *Saccharomyces cerevisiae* yeast strains. Shirasu *et al.* (1976) screened pesticides for their mutagenic potential without metabolic activation. Simazine was not mutagenic in a sensitivity test (recombination assay) using strains of *Bacillus subtilis*.

Others have reported that simazine is weakly mutagenic. Sharma and Panneerselvam (1987) reported that simazine inhibited germination and reduced the mitotic index in barley progenies following a single 6-h pulse exposure of the seeds (concentration not given) at the time of peak DNA synthetic activity. It was concluded that simazine is mutagenic and may

be karyotoxic. Simazine was non-mutagenic in a number of microbial mutagenicity systems (employing *S. typhimurium, Escherichia coli, B. subtilis,* and *Serratia marcescens*) but was weakly mutagenic in the fruitfly (*Drosophila melanogaster*). Other mutagenicity and carcinogenicity studies are summarized in U.S. EPA (1987).

The U.S. EPA (1988) stated that data gaps exist for the oncogenic and chronic toxicity of simazine in rodents and dogs and for mutagenicity testing and metabolism studies. The WHO (1988) reported that simazine appears to be devoid of significant mutagenic or genotoxic activity; however, the International Agency for Research on Cancer has not yet evaluated simazine, as the information is apparently inadequate for a full evaluation.

In teratology studies, the U.S. EPA (1988) reported a three-generation reproduction study with rats fed 100 $mg \cdot kg^{-1}$ in the diet (approximately 5 $mg \cdot kg^{-1}$ b.w.$\cdot d^{-1}$) for 93 weeks, which produced a NOEL of greater than 100 $mg \cdot kg^{-1}$; no specific end point besides "reproductive performance" was mentioned.

Metabolism and Depuration

No accumulation of simazine in the tissues of livestock animals has been noted, although the U.S. EPA (1988) indicated the need for long-term studies that include analysis of simazine and its metabolites in meat, milk, poultry, eggs, and other commodities. Tekel *et al.* (1988) reported traces of simazine in commercial milk and butter samples in Czechoslovakia (maximum concentration of 0.01 $mg \cdot kg^{-1}$ in the butter and 0.002 $mg \cdot kg^{-1}$ in the milk). Víden *et al.* (1987) found trace amounts of simazine in milk (approximately 0.002 $mg \cdot kg^{-1}$) in Czechoslovakia. These levels were well below the Canadian negligible residue limit of 0.1 $mg \cdot kg^{-1}$ (Health and Welfare Canada, 1989b).

Guideline

The derivation of a water quality guideline for livestock watering requires valid chronic toxicity and bioaccumulation data for livestock consuming simazine in their dietary water. Except for two reports mentioning that trace amounts of simazine have been found in dairy samples (Víden *et al.* 1987; Tekel *et al.* 1988), no other evidence of simazine residues in livestock products has been found. (As simazine is readily metabolized and excreted by mammals [U.S. Department of Agriculture, 1984], the specific concern related to this compound should be excretion in milk.) No data are available concerning the chronic toxicity of simazine to livestock; the U.S. Department of Agriculture (1984) provided a NOEL for cattle of 10 $mg \cdot kg^{-1} \cdot d^{-1}$, but this was a short-term NOEL from a 10-d study.

In the absence of the required information, the derivation of a guideline for livestock watering requires use of the raw drinking water guideline. This procedure provides a margin of safety for livestock and prevents the accumulation of unacceptable residues in animal products (CCREM, 1987). As an IMAC of 10 $\mu g \cdot L^{-1}$ for simazine in raw water for drinking water supply has been proposed (Health and Welfare Canada, 1989a), this value is also proposed as an interim guideline for water used for livestock watering.

Irrigation

Toxicity to Crops

Simazine has been found in irrigation water with maximum concentrations ranging from 0.25 $mg \cdot L^{-1}$ (Anderson *et al.*, 1978) to 0.70 $mg \cdot L^{-1}$ (Smith *et al.*, 1975), the latter in the first ponding water after a ditchbank application of 22.4 $kg \cdot ha^{-1}$. A concentration of 0.15 $mg \cdot L^{-1}$ is known to injure alfalfa and brome grass (Korven, 1975). To protect sensitive crops, therefore, these limited data suggest a simazine concentration in irrigation water below 0.15 $mg \cdot L^{-1}$.

Pringle *et al.* (1978) studied the impact on six crops of simazine residues in irrigation water collected from ditchbanks. The herbicide was applied at concentrations of 0.01 and 0.10 $mg \cdot L^{-1}$ in the irrigation water. These concentrations were what the authors assumed were the maximum amount and 10 times the maximum amount that would likely be expected after ditchbank application (cf. Smith *et al.*, 1975). Crops were harvested 7 and 30 d after treatment; growth and productivity were not measured. No simazine residues were found in corn grain or pinto bean pods, whereas trace amounts were found in pinto bean foliage and cucumbers. Concentrations ranging from 0.6 to 2.9 $\mu g \cdot kg^{-1}$ (detection limits not given) were reported in tomatoes, sugar beets, and corn foliage. The highest residues of simazine were found in alfalfa (6.4 $\mu g \cdot kg^{-1}$ at the 0.10 $mg \cdot L^{-1}$ irrigation dose). These concentrations were well below the Canadian negligible residue limit of 100 $\mu g \cdot kg^{-1}$ (Health and Welfare Canada, 1989b). The authors suggested that simazine

concentrations of up to 0.10 $m \cdot L^{-1}$ in irrigation water would result in little simazine accumulation in a variety of crops.

Wile (1967) collected water from a pond that had been treated 28 d previously with 3.0 $mg \cdot L^{-1}$ of simazine (starting concentration added to the water) and used this water to irrigate tomato and soybean plants. The simazine-contaminated water killed all the tomato plants and damaged the soybeans, but the actual concentrations of simazine in the irrigation water were not measured.

Guideline

The U.S. EPA (1977) recommended that triazine herbicides should have stringent restrictions placed on their presence in irrigation water to protect sensitive crops. This limit was set at 10 $\mu g \cdot L^{-1}$. The Ontario Ministry of the Environment (OMOE, 1984) recommended a limit of 0.5 $\mu g \cdot L^{-1}$ as a general guideline for triazine herbicides in irrigation water to prevent damage to seedling crops, because injury has been shown with seedling crops irrigated with water containing this concentration of triazine herbicides.

Although information is limited, the OMOE (1984) recommendation of a concentration of 0.5 $\mu g \cdot L^{-1}$ simazine in irrigation water would appear adequate for the protection of non-target plants. Therefore, this level is proposed as a Canadian water quality interim guideline for simazine in irrigation water. Levels of simazine in irrigation waters may be elevated after applications for weed control to the banks of irrigation canals. As outlined above, these waters must also be maintained for freshwater aquatic life.

Recreational Water Quality and Aesthetics

Guideline

The U.S. Department of Agriculture (1984) reported that water containing 0.1–5.0 $mg \cdot L^{-1}$ simazine did not differ from control samples in terms of its "sensory qualities." Water with simazine at 50 $mg \cdot L^{-1}$ or greater had drastically altered taste and odour qualities. No other evidence was found in the literature to suggest that the presence of simazine in water would result in any aesthetic impairment at concentrations that would be deleterious for other water uses. In the absence of other information, a recommended limit for simazine in waters used for recreation has not been derived.

Industrial Water Supplies

Guideline

There is no indication that simazine poses or has the potential to pose a threat to the quality of water used for industry when used according to registered use patterns. Although of potential concern if found in water supplies, a water quality guideline for simazine in industrial water supplies has not been recommended.

SUMMARY

After an evaluation of the published information on the triazine herbicide simazine, the following water quality guidelines were derived (Table 10). The background information on simazine in terms of uses and production, occurrence in the aquatic environment, and persistence and degradation was reviewed. The rationale employed for the development of the recommended guidelines was summarized.

Table 10. Recommended Water Quality Guidelines for Simazine

Uses	Guidelines
Raw water for drinking water supply	10 $\mu g \cdot L^{-1}$ (IMAC)*
Freshwater aquatic life	10 $\mu g \cdot L^{-1}$
Agricultural uses	
Livestock watering	10 $\mu g \cdot L^{-1}$ (interim guideline)
Irrigation	0.5 $\mu g \cdot L^{-1}$ (interim guideline)
Recreational water quality and aesthetics	No recommended guideline
Industrial water supplies	No recommended guideline

* Health and Welfare Canada, 1989a.

ACKNOWLEDGMENTS

The authors gratefully acknowledge the assistance and comments of the members of the Canadian Council of Ministers of the Environment (CCME) and the scientific reviewers from Environment Canada (M.C. Taylor of the Water Quality Branch, J. Maguire of the National Water Research Institute, C. Boutin of the Canadian Wildlife Service, and R.J. Sebastien of Environmental Protection), V. Zitko of Fisheries and Oceans Canada, and G. Wood of Health and Welfare Canada.

REFERENCES

AEC (Alberta Environmental Centre). 1989. Data from Oldman, Bow, Red Deer, Athabasca, Peace and North Saskatchewan river basins (1984–1988). Unpublished data provided to Environment Canada.

Agriculture Canada. 1989. Regulatory Information on Pesticide Products. RIP Database (CCINFODISK). Produced by Agriculture Canada and distributed by the Canadian Centre for Occupational Health and Safety (CD-ROM).

Alabaster, J.S. 1969. Survival of fish in 164 herbicides, insecticides, fungicides, wetting agents and miscellaneous substances. Int. Pest Control, 11(2): 29–35.

Albanis, T.A., P.J. Pomonis, and A.T. Sdoukos. 1986. Seasonal fluctuations of organochlorine and triazine pesticides in the aquatic system of Ioannina Basin (Greece). Sci. Total Environ., 58: 243–253.

Allott, D.J. 1969. The persistence of simazine applied annually in the prolonged absence of soil cultivation. Weed Res., 9: 279–287.

Anderson, K.J., E.G. Leighty, and M.T. Takaashi. 1972. Evaluation of herbicides for possible mutagenic properties. J. Agric. Food Chem., 20(3): 649–656.

Anderson, L.W.J., J.C. Pringle, R.W. Raines, and D.A. Sisneros. 1978. Simazine residue levels in irrigation water after ditchbank application for weed control. J. Environ. Qual., 7(4): 574–579.

Anonymous. 1987. Quebec survey affirms high levels of drinking water quality. Eco/Log Week, 15(29): 2.

Bagnati, R., E. Benfenati, E. Davoli, and R. Fanelli. 1988. Screening of 21 pesticides in water by single extraction with C18 silica bonded phase columns and HRGC-MS. Chemosphere, 17(1): 59–65.

Bailey, H.S. 1985. '1983 Toxic Chemical Survey: A survey of nine impoundments in the Saint John River basin. IWD-AR-WQB-85-88, Inland Waters Directorate (Atlantic Region), Environment Canada, Ottawa. 48 pp.

Baker, D.B. 1985. Regional water quality impacts of intensive row-crop agriculture: A Lake Erie basin case study. J. Soil Water Conserv. 40(1): 125–132.

Bouck, G.R., and D.A. Johnson. 1979. Medication inhibits tolerance to seawater in coho salmon smolts. Trans. Am. Fish. Soc., 108: 63–66.

Bryfogle, B.M., and W.F. McDiffett. 1979. Algal succession in laboratory microcosms as effected by an herbicide stress. Am. Midl. Nat., 101(2): 344–354.

Burkhard, N., and J.A. Guth. 1981. Chemical hydrolysis of 2-chloro-4,6-bis-(alkylamino)-1,3,5-triazine herbicides and their breakdown in soil under the influence of adsorption. Pestic. Sci., 12: 45–52.

Burnside, O.C., E.L. Schmidt, and R. Behrens. 1961. Dissipation of simazine from the soil. Weeds, 9: 477–484.

Burnside, O.C., C.R. Fenster, and G.A. Wicks. 1963. Dissipation and leaching of monuron, simazine and atrazine in Nebraska soils. Weeds, 11: 209–213.

Caro, J.H. 1976. Pesticides in agricultural runoff. *In* Control of Water Pollution from Cropland, Vol. II, An Overview, ed. B.A. Stewart. NTIS PB-277 647, Agricultural Research Service, U.S. Department of Agriculture, Washington, D.C., pp. 91–119.

CCREM (Canadian Council of Resource and Environment Ministers). 1985. Inventory of Water Quality Guidelines and Objectives 1984. Prepared by the Canadian Council of Resource and Environment Ministers Task Force on Water Quality Guidelines.

CCREM (Canadian Council of Resource and Environment Ministers). 1987. Canadian Water Quality Guidelines. Prepared by the Task Force on Water Quality Guidelines of the Canadian Council of Resource and Environment Ministers.

Chen, Y.-L., J.-R. Duh, and Y.-S. Wang. 1983. The influence of climate and soil properties on the degradation of simazine in soils in Taiwan. Proc. Natl. Sci. Counc. Repub. China (A), 7(1): 36–41.

Clay, D.V., and C.E. McKone. 1968. The persistence of chlorthiamid, lenacil and simazine in uncropped soil. *In* Proc. 9th Br. Weed Control. Conf., 18–21 November, Brighton, England, pp. 933–938.

Cohen, S.Z., S.M. Creeger, R.F. Cassel, and C.G. Enfield. 1984. Potential pesticide contamination of groundwater from agricultural uses. *In* Treatment and Disposal of Pesticide Wastes, ed. R.F. Krueger and J.N. Seiber. Am. Chem. Soc. Symp. Ser. No. 259, Washington, D.C., pp. 294–325.

Cohen, S.Z., C. Eiden, and M.N. Lorber. 1986. Monitoring ground water for pesticides. *In* Evaluation of Pesticides in Ground Water, ed. W.Y. Garner, R.C. Honeycutt, and H.N. Nigg. Am. Chem. Soc. Symp. Ser. No. 315, Washington, D.C. pp. 170–196.

Comes, R.D., and F.L. Timmons. 1965. Effect of sunlight on the phytotoxicity of some phenylurea and triazine herbicides on a soil surface. Weeds, 13: 81–84.

Cook, J.D., and B.W. Smith. 1976. Sensitivity of striped bass fingerlings to simazine. Paper presented at the National Fish Culture Workshop, 13–15 January, Springfield, Mo. (Cited in McCann, 1980.)

Dabydeen, S., and J.R.C. Leavitt. 1981. Adsorption and effect of simazine and atrazine on *Elodea canadensis*. Bull. Environ. Contam. Toxicol., 26: 381–385.

Davis, D.E., H.H. Funderburk, Jr., and N.G. Sansing. 1959. Absorption, translocation, degradation and volatilization of radioactive simazine. Proc. S. Weed Conf., 12: 172–173 (abstract).

Dawson, J.H., V.F. Burns, and W.J. Clore. 1968. Residual monuron, diuron, and simazine in a vineyard soil. Weed Sci., 16: 63–65.

Day, B.E., L.S. Jordan, and V.A. Jolliffe. 1968. The influence of soil characteristics on the adsorption and phytotoxicity of simazine. Weed Sci., 16: 209–213.

Dodson, J.J., and C.I. Mayfield. 1979. Modification of the rheotropic response of rainbow trout (*Salmo gairdneri*) by sublethal doses of the aquatic herbicides diquat and simazine. Environ. Pollut., 18: 147–157.

Dubach, P. 1970. Introduction to triazine–soil interactions. Residue Rev., 32: 19–28.

Egyed, M.N., and A. Shlosberg. 1977. Some considerations in the evaluation of a herbicide (simazine–aminotriazole) poisoning in sheep and horses. Vet. Hum. Toxicol., 19(2): 83–84.

Emnova, E.E., G.V. Mereniuk, and L.G. Tsurkan. 1987. Genetic activity of sim-triazine herbicides on *Saccharomyces cerevisiae* yeast strains. Tsitol. Genet., 21(2): 127–131.

Environmental Studies Board. 1973. Water Quality Criteria 1972. A report of the Committee on Water Quality Criteria. EPA-R3-73-033, U.S. Environmental Protection Agency, Washington, D.C. 594 pp.

Environment Canada. 1979. Water Quality Sourcebook—A Guide to Water Quality Parameters. Water Quality Branch, Inland Waters Directorate, Ottawa.

Esser, H.O., G. Dupuis, E. Ebert, G.J. Marco, and C. Vogel. 1975. S-triazines. *In* Herbicides. Chemistry, Degradation and Mode of Action, ed. P.C. Kearney and D.D. Kaufman. Vol. 1, 2nd ed., 129–208. New York: Marcel Dekker.

Fink, R.J. 1975. The effect of simazine on the reproductive capability of mallard ducks. Toxicol. Appl. Pharmacol., 33(1): 188–189 (abstract).

Fishel, D.K., and P.L. Lietman. 1986. Occurrence of nitrate and herbicides in ground water in the upper Conestoga River basin, Pennsylvania. U.S. Geological Survey Rep. 85–4202, Harrisburg, Pa. (Cited in Hall *et al.*, 1989.)

Fitzmayer, K.M., J.G. Geiger, and M.J. van den Avyle. 1982a. Effects of chronic exposure to simazine on the cladoceran, *Daphnia pulex*. Arch. Environ. Contam. Toxicol., 11: 603–609.

Fitzmayer, K.M., J.G. Geiger, and M.J. van den Avyle. 1982b. Acute toxicity effects of simazine on *Daphnia pulex* and larval striped bass. Proc. Annu. Conf. Southeast. Assoc. Fish Wildl. Agencies, 1982: 146–156.

Foy, C.L. 1964. Volatility and tracer studies with alkylamino-s-triazines. Weeds, 12: 103–108.

Foy, C.L., and H. Hiranpradit. 1977. Herbicide movement with water and effects of contaminant levels on non-target organisms. Virginia Polytechnic Institute and State University, Blacksburg, Va. 89 pp.

Frank, R. 1981. Pesticides and PCB in the Grand and Saugeen river basins. J. Great Lakes Res., 7(4): 440–454.

Frank, R. 1986. Rural water quality and pesticides. Highl. Agric. Res. Ont., 9(4): 20–25.

Frank, R., and L. Logan. 1988. Pesticide and industrial chemical residues at the mouth of the Grand, Saugeen and Thames rivers, Ontario, Canada, 1981–85. Arch. Environ. Contam. Toxicol., 17: 741–754.

Frank, R., G.J. Sirons, R.L. Thomas, and K. McMillan. 1979. Triazine residues in suspended solids (1974–1976) and water (1977) from the mouths of Canadian streams flowing into the Great Lakes. J. Great Lakes Res., 5(2): 131–138.

Frank, R., H.E. Braun, M. van Hove Holdrinet, G.J. Sirons, and B.D. Ripley. 1982. Agriculture and water quality in the Canadian Great Lakes Basin: V. Pesticide use in 11 agricultural watersheds and presence in stream water, 1975–1977. J. Environ. Qual. 11(3): 497–505.

Frank, R., B.D. Ripley, H.E. Braun, B.S. Clegg, R. Johnston, and T.J. O'Neill. 1987a. Survey of farm wells for pesticide residues, southern Ontario, Canada, 1981–1982, 1984. Arch. Environ. Contam. Toxicol., 16: 1–8.

Frank, R., B.S. Clegg, B.D. Ripley, and H.E. Braun. 1987b. Investigations of pesticide contaminations in rural wells, 1979–1984, Ontario, Canada. Arch. Environ. Contam. Toxicol., 16: 9–22.

Frank, R., H.E. Braun, B.S. Clegg, B.D. Ripley, and R. Johnson. 1990a. Survey of farm wells for pesticides, Ontario, Canada, 1986 and 1987. Bull. Environ. Contam. Toxicol., 44: 410–419.

Frank, R., H.E. Braun, B.D. Ripley, and B.S. Clegg. 1990b. Contamination of rural ponds with pesticide, 1971–85, Ontario, Canada. Bull. Environ. Contam. Toxicol. 44: 401–409.

Fryer, J.D., and K. Kirkland. 1970. Field experiments to investigate long-term effects of repeated applications of MCPA, Tri-allate, simazine and linuron: Report after 6 years. Weed Res., 10: 133–158.

Funderburk, H.H., Jr., and D.E. Davis. 1963. The metabolism of ^{14}C chain- and ring-labeled simazine by corn and the effect of atrazine on plant respiratory systems. Weeds, 11: 101–104.

Gaines, T.B., and R.E. Linder. 1986. Acute toxicity of pesticides in adult and weanling rats. Fundam. Appl. Toxicol., 7: 299–308.

Galassi, S., L. Guzzella, and S. Sora. 1989. Mutagenic potential of drinking waters from surface supplies in northern Italy. Environ. Toxicol. Chem., 8: 109–116.

Garrett, N.E., H.F. Stack, and M.D. Waters. 1986. Evaluation of the genetic activity profiles of 65 pesticides. Mutat. Res., 168(3): 301–325.

Gaynor, J.D., and D.C. MacTavish. 1981. Movement of granular simazine by wind erosion. HortScience, 16(6): 756–757.

Ghassemi, M., L. Fargo, P. Painter, P. Painter, S. Quinlivan, R. Scofield, and A. Takata. 1981. Environmental fates and impacts of major forest use pesticides. TRW Environmental Division for U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances, Washington, D.C., pp. A-268–298.

Gilderhus, P.A. 1969. Some effects of long-term exposure to simazine on goldfish, bluegills and aquatic invertebrates. Unpublished report, U.S. Department of the Interior, Wis. (Cited in Mauck, 1974.)

Gile, J.D., J.C. Collins, and J.W. Gillett. 1980. Fate of selected herbicides in a terrestrial laboratory microcosm. Environ. Sci. Technol., 14(9): 1124–1128.

Glenn, S., and J.S. Angle. 1987. Atrazine and simazine in runoff from conventional and no-till corn watersheds. Agric. Ecosyst. Environ., 18(4): 273–280.

Glotfelty, D.E., A.W. Taylor, A.R. Isensee, J. Jersey, and S. Glenn. 1984. Atrazine and simazine movement to Wye River estuary. J. Environ. Qual., 13(1): 115–121.

Glotfelty, D.E., J.N. Seiber, and L.A. Liljedahl. 1987. Pesticides in fog. Nature (London), 325(6105): 602–605.

Goldsborough, L.G., and G.G.C. Robinson. 1983. The effect of two triazine herbicides on the productivity of freshwater marsh periphyton. Aquat. Toxicol., 4: 95–112.

Goldsborough, L.G., and G.G.C. Robinson. 1986. Changes in periphytic algal community structure as a consequence of short herbicide exposures. Hydrobiologia, 139: 177–192.

Hall, J.K., and N.L. Hartwig. 1990. Triazine herbicide fate in a no-tillage corn (*Zea mays* L.)–crownvetch (*Coronilla varia* L.) "living mulch" system. Agric. Ecosyst. Environ., 30(3–4): 281–293.

Hall, J.K., M.R. Murray, and N.L. Hartwig. 1989. Herbicide leaching and distribution in tilled and untilled soil. J. Environ. Qual., 18: 439–449.

Hance, R.J. 1984. Variations in observations of pesticide behaviour between laboratory and long-term field studies. *In* Proc. les Colloques de l'INRA, No. 31, Behaviour and Side Effects of Pesticides in Soil, 4–8 June, Versailles, pp. 57–65.

Hapke, H.J. 1968. Untersuchungen zur toxikologie des unkrautvernichters simazin. Berl. Muench. Tieraerztl. Wochenschr, 15: 301–303.

Harris, C.I. 1967. Fate of 2-chloro-s-triazine herbicides in soil. J. Agric. Food Chem., 15(1): 157–162.

Hashimoto, Y., and Y. Nishiuchi. 1981. Establishment of bioassay methods for the evaluation of acute toxicity of pesticides to aquatic organisms. J. Pestic. Sci., 6: 257–264. (In Japanese with English summary.)

Hawxby, K.W., and R. Mehta. 1979. The fate of aquazine in a small pond. Proc. Okla. Acad. Sci., 59: 16–19.

Health and Welfare Canada. 1989a. Guidelines for Canadian Drinking Water Quality. 4th ed. Prepared by the Federal–Provincial Subcommittee on Drinking Water of the Federal–Provincial Advisory Committee on Environmental and Occupational Health. Canadian Government Publishing Centre, Ottawa. 25 pp.

Health and Welfare Canada. 1989b. Canadian Food and Drugs Act and Regulations. Maximum Residue Limits for Agricultural

Chemicals. Division 15, Table II, Food and Drug Regulations. p. 534.

Helling, C.S. 1970. Movement of *s*-triazine herbicides in soils. Residue Rev., 32: 175–210.

Hill, E.F., and M.B. Camardese. 1986. Lethal dietary toxicities of environmental contaminants and pesticides to *Coturnix*. Fish Wildl. Tech. Rep. 2, U.S. Fish and Wildlife Service. 147 pp.

Hogue, U.J., S.U. Khan, and A. Gaunce. 1981. Leaching of four orchard herbicides in soil columns. Can. J. Soil Sci., 61: 401–407.

Hormann, W.D., J.C. Tournayre, and H. Egli. 1978. Triazine herbicide residues in central European streams. Pestic. Monit. J., 13(3): 128–131.

Howell, G.S., and S.K. Ries. 1972. The influence of herbicides used on horticultural crops on stream ecology. Report to Michigan State University, East Lansing, Mich. 16 pp.

Ivey, M.J., and H. Andrews. 1965. Leaching of simazine, atrazine, diuron and DCPA in soil columns. Proc. South. Weed Sci. Soc., 18: 670.

Jenkins, D.G., and A.L.J. Buikema. 1990. Response of a winter plankton food web to simazine. Environ. Toxicol. Chem., 9(6): 693–705.

Jensen, K.I.N., and E.R. Kimball. 1982. The comparative behavior of simazine and terbacil in soils. Weed Res., 22: 7–12.

Jordan, L.S., J.D. Mann, and B.E. Day. 1965. Effects of ultraviolet light on herbicides. Weeds, 13: 43–46.

Jordan, L.S., W.J. Farmer, J.R. Goodin, and B.E. Day. 1970. Non-biological detoxication of the s-triazine herbicides. Residue Rev., 32: 267–286.

Jury, W.A., D.D. Focht, and W.J. Farmer. 1987. Evaluation of pesticide groundwater pollution potential from standard indices of soil–chemical adsorption and biodegradation. J. Environ. Qual., 16(4): 422–428.

Kalouskovà, N. 1986. Kinetics and mechanisms of interaction of simazine with humic acids. J. Environ. Sci. Health, B21(3): 251–268.

Kapur, K., and N.K. Yadav. 1982. The effect of some herbicides on the hatching of eggs in common carp, *Cyprinus carpio* var. *communis*. Acta Hydrobiol., 24(1): 87–92.

Kaufman, D.D., and P.C. Kearney. 1970. Microbial degradation of s-triazine herbicides. Residue Rev., 32: 235-265.

Kaufman, D.D., P.C. Kearney, and T.J. Sheets. 1965. Microbial degradation of simazine. J. Agric. Food Chem., 13: 238–242.

Kearney, P.C., T.J. Sheets, and J.W. Smith. 1964. Volatility of seven s-triazines. Weeds, 12: 83–87.

Khan, S.U., and P.B. Marriage. 1979. Residues of simazine and hydroxysimazine in an orchard soil. Weed Sci., 27(2): 238–241.

Knüsli, E. 1970. History of the development of triazine herbicides. Residue Rev., 32: 1–9.

Korven, H.C. 1975. Irrigation ditch maintenance with chemicals and grasses. Can. Agric. Eng., 17: 39–43.

Kosanke, G.J., W.W. Schwippert, and T.W. Beneke. 1988. The impairment of mobility and development in freshwater snails (*Physa fontinalis* and *Lymnaea stagnalis*) caused by herbicides. Comp. Biochem. Physiol., 90C(2): 373–379.

Leblová, S., and J. Rovenská. 1987. Effect of pollutants on alcohol dehydrogenase isolated from peanut (*Arachis hypogaea*). Biologia (Bratislava), 42(4): 347–357.

Lee, H.-B., and Y.D. Stokker. 1986. Analysis of eleven triazines in natural waters. J. Assoc. Off. Anal. Chem., 69(4): 568–572.

Marchini, S., L. Passerini, D. Cesareo, and M.L. Tosato. 1988. Herbicidal triazines: Acute toxicity on *Daphnia*, fish, and plants and analysis of its relationships with structural factors. Ecotoxicol. Environ. Saf., 16: 148–157.

Markova, N., N. Cherepova, and M. Shabanov. 1985. Ultrastructural changes of *S. typhimurium* under treatment with the pesticide simazine. Dokl. Bolg. Akad. Nauk., 38(10): 1367–1370.

Mauck, W.L. 1974. A review of the literature on the use of simazine in fisheries. Fish Control Laboratory, Bureau of Sport Fisheries and Wildlife. La Crosse, Wis. 62 pp.

Mauck, W.L., F.L. Mayer, Jr., and D.D. Holz. 1976. Simazine residue dynamics in small ponds. Bull. Environ. Contam. Toxicol., 16: 1–8.

Mayer, F.L., Jr., and M.R. Ellersieck. 1986. Manual of acute toxicity: Interpretation and data base for 410 chemicals and 66 species of freshwater animals. Resource Publication 160, Fish and Wildlife Service, U.S. Deptartment of the Interior, Washington, D.C. 574 pp.

Mayer, F.L., and H.O. Sanders. 1977. Simazine effects on non-target aquatic organisms: A preliminary report. Proc. South. Weed Sci. Soc., 30: 333 (abstract).

McCall, P.J., D.A. Laskowski, R.L. Swann, and H.J. Dishburger. 1981. Measurement of sorption coefficients of organic chemicals and their use in environmental fate analysis. *In* Test Protocols for Environmental Fate and Movements of Toxicants. Proceedings of a Symposium. Association of Official Analytical Chemists, Washington, D.C., pp. 89–109.

McCann, J.A. 1980. Simazine toxicity to fingerling striped bass. Prog. Fish-Cult., 42(3): 180–181.

McGee, B. 1984. Survey of Pesticide Use in Ontario, 1983. Estimates of pesticides used on field crops, fruits, vegetables and in roadside weed control. Economics Information Report No. 84-05, Economics and Policy Coordination Branch, Ontario Ministry of Agriculture and Food, Toronto. 39 pp.

Mehta, R.S., and K.W. Hawxby. 1979. Effects of simazine on the blue-green alga *Anacystis nidulans*. Bull. Environ. Contam. Toxicol., 23: 319–326.

Miltner, R.J., D.B. Baker, T.F. Speth, and C.A. Frank. 1988. Treatment of seasonal pesticides in surface waters. NTIS/PB88-225008, Drinking Water Research Division. U.S. Environmental Protection Agency, Cincinnati, Ohio. 47 pp.

Moreland, D.E., W.A. Gentner, J.L. Hilton, and K.L. Hill. 1959. Studies on the mechanism of herbicidal action of 2-chloro-4,6 bis(ethylamino)-s-triazine. Plant Physiol., 34(4): 432–435.

Moxley J. 1989. Survey of Pesticide Use in Ontario, 1988. Estimates of pesticides used on field crops, fruits, vegetables and in roadside weed control. Economics Information Report No. 89-08, Economics and Policy Coordination Branch, Ontario Ministry of Agriculture and Food, Toronto. 40 pp.

Muir, D.C.G. 1990. Persistence and transformation in water and sediments. *In* Environmental Chemistry of Herbicides, Vol. 2, ed. R. Grover. Boca Raton, Fla.: CRC Press.

NAQUADAT. 1989. National Water Quality Data Bank. Water Quality Branch, Inland Waters Directorate, Environment Canada, Ottawa.

NAS (National Academy of Sciences). 1977. Drinking Water and Health. Safe Drinking Water Committee, National Research Council, Washington, D.C. 939 pp.

Nearpass, D.C. 1966. Effects of soil acidity on the adsorption, penetration and persistence of simazine. Weeds, 13: 341–346.

Niimi, A.J. 1987. Biological half-lives of chemicals in fishes. Rev. Environ. Contam. Toxicol., 99: 1–46.

Norton, J.A., and J.F. Ellis. 1977. Factors that might affect the sensitivity of rainbow trout to simazine. Proc. South. Weed Sci. Soc., 30: 332 (abstract).

OMOE (Ontario Ministry of the Environment). 1984. Water Management—Goals, Policies, Objectives and Implementation Procedures of the Ministry of the Environment. Toronto. 70 pp.

OMOE (Ontario Ministry of the Environment). 1987a. Pesticides in Ontario Drinking Water—1985. August 1987, Toronto. 31 pp.

OMOE (Ontario Ministry of the Environment). 1987b. Pesticides in Ontario Drinking Water—1986. November 1987, Toronto. 56 pp.

O'Neal, S.W., and C.A. Lembi. 1983. Effect of simazine on photosynthesis and growth of filamentous algae. Weed Sci., 31(6): 899–903.

O'Neill, H.J., and H.S. Bailey. 1987. 1986 New Brunswick Pesticide Survey: A survey of three streams draining agricultural areas. Tech. Rep. IW/L-AR-WQB-87-132. Inland Waters Directorate, Environment Canada, Ottawa. 47 pp.

Ontario Ministry of Agriculture and Food. 1989. 1990 Guide to Chemical Weed Control. RV-11-89-62M, Toronto, Ontario. Queen's Printer for Ontario. 209 pp.

Palmer, J.S., and R.D. Radeleff. 1972. The toxicologic effects of certain fungicides and herbicides on sheep and cattle. Ann. N.Y. Acad. Sci., 1972: 729–736.

Paromenskaya, L.N., and G.N. Lyalin. 1968. Effect of simazine on photosynthetic pigments of green algae. Sov. Plant Physiol., 15(6): 842–846.

Pionke, H.B., D.E. Glotfelty, and J.D. Urban. 1986. Pesticide contamination of ground water in a rural Pennsylvania watershed. *In* Proc. Agricultural Impacts on Ground Water, Omaha, Nebr. Natl. Water Well Assoc., Dublin, Ohio., pp. 542–563 (Cited in Hall *et al.*, 1989.)

Pionke, H.B., D.E. Glotfelty, A.D. Lucas, and J.B. Urban. 1988. Pesticide contamination in the Mohantango Creek watershed. J. Environ. Qual., 17(1): 76–84.

Pringle, J.C. Jr., L.W.J. Anderson, and R.W. Raines. 1978. Residues in crops irrigated with water containing simazine. J. Agric. Food Chem., 26(5): 1143–1147.

Prowse, G.A. 1960. Notes on the toxicity to fish of some common agricultural insecticides and weedicides. Pestic. Abstr. News Summ. A, 5(4): 508–509.

Rao, K.S., and N.K. Dad. 1979. Studies of herbicide toxicity in some freshwater fishes and ectoprocta. J. Fish Biol., 14: 517–522.

Raszyk, J. 1986. Physical, chemical and biological study of dust from large-scale pig farms. Vet. Med. (Praha), 31(4): 233–244. (In Russian.)

Reed, E.H., and H.A. Holt. 1982. Leaching of selected total vegetation control herbicides. Proc. 36th Annu. Meet. Northeast. Weed Sci. Soc., 1982: 39–43.

Reinert, K.H., and J.H. Rodgers. 1987. Fate and persistence of aquatic herbicides. Environ. Contam. Toxicol., 98: 61–98.

Richards, R.P., J.W. Kramer, D.B. Baker, and K.A. Krieger. 1987. Pesticides in rainwater in the northeastern United States. Nature (London), 327(6118): 129–131.

Ripley, B.D., B.S. Clegg, and R. Frank. 1986. Survey of triazine and chloroacetamide herbicides in well water in Ontario, Canada, 1985. Proc. 6th Int. Congr. Pestic. Chem. (IUPAC), 10–15 August, 5F-07 (abstract).

Roadhouse, F.E.B., and L.A. Birk. 1961. Penetration of and persistence in soil of the herbicide 2-chloro-4,6-bis(ethylamino)-s-triazine (simazine). Can. J. Plant Sci., 4: 252–260.

Roberts, G.C., G.J. Sirons, R. Frank, and H.E. Collins. 1979. Triazine residues in a watershed in southwestern Ontario (1973–75). J. Great Lakes Res., 5(3–4): 246–255.

Rodgers, C.A. 1970. Uptake and elimination of simazine by green sunfish (*Lepomis cyanellus* Raf.). Weed Sci., 18: 134–136.

Roller, N.F. 1979. Survey of Pesticide Use in Ontario, 1978. Economics Branch, Ontario Ministry of Agriculture and Food, Toronto. 35 pp.

Sanders, H.O. 1970. Toxicities of some herbicides to six species of freshwater crustaceans. J. Water Pollut. Control Fed., 42(8): 1544–1550.

Schwartz, S.S., D.W. Blinn, and G. Johnson. 1981. The physical-chemical and planktonic response of an algicide-treated shallow mountain lake in Arizona. Int. Rev. Gesamten Hydrobiol., 66: 249–262.

Scott, H.D., and J.F. Lutz. 1971. Release of herbicides from clay minerals as a function of water content: I. Kaolinite. Soil Sci. Soc. Am. J., 35: 374–379.

Selim, S.A., S.W. O'Neal, M.A. Ross, and C.A. Lembi. 1989. Bioassay of photosynthetic inhibitors in water and aqueous soil extracts with Eurasian watermilfoil (*Myriophyllum spicatum*). Weed Sci., 37(6): 810–814.

Sharma, C.B.S.R., and N. Panneerselvam. 1987. Mutagenicity of pesticides in barley progeny test system. Environ. Mutagen., 9 (Suppl. 8): 99 (abstract).

Shirasu, Y., M. Moriya, K. Kato, A. Furuhashi, and T. Kada. 1976. Mutagenicity screening of pesticides in the microbial system. Mutat. Res., 40: 19–30.

Singh, S.P., and N.K. Yadav. 1978. Toxicity of some herbicides to major carp fingerlings. Indian J. Ecol., 5(2): 141–147.

Slack, C.H., R.L. Blevins, and C.E. Rieck. 1978. Effect of soil pH and tillage on persistence of simazine. Weed Sci., 26(2): 145–147.

Smith, A.E., 1982. Herbicides and the soil environment in Canada. Can. J. Soil Sci., 62(3): 433–460.

Smith, A.E. 1985. Persistence of triazine herbicides in Canadian soils. Can. J. Plant Sci., 65: 811–818.

Smith, A.E., and B.J. Hayden. 1976. Field persistence studies with eight herbicides commonly used in Saskatchewan. Can. J. Plant Sci., 56: 769–771.

Smith, A.E., R. Grover, G.S. Esmond, and H.C. Korven. 1975. Persistence and movement of atrazine, bromacil, monuron, and simazine in intermittently-filled irrigation ditches. Can. J. Plant Sci., 55: 809–816.

Smith, A.E., D.C.G. Muir, and R. Grover. 1982. The triazine herbicides. *In* Analysis of Pesticides in Water, Vol. III. CRC Publications, pp. 213–239.

Snow, J.R. 1963. Simazine as an algicide for bass ponds. Prog. Fish-Cult., 25 (1): 34–36.

Statistics Canada. 1987. Imports. Commodity by Country. C.I.T.C. Detail 1986–1987. International Trade Division.

Statistics Canada. 1988. Imports. Commodity by Country. C.I.T.C. Detail 1987–1988. International Trade Division.

Stroube, E.W., and D.D. Bondarenko. 1960. Persistence and distribution of simazine applied in the field. Proc. North Cent. Weed Control. Conf. 17: 40–41.

Suntio, L.R., W.Y. Shiu, D. Mackay, J.N. Seiber, and D. Glotfelty. 1988. Critical review of Henry's law constants for pesticides. Rev. Environ. Contam. Toxicol., 109: 1–59.

Sutton, D.L., D.A. Durham, S.W. Bingham, and C.L. Foy. 1968. Influence of simazine on apparent photosynthesis of aquatic plants and herbicide residue removal from water. Weed Sci., 1968: 56–59.

Talbert, R.E., and O.H. Fletchall. 1964. Inactivation of simazine and atrazine in the field. Weeds, 12: 33–37.

Talbert, R.E., and O.H. Fletchall. 1965. The adsorption of some s-triazines in soils. Weeds, 13: 46–52.

Technical Database Services Inc. 1986. LOG P and Related Parameters Database. Pomona College, Calif., and Technical Database Services Pomona College Medicinal Chemistry Project, Claremont, Calif.

Tekel, J., P. Farkas, K. Schultzová, J. Kovaciova, and A. Szokolay. 1988. Analysis of triazine herbicides residues in butter and pasteurized milk. Z. Lebensm. Unters. Forsch., 186(4): 319–322.

Thomas, R.E. 1967. Simazine treatment of Ravenna Lake for control of aquatic vegetation. Nebraska Game, Forestation and Fish Commission, 1 July, 9 pp. (Cited in Mauck 1974.)

Torstensson, L. 1974. Effects of MCPA, 2,4,5-T, linuron and simazine on some functional groups of soil microorganisms. Swed. J. Agric. Res., 4: 151–160.

Triplett, G.B., Jr., B.J. Conner, and W.M. Edwards. 1978. Transport of atrazine and simazine in runoff from conventional and no-tillage corn. J. Environ. Qual., 7(1): 77–84.

Tucker, C.S., and C.E. Boyd. 1978a. Consequences of periodic applications of copper sulfate and simazine for phytoplankton control in catfish ponds. Trans. Am. Fish. Soc., 10 (107): 316–320.

Tucker, C.S., and C.E. Boyd. 1978b. Effects of simazine treatment on channel catfish and bluegill production in ponds. Aquaculture, 15: 345–352.

Tucker, C.S., and C.E. Boyd. 1981. Relationships between pond sediments and simazine loss from waters of laboratory systems. J. Aquat. Plant Manage., 19: 55–57.

Tucker, C.S., R.L. Busch, and S.W. Lloyd. 1983. Effects of simazine treatment on channel catfish production and water quality in ponds. J. Aquat. Plant Manage., 21: 7–11.

Turbak, C.S., S.B. Olson, and G.A. McFeters. 1986. Comparison of algal assay systems for detecting waterborne herbicides and metals. Water Res., 20(1): 91–96.

Upchurch, R.P., F.L. Selman, D.D. Mason, and E.J. Kamprath. 1966. The correlation of herbicidal activity with soil and climatic factors. Weeds, 14: 42–49.

U.S. Department of Agriculture. 1984. Simazine. *In* Pesticide Background Statements. Agricultural Handbook No. 633., Vol. 1, Herbicides. U.S. Department of Agriculture Forest Service, Washington, D.C., pp. S1–S92.

U.S. EPA (Environmental Protection Agency). 1977. Silviculture and chemicals and protection of water quality. PB 271 923/5, Region X, Seattle, Wash. 224 pp.

U.S. EPA (Environmental Protection Agency). 1984. Guidance for the reregistration of products containing simazine as the active ingredient. PB84-212349. Office of Pesticide Programs, Washington, D.C.

U.S. EPA (Environmental Protection Agency). 1987. Health advisories for 50 pesticides. NTIS/PB88-113543, U.S. Environmental Protection Agency, Washington, D.C. 818 pp.

U.S. EPA (Environmental Protection Agency). 1988. Simazine Fact Sheet No. 23. In U.S. Environmental Protection Agency Pesticide Fact Handbook. Noyes Data Corp., Park Ridge, N.J., pp. 703–707.

Vance, B.D., and D.L. Smith. 1969. Effects of five herbicides on three green algae. Tex. J. Sci., XX(4): 329–335.

Víden, I., Z. Rathouská, J. Davidek, and J. Hajslová. 1987. Use of gas liquid chromatography/mass spectrometry for triazine herbicide residues analysis in forage and milk. Z. Lebensm. Unters. Forsch., 185(2): 98–105.

Walker, A. 1976a. Simulation of herbicide persistence in soil. I. Simazine and prometryne. Pestic. Sci., 7: 41–49.

Walker, A. 1976b. Simulation of herbicide persistence in soil. II. Simazine and linuron in long-term experiments. Pestic. Sci., 7: 50–58.

Walker, A. 1978. Simulation of the persistence of eight soil-applied herbicides. Weed Res., 18: 305–313.

Walker, A., R.J. Hance, J.G. Allen, G.G. Briggs, Y.-L. Chen, J.D. Gaynor, E.J. Hogue, A. Malquori, K. Moody, J.R. Moyer, W. Pestemer, A. Rahman, A.E. Smith, J.C. Streibig, N.T.L. Torstensson, L.S. Widyanto, and R. Zandvoort. 1983. EWRS Herbicide–Soil Working Group: Collaborative experiment on simazine persistence in soil. Weed Res., 23: 373–383.

Walker, C.R. 1962. Toxicological effects of herbicides on the fish environment. *In* Proc. Annu. Air Water Pollut. Conf., 12 November, Columbia, Mo., Vol. 8., pp. 17–34.

Walker, C.R. 1964. Simazine and other s-triazine compounds as aquatic herbicides in fish habitats. Weeds, 12(2): 134–149.

Weed Science Society of America. 1983. Herbicide Handbook. 5th ed. Weed Science Society of America, Champaign, Ill.

Wellborn, T.R. 1969. The toxicity of nine therapeutic and herbicidal compounds to striped bass. Prog. Fish-Cult., 31(1): 27–32.

Whitley, J.R. 1966. Control of undesirable aquatic vegetation. Unpublished report, Fisheries Research and Training, Mo. (Cited in Mauck, 1974.)

WHO (World Health Organization). 1988. Drinking-water Quality Guidelines for Selected Herbicides. NTIS/PB88-204136, World Health Organization, Copenhagen, Denmark. 32 pp.

Wile, I. 1967. Aquatic plant control with diquat, fenac, and simazine in Ontario farm ponds. Publication No. 31, The Ontario Water Resources Commission, Toronto. 19 pp.

Wnuk, M., R. Kelley, G. Breuer, and L. Johnson. 1987. Pesticides in water supplies using surface water sources. NTIS PB88-136916, Iowa Department of Natural Resources, Des Moines, Iowa. 43 pp.

Worthing, C.R., and S.B. Walker (eds.). 1987. The Pesticide Manual, A World Compendium, 8th ed. British Crop Protection Council, Thornton Heath, U.K. 1081 pp.

Zimdahl, R.L., V.H. Freed, M.L. Montgomery, and W.R. Furtick. 1970. The degradation of triazine and uracil herbicides in soil. Weed Res., 10: 18–26.

Zurawski, H., and M. Ploszynski. 1968. Investigations on the disappearance of simazine from light soil. *In* Proc. 9th Br. Weed Control Conf., 18–21 November, Brighton, England, pp. 115–118.

Appendix A.

Concentrations of Simazine in Water, Sediment, and Biota

Table A-1. Concentrations of Simazine in Water, Sediment, and Biota

Medium	Location	Mean concentration ($\mu g \cdot L^{-1}$)*	Range ($\mu g \cdot L^{-1}$)	No. of detections/ No. of samples	Year(s)	Reference
Surface water	Hillman Creek drainage, southwestern Ontario agricultural watershed	—	ND–3.6 (DL = 0.02 $\mu g \cdot L^{-1}$	132/360	1973–75	Roberts *et al.*, 1979
Surface water	11 agricultural watersheds in southern Ontario	0.02	ND–0.3	—†	1975–76	Frank *et al.*, 1982
		0.06	ND–3.4	—†	1976–77	
Surface water	Grand River, Ontario, agricultural watershed	0.0012	ND–0.01	20‡	1975–76	Frank, 1981
		0.0003	ND–0.01	30‡	1976–77	
Surface water	Saugeen River, Ontario, agricultural watershed	ND (DL < 1 $ng \cdot L^{-1}$)	ND	0/14	1975–76	Frank, 1981
		ND (DL < 1 $ng \cdot L^{-1}$)	ND	0/20	1976–77	
Surface water	Mouths of 92 streams in southern Ontario draining into the Great Lakes	0.2	ND–6.0	26/92	July 1977	Frank *et al.*, 1979
Surface water	Nine streams in central Europe	—	<0.4 0.4–1.0 1.0–10 >10	86 25 7 0 (detected in 70% of the samples)	1976–77	Hormann *et al.*, 1978
Surface water	Ioannina basin, Greece, agricultural watershed				Sept. 1984–Sept. 1985	Albanis *et al.*, 1986
	- river stations	—	ND–14.8	13/15		
	- canal stations	—	ND–8.2	8/10		
	- lake stations	—	ND–80.2 (DL = 0.2 $\mu g \cdot L^{-1}$)	27/36		
Surface water	Grand River, Ontario, agricultural watershed	ND	(DL = < 0.02 $\mu g \cdot L^{-1}$)	0/95	1981–85	Frank and Logan, 1988
Surface water	Saugeen River, Ontario, agricultural watershed	0.1	—	1/143	1981–85	Frank and Logan, 1988

ND = not detected
DL = detection limit
*Unless otherwise indicated.
†Number of detections and number of samples not recorded.
‡Number of detections not recorded.

Table A-1. Continued

Medium	Location	Mean concentration ($\mu g \cdot L^{-1}$)*	Range ($\mu g \cdot L^{-1}$)	No. of detections/ No. of samples	Year(s)	Reference
Surface water	Thames River, Ontario, agricultural watershed	0.1	—	8/202	1981–85	Frank and Logan, 1988
Surface water	Po River, Italy	—	<0.1–0.6	Above 0.01 $\mu g \cdot L^{-1}$ in 7 0f 12 samples	1986	Galassi *et al.*, 1989
Surface water	United States	—	ND–1300	877/5067	—	U.S. EPA, 1987
Surface water	Iowa, untreated surface water	—	0.27 (DL = 0.2 $\mu g \cdot L^{-1}$)	1/130	1985–86	Wnuk *et al.*, 1987
Surface water	Iowa, treated surface water	—	ND	0/140	1985–86	Wnuk *et al.*, 1987
Groundwater	Rural farm wells in southern Ontario, mineral soils	—	ND (DL = 0.1 $\mu g \cdot L^{-1}$)	0/91 (only used on 1 of 91 farms)	1984	Frank *et al.*, 1987a
Groundwater	Rural farm wells in southern Ontario suspected of contamination	—	ND–8.8 (DL = 0.1 $\mu g \cdot L^{-1}$)	Detected in 10 of 160 wells suspected of contamination	1987–84	Frank *et al.*, 1987b
Groundwater	291 farm wells in Ontario	—	—	Detected in 6 of 291 samples at a concentration above 1.0 $\mu g \cdot L^{-1}$	1985	Ripley *et al.*, 1986
Groundwater	California	—	0.5–3.5	Detected in 6 of 106 wells	—	Cohen *et al.*, 1984
Groundwater	United States (California, Pennsylvania, Maryland)	—	0.2–3.0	—	—	Cohen *et al.*, 1986
Groundwater	United States	—	ND–800	229/2282	—	U.S. EPA, 1987
Groundwater	Pennsylvania	—	0.01–0.170	Detected in 14 of 38 wells and piezometer samples	1985–86	Pionke *et al.*, 1988
Groundwater	Province of Bergamo, northern Italy	—	ND–0.2	—	—	Bagnati *et al.*, 1988
Suspended solids	Mouths of 12 Ontario streams flowing into the Great Lakes	ND (DL = 0.05 $\mu g \cdot L^{-1}$)	—	0/45	1974–76	Frank *et al.*, 1979
Stream bed sediments	Mouths of Grand and Saugeen rivers, Ontario	ND (DL = 0.05 $\mu g \cdot L^{-1}$)	—	0/6	1976–77	Frank, 1981
Fish (Brown bullhead [*Ictalurus nebulosus*] grizzard shad [*Dorosoma cepedianum*], black crappie [*Pomoxis nigromaculatus*]	Hillman Creek drainage, southwestern Ontario, agricultural watershed	ND (DL not given)	—	0/33	1974	Roberts *et al.*, 1979

Appendix B.
Simazine Persistence in Soil

Table B-1. Simazine Persistence in Soil

Location/soil type	Application rate ($kg \cdot ha^{-1}$)*	Soil depths measured (cm)	Concentration of residues	Time after treatment	Results and comments	Reference
FIELD STUDIES						
Columbus, Ohio	2.2	0–6	0.3 $kg \cdot ha^{-1}$	5 months	Residues measured using an oat bioassay. Simazine at 2.2 $kg \cdot ha^{-1}$ did not reduce the yield of any of the crops following corn	Stroube and Bondarenko, 1960
Silty clay loam	2.2	0–15	ND	1 year		
OM = 6.3%	4.5	0–15	0.5 $kg \cdot ha^{-1}$	1 year		
pH = 6.4	9	0–15	3.1 $kg \cdot ha^{-1}$	1 year		
Begbroke, England						
Sandy loam soil	1.8	0–10	Phytotoxic residues persisted for 12 months post-treatment on uncropped plots		Phytotoxicity (as determined by turnip bioassay) half-life = 2–4 weeks	Clay and McKone, 1968
Sand = 74.4%						
Silt = 10.4%						
Clay = 15.2%						
OM = 3.1%						
pH = 6.7						
Oxford, U.K.						
Coarse, sandy loam	1.68, for 6 years to maize	0–15	0.02–0.03 $\mu g \cdot g^{-1}$	1 year	Residues determined by turnip bioassay and some confirmed by GLC. No accumulation of residues as result of successive applicants. Residues concentrated in top 5 cm soil. After 6 weeks, 75–80% decline in phytotoxicity. Remainder declined more slowly.	Fryer and Kirkland, 1970
Sand = 75%						
Silt = 12%						
Clay = 11%						
OM = 2%						
pH = 7						
Oxford, U.K.						
Coarse, sandy loam	3.4, applied twice annually to uncropped field for 6 years	0–15	0.6–1.9 $kg \cdot ha^{-1}$ prior to subsequent sprayings	22–29 weeks	No accumulation as a result of successive applications.	
Sand = 75%						
Silt = 12%						
Clay = 11%						
OM = 2%						
pH = 7						
Southern Ontario	4.5, for 9 years to peach orchard	0–15	0.108 $kg \cdot ha^{-1}$	1 month	Residues determined by GLC/MS. No accumulation of residues as a result of successive applications; some persistence of metabolite hydroxysimazine noted. Annual simazine loss over 95%.	Khan and Marriage, 1979
Sandy loam soil			0.041 $ka \cdot ha^{-1}$	4 months		
			0.018 $kg \cdot ha^{-1}$	9 months		
			0.001 $kg \cdot ha^{-1}$	28 months		

OM = organic matter
ND = not detected
* Unless otherwise indicated.

Table B-1. Continued

Location/soil type	Application rate ($kg \cdot ha^{-1}$)*	Soil depths measured (cm)	Concentration of residues	Time after treatment	Results and comments	Reference
Saskatchewan field plots Sandy loam OM = 3.2% pH = 6.7	4.5	0–5	30% ± 16%†	5 months	Residues detected at end of growing season. Minimum degradation over winter months. Less than 2% of applied rate detected at 5- to 10-cm depth, indicating minimal leaching.	Smith and Hayden, 1976
Heavy clay OM = 4.2% pH = 7.3	4.5	0–5	43% ± 15%	5 months		
Silty loam OM = 11.7% pH = 6.2	4.5	0–5	30% ± 14%	5 months		
Wellesbourne, U.K. Sandy loam Clay = 18% OM = 2% pH = 6.2						Walker, 1967a
• incorporated (3–4 cm)	2.0	0–7.5	~20% remaining	200 d	Little differences in degradation noted between incorporated and unincorporated herbicide applications. Half-life range (interpolated from graphs) 3–5 months.	
• unincorporated (surface)	2.0	0–7.5	~20% remaining	200 d		
Wellesbourne, U.K. Sandy loam Clay = 18% OM = 2% pH = 7	2.0	0–7.5	—	—	Half-life range 42–75 d. After 120 d, 20%–30% of initial application remaining. Slower rate corresponds to cool soil conditions.	Walker, 1978
Lexington, Kentucky Maury silt loam • no-till corn OM = 4.3%–5.1% pH = 4.3–6.3	3.4	0–8	—	—	Oat bioassays indicated more rapid decrease in phototoxicity under no-tillage compared with conventional tillage. Under no-tillage, lower persistence noted at lower pH values.	Slack *et al.*, 1978
• conventional till corn OM = 2.7%–3.1% pH = 4.9–6.2	3.4	0–8	—	—		
Annapolis Valley, Nova Scotia 4 typical soil types with compositions ranging: Sand- 44.8% to 92.4% Clay - 2.0 to 35% OM - 3.0 to 7.2% pH - 4.8 to 6.1	3 $kg \cdot ha^{-1}$	0–25 cm	2%–5%	one year	Most of the measured residues located in the 0–10 cm soil horizon, however, some was recovered between 15 and 25 cm in 3 of the 4 soils.	Jensen and Kimball, 1982

†Mean and standard deviation for 3-year study.

Table B-1. Continued

Location/soil type	Application rate ($kg \cdot ha^{-1}$)*	Soil depths measured (cm)	Concentration of residues	Time after treatment	Results and comments	Reference
Prosser, Washington Warden silt loam - grapes Sand - 38.4% Clay - 8.0% Silt - 53.6% OM - 2.0% pH - 7.3	1.1 $kg \cdot ha^{-1}$ 6 years to grapes	0–30 cm	0–67 $kg \cdot ha^{-1}$	1 year	Most of the measured residues located in the 0–10 cm soil horizon.	Dawson *et al.*, 1968
	3.4 $kg \cdot ha^{-1}$ 6 years to grapes	0–30 cm	2.24 $kg \cdot ha^{-1}$	1 year		
Nebraska Silty clay loam and two clay loam soils with varying compositions	2.8	0–8	1.83 $\mu g \cdot g^{-1}$ 0.23 $\mu g \cdot g^{-1}$	4 months 16 months	Residue levels determined with oat and soybean bioassays.	Burnside *et al.*, 1963
Sand = 16.1%–49.1% Clay = 19.3%–30.7% OM = 2.2%–3.0% Silt = 31.6%–53.2% pH = 5.2–7.1	5.6	0–8	1.67 $\mu g \cdot g^{-1}$ 0.43 $\mu g \cdot g^{-1}$	4 months 16 months		
	11.2	0–8	2.00 $\mu g \cdot g^{-1}$ 0.63 $\mu g \cdot g^{-1}$	4 months 16 months		
Taichung, Taiwan Clay loam Sand = 42.5% Silt = 26.7% OM = 1.43% pH = 5.15	2	1.5–10	ND	112 d, summer and winter	Summer half-life = 18 d (hot, wet). Winter half-life = 24 d (cooler, dryer). No residues detected below 10–cm depth.	Chen *et al.*, 1983
Taipei, Taiwan loam soil, Sand = 32% Silt = 47% OM = 1.8% pH = 4.3	2	1.5–10	ND	84 d	Autumn half-life = 14 d. No residues detected below 10–cm depth.	Chen *et al.*, 1983
Britain	112 (simulated spill)	0–5.1 5.1–10.2 10.2–15.3 15.3–30.6	93% 3.3% 1.1% 1.3%	29 months 29 months 29 months 29 months	Percentage of original application remaining 29 months after simulated spill.	Hance, 1984
LABORATORY STUDIES						
Sandy loam Clay = 18% OM = 2% pH = 7	8/4/2 $\mu g \cdot g^{-1}$ (initial concentration)	—	—	—	Laboratory incubation studies at 25°C and 11%–12% soil moisture. Observed half-lives = 43/39/36 d for initial concentrations indicated, respectively.	Walker, 1976a

Table B-1. Continued

Location/soil type	Application rate ($kg \cdot ha^{-1}$)*	Soil depths measured (cm)	Concentration of residues	Time after treatment	Results and comments	Reference
Sandy loam Clay = 18% OM = 2% pH = 7	8 $\mu g \cdot g^{-1}$ (initial concentration)	—	—	—	Laboratory incubation studies at 25°C and moisture contents ranging from 13.2% to 4.8%. Half-life range observed for this interval 37–85 d.	Walker, 1976a
Oxford, U.K. Coarse sandy loam Sand = 75% Silt = 12% Clay = 11% OM = 2% pH = 7	4 $\mu g \cdot g^{-1}$ (initial concentration)	—	—	—	Laboratory incubation studies. Temperature change from 30°C to 5°C yielded half-life change from 29 to 209 d at 4% moisture content and 16–125 d at 12% moisture content.	Walker, 1976b
Neuhofen, Germany Sandy loam Sand = 83.8% Clay = 7.3% OM = 3.8% pH = 6.5	10 $\mu g \cdot g^{-1}$ (initial concentration)	—	—	—	Half-life = 100 d at 22°C and 40% moisture capacity in laboratory incubation study.	Burkhard and Guth, 1981
Hutzenbuhl, Germany Loamy sand Sand = 77.1% Clay = 10.5% OM = 1.8% pH = 4.8	10 $\mu g \cdot g^{-1}$ (initial concentration)	—	—	—	Half-life = 45 d at 22°C and 40% moisture capacity in laboratory incubation study.	Burkhard and Guth, 1981
SOIL COLUMN LEACHING TESTS						
Sandy loam Sand = 70.1% Silt = 25.1% Clay = 4.8% OM = 1.4% pH = 4.6	5(80% ai)	10	93% 35%		Proportion of chemical remaining after irrigation with 20, 40, and 80 cm water, respectively.	Hogue *et al.*, 1981
Loam Sand = 39.5% Silt = 38.1% Clay = 22.4% OM = 3.8% pH = 7.5	5	10	92% 49% 16%		Proportion of chemical remaining after irrigation with 20, 40, and 80 cm water, respectively.	

Appendix C.

Observed and Calculated Bioconcentration Factors for Simazine in Aquatic Organisms

Table C-1. Observed and Calculated Bioconcentration Factors for Simazine in Aquatic Organisms

Species	Exposure medium/ concentration	Formulation	Tissue/ concentration	Bioconcentration factor (BCF)*	Time span	Comments	Reference
Fish (freshwater) Green sunfish (*Lepomis cyanellus*)	Water/1 $\mu g \cdot g^{-1}$	—	Whole body/0.95 $\mu g \cdot g^{-1}$	0.95	3 weeks		Rodgers, 1970
	Water/3 $\mu g \cdot g^{-1}$	—	Whole body/2.29 $\mu g \cdot g^{-1}$	0.76	3 weeks		
	Diet/3 $\mu g \cdot g^{-1}$ b.w.	—	Whole body/0.0 $\mu g \cdot g^{-1}$	—	3 weeks	Force fed, weekly analyses 72 h after last feeding.	
	Diet/10 $\mu g \cdot g^{-1}$ b.w.	—	Whole body/6 $\mu g \cdot g^{-1}$	—	3 weeks	Force fed, weekly analyses 72 h after last feeding.	
	Diet/10 $\mu g \cdot g^{-1}$ b.w.	—	Whole body/0.0 $\mu g \cdot g^{-1}$	—	3 weeks	As above, but 168 h after last feeding.	
	Depuration in clean water	—	Whole body/0.0 $\mu g \cdot g^{-1}$		7 d		
Various fish (freshwater) (*Lepomis macrochirus*) (Bullhead bluegill) (*Ictaliurus sp.*), green sunfish (*Lepomis cyanellus*), pumpkinseed (*Lepomis gibbosus*), goldfish (*Carassius auratus*)	Water/1.7 $mg \cdot L^{-1}$	—	Viscera/1.1 $\mu g \cdot g^{-1}$ (1 d post-treatment), 0.47 $\mu g \cdot g^{-1}$ (30 d post-treatment)	<1	30 d	Similar pattern in fish meat; however, residues in skin reached a high of 0.95 $\mu g \cdot g^{-1}$ (8 d) and 0.56 $\mu g \cdot g^{-1}$ (30 d) post-treatment.	Thomas, 1967
Fish (freshwater) Bluegill, (*Lepomis macrochirus*)	Water	—	—	5	28 d	—	Reinert and Rodgers, 1987
Fish (freshwater) Bluegill (*Lepomis macrochirus*)	Water/single dose, initial concentration 3 $mg \cdot L^{-1}$	—	Whole body	<1	Long-term exposure in treatment ponds	Concentration in fish generally less than exposure concentration throughout 2-year study in treatment ponds.	Mauck *et al.*, 1976
Fish (freshwater) Catfish (*Ictalrus sp.*)	Water		—	2	28 d	—	Reinart and Rodgers, 1987
Fish (freshwater) Rainbow trout (*Salmo gairdneri*) (1 year old)	Water/static, nominal	Technical grade: 1 $\mu g \cdot g^{-1}$ (ai) 4 $\mu g \cdot g^{-1}$ (ai) 12.5 $\mu g \cdot g^{-1}$ (ai)	Flesh	0.15 0.26 0.27	24 h	Bioaccumulation generally proportional to exposure concentration.	Dodson and Mayfield, 1979

b.w. = body weight

*BCF (bioconcentration factor) = concentration in tissue/concentration in medium (water or diet).

Table C-1. Continued

Species	Exposure medium/ concentration	Formulation	Tissue/ concentration	Bioconcentration factor (BCF)*	Time span	Comments	Reference
		Princep 80W:				Fish residues well below water concentration.	
		1 $\mu g \cdot g^{-1}$ (ai)		0.08			
		4 $\mu g \cdot g^{-1}$ (ai)		0.30			
		12.5 $\mu g \cdot g^{-1}$ (ai)		0.18			
Fish (freshwater) Fathead minnow (*Pimephales promelas*)	Water/continuous exposure to 1.7 $mg \cdot L^{-1}$	—	—	55	—	Abstract only available. No simazine residues after 3-d exposure to uncontaminated water.	Mayer and Sanders, 1977
Benthic invertebrate Mayfly (*Hexagenia* sp.)	Water/single dose, initial concentration 3 $mg \cdot L^{-1}$	—	Whole body	92	8 d	Maximum reported BCF from treated pond. BCF based on measured residues in water and mayflies 8 d post-treatment. BCF subsequently declined throughout remainder of year.	Mauck *et al.*, 1976
Aquatic plant (*Elodea canadensis*)	Water/5 $mg \cdot L^{-1}$	98%	Stems and leaves	Residues ($ng \cdot mg^{-1}$ dry wt.):	Exposures for:	Maximum BCF = 4.4.	Dabydeen and Leavitt, 1981
				4	0 min		
				14	5 min		
				17	20 min		
				22	60 min		
				22	120 min		

Appendix D.

Acute and Chronic Toxicity of Simazine to Freshwater Aquatic Organisms

Table D-1. Acute and Chronic Toxicity of Simazine to Freshwater Aquatic Organisms

Species	Test conditions	Temperature (°C)	pH	Water hardness (mg $CaCO_3 \cdot L^{-1}$)	Formulation (% active)	Toxicity parameter	Reference
Fish Striped bass fingerlings (*Morone saxatilis*)	96-h static test; no solvents used; at highest concentrations (180 $mg \cdot L^{-1}$), precipitate formed on bottom of containers	21	6.9	35 and 137 (2 tests)	80% wettable powder	96-h LC_{50} > 180 $mg \cdot L$-1 (144 $mg \cdot L$-1) as active ingredient) (no fish died at 2 highest concentrations: 100, 180 $mg \cdot L^{-1}$	McCann, 1980
Fish Striped bass (*Morone saxatilis*)	48-h static test; no aeration; simazine concentration measured at beginning and end; organic solvents not used. Note: up to 30% mortality in controls	20 ± 1	7.7	120 ± 15 (soft)	Commercial preparation	48-h LC_{50} = 16 $mg \cdot L^{-1}$	Fitzmayer *et al.*, 1982b
		20 ± 1	8.1	220 ± 11 (hard)	80% active; test concentrations based on active ingredient	48-h LC_{50} = 18 $mg \cdot L^{-1}$	
3-d-old larvae		20 ± 1	7.7	120 ± 15 (soft)		48-h LC50 > 100 $mg \cdot L^{-1}$	
7-d-old larvae		20 ± 1	8.1	20 ± 11 (hard)		48-h LC50 > 100 $mg \cdot L^{-1}$	
Fish Striped bass fingerlings (*Roccus saxatilis*)	—	—	—	—	80% wettable powder	96-h LC_{50} = 0.25 $mg \cdot L^{-1}$	Wellborn, 1969
Fish Striped bass fingerlings (*Morone saxatilis*)	3 $mg \cdot L^{-1}$ in aquaria exposure	—	—	—	80% wettable powder	No mortality at 3 $mg \cdot L^{-1}$	Cook and Smith, 1976
Fish Striped bass fingerlings (*Morone saxatilis*)	3 $mg \cdot L^{-1}$ applied 3 times at 7-d intervals in pond exposure	—	—	—	80% wettable powder	No mortality at 3 $mg \cdot L^{-1}$	Cook and Smith, 1976
Fish Fathead minnow (*Pimephales promelas*)	96-h static test	25	7.4	44	4% granular	96-h LC_{50} = 5 $mg \cdot L^{-1}$ (range = 3.5–7.2 $mg \cdot L^{-1}$)	Mayer and Ellersieck, 1986
Fish Fathead minnow (*Pimephales promelas*)	96-h static test	25	7.4	44	80% wettable powder	96-h LC_{50} = 510 $mg \cdot L^{-1}$ (range = 373–698 $mg \cdot L^{-1}$)	Mayer and Ellersieck, 1986
Fish Bluegill (*Lepomis macrochirus*)	—	—	—	—	—	LD_{50} > 100 $mg \cdot L^{-1}$	Sanders, 1970
Fish Bluegill (*Lepomis macrochirus*)	—	—	—	—	—	LD_{50} > 90 $mg \cdot L^{-1}$	Worthing and Walker, 1987
Fish Sunfish (*Lepomis spp.*)	Static, salt used as a carrier; numerous varieties of sunfish tested; duration of tests not specified	—	—	—	—	LC_{50} range = 11–695 $mg \cdot L^{-1}$	Walker, 1964

Table D-1. Continued

Species	Test conditions	Temperature (°C)	pH	Water hardness (mg $CaCO_3 \cdot L^{-1}$)	Formulation (% active)	Toxicity parameter	Reference
Fish Rainbow trout (*Salmo gairdneri*)	Static, 40–L aquaria	18	7.2	250	50–100	24-h TL_m = 95 mg•L^{-1}	Alabaster, 1969
Fish Rainbow trout (*Salmo gairdneri*)	Static, 40–L aquaria	18	7.2	250	50–100	48-h TL_m = 43 mg•L^{-1}	Alabaster, 1969
Fish Rainbow trout (*Salmo gairdneri*)	Static	12	7.4	44	Technical grade	96-h LC_{50} > 100 mg•L^{-1}	Mayer and Ellersieck, 1986; Worthing and Walker, 1987
Fish Major carp fingerlings (*Cirrhina mrigala*)	Static, aerated, unmeasured	—	—	—	—	96-h LC_{50} between 25 and 50 mg•L^{-1}	Singh and Yadav, 1978
Fish Major carp fingerlngs (*Cirrhina mrigala*)	24-d static test in 90–L aquarium with aquatic plants; not aerated; not measured; aquatic plants died post-treatment	—	—	—	—	% mortality vs. initial simazine concentration 25%–2.5 mg•L^{-1} 35%–5.0 mg•L^{-1} 50%–7.5 mg•L^{-1} Note: water quality parameters not measured, potential deoxygenation noted	Singh and Yadav, 1978
Fish Common carp (*Cyprinus carpio*)	Static egg hatching tests, unmeasured Note: no apparent correction for control data	16	7.5	360	—	50% hatching at 40 mg•L^{-1} % deformed larvae vs. concentration 9%–20 mg•L^{-1} 12%–30 mg•L^{-1} 21%–40 mg•L^{-1} 53%–60 mg•L^{-1} no hatch–80 mg•L^{-1}	Kapur and Yadav, 1982
Aquatic earthworms (*Oligochaeta*)	Static	70	Slightly alkaline	21–24	80% wettable powder	96-h LC_{50} = 28 mg•L^{-1}	Walker, 1964
Common midge larvae (*Tendipedidae*)	Static	70	Slightly alkaline	21–24	80% wettable powder	96-h LC_{50} = 28 mg•L^{-1}	Walker, 1964

Table D-1. Continued

Species	Test conditions	Temperature (°C)	pH	Water hardness (mg $CaCO_3 \cdot L^{-1}$)	Formulation (% active)	Toxicity parameter	Reference
Crustacean (*Daphnia pulex*)	Static, unmeasured	20 ± 1	7.4–7.7	105–120	Commercial preparation 80% active; test concentrations reported as active ingredient	48–h LC_{50} = 5.3 mg•L^{-1} 48-h LC_{50} = 21.3 mg•L^{-1} 48-h LC_{50} = 92.1 mg•L^{-1}	Fitzmayer *et al.*, 1982a
Crustacean (*Daphnia pulex*)	Static with daily replacement of test solution, cleaning of containers, and food replenishment; concentrations of 4 mg•L^{-1} and 20 mg•L^{-1}, plus controls	20 ± 1	7.4–7.7	105–120	Commercial preparation 80% active; test concentrations reported as active ingredient	Average survival times: at 20 mg•L^{-1}–9.6 d; 4 mg•L^{-1}–65% dead after 25 d, with most mortality during 21–25 d (growth suppression, reproduction delayed)	Fitzmayer *et al.*, 1982a
Crustacean (*Daphnia pulex*)	48-h static; no aeration; simazine measured at beginning and end for highest concentrations	20 ± 1 20 ± 1	7.7 8.1	120 ± 15 120 ± 11	Commercial preparation 80% active; test concentrations reported as active ingredient	48-h LC_{50} > 50 mg•L^{-1} 48-h LC_{50} > 50 mg•L^{-1}	Fitzmayer *et al.*, 1982b
Crustacean *Daphnia magna*	Static	21	7.4	272	Technical grade	Immobilization at 1 mg•L^{-1} (48h)	Sanders, 1970
Seed Shrimp (*Cypridopsis vidua*)	Static	21	7.4	272	Technical grade	Immobilization at 3.2 mg•L^{-1} (48 h)	Sanders, 1970
Crustacean *Daphnia magna*	Static	21 ± 1	8.4	250	Technical grade	48-h LC_{50} > 3.5 mg•L^{-1}	Marchini *et al.*, 1988
Aquatic plant (*Elodea canadensis*)	Static, unmeasured; morphological and structural changes examined	—	—	—	Technical grade (98%)	24-h LOEL = 3 mg•L^{-1} (Note: 1 mg•L^{-1} produced no visible change after 24–96 h)	Dabydeen and Leavitt, 1981
Periphytic algal community	Limnocorrals *in situ* Delta Marsh, treated with 0.1, 1.0, and 5.0 mg•L^{-1} simazine just prior to algal colonization	—	—	—	Technical grade	Community LC_{50} (herbicide concentration causing 50% reduction in biovolume) between 0.1 and 1.0 mg•l^{-1}	Goldsborough and Robinson, 1986
Filamentous algae	Static test flasks innoculated with simazine to produce test concentrations of 0.1–1.0 mg•L^{-1}; photosynthesis rates measured for 5–6 min before and after simazine injection; light intensity 100 µE•m^{-2}•s^{-1}	25 ± 0.1	—	—	—	50% inhibition of photosynthesis:	O'Neal and Lembi, 1983
Cladophora glomerata						0.77 mg•L^{-1}	
Pithophora oedogonia						0.61 mg•L^{-1}	
Siprogyra jurgensii						0.22 mg•L^{-1}	
Planktonic algae (*Ankistrodesmus brainii*)						0.95 mg•L^{-1}	

Table D-1. Continued

Species	Test conditions	Temperature (°C)	pH	Water hardness (mg $CaCO_3 \cdot L^{-1}$)	Formulation (% active)	Toxicity parameter	Reference
Green algae *Selenastrum capricornutum*	Static, unmeasured, used algal assay medium; when stream water used, no inhibitory response equivalent to 50% of control occurred with concentration range tested (0.01–10 $\mu g \cdot L^{-1}$)	—	7.7	—	Princep 4G	EC_{50} = 2.24 $\mu g \cdot L^{-1}$ (using 24-h oxygen evolution assay) EC_{50} = 0.164 $\mu g \cdot L^{-1}$ (using 21-d EPA bottle test)	Turbak *et al.*, 1986
Green algae (*Chlamydomonas sp.*)	Static, unmeasured	20	—	—	Technical grade	At 0.052 and 0.104 $mg \cdot L^{-1}$, stimulatory effect on chlorophyll in comparison with control (3.4% and 6.9%, respectively); at 0.208 $mg \cdot L^{-1}$, inhibitory effect (i.e., -64.7%) on chlorophyll	Foy and Hiranpradit, 1977
Green algae (*Chlorella sp.*)	Static, unmeasured	20			Technical grade	At 0.52 $mg \cdot L^{-1}$, stimulatory effect on chlorophyll in comparison with control (56.4%); at 0.208 $mg \cdot L^{-1}$, inhibitory effect (i.e., -62.7%) on chlorophyll	
Planktonic algae *Scenedesmus quadricauda*	Static test flasks with continuous shaking: simazine concentrations (up to 200 $mg \cdot L^{-1}$) reported as active ingredients, based on innoculation and not measured *in situ*; results determined 4 d after innoculation; results based on differences in populations determined by visual comparison by colour with controls.	25	7	—	Commercial preparation 80% active, test concentrations based on active ingredient	Not toxic at concentrations up to 200 $mg \cdot L^{-1}$	Vance and Smith, 1969
Chlamydomonas eugametos						Increased growth at concentrations up to 200 $mg \cdot L^{-1}$	
Chlorella pyrenoidosa						Not toxic at concentrations up to 200 $mg \cdot L^{-1}$	