E X T O X N E T

Extension Toxicology Network

A Pesticide Information Project of Cooperative Extension Offices of Cornell University, Michigan State University, Oregon State University, and University of California at Davis. Major support and funding was provided by the USDA/Extension Service/National Agricultural Pesticide Impact Assessment Program.

Pesticide
Information
Profile

Simazine

Publication Date: 9/93

TRADE OR OTHER NAMES

Some trade names include Aquazine, Cekusan, Cekusima, Framed, G-27692, Gesatop, Primatol, Princep, Simadex, Simanex, Tanzine and Totazina (24, 25, 26).

REGULATORY STATUS

Simazine is a general use pesticide. Products containing simazine must bear the signal word "Caution" (25).

INTRODUCTION

Simazine is a selective triazine herbicide. It is used to control broad- leaved weeds and annual grasses in field, berry fruit, vegetable and ornamental crops, on turfgrass, and in orchards and vineyards. At higher rates, it is used for nonselective weed control in industrial areas. Before 1992, simazine was used to control submerged weeds and algae in large aquariums, farm ponds, fish hatcheries, swimming pools, ornamental ponds and cooling towers. Simazine is available in wettable powder, water dispersible granule, liquid and granular formulations (5, 25).

TOXICOLOGICAL EFFECTS

ACUTE TOXICITY

Simazine is highly toxic if inhaled, moderately toxic if ingested, and slightly toxic via dermal exposure (29). No cases of poisoning in humans have been reported from ingestion of simazine (29). Rats given an oral dose of 5,000 mg/kg exhibited drowsiness and irregular breathing. A single oral dose of 4,200 mg/kg produced anorexia, weight loss and some deaths in rats within 4 to 10 days (26).

Rashes and dermatitis from occupational exposure to simazine have occurred (24). Simazine is slightly irritating to the skin and moderately irritating to the eyes of rabbit (29). Patch tests on humans have shown that simazine is not a skin irritant, fatiguing agent or sensitizer (24, 25). In rabbits, 80 mg of simazine produced irritation in the eye. No systemic toxicity was observed in a 21 day dermal study with rabbits exposed to doses of up to 1 gm/kg (30).

The triazine herbicides disturb energy metabolism (thiamin and riboflavin functions). Toxicity symptoms include difficulty in walking, tremor, convulsions, paralysis, cyanosis, slowed respiration, miosis (pin point pupils), gut pain, diarrhea and impaired adrenal function (14).

The amount of a chemical that is lethal to one-half (50%) of experimental animals fed the material is referred to as its acute oral lethal dose fifty, or LD50. The oral LD50 for technical simazine in rats and mice is >5,000 mg/kg. The dermal LD50 in rabbits is 3,100 to 10,000 mg/kg. The lethal concentration fifty, or LC50, is that concentration of a chemical in air or water that kills half of the experimental animals exposed to it for a set time period. The 4-hour inhalation LC50 in rats is 2 mg/m3, and the 1-hour LC50 in rats is 9800 mg/m3. The LClo in rats is 580 mg/m3 (24, 25, 28, 29, 30). An LClo is the lowest concentration which causes death in test animals.

For unknown reasons, sheep and cattle are especially susceptible to poisoning by simazine. Doses of 500 mg/kg were fatal in sheep with death delayed for 5 to 16 days (30). Symptoms exhibited by poisoned sheep include lower food intake, higher water intake, incoordination, tremors, and weakness, especially in the hindquarters (24).

CHRONIC TOXICITY

Two-year chronic oral feeding studies in which rats were given daily dosages at various rates as high as 5 mg/kg for simazine in the diet, resulted in no gross or microscopic signs of toxicity due to ingestion (29).

When rats were given repeated doses of 15 mg/kg/day, some liver cells degenerated during the first 3 days, but the condition did not progress. Instead, the liver adapted and the compound was metabolized (24).

The EPA has set a Lifetime Health Advisory (LHA) for Simazine in drinking water at 1 ug/l. EPA believes that water containing simazine at or below this level is acceptable for drinking every day over the course of one's lifetime, and does not pose any health concerns. However consuming high levels well above the LHA level over a long period of time has caused tremors, damage to the testes, kidneys, liver and thyroid, disturbances in sperm production, and gene mutations in laboratory animals (31).

Rats and guinea pigs fed 100 mg/kg daily for 6 months had decreased weight gain, increased white blood cell counts, decreased blood cholinesterase activity and deterioration and inflammation of the stomach. In a 28-day study, oral doses of 2,500 mg/kg/day to rats resulted in stomach ulcers, damage to the small intestine, and death (30).

Reproductive Effects

No adverse effects on reproductive capacity or development were observed in a three generation study of rats fed 5 mg/kg/day simazine (26). Chronic inhalation of a cumulative dose of 17 mg/m3 for 2 hr/day for 8 days in pregnant rats resulted in toxic effects on the fetuses and developmental abnormalities. Decreased weights and increased skeletal abnormalities were noted in the fetuses of pregnant rabbits fed 200 mg/kg/day (30).

Teratogenic Effects

No teratogenic effects were observed when rabbits were given daily doses of 5, 75 or 200 mg/kg for days 7 through 19 of pregnancy (26).

Mutagenic Effects

Simazine has shown negative results in a variety of mutagenicity tests on bacterial cultures (26). Tests for mutagenicity on human lung cell cultures have produced both positive and negative results (26). When injected into adult male fruitflies, simazine increased the frequency of sex-linked lethal mutations, but failed to do so when fed to larvae. Other tests for mutagenicity in fruitflies were negative (24).

Carcinogenic Effects

EPA has classified simazine as a possible human carcinogen because it may have caused cancer in test animals which received high doses over the course of their lifetimes (14, 26). Because simazine in drinking water may possibly increase the risk of cancer in humans, the Lifetime Health Advisory level set by EPA includes an additional margin of safety (26). Simazine did not produce tumors in mice given 215 mg/kg/day, the highest dose tolerated, for 18 months. Simazine produced thyroid and mammary tumors in female rats fed 5 mg/kg, the highest dietary dose tested (24, 26).

Organ Toxicity

Consuming high levels well above the LHA level over a long period of time has caused tremors, damage to the testes, kidneys, liver, and thyroid, and disturbances in sperm production in laboratory animals (31). Fate in Humans and Animals

Simazine stimulates its own breakdown in the liver (6). Some accumulation occurs in the fat (14). Anywhere from 67-97% of the simazine in the body is excreted through the urine within 24 hours. Rats were given single oral doses of 0.5 or 200 mg/kg. Seven days later, those receiving the low dose had excreted 13 to 19% of the dose in the feces, 51 to 62% in the urine, and an additional 12% was found in their body tissues. At the higher dose, approximately 2% of the dose was excreted in the feces, 22% was excreted in the urine, and another 2% was found in body tissues. Simazine was found in the rats' red blood cells, liver, kidney, fat, plasma and bone. When a cow was fed 5 ppm for 3 days, no simazine was found in the cow's milk during the next 3 days. It has been reported that simazine residues were present in the urine of sheep for up to 12 days after administration of a single oral dose. The maximum concentration in the urine occurred from 2 to 6 days after administration (26).

ECOLOGICAL EFFECTS

Effects on Birds

Birds are very tolerant to simazine (5, 8). The LC50 for 10 day old mallards and pheasants is greater than 5,000 ppm (Lethal Diet Tox. Environ. Poll. Birds. 1975). The 8-day dietary LC50 for bobwhite quail is 8,800 ppm and for mallard ducks is 51,200 ppm (29).

Effects on Aquatic Organisms

Simazine has very low toxicity to all aquatic species reviewed (5, 8). The 48-hour LC50 for simazine in rainbow trout is 56 mg/l (25). The 96-hour LC50 for simazine in rainbow trout is 2.8 ppm, 16.0 ppm in bluegill sunfish, and greater than 1.0 ppm in oysters (29).

Effects on Other Animals (Nontarget species)

Most mammals tend to be insensitive to simazine (8). Sheep and cattle are especially sensitive (6). Simazine is non-toxic to bees (5, 8, 25). Limited studies have shown some minor fungicidal and nematocidal activity but no insecticidal activity (29).

ENVIRONMENTAL FATE

Breakdown of the Chemical in Soil and Groundwater

Simazine is persistent and does not adsorb strongly to soil particles (Koc = 138 g/ml). In combination with a lengthy soil half-life, these factors suggest that simazine is likely to contaminate groundwater. Its tendency to leach is limited by its low solubility in water (6.2 ug/ml). Soil half-lives of 36 to 234 days on sandy loam, 16.3 to 25.5 weeks on loamy sand and silt loam, and 75 days on an unspecified soil type have been reported. Residual activity remains for 2-7 months (2-4 kg/ha) after application. Simazine does adsorb to clays and mucks (26, 27, 29).

Simazine is subject to decomposition by ultraviolet radiation, but this effect is small under normal field conditions. Loss from volatilization is also insignificant. In soils, microbial activity probably accounts for decomposition of a significant amount of simazine. Simazine has little if any lateral movement in soil, but can be washed along with soil particles (29).

Breakdown of the Chemical in Water

The average half life of simazine in ponds where it has been applied is 30 days, with the actual half life dependent on the level of algae present, the degree of weed infestation and other factors (29).

A 5-year survey of drinking water wells did not find any drinking water systems containing simazine at concentrations at or above the EPA's Lifetime Health Advisory for simazine of 1 ug/l. Lower concentrations were detected in 1.1% of the community water system wells and in 0.2% of the rural domestic wells tested nationwide (31).

Simazine has been found in surface water in 16 states and in groundwater in 8 states. The maximum levels detected were 1,300 ug/l in surface water and 800 ug/l in ground water (26).

Breakdown of the Chemical in Vegetation

Plants absorb simazine mainly through the roots, with little or no foliar penetration. From the roots, it is translocated upward to the stems, leaves and growing shoots of the plant (2, 29). It acts to inhibit photosynthesis (8). Resistant plants readily metabolize simazine to possibly mutagenic by- products (Menzie. Metab. Pesticides. 1974, 2; Environ. Health Perspect. 27:45. 1978).

Plants that are sensitive to simazine accumulate it unchanged (2). It is possible that livestock or wildlife grazing on these plants could be poisoned.

PHYSICAL PROPERTIES AND GUIDELINES

Simazine is a white crystalline solid (24). It is stable to natural light and extremes of temperature (2). It is stable in neutral to slightly basic or acidic media, but is hydrolyzed by stronger acids and bases (24, 30). Simazine presents a slight fire hazard if exposed to heat and flame. It may burn, but

does not readily ignite. Thermal decomposition of simazine may release toxic oxides of nitrogen and carbon, and toxic and corrosive fumes of chlorides (30).

Occupational Exposure Limits:

No occupational exposure limits for simazine have been set by OSHA, NIOSH, or ACGIH (30).

Physical Properties:

CAS #:	122-34-9
Specific gravity:	1.302 (30)
H20 solubility:	3.5 mg/l at 20 degrees C, or 5 ppm at 20-22 degrees C (24, 25)
Solubility in other solvents:	Solubility at 20 degrees C
	Chloroform 900 ppm (1)
	Light petroleum 2 ppm (6)
	Methanol 400 ppm (6)
Melting point:	225-227 degrees C (439-441 degrees F) (30, 2)
Vapor pressure:	6.1 x 10 to the minus 9 mm Hg at 20 degrees C (5)
Density:	1.302 g/cm3 (7)
Koc:	138 g/ml (27)
Chemical Class/Use:	triazine herbicide

BASIC MANUFACTURER

Ciba-Geigy Corporation
Agricultural Division
PO Box 18300
Greensboro, NC 27419-8300

Review by Basic Manufacturer:

Comments solicited: January, 1992
Comments received: April, 1992

REFERENCES

1. Meister, R.T. (ed.) 1987. Farm Chemicals Handbook. Willoughby, OH: Meister Publishing Co.
2. WSSA Herbicide Handbook Committee. 1983. Herbicide Handbook of the Weed Science Society of America. 5th Ed. WSSA, Champaign, IL.
3. Hazardous Materials Advisory Committee. 1974. EPA-SAB-74-001 Herbicide Report Chemical Analysis, Environmental Effects, Agriculture and Other Applied Uses. EPA: May.
4. Tucker, Richard. 1970. Handbook of toxicity of pesticides to wildlife. USDI Fish & Wildlife Service.
5. Worthing, C.R. (ed.). 1987. The pesticide manual: A world compendium. 8th Ed. The British Crop Protection Council. Croydon, England.
6. Hayes, Wayland, Jr. 1982. Pesticides studied in man. Baltimore, MD: Williams & Wilkins.

7. Kearney, P.C. & D.D. Kaufman (eds.). 1975. Herbicides: chemistry, degradation, and mode of action. 2nd Ed. Vol. 1 & 2. New York: M. Dekker.
8. Hartley, D. and H. Kidd, (eds.) 1983. The Agrochemicals Handbook. Nottingham, England: Royal Society of Chemistry.
9. Crop Protection Chemicals Reference. 1986. 2nd Ed. New York: Chemical and Pharmaceutical Pub. Co.
10. Shepard, T.H. 1973. Catalog of teratogenic agents. Baltimore, MD: John Hopkins University Press.
11. Schardein, James. 1985. Chemically induced birth defects. New York: Marcel Dekker.
12. U.S. Department of Health, Education and Welfare. 1976. Suspected Carcinogens. A subfile of the registry of toxic effects of chemical substances. Washington, DC: EPA.
13. Department of Transportation. 1984. Emergency Response Guidebook: Guidebook for hazardous materials incidents. Washington, DC: U.S. DOT.
14. Hallenbeck, W.H. & K.M. Cunningham-Burns. 1985. Pesticides and human health. New York: Springer-Verlag.
15. Lef'evre, M.J. 1980. First aid manual for chemical accidents. New York: Van Nostrand Reinhold.
16. TOXNET. 1985. National library of medicine's toxicology data network. Hazardous Substances Databank. Public Health Service. National Institute of Health. U.S. Department of Health and Human Services. Bethesda, MD: NLM.
17. Sax, N.I. 1975. Dangerous properties of industrial materials. 4th Ed. New York: Van Nostrand Reinhold Co.
18. Casarett, L.J. 1980. Casarett & Doull's Toxicology: the basic science of poisons. 2nd Ed. New York: Macmillan.
19. Gosselin, R.E. 1984. Clinical toxicology of commercial products. 5th Ed. Baltimore, MD: Williams & Wilkins.
20. National Fire Protection Association (NFPA). 1978. Fire Protection Guide. Hazardous Materials.
21. Morgan, D.P. 1982. Recognition and management of pesticide poisonings. Iowa Pesticide Hazardous Assessment Project. 1982. Iowa City, IA.
22. Windholz, M. (ed.) 1976. The Merck Index: an encyclopedia of chemicals and drugs. 9th Ed. Rahway, NJ: Merck.
23. Sunshine, Irving. 1969. Handbook of analytical toxicology. Cleveland, OH: Chemical Rubber Co.
24. Hayes, W.J. and E.R. Laws (ed.). 1990. Handbook of Pesticide Toxicology, Vol. 3, Classes of Pesticides. Academic Press, Inc., NY.
25. Meister, R.T. (ed.). 1992. Farm Chemicals Handbook '92. Meister Publishing Company, Willoughby, OH.
26. U.S. Environmental Protection Agency. 1988 (Aug.). Simazine: Health Advisory. Office of Drinking Water, US EPA, Washington, DC.
27. U.S. Department of Agriculture, Soil Conservation Service. 1990 (Nov.). SCS/ARS/CES Pesticide Properties Database: Version 2.0 (Summary). USDA - Soil Conservation Service, Syracuse, NY.
28. Ciba-Geigy Corp. 1990 (May 3). Toxicological Overview for Simazine Technical. Agricultural Div., Ciba-Geigy Corp., Greensboro, NC.
29. WSSA Herbicide Handbook Committee. Herbicide Handbook of the Weed Science Society of America, 6th Ed. WSSA, Champaign, IL. 1989.
30. Occupational Health Services, Inc. 1991 (Feb. 21). MSDS for Simazine. OHS Inc., Secaucus, NJ.
31. U.S. Environmental Protection Agency. 1990 (fall). National Pesticide Survey: Simazine. Office of Water, Office of Pesticides and Toxic Substances, US EPA, Washington DC.