1989

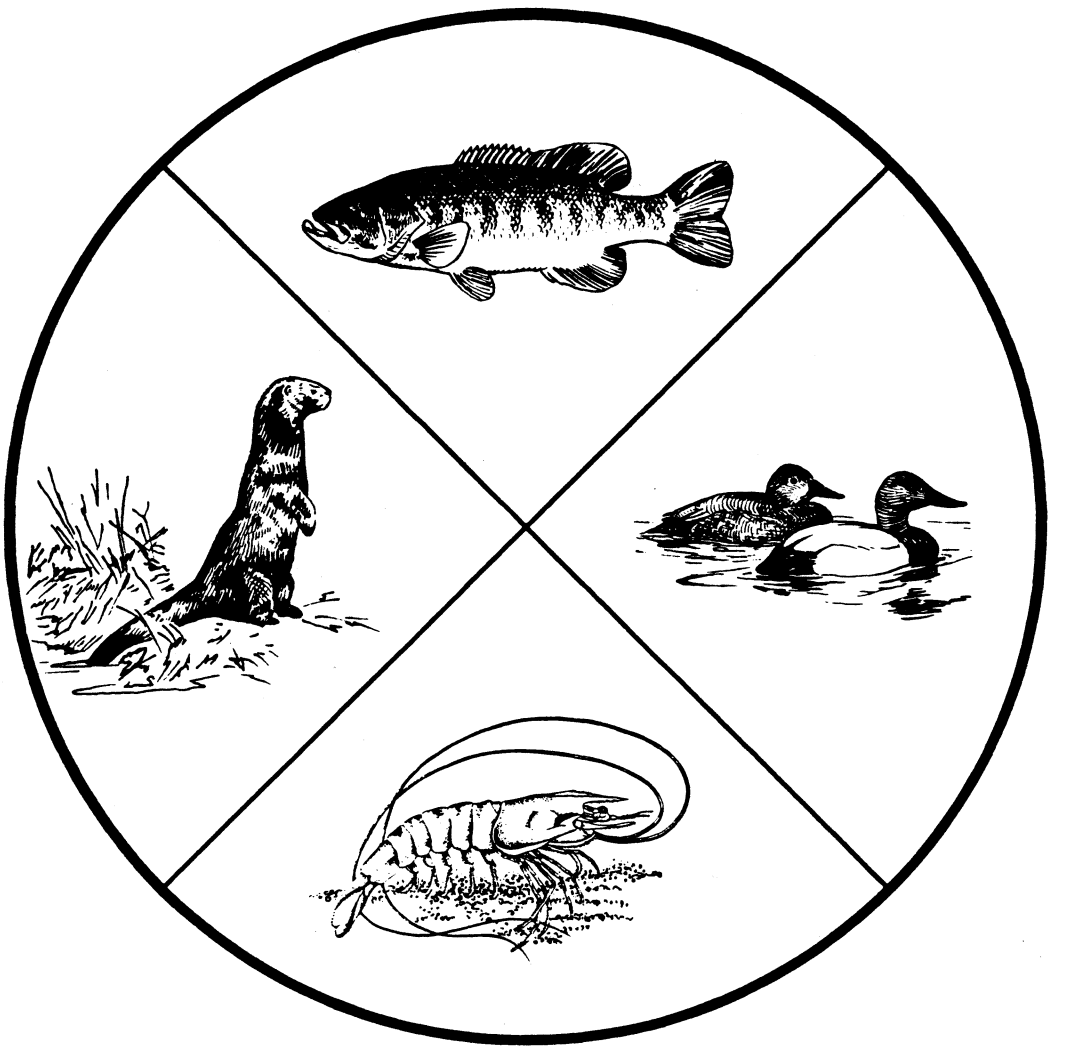
Atrazine Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review

Ronald Eisler
U.S. Fish and Wildlife Service

BIOLOGICAL REPORT 85(1.18)
MAY 1989

CONTAMINANT HAZARD REVIEWS
REPORT NO. 18

ATRAZINE HAZARDS TO FISH, WILDLIFE, AND INVERTEBRATES: A SYNOPTIC REVIEW



Fish and Wildlife Service

U.S. Department of the Interior

Other publications in the Contaminant Hazard Review series[a]

Subject	Publication date	Publication number
Mirex	March 1985	85(1.1)
Cadmium	July 1985	85(1.2)
Carbofuran	August 1985	85(1.3)
Toxaphene	August 1985	85(1.4)
Selenium	October 1985	85(1.5)
Chromium	January 1986	85(1.6)
Polychlorinated Biphenyls	April 1986	85(1.7)
Dioxins	May 1986	85(1.8)
Diazinon	August 1986	85(1.9)
Mercury	April 1987	85(1.10)
Polycyclic Aromatic Hydrocarbons	May 1987	85(1.11)
Arsenic	January 1988	85(1.12)
Chlorpyrifos	March 1988	85(1.13)
Lead	April 1988	85(1.14)
Tin	January 1989	85(1.15)
Index to Species	February 1989	85(1.16)
Pentachlorophenol	April 1989	85(1.17)

[a]Copies of individual reviews, if available, can be obtained by writing the Section of Information Management, Patuxent Wildlife Research Center, U.S. Fish and Wildlife Service, Laurel, Maryland 20708.

Biological Report 85(1.18)
May 1989

Contaminant Hazard Reviews
Report No. 18

ATRAZINE HAZARDS TO FISH, WILDLIFE, AND INVERTEBRATES: A SYNOPTIC REVIEW

by

Ronald Eisler

U.S. Fish and Wildlife Service
Patuxent Wildlife Research Center
Laurel, MD 20708

Fish and Wildlife Service
U.S. Department of the Interior

DISCLAIMER

Mention of trade names or commercial products does not constitute endorsement or recommendation for use by the U.S. Government.

Suggested citation:

Eisler, R. 1989. Atrazine hazards to fish, wildlife, and invertebrates: a synoptic review. U. S. Fish Wildl. Serv. Biol. Rep. 85(1.18). 53 pp.

SUMMARY

The herbicide atrazine (2-chloro-4-ethylamino-6-isopropylamino-1,3,5-triazine) is the most heavily used agricultural pesticide in North America. In the United States alone, more than 50 million kg (110 million pounds) are applied annually to more than 25 million ha (62 million acres), primarily to control weeds in corn and sorghum. Residues have been detected at phytotoxic concentrations in groundwater, lakes, and streams as a result of runoff from treated fields. Atrazine degrades rapidly, usually by way of hydrolysis, nitrogen dealkylation, and splitting of the triazine ring to less toxic compounds not normally inhibitory to plants and animals. The half-time persistence of atrazine in soils is usually about 4 days, but may range up to 385 days in dry, sandy, alkaline soils, under conditions of low temperature and low microbial densities. Half-time persistence is about 3 days in freshwater, 30 days in marine waters, 35 days in marine sediments, and less than 72 hours in vertebrate animals.

Sensitive species of aquatic plants experience temporary, but reversible, adverse effects at concentrations in the range of 1 to 5 ug atrazine/l. However, potentially harmful phytotoxic concentrations of atrazine, i.e., >10 ug/l for extended periods, have not been documented in the environment, and are probably unrealistic under current application and degradation rates. Aquatic fauna are indirectly affected at atrazine concentrations of 20 ug/l and higher, partly through reduction of the food supply of herbivores, and partly through loss of macrophyte habitat. Direct adverse effects to aquatic invertebrates and fishes were measured at 94 ug/l and higher. Bioaccumulation of atrazine is limited, and food chain biomagnification is negligible in aquatic ecosystems. Birds are comparatively resistant to atrazine, having a low probability for uptake and retention. Known acute oral LD-50 values for birds are >2,000 mg/kg body weight, and dietary LD-50s are >5,000 mg/kg ration. However, indirect ecosystem effects of atrazine on seed- and insect-eating birds are unknown, and should be investigated. Data are lacking for atrazine toxicity to mammalian wildlife, but tests with domestic livestock and small laboratory animals indicate that this group is also comparatively resistant. Acute oral LD-50s for mammals are >1,750 mg/kg body weight; no adverse effects were measured at chronic dietary levels of 25 mg/kg (about 1.25 mg/kg body weight) and, for some species, 100 mg/kg diet.

Proposed criteria for aquatic life protection include <5 ug atrazine/l for sensitive species of aquatic flora, and <11 ug/l for most species of

aquatic plants and animals. No criteria have been promulgated for human or animal health protection, although it has been suggested that <7.5 ug/l in drinking water, and <0.0375 mg atrazine/kg body weight (<2.25 mg daily for a 60 kg adult, <1.5 mg/kg diet based on consumption of 1.5 kg food daily) would pose a negligible risk to human health. Additional data are needed on toxicity, environmental fate, and chemistry of atrazine and its metabolites in order to maintain existing registrations or to permit new registrations. In particular, more research is needed on possible synergistic or additive effects of atrazine with other agricultural chemicals in aquatic environments.

CONTENTS

TABLES

ACKNOWLEDGMENTS

I thank Nancy A. Bushby, Lynda J. Garrett, and Joyce Haber Corley for literature search and retrieval; Barbara A. Roberts for secretarial help; W. James Fleming, Gary H. Heinz, A. Dennis Lemly, Harry M. Ohlendorf, and David L. Trauger for technical and scientific review; and Paul H. Eschmeyer and James R. Zuboy for editorial services.

INTRODUCTION

Atrazine (2-chloro-4-ethylamino-6-isopropylamino-1,3,5-triazine) is the most heavily used agricultural pesticide in North America (DeNoyelles et al. 1982; Stratton 1984; Hamilton et al. 1987), and is currently registered for use in controlling weeds in numerous crops, including corn (Zea mays), sorghum (Sorghum vulgare), sugarcane (Saccharum officinarum), soybeans (Glycine max), wheat (Triticum aestivum), pineapple (Ananas comusus), and various range grasses (Reed 1982). Atrazine was first released for experiment station evaluations in 1957 and became commercially available in 1958 (Hull 1967; Jones et al. 1982). In 1976, 41 million kg (90 million pounds) were applied to 25 million ha (62 million acres) on farms in the United States, principally for weed control in corn, wheat, and sorghum crops; this volume represented 16% of all herbicides and 9% of all pesticides applied in the United States during that year (DeNoyelles et al. 1982; Hamala and Kollig 1985). By 1980, domestic usage had increased to 50 million kg (Reed 1982). In Canada, atrazine was the most widely used of 77 pesticides surveyed (Frank and Sirons 1979). Agricultural use of atrazine has also been reported in South Africa, Australia, New Zealand, Venezuela, and in most European countries (Reed 1982). Resistance to atrazine has developed in various strains of weeds typically present in crop fields--sometimes in less than two generations (Bettini et al. 1987; McNally et al. 1987)--suggesting that future agricultural use of atrazine may be limited.

Atrazine has been detected in lakes and streams at levels ranging from 0.1 to 30.3 ug/l; concentrations peak during spring, which coincides with the recommended time for agricultural application (Hamilton et al. 1987). In runoff waters directly adjacent to treated fields, atrazine concentrations of 27 to 69 ug/l have been reported, and may reach 1,000 ug/l (DeNoyelles et al. 1982). Some of these concentrations are demonstrably phytotoxic to sensitive species of aquatic flora (DeNoyelles et al. 1982; Herman et al. 1986; Hamilton et al. 1987). Although atrazine runoff from Maryland corn fields was suggested as a possible factor in the decline of submerged aquatic vegetation in Chesapeake Bay, which provides food and habitat for large populations of waterfowl, striped bass (Morone saxatilis), American oysters (Crassostrea virginica), and blue crabs (Callinectes sapidus), it was probably not a major contributor to this decline (Forney 1980; Menzer and Nelson 1986).

This report was prepared in response to requests for information from environmental specialists of the U. S. Fish and Wildlife Service. It is part of a continuing series of brief reviews on hazards of selected chemicals to natural resources.

ENVIRONMENTAL CHEMISTRY

Atrazine is a white crystalline substance that is sold under a variety of trade names for use primarily as a selective herbicide to control broadleaf and grassy weeds in corn and sorghum (Table 1). It is only slightly soluble in water (~33 mg/l at 27 °C), but soluble (360 to 183,000 mg/l) in many organic solvents. Atrazine is usually applied in a water spray at concentrations of 2.2 to 4.5 kg/ha before weeds emerge. Stored atrazine is stable for several years, but degradation begins immediately afer application (Table 1). The chemical is available as a technical material at 99.9% active ingredient and as a manufacturing-use product containing 80% atrazine for formulation of wettable powders, pellets, granules, flowable concentrates, emulsifiable concentrates, or tablets (EPA 1983).

There are three major atrazine degradation pathways: hydrolysis at carbon atom 2, in which the chlorine is replaced with a hydroxy group; N-dealkylation at carbon atom 4 (loss of the ethylpropyl group) or 6 (loss of the isopropyl group); and splitting of the triazine ring (Knuesli et al. 1969; Reed 1982). The major atrazine metabolite in both soil and aquatic systems is hydroxyatrazine. In soils, it accounts for 5% to 25% of the atrazine originally applied after several months compared to 2% to 10% for all dealkylated products combined, including deethylated atrazine and deisopropylated atrazine (Stratton 1984; Schiavon 1988a,b). Atrazine may be converted to nonphytotoxic hydroxyatrazine by chemical hydrolysis, which does not require a biological system (Dao 1977; Wolf and Jackson 1982). Bacterial degradation, however, proceeds primarily by N-dealkylation (Giardi et al. 1985). In animals, N-dealkylation is a generally valid biochemical degradation mechanism (Knuesli et al. 1969). In rats, rabbits, and chickens, most atrazine is excreted within 72 hours; 19 urinary metabolites--including hydroxylated, N-dealkylated, oxidized, and conjugated metabolites--were found (Reed 1982). There is general agreement that atrazine degradation products are substantially less toxic than the parent compound and not normally present in the environment at levels inhibitory to algae, bacteria, plants, or animals (DeNoyelles et al. 1982; Reed 1982; Stratton 1984).

Residues of atrazine rapidly disappeared from a simulated Northern Prairie freshwater wetland microcosm during the first 4 days, primarily by way of adsorption onto organic sediments (Huckins et al. 1986). This is consistent with the findings of others who report 50% loss ($Tb\frac{1}{2}$) from freshwater in 3.2 days (Moorhead and Kosinski 1986), 82% loss in 5 days, and 95% loss in 55 days (Lay et al. 1984), although one report presents evidence of a 300-day half-life for atrazine (Yoo and Solomon 1981). In estuarine

Table 1. Chemical and other properties of atrazine (Anon. 1963; Hull 1967; Knuesli et al. 1969; Gunther and Gunther 1970; Reed 1982; Beste 1983; Hudson et al. 1984; Huber and Hock 1986; Huckins et al. 1986; EPA 1987).

Variable	Datum
Chemical name	2-chloro-4-ethylamino-6-isopropylamino-1,3,5-triazine.
Alternate names	CAS 1912-24-9, ENT 28244, G-30027, Aatrex, Aatrex 4L, Aatrex 4LC, Aatrex Nine-O, Aatrex 80W, Atranex, Atratol, Atratol 8P, Atratol 80W, Atrazine 4L, Atrazine 80W, Atred, Bicep 4.5L, Co-Op, Co-Op Atra-pril, Cristatrina, Crisazine, Farmco atrazine, Gesaprim, Griffex, Primatol A, Shell atrazine herbicide, Vectal, Vectal SC.
Primary uses	Selective herbicide for control of most annual broadleaf and grassy weeds in corn, sugar cane, sorghum, macadamia orchards, rangeland, pineapple, and turf grass sod. Nonselective herbicide for weed control on railroads, storage yards, along highways, and industrial sites. Sometimes used as selective weedicide in conifer reforestation, Christmas tree plantations, and grass seed fields.
Major producer	Ciba-Geigy Corporation
Application methods	Usually as water spray or in liquid fertilizers applied preemergence, but also may be applied preplant or postemergence. Rates of 2 to 4 pounds/acre (2.24 to 4.48 kg/ha) are effective for most situations; higher rates are used for nonselective weed control, and on high organic soils.
Compatibility with other pesticides	Compatible with most other pesticides and fertilizers when used at recommended rates. Sold in formulation with Lasso®, Ramrod®, and Bicep®.

Table 1.(Continued)

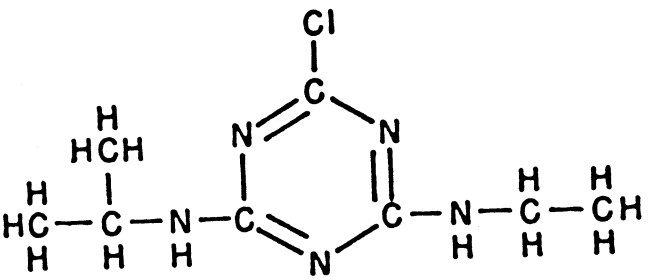
Variable	Datum
Stability	Very stable over several years of shelf life, under normal illumination and extreme temperatures. Stable in neutral, slightly acid or basic media. Sublimes at high temperatures and when heated, especially at high temperatures in acid or basic media, hydrolyzes to hydroxyatrazine (2-hydroxy-4-ethylamino-6-isopropylamino-s-triazine), which has no herbicidal activity.
Empirical formula	$C_8H_{14}ClN_5$
Structural formula	
Molecular weight	215.7
Melting point	173 °C to 175 °C
Vapor pressure	5.7×10^{-8} mm mercury at 10 °C, 3.0×10^{-7} at 20 °C, $1.4_5 \times 10^{-6}$ at 30 °C, and 2.3×10^{-5} at 50 °C.
Henry's Law constant	6.13×10^{-8} to 2.45×10^{-7} atm-m^3/mole
Physical state	The technical material is a white, crystalline, noncombustible, noncorrosive, substance.

Table 1. (Concluded)

Variable	Datum
Purity	No impurities or contaminants that resulted from the manufacturing process were detected.
Solubility	
Water	22 mg/l at 0 °C, 32 mg/l at 25 °C, 320 mg/l at 85 °C
N-pentane	360 mg/l at 27 °C
Petroleum ether	12,000 mg/l at 27 °C
Methanol	18,000 mg/l at 27 °C
Ethyl acetate	28,000 mg/l at 27 °C
Chloroform	52,000 mg/l at 27 °C
Dimethyl sulfoxide	183,000 mg/l at 27 °C
Log Kow	2.71

waters and sediments, atrazine is inactivated by adsorption and metabolism: half-time persistence in waters has been estimated to range between 3 and 30 days, being shorter at elevated salinities; for sediments this range was 15 to 35 days (Jones et al. 1982; Stevenson et al. 1982; Glotfelty et al. 1984; Isensee 1987). The comparatively rapid degradation of atrazine to hydroxyatrazine in estuarine sediments and water column indicates a low probability for atrazine accumulation in the estuary, and a relatively reduced rate of residual phytotoxicity in the estuary for the parent compound (Jones et al. 1982).

Atrazine is leached into the soil by rain or irrigation water. The extent of leaching is limited by the low water solubility of atrazine and by its adsorption onto certain soil constituents (Anon. 1963). Runoff loss in soils ranges from 1.2% to 18% of the total quantity of atrazine applied, but usually is less than 3% (Wolf and Jackson 1982). Surface runoff of atrazine from adjacent conventional tillage and no-tillage corn watersheds in Maryland was measured after single annual applications of 2.2 kg/ha for 4 years (Glenn and Angle 1987). Most of the atrazine in surface runoff was lost during the first rain after application. In 1979, the year of greatest precipitation, 1.6% of the atrazine applied moved from the conventional tillage compared to 1.1% from the no-tillage watershed, suggesting that no-tillage should be encouraged as an environmentally sound practice (Glenn and Angle 1987). Lateral and downward movement of atrazine was measured in cornfield soils to a depth of 30 cm when applied at 1.7 kg/ha to relatively moist soils; in lower elevation soils, atrazine accumulated by way of runoff and infiltration (Wu 1980). Downward movement of atrazine through the top 30 cm of cornfield soils indicates that carryover of atrazine to the next growing season is possible: between 5% and 13% of atrazine was available one year after application (Wu 1980; Wu and Fox 1980). Atrazine is not usually found below the upper 30 cm of soil in detectable quantities, even after years of continuous use; accordingly, groundwater contamination by atrazine is not expected at recommended application rates (Anon. 1963; Hammons 1977; Wolf and Jackson 1982; Beste 1983).

Atrazine persistence in soils is extremely variable: reported $Tb\frac{1}{2}$ values ranged from 20 to 100 days in some soils to 330 to 385 days in others (Jones et al. 1982); intermediate values were reported by Forney (1980), Stevenson et al. (1982), and Stratton (1984). Atrazine activity and persistence in soils is governed by many physical, chemical, and biological factors. In general, atrazine loss was more rapid under some conditions than others: it was more rapid from moist soils than from dry soils, during periods of high temperatures than during periods of low temperatures, from high organic and high clay content soils than from sandy mineral soils, during summer than in winter, from soils with high microbial densities than from those with low densities, from soils of acidic pH than from those of neutral or alkaline pH, during storm runoff events than during normal flows, at shallow soil depths than at deeper depths, and under conditions of increased ultraviolet irradiation (Anon. 1963; McCormick and Hiltbold 1966; Hull 1967; Gunther and Gunther 1970; Dao 1977; Hammons 1977; Frank and Sirons 1979; Forney 1980; Stevenson et al. 1982; Wolf and Jackson 1982; Beste 1983; EPA 1987). Microbial action, usually by way of N-dealkylation and hydrolysis to hydroxyatrazine, probably accounts for the major breakdown of atrazine in the soil, although nonbiological degradation pathways of volatilization, hydroxylation, dealkylation, and photodecomposition are also important (Hull 1967; Gunther and Gunther 1970; Reed 1982; Menzer and Nelson 1986).

BACKGROUND CONCENTRATIONS

Atrazine concentrations in human foods are negligible. Monitoring of domestic and imported foods in the human diet by the U.S. Food and Drug Administration between 1978 and 1982 showed that only 3 of 4,500 samples analyzed had detectable atrazine residues. Two samples in 1980 contained 0.01 and 0.08 mg atrazine/kg and one in 1978, following a known contamination incident, contained 47 mg/kg (Reed 1982).

Atrazine and its metabolites have been observed in freshwater streams contiguous to agricultural lands; 0.1% to 3% of the atrazine applied to the fields was lost to the aquatic environment (Jones et al. 1982). Atrazine concentrations in runoff waters from treated cornfields may exceed 740 ug/l (Table 2). Elevated levels were associated with high initial treatment rates, major storms shortly after application, conventional tillage practices (vs. no tillage), and increased flow rates, increased suspended solids, and increased dissolved nitrates and nitrites. Concentrations in runoff water usually declined rapidly within a few days (Forney 1980; Setzler 1980; Stevenson et al. 1982). Groundwater contamination by way of atrazine treatment of cornfields has been unexpectedly reported in parts of Colorado, Iowa, and Nebraska. Contamination was most pronounced in areas of highly permeable soils that overlie groundwater at shallow depths (Wilson et al. 1987).

The total amount of atrazine reaching the Wye River, Maryland, estuary depended on the quantity applied in the watershed and the timing of runoff. In years of significant runoff, 2% to 3% of the atrazine moved to the estuary within 2 weeks after application and effectively ceased after 6 weeks (Glotfelty et al. 1984). In Chesapeake Bay waters, a leakage rate of 1% of atrazine from agricultural soils resulted in aqueous concentrations averaging 17 ug/l--concentrations potentially harmful to a variety of estuarine plants (Jones et al. 1982). The maximum recorded atrazine concentration in runoff water entering Chesapeake Bay was 480 ug/l (Forney 1980). However, these concentrations seldom persisted for significant intervals, and only rarely approached those producing long-term effects on submerged aquatic vegetation (Glotfelty et al. 1984).

Atmospheric transport of atrazine-contaminated aerosol particulates, dusts, and soils may contribute significantly to atrazine burdens of terrestrial and aquatic ecosystems. The annual atmospheric input of atrazine in rainfall to the Rhode River, Maryland, as one example, was estimated at 1,016 mg/surface ha in 1977, and 97 mg/ha in 1978 (Wu 1981). A similar situation exists with fog water. When fog forms, exposed plant surfaces become saturated with liquid for the duration of the fog (Glotfelty et al. 1987).

Table 2. Atrazine concentrations in selected watersheds.

Locale and other variables	Concentration[a], in ug/l or ug/kg	Reference[b]
ATRAZINE-TREATED CORNFIELDS		
Iowa, shortly after application		
Runoff water	4,900	1
Sediments	7,350	1
Kansas, 1974		
Runoff water		
May	1,074	1
June	739	1
Soil from drainage canal	50	1
Water from drainage canal		
Summer	100	1
Winter	10	1
Ontario, Canada (1.7 kg/ha)		
Clay-dominated soils	Max. 25	2
Loam-dominated soils	Max. 14	2
Sand-dominated soils	Max. 4	2
STREAMWATER, Quebec		
Atrazine	(0.01 to 26.9)	3
Metabolites	(<0.01 to 1.3)	3
NORTHERN OHIO STREAMS, 1980		
Sandusky River Basin	(1.0 to 45.7)	4
Others	(0.1 to 23.2)	4
STREAMS ENTERING GREAT LAKES FROM CANADA		
To Lake Erie	4.0	2
To Lake Huron	1.4	2
To Lake Ontario	1.1	2

Table 2. (Continued)

Locale and other variables	Concentration[a], in ug/l or ug/kg	Reference[b]
SUSQUEHANNA DRAINAGE BASIN,		
Pennsylvania, 1980		
May	Max. 67.8	2
Other months	(1.1 to 2.5)	2
DRINKING WATER		
Colorado	Usually <1.8, Max. 2.3	5
Tiffin, Ohio, 1980		
May 30	16.4	4
June 16	7.2	4
June 26	5.3	4
July 1	3.3	4
FOG WATER, Beltsville, Maryland	(0.27 to 0.82)	6
CHESAPEAKE BAY WATERSHED		
Runoff water	Max. 480.0	1
Chesapeake Bay, 1980		
April	Max. 0.3	2
June	Max. 1.1	2
July	Max. 0.4	2
Chesapeake Bay tributaries		
Horn Point		
May-July, 1980	(0.1 to 18.3)	7
May, 1981	(0.7 to 46.0)	7
Choptank estuary		
May-July, 1980	(0.0 to 0.8)	7
May, 1981 (runoff event)	(0.2 to 9.3)	7
Wye River, Maryland	Usually <3.0 at peak loadings; Max. ~15.0	8

Table 2. (Concluded)

Locale and other variables	Concentration[a], in ug/l or ug/kg	Reference[b]
Rhode River, Maryland		
1977-1978		
Water column, depth ~0.3 m	.04 (0.003 to 0.19)	9
Microsurface layer	0.13 (0.01 to 3.3)	9
Rainwater, May	Max. 2.2	10

[a] Concentrations are shown as mean, range (in parentheses), and maximum (Max.).

[b] References: 1, Forney 1980; 2, Stevenson et al. 1982; 3, Frank and Sirons 1979; 4, Setzler 1980; 5, Wilson et al. 1987; 6, Glotfelty et al. 1987; 7, Kemp et al. 1985; 8, Glotfelty et al. 1984; 9, Lu et al. 1980; 10, Wu 1981.

LETHAL AND SUBLETHAL EFFECTS

GENERAL

In terrestrial ecosystems, atrazine effectively inhibits photosynthesis in target weeds, and may also affect certain sensitive crop plants. Atrazine metabolites are not as phytotoxic as the parent compound. Degradation is usually rapid, although atrazine may persist in soils for more than one growing season. Soil fauna may be adversely affected shortly after initial atrazine application at recommended levels, but long-term population effects on this group are considered negligible.

As discussed later, sensitive species of aquatic flora experience temporary adverse effects at concentrations as low as 1.0 to 5.0 ug/l; however, most authorities agree that potentially harmful levels, i.e., >10 ug/l for long periods, have not been documented and are probably unrealistic under current application protocols and degradation rates. The observed declines in submerged aquatic vegetation in the Chesapeake Bay are not now directly attributable to atrazine use. Atrazine indirectly affects aquatic fauna at concentrations of 20 ug/l and higher by reducing the food supply of herbivores and, to some extent, their macrophyte habitat. Direct adverse effects on growth and survival of aquatic fauna were evident in the range of 94 to 500 ug/l. Bioaccumulation of atrazine is limited and food chain biomagnification is negligible in aquatic ecosystems.

Birds show a low probability for atrazine uptake and accumulation. Known acute oral LD-50s and dietary LD-50s are high: >2,000 mg/kg body weight, and >5,000 mg/kg diet. Indirect ecosystem effects of atrazine on insect- and seed-eating birds are not known, and seem to merit study.

Data are lacking for mammalian wildlife, but tests with domestic livestock and small laboratory animals strongly indicate that this group is comparatively resistant to atrazine. Acute oral LD-50s are >1,750 mg/kg body weight, and no adverse effects are evident at dietary levels of 25 mg/kg food (about 1.25 mg/kg body weight) and sometimes 100 mg/kg food over extended periods.

TERRESTRIAL PLANTS AND INVERTEBRATES

Atrazine enters plants primarily by way of the roots and secondarily by way of the foliage, passively translocated in the xylem with the transpiration stream, and accumulates in the apical meristems and leaves (Hull 1967; Forney 1980; Reed 1982; Wolf and Jackson 1982). The main phytotoxic effect is the inhibition of photosynthesis by blocking the electron transport during Hill reaction of photosystem II. This blockage leads to inhibitory effects on the synthesis of carbohydrate, a reduction in the carbon pool, and a buildup of carbon dioxide within the leaf, which subsequently causes closure of the stomates, thus inhibiting transpiration (Stevenson et al. 1982; Jachetta et al. 1986; Shabana 1987).

Atrazine is readily metabolized by tolerant plants to hydroxyatrazine and amino acid conjugates. The hydroxyatrazine can be further degraded by dealkylation of the side chains and by hydrolysis of resulting amino groups on the ring and some carbon dioxide production (Hull 1967; Reed 1982; Beste 1983). Resistant plant species degrade atrazine before it interferes with photosynthesis. Corn, for example, has an enzyme (2,4-dihydroxy-7-methoxy-1, 4 [2H]-berzoxazin-3 [4H]-one) that degrades atrazine to nonphytotoxic hydroxyatrazine (Wu 1980; Stevenson et al. 1982). In sensitive plants, such as oats, cucumber, and alfalfa, which are unable to detoxify atrazine, the compound accumulates, causing chlorosis and death (Anon. 1963; Hull 1967). Corn and sorghum excrete about 50% of accumulated atrazine and metabolize the rest to insoluble residues that are indigestible to sheep (Ovis aries) and rats (Rattus sp.). These results strongly suggest that the final disposition of atrazine metabolites does not occur in either plants or animals, but ultimately through microbial breakdown (Bakke et al. 1972b).

Long-term applications of atrazine for weed control in corn result in degradation products, mainly hydroxylated analogues, that may persist in soil for at least 12 months after the final herbicide application, and may enter food crops planted in atrazine-treated soil in the years after cessation of long-term treatment (Frank and Sirons 1979; Kulshrestha et al. 1982). In one example, atrazine was applied to a corn field for 20 consecutive years at rates of 1.4 to 2.2 kg/ha (Khan and Saidak 1981). Soils collected 12 months after the last application contained atrazine (55 ug/kg dry weight), hydroxyatrazine (296 ug/kg), and various mono dealkylated hydroxy analogues (deethylatrazine at 14 ug/kg, deethylhydroxyatrazine at 17 ug/kg, and deisopropylhydroxyatrazine at 23 ug/kg). Oat (Avena sativa) seedlings grown in this field contained hydroxyatrazine (64 to 73 ug/kg fresh weight) and deisopropylhydroxyatrazine (84 to 116 ug/kg); similar results were obtained with timothy, Phleum pratense (Khan and Saidak 1981). In areas with a relatively long growing season, a double cropping of soybeans (Glycine max)--planted after corn is harvested for silage or grain--is gaining acceptance. Under conditions of warm weather, relatively high rainfall, and sandy soils, soybeans can be safely planted after corn (14 to 20 weeks after atrazine

application) when rates of atrazine normally recommended for annual weed control (1.12 to 4.48 kg/ha) are used (Brecke et al. 1981).

Seed germination of sensitive species of plants was reduced by 50% at soil atrazine concentrations between 0.02 and 0.11 mg/kg (Table 3). Mustard (Brassica juncea) was especially sensitive, and died shortly after germination. Soil atrazine residues of this magnitude were typical of those remaining at the beginning of a new growing season following corn in sandy loam under tropical conditions (Kulshrestha et al. 1982). Reduction in seed germination was also noted at soil atrazine concentrations of 0.25 to 0.46 mg/kg for the lentil Lens esculenta, the pea Pisum sativum, and the gram Cicer arietinum (Kulshrestha et al. 1982). Many species of mature range grasses are tolerant of atrazine but are susceptible as seedlings; seedlings of the most sensitive three species of eight tested were adversely affected in soils containing 1.1 mg atrazine/kg (Bahler et al. 1984; Table 3).

Soil fungi and bacteria accumulated atrazine from their physicochemical environment by factors between 87X and 132X (Wolf and Jackson 1982), probably through passive adsorption mechanisms. Atrazine stimulated the growth of at least two common species of fungal saprophytes known to produce antibiotics: Epicoccum nigrum and Trichoderma viride (Richardson 1970). Trichoderma, for example, grew rapidly at all treatments tested (up to 80 mg/kg soil) and showed optimal growth 3 to 10 days postinoculation (Rodriguez-Kabana et al. 1968). Atrazine suppressed the growth of various species of soil fungi, including Rhizoctonia solani, Sclerotium rolfsii, and Fusarium spp., and stimulated the growth of other species known to be antagonistic to Fusarium. This selectivity is likely to induce a shift in the fungal population of atrazine-treated soil that would be either harmful or beneficial to subsequent crops, depending on whether saprophytic or pathogenic fungi attained dominance (Richardson 1970).

At 2.5 mg atrazine/kg soil, equivalent to 2 kg/ha in the top 10 cm, field and laboratory studies demonstrated that mortality in arthropod collembolids (Onchiurus apuanicus) was 47% in 60 days; however, fecundity was not affected at dose levels up to 5.0 mg/kg soil. It was concluded that atrazine applications at recommended treatment levels had negligible long-term population effects on sensitive species of soil fauna (Mola et al. 1987). At 5 or 8 kg atrazine/ha, all species of soil fauna tested, except some species of nematodes, were adversely affected (Popovici et al. 1977). One month postapplication, population reductions of 65% to 91% were recorded in protozoa, mites, various insect groups, and collembolids at 5 kg/ha; after 4 months, populations were still depressed by 55% to 78% (Popovici et al. 1977). At 9 kg atrazine/ha, soil faunal populations of beetles, collembolids, and earthworms remained depressed for at least 14 months after initial treatment (Mola et al. 1987).

Table 3. Atrazine effects on selected species of terrestrial plants.

Species, dose, and other variables	Effect and reference
Soil alga, *Chlorella vulgaris*	
0.1 and 0.5 ug/l soil water	Chlorophyll production stimulated (Torres and O'Flaherty 1976).
1.0 ug/l and higher	Chlorophyll production inhibited; more-than-additive toxicity observed in combination with simazine and malathion (Torres and O'Flaherty 1976).
Mustard, *Brassica juncea*	
20 ug/kg dry weight soil	Seed germination reduced 50%; death shortly thereafter (Kulshrestha et al. 1982).
Cyanobacteria, 4 species, isolated from rice-cultivated soils in Egypt	
50 ug/l soil water for 7 days	Suppressed pigment biosynthesis in *Aulosira fertissima* and *Tolypothrix tenuis*, reduced growth in *Anabaena oryzae* and *Nostoc muscorum*, and reduced carbohydrate content in *Nostoc* and *Tolypothrix* (Shabana 1987).
100 to 500 ug/l soil water for 7 days	All variables affected in all species (Shabana 1987).
Barley, *Hordeum vulgare*	
50 ug/kg dry weight soil	Seed germination reduced 50% (Kulshrestha et al. 1982).
Oat, *Avena sativa*	
70 ug/kg dry weight soil	Seed germination reduced 50% (Kulshrestha et al. 1982).
Wheat, *Triticum aestivum*	
110 ug/kg dry weight soil	Seed germination reduced 50% (Kulshrestha et al. 1982).
0.6 kg/ha	Effectively controls weeds in wet sandy soils; some damage to crop possible in dry clay soils (Amor et al. 1987)

Table 3. (Concluded)

Species, dose, and other variables	Effect and reference
Range grasses, four species, seedlings	
1.1 mg/kg soil	Survival reduced, and growth reduced in surviving seedlings (Bahler et al. 1984).
Weed, Chenopodium album, seedlings from French garden never treated with chemicals	
0.5 kg/ha	Survival 12%; progeny of these survivors were resistant to 1 kg/ha treatment (Bettini et al. 1987).
1.0 kg/ha	Fatal to 100% (Bettini et al. 1987).
Corn, Zea mays	
1.25 kg/ha	No effect on growth or yield (Malan et al. 1987).
5.0 kg/ha	Severe phytotoxicity 25 to 30 days after planting; growth inhibition during early development. Recovery, with no negative effect on final yield (Malan et al. 1987).
Soybean, Glycine max, planted after corn, Zea mays	
2.24 kg/ha	No effect on yield when planted at least 8 weeks after atrazine application (Brecke et al. 1981).
4.48 kg/ha	At least 10-week interval required after atrazine application for successful germination (Brecke et al. 1981).

AQUATIC PLANTS

Since the mid-1960's, seagrasses and freshwater submersed vascular plants have declined in many aquatic systems, especially Chesapeake Bay (Forney and Davis 1981; Stevenson et al. 1982; Kemp et al. 1983; Cunningham et al. 1984). These plants provide food and habitat to diverse and abundant animal populations. In Chesapeake Bay, this decline has been associated with an overall decline in the abundance of fish and wildlife, and has been interpreted as an indication of serious disturbance in the ecological balance of the estuary. More than 10 native species of submerged aquatic plants in Chesapeake Bay have decreased in abundance. In the upper estuary, this decline was preceded by an invasion of Eurasian watermilfoil (Myriophyllum spicatum), which eventually also died back (Kemp et al. 1983). Runoff of herbicides, including atrazine, from treated agricultural lands has been suggested as a possible factor involved in the disappearance of Chesapeake Bay submerged vegetation. During the past 20 years, the most widely used herbicide in the Chesapeake Bay watershed--and in the surrounding coastal plain--has been atrazine. Since its introduction into the region in the early 1960's, atrazine use has grown to about 200,000 kg annually in Maryland coastal communities alone (Kemp et al. 1983). Potentially phytotoxic concentrations of atrazine would be expected in estuaries with the following characteristics (which seem to apply in most of upper Chesapeake Bay): immediately adjacent to cornfields in the watershed; rains occur shortly after atrazine application; clay soils in fields producing more rapid runoff; soils with circumneutral pH and relatively low organic content; and large estuarine areas of low salinity and poor mixing (Stevenson et al. 1982).

At this time, most authorities agree that atrazine could induce some loss in aquatic vegetation but was not likely to have been involved in the overall decline of submerged plants in Chesapeake Bay (Forney 1980; Plumley and Davis 1980; Forney and Davis 1981; Kemp et al. 1983, 1985; Jones et al. 1986), and that nutrient enrichment and increased turbidity probably played major roles (Kemp et al. 1983, 1985). In the open waters of Chesapeake Bay, atrazine concentrations have rarely exceeded 1 ug/l; in major tributaries, such as the Choptank and Rappahanock Rivers, concentrations of 5 ug/l may occur after a major spring runoff. These runoffs sometimes generate transient, 2- to 6-hour concentrations up to about 40 ug/l in secondary tributaries (Kemp et al. 1983). In some small coves on the Chesapeake Bay, submerged plants may be exposed periodically to atrazine concentrations of 5 to 50 ug/l for brief periods during runoffs; however, dilution, adsorption, and degradation tend to reduce concentrations in the water phase to <5 ug/l within 6 to 24 hours (Jones et al. 1986). Since atrazine degrades rapidly in estuarine conditions ($Tb\frac{1}{2}$ 1 to 6 weeks), concentrations of atrazine on suspended and deposited estuarine sediments were seldom >5 ug/kg, suggesting little potential for accumulation (Kemp et al. 1983). The photosynthesis of redheadgrass (Potamogeton perfoliatus) was significantly inhibited by atrazine concentrations of 10 to 50 ug/l; however, it returned to normal levels within

1 hour after atrazine was removed (Jones et al. 1986). Recovery of redheadgrass within several weeks has also been documented after exposure to 130 ug/l for 4 weeks (Cunningham et al. 1984). In Chesapeake Bay, potential long-term exposure of submersed aquatic plants to concentrations of atrazine in excess of 10 ug/l is doubtful; therefore, any observed reductions in photosynthesis by these plants under such conditions would be minor and reversible (Jones et al. 1986).

Some authorities, however, suggest that the effects of atrazine on aquatic plants may be substantial. For example, atrazine concentrations between 1 and 5 ug/l adversely affect phytoplankton growth and succession; this, in turn, can adversely affect higher levels of the food chain, beginning with the zooplankton (DeNoyelles et al. 1982). Also, exposure to environmentally realistic concentrations of 3.2 to 12 ug atrazine/l for about 7 weeks was demonstrably harmful to wildcelery (Vallisneria americana), a submersed vascular plant in Chesapeake Bay (Correll and Wu 1982). At higher concentrations of 13 to 1,104 ug/l for 3 to 6 weeks, growth of representative submerged macrophytes in Chesapeake Bay was significantly depressed, and longer exposures were fatal to most species (Forney 1980). Atrazine concentrations of 100 ug/l reportedly cause permanent changes in algal community structure after exposure for 14 days, including decreased density and diversity, altered species composition, and reduced growth (Hamala and Kollig 1985). It seems that additional research is needed on the role of atrazine and on its interactions with other agricultural chemicals in regard to observed declines in submerged plants. It is emphasized that degradation products of atrazine did not play a role in the disappearance of the submerged vascular plants from the Chesapeake Bay. For example, 500 ug/l of deethylated atrazine was needed to produce 20% to 40% photosynthetic inhibition in four major species of submerged macrophytes in 2 hours, but only 95 ug/l of the parent atrazine caused 50% inhibition in a similar period (Jones and Winchell 1984).

Many studies have been conducted on the effects of atrazine on various species of aquatic flora under controlled conditions (Table 4). At concentrations of 1 to 5 ug/l, and exposure periods of 5 minutes to 7 weeks, documented adverse effects in sensitive species included inhibition in photosynthesis, growth, and oxygen evolution (Table 4). Higher concentrations were associated with altered species composition, reduced carbon uptake, reduced reproduction, high accumulations of atrazine, decreased chlorophyll a production, ultrastructural changes on chloroplasts, and death (Table 4). Phytotoxic effects were significantly increased at elevated levels of incident illumination, elevated water temperatures, decreased water pH, decreased dissolved oxygen concentrations, decreased nutrient content, and at increasing atrazine concentrations in the water column (Forney and Davis 1981; Karlander et al. 1983; Jones and Estes 1984; Mallanchuk and Kollig 1985; Mayasich et al. 1986). Phytotoxicity was not significantly influenced by atrazine concentrations in the sediments or hydrosoils, or by the salinity of the medium (Forney 1980; Forney and Davis 1981; Jones and Estes 1984; Huckins et

Table 4. Atrazine effects on selected species of aquatic plants.

Species, dose, and other variables	Effect and reference
Phytoplankton communities in experimental microcosms	
0.5 to 5.0 ug/l, 39 weeks	No measurable adverse effects (Brockway et al. 1984).
1.0 to 5.0 ug/l, several days	Reduced photosynthesis in sensitive species (DeNoyelles et al. 1982).
>17.9 ug/l, 21 days	Decreased oxygen production, decreased content of calcium and magnesium (Pratt et al. 1988).
20 ug/l, 20 days	Altered species composition (DeNoyelles and Kettle 1985).
20 ug/l, 136 days	Reduced growth, altered succession; atrazine resistant species now dominant (DeNoyelles et al. 1982).
50 ug/l, 12 days	Oxygen production decreased 20% to 30% (Brockway et al. 1984).
100 ug/l, 14 days	Algal densities and biomass reduced, diversity decreased, and species composition altered. Within 16 days after removal of atrazine stress, net productivity was indistinguishable from controls, but community structure remained altered at day 21 (Hamala and Kollig 1985).
100 ug/l, 20 days	Carbon uptake reduced >40% (DeNoyelles and Kettle 1985).
500 ug/l, 53 days	Immediate decline in primary productivity and community metabolism; no recovery (Stay et al. 1985).

Table 4. (Continued)

Species, dose, and other variables	Effect and reference
5,000 ug/l, 12 days	Death (Brockway et al. 1984).
Alga, Cyclotella meneghiniana	
1.0 ug/l, 5 minutes	Some inhibition in oxygen evolution (Millie and Hersh 1987).
99 to 243 ug/l, 5 minutes	Oxygen evolution reduced 50% (Millie and Hersh 1987).
500 ug/l, 5 minutes	Oxygen evolution 100% inhibited (Millie and Hersh 1987).
Wildcelery, Vallisneria americana	
1.3 ug/l, 47 days	No measurable effect (Correll and Wu 1982).
3.2 ug/l, 49 days	Some reduction in leaf area (Correll and Wu 1982).
12 ug/l, 47 days	LC-50; reduced reproduction and leaf area in survivors (Correll and Wu 1982).
75 ug/l, 12 to 28 days	Inhibited photosynthesis (Correll and Wu 1982).
100 ug/l, 6 weeks	Growth inhibited 29% (Forney and Davis 1981).
120 ug/l, 30 days	LC-100 (Correll and Wu 1982).
163 ug/l, 21 to 42 days	Growth inhibition of 50% (Forney 1980).
320 ug/l, 6 weeks	Growth inhibited 36% (Forney and Davis 1981).

Table 4. (Continued)

Species, dose, and other variables	Effect and reference
Elodea, Elodea canadensis	
3.2 ug/l, 3 to 4 weeks	Growth inhibited 1% (Stevenson et al. 1982).
13 ug/l, 21 to 42 days	Growth inhibited 50% (Forney 1980).
32 ug/l, 3 to 4 weeks	Growth inhibited 15% to 39% (Forney and Davis 1981).
100 ug/l, 3 to 4 weeks	Growth inhibited 53% (Forney and Davis 1981).
Redheadgrass, Potamogeton perfoliatus	
4 ug/l, 4 weeks	Photosynthesis reduced 10% (Kemp et al. 1985).
10 ug/l, 3 weeks	Growth inhibited 15% (Forney and Davis 1981).
50 ug/l, 2 hours	Equilibrium reached within 15 minutes, maximum residues of 3.5 mg/kg dry weight (Jones et al. 1986).
55 ug/l, 4 weeks	Photosynthesis reduced 50% (Kemp et al. 1985; Larsen et al. 1986).
80 ug/l, 2 hours	Photosynthesis inhibited 50% (Jones et al. 1986).
100 ug/l, 2 hours	Photosynthesis inhibition and residues of about 9.0 mg/kg dry weight; recovery rapid in atrazine-free medium but some photosynthetic depression for up to 77 hours (Jones et al. 1986).

Table 4. (Continued)

Species, dose, and other variables	Effect and reference
100 ug/l, 4 weeks	Photosynthesis inhibition; water levels of 87 ug atrazine/l at 4 weeks; recovery in 2 to 3 weeks in atrazine-free medium (Kemp et al. 1985).
130 ug/l, 4 weeks	Decreased oxygen production immediately on exposure; significant recovery within 2 weeks despite constant atrazine concentrations (Cunningham et al. 1984).
320 ug/l, 3 weeks	Growth inhibited 45% to 54% (Forney and Davis 1981).
450 to 650 ug/l, 2 hours	Photosynthesis inhibited 87%; residues of about 5 mg/kg dry weight (Jones et al. 1986).
474 ug/l, 21 to 42 days	Growth reduced 50% (Forney 1980).
1,200 ug/l, 4 weeks	Pronounced phytotoxic effects; no recovery (Cunningham et al. 1984).
Eurasian watermilfoil, Myriophyllum spicatum	
5 ug/l, 4 weeks	Enhanced oxygen production (Kemp et al. 1985).
11 ug/l, 4 weeks	Photosynthesis reduced 1% (Kemp et al. 1985).
50 ug/l, 4 weeks	Oxygen production depressed (Kemp et al. 1985).
117 ug/l, 4 weeks	Photosynthesis reduced 50% (Kemp et al. 1985; Larsen et al. 1986).
320 ug/l, 4 weeks	Growth inhibited 22% (Forney and Davis 1981).

Table 4. (Continued)

Species, dose, and other variables	Effect and reference
1,000 ug/l, 4 weeks	Growth inhibited 62% (Forney and Davis 1981).
1,000 ug/l, 4 weeks	Residues <1 ug/kg (Kemp et al. 1985).
1,104 ug/l, 21 to 42 days	Growth inhibited 50% (Forney 1980).
Common cordgrass, *Spartina alterniflora*	
10 ug/l, 3 to 4 weeks	Biomass reduction of 6% (Stevenson et al. 1982).
100 ug/l, 3 to 4 weeks	Biomass reduction of 34% (Stevenson et al. 1982).
1,000 ug/l, 3 to 4 weeks	Biomass reduction of 46% (Stevenson et al. 1982).
Shoal grass, *Halodule wrightii*	
10, 40, or 120 ug/l, 22 days	Enhanced growth when compared to controls (Mitchell 1985).
420 ug/l, 22 days	Above ground biomass reduced 26% (Mitchell 1985).
1,490 ug/l, 22 days	Above ground biomass reduced 45% compared to controls (Mitchell 1985).
Marine alga, *Nannachloris oculata*	
15 ug/l, 7 days	Growth reduction (Mayasich et al. 1987).
50 ug/l, 72 hours	Some growth inhibition; inhibition greatest under conditions of elevated temperature and illumination (Karlander et al. 1983).

Table 4. (Continued)

Species, dose, and other variables	Effect and reference
Algae and macrophytes, various species	
20 ug/l, 6 weeks	Bioconcentration factors up to 32X (Huckins et al. 1986).
Submerged aquatic macrophytes, 4 species: Potamogeton sp., Ruppia sp., Myriophyllum sp., Zannichellia sp.	
20 ug/l, 2 hours	Photosynthesis inhibition of about 1% (Jones and Winchell 1984).
95 ug/l, 2 hours	Photosynthesis inhibition 50%; atrazine significantly more effective than deethylated atrazine, deisopropylated atrazine, and hydroxyatrazine, in that order, in effecting inhibition (Jones and Winchell 1984).
Algae, various species	
22 ug/l, 7 days	No effect on photosynthesis rate, chlorophyll content, or cell numbers (Plumley and Davis 1980).
37 to 308 ug/l, 24 hours	Carbon uptake reduced 50% (Larsen et al. 1986).
60 to 100 ug/l, 72 hours	Growth inhibited 50% in seven species (Mayer 1987).
60 to 460 ug/l, 1 hour	Oxygen evolution inhibited 50% in 18 species (Hollister and Walsh 1973).
77 to 102 ug/l, 24 hours	Photosynthesis reduction of 50% (Larsen et al. 1986).
80 to 907 ug/l, 3 weeks	Growth inhibited 50% (Larsen et al. 1986).

Table 4. (Continued)

Species, dose, and other variables	Effect and reference
100 ug/l, 2 hours	Growth inhibited 50% in three species (Mayer 1987).
100 ug/l, 3 days	Reduced productivity; complete recovery by day 7 (Moorhead and Kosinski 1986).
100 to 300 ug/l, 10 days	Growth inhibited 50% in four species (Mayer 1987).
100 to 460 ug/l, 72 hours	Growth inhibited 50% in eight species (Mayer 1987).
220 ug/l, 7 days	Reduced photosynthesis; no effect on chlorophyll production and cell division rate in three estuarine species (Plumley and Davis 1980).
Algae, Chlorella spp.	
54 ug/l, 10 days	Growth reduction of 30% (Gonzalez-Murua et al. 1985).
200 ug/l, 48 hours	Photosynthesis reduced 30%, but no effect on growth (Lay et al. 1984).
250 ug/l, 7 days	Growth reduction; 90% of atrazine passively accumulated within 1 hour (Veber et al. 1981).
Submersed vascular plant, Zannichellia palustris	
75 ug/l, 21 to 42 days	Photosynthesis inhibition (Correll and Wu 1982).
Submersed vascular plant, Potamogeton pectinatus	
75 ug/l, 21 to 42 days	Photosynthesis stimulation (Correll and Wu 1982).

Table 4. (Concluded)

Species, dose, and other variables	Effect and reference
650 ug/l, 21 to 42 days	Photosynthesis inhibition (Correll and Wu 1982).
Submersed vascular plant, Zostera marina	
75 ug/l, 21 to 42 days	Photosynthesis stimulation (Correll and Wu 1982).
650 ug/l, 21 to 42 days	Photosynthesis inhibition (Correll and Wu 1982).
Periphyton communities in freshwater enclosures	
80 to 1,560 ug/l, 10 months	Declines in net production, cell numbers, biomass, number of taxa, and chlorophyll activity; larger algal species (Mougeotia, Oedogonium, Tolypothrix, Epithemia) were the most sensitive. At higher concentrations, population shifted from a chlorophyte-dominated to a diatom-dominated community (Hamilton et al. 1987).
100 ug/l, 2 treatments, 6 weeks apart	After initial application, all blue green algae disappeared and organic matter significantly decreased. Within 3 weeks of second treatment, a 36% to 67% reduction in organic matter, chlorophyll, algal biomass, and rate of carbon assimilation was measured. Some species of green algae decreased in abundance, but others increased (Herman et al. 1986).
Duckweed, Lemna minor	
250 ug/l, 15 days	Ultrastructural changes on chloroplasts of mesophyll cells; no effect on chlorophyll and lipid distribution (Beaumont et al. 1980; Grenier et al. 1987).

al. 1986).

Atrazine was 4 to 10 times more effective than its degradation products in producing growth reduction, photosynthesis inhibition, and acetylene-reducing ability in two species of green algae, Chlorella pyrenoidosa, Scenedesmus quadricauda, and three species of cyanobacteria, Anabaena spp. (Stratton 1984). Atrazine reduced growth 50% at 0.03 to 5.0 mg/l and inhibited photosynthesis 50% at 0.1 to 0.5 mg/l. Comparable values for deethylated atrazine were 1.0 to 8.5 mg/l for growth reduction, and 0.7 to 4.8 mg/l for photosynthesis inhibition; for deisopropylated atrazine, these values were 2.5 to >10 mg/l for growth reduction and 3.6 to 9.3 mg/l for photosynthesis inhibition; hydroxyatrazine and diaminoatrazine were nontoxic to most cultures tested (Stratton 1984). Smooth cordgrass (Spartina alterniflora), the major emergent species in North American salt marshes, is only slightly affected by relatively high levels of atrazine, due possibly to its ability to metabolize this compound (Davis et al. 1979; Forney and Davis 1981; Stevenson et al. 1982). Studies with radiolabled atrazine and Spartina roots were conducted during 2-day exposures, followed by 28 days in atrazine-free solution (Pillai et al. 1977; Weete et al. 1980). After 2 days, 90% of the atrazine had translocated to the shoots. Atrazine was readily metabolized to chloroform-soluble substances, then to water-soluble substances, and finally to insoluble substances. Atrazine in the chloroform-soluble fraction decreased from 85% to 24% by day 28; the aqueous fraction contained 15% at the start and 60% at day 28. The basis of Spartina resistance is due primarily to its ability to convert atrazine to N-dealkylation products, such as 2-chloro-4-amino-6-isopropylamino-s-triazine. However, at least 14 water-soluble metabolites were isolated; about half contained the fully alkylated triazine rings, and most of the others had the 4-amino-6-isopropylamino derivative. Acid hydrolysates of the metabolites contained small amounts of amino acids, suggesting that a conjugation pathway, in addition to N-deakylation, may be operative in Spartina.

Estuarine fungi contribute substantially to plant detritus due to their abundance and degradative potential. Fungi are known to accumulate soluble atrazine from seawater through sorption, and release up to 2.2% as hydroxyatrazine and other atrazine metabolites; another 4.6% is more tightly associated and less available to the external environment. The combined processes result in atrazine accumulation, and may contribute to its transport and redistribution through the estuary (Schocken and Speedie 1982, 1984).

AQUATIC FAUNA

A marine copepod (Acartia tonsa) was the most sensitive aquatic animal tested against direct effects of atrazine, having a 96-hour LC-50 of 94 ug/l (Table 5). Adverse effect levels to selected species of aquatic invertebrates

Table 5. Lethal and sublethal effects of atrazine on selected species of aquatic fauna. Concentrations listed are in micrograms of atrazine per liter of medium.

Ecosystem, organism, and other variables	Concentration (ug/l)	Effect	Reference[a]
FRESHWATER INVERTEBRATES			
Midge, Chirononmus riparius Adults	20	Whole body residue of 160 ug/kg in 6 weeks	1
Larvae	20	Whole body residue of 569 ug/kg in 6 weeks	1
Cladoceran, Daphnia magna	20	After 6 weeks, whole body residue of 300 ug/kg	1
D. magna	200	Exposure for six generations. Number of young per female in 21 days did not differ from controls in generations 1, 2, and 3, but significant reduction measured in generations 4, 5, and 6.	2
D. magna	6,900 (5,200 to 8,100)	LC-50 (48 hours).	3
Scud, Gammarus fasciatus	60 to 140	MATC[b]	3
G. fasciatus	>2,400	Some deaths in 48 hours.	3
G. fasciatus	5,700 (3,600 to 8,000)	LC-50 (48 hours).	3
Midge, Chironomus tentans	110 to 230	MATC[b]	3
C. tentans	500	Some deaths in 48 hours.	3
C. tentans	720 (360 to 1,440)	LC-50 (48 hours).	3

Table 5. (Continued)

Ecosystem, organism, and other variables	Concentration (ug/l)	Effect	Reference[a]
Leeches, two species (Glossiphonia complanata, Helobdella stagnalis)	<1,000	Adverse effects on growth, food intake, and egg production.	11
Leeches, two species	6,300 to 9,900	LC-50 (28 days).	11
Leeches, two species	16,000	No deaths in 96 hours.	11
FRESHWATER FISHES			
Brook trout, Salvelinus fontinalis	60 to 120	MATC[b]	3
S. fontinalis	450	Reduced incubation time of developing embryos.	3
S. fontinalis	740	After 44 weeks, concentration in muscle <0.2 mg/kg fresh weight.	3
S. fontinalis	6,300 (4,100 to 9,700)	LC-50 (8 days)	3
Bluegill, Lepomis macrochirus	90 to 500	MATC[b]	3
L. macrochirus	94	After 78 weeks, concentration in muscle <0.2 mg/kg fresh weight.	3
L. macrochirus	500	At 28 days, fish were lethargic and ate poorly, and swam erratically.	3
L. macrochirus	6,700 (5,400 to 8,400)	LC-50 (7 days).	3

Table 5. (Continued)

Ecosystem, organism, and other variables	Concentration (ug/l)	Effect	Reference[a]
L. macrochirus	8,000 to 42,000	LC-50 (96 hours).	3,4,5,6
L. macrochirus	46,000	LC-50 (24 hours)	6
Fathead minnow, Pimephales promelas	210	After 43 weeks, concentration in eviscerated carcass was <1.7 mg/kg fresh weight.	3
P. promelas	210 to 520	MATC[b]	3
P. promelas, fry	520	LC-25 (96 hours).	3
P. promelas	15,000 (11,000 to 20,000)	LC-50 (8 days).	3
Rainbow trout, Salmo gairdneri	4,500 to 24,000	LC-50 (96 hours)	4,6
MARINE INVERTEBRATES			
Mysid shrimp, Mysidopsis bahia	80 to 190	MATC[b]	7
M. bahia	1,000 (650 to 3,100)	LC-50 (96 hours).	7
Copepod, Acartia tonsa	94 (52 to 167)	LC-50 (96 hours).	7

Table 5. (Continued)

Ecosystem, organism, and other variables	Concentration (ug/l)	Effect	Reference[a]
Brown shrimp, Penaeus aztecus	1,000	50% immobilized in 48 hours.	8
American oyster, Crassostrea virginica,	1,000	No effect on survival or growth.	9
C. virginica	>1,000	Growth reduced 50% in 96 hours.	8
C. virginica	>30,000	No effect on development in 48 hours.	7
"Shrimp"	1,000	LC-30 (96 hours).	9
Pink shrimp, Penaeus duorarum	6,900	LC-50 (96 hours).	7
Grass shrimp, Palaemonetes pugio	9,000	LC-50 (96 hours).	7
Fiddler crab, Uca pugilator	>29,000	LC-50 (96 hours).	7
Fiddler crab, Uca pugnax	100,000	Interfered with escape resonse when exposed in August; negligible effects in November; young males most sensitive.	10
U. pugnax	1,000,000 to 10,000,000	Reduced survival after 10 weeks.	10

Table 5. (Concluded)

Ecosystem, organism, and other variables	Concentration (ug/l)	Effect	Reference[a]
Mud crab, *Neopanope texana*	750,000	No deaths in 96 hours.	9
N. texana	1,000,000	LC-50 (96 hours).	9
MARINE FISHES			
Sheepshead minnow, *Cyprinodon variegatus*	1,900 to 3,400	MATC[b]	7
C. variegatus	>16,000	LC-50 (96 hours).	7
Spot, *Leiostomus xanthurus*	8,500	LC-50 (96 hours).	7

[a]References: 1, Huckins et al. 1986; 2, Kaushik et al. 1985; 3, Macek et al. 1976; 4, Beste 1983; 5, Klaasen and Kadoum 1979; 6, Mayer and Ellersieck 1986; 7, Ward and Ballantine 1985; 8, Mayer 1987; 9, Stevenson et al. 1982; 10, Plumley et al. 1980; 11, Streit and Peter 1978.

[b] Maximum acceptable toxicant concentration. Lower value in each pair indicates highest concentration tested producing no measurable effect on growth, survival, reproduction, or metabolism during chronic exposure; higher value indicates lowest concentration tested producing a measurable effect.

and fishes ranged from 120 ug/l to 500 ug/l, based on lifetime exposure studies (Table 5). The most sensitive criterion measured during long-term chronic exposure varied among species. Among freshwater invertebrates, for example, the most useful criterion was survival for Gammarus, the number of young produced for Daphnia, and developmental retardation for Chironomus (Macek et al. 1976).

Ambient concentrations as low as 20 ug atrazine/l have been associated with adverse effects on freshwater aquatic fauna, including benthic insects (Dewey 1986) and teleosts (Kettle et al. 1987), although effects were considered indirect. For example, the abundance of emerging chironomids (Labrundinia pilosella), and other aquatic insects declined at 20 ug atrazine/l (Dewey 1986). Richness of benthic insect species and total emergence declined significantly with atrazine addition. The effects were primarily indirect, presumably by way of reduction in food supply of nonpredatory insects, and to some extent their macrophyte habitat. Dietary habits and reproductive success were negatively affected in three species of fish after exposure for 136 days in ponds containing 20 ug atrazine/l (Kettle et al. 1987). About 70% of the original concentration applied was present in water at the end of the study. The reproduction of channel catfish (Ictalurus punctatus) and gizzard shad (Dorosoma cepedianum) failed, and that of bluegills, as measured by number of young per pond, was reduced more than 95%. Also, the number of prey items in the stomachs of bluegills were significantly higher in control ponds (25.6) than in a treated pond (3.8), and number of taxa represented were significantly greater. Macrophyte communities in treated ponds were reduced more than 60% in 2 months. The authors concluded that the effects of atrazine on bluegills were probably indirect, and that the reduction of macrophytes that had provided habitat for food items led to impoverished diets and more cannibalism by adult bluegills (Kettle et al. 1987).

Bioaccumulation of atrazine from freshwater is limited, and food chain biomagnification is negligible. In a farm pond treated once with 300 ug atrazine/l, residues at 120 days posttreatment ranged between 204 and 286 ug/kg in mud and water, and from not detectable in bullfrog (Rana catesbeiana) tadpoles to 290 ug/kg (all fresh weights) in whole bluegills; values were intermediate in zooplankton and clams. No residues were detectable in biological components one year posttreatment, when residues were <21 ug/kg in water and mud (Klaasen and Kadoum 1979). In a laboratory stream treated four times with 25 ug atrazine/l for 30 days, followed by depuration for 60 days, maximum accumulation factors ranged from about 4X in annelids to 480X in mayfly nymphs; however, residue concentrations declined to posttreatment levels within a few days after depuration began. Maximum atrazine concentrations recorded, in mg/kg whole organism fresh weight, were 0.2 in the clam Strophitis rugosus, 0.4 in the snail Physa sp., 0.9 in crayfish, Orconectes sp., 2.4 in the mottled sculpin Cottus bairdi, 3.0 in the amphipod Gammarus pseudolimnaeus, and 3.4 in mayflies, Baetis sp. (Lynch et al. 1982). In studies with the freshwater snail Ancylus fluviatilis and fry of the

whitefish Coregonus fera, atrazine was rapidly accumulated from the medium by both species and saturation was reached within 12 to 24 hours; bioconcentration factors were 4X to 5X at ambient water concentrations of 50 to 250 ug atrazine/l (Gunkel and Streit 1980; Gunkel 1981). Elimination of atrazine was rapid: 8 to 62 minutes for Coregonus, and 18 minutes for Ancylus. No accumulation of atrazine was recorded in molluscs, leeches, cladocerans, or fish when contamination was by way of the diet (Gunkel and Streit 1980; Gunkel 1981). Atrazine accumulations in Daphnia pulicaria were significantly correlated with whole body protein content at low (8 °C) water temperatures, and with fat content at elevated (20 °C) water temperatures (Heisig-Gunkel and Gunkel 1982).

Atrazine is rapidly degraded in boxcrabs (Sesarma cinereum) feeding on smooth cordgrass (Spartina alterniflora) grown in a radiolableled atrazine solution. After 10 days, only 1.2% of the total radioactivity in the crab was unchanged atrazine, compared to 24% in the food source. The accumulation of water-soluble atrazine metabolites (86% of total radioactivity) in Sesarma suggested that glutathione conjugation, or a comparable pathway, was responsible for the almost complete degradation and detoxification of atrazine in crabs (Davis et al. 1979; Pillai et al. 1979). Atrazine does not appear to be a serious threat to crabs in Chesapeake Bay, where water concentrations of 2.5 ug/l have been recorded, although it could have an indirect effect on crabs by decreasing the algal population, which composes a portion of their diet (Plumley et al. 1980).

BIRDS

Atrazine is not acutely lethal to birds at realistic environmental levels, i.e., oral LD-50 values were >2,000 mg/kg BW and dietary LC-50s were >5,000 mg/kg (Table 6). Also, the probability is low for chronic effects of atrazine on wetland aquatic organisms and for biomagnification of toxic residues through waterfowl food chains (Huckins et al. 1986). However, indirect effects of atrazine on insect- and seed-eating birds have not been investigated, and this may be critical to the survival of certain species during nesting and brood-rearing. Studies are needed on the potential indirect ecosystem effects of atrazine, with special reference to seed-eating birds.

Domestic chickens (Gallus sp.) rapidly metabolized atrazine by way of partial N-dealkylation accompanied by hydrolysis; dealkylation occurred mainly at the ethylamino group, resulting in intermediate degradation products (Foster and Khan 1976; Khan and Foster 1976). In vitro studies with bird liver homogenates also demonstrated active transformation of atrazine and its metabolites. Chicken liver homogenates released nonextractable atrazine residues that had accumulated in corn plants, present mainly as 2-chloro mono

Table 6. Atrazine effects on selected species of birds.

Species, dose, and other variables	Effect and reference
Chicken, *Gallus* sp.	
Laying hens were fed diets containing 100 mg/kg for 7 days.	No visible adverse physiological effects or signs of toxicity. No effect on egg production or growth. No residues of atrazine or its metabolites detected in eggs. In excreta, however, atrazine and atrazine metabolites were detected after 24 hours on treated diet and remained measurable until day 11, or after 4 days on an untreated diet (Foster and Khan 1976; Reed 1982).
Adults fed diets containing 100 mg/kg for 7 days, followed by uncontaminated diet for 7 days. Residues of atrazine and its metabolites were determined in selected tissues.	Residues, in mg/kg FW, were as follows: atrazine, 38.8 in abdominal fat and 0.04 in muscle; hydroxyatrazine, 16.2 in liver, 4.3 in kidney 2.5 in oviduct, 0.7 in abdominal fat, and 0.5 in gizzard; and deethylhydroxyatrazine in liver, 2.3 in kidney, 0.8 to 1.8 in muscle, and 0.3 in gizzard (Khan and Foster 1976).
Ring-necked pheasant, *Phasianus colchicus*	
Males, age 3 months, given 2,000 mg/kg body weight (BW), administered orally.	Survivors showed weakness, hyperexcitability, ataxia, and tremors; remission by day 5 posttreatment (Hudson et al. 1984).
Mallard, *Anas platyrhynchos*	
Females, age 6 months, given 2,000 mg/kg BW, administered orally.	Survivors showed weakness, tremors, ataxia, and weight loss. Signs of poisoning appeared within one hour posttreatment and persisted up to 11 days (Tucker and Crabtree 1970; Hudson et al. 1984).
19,650 mg/kg diet for 8 days.	LD-50 (Beste 1983).

Table 6. (Concluded)

Species, dose, and other variables	Effect and reference
Coturnix, *Coturnix japonica* Chicks, age 7 days, given diets containing 5,000 mg/kg for 5 days plus 3 days on untreated feed.	One of 14 birds tested died on day 3 of feeding; no other adverse effects reported (Hill and Camardese 1986).
Northern bobwhite, *Colinus virginianus* 5,760 mg/kg diet for 8 days.	LD-50 (Beste 1983).

N-dealkylated compounds, and subsequently metabolized them to 2-hydroxy analogues (Khan and Akhtar 1983). Liver homogenates in the goose (Anser sp.) contained enzyme systems that metabolized atrazine by partial N-dealkylation and hydrolysis; hydrolysis predominated and resulted in the formation of hydroxyatrazine, which does not undergo further degradation by dealkylation. But partly N-dealkylated metabolites, such as deethylatrazine and deisopropylatrazine, were further hydrolyzed to the corresponding hydroxy analogues (Foster et al. 1980).

MAMMALS

Data are lacking for atrazine's effects on mammalian wildlife, although there is a growing body of evidence on domestic and small laboratory mammals. Available data demonstrate that mammals are comparatively resistant to atrazine, and that the compound is not carcinogenic, mutagenic, or teratogenic (Reed 1982; Table 7). There have been no established cases of skin irritation resulting from experimental or commercial applications of atrazine, and no documented cases of poisoning in man (Anon. 1963; Hull 1967). No observable ill effects were detected in cattle, dogs, horses, or rats fed diets that included 25 mg atrazine/kg food over extended periods (Beste 1983). Most members of the triazine class of herbicides, including atrazine, have low acute oral toxicities--usually >1,000 mg/kg body weight (Murphy 1986; Table 7). But at dosages bordering on lethality, rats showed muscular weakness, hypoactivity, drooped eyelids, labored breathing, prostration (Beste 1983), altered liver morphology and renal function (Santa Maria et al. 1986, 1987), and embryotoxicity (Peters and Cook 1973).

Animals feeding on atrazine-treated crops are at limited toxicological risk. Crop plants metabolize atrazine to hydroxyatrazine, dealkylated analogues, and cysteine-and glutathione-conjugates of atrazine; mature plants contain little unchanged atrazine. Bound atrazine residues in plants are of limited bioavailability to animals (Bakke et al. 1972a; Khan and Akhtar 1983; Khan et al. 1985). Metabolic degradation of atrazine in mammals is usually rapid and extensive; unchanged atrazine was recovered only from the feces (Anon. 1963). Liver enzyme systems in pigs, rats, and sheep metabolize atrazine by partial N-dealkylation and hydrolysis (Bakke et al. 1972a; Dauterman and Muecke 1974; Foster et al. 1980). However, atrazine is reportedly converted in vivo to N-nitrosoatrazine in mice, Mus sp. (Krull et al. 1980). Since N-nitrosoatrazine is carcinogenic and mutagenic to laboratory animals (Krull et al. 1980), more research is recommended on the extent of nitrosation of atrazine in the environment.

Table 7. Lethal and sublethal effects of atrazine on selected species of mammals.

Organism, dose, and other variables	Effect and reference
Cattle, Cow, *Bos* spp.	
30 mg atrazine/kg diet for 21 days	Tissue residues <0.1 mg/kg fresh weight (Reed 1982).
100 mg atrazine/kg diet for 21 days	No detectable atrazine (<0.04 mg/kg) or hydroxyatrazine (<0.05 mg/kg) found in milk (Reed 1982).
Domestic sheep, *Ovis aries*	
30 mg atrazine/kg diet for 28 days	Tissue residues <0.1 mg/kg fresh weight (Reed 1982).
100 mg atrazine/kg diet for 28 days	No adverse effects (Reed 1982).
Mice, *Mus* spp.	
46.4 mg/kg body weight (BW) given daily on days 6 through 14 of pregnancy	No effect on reproduction (Peters and Cook 1973).
82 mg/kg diet for 18 months	Negative oncogenicity results (Reed 1982).
1,750 to 3,900 mg/kg BW	Acute oral LD-50 value (Anon. 1963; Hull 1967; Reed 1982).
Dog, *Canis familiaris*	
150 mg/kg diet for two years, equivalent to 3.75 mg/kg BW daily	No observable effect level (Reed 1982).
1,500 mg/kg diet for two years	No oncogenic effects; decreased body weight, reduced hemoglobin and hematocrit (Reed 1982).

Table 7. (Continued)

Organism, dose, and other variables	Effect and reference
Rat, Rattus spp.	
Inhalation exposure to a dust aerosol of Atrazine 80W (80% wettable powder) for one hour to concentrations between 1.8 and 4.9 mg/l atmosphere	No deaths, or signs of toxicological or pharmacological effects (Hull 1967).
100 mg/kg diet for 2 years, equivalent to 5 mg/kg BW daily	No gross microscopic signs of toxicity (Anon. 1963; Reed 1982; Beste 1983).
100 mg/kg diet for three generations, equivalent to 5 mg/kg BW daily	No teratogenic or reproductive effects (Reed 1982).
Daily oral administration on days 6 to 15 of gestation, in mg/kg BW	
10	No adverse maternal or fetal effects (Infurna et al. 1988).
70	Increased salivation; initial reduction in feed consumption (Infurna et al. 1988).
700	Mortality 78% before necropsy; increased incidences of salivation, ptosis, bloody ulva, swollen abdomen, and fetal skeletal malformations (Infurna et al. 1988).
100, 200, 400, or 600 mg/kg BW daily, given orally for 14 days	All dose levels increased elimination of sodium, potassium, chloride, and urine protein; interference with creatinine clearance at 200 mg/kg BW and higher (Santa Maria et al. 1986).

Table 7. (Continued)

Organism, dose, and other variables	Effect and reference
100, 200, 400 or 600 mg/kg BW daily for 14 days	At 100 mg/kg, significant increases in serum lipids, serum alkaline phosphatase, and serum alanine aminotransferase; no liver histopathology. At 200 mg/kg, a significant reduction in body weight. At 400 mg/kg, liver enlargement and loss in body weight. A dose-dependent decrease in growth and in serum glucose and a dose-related increase in total serum lipids were recorded. At 600 mg/kg, liver histopathology (Santa Maria et al. 1987).
100, 300, or 900 mg/kg diet for 3 weeks	Except for lymphopenia, which was observed at all dose levels, no other effects were measured in the 100 and 300 mg/kg groups. At 900 mg/kg, significant decreases occurred in body weight, food intake, blood lymphocytes and thymus weight, and significant increases occurred in thyroid weight, mesenteric lymph nodes, and histopathology (Vos et al. 1983).
200 mg/kg BW injected subcutaneously on days 3, 6, and 9 of gestation	No effect on number of pups per litter or on weight at weaning (Peters and Cook 1973).
800, 1,000, or 2,000 mg/kg BW injected subcutaneously on days 3, 6, and 9 of gestation	At 2,000 mg/kg BW, most pups born dead; at 800 and 1,000 mg/kg BW, litter size reduced 50% to 100% (Peters and Cook 1973).
1,000 mg atrazine/kg diet from first day of pregnancy throughout gestation	No effect on number of pups per litter or on weight at weaning (Peters and Cook 1973).
1,000 mg/kg diet for two years, equivalent to 50 mg/kg BW daily	No signs of oncogenicity, but reduced food intake and lower body weight (Reed 1982).
1,800 to 5,100 mg/kg BW	Acute oral LD-50 (Anon. 1963; Hull 1967; Reed 1982; Beste 1983).

Table 7. (Concluded)

Organism, dose, and other variables	Effect and reference
White rabbit, Oryctolagus cuniculus	
Daily oral administration on gestational days 7 through 19, in mg/kg BW	
1	No adverse maternal or fetal effects (Infurna et al. 1988).
5	Moderate reductions in food consumption and body weight gain (Infurna et al. 1988).
75	Increased abortion rate; no death of does. Weight loss, reductions in feed consumption, and fetal and embryotoxic effects, including reduced fetal weight and increased incidence in skeletal variations (Infurna et al. 1988).
9,300 mg/kg BW	Acute dermal LD-50 (Beste 1983).

CURRENT RECOMMENDATIONS

Labels on products containing atrazine are required to contain information on acceptable uses and potential hazards to groundwater and to fish and wildlife (EPA 1983). At present, atrazine is approved for use as an herbicide to control broadleaf and grassy weeds on corn, sorghum, sugarcane, pineapple, macadamia nuts, rangeland, turf grass sod, conifer reforestation areas, Christmas tree plantations, grass seed fields, noncrop land, guava, grass in orchards, millet, perennial ryegrass, and wheat. Because atrazine is expected to leach into groundwater, it was recommended (EPA 1983) that labels of atrazine products bear the following statement: "Atrazine leaches readily and accepted label rates have been found to result in contamination of water supplies by way of groundwater. Therefore users are advised to avoid use of atrazine in well drained soils, particularly in areas having high groundwater tables." Cautionary statements on potential hazards to living resources is another labeling requirement: "This pesticide is toxic to aquatic invertebrates. Do not apply to water or wetlands. Runoff and drift from treated areas may be hazardous to aquatic organisms in neighboring areas. Do not contaminate water by cleaning of equipment or disposal of wastes. Do not discharge into lakes, streams, ponds, or public water supplies unless in accordance with an [approved EPA] permit" (EPA 1983).

Current permissible tolerances for atrazine range from 0.02 mg/kg (the limit of detection of the analytical method) in meat, milk, and eggs, to 15 mg/kg in orchard grass forage, fodder, and hay (Reed 1982; EPA 1983). However, the current 15 mg/kg tolerance in forage is considered high, and a new upper limit of 4 mg/kg is proposed. This limit would be expressed in terms of atrazine and three major metabolites: 2-amino-4-chloro-6-isopropylamino-1, 3, 5-triazine; 2-amino-4-chloro-6-ethylamino-1, 3, 5-triazine; and 2-chloro-4, 6-diamino-1, 3, 5-triazine (Reed 1982; EPA 1983).

The maximum recommended safe level of atrazine to algal diatoms is 10 ug/l (Karlander et al. 1983), although temporary inhibition of chlorophyll production in sensitive algal species has been reported in the range of 1 to 5 ug/l (Torres and O'Flaherty 1976). Proposed atrazine concentrations for aquatic life protection range from about 1 to 11 ug/l: 1 to 2 ug/l for protection of estuarine productivity (Stevenson et al. 1982; Ward and Ballantine 1985); 1 to 7 ug/l for no adverse effect levels to most species of submerged aquatic vegetation (Glotfelty et al. 1984); 5 to 10 ug/l for minor reductions in photosynthesis in sensitive species of aquatic macrophytes (Glotfelty et al. 1984); 9 ug/l for sensitive aquatic invertebrates, as judged by an uncertainty factor of 10 applied to a 96-hour LC-50 (Ward and Ballantine

1985); and 11 ug/l for salt marsh algae, based on the least effect level of 110 ug/l, and an uncertainty factor of 10 (Plumley and Davis 1980). Atrazine concentrations >11 ug/l sometimes occur during periods of runoff and non-flushing (Stevenson et al. 1982), but rarely persist at levels necessary to markedly inhibit photosynthesis in aquatic plants, i.e., 60 to 70 ug/l (Glotfelty et al. 1984).

In laboratory animals, atrazine is only slightly toxic on an acute basis; no carcinogenic, mutagenic, or reproductive effects have been seen at low doses, and reduced food intake and body weight were the primary adverse effects seen at high doses in chronic studies with rats and dogs (Reed 1982). However, data are lacking on indirect ecosystem effects of atrazine application on terrestrial wildlife--especially on insectivores and granivores; studies should be initiated this subject area.

No allowable daily intake of atrazine in the human diet has been established, although 0.0375 mg/kg body weight daily has been proposed --equivalent to 2.25 mg daily for a 60-kg adult, or 1.5 mg/kg diet based on 1.5 kg food daily (Reed 1982). In man, the theoretical maximum residue contribution (TRMC)--a worst case estimate of dietary exposure--is 0.77 mg daily, assuming 1.5 kg of food eaten daily; this is equivalent to 0.51 mg/kg diet, or 0.013 mg/kg body weight daily for a 60-kg person (EPA 1983). Another TRMC calculation is based on 0.233 mg daily per 1.5 kg diet, equivalent to 0.156 mg/kg diet, or 0.0039 mg/kg body weight daily for a 60-kg person (Reed 1982). Both TRMC estimates are substantially below the proposed limit of 0.0375 mg/kg body weight daily. Lifetime exposure to drinking water concentrations of 2.3 ug atrazine/l poses negligible risk to human health, as judged by the no adverse effect level of 7.5 ug/l when 1% of the allowable daily intake is obtained from this source (EPA 1987; Wilson et al. 1987). Higher allowable concentrations are proposed over short periods: 123 ug/l for adults and 35 ug/l for children over a 10-day period (EPA 1987).

Additional data are needed on toxicity, environmental fate, and chemistry of atrazine in order to maintain existing registrations or to permit new registrations (EPA 1983). Specifically, data are needed on mobility and degradation rates of atrazine and its metabolites in soils; accumulation studies in rotational crops, fish, and aquatic invertebrates; and chronic testing with representative flora and fauna on survival, reproduction, carcinogenesis, teratogenesis, and mutagenesis (EPA 1983). Animal metabolism studies are required if tolerances for residues in animal products are expressed in terms of atrazine and its metabolites (EPA 1983). Finally, more research on aquatic species is merited on synergistic and additive effects of atrazine in combination with other agricultural chemicals at realistic environmental levels of 1 to 50 ug/l, and on the toxic effects of dealkylated atrazine metabolites (Stevenson et al. 1982).

LITERATURE CITED

Amor, R. L., A. Kent, P. E. Ridge, and R. M. Binns. 1987. Persistence of atrazine in chemical fallows in the Victorian Wimmera and Mallee. Plant Protection Q. 21:38-40.

Anon. 1963. Atrazine. Geigy Agric. Chem. Tech. Bull. 63-1. 14 pp. Avail. from Geigy Chem. Corp., P.O. Box 430, Yonkers, New York.

Bahler, C. C., K. P. Vogel, and L. E. Moser. 1984. Atrazine tolerance in warm-season grass seedlings. Agron. J. 76:891-895.

Bakke, J. E., J. D. Larson, and C. E. Price. 1972a. Metabolism of atrazine and 2-hydroxyatrazine by the rat. J. Agric. Food Chem. 20:602-607.

Bakke, J. E., R. H. Shimabukuro, K. L. Davison, and G. L. Lamoureux. 1972b. Sheep and rat metabolism of the insoluble ^{14}C-residues present in ^{14}C-atrazine-treated sorghum. Chemosphere 1:21-24.

Beaumont, G., A. Lord, and G. Grenier. 1980. Effets physiologiques de l'atrazine a doses subletales sur Lemna minor. V. Influence sur l'ultrastructure des chloroplastes. Can. J. Bot. 58:1571-1577.

Beste, C. E. (ed.). 1983. Herbicide handbook of the Weed Science Society of America. Weed Science Society of America, 309 West Clark Street, Champaign, Illinois. 515 pp.

Bettini, P., S. McNally, M. Sevignac, H. Darmency, J. Gasquez, and M. Dron. 1987. Atrazine resistance in Chenopodium album. Plant Physiol. 84:1442-1446.

Brecke, B. J., W. L. Cuney, and D. H. Teem. 1981. Atrazine persistence in a corn-soy bean doublecropping system. Agron. J. 73:534-537.

Brockway, D. L., P. D. Smith, and F. E. Stancil. 1984. Fate and effects of atrazine in small aquatic microcosms. Bull. Environ. Contam. Toxicol. 32:345-353.

Correll, D. L., and T. L. Wu. 1982. Atrazine toxicity to submersed vascular plants in simulated estuarine microcosms. Aquat. Bot. 14:151-158.

Cunningham, J. J., W. M. Kemp, M. R. Lewis, and J. C. Stevenson. 1984. Temporal responses of the macrophyte Potamogeton perfoliatus L., and its associated autotrophic community to atrazine exposure in estuarine microcosms. Estuaries 7(4B):519-530.

Dao, T. H. 1977. Factors affecting atrazine adsorption, degradation, and mobility in soil. Ph. D. Thesis. University of Nebraska, Lincoln. 68 pp.

Dauterman, W. C., and W. Muecke. 1974. In vitro metabolism of atrazine by rat liver. Pestic. Biochem. Physiol. 4:212-219.

Davis, D. E., J. D. Weete, C. G. P. Pillai, F. G. Plumley, J. T. McEnerney, J. W. Everest, B. Truelove, and A. M. Diner. 1979. Atrazine fate and effects in a salt marsh. U. S. Environ. Protection Agency Rep. 600/3-79-111. 84 pp.

DeNoyelles, F., Jr., and W. D. Kettle. 1985. Experimental ponds for evaluating bioassay predictions. Pages 91-103 in T. P. Boyle (ed.). Validation and predictability of laboratory methods for assessing the fate and effects of contaminants in aquatic ecosystems. ASTM Spec. Tech. Publ. 865. American Society for Testing and Materials, 1916 Race Street, Philadelphia, Pennsylvania 19103.

DeNoyelles, F., W. D. Kettle, and D. E. Sinn. 1982. The responses of plankton communities in experimental ponds to atrazine, the most heavily used pesticide in the United States. Ecology 63:1285-1293.

Dewey, S. L. 1986. Effects of the herbicide atrazine on aquatic insect community structure and emergence. Ecology 67:148-162.

EPA. 1983. Guidance for the reregistration of pesticide products containing atrazine as the active ingredient. U. S. Environ. Protection Agency Rep. 540-RS-83-002. 90 pp.

EPA. 1987. Atrazine. Health advisories for 50 pesticides. Draft report. Office of Drinking Water, U. S. Environ. Protection Agency, 401 M Street SW, Washington, DC 20460. 21 pp.

Forney, D. R. 1980. Effects of atrazine on Chesapeake Bay aquatic plants. M. S. Thesis. Auburn University, Auburn, Alabama. 76 pp.

Forney, D. R., and D. E. Davis. 1981. effects of low concentrations of herbicides on submersed aquatic plants. Weed Sci. 29:677-685.

Foster, T. S., and S. U. Khan. 1976. Metabolism of atrazine by the chicken. J. Agric. Food Chem. 24:566-570.

Foster, T. S., S. U. Khan, and M. H. Akhtar. 1980. Metabolism of deethylatrazine, deisopropylatrazine, and hydroxyatrazine by the soluble fraction (105000 g) from goose liver homogenates. J. Agric. Food Chem. 28:1083-1085.

Frank, R., and G. J. Sirons. 1979. Atrazine: its use in corn production and its loss to stream waters in southern Ontario, 1975-1977. Sci. Total Environ. 12:223-239.

Giardi, M. T., M. C. Giardina, and G. Filacchioni. 1985. Chemical and biological degradation of primary metabolites of atrazine by a Nocardia strain. Agric. Biol. Chem. 49:1551-1558.

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

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

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. 13:115-121.

Gonzalez-Murua, C., A. Munoz-Rueda, F. Hernando, and M. Sanchez-Diaz. 1985. Effect of atrazine and methabenzthiazuron on oxygen evolution and cell growth of Chlorella pyrenoidosa. Weed Res. 25:61-66.

Grenier, G., L. Proteau, J. P. Marier, and G. Beaumont. 1987. Effects of a sublethal concentration of atrazine on the chlorophyll and lipid composition of chlorophyll-protein complexes of Lemna minor. Plant Physiol. Biochem. 25:409-413.

Gunkel, G. 1981. Bioaccumulation of a herbicide (atrazine, s-triazine) in the whitefish (Coregonus fera J.): uptake and distribution of the residue in fish. Arch. Hydrobiol. Suppl. 59(2/3):252-287.

Gunkel, G., and B. Streit. 1980. Mechanisms of a herbicide (atrazine, s-triazine) in a freshwater mollusc (Ancylus fluviatilis Mull.) and a fish (Coregonus fera Jurine). Water Res. 14:1573-1584.

Gunther, F. A., and J. D. Gunther (eds.). 1970. The triazine herbicides. Residue Rev. 32:1-413.

Hamala, J. A., and H. P. Kollig. 1985. The effects of atrazine on periphyton communities in controlled laboratory ecosystems. Chemosphere 14:1391-1408.

Hamilton, P. B., G. S. Jackson, N. K. Kaushik, and K. R. Solomon. 1987. The impact of atrazine on lake periphyton communities, including carbon uptake dynamics using track autoradiography. Environ. Pollut. 46:83-103.

Hammons, R. H. 1977. Atrazine persistence in a Valentine loamy fine sand profile. M. S. Thesis. Univ. Nebraska, Lincoln. 48 pp.

Heisig-Gunkel, G., and G. Gunkel. 1982. Distribution of a herbicide (atrazine, s-triazine) in Daphnia pulicaria: a new approach to determination. Arch. Hydrobiol. Suppl. 59(4):359-376.

Herman, D., N. K. Kaushik, and K. R. Solomon. 1986. Impact of atrazine on periphyton in freshwater enclosures and some ecological consequences. Can. J. Fish. Aquat. Sci. 43:1917-1925.

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

Hollister, T. A., and G. E. Walsh. 1973. Differential responses of marine phytoplankton to herbicides: oxygen evolution. Bull. Environ. Contam. Toxicol. 9:291-295.

Huber, S. J., and B. Hock. 1986. Atrazine in water. Pages 438-451 in H. U. Bergmeyer, J. Bergmeyer, and M. Grassl (eds.). Methods of enzymatic analysis. Vol. XII. Drugs and pesticides, third edition. VCH Publishers, Deerfield Beach, Florida.

Huckins, J. N., J. D. Petty, and D. C. England. 1986. Distribution and impact of trifluralin, atrazine, and fonofos residues in microcosms simulating a northern prairie wetland. Chemosphere 15:563-588.

Hudson, R. H., R. K. Tucker, and M. A. Haegele. 1984. Handbook of toxicity of pesticides to wildlife. U. S. Fish Wildl. Serv. Resour. Publ. 153. 90 pp.

Hull, H. M. 1967. Herbicide handbook of the Weed Society of America. W. F. Humphery Press, Geneva, New York. 293 pp.

Infurna, R., B. Levy, C. Meng, E. Yau, and V. Traina. 1988. Teratological evaluations of atrazine technical, a triazine herbicide, in rats and rabbits. J. Toxicol. Environ. Health 24:307-319.

Isensee, A. R. 1987. Persistence and movement of atrazine in a salt marsh sediment microecosystem. Bull. Environ. Contam. Toxicol. 39:516-523.

Jachetta, J., A. P. Appleby, and L. Boersma. 1986. Apoplastic and

symplastic pathways of atrazine and glyphosate transport in shoots of seedling sunflower. Plant Physiol. 82:1000-1007.

Jones, T. W., and P. S. Estes. 1984. Uptake and phytotoxicity of soil-sorbed atrazine for the submerged aquatic plant, Potamogeton perfoliatus L. Arch. Environ. Contam. Toxicol. 13:237-241.

Jones, T. W., W. M. Kemp, P. S. Estes, and J. C. Stevenson. 1986. Atrazine uptake, photosynthetic inhibition, and short-term recovery for the submersed vascular plant, Potamogeton perfoliatus L. Arch. Environ. Contam. Toxicol. 15:277-283.

Jones, T. W., W. M. Kemp, J. C. Stevenson, and J. C. Means. 1982. Degradation of atrazine in estuarine water/sediment systems and soils. J. Environ. Qual. 11:632-638.

Jones, T. W., and L. Winchell. 1984. Uptake and photosynthetic inhibition by atrazine and its degradation products on four species of submerged vascular plants. J. Environ. Qual. 13:243-247.

Karlander, E. P., J. M. Mayasich, and D. E. Terlizzi. 1983. Effects of the herbicide atrazine on an oyster-food organism. Univ. Maryland Water Resour. Res. Cen., Tech. Rep. 73. 20 pp. Univ. Maryland, College Park.

Kaushik, N. K., K. R. Solomon, G. Stephenson, and K. Day. 1985. Assessment of sublethal effects of atrazine on zooplankton. Can. Tech. Rep. Fish. Aquat. Sci. 1368:377-379.

Kemp, W. M., W. R. Boynton, J. J. Cunningham, J. C. Stevenson, T. W. Jones, and J. C. Means. 1985. Effects of atrazine and linuron on photosynthesis and growth of the macrophytes, Potamogeton perfoliatus L. and Myriophyllum spicatum L. in an estuarine environment. Mar. Environ. Res. 16:255-280.

Kemp, W. M., W. R. Boynton, J. C. Stevenson, J. C. Means, R. R. Twilley, and T. W. Jones (eds.). 1983. Submerged aquatic vegetation in upper Chesapeake Bay: studies related to possible causes of the recent decline in abundance. Final report submitted to U. S. Environ. Protection Agency, 2083 West Street, Annapolis, Maryland 21401. 331 pp.

Kettle, W. D., F. DeNoyelles, Jr., B. D. Heacock, and A. M. Kadoum. 1987. Diet and reproductive success of bluegill recovered from experimental ponds treated with atrazine. Bull. Environ. Contam. Toxicol. 38:47-52.

Khan, S. U., and M. H. Akhtar. 1983. In vitro release of bound

(nonextractable) atrazine residues from corn plants by chicken liver homogenate and bovine rumen liquor. J. Agric. Food Chem. 31:641-644.

Khan, S. U., and T. S. Foster. 1976. Residues of atrazine (2-chloro-4-ethylamino-6-isopropylamino-s -triazine) and its metabolites in chicken tissues. J. Agric. Food Chem. 24:768-771.

Khan, S. U., S. Kacew, and S. J. Molnar. 1985. Bioavailability in rats of bound ^{14}C residues from corn plants treated with [^{14}C] atrazine. J. Agric. Food Chem. 33:712-717.

Khan, S. U., and W. J. Saidak. 1981. Residues of atrazine and its metabolites after prolonged usage. Weed Res. 21:9-12.

Klaasen, H. E., and A. M. Kadoum. 1979. Distribution and retention of atrazine and carbofuran in farm pond ecosystems. Arch. Environ. Contam. Toxicol. 8:345-353.

Knuesli, E., D. Berrer, G. Depuis, and H. Esser. 1969. s-Triazines. Pages 51-78 in P. C. Kearney and D. D. Kaufman (eds.). Degradation of herbicides. Marcel Dekker, New York.

Krull, I. S., K. Mills, G. Hoffman, and D. H. Fine. 1980. The analysis of N-nitrosoatrazine and N-nitrosocarbaryl in whole mice. J. Anal. Toxicol. 4:260-262.

Kulshrestha, G., N. T. Yaduraju, and V. S. Mani. 1982. The relative toxicity of the s -triazine herbicides atrazine and simazine to crops. J. Environ. Sci. Health B17:341-354.

Larsen, D. P., F. DeNoyelles, Jr., F. Stay, and T. Shiroyama. 1986. Comparisons of single-species, microcosm and experimental pond responses to atrazine exposure. Environ. Toxicol. Chem. 5:179-190.

Lay, J. P., A. Muller, L. Peichl, W. Klein, and F. Korte. 1984. Longterm effects of the herbicides atrazine and dichlobenil upon the phytoplankton density and physico-chemical conditions in compartments of a freshwater pond. Chemosphere 13:821-832.

Lu, T. W., L. Lambert, D. Hastings, and D. Banning. 1980. Enrichment of the agricultural herbicide atrazine in the microsurface water of an estuary. Bull. Environ. Contam. Toxicol. 24:411-414.

Lynch, T. R., H. E. Johnson, and W. J. Adams. 1982. The fate of atrazine and a hexachlorobiphenyl isomer in naturally-derived model stream ecosystems. Environ. Toxicol. Chem. 1:179-192.

Macek, K. J., K. S. Buxton, S. Sauter, S. Gnilka, and J. W. Dean. 1976.

Chronic toxicity of atrazine to selected aquatic invertebrates and fishes. U. S. Environ. Protection Agency Rep. 600/3-76-047. 58 pp.

Malan, C., J. H. Visser, and H. A. van de Venter. 1987. Atrazine metabolism in field-grown maize and the effect of atrazine phytotoxicity on yield. S. Afr. J. Plant Soil 4:7-11.

Malanchuk, J. L., and H. P. Kollig. 1985. Effects of atrazine on aquatic ecosystems: a physical and mathematical modeling assessment. Pages 212-224 in T. P. Boyle (ed.). Validation and predictability of laboratory methods for assessing the fate and effects of contaminants in aquatic ecosystems. ASTM Spec. Tech. Publ. 865. American Society for Testing and Materials, 1916 Race Street, Philadelphia, Pennsylvania 19103.

Mayasich, J. M., E. P. Karlander, and D. E. Terlizzi, Jr. 1986. Growth responses of Nannochloris oculata Droop and Phaeodactylum tricornutum Bohlin to the herbicide atrazine as influenced by light intensity and temperature. Aquat. Toxicol. 8:175-184.

Mayasich, J. M., E. P. Karlander, and D. E. Terlizzi, Jr. 1987. Growth responses of Nannochloris oculata Droop and Phaeodactylum tricornutum Bohlin to the herbicide atrazine as influenced by light intensity and temperature in unialgal and bialgal assemblage. Aquat. Toxicol. 10:187-197.

Mayer, F. L. 1987. Acute toxicity handbook of chemicals to estuarine organisms. U. S. Environ. Protection Agency Rep. 600/8-87/017. 274 pp.

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. U. S. Fish Wildl. Serv. Resour. Publ. 160. 579 pp.

McCormick, L. L., and A. E. Hiltbold. 1966. Microbiological decomposition of atrazine and diuron in soil. Weeds 14:77-82.

McNally, S., P. Bettini, M. Sevignac, H. Darmency, J. Gasquez, and M. Dron. 1987. A rapid method to test for chloroplast DNA involvement in atrazine resistance. Plant Physiol. 83:248-250.

Menzer, R. E., and J. O. Nelson. 1986. Water and soil pollutants. Pages 825-853 in C. D. Klaassen, M. O. Amdur, and J. Doull (eds.). Casarett and Doull's toxicology, third edition. Macmillan Publ. Co., New York.

Millie, D. F., and C. M. Hersh. 1987. Statistical characterizations of the atrazine-induced photosynthetic inhibition of Cyclotella meneghiniana

(Bacillariophyta). Aquat. Toxicol. 10:239-249.

Mitchell, C. A. 1985. Effects of atrazine on Halodule wrightii Ascherson in the laboratory. M. S. Thesis. Corpus Christi State Univ., Corpus Christi, Texas. 82 pp.

Mola, I., M. A. Sabatini, B. Fratello, and R. Bertolani. 1987. Effects of atrazine on two species of Collembola (Onychiuridae) in laboratory tests. Pedobiologia 30:145-149.

Moorhead, D. L., and R. J. Kosinski. 1986. Effect of atrazine on the productivity of artificial stream algal communities. Bull. Environ. Contam. Toxicol. 37:330-336.

Murphy, S. D. 1986. Toxic effects of pesticides. Pages 519-581 in C. D. Klaassen, M. O. Amdur, and J. Doull (eds.). Casarett and Doull's toxicology, third edition. Macmillan Publ. Co., New York.

Peters, J. W., and R. M. Cook . 1973. Effects of atrazine on reproduction in rats. Bull. Environ. Contam. Toxicol. 9:301-304.

Pillai, C. G. P., J. D. Weete, and D. E. Davis. 1977. Metabolism of atrazine by Spartina alterniflora. I. Chloroform-soluble metabolites. J. Agric. Food Chem. 25:852-855.

Pillai, P., J. D. Weete, A. M. Diner, and D. E. Davis. 1979. Atrazine metabolism in box crabs. J. Environ. Qual. 8:277-280.

Plumley, F. G., and D. E. Davis. 1980. The effect of a photosynthesis inhibitor atrazine, on salt marsh edaphic algae, in culture, microecosystems, and in the field. Estuaries 3:271-277.

Plumley, F. G., D. E. Davis, J. T. McEnerney, and J. W. Everest. 1980. Effects of a photosynthesis inhibitor, atrazine, on the salt-marsh fiddler crab, Uca pugnax (Smith). Estuaries 3:217-223.

Popovici, I., G. Stan, V. Stefan, R. Tomescu, A. Dumea, A. Tarta, and F. Dan. 1977. The influence of atrazine on soil fauna. Pedobiologia 17:209-215.

Pratt, J. R., N. J. Bowers, B. R. Niederlehner, and J. Cairns, Jr. 1988. Effects of atrazine on freshwater microbial communities. Arch. Environ. Contam. Toxicol. 17:449-457.

Reed, D. 1982. Atrazine. (Unpubl. mimeo.) Available from U. S. Food and Drug Administration, Bureau of Foods, Washington, DC. 6 pp.

Richardson, L. T. 1970. Effects of atrazine on growth response of soil

fungi. Can. J. Plant Sci. 50:594-596.

Rodriquez-Kabana, R., E. A. Curl, and H. H. Funderburk, Jr. 1968. Effect of atrazine on growth activity of Sclerotium rolfsii and Trichoderma viride in soil. Can. J. Microbiol. 14:1283-1288.

Santa Maria, C., J. Moreno, and J. L. Lopez-Campos. 1987. Hepatotoxicity induced by the herbicide atrazine in the rat. J. Appl. Toxicol. 7:373-378.

Santa Maria, C., M. G. Vilas, F. G. Muriana, and A. Relimpio. 1986. Subacute atrazine treatment effects on rat renal functions. Bull. Environ. Contam. Toxicol. 36:325-331.

Schiavon, M. 1988a. Studies of the leaching of atrazine, of its chlorinated derivatives, and of hydroxyatrazine from soil using ^{14}C ring-labeled compounds under outdoor conditions. Ecotoxicol. Environ. Safety 15:46-54.

Schiavon, M. 1988b. Studies of the movement and the formation of bound residues of atrazine, of its chlorinated derivatives, and of hydroxyatrazine in soil using ^{14}C ring-labeled compounds under outdoor conditions. Ecotoxicol. Environ. Safety 15:55-61.

Schocken, M. J., and M. K. Speedie. 1982. Interaction of higher marine fungi with the herbicide atrazine. II. Sorption of atrazine to four species of marine fungi. Bull. Environ. Contam. Toxicol. 29:101-106.

Schocken, M. J., and M. K. Speedie. 1984. Physiological aspects of atrazine degradation by higher marine fungi. Arch. Environ. Contam. Toxicol. 13:707-714.

Setzler, J. V. 1980. Atrazine residues in northern Ohio streams--1980. U. S. Army Corps Engineers, Tech. Rep., Lake Erie Wastewater Manage. Study, 1776 Niagara St., Buffalo, New York. 29 pp.

Shabana, E. F. 1987. Use of batch assays to assess the toxicity of atrazine to some selected cyanobacteria. I. Influence of atrazine on the growth, pigmentation and carbohydrate contents of Aulosira fertilissima, Anabaena oryzae, Nostoc muscorum and Tolypothrix tenuis. J. Basic Microbiol. 2:113-119.

Stay, F. S., D. P. Larsen, A. Katko, and C. M. Rohm. 1985. Effects of atrazine on community level responses in Taub microcosms. Pages 75-90 in T. P. Boyle (ed.). Validation and predictability of laboratory methods for assessing the fate and effects of contaminants in aquatic ecosystems. ASTM Spec. Tech. Publ. 865. American Society for Testing and Materials, 1916 Race Street, Philadelphia, Pennsylvania 19103.

Stevenson, J. C., T. W. Jones, W. M. Kemp, W. R. Boynton, and J. C. Means. 1982. An overview of atrazine dynamics in estuarine ecosystems. Pages 71-94 in Proceedings of the workshop on agrichemicals and estuarine productivity, Beaufort, North Carolina, September 18-19, 1980. Avail. from Natl. Ocean. Atmos. Admin., Off. Mar. Pollut. Assess., Boulder Colorado.

Stratton, G. W. 1984. Effects of the herbicide atrazine and its degradation products, alone and in combination, on phototrophic microorganisms. Arch. Environ. Contam. Toxicol. 13:35-42.

Streit, B., and H. M. Peter. 1978. Long-term effects of atrazine to selected freshwater invertebrates. Arch. Hydrobiol. Suppl. 55:62-77.

Torres, A. M. R., and L. M. O'Flaherty. 1976. Influence of pesticides on Chlorella, Chlorococcum, Stigeoclonium (Chlorophyceae), Tribonema, Vaucheria (Xanthophyceae) and Oscillatoria (Cyanophyceae). Phycologia 15:25-36.

Tucker, R. K., and D. G. Crabtree. 1970. Handbook of toxicity of pesticides to wildlife. U. S. Fish Wildl. Serv. Resour. Publ. 84. 131 pp.

Veber, K., J. Zahradnik, I. Breyl, and F. Kredi. 1981. Toxic effect and accumulation of atrazine in algae. Bull. Environ. Contam. Toxicol. 27:872-876.

Vos, J. G., E. I. Krajnc, P. K. Beekhof, and M. J. van Logten. 1983. Methods for testing immune effects of toxic chemicals: evaluation of the immunotoxicity of various pesticides in the rat. Pages 497-504 in J. Miyamoto and P. C. Kearney (eds.). Pesticide chemistry, human welfare and the environment. Vol. 3. Mode of action metabolisms and toxicology. Pergamon Press, Oxford, England.

Ward, G. S., and L. Ballantine. 1985. Acute and chronic toxicity of atrazine to estuarine fauna. Estuaries 8:22-27.

Weete, J. D., P. Pillai, and D. D. Davis. 1980. Metabolism of atrazine by Spartina alterniflora. 2. Water-soluble metabolites. J. Agric. Food Chem. 28:636-640.

Wilson, M. P., E. P. Savage, D. D. Adrian, M. J. Aaronson, T. J. Keefe, D. H. Hamar, and J. T. Tessari. 1987. Groundwater transport of the herbicide, atrazine, Weld County, Colorado. Bull. Environ. Contam. Toxicol. 39:807-814.

Wolf, D. C., and R. L. Jackson. 1982. Atrazine degradation, sorption and

50272-101

REPORT DOCUMENTATION PAGE	1. REPORT NO. Biological Report 85 (1.18)	2.	3. Recipient's Accession No.

4. Title and Subtitle
Atrazine hazards to fish, wildlife and invertebrates: a synoptic review

5. Report Date
May 1989

6.

7. Author(s)
Ronald Eisler

8. Performing Organization Rept. No.

9. Performing Organization Name and Address
Patuxent Wildlife Research Center
U. S. Fish and Wildlife Service
Laurel, MD 20708

10. Project/Task/Work Unit No.

11. Contract(C) or Grant(G) No.
(C)
(G)

12. Sponsoring Organization Name and Address

13. Type of Report & Period Covered

14.

15. Supplementary Notes

16. Abstract (Limit: 200 words)

The herbicide atrazine (2-chloro-4-ethylamino-6-isopropylamino-1,3,5-triazine) is the most heavily used agricultural pesticide in North America. Domestically, more than 50 million kg are applied yearly to more than 25 million ha, primarily to control weeds in corn and sorghum crops. Atrazine residues have been detected in runoff from treated fields in lakes and streams at phytotoxic levels. Birds and mammals were comparatively resistant, with a low probability for atrazine accumulation and retention. Data are lacking on indirect effects of atrazine on wildlife granivores and insectivores. Direct effects to aquatic fauna occur at 94 ug/l, and higher; however, indirect effects may occur at 20 ug/l, and higher, partly through reduction of the food supply of herbivores, and partly through loss of macrophyte habitat.

Ecological and toxicological aspects of atrazine in the environment are briefly reviewed, with special emphasis on fishery and wildlife resources. Subtopics include enviromental chemistry, background concentrations, lethal and sublethal effects, and current recommendations for the protection of sensitive species.

17. Document Analysis a. Descriptors

Pesticides	Toxicity	Birds	Invertebrates
Herbicides	Submersed aquatic vegetation	Mammals	Natural resources
Contaminants	Aquatic fauna	Fishes	Contaminants

b. Identifiers/Open-Ended Terms

Atrazine	Chesapeake Bay	Sublethal effects
Triazines	Metabolism	Criteria

c. COSATI Field/Group

18. Availability Statement	19. Security Class (This Report)	21. No. of Pages
Release unlimited	Unclassified	53
	20. Security Class (This Page) Unclassified	22. Price

(See ANSI-Z39.18) See Instructions on Reverse OPTIONAL FORM 272 (4 (Formerly NTIS-35) Department of Comme

47284 ☆U.S. GOVERNMENT PRINTING OFFICE: 1989-675-447/5081