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Annual Review

ECOLOGICAL RISK ASSESSMENT OF ATRAZINE IN NORTH AMERICAN SURFACE WATERS

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Abstract—The triazine herbicide atrazine (2-chloro-4-ethylamino-6-isopropyl-amino-*s*-triazine) is one of the most used pesticides in North America. Atrazine is principally used for control of certain annual broadleaf and grass weeds, primarily in corn but also in sorghum, sugarcane, and, to a lesser extent, other crops and landscaping. Atrazine is found in many surface and ground waters in North America, and aquatic ecological effects are a possible concern for the regulatory and regulated communities. To address these concerns an expert panel (the Panel) was convened to conduct a comprehensive aquatic ecological risk assessment. This assessment was based on several newly suggested procedures and included exposure and hazard subcomponents as well as the overall risk assessment. The Panel determined that use of probabilistic risk assessment techniques was appropriate. Here, the results of this assessment are presented as a case study for these techniques. The environmental exposure assessment concentrated on monitoring data from Midwestern watersheds, the area of greatest atrazine use in North America. This analysis revealed that atrazine concentrations rarely exceed 20 μg/L in rivers and streams that were the main focus of the aquatic ecological risk assessment. Following storm runoff, biota in lower-order streams may be exposed to pulses of atrazine greater than 20 μg/L, but these exposures are short-lived. The assessment also considered exposures in lakes and reservoirs. The principal data set was developed by the U.S. Geological Survey, which monitored residues in 76 Midwestern reservoirs in 11 states in 1992–1993. Residue concentrations in some reservoirs were similar to those in streams but persisted longer. Atrazine residues were widespread in reservoirs (92% occurrence), and the 90th percentile of this exposure distribution for early June to July was about 5 μg/L. Mathematical simulation models of chemical fate were used to generalize the exposure analysis to other sites and to assess the potential effects of reduction in the application rates. Models were evaluated, modified, and calibrated against available monitoring data to validate that these models could predict atrazine runoff. PRZM-2 overpredicted atrazine concentrations by about an order of magnitude, whereas GLEAMS underpredicted by a factor of 2 to 5. Thus, exposure models were not used to extrapolate to other regions of atrazine use in this assessment. The effects assessment considered both freshwater and saltwater toxicity test results. Phytoplankton were the most sensitive organisms, followed, in decreasing order of sensitivity, by macrophytes, benthic invertebrates, zooplankton, and fish. Atrazine inhibits photophosphorylation but typically does not result in lethality or permanent cell damage in the short term. This characteristic of atrazine required a different model than typically used for understanding the potential impact in aquatic systems, where lethality or nonreversible effects are usually assumed. In addition, recovery of phytoplankton from exposure to 5 to 20 μg/L atrazine was demonstrated. In some mesocosm field experiments, phytoplankton and macrophytes were reduced after atrazine exposures greater than 20 μg/L. However, populations were quickly reestablished, even while atrazine residues persisted in the water. Effects in field studies were judged to be ecologically important only at exposures of 50 μg/L or greater. Mesocosm experiments did not reveal disruption of either ecosystem structure or function at atrazine concentrations typically encountered in the environment (generally 5 μg/L or less). Based on an integration of laboratory bioassay data, field effects studies, and environmental monitoring data from watersheds in high-use areas in the Midwestern United States, the Panel concluded that atrazine does not pose a significant risk to the aquatic environment. Although some inhibitory effects on algae, phytoplankton, or macrophyte production may occur in small streams vulnerable to agricultural runoff, these effects are likely to be transient, and quick recovery of the ecological system is expected. A subset of surface waters, principally small reservoirs in areas with intensive use of atrazine, may be at greater risk of exposure to atrazine. Therefore, it is recommended that site-specific risk assessments be conducted at these sites to assess possible ecological effects in the context of the uses to which these ecosystems are put and the effectiveness and cost–benefit aspect of any risk mitigation measures that may be applied.

Keywords—Risk assessment Atrazine Aquatic Surface water North America

INTRODUCTION

The herbicide atrazine is widely used for control of certain annual broadleaf and grass weeds, primarily in corn but also in sorghum, sugarcane, other crops, and landscape vegetation to some extent. In North America, the greatest quantity is used on corn. Atrazine was developed from research on derivatives of symmetrical triazines begun in 1952 by the Geigy Chemical Company of Basel, Switzerland [1]. Atrazine was patented in Switzerland in 1958 and registered for commercial use in the United States in 1959 [2]. Atrazine has been a major herbicide used worldwide since that time.

On November 23, 1994, the U.S. Environmental Protection

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Agency (EPA) began a special review of atrazine by publishing ''Atrazine, Simazine and Cyanazine; Notice of Initiation of Special Review'' in the *Federal Register* [2]. This notice indicated that, although ecological effects were not a trigger in the Special Review (which is currently based on human health issues), the Agency was nonetheless concerned about the existence of widespread residues ''because they may have the potential to affect aquatic organisms and terrestrial plants and their ecosystems.'' The notice concluded with a request to registrants and the public for information on a number of topics, including ecological effects.

To address the concerns expressed by the EPA, and to respond to the request for additional information, Ciba-Geigy requested that The Institute of Wildlife and Environmental Toxicology (TIWET) at Clemson University form a multidisciplinary expert panel (the Panel) to conduct a comprehensive aquatic ecological risk assessment (ERA) of atrazine. This paper is based on the report of the Panel. The assessment was based on the procedures prescribed in the *Framework for Ecological Risk Assessment* [3] and the *Report of the Aquatic Risk Assessment and Mitigation Dialogue Group* (ARAMDG) [4]. The information presented here is based on the report of this Panel [5].

Overview

In accordance with the recommendations of the *Framework for Ecological Risk Assessment* [3], the risk assessment was a three-step process with the following distinct phases: problem formulation, analysis, and risk characterization (Fig. 1; discussed in the following section). The report of the ARAMDG [4] provided guidance in the use of a tiered approach for specific technical recommendations for exposure and effects determinations and for risk characterization, in particular the use of probabilistic expressions of effect endpoints and environmental exposure concentrations. For risk characterization, this approach involves comparison of probability distributions of environmental concentrations with probability distributions of species response data as determined from laboratory single-species toxicity tests, field studies or microcosm studies. The overlap of these distributions is a measure of the risk to aquatic life. This approach has a number of advantages over assessments based on single hazard quotients based on the response concentration for the most sensitive species and the maximum environmental concentration. Most importantly, it allows estimation of the magnitude and likelihood of potential ecosystem impacts as opposed to ''worst case scenarios.''

Finally, although probabilistic procedures have improved risk characterization based on laboratory studies, atrazine has also been the subject of several aquatic field studies in microcosms and mesocosms. These studies provided information for the highest tier of our analysis.

Problem formulation

Stressor characteristics. The stressor considered here is atrazine and its primary degradation products. For the purpose of problem formulation, a preliminary assessment considered the following characteristics of atrazine important in determining exposure and potential effects on biota.

Exposure: Persistence; magnitude (environmental concentration); duration of residue pulses; frequency; seasonality; metabolism; and kinetics of accumulation and depuration.

Effects: Among-species tolerance distributions; population and community responses (microcosms/mesocosms); inhibitory nature of the effects on aquatic plants; interaction of duration and intensity of exposure on effects; and recovery rates.

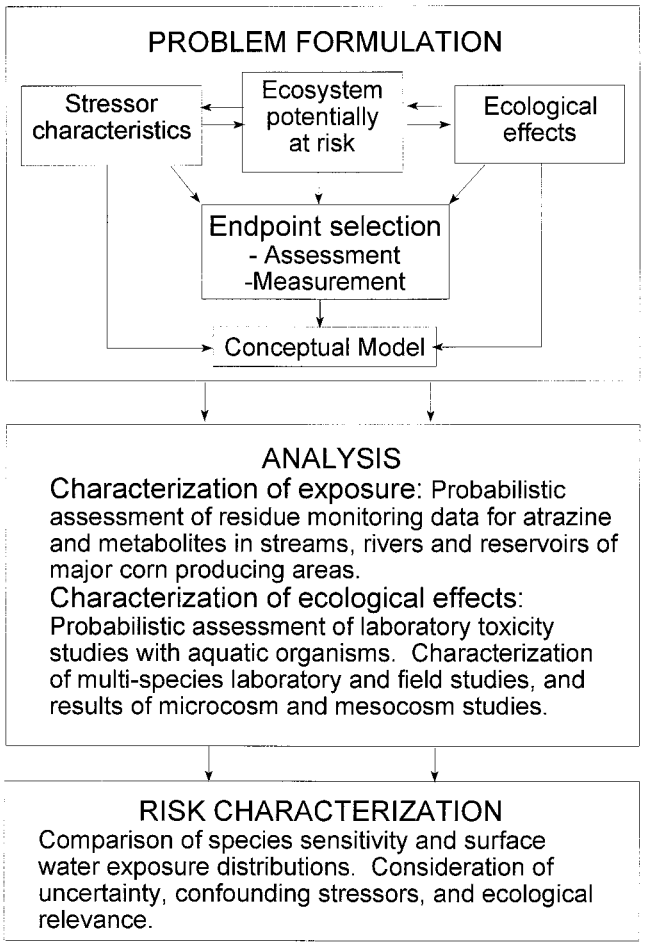

Fig. 1. Structure for risk assessment of atrazine in aquatic ecosystems.

Atrazine is a relatively mobile herbicide that produces a reversible inhibition of photosynthesis. The compound does not bioaccumulate appreciably and, although relatively persistent, is subject to abiotic and biotic breakdown. Some degradates are phytotoxic but much less so than the parent compound. Residue concentrations in small streams and rivers in agricultural watersheds are episodic, with major peaks in spring and early summer following applications, typically in May and June.

Use rates and geographic distribution. The estimated use of atrazine in 1993 was 32,000 to 34,000 t active ingredient [6]. Most atrazine use occurs in the Midwestern U.S. and southern Ontario (Table 1 and Fig. 2). Total 1993 atrazine use in the 12-state corn belt accounted for 83% of total corn applications and 69% of total atrazine use in the U.S. The Midwestern states also comprise the area of greatest number of acres planted in corn (Fig. 3). Applications of atrazine in the Midwestern states of Illinois, Iowa, Nebraska, and Indiana accounted for 52% of corn use (Table 1) and 43% of the total atrazine applied in the U.S. in 1993. Crops other than corn represented less than 20% of total atrazine applications (Table 2).

Much of the Midwestern U.S. receives high amounts of rainfall during the critical growing season (Fig. 4). We combined data on rainfall and atrazine use in a geographic information

Table 1. Geographic distribution of atrazine use on corn, 1993[a]

State	Tonnes used	% of use
Illinois	5,000	17
Iowa	4,200	14
Nebraska	3,200	11
Indiana	3,000	10
Ohio	1,700	5.6
Missouri	1,300	4.3
Michigan	1,100	3.7
Texas	1,100	3.6
Minnesota	1,100	3.5
Wisconsin	1,100	3.3
Kansas	1,000	3.3
Kentucky	1,000	3.2
Pennsylvania	750	2.5
New York	580	1.9
Tennessee	500	1.7
All other states	3,400	11
Ontario, Canada	584	NA

[a] Source [117,118].

system to define climate-use areas of greatest risk of atrazine runoff (Fig. 5).

Registered application rates (label use rates) of atrazine were reduced from 5.6 to 3.4 kg/ha in 1990. In a continuation of exposure reduction measures, current maximum preemergent label use rates on corn and sorghum are 1.8 to 2.2 kg/ha, depending on soil erosion characteristics and soil coverage by plant residues. Maximum total pre- and postemergent use rates of 2.2 to 2.8 kg/ha are currently allowed by the label. These allowed use rates were in effect for the 1993 season [7].

Although recommended label application rates for use in corn have been reduced to less than half of their pre-1990 rates, a corresponding reduction in total mass of atrazine used on corn has not occurred. This is primarily because application rates actually used in the field were often already less than the maximum label rates. In 1984, field use rates averaged slightly more than 2.2 kg/ha, and applications in the U.S. on corn totaled approximately 44,000 t (Fig. 6). Use rates dropped to slightly more than 1.6 kg/ha by 1987 and then ranged between 1.3 and 1.6 kg/ha through 1994. Total amounts of atrazine applied to corn during this period ranged from 28,000 to 34,000 t. The reduction in use rates between 1984 and 1987 is attributed to the introduction of formulations combining atrazine with the herbicide metolachlor. This combination requires less total herbicide than for either herbicide alone. With lesser label rates, steady or declining averages for actual use rates, implementation of expected regional use restrictions in sensitive areas, and continued introduction of herbicides in competition with atrazine, total application to corn may decline in the future.

Table 2. 1993 atrazine use by crop[a]

Crop	Tonnes applied	% of total use
Corn, U.S.	30,000	83
Corn, Ontario	584	99
Sorghum	4,000	11
Sugarcane	1,400	4
Other	720	2
Total U.S.	36,000	

[a] Source [118].

Physical/chemical properties and environmental behavior

Atrazine (Fig. 7) is sold as the active ingredient in a number of flowable, wettable powder, and water-dispersible granular formulations under a variety of trade names as a selective herbicide [8]. The herbicide is usually applied preemergence as a water-dispersed spray or in liquid fertilizer, although preplant and postemergent applications are occasionally used. Typically, a single application is made by ground equipment [6].

Movement in the environment. Atrazine has both a low vapor pressure (2.89×10^{-7} mm at 25°C) and low Henry's law constant (2.48×10^{-9} atm m^3 mol^{-1}), and thus volatilization from surfaces and water is negligible (Table 3). The moderate water solubility of atrazine (33 μg/mL at 22°C) and small K_d and K_{oc} (0.19 to 2.46 and 25 to 155, respectively, Table 4) favor movement of the chemical in the dissolved state from treated soil in surface or subsurface waters during irrigation or rain events. Based on these properties, atrazine is not expected to adsorb strongly to sediments and may partition only moderately from the water column. Once atrazine is in the water, the relatively small hydrolysis and aqueous photolysis rates (Table 3) can result in an extended presence in stationary water. Long-term concentrations in receiving waters are thus highly dependent on dilution and hydraulic residence time.

Persistence in aqueous systems. The fate of atrazine in aqueous systems is influenced by the *s*-triazine ring, which makes this herbicide resistant to microbial attack [9]. Due to this property, biodegradation may be less important than chemical degradation in the environment. Chemical degradation occurs by hydrolysis at carbon 2, by *N*-dealkylation at carbon 4, and by splitting the triazine ring [10]. In laboratory studies at 25°C over a pH range of 5 to 9, atrazine was found to be stable with no changes in concentration observed after 30 d [11] (Table 5). The environmental half-life has been reported to be 244 d at 25°C and a pH of 4; however, the addition of 2% humic acid decreased the half-life to 1.73 d (Table 5) [12]. This suggests that hydrolysis of atrazine may be catalyzed [9]. Half-lives of 34.8, 174, 398, and 742 d at pHs of 2.9, 4.5, 6.0, and 7.0, respectively, with the addition of 5 mg/L of fulvic acid (a concentration naturally occurring in surface waters) have been reported [13]. Hydrolysis of atrazine to the transformation product hydroxyatrazine followed first-order kinetics [13].

Photolysis of atrazine does not occur in water at wavelengths greater than 300 nm [14]. Some investigators have reported that, at a wavelength of 290 nm, the photolysis half-life of atrazine at a concentration of 10 mg/L at 15°C was 25 h, compared to a half-life of 4.9 h for identical conditions with acetone sensitizer added at a concentration of 1 mL/100 mL [15] (Table 5). The half-lives of sunlight-exposed ring-labeled atrazine at concentrations of 0.1 mg/L, under aerobic conditions, were reported to be 15 and 20 d for two estuarine sediments and 3 to 12 d for estuarine water [16]. The major short-term degradate was hydroxyatrazine, confirming previous observations [13].

Other environmental factors also contribute to the dissipation of atrazine in the environment. Atrazine has been reported to be inactivated by adsorption and degradation in estuarine water and sediments [16–19]. The half-life in water has been estimated to range from 3 to more than 90 d (shorter at elevated salinities). For sediment, the range was 15 to 35 d. The presence of hydroxyatrazine in sunlight-exposed estuarine sediments and water implies a low probability for atrazine accumulation in estuaries and a reduced rate of residual phytotoxicity in estuaries for the parent compound [16]. During a 3-year study, in situ atrazine

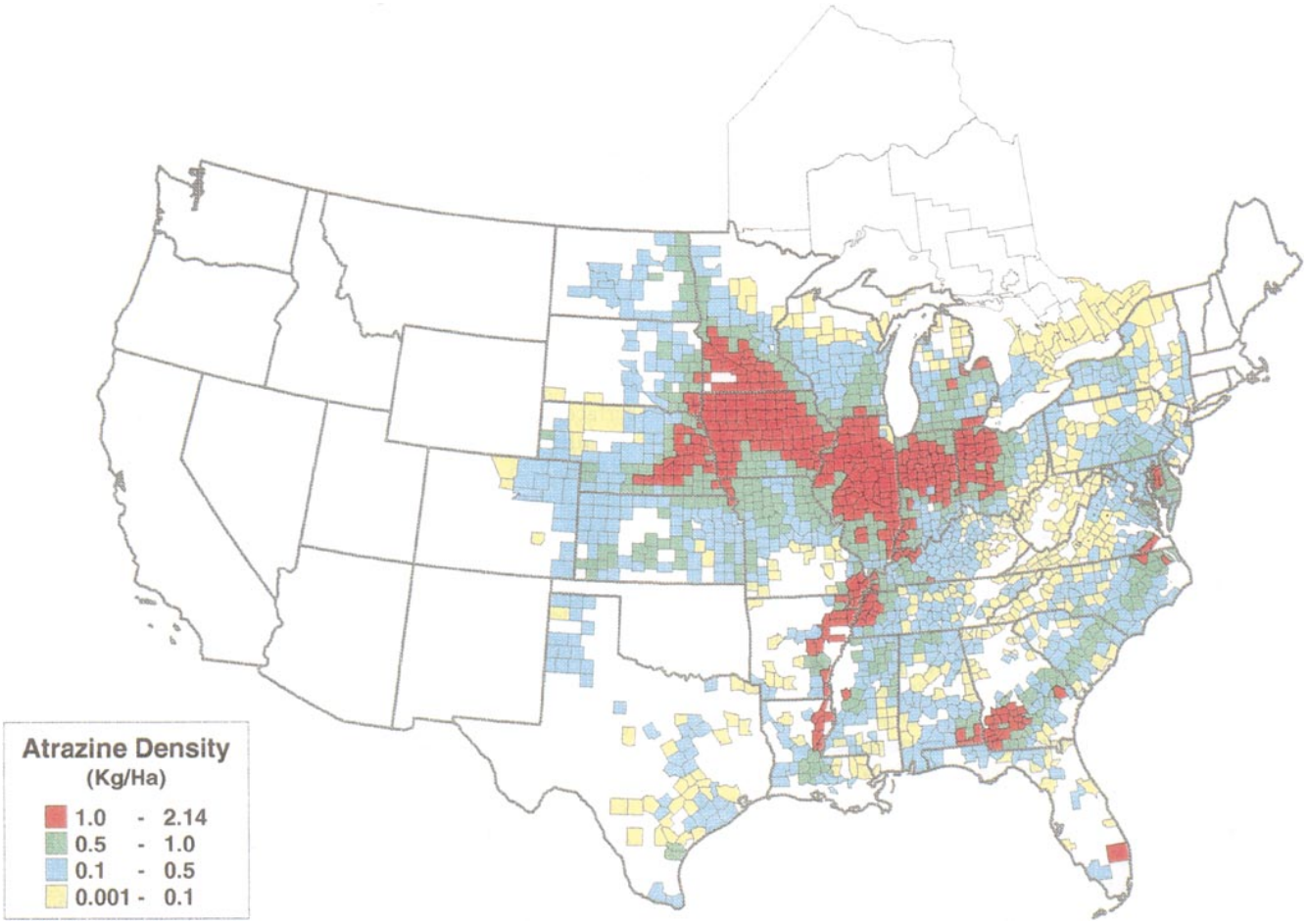

Fig. 2. Atrazine usage on corn per county, 1985–1988.

concentrations in the Wye River estuary suggested that atrazine degradation conforms to a first-order half-life of 30 d [18]. A 30-d half-life for atrazine has been reported in natural estuarine conditions [20]. Two microcosm studies with artificial lighting and constant temperatures estimated first-order half-lives of 3 to 4 months in estuarine water with plants and sediment [21,22]. The effect of salinity (5, 15, and 25 ppt) on degradation of atrazine at concentrations of 5, 50, and 500 μg/L was studied in estuarine and marine autoclaved water without sediment [23]. Atrazine did not exhibit significant loss after 128 d in any of the test conditions prepared with autoclaved water. A 17% loss was reported during a 128-d period in ambient Wye River water from the Chesapeake Bay watershed. Although discrepancies exist in the half-life estimates (Table 5), differences in the temperature, light source, sediment type, and concentration may account for much of the variation.

Transformation products. Known transformation products of atrazine are deethylatrazine (DEA); hydroxyatrazine (HA); deisopropylatrazine (DIA); diaminochlorotriazine (DAC); and two dealkylated hydroxytriazines, deethylhydroxyatrazine (DEHA) and deisopropylhydroxyatrazine (DIHA) (Fig. 7). Formation of these transformation products has been measured in laboratory studies, and some data are available for transformation products detected in the aquatic environment.

In aqueous photolysis studies using natural light, degradate amounts calculated as percentage of initial atrazine concentrations were determined to be 2.8, 2.6, 1.2, 0.9, 1.2, and 0.4% for DEA, HA, DIA, DAC, DIHA, and DEHA, respectively (Table 6) [11]. Slightly greater proportions of DEA, HA, and DIA (6.4, 6.6, and 2.6%, respectively) were observed in an anaerobic aqueous metabolism study with Georgia sandy clay. Atrazine and its analogues are also metabolized in plants, particularly in resistant plants such as corn [24]. Prior exposure to atrazine and other triazines in plants appears to stimulate production or activity of the enzymes (glutathione-*s*-transferases) responsible for detoxification. Thus, short exposures to small, nontoxic concentrations will protect the plant from subsequent exposure concentrations that would normally be toxic [25].

DEA would be expected to be more mobile in the aquatic environment than parent atrazine or other degradates because of its lesser K_d and K_{oc} values (Table 4). According to measured K_d and K_{oc} values, HA is expected to be least mobile in soil/water systems and would be transported in aqueous systems with greater proportions bound to suspended particulate matter.

Information on persistence of atrazine transformation products is limited. Soil half-lives for DEA, DIA, DAC, and HA have been reported to be 26, 17, 19, and 121 d, respectively [26]. However, aqueous half-lives of atrazine degradates are not available. Bioconcentration data for atrazine transformation products are also limited, because studies reporting biological concentrations focus on metabolite production in biota upon exposure to atrazine, rather than uptake of these degradates from environmental media.

Bioconcentration and bioaccumulation. Based on measures of bioconcentration factors (BCFs) and uptake data presented in the literature [27], atrazine is not expected to bioconcentrate. Food-chain biomagnification is also expected to be negligible. Its relatively small octanol–water partition coefficient (log K_{ow} = 2.68 at 25°C, Table 3) and its susceptibility to metabolism and rapid elimination combine to produce small BCFs in most tested species (Table 7). In addition to reported BCF values for

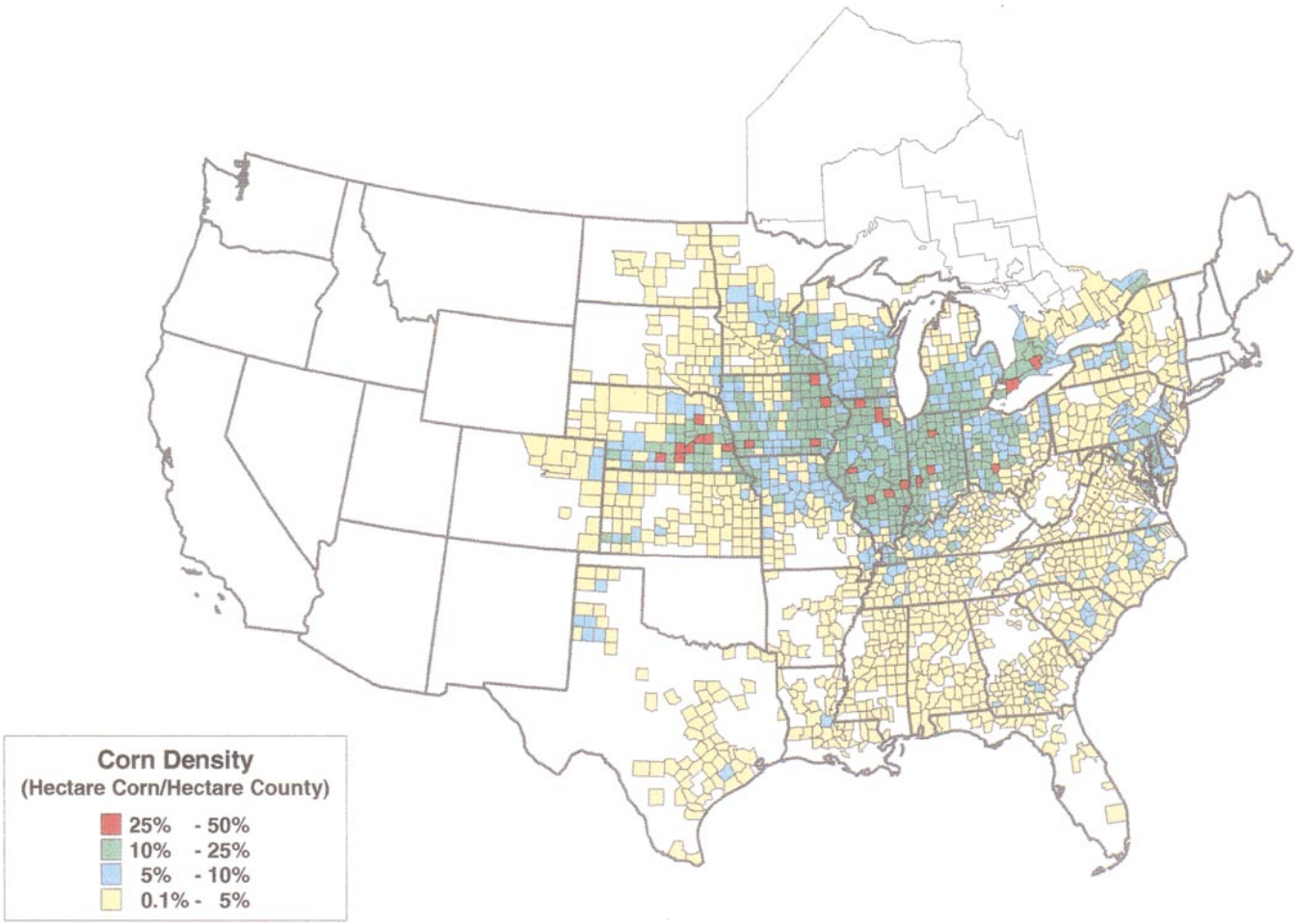

Fig. 3. Area planted in corn per county, 1985–1988.

aqueous exposure, no accumulation was seen in mollusks, leeches, Cladocera, or fish for atrazine exposure via the diet [28, 29].

Ecosystems potentially at risk

To determine ecosystems potentially at risk from atrazine, we considered (1) where, when, and in what quantities atrazine is used, (2) available surface water monitoring data, and (3) the stressor characteristics discussed previously. Based on these considerations, the ecosystems at greatest risk are associated with streams, rivers, and reservoirs of the Midwestern corn-growing regions of North America. These water bodies annually receive runoff from agricultural fields treated with atrazine. During the postapplication period, peak atrazine concentrations in these ecosystems may be within the ranges that cause effects in laboratory bioassays. The most susceptible organisms within these systems are phytoplankton, periphyton, and macrophytes, which may be affected directly. Aquatic animals may be affected indirectly via effects on food supply or changes in the physical habitat (macrophyte beds).

Because of where atrazine is used and the time of year that it is applied, algae and macrophytes in first- and second-order streams are expected to be exposed to the greatest concentrations. However, these exposures are expected to be of relatively short duration and, over exposure periods of a few days, the effects of atrazine on plants may be completely reversible. Aquatic plants in impounded waters may be exposed for longer periods of time. The assessment addresses both lentic and lotic situations.

Estuaries and nearshore coastal regions were also considered. Although atrazine monitoring data are limited for these areas, such as mainstem Chesapeake Bay and the Gulf of Mexico, available information indicates that exposures in these areas are typically much smaller than in the greater Mississippi basin and appear to be in less than toxic concentrations (K. Balu, personal communication) [30,31]. We determined, therefore, that the risk assessment for atrazine should focus on freshwater systems in the U.S. Midwest.

Effects on nontarget terrestrial systems were evaluated briefly. The acute and chronic toxicity of atrazine to terrestrial animals, the typical method of application (a single, ground-applied, soil-directed spray), its lack of bioaccumulation potential, and relative ease of metabolism and excretion indicated reduced cause for concern in these environments. Some drift to nearby nontarget vegetation may occur. This risk was not evaluated quantitatively. However, in the judgment of the Panel, field edges are typically disturbed communities of abundant, widespread species. Occasional localized and reversible impacts on plant growth may affect physical habitat for some wildlife but were judged to be a minor concern relative to aquatic risk.

Endpoints. Two types of endpoints were distinguished in the *Framework for Ecological Risk Assessment* [3]: assessment endpoints, which are explicit expressions of the actual environmental value that is to be protected; and measurement endpoints, which are measurable responses to a stressor that can be correlated with or used to predict changes in the assessment endpoints [32].

We focused on primary productivity, sustainability of aquatic macrophyte community structure, and long-term viability of fish populations as assessment endpoints that address the integrity of ecosystem structure and function.

Within each stratum of laboratory and field testing, measurement endpoints differ, whereas assessment endpoints remain

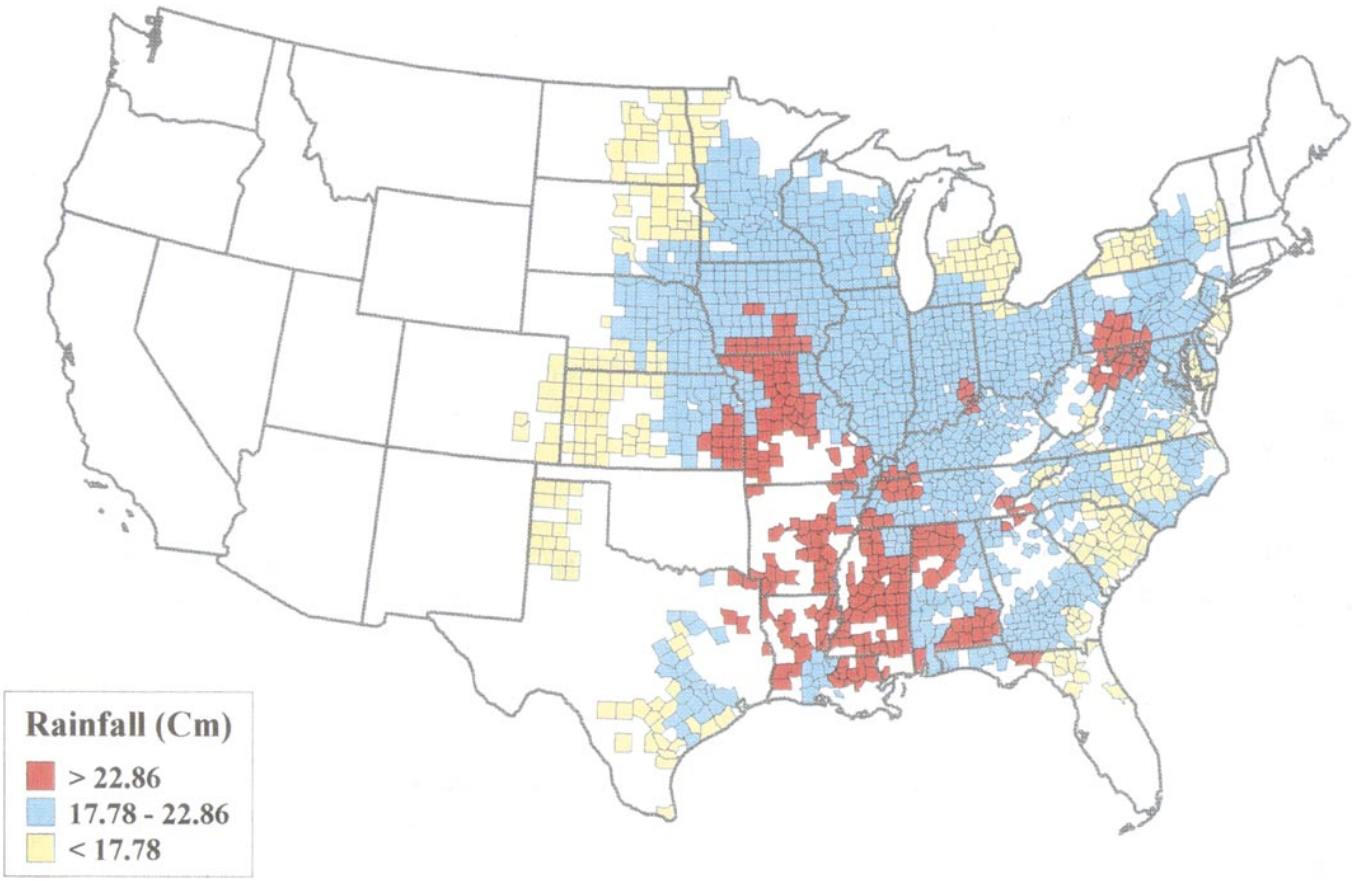

Fig. 4. Average cumulative rainfall during the first 60 days following corn planting.

the same (Table 8). In characterizing effects with laboratory studies, measurement endpoints included acute and chronic toxic responses such as survival, growth, and reproduction. When considering the more complex tests such as microcosms and mesocosms, measured responses converged toward assessment endpoints and included population trends over time, plant biomass changes, and community/ecosystem productivity.

If the assessment had been based on laboratory testing alone, estimates of risk would have to be conservative (i.e., wider margins of uncertainty). However, because the risk assessment encompassed more data on actual exposure and effects, the panel believed more conservative assumptions were unnecessary and more accurate assessments of risk were possible. That is, field test data allowed direct inferences about population- and ecosystem-level effects. The uncertainty associated with assessment endpoints was thus reduced, resulting in more accurate estimates of risk and greater understanding of and confidence in cause–effect interpretations. This does not imply that laboratory studies play a subsidiary role or are discounted in the ecological risk assessment. In fact, they are essential to probabilistic risk assessment.

Conceptual model

The problem formulation culminated with development of a conceptual model where the preliminary analysis of the ecosystems at risk, stressor characteristics, and ecological effects was used to define possible exposure and effect scenarios. The major goal was development of a series of working hypotheses regarding how atrazine might affect exposed ecosystems. The conceptual model was based on information about the ecosystems potentially at risk and the relationship between measurement and assessment endpoints. Although several hypotheses can be generated during problem formulation, only those considered most likely to be related to risks were selected for further evaluation in the analysis phase. As acknowledged in the *Framework for Ecological Risk Assessment* [3], professional judgment is necessary in the selection of risk hypotheses. The conceptual model describes the approach that will be used for the analysis phase and the types of data and analytical tools that will be needed.

Under the conceptual model, a wide range of hypotheses about the effects of atrazine on aquatic ecosystems could be considered, including interactions with the abiotic environment and impacts on ecosystem structure and function. The principal hypotheses considered were the following:

Atrazine may cause temporary, reversible reductions in plant productivity. If exposures are long enough, and the periods between exposures are short enough, total plant biomass may also be reduced.

Atrazine may cause damage to the community structure of macrophytes and reduce the ability of the aquatic habitat to sustain populations of other organisms, such as invertebrates and fish.

Direct effects of atrazine on aquatic plants may result in adverse affects on survival, growth, or reproduction of herbivores and predators, including fish.

These hypotheses were tested by assessing laboratory, field, and other data and are presented in the detailed description of

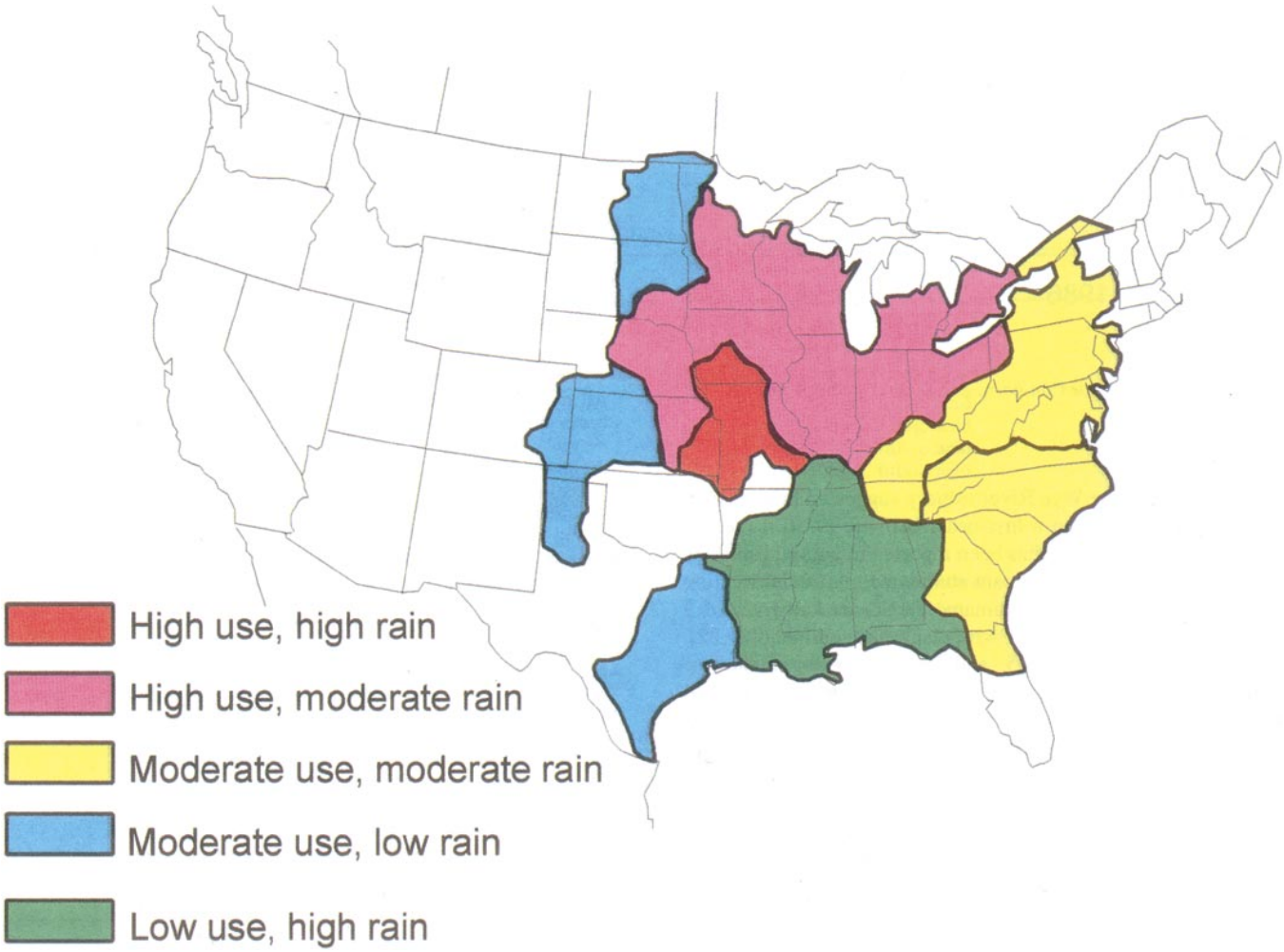

Fig. 5. Regions of potential risk of atrazine runoff based on climate and atrazine use level.

the exposure and effects characterization and the risk assessment (Fig. 1).

The response of organisms to chemical exposure depends on the mechanism of action and the interaction between duration and intensity of exposure [33]. The primary mechanism of action of atrazine on plants is lack of photosynthate. Plants can recover from effects of phytostatic compounds such as atrazine if the duration of exposure is short enough and the period between exposures—when concentrations are less than the threshold for inhibition—is long enough for the plants to recover. This is particularly so if the plants are able to detoxify the atrazine. The response of plants to the effects of atrazine is therefore a function not only of dose (concentration) but of dose rate (concentration over time). Plants can be exposed to pulsed exposures of inhibitory compounds without adverse effect on overall biomass, but total productivity may be reduced. With the short generation times of most algae and the rapid growth rates of aquatic macrophytes, atrazine effects would not be expected to cause secondary effects on higher trophic levels unless exposure was of sufficiently long duration to irrevocably damage organisms.

The phytostatic nature of atrazine makes assessing ecological risk difficult because of uncertainty about duration of exposure relative to the ability of primary producers to recover photosynthetic capacity when exposure ceases. For toxicants that cause acute lethality or effects on growth or reproduction from which individual organisms cannot recover, the effects of pulsed exposures are usually cumulative; the cumulative dose can be calculated as the product of duration and intensity of exposure (concentration). However, because some plants can completely recover from the effects of atrazine, this model is not appropriate unless the time between exposures is short.

The effects characterization considered all available acute and chronic effects data. Typically, acute data were reported for exposures less than or equal to 4 d, whereas chronic data were reported for exposures longer than 9 d. The risk assessment compared toxicity data with appropriate exposure data. The techniques used are discussed in more detail in the sections on the characterization of exposure and effects.

It is known that nutrient supply and light intensity are water-quality parameters critical for aquatic plant growth [32]. Moreover, these parameters fluctuate seasonally and with agricultural practice. A simple model was used to evaluate the effects of atrazine on algae during the spring runoff period when the concentrations of atrazine and suspended solids in surface waters are greatest. This approach enabled interpretation of the effects of atrazine in the context of light limitation due to turbidity caused by suspended solids in the water column. The recovery of algae from temporary effects of xenobiotics is rapid compared to zooplankton or fish. Doubling times of phytoplankton range from a few days to as little as 3 h [34–36].

Effects on primary producers, such as reduced fixation of

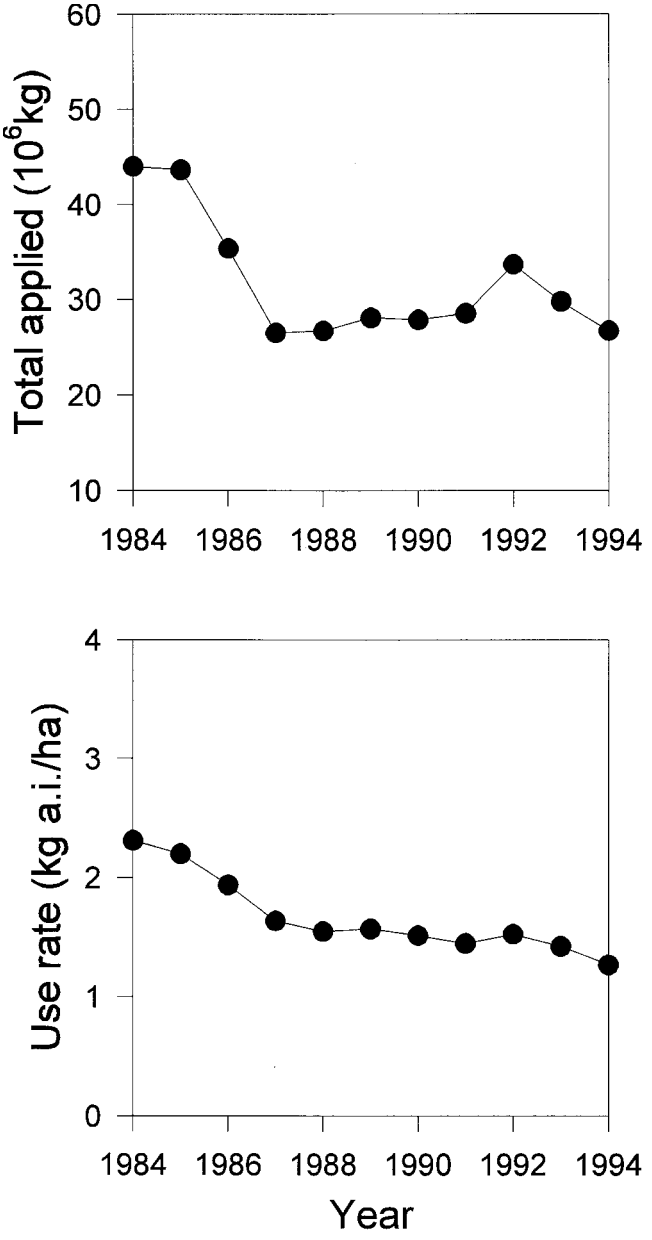

Fig. 6. Atrazine use rates on corn, and total amount of atrazine applied, 1984–1994.

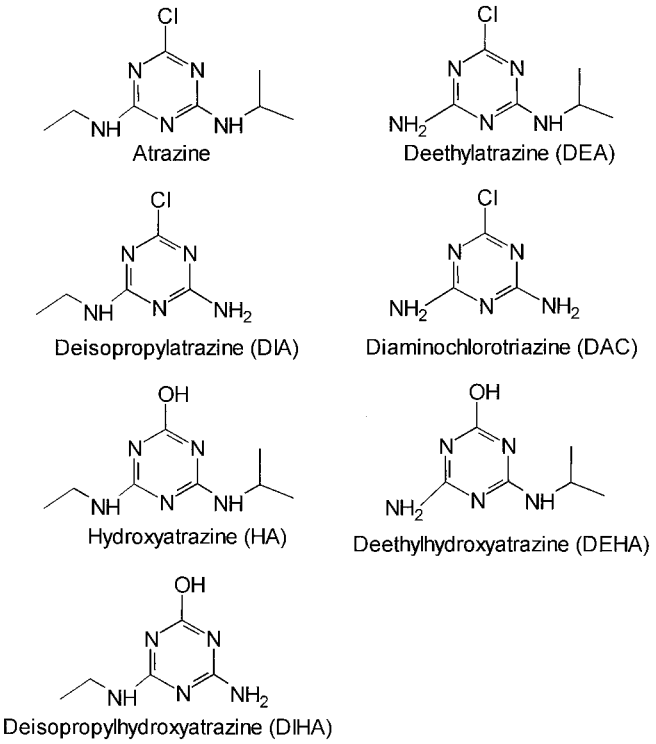

Fig. 7. Chemical structures of atrazine and degradation products.

carbon, leading to reduced carbohydrate synthesis may be transmitted from herbivorous animals through other secondary producers and, finally, to top predators such as fish. This linkage between primary production and fish production [37] is an important aspect of potential effects of atrazine on aquatic communities. Survivorship of young fish in the 1 or 2 weeks posthatch is typically low, even in uncontaminated systems. Small changes in ultimate survival rates at this stage may have great effects on the size and condition of the subsequent adult population, depending on the reproductive strategy [37]. Survival of young fish is strongly influenced by the success of juveniles feeding on phytoplankton and zooplankton. Phytoplankton clearly can be affected directly by atrazine, and zooplankton may be affected indirectly by food reduction or by modification of the physical habitat via removal or reduction of macrophytes [38]. Loss of physical habitat appears to influence the survivorship of zooplankton by increasing the frequency with which they are captured (fewer refugia). These examples demonstrate the complexity of ecological interactions and the usefulness of ecosystem-level studies for evaluation of impacts. Atrazine may be one of the few pesticides for which sufficient field testing exists to address such risks, and this analysis constituted the highest tier of our assessment.

Exposure

This exposure characterization evaluated the agricultural use pattern for atrazine, its physical and chemical properties, degradation products, and available surface water monitoring stud-

Table 3. Physical and chemical characteristics of atrazine[a]

CAS number	1912-24-9
Chemical name	2-chloro-4-ethylamino-6-isopropyl-amino-1-*s*-triazine
Ciba number	G-30027
Molecular weight	215.70 g/mol
Molecular formula	$C_8H_{14}N_5Cl$
Melting point	175–177°C
Water solubility	33 μg/mL at 22°C
Vapor pressure	2.89×10^{-7} mm Hg at 25°C
Henry's law constant	2.48×10^{-9} atm m^3 mol^{-1}
Log K_{ow}	2.68 at 25°C
Hydrolysis	stable for 30 d at pH 5–9 at 25°C
Aqueous photolysis	Natural light: $t_{½}$ 335 d at pH 7 Mercury lamp: $t_{½}$ 17.5 h at pH 7
Soil photolysis	Natural light: $t_{½}$ 12 d Mercury lamp: $t_{½}$ 5 d Xenon lamp: $t_{½}$ 45 d
Aerobic soil metabolism	$t_{½}$ 146 d, CA loam
Anaerobic soil metabolism	$t_{½}$ 77 d, CA sandy loam $t_{½}$ 159 d, CA loam
Anaerobic aqueous metabolism	$t_{½}$ 608 d, GA sandy clay

[a] Source [11].

Table 4. Partition coefficients for atrazine and major metabolites[a]

	ATZ		DEA		HA		DIA		DAC	
Soil type[b]	K_d	K_{oc}	K_d	K_{oc}	K_d	K_{oc}	K_d	K_{oc}	K_d	K_{oc}
MD clay	2.46	87.0	1.02	36.1	389	13,797	2.73	97	1.56	55
MD sand	0.20	39.0	0.06	12.2	1.98	374	0.16	30	0.16	31
MD sandy loam	0.79	70.0	0.36	31.8	6.52	583	0.51	45	0.65	58
CA loam	0.73	155.0	0.21	44.9	12.1	2,573	0.27	58	0.36	76
CA sandy loam	0.19	25.3	NA	NA	NA	NA	NA	NA	NA	NA

[a] ATZ = atrazine, DEA = deethylatrazine, HA = hydroxyatrazine, DIA = deisopropylatrazine, DAC = diaminochlorotriazine.
[b] MD clay = 4.8% OM[1], 2.8% OC[2], pH 5.9; MD sand = 0.9% OM, 0.5% OC, pH 6.5; MD sandy loam = 1.9% OM, 1.1% OC, pH 7.5; CA loam = 0.8% OM, 0.5% OC, pH 6.7; CA sandy loam = 1.3% OM, 0.76% OC, pH 7.8. Source [11].

ies. Chemical fate and transport models were also evaluated as a tool for predicting surface water exposures. Where possible, data were analyzed to provide acute (4-d) and chronic (21-d) exposure estimates to facilitate comparison of exposure estimates with toxicity data with similar exposure times. In addition, probabilistic techniques were used to characterize high percentile of toxicologically relevant exposures as a preferred alternative to instantaneous maximum concentrations.

For a complete risk characterization of any chemical, a close integration between the measurement endpoints typically represented in the toxicity databases and the assessment endpoints of importance to the ecosystem, the public, and resource managers is necessary. Measurement endpoints (Table 8) used in this atrazine risk assessment included mortality, cell growth, biomass, and chlorophyll *a* biomass. The risk characterization process evaluates these measurement endpoints in relation to the assessment endpoints. Because of the reversible nature of phytoplankton and periphyton responses to atrazine in the environment, it is appropriate to place a large degree of confidence in the results of micro- and mesocosm tests that integrate responses to realistic exposures of an appropriate duration.

Effects

Through literature searches, review papers, and test results, a comprehensive database of ecological effects information, including toxicity testing with 47 saltwater species and 85 freshwater species, was compiled. Acute and chronic single-species laboratory tests with aquatic plants, invertebrates, amphibians, and fish were evaluated. This review showed decreasing taxon sensitivity, based on geometric means of acute values for freshwater organisms, as follows: phytoplankton > aquatic macrophytes > benthos > zooplankton > fish. Effects have been reported for atrazine concentrations as small as 1 μg/L for a freshwater phytoplankton, 5 μg/L for a saltwater macrophyte, and greater than 220 μg/L for freshwater benthos and fish. We also considered 20 multispecies microcosm and field mesocosm studies. These studies showed that atrazine impacts on aquatic communities required greater concentrations than would be predicted from single-species laboratory bioassays.

Probabilistic risk assessment

As pointed out in the report of the ARAMDG [4], regulatory decisions with regard to registration of new pesticides are usu-

Table 5. Atrazine half-lives in aqueous systems

Study	$t_{½}$ (d)	Conditions
Laboratory hydrolysis studies		
Li and Feldbeck [12]	244	25°C, pH 4
	1.73	25°C, pH 4, 2% humic acid
Khan [13]	34.8	pH 2.9, 5 mg/L fulvic acid
	174	pH 4.5, 5 mg/L fulvic acid
	398	pH 6.0, 5 mg/L fulvic acid
	742	pH 7.0, 5 mg/L fulvic acid
Ciba [11]	No change after 30 d	pH 5–7
Aqueous photolysis studies		
Burkhead and Guth [15]	1	15°C, 290 nm
	0.2	15°C, 290 nm, 1 ml/L acetone
Jones et al. [16]	15–20	Aerobic, natural light, estuarine sediment
	3–12	Aerobic, natural light, estuarine water
Ciba [11]	335	pH 7, natural light
	0.73	pH 7, mercury lamp
Combined effects		
Hall et al. [23]	No loss after 128 d	Autoclaved estuarine and marine water
	17% loss during 128	Wye River water
Ciba [11]	608	Anaerobic with GA sandy clay
Field or microcosm evaluations		
Glotfelty et al. [18]	30	Wye River estuary
Ballantine et al. [20]	30	Estuarine conditions
Cunningham et al. [21]	90–120	Estuarine microcosm
Kemp et al. [22]	90–120	Estuarine microcosm

Table 6. Occurrence of atrazine transformation products in laboratory tests[a]

Test	Maximum % of applied atrazine found (day of maximum concn.)					
	DEA	HA	DIA	DAC	DIHA	DEHA
Aqueous photolysis						
Natural light	2.8 (15)	2.6 (15)	1.2 (6.9)	0.9 (15)	1.2 (6.9)	0.4 (15)
Mercury lamp	1.5 (0.062)	65 (0.21)	1.2 (0.33)	10 (2)	3.9 (2)	0.6 (2)
Soil photolysis						
Natural light	19.2 (3.5)	—	7.9 (7)	6.8 (22)	—	—
Mercury lamp	9.2 (2)	9.6 (3)	—	4.1 (3)	—	—
Xenon lamp	13.3 (30)	—	—	11.9 (30)	—	—
Aerobic soil metabolism						
CA loam	4.18 (244)	0.7 (62)	1.61 (244)	0.7 (3)	—	—
Anaerobic soil metabolism						
CA loam	2.1 (32)	0.4 (94)	0.7 (32)	0.3 (32)	—	—
Anaerobic aqueous metabolism						
GA sandy clay	6.4 (275)	6.6 (183)	2.6 (275)	—	—	—

[a] DEA = deethylatrazine, HA = hydroxyatrazine, DIA = deisopropylatrazine, DAC = diaminochlorotriatrazine, DIHA = deisopropylhydroxyatrazine, DEHA = deethylhydroxyatrazine. Source [11].

ally made on the basis of (1) estimates of exposure through the use of models, (2) responses from single-species laboratory toxicity tests, and, in some cases, (3) field studies. Throughout the useful life of a pesticide, risk may be reevaluated on a regular basis or as needed. At these times, additional studies of exposure or responses, that can now be derived from actual field measurements, can be used to refine risk estimates.

The estimation of exposure concentrations of pesticides to aquatic organisms is an important part of the risk characterization process for aquatic ecosystems. In the currently used processes of risk characterization, exposure concentrations can be developed in several ways. These range from actual measured environmental concentrations (MECs) in environmental matrices to estimated environmental concentrations (EECs) derived from computer-simulated scenarios. However, most of these procedures result in single concentrations. The likelihood of occurrence of these concentrations is unknown if they are not placed in a probabilistic framework that takes into account their variability. Recently, procedures have been proposed that take this variation into account by providing distributions of environmental concentrations rather than single values [4,39–42]. Such probabilistic environmental concentration distributions can be used to estimate how frequently the concentrations of pesticides will exceed any given toxicity threshold.

In protecting aquatic environments, the range of sensitivity

Table 7. Reported bioconcentration factors (BCFs) for atrazine

Species	BCF	Reference
Bluegill sunfish	12	[11]
Bluegill sunfish	<2.1	[119]
Whitefish fry	4–5	[28,29]
Brook trout	<0.27	[119]
Fathead minnows	<2.1	[120]
Fathead minnows	0.9	[121]
Mottled sculpin	2.0	[122]
Golden ide	1.0	[123]
Black bullhead	0.3	[119]
Fish	11	[124]
Bullfrog tadpoles	Not detectable	[125]
Annelids	4	[122]
Mayfly nymphs	480	[122]
Freshwater snail	4–5	[28]
Snails	7.5	[124]
Daphnids	2.2–4.4	[28]
Algae	76	[124]
Soil fungi and bacteria	87–132	[126]

Table 8. Assessment and measurement endpoints used in assessing the risk associated with atrazine residues in surface waters in North America

Assessment endpoints	Measurement endpoints
Primary productivity of phytoplankton and periphyton	Lower tier, laboratory-based data Sensitivity of phytoplankton and periphyton to atrazine using endpoints related to population growth (increase in numbers) Higher tier, field-based data Population trends over time, including recovery Changes in community biomass, chlorophyll content, and photosynthesis rates Sensitivity of phytoplankton and periphyton to confounding stressors that may be present at the same time as atrazine
Sustainability of aquatic macrophyte community structure	Lower tier, laboratory-based data Sensitivity of macrophytes to atrazine using endpoints related to survival and growth Higher tier, field-based data Population trends over time Changes in biomass, chlorophyll content and photosynthesis rates
Long-term viability of fish populations	Lower tier, laboratory-based data Sensitivity of fish to atrazine using endpoints related to survival, growth and reproduction Sensitivity of fish prey organisms (zooplankton and benthos) to atrazine using endpoints related to survival, growth and reproduction Higher tier, field-based data Fish survival and population trends over time Population trends of prey organisms over time

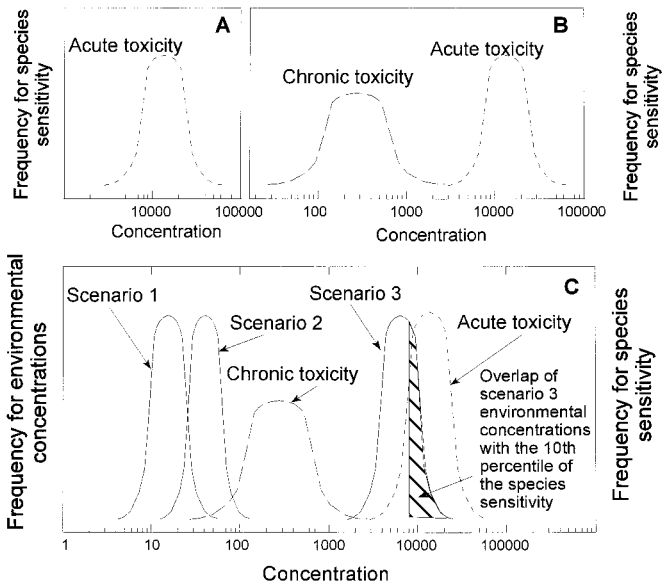

Fig. 8. Diagram illustrating distributions of stressor concentrations and species sensitivity. The degree to which the distribution of environmental concentrations overlaps with the distribution of acute or chronic sensitivity indicates the risk.

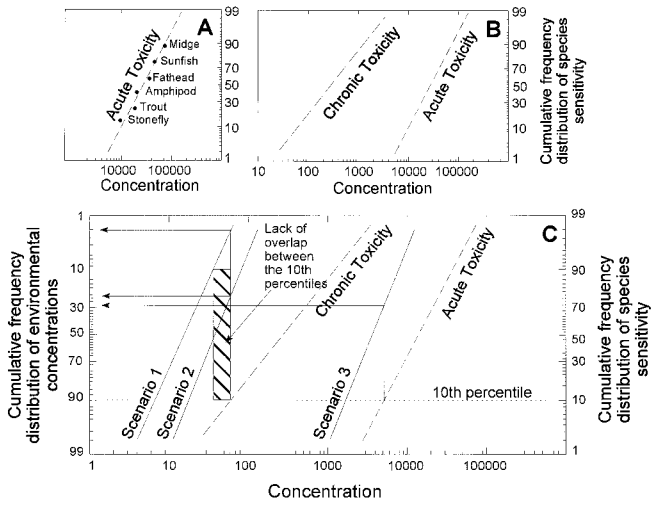

Fig. 9. Diagram illustrating the comparison of cumulative frequency distributions of environmental concentrations of a stressor with concentrations causing acute and chronic toxicity. Lines plotted using log and Pearson Type III scales on the *x*- and *y*-axes, respectively. The rectangle illustrates the lack of overlap between the 10th percentile of the chronic sensitivity distribution and the 90th percentile of the environmental concentration distribution for scenario 1. The horizontal arrows indicate the intercept of the 10th percentile of the sensitivity distribution with the environmental concentration distribution and indicate the probability that this concentration will be exceeded.

of species to substances must also be taken into account. Traditionally, risk assessments have been based on the effect concentration of the most sensitive organism or group of organisms. This may be made more conservative by the use of a safety or application factor [43]. In the absence of an adequate range of toxicity tests, these risk assessments may be under- or overprotective. However, where an acceptable range of toxicity data is available, the inherent variation in receptor response is better defined and use of safety factors may not be warranted. As with the environmental concentrations, assessment of toxicity should include the distribution of sensitivities.

Expressing the results of a refined risk characterization analysis as a distribution of toxicity values rather than a single-point estimate is an approach presently being used by the Dutch government [39] and others [41,44] and has been recommended for assessing risk from pesticides in the aquatic environment [4]. A major advantage of this approach is that it uses all relevant single-species toxicity data and, when combined with exposure distributions, allows quantitative estimations of risks to aquatic organisms.

The principle is illustrated in Figures 8 and 9, adapted from [40] and [4]. The distributions of measured acute and chronic sensitivity are assumed to represent the universe of species and to be log-normal with respect to concentration, shown as log-normal distributions in Figure 8A and B and cumulative frequency distributions in Figure 9A and B. The degree of overlap of the exposure curve with the effects curves can be used to estimate the probability that a percentile of species may be adversely affected on a percentile of occasions (Fig. 8C, scenario 3). It is easier to estimate the intercepts and overlaps of these frequency distributions (Fig. 8) when they are linearized via the cumulative frequency distribution approach (Fig. 9).

There are several possible outcomes of this approach. The distribution of environmental concentrations (scenario 1, Figs. 8 and 9C) has little overlap with the chronic toxicity distribution, which suggests that there would be little impact from this exposure situation. Reading from the intercept of the 10th percentile of the chronic sensitivity distribution shows that this concentration is exceeded only 2% of the time. The distribution of scenario 2 (Figs. 8 and 9C) is such that more overlap is present. Reading from the intercept of the 10th percentile of the chronic sensitivity distribution shows that this concentration is exceeded 22% of the time. In this situation, it is likely that chronic responses would be observed quite frequently in some species unless mitigation measures can shift the entire environmental concentration distribution to lesser concentrations or increase the steepness of the slope. This could be done by identifying either situations that lead to the greater environmental concentrations or the more sensitive species and restricting the use of the product in those situations or in areas where the more sensitive species occur. In the case of scenario 3 (Figs. 8 and 9C), chronic responses will likely be observed in almost all instances (complete overlap with the chronic sensitivity distribution). Reading from the intercept of the 10th percentile of the acute sensitivity distribution shows that this concentration is exceeded 30% of the time and major mitigatory measures may be required.

In the Dutch system [39], distributions of chronic toxic effects no-observed-effect concentrations (NOECs) are used to estimate the concentration of a chemical at which only 5% of the species would be affected. In other words, at the chosen level of protection, there is a chance that 5% of the species may be affected. In using overlapping distributions, there is an assumption that protecting a certain percentage (i.e., 5 or 10%) of species for a certain proportion of occasions will also preserve ecosystem structure and function. Given the natural variability between sites within ecosystems and even between ecosystems, this assumption will probably hold. There are intrinsic limitations to this approach [4,39]. For example, the choice of protection level may not be socially or politically acceptable, especially if the potentially affected species include organisms of high ecological, commercial, or recreational value.

The selection of toxicity and exposure criteria may vary depending on characteristics of the stressor and the ecosystem

at risk. The panel chose to follow the ARAMDG [4] recommendation for using the 90th percentile of exposure and 10th percentile of sensitivity. In this assessment, these values were compared as a probabilistically derived quotient. In addition, the probability of exposures exceeding the 10th percentile of the sensitivity distribution was also considered. In view of the specificity of action of atrazine, the nature of the effects, and the redundancy of the more sensitive organisms in the ecosystems at risk, these criteria were judged appropriate for this risk assessment.

Use of a sensitivity distribution in developing criteria requires that the species selected for these tests be truly representative of the universe of species that may be present in the environment. More sensitive species must be included to make sure that species representative of greater sensitivity are present. This avoids wrongly concluding that the risk is low. Less sensitive species are required to ensure that the distribution is not artificially skewed to overrepresent the more sensitive species. This avoids concluding that the risk is high when, in fact, it is low.

Similarly, use of a concentration distribution requires that the concentrations selected be representative of the universe of concentrations that may be present in the environment. Locations or times of year with greater concentrations and others with lesser concentrations should be included to ensure that the full range of concentrations is adequately represented. Furthermore, concentrations at one location should be representative of the universe of concentrations at that location, but unfortunately, this is generally not the case. Some sampling programs emphasize concentrations present during the herbicide runoff season. Use of data sets from such programs without adjustment for the nonuniform sampling is likely to lead to substantial overestimation of the intended criterion, for example, the 90th percentile.

EXPOSURE CHARACTERIZATION

Exposure characterization using model simulations

Mathematical simulation models of fate and transport have been identified by a number of regulatory work groups, including the ARAMDG [4] and the FIFRA Exposure Modelling Work Group [45], as potentially valuable tools for improving regulatory decisions for pesticide registration. This is also expressed in the ''New Paradigm'' of the EPA Office of Pesticide Programs, where greater use of computer modeling is proposed rather than field monitoring studies [cited in 4]. A probabilistic modeling study was initiated as part of the ecological risk assessment of atrazine in order to evaluate exposure to aquatic nontarget organisms over a wider range of product use areas and climatic conditions than were available in known field data. Model scenarios consisted of simulating runoff of atrazine from treated fields using the EPA's Pesticide Root Zone Model (PRZM-2) [46] and the USDA's GLEAMS [47] as recommended by the ARAMDG [4].

Phase I of the model validation study investigated how well PRZM-2 and GLEAMS reproduced observed water and chemical runoff. To the extent possible, input files were structured from measurements taken at the test site during the field study. In the absence of site-specific data, best available sources were used such as temperature data from a nearby National Oceanic and Atmospheric Admistratation (NOAA) weather station. Input parameters related to physicochemical properties for atrazine were based on environmental fate summaries provided by Ciba-Geigy [20]. An average organic carbon–water partition coefficient (K_{oc}) of 87.8 was calculated from the results of mobility studies for parent atrazine for four soils: clay, sand, sandy loam, and loam. Soil–water partition coefficients (K_d) used in the models were calculated for each soil horizon at each test site based on the average K_{oc} multiplied by the organic carbon content of the horizon. Half-lives were calculated for each soil horizon using an equation derived from aerobic and anaerobic metabolism studies for parent atrazine in which atrazine degradation is expressed as a function of organic matter content:

$$t_{1/2} = k/om$$

in which $t_{1/2}$ is the degradation half-life for the soil horizon, k is an empirically derived coefficient, and om is the organic matter content of the horizon expressed as a fraction. The coefficient k was determined to be 2.12 ± 0.16 d from aerobic soil metabolism studies and 1.97 ± 0.04 d from anaerobic soil metabolism studies. An average k of 2.0 d was used for estimating the degradation half-life in all horizons. A plant uptake efficiency factor of 1.0 was assumed because of the solubility of atrazine in water. A value of 1.0 indicates that plant uptake of dissolved pesticide is directly related to the transpiration rate. These simulations are referred to hereafter as ''base runs.''

Discrepancies between observed and predicted chemical loss may result from a number of error sources, including the predictive capabilities in simulating hydrologic processes or associated pesticide physicochemical behavior. Phase II of the model validation involved calibrating the hydrologic portion only of each data set in order to minimize error related to water balance. Physicochemical properties for atrazine remained unchanged. Water runoff was calibrated by adjusting the Soil Conservation Service (SCS) curve number and initial soil-moisture content. The curve number provides an index of runoff potential and takes into account soil texture, land cover, and land management factors [48].

Phase III involved varying physicochemical properties for atrazine to determine the relative influence of specific properties on predicted atrazine runoff. These simulations were performed using PRZM-2. Sensitivity analyses were conducted by systematically changing K_{oc}, degradation half-lives, and compartment size in each hydrologic calibration data set to determine the influence of each parameter on event and cumulative atrazine loss in runoff. Details related to the model configuration and results for each study site have been omitted, and, for brevity, only the results of the hydrologic calibration are presented below.

These results were then validated against data collected from field studies. In 1985, Memphis State University performed a runoff monitoring study on an agricultural watershed in Shelby County, Tennessee [49,50]. Two runoff events containing measurable concentrations of atrazine occurred within the first 23 d of application. These events corresponded to rainfall amounts of 4.5 and 7.8 cm. Atrazine masses associated with these events were calculated to be 88.6 and 19.2 g, respectively. Initial model simulations (base runs) with both PRZM-2 and GLEAMS resulted in predicted water runoff within approximately 25% of observed volumes for each event. With PRZM-2, predicted losses of atrazine in runoff were greater than observed masses (1,318 g as opposed to 89 g for the first event). Atrazine runoff predicted by GLEAMS provided a better match to observed data (103 g compared to 89 g).

Hydrologic calibration involved decreasing the curve number by approximately 10% for both models and adjusting the initial water content in the soil. Results are shown in Figure 10.

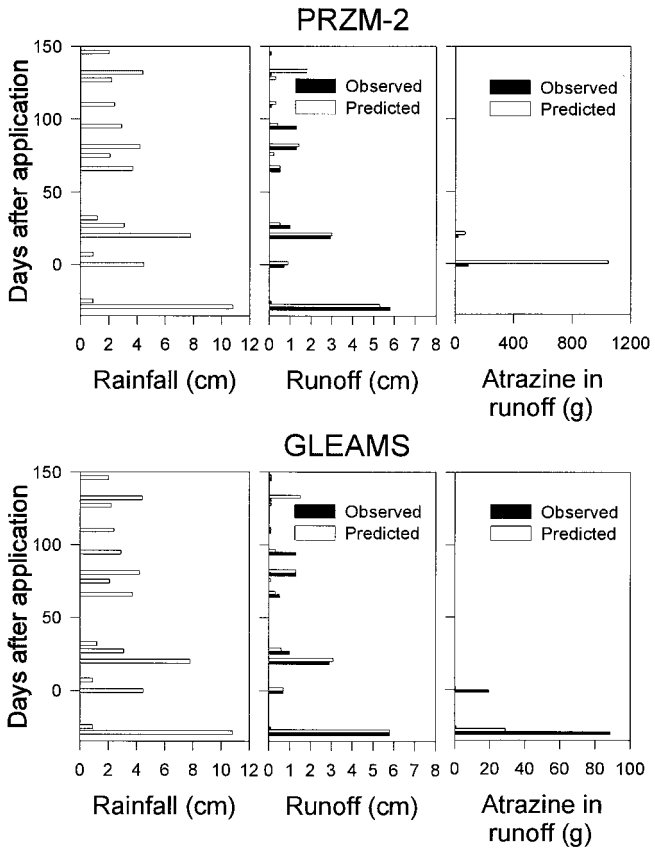

Fig. 10. Results of GLEAMS and PRISM II runoff models for Shelby County, Tennessee compared to actual measurements.

The predicted water runoff from both models was in good agreement with the experimentally measured values at the test site. The predicted atrazine losses in runoff were slightly improved for PRZM-2 (1,046 g) but noticeably worse for GLEAMS (28 g as opposed to observed mass of 89 g; D in Fig. 10). These results indicate that PRZM-2 overpredicted atrazine runoff and GLEAMS underpredicted atrazine runoff using soil adsorption and degradation rates based on laboratory data.

During the 1970s the EPA and the USDA collaborated on a number of field studies to generate a database for development and testing of mathematical models of pesticide and nutrient transport from agricultural lands [51]. Data were collected from four small watersheds ranging in size from 1.3 to 2.7 ha, located near Watkinsville, Georgia, in the northeastern part of the state. The sites were instrumented for monitoring environmental conditions including precipitation, air temperature, soil moisture and temperature, and water and sediment runoff. Atrazine was applied to two of the watersheds from 1973 to 1975. Site P2 (1.3 ha) was treated at rates of 3.36, 3.81, and 1.54 kg/ha for 1973, 1974, and 1975, respectively. The greater applications are consistent with the greatest recommended rates used at that time. Atrazine was applied to site P4 (1.4 ha) at rates of 3.36, 4.03, and 1.45 kg/ha. Soils at both sites were Cecil sandy loam, classified as hydrologic soil group B, having moderate runoff potential.

Certain model tendencies that had been observed in the Tennessee simulations were also observed with the Georgia simulations. With PRZM-2, base runs resulted in runoff mass of atrazine greater than observed for all years at both sites. Atrazine runoff predicted by GLEAMS was less than observed except where predicted water runoff was great. Runoff volumes were underpredicted by both models for 1973 and 1975 and near observed volumes for 1974. Hydrologic calibration was conducted only for 1973 and 1975 and involved increasing the curve number by approximately 10%. After hydrologic calibration, predicted atrazine runoff increased, with additional mass generated from relatively small volume precipitation events (1 to 2 cm) within the first week or two following application.

Although simulations included base runs in which input parameters were selected from commonly accepted and recommended data sources, in general, atrazine runoff was overpredicted by PRZM-2, typically by an order of magnitude. Atrazine runoff was typically underpredicted by GLEAMS. Runoff water volumes predicted by PRZM-2 and GLEAMS were generally similar to each other and similar to observed volumes. Calibrating water volumes affected peak event atrazine runoff by as much as 200%.

To determine the relative influences that specific variables have on predicted atrazine runoff, sensitivity analyses were performed on atrazine adsorption coefficients (K_{oc}), degradation rates ($t_{1/2}$), and model compartment size. Physicochemical properties for atrazine were changed to reflect ranges observed in mobility and metabolism studies. Compartment sizes were changed to reflect values typically used with PRZM-2. Predicted concentrations of atrazine runoff varied depending on the site and year simulated, but common trends occurred. Changes in K_{oc} had little impact on total atrazine runoff but profound impact on the distribution of mass between events. Higher K_{oc} tended to reduce atrazine runoff mass in early events but increase runoff mass in later events because additional compound was available for runoff. Changes in half-life had relatively little impact on initial runoff events but greater influence on events several weeks later. Compartment size had the most dramatic effect on atrazine runoff. The best results were achieved with the smallest compartment size (1 mm). Increased compartment sizes resulted in greater amounts of atrazine runoff, especially later in the season. This indicates that extremely small compartment sizes or the inclusion of nonlinear mixing zone in the soil in contact with the runoff water may be required in the current version of PRZM-2 to limit the depth from which atrazine is available for runoff.

Model predictions of atrazine concentrations, both in-stream and field-edge, were about an order of magnitude greater than concentrations observed in surface water monitoring studies [52,53] and field runoff studies [49,51]. Probabilistic modeling following the originally planned procedures for parameter estimation would likely have resulted in estimated exposure concentrations an order of magnitude greater than would occur in nature using PRZM-2 and unrealistically small using GLEAMS. These results underscore the need to work with a calibrated model. Improvements to the pesticide extraction algorithms in the models are recommended before either model can be used to broadly estimate runoff losses for pesticides having physicochemical properties similar to atrazine. Such improvements are currently being developed and tested against the above field data sets.

Model scenarios were therefore not utilized in this current risk assessment. Following a more thorough investigation into the predictive abilities of simulation models and their reliability for risk assessment, it may be possible to use these types of models to generalize the results of risk assessments in both space and time.

Table 9. Description of river data sets

Designation	Watershed size (km²)	Atrazine use (tons)	% in cropland
Maumee River, OH[a]	16,000	NA[c]	76
Sandusky River, OH[a]	3,200	NA	80
Honey Creek, OH[a]	390	NA	83
Rock Creek, OH[a]	88	NA	81
Lost Creek, OH[a]	11	NA	83
Cedar River, IA[b]	12,000	330	64
Iroquois River, IL[b]	5,200	210	71
West Fork, Blue River, NE[b]	3,000	160	61
Sangamon River, IL[b]	1,400	52	75
Silver Creek, IL[b]	1,200	21	53
Delaware River, IA[b]	960	24	46
Huron River, OH[b]	930	22	53
Old Mans Creek, IA[b]	500	10	53
Roberts Creek, IA[b]	260		47

Source: D.B. Baker and R.P. Richards (personal communication). Sampling years: 1983–1993. Sampling and analysis: Three samples per day were collected April 15–August 15; during runoff events, all three were analyzed; during low flow, two per week were analyzed; minimum of two samples per month were collected and analyzed at the balance of year. Whole water samples were analyzed. Results were not corrected for analytical recovery.
Source [55]. Sampling years: 1990–1991. Sampling and analysis: Storm events sampled every 3 to 8 h. Base flow was sampled either weekly or biweekly. Filtered water samples were analyzed. Results were not corrected for analytical recovery.
[c] Not available.

Methods for determination of stream and river exposures

Pesticide exposure in river and stream ecosystems can be influenced greatly by stream order and watershed size. Agricultural drainage ditches and lower-order streams draining small watersheds are subject to peak pesticide concentrations for short durations. Higher-order streams and rivers draining large watersheds typically exhibit lesser peak pesticide concentrations, but pesticides are detectable for longer periods of time. These phenomena are a direct result of differences in hydrological response time and patterns of merging of tributaries into the main stem. The shorter response time of a small stream results in a hydrograph duration on the order of hours or less and rapidly changing herbicide concentrations. Nearly simultaneous runoff of herbicides from all contributing areas leads to greater peak concentrations. In larger rivers, the hydrograph lasts for days or weeks, and the peak herbicide concentrations from different subbasins enter at different times, leading to an attenuated herbicide chemograph.

Field monitoring data can be used to describe exposure patterns provided that the monitoring data set is adequate to allow for accurate characterization of pesticide concentration distribution during a storm event (see discussion of error associated with sampling, below). Data sets from Ohio [54] and Iowa, Illinois, Nebraska, and Ohio [55] that meet these sampling frequency criteria (Table 9) were used in development of the stream and river exposure analyses. Because the effects of atrazine are reversible for short-term exposures, the entire data sets were used rather than the 90th percentiles of the 10-year maxima as suggested in [4].

Screening of stream and river raw data

All analytical concentration data were retained, including those with results less than the detection limits. Although results less than the detection limits are generally considered unreliable, they are the least likely concentrations to be of toxicological concern. When small-concentration data are censored or excluded from the analysis, statistical treatments of the concentration data sets contain biases. This can be avoided by retaining the data and assigning a zero or threshold value to the results for the purpose of determining distributions.

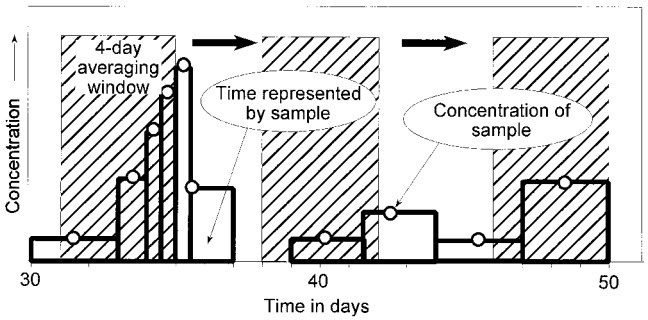

Fig. 11. Illustration of the window technique for calculating time-weighted mean concentrations. Only a portion of the record is shown and, for illustrative purposes, the times each sample represents has been set at a maximum of 1.5 days. The averaging window is moved through one day at a time.

Discrete exposure data. Atrazine concentrations were measured in discrete samples obtained during stream monitoring. Samples were collected more frequently during the atrazine runoff season than during the fall, winter, or early spring, and more frequently during runoff events than during low-flow periods within the atrazine runoff season. In order to normalize for this irregular sampling, each sample was associated with a time interval that the sample would represent. This time interval extended from a point in time halfway between the sample and the preceding sample to a point in time halfway between the sample and the following sample. Neither ''half-window'' was permitted to exceed 7 d in length. Time that did not fall within the time window of a sample was considered uncharacterized and was assigned a concentration of 0.1 μg/L.

Because of the structure of the sampling program, this ''uncharacterized'' time fell almost entirely during the fall and winter when concentrations were small [54]. Because the sampling program was stratified to collect samples more frequently during the spring period, when atrazine concentrations were greatest, the assignment of 0.1 μg/L to ''missing'' time periods did not affect the shape of the concentration-probability curves at greater atrazine concentrations, where overlap with the sensitivity distribution is more likely. The exposure values generated by this window technique were sorted by decreasing concentration and plotted as a function of cumulative time represented by samples. Probabilities of exceeding these values were determined with reference to the total time.

4-d and 21-d average exposures. To calculate 4-d and 21-d moving averages, time windows were assigned to the instantaneous observations in the manner described above. Windows 4 d or 21 d wide were moved through the time series, with the beginning of the window placed in succession on each day of the time series. The moving average was calculated from the atrazine concentrations data at each such position (Fig. 11). A data set with a duration of n days would produce $(n - 3)$ 4-d averages and $(n - 20)$ 21-d averages. The resulting data sets of averages were sorted in decreasing order and exceeded values were derived directly from percentiles of the data. For the Ohio rivers, each analysis led to nearly 4,000 estimated 4-d or 21-d averages.

Pulsed atrazine exposure periods. Eleven years of atrazine data for the Maumee River, Sandusky River, and Honey Creek in Ohio were analyzed to characterize the duration of exposure

Table 10. Atrazine 90th percentile values from probability distributions of instantaneous, 4-d, and 21-d average concentrations

River	Drainage area (km^2)	Concn. (μg/L)		
		Instantaneous	4 d	21 d
Lost Creek, OH	11	1.6	1.96	2.71
Rock Creek, OH	88	2.43	2.97	3.69
Roberts Creek, IA	260	—	2.73	9.07
Honey Creek, OH	390	4.22	4.69	4.75
Old Mans Creek, IA	500	—	4.37	7.09
Huron River, OH	930	—	5.60	5.73
Delaware River, KS	960	—	5.78	6.07
Silver Creek, IL	1,200	—	9.00	7.71
Sangamon River, IL	1,400	—	0.56	0.52
West Fork, Big Blue River, NE	3,000	—	1.79	2.29
Sandusky River, OH	3,200	3.13	3.51	3.86
Iroquois River, IL	5,200	—	3.40	3.52
Cedar River, IA	12,000	—	2.14	2.40
Maumee River, OH	16,000	3.40	3.47	3.44

periods. Atrazine concentrations were plotted as a function of Julian day for each year and water body with linear interpolation between data points. Data were analyzed during the annual peak exposure period only (May, June, July, and August). Exposures were calculated for periods when atrazine concentrations exceeded 2, 5, 10, or 20 μg/L (Fig. 12). These atrazine concentrations were in the range of concentrations reported to have effects on green algae, specifically *Selenastrum capricornutum.*

Estimates of exposure. Instantaneous, 4-d, and 21-d average exposures were estimated. Instantaneous 90th percentile values for the 14 systems were determined from the frequency distributions and ranged from 9.07 μg/L for Silver Creek, Illinois, to 0.89 μg/L for the Sangamon River, Illinois. The greatest and least 4-d 90th percentile values were 9.00 and 0.56 μg/L for Silver Creek and the Sangamon River, respectively. The greatest

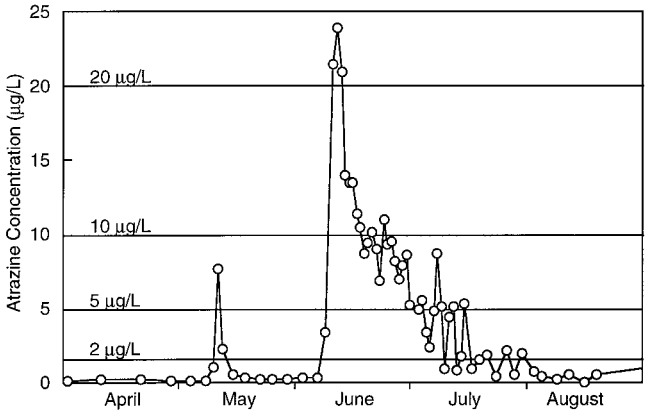

Fig. 12. Concentrations of atrazine in the Sandusky River, 1993.

and least 21-d 90th percentile concentrations were 9.07 and 0.52 μg/L for Roberts Creek, Iowa, and the Sangamon River, respectively. Instantaneous, 4-d, or 21-d 90th percentile concentration values were generally within a factor of 2 for a single river. As stream order increased, differences between these values lessened. In the curves for instantaneous, 4-d, or 21-d averages from discrete samples for Lost Creek, Ohio, and the Maumee River, Ohio, the smallest and largest rivers monitored, the 90th percentile values were similar for both systems (Table 10).

The curves also provide an estimate of the duration of time a given concentration is exceeded in each aquatic system. Table 11 illustrates the approximate percentage of time each river exceeded concentrations of 2, 5, 10, or 20 μg/L for the months of May through August.

There were few differences in the frequency of exceeded concentrations among the systems analyzed. Four-day and 21-d frequencies were similar within each system, with differences generally less than a factor of 2 between 4- and 21-d averages.

Table 11. Atrazine concentration exceedency frequencies for 4-d and 21-d averages in several watersheds[a]

Watershed and size (km^2)	% of time that concn. exceeds:							
	2 μg/L		5 μg/L		10 μg/L		20 μg/L	
	4 d	21 d	4 d	21 d	4 d	21 d	4 d	21 d
<100								
Lost Creek, OH	10	12	5	6	3	2	1	0
Rock Creek, OH	13	16	7	7	2	2	1	0
101–1,000								
Roberts Creek, IA	12	12	5	11	4	8	3	6
Honey Creek, OH	20	22	9	9	4	4	1	0
Old Mans Creek, IA	12	15	10	12	3	0	1	0
Huron River, OH	30	32	12	14	2	0	0	0
Delaware River, KS	16	20	12	13	8	5	0	0
1,001–10,000								
Silver Creek, IL	30	32	12	20	8	6	1	0
Sangamon River, IL	5	5	1	0	0	0	0	0
Big Blue River, NE	10	11	5	5	2	2	1	0
Sandusky River, OH	15	16	8	8	2	1	0	0
Iroquois River, IL	15	23	5	0	0	0	0	0
10,001–1,000,000								
Cedar River, IA	10	15	3	1	0	0	0	0
Maumee River, OH	17	18	7	7	2	1	0	0

[a] Source: Data of D.B. Baker and R.P. Richards (personal communication) for the years 1989–1992 and Scribner et al. [55] for the years 1990–1992.

Table 12. Concentrations of transformation products as a percent of parent atrazine in rivers[a]

	Mean (μg/L)		Median (μg/L)		75th Percentile		95th Percentile	
River	DEA[b]	DIA[b]	DEA	DIA	DEA	DIA	DEA	DIA
Cedar River (n = 43)	20	11	14	9.0	22	12	54	20
Iroquois River (n = 52)	23	12	19	11	33	19	44	21
Big Blue River (n = 37)	15	7.1	11	4.7	20	9.3	31	14
Sangamon River (n = 55)	23	15	16	11	27	20	53	31
Silver Creek (n = 35)	17	12	15	11	23	18	27	21
Delaware River (n = 32)	12	4.9	9.8	4.2	13	6.6	20	8.3
Huron River (n = 59)	28	12	31	8.9	41	17	55	23
Old Mans Creek (n = 55)	20	10	14	6.9	23	17	47	17
Roberts Creek (n = 23)	20	10	16	5.7	34	9.3	45	26

Source [55]. Samples were taken in April–August 1990.
DEA = deethylatrazine, DIA = deisopropylatrazine.

The greatest frequency for a 4-d average concentration of 2 μg/L was 30% in Silver Creek and the Huron River, Ohio. Both systems exceeded 21-d average concentrations of 2 μg/L 32% of the time. The least frequency of exceeding an average concentration of 2 μg/L was in the Sangamon River, in which this concentration was exceeded 5% of the time for both 4- and 21-d averages. The analyzed systems exceeded 4- or 21-d concentrations of 2 μg/L an average of 15 and 18% of the time, respectively.

Frequencies for exceeded concentrations of 5 μg/L were approximately half of those for 2 μg/L. Silver Creek and the Delaware (KS) and Huron rivers all exceeded this 4-d average concentration 12% of the time. Silver Creek exceeded this concentration in 21-d averages 20% of the time. The Sangamon River at 1% and the Sangamon and Iroquois rivers (IL) at 0% showed the least frequently exceeded values for 4- and 21-d averages, respectively. Frequencies for exceeded concentrations of 5 μg/L for 4 or 21 d averaged 7% for all rivers analyzed.

Three of the 14 rivers analyzed did not exceed 10 μg/L in 4-d averages, and five of the rivers did not exceed 10 μg/L in 21-d averages. The maximum frequency at which this concentration was exceeded was 8% in both the Delaware River and Silver Creek for 4-d averages and in Roberts Creek for 21-d averages. Half of the rivers did not exceed 4-d average concentrations of 20 μg/L, and only one, Roberts Creek, exceeded a 21-d average concentration as great as 20 μg/L.

Concentrations of atrazine transformation products. Atrazine degradation products were found in nine rivers and streams in several Midwestern U.S. states [55]. As a percentage of parent atrazine, DEA and DIA had concentrations 12 to 28% and 4.9 to 15%, respectively (Table 12). Extreme values (90th percentiles) indicated that the sum of the two metabolites rarely exceeded 80% of the parent compound concentration. The proportion of total concentration that was made up of atrazine metabolites became greater as a function of time postapplication.

In studies of other streams and rivers, concentrations of DEA, DIA, or DAC were generally less than 40% of atrazine. The fractional proportions of DEA, DIA, or DAC were 43 and 39% in Chesapeake Bay and 16, 15, and 13%, respectively, in the tributaries of the Bay (M. Cheung, personal communication). Total loading of atrazine, DEA, and DIA to the Mississippi River and several tributaries has been estimated [56]. DEA was 9.2 and 22% of atrazine loading in the Platte and Ohio rivers, respectively. DIA loading was much smaller, 0.11 and 3.0% in the Illinois and Missouri rivers, respectively.

Co-occurrence of suspended sediment and atrazine loading. Export of suspended sediment from a 10-ha conventional tillage corn field in western Tennessee has been reported [50]. Concentrations of suspended solids for three runoff events in 1985 that contributed atrazine ranged from an average of 398 to 2,565 mg/L. It should be noted that data from other runoff events during 1985, in which no atrazine was detected, were reported with average suspended solids concentrations greater than 7,000 mg/L. Hence, western Tennessee agricultural fields under conventional tillage contribute a significant amount of sediment to receiving streams.

Average suspended solids concentrations in waters with atrazine concentrations equal to or greater than 1 μg/L ranged from 95.9 (Honey Creek, OH) to 145 mg/L (Sandusky River, OH) over an 11-year period. Based on the low correlations (Fig. 13), the relationship between atrazine concentrations and other water quality parameters, including suspended sediment, was weak.

Regional differences in atrazine loadings. Soil characteristics, as well as stream order, affect atrazine concentrations in

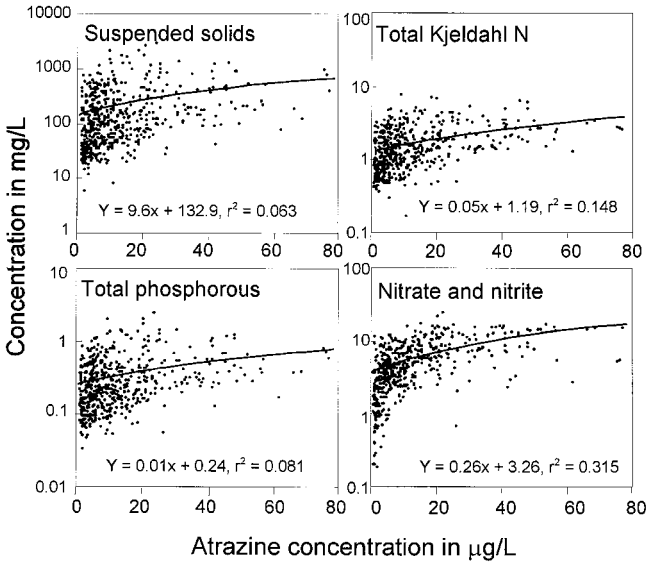

Fig. 13. Relationship between concentration of atrazine and concentration of nutrients and sediment in Rock Creek samples with atrazine concentrations exceeding 1 μg/L.

Table 13. Description of lake and reservoir data sets

Study	No. locations	Sampling period	Analysis	Sampling
Goolsby et al. [59]	13	NA	30% by GC-MS, balance by ELISA	Sampling criteria not given. Five reservoirs and eight lakes sampled in Illinois (5), Iowa (5), Kansas (1), and Missouri (2). Two of 13 localities sampled twice, for 15 samples in all
Goolsby et al. [59]	76	Bimonthly, April–November 1992	Analysis by GC-MS	Sampling at reservoir outflow. Lakes/reservoirs lokcated in North Dakota (1), South Dakota (1), Nebraska (10), Kansas (9), Minnesota (7), Iowa (5), Missouri (7), Wisconsin (11), Illinois (7), Indiana (11), and Ohio (7)
Williams [127]	1	Monthly, 1984–1991, with several omissions	Analysis by GC/MS	Total sample size 81. Hoover Reservoir, OH
Kloibel [57]	1	5 times, April–December 1990	Analysis method not known	Sampling criteria not known. Rathbun Reservoir, IA
Witt and Randtke [128]	6	Monthly, November 1990–March 1992	Analysis by GC/MS	Typically monthly sampling, total sample size 78 Lake/reservoir sites in Kansas (4), Pennsylvania (1), Iowa (1)

aquatic systems. Atrazine in runoff from an 18-ha corn field in western Tennessee was not associated with particulate matter. Concentrations never exceeded 250 μg/L at the field edge [49]. Atrazine loss from the field occurred only during the first month after application. Atrazine concentrations were less than the detection limit after the first three storm events. Exposure under these conditions was limited to 1 month of the year. The reason for this is that, unlike the fine- to medium-textured glacial till-deposited soils in northwestern Ohio, where atrazine moves into surface waters between storm events due to interflow and tile drainage, atrazine migration in western Tennessee occurs primarily by overland flow. This is probably the case for other regions with similar soil types.

Lakes and reservoirs. A less extensive body of data exists for atrazine concentrations in lakes and reservoirs than in streams and rivers. Data are generally collected less frequently in lentic waters than in streams and rivers because concentration fluctuations are of longer duration. Because most concentration changes in reservoirs are due to inflow during rain events, sampling usually concentrates on spring and summer periods following herbicide application. This part of the assessment focused on lake or reservoir studies that included multiple sampling at single or multiple sites (Table 13).

Atrazine is often found in Midwestern reservoirs (Table 14). Atrazine was at concentrations greater than 0.05 μg/L in 74 to 92% of samples collected from 76 reservoirs between April and November in 1992 [56]. The range of atrazine concentrations in these studies was smaller than those in the rivers and streams discussed above, and peak concentrations were less. Median atrazine concentrations in reservoirs ranged from 0.37 [56] to 4.72 μg/L [57]. Atrazine appeared to persist in some reservoirs at greater concentrations throughout the year, governed by the hydraulic residence times of the specific reservoirs. The mean atrazine concentration in 13 reservoirs sampled from December to February was 1.85 μg/L, and the median concentration was 0.60 μg/L. Much of the reservoir water inflow occurs in the spring, when atrazine applications on the surrounding watershed may result in greater amounts of atrazine accumulating in the reservoirs. Later in the season, when stream and river concentrations are less, water inputs into the reservoirs are also smaller, resulting in little dilution of atrazine concentrations.

The persistence of atrazine in water may result in reduced fluctuations in some reservoir concentrations over the year. When assessing overall atrazine mass balance in a monitored reservoir, Ulrich et al. [58] estimated that 5% of atrazine losses were accounted for by hydrolysis, photolysis, volatilization, or microbial processes. The majority (95%) of losses of atrazine from the reservoir were accounted for by reservoir outflow. Atrazine input was confined to a 2-month postapplication period. During several months postapplication, the upper levels of the lake increased in atrazine concentration, but concentrations remained approximately 0.2 μg/L at depths between 10 and 30 m. Surface concentrations increased to 0.4 μg/L at the end of June, decreased to 0.3 μg/L by mid-August, and to 0.25 μg/L by mid-October.

Concentrations of atrazine transformation products. Concentrations of DEA and DIA as a percentage of atrazine were somewhat greater in the reservoirs studied (Table 15) than in the streams and rivers. As in the rivers, concentrations of trans-

Table 14. Atrazine concentrations in reservoirs

Time period	No. of samples	Samples with detections (%)	Median (μg/L)	Two highest (μg/L)	90th Percentile (μg/L)[a]
April–May[b]	76	74	0.37	4.95, 4.04	2.56
June–July[b]	76	92	1.23	12.4, 12.1	4.63
August–September[b]	75	85	0.74	4.64, 4.20	2.71
October–November[b]	76	80	0.46	3.18, 2.29	2.50
April–December[c]	5	100	4.72	4.94, 4.31	—[d]
December–February[e]	15	100	0.60	10.0, 4.0	6.4[f]
Year-round[g]	81	100	0.73	11.9, 10.3	7.2

[a] Ninetieth percentile of lake/reservoir instantaneous concentrations, not time-weighted but approximately evenly distributed within the time period listed.
[b] Goolsby et al. [59] 76 locations, 1992.
[c] Kloibel [57] one location, 1990.
[d] With $n = 5$, a 90th percentile estimate cannot be calculated.
[e] Goolsby et al. [59] 13 locations, 1990–1992.
[f] With $n = 15$, the 90th percentile is ill-defined and the median is somewhat uncertain; these values were calculated (with interpolation) using the program DataDesk.
[g] W.M. Williams (personal communication), one location, 1985–1991.

Table 15. Atrazine transformation product concentrations in midwestern reservoirs[a]

Compound	Time period	Median (μg/L)	% of atrazine[b]	90th Percentile (μg/L)	% with detections
Goolsby et al. [59], 76 reservoirs					
DEA	April–May	0.14	38	0.49	64
	June–July	0.24	20	0.94	78
	August–September	0.22	30	0.73	73
	October–November	0.18	39	0.65	70
DIA	April–May	0.08	22	0.35	59
	June–July	0.14	11	0.65	70
	August–September	0.11	15	0.37	63
	October–November	0.11	24	0.33	62
Witt and Randtke [128], 6 reservoirs					
DEA		0.38	18	0.99	82
DIA		0.10	5	0.50	56
HA		0.80	38	1.36	85

[a] DEA = deethylatrazine, DIA = deisopropylatrazine, HA = hydroxyatrazine.
[b] Median transformation product concentration as a percent of median atrazine concentration for the same period.

formation products were greatest relative to atrazine when atrazine concentrations were least. Hydroxyatrazine was also found in reservoirs, sometimes at concentrations approaching that of parent atrazine.

Community water supplies. In 1991 and 1992, 114 community water supplies in Illinois participated in a voluntary atrazine monitoring program. The majority of these water supplies (83) withdraw their raw water from reservoirs, but about 25% withdraw raw water from rivers (D. Tierney, personal communication). Samples were taken quarterly, and the data set for individual water treatment plants ranged from one to six samples.

Based on the results of the Illinois EPA study, 10 water treatment plants with elevated atrazine concentrations were selected for a more intensive monitoring program conducted by Ciba-Geigy in 1993 to 1994 (D. Tierney, personal communication). These plants were sampled 15 times between the middle of June and the end of the year, with sampling intervals initially weekly, then increasing to every 2 weeks, and reaching every 3 weeks at the end of the study. Time-weighted mean concentrations and 90th percentile concentrations (by time) for these stations were calculated for the period of record and are given in Table 16. Although these values were calculated for a 6-month period, they should be approximately valid as estimates of annual values because the period of sampling covers about half the typical pesticide runoff season and half the nonrunoff season.

Table 16. Time-weighted mean concentrations (TWMC) and 90th percentile concentrations for 10 reservoirs in Illinois between June 1993 and June 1994[a]

Location	TWMC (μg/L)	90th Percentile (μg/L)
ADGPTV	5.74	14.0
Carlinville	3.57	11.3
Centralia	4.18	15.0
Highland	7.40	26.0
Hillsboro	4.28	11.0
Nashville	6.97	14.0
Salem	10.2	48.0
SAVE Site	2.11	6.4
Waverly[b]	1.23	3.8
Wayne City	3.94	13.0

[a] Source: D. Tierney, personal communication.
[b] Based on 6 months of data only.

This data set is different from the others used in this assessment because the water supplies chosen for the study were those that had been found to have the greatest concentrations of atrazine in the initial study. As such, they are not representative of the entire set of 114 water supplies, nor presumably of water supplies in general. Rather, they were drawn selectively from the upper end of the distribution (Fig. 14). This is an important consideration when these results are used in risk assessment. Although the data from other sampling sites reported in this study were not intentionally chosen to reflect elevated concentrations, analysis showed that the entire set of sites is almost as strongly concentrated in the upper end of the atrazine

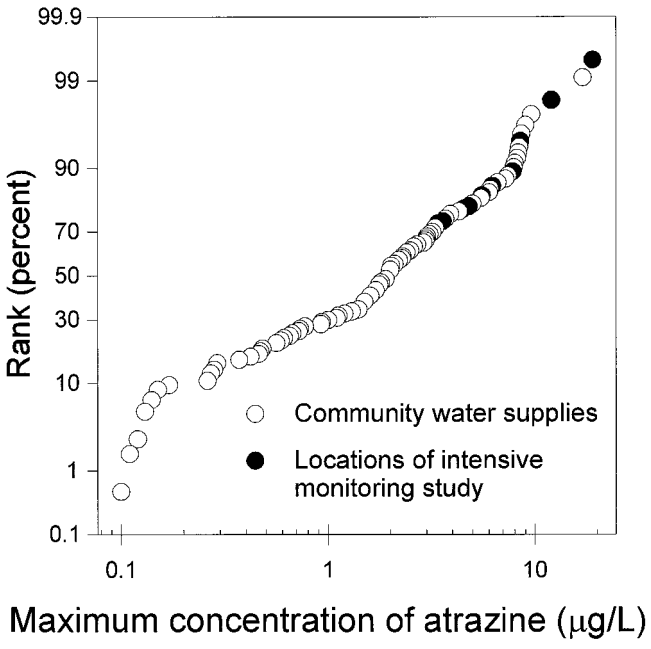

Fig. 14. Maximum concentration of atrazine (μg/L) observed at 114 community water supplies in Illinois during 1991 and 1992. The solid symbols represent the water supplies chosen for the Ciba Intensive Monitoring Study during 1993 and 1994.

concentration distribution as are the Illinois EPA sites. Overall, therefore, the exposures described throughout this section are disproportionately representative of sites with greater exposure potential.

Analysis of exposure data

In the above discussion, it was pointed out that the greatest concentrations and the greatest frequency of elevated concentrations were associated with low-order streams and with small reservoirs that are impoundments of such streams and have minimal outflow, at least seasonally. Because use of atrazine is concentrated in the corn belt of the Midwest, streams, reservoirs, and small rivers in this region present scenarios with greatest potential exposure, and these were used in part of the assessment. These data sets included the data sets of Baker and Richards (D. Baker, personal communication) on concentrations in Ohio rivers, the U.S. Geological Survey (USGS) data on rivers in the central Midwest [55], USGS data on Midwestern reservoirs [59], and a general sampling taken from 114 water treatment plant intakes in Illinois [60] plus a subset of these streams and reservoirs selected for their high potential for exposure (D. Tierney, personal communication). In the case of the survey data on the rivers in the central Midwest [55], data from enzyme-linked immunosorbent assays (ELISA) and gas chromatography with mass-spectrometer detection (GC-MS) were presented. Where GC-MS data were available, they were used in preference to ELISA data; where GC-MS data were unavailable, ELISA data were used.

To the extent possible, data sets were chosen that had a long duration, a dense sampling frequency, and adequate coverage of storm runoff events when concentrations are likely to be greatest in streams and rivers. An effort was made to select river and stream data sets covering a range of stream orders, to allow evaluation of scale effects. The main sources of data were the Ohio databases of Baker and Richards and a number of additional data sets from Canada [61–64] and large rivers in the U.S. (K. Balu, personal communication). These combinations of choices yielded a total data set that emphasized the upper portion of the concentration distribution of possible data sets and likely added conservatism to the risk assessment.

These data sets were analyzed in three ways. For some data sets with relatively few values, and for data sets for which sample time information was not available, distributions of measured data were plotted on the assumption that the N data points represented a universe of $N + 1$ measurements, and 90th percentile concentrations were obtained by regression [42], as recommended in the ARAMDG report [4]. For data sets that were clearly not log-normally distributed or were products of seasonal sampling, 90th percentiles were estimated nonparametrically from the time-weighted data. For larger and more complete data sets (Ohio databases of Baker and Richards [D. Baker, personal communication] and Scribner et al. [55]), 4- and 21-d running averages were calculated for the period of record, and 90th percentile concentrations were extracted from the distribution of these running averages, without the use of regression. Because each 4- or 21-d average represents an equal interval of time, no time-weighting is needed with these data. The use of time-weighted concentrations is most realistic for comparison with toxicity data derived from exposures over the same or similar time periods. Distributions of instantaneous measurements, or those taken only in the use season, are likely to overestimate exposures unless samples are taken year-round at fixed intervals.

The data sets contained measurements collected over a variety of time scales. Some spanned only the growing season, others 1 or 2 years, and a few spanned up to 12 years of observations. Data sets representing longer time periods would be most likely to capture rare events such as very heavy rainfalls leading to greater contamination. These data sets are thus more realistic for assessing likely maximum concentrations of atrazine in aquatic systems.

Influence of cultural practices on trends in exposure

The generic and runoff-specific mitigation techniques discussed in the final report of the ARAMDG [4] have been initiated relatively recently for atrazine. Reduction in atrazine exposures, through reductions in use rates and total tonnage used, is likely to continue.

Conservation tillage. A review of the effects of conservation tillage on pesticide runoff [65] includes several studies on atrazine. In studies of the impact of conservation tillage on herbicide and water runoff in rainfall simulations, Baker et al. [66] reported 28 to 52% reductions in water loss and 17 to 59% reductions in loss of atrazine for disk conservation tillage as compared to conventional systems under moderate rainfall conditions (127 mm in 2 h). Losses of both water and atrazine were less when residual crop cover was greatest. Under greater rainfall conditions (272 mm in 2 h and 146 mm in 2 h), Saurer and Daniel [67] reported a 20% reduction in atrazine loss under chisel conservation tillage and an 11% increase in atrazine loss under both ridge plant and no-till systems compared to conventional tillage.

Studies using natural rainfall have, in most cases, found reductions in water and pesticide losses in runoff from fields under conservation tillage. Of the 13 trials reviewed [65], movement of atrazine from fields under various conservation tillage systems averaged 44% of the atrazine movement from fields under conventional tillage. Atrazine loss in runoff was greater (198%) under conservation tillage in only one of these trials; in three tests no atrazine loss in runoff was detected.

Setbacks and application buffer requirements. Label specifications in force for the 1993 planting season included new requirements for setback and application buffers. Applications are not to take place within 61 m of lakes or reservoirs. In addition, a 20-m application buffer is required from points where field surface water enters intermittent or perennial streams or rivers. These measures may result in edge-of-field reductions in atrazine loss in runoff and thus lessen aquatic loading. Runoff studies are required to measure the extent of these reductions.

Detection of trends in exposure. The gradual increase in the use of conservation tillage, with subsequent reduction in water and pesticide runoff, may have already reduced atrazine concentrations in aquatic systems. It is unlikely that this reduction will be discernable in the monitoring data in the near future. Climatic variability and resultant variability in runoff may, in the short term, obscure all but the most dramatic changes in concentrations resulting from alterations in use rates or management practices. The concurrent impact of atrazine use rate reduction as well as the effects of setback and application buffer requirements are also unlikely to be detectable in current monitoring data.

EFFECTS CHARACTERIZATION

Ecological effects

Mode of action. The mechanism of action of atrazine is im-

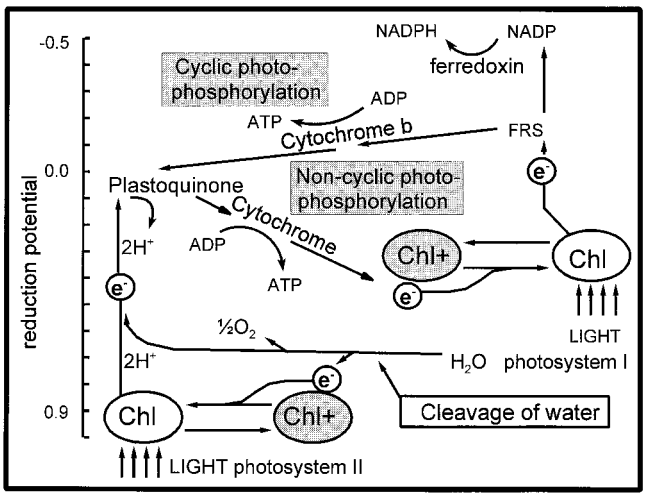

Fig. 15. Photophosphorylation.

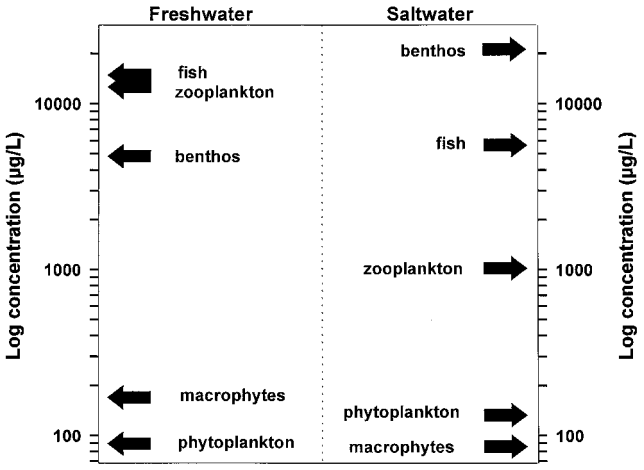

Fig. 16. Geometric means for acute atrazine toxicity data in freshwater and saltwater organisms.

portant in the risk assessment. Atrazine enters plants primarily through roots and foliage of rooted plants and across cell surfaces of unicellular plants [68]. In photosynthesis, light energy (photons) oxidizes chlorophyll molecules, which obtain replacement electrons from the cleavage of water, thus producing oxygen as a byproduct [69]. Atrazine acts by inhibiting photosynthesis via blockage of electron transport in photosystem II [69,70]. The blockage leads to chlorophyll destruction, inhibition of carbohydrate synthesis, a reduction in the carbon pool, and a buildup of CO_2 within the cell [71]. Because this photosynthetic metabolic pathway is found in plants and not animals, atrazine is much more toxic to plants than to animals. Like most of the herbicides known to be photosynthetic inhibitors (e.g., triazines, ureas, and uracils), atrazine competes with plastoquinone II at its binding site and blocks the transport of electrons from photosystem II, which utilizes water as an electron donor (Fig. 15). Without replacement electrons from water, electron flow mediated by light can occur only until all of the chlorophyll molecules in photosystem II are oxidized. After this, photophosphorylation (ATP production), reduction of photosystem I chlorophyll molecules, cyclic photophosphorylation, production of NADPH, and eventually the reduction (fixation) of carbon dioxide in the dark reactions all cease. Extended exposure to light in the presence of blockers of photosynthesis such as atrazine results in damage to the chlorophyll molecules. In the absence of light, atrazine does not affect plants.

The binding of atrazine to the plastoquinone II binding site is reversible. When atrazine-exposed plants are removed to uncontaminated medium, levels of photosynthetic activity increase. This demonstrates that processes such as metabolic detoxification, vacuolization within the plant, or diffusion from the plant back into the matrix must result in permanent removal of atrazine from sites for photosynthesis in chloroplasts. This results in recovery [72].

Effects of atrazine in laboratory toxicity tests. A comprehensive review of freshwater and saltwater laboratory tests of the toxicity of atrazine was conducted. This review is summarized in Figure 16, which illustrates the difference in geometric mean response among groups of saltwater and freshwater organisms. Information on the acute or chronic aquatic toxicity of atrazine was available for 85 aquatic species tested in freshwater (Tables 17 to 21). The order of sensitivity from most to least sensitive for the trophic groups was as follows: phytoplankton > aquatic macrophytes > benthos > zooplankton > fish (Fig. 17). The geometric means by trophic group (Fig. 16) were generated from acute data except for aquatic macrophytes, for which only chronic data were available. Due to limited data for amphibians, a geometric mean was not developed for this trophic group. However, the limited toxicity data suggest that amphibians are tolerant of atrazine.

Acute and chronic saltwater aquatic toxicity data for atrazine were available for 47 species. The order of sensitivity from most to least sensitive based on geometric means of acute toxicity data for the various trophic groups was aquatic macrophytes > phytoplankton > zooplankton > fish > benthos (Fig. 18 and Tables 22 to 26).

Toxicity of transformation products to aquatic plants. Atrazine was more inhibitory to photosynthesis than were its transformation products [73]. Although DEA and DIA were the most toxic transformation products of the four tested, atrazine was 7 to 10 times more inhibitory to blue-green algae and 4 to 6 times more inhibitory to green algae than the most potent transformation product, DEA. Atrazine was 20 to 50 times more inhibitory to blue-green algae and 7 to 13 times more inhibitory to green algae than DIA. DAC and HA were relatively nontoxic to aquatic plants; atrazine was 200 to >1,000 times more inhibitory than either of these products. This conclusion was supported by a study of the toxicity of DEA to submerged macrophytes, in which atrazine produced photosynthetic inhibition at less than one-fifth the concentration required for DEA [74]. An extensive in vitro study of the inhibition of the Hill reaction by more than 100 triazine analogues supports the relative potencies for the transformation products tested [75]. In his general review, Eisler [27] concluded that ''there is general agreement that atrazine degradation products are substantially less toxic than the parent compound and not normally present in the environment at concentrations inhibitory to algae, bacteria, plants, or animals.''

Of the species tested by Stratton [73], the green alga *Chlorella pyrenoidosa* was more sensitive to the atrazine metabolites DEA and DIA than to atrazine itself. EC50s for atrazine, DEA, and DIA were 500, 1,800, and 3,600 µg/L, respectively. Based on these values, potency ratios of 3.6 (DEA) and 7.2 (DIA) were calculated (metabolite EC50/atrazine EC50). Using this worst case relationship in potency, it is possible to predict the increased risk to aquatic photosynthetic plants for different ex-

Table 17. Toxicity of atrazine (μg/L) to freshwater phytoplankton

Species	EC50	Duration	Comment	Reference
Chlorella vulgaris	25	11 d	—	[129]*
C. vulgaris	—	7 d	NOEC = 500	[130]
C. vulgaris	—	5, 14 d	500 μg/L caused slight growth reduction	[131]
C. vulgaris	—	7 d	1 μg/L decreased chlorophyll production	[132]
C. vulgaris	42–53	24 h	—	[133]
Chlorella pyrenoidosa	125	24 h	—	[134]
C. pyrenoidosa	—	10 d	55 μg/L decreased growth and O_2 production	[135]
C. pyrenoidosa	—	5, 24 d	500 μg/L inhibited growth	[131]
C. pyrenoidosa	175	5 d	Growth	[136]*
C. pyrenoidosa	139	1 h	Photosynthesis inhibition	[137]
C. pyrenoidosa	500	3 h	Photosynthesis	[73]
	1,000	14 d	Growth	
C. pyrenidosa	—	14 h	100 μg/L slightly inhibited cell growth	[138]
C. pyrenidosa	282	5 d	—	[77]*
Chlorella sp.	35–42	5 min	—	[139]
Chlorella sp.	—	12 d	1,000 μg/L inhibited photosynthesis	[140]
Ankistrodesmus braunii	60	11 d	—	[129]*
A. braunii	61–219	24 h	—	[133]
Franceia sp.	430–774	5 min	—	[139]
Chlorococcales sp.	48–162	5 min	—	[139]
Scenedesmus obliquus	38–57	24 h	—	[133]
Scenedesmus subspicatus	110	96 h	—	[141]*
Scenedesmus quadricauda	—	?	1,000 μg/L prevented growth	[142]
S. quadricauda	—	72–96 h	500 and 800 μg/L reduced growth	[143]
S. quadricauda	300	3 h	—	[73]
S. subspicatus	21	96 h	—	[144]*
Scenedesmus sp.	—	12 d	1,000 μg/L inhibited photosynthesis	[140]
Chlamydomonas eugametos	—	14 d	NOEC = 500	[131]
C. reinhardi	—	14 d	500 μg/L completely inhibited growth	[131]
C. reinhardi	45–484	5 min	—	[139]
	—	48 h	216 μg/L inhibited growth	
C. reinhardi	—	14 d	1,000 μg/L inhibited growth	[145]
C. reinhardi	—	72–96 h	50 μg/L reduced growth	[143]
C. reinhardi	19–48	24 h	—	[133]
Chlamydomonas sp.	—	12 d	1,000 μg/L inhibited growth	[140]
Selenastrum capricornutum	214	7 d	—	[146]*
S. capricornutum	42–53	24 h	—	[133]
S. capricornutum	69.7	24 h	Algal medium	[147]
	854	24 h	Montana water	
S. capricornutum	—	5 d	MAC = 200 μg/L	[148]
S. capricornutum	—	30 d	10 μg/L reduced GPP; NOEC <100	[100]
S. capricornutum	120	120 h	—	[77]*
	53	—	—	
S. capricornutum	130	96 h	—	[149]*
S. capricornutum	95	5 d	—	[150]*
S. capricornutum	50	96 h	—	[151]*
S. capricornutum	55	120 h	NOEC = 16	[152]*
S. capricornutum	—	96 h	Dry weight; NOEC = 0.5; LOEC = 1	[153]*
	4	96 h	Cell count; NOEC = 0.5; LOEC = 1	
S. capricornutum	9.5	7 d	Cell count IC50	[154]
Stigeoclonium tenue	—	7 d	1 μg/L decreased chlorophyll production	[132]
Anabaena inaequalis	300	3 h	Photosynthesis	[73]
	100	14 d	Growth	
A. cylindrica	500	3 h	—	[73]
	178–253	24 h	—	[133]
A. variabilis	100	3 h	—	[133]
A. flos-aquae	230	5 d	Phytostatic concn. = 4,970 μg/L; phytocidal concn. = >3,200 μg/L; NOEC <100	[155]*
A. flos-aquae	58	3 d	—	[146]*
	469	5 d	—	
	776	7 d	—	
Microcystis aeruginosa	—	5 d	MAC = 400 μg/L	[148]
Oscillatoria lutea	—	7 d	1 μg/L decreased chlorophyll	[132]
Plectonema boryanam	—	3 d	NOEC = 10,000	[156]
Narnochloris oculata	—	8 d	15 μg/L reduced growth	[157]
Bumilleriopsis filiformis	220	?	Oxygen evolution	[158]
Cyclotella meneghiniana	74–243	5 min	—	[159]
Phaeodactyum tricornutum	—	8 d	NOEC = 15	[157]
Navicula pelliculosa	60	5 d	Phytostatic concn. = 1,710 μg/L; phytocidal concn. >3,200 μg/L	[155]
Porphyridium cruentum	308	5 d		[77]

* References used in the risk characterization.

Table 18. Toxicity of atrazine (μg/L) to freshwater aquatic macrophytes

Species	EC50	Duration	Comment	Reference
Potamogeton pectinatus	—	30 d	100 μg/L reduced growth	[160]*
P. perfoliatus	474	21 d	Leaf growth	[70,161]
	53	21 d	Mortality	
Elodea canadensis	80	28 d	Leaf growth	[70,161]
	163	42 d	Leaf growth	[70,161]
Myriophyllum spicatum	1,104	28 d	Leaf growth	[70,161]
		5 d	3,700 μg/L reduced number of branches	[162]*
Lemna sp., *Ceratophyllum* *Elodea*	—	30 d	1,000 μg/L reduced biomass	[100]
Lemna gibba	50	14 d	Frond density, NOEC = 8.3	[163]*
	22	14 d	Frond biomass; NOEC = 8.3	
L. gibba	180	7 d	Frond production	[164]*
L. gibba	37	14 d	Frond density; NOEC <3.4	[165]*
	45	14 d	Frond biomass; NOEC = 7.7	
L. gibba	170	5 d	—	[77]*
L. minor	8,700	14 d	NOEC = 10; LOEC = 100	[153]*
Thallassia testudinum	320	40 h	—	[153]*
Elodea canadensis	1,200	10 d	NOEC = 10; LOEC = 100	[166]
Hydrilla verticillata	430	14 d	Shoot length	
	110,220	14 d	New shoot growth	
	80,250	4 d	Dehydrogenase activity	

* References used in the risk characterization.

Table 19. Toxicity of atrazine (μg/L) to freshwater benthos (μg/L)

Species	LC50	Duration	Comment	Reference
Ancyclus fluviatilis	—	30 d	1,000 μg/L decreased hatch	[167]*
Glossiphonia complanata	6,300	28 d	—	[167]
Helobdella stagnalis	9,900	27 d	—	[167]
Anodonta imbecilis	>60,000	24 h	—	[168]
Chironomus riparius	18,900	240 h	—	[169]*
C. riparius	—	48 h	EC50 = 1,000	[100]*
C. tentans	720	48 h	MATC = 100–230 μg/L	[120]*
Gammarus fasciatus	5,700	48 h	MATC = 60–140	[120]*
Paratya australiensis	—	10 d	NOEC = 340	[170]

* References used in the risk characterization.

Table 20. Toxicity of atrazine (μg/L) to freshwater zooplankton

Species	LC50	Duration	Comment	Reference
Daphnia pulex	—	28 d, 70 d	1,000 μg/L caused significant effects on population	[171]*
D. pulex	>40,000	3 h	—	[172]
D. pulex	—	48 h	EC50 = 36,000–46,500	[173]*
D. pulex	33,000	48 h	—	[174]*
Daphnia magna	3,600	26 h	—	[175]
D. magna	9,400	48 h	—	[168]*
D. magna	—	48 h	EC50 = 3,600	[120]*
D. magna	—	24 h, 48 h	EC50 >39,000	[176]*
D. magna	6,900	48 h	MATC = 140–250 μg/L	[120]*
D. macrocopa	>40,000	3 h	—	[172]
Moina macrocopa	—	30–45 d	1,000 μg/L did not affect development	[177]*
M. macrocopa	—	30–45 d	1,000 μg/L increased mortality	[178]*
Ceriodaphnia quadrangula	—	30–45 d	1,000 μg/L reduced productivity	[178]*
C. quadrangula	—	30–45 d	1,000 μg/L reduced development	[177]
C. dubia	>30,000	48 h	—	[179]*
	—	4 d	Chronic value = 6,900 μg/L; NOEC = 5,000–10,000; LOEC = 10,000–20,000	
	—	7 d	Chronic value = 3,500 μg/L; NOEC = 2,500; LOEC = 5,000	
C. dubia	—	7 d	NOEC = 5,000	[180]*
	2,000	7 d	—	
Scapholeberis mucronata	—	?	1,000 μg/L reduced fecundity after the first generation	[178]
S. mucronata	—	30–45 d	Embryonic development reduced at 1,000 μg/L	[177]*
Acanthamoeba castellanii	—	6 d	10,000 μg/L reduced growth	[181]
Chlorohydra viridissima	—	21 d	5,000 μg/L reduced budding rate	[182]*
Aedes aegypti	—	24 h	NOEC = 10,000	[183]
Colpidium campylum	>50,000	24 h	—	[150]

* References used in the risk characterization.

Table 21. Toxicity of atrazine (μg/L) to freshwater fish

Species	LC50	Duration	Comment	Reference
Salmo gairdneri	8,800	96 h	—	[184]*
S. gairdneri	4,500	96 h	—	[185]*
S. gairdneri	—	10 d	NOEC >340	[186]
S. gairdneri	3,500–5,700	96 h	—	[187]*
S. gairdneri	—	28 d	5–40 μg/L caused alteration of renal corpuscles and renal tubules	[188]*
S. gairdneri	870	28 d	—	[189]
S. gairdneri	870–1,110	28 d	—	[190]
S. gairdneri	26,400	72 h	—	[191]*
Salvelinus fontinalis	6,300	96 h	MATC = 60–120 μg/L	[120]*
Oncorhynchus kisutch	15,000	96 h	—	[192]*
Carassius carassius	76,000	96 h	—	[184]*
C. carassius	>100,000	96 h	—	[185, 187]*
Cyprinus carpio	50,000	48 h	—	[191]*
C. carpio	>10,000	48 h	—	[172]*
C. auratus	>10,000	48 h	—	[172]*
Leuciscus idus	44,000	96 h	—	[193]*
Brachydanio rerio	—	35 d	1,300 μg/L increased number of edemas	[194]
B. rerio	1,200	35 d	—	[195]*
Ictalurus punctatus	7,600	96 h	—	[184]*
I. punctatus	220	10 d	—	[189]
I. punctatus	220–340	8.5 d	—	[190]
Lepomis macrochirus	16,000	96 h	—	[184]*
L. macrochirus	—	8 d	NOEC >10,000	[196]*
L. macrochirus	8,000	96 h	MATC = 90–500 μg/L	[120]*
L. cyanellus	—	8 d	NOEC >10,000	[196]
Micropterus dolomieui	—	3 d	Survived 10,000 μg/L	[196]
Lebistes reticulata	4,300	96 h	—	[184]*
Poecilia reticulata	71,000	72 h	—	[191]*
Gambusia affinis	—	48 h	NOEC > 10,000	[197]
Tilapia sparrmanii	—	21–28 d	3,400–4,800 μg/L increased bioconcn.	[198]
T. sparrmanii	—	72 h	Oxygen consumption; NOEC = 8,100	[199]
Erimyzon sucetta	—	8 d	NOEC >10,000	[186]
Perca sp.	16,000	96 h		[193]*
Pimephales promelas	15,000	96 h	MATC = 210–520 μg/L	[120]*
P. promelas	—	7 d	IC40 = 2,700 μg/L	[127]
P. promelas	20,000	96 h	ACR = 59 μg/L	[200]
	—	274 d	LOEC = 460	
Oryzias latipes	>10,000	48 h	—	[172]*
Galaxias maculatus	—	10 d	NOEC >340	[170]*
Pseudophritis urvillii		10 d	NOEC >340	[170]
Gnathonemus petersi		15–45 min	Electric organ discharge (EOD) modified at 100 μg/L	[201]
G. tamandau	—	45–60 min	EOD modified at 50 μg/L	[201]

* References used in the risk characterization.

Table 22. Toxicity of atrazine (μg/L) to saltwater macrophytes

Species	Duration	Comment	Reference
Zannichellia palustris	21–42 d	Oxygen production inhibited at 75 μg/L	[202]
Z. palustris	2 h	Photosynthetic efficiency and oxygen production; EC50 = 91 μg/L	[74]
Potamogeton pectinatus	21–42 d	Oxygen production inhibited	[202]
P. perfoliatus	2 h	Photosynthetic efficiency; EC50 = 77 μg/L	[74]
P. perfoliatus	4 h	EC50 = 80 μg/L	[203]
P. perfoliatus	7 d	130 μg/L decreased oxygen production; algacidal concentration = 1,200 μg/L	[21]
P. perfoliatus	2–4 wk	5 μg/L reduced photosynthesis in 2 out of 4 weeks; 50 μg/L reduced photosynthesis in all 4 weeks	[22]
P. perfoliatus	4 h	Photosynthesis reduced by 69% at 100 μg/L	[204]
Zostera marina	21 d	10 μg/L reduced growth; LC50 = 100–540 μg/L	[205]
Z. marina	21–42 d	Oxygen production inhibited at 650 μg/L	[202]
Z. marina	6 h	Net productivity reduced at 100 μg/L; NOEC = 10 μg/L	[206]
Z. marina	21 d	LC50 = 100–540 μg/L	
Vallisneria americana	9 wk	Growth reduced at 100 μg/L	[70]*
V. americana	21–42 d	Oxygen production slightly inhibited at 75 μg/L; significantly reduced at 650 μg/L	[204]*
V. americana	?	4 μg/L reduced tuber development; 8 μg/L reduced growth	[207]
Myriophyllum spicatum	28 d	5 μg/L enhanced photosynthesis; 50 μg/L reduced photosynthesis in 2 out of 4 wk	[22]*
M. spicatum	2 h	Photosynthetic efficiency; EC50 = 104 μg/L	[74]
M. spicatum	5 d	50% reduction in number of branches at 3,700 μg/L	[162]
Spartina alterniflora	45 d	Minor reductions in growth at 110 μg/L; significant reductions at 1,110 μg/L	[208]*
Ruppia maritima	2 h	Photosynthetic efficiency and oxygen production; EC50 = 102 μg/L	[74]

* References used in the risk characterization.

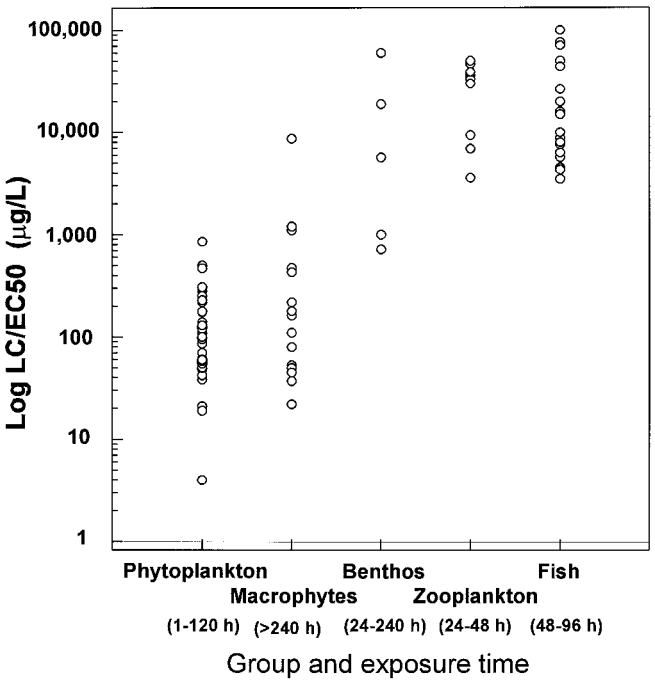

Fig. 17. Range of acute toxicity data for a number of groups of freshwater organisms.

posure scenarios of atrazine + DEA + DIA. USGS monitoring of atrazine and these metabolites in nine Midwestern rivers [55] (see discussion above) is one source of exposure data that can be used for this purpose. In the nine rivers studied, the maximum mean concentrations of DEA and DIA, as percentage of atrazine, were 28 and 15%, respectively. When combined in a relative risk calculation (Table 27), it appears that the combined toxicity of atrazine and metabolites will typically be only slightly greater (<10%) than the toxicity of atrazine alone.

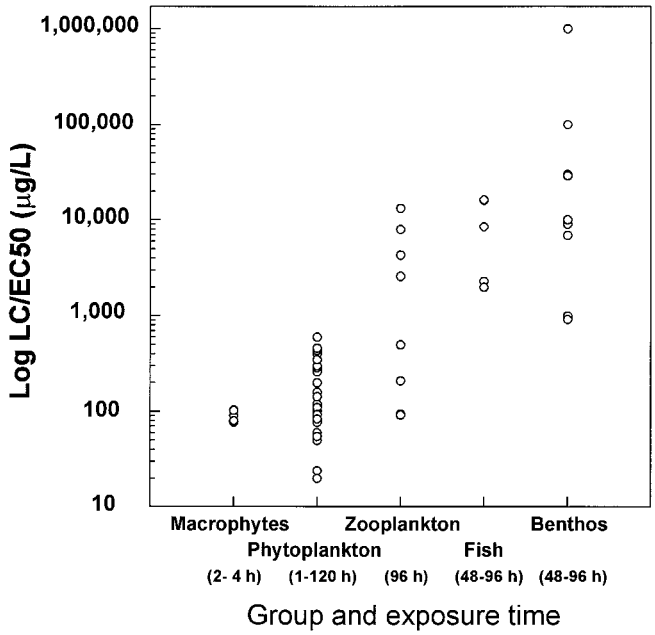

Fig. 18. Range of acute toxicity data for the groups of saltwater organisms.

Analysis of toxicity data

Risk characterization involves the integration of data on ecological effects with data on environmental exposures. In the characterization of effects, numerous laboratory, microcosm, and mesocosm studies of atrazine effects on a wide range of biota were summarized. In both the saltwater and the freshwater environments, phytoplankton, periphyton, and some other species of plants were found to be most sensitive to atrazine exposure. Data from these effects studies were placed in a probabilistic risk framework.

Data selection. There was considerable information on the toxicity of atrazine to aquatic organisms. Although many of these bioassays were not conducted under Good Laboratory Practice (GLP) guidelines, these data are still useful because they represent the range of sensitivity that may occur under a wide variety of experimental and environmental conditions. Many older tests were conducted without analysis of the exposure matrix, and the results are thus based on nominal concentrations. Atrazine is relatively persistent in water, and concentrations would not be expected to vary greatly over time, especially in short-term bioassays. On this basis, data were used from studies based on nominal as well as measured concentrations.

As discussed in the development of the conceptual model above, acute and chronic toxicity data were selected from the literature (Tables 17 to 26) for the risk characterization. For acute toxicity, all tests for which 48- to 96-h LC50/EC50 data (120 to 168 h for algal tests) were available were used. For chronic toxicity, all tests for which 10- to 36-d chronic NOECs, Minimum Algistatic Concentrations (MACs), or Maximum Allowable Toxicant Concentrations (MATCs) were available were used. For both sets of data, endpoints included growth and morbidity but excluded biomarker-type measurement endpoints such as oxygen evolution, enzyme activity, or chlorophyll content.

Acute toxicity. The data on acute toxicity were analyzed as a distribution on the assumption that the data represented the universe of species. Because it was not possible to test all the species in the universe, an approximation was made. This approximation assumed that the number of species tested (N) is one less than the number in the universe. To obtain symmetrical graphic distributions from smaller data sets, percentages were calculated from the formula ($100 \times n/[N+1]$) (adapted from [41]), where n is the rank number of the datum point and N is the total number of data points in the set. This formula compensates for the size of the data set. Small (more uncertain) data sets give a wider distribution with more chance of overlap than larger (more certain) data sets. Where multiple data points were available for a single species, the smallest value was used for regression analysis of the cumulative frequency distribution. Data were plotted using a log-Pearson type III distribution [42] using SigmaPlot [76], which also gave regression coefficients and the equations for the regression lines (Table 28).

The distribution for the acute toxicity data used in the assessment was obtained by plotting all data, but only the least toxicity values for each species were used for the regression. Included in Figure 19 are LC5 or EC5 values, obtained from extrapolation of aquatic plant bioassays, that have been critically evaluated by the EPA and are part of the database maintained for product registrations [77]. Many of these studies were conducted specifically for pesticide registration under GLP guidelines (Table 29). The EPA data are based on short-term toxicity

Table 23. Toxicity of atrazine (μg/L) to saltwater phytoplankton

Species	EC50	Duration	Comment	Reference
Chlorella pyrenoidosa	—	5 d	Minimum algistatic concn. (MAC) = 520	[148]
Dunaliella tertiolecta	—	5 d	MAC = 1,100	[148]
D. tertiolecta	170	5 d	—	[155]*
D. tertiolecta	—	5 d	NOEC <100	[155]
	170	14 d	Phytostatic concn. = 1,400; phytocidal concn. = 3,200	
D. tertiolecta	600	90 min	—	[209]*
	400	10 d	—	
D. tertiolecta	159	1 h	—	[210]
D. tertiolecta	300	10 d	—	[211]*
Chlamydomonas sp.	60	1 h	—	
Platymonas	102	1 h	—	
Chlorella sp.	143	1 h	—	
Neochloris sp.	82	1 h	—	[210]
Chlorococcum	80	1 h	—	[210]*
	400	90 min	—	
	100	10 d	—	
Porphridium cruentum	—	5 d	MAC = 780	[148]
P. cruentum	79	1 h	—	[210]
Isochrysis galbana	200	90 min	—	[209]*
	100	10 d	—	
	200	10 d	—	
I. galbana	—	5 d	13 μg/L decreased growth and fluorescence	[148]
Monochrysis luteri	77	1 h	—	[210]
I. galbana	100	1 h	—	
Phaeodactylum tricornutum	100	1 h	—	[210]
P. tricornutum	200	90 min	—	[209]*
	200	10 d	—	
Laminaria saccarina	—	48 h	Chronic value = 48.96; NOEC = 33.2; LOEC = 72.2	[212]
Skeletonema costatum	265	48 h	—	[213]*
S. costatum	260	48 h	—	[211]*
S. costatum	50	48 h	—	[214]*
S. costatum	—	5 d	Decrease in growth and fluorescence at 22 and 13 μg/L	[148]*
S. costatum	55	120 h	NOEC = 14	[152]*
S. costatum	24	96 h	—	[77]*
Minutocellus polymorphus	20	48 h	—	[214]*
Thalassiosira fluviatilis	—	7 d	Chronic value = 69.6 μg/L (reduced photosynthesis) and 645.7 μg/L (reduced cell number)	[88]
T. fluviatilis	110	1 h	—	[210]
Nitzschia sigma	—	7 d	Chronic value = 69.6 μg/L (reduced photosynthesis) and 645.7 μg/L (reduced cell number)	[88]
N. closterium	287	1 h	—	[210]
Nitzschia	434	1 h	—	
Navicula inserta	460	1 h	—	
Amphora exigua	300	1 h	—	
Achnanthes brevipes	93	1 h	—	
Staureneis amphoroides	348	1 h	—	
Cyclotella nana	84	1 h	—	

* References used in the risk characterization.

Table 24. Toxicity of atrazine (μg/L) to saltwater zooplankton

Species	LC50	Duration	Comment	Reference
Acartia tonsa	94	96 h	—	[215]*
A. tonsa	210	96 h	—	[216]*
A. tonsa	92	96 h	—	[216]*
A. tonsa	4,300	96 h	NOEC <2,900	P.C. McNamara, personal communication*
Acartia clausi	7,945	96 h	–	[216]*
Eurytemora affinis	500	96 h	5 ppt test condition	L.W. Hall, Jr., personal communication*
	2,600	96 h	15 ppt test condition	L.W. Hall, Jr., personal communication*
	13,200	96 h	25 ppt test condition	L.W. Hall, Jr., personal communication*
E. affinis	14,600	8 d	5 ppt test conditon	[217]*
	20,900	8 d	15 ppt test condition	[217]*
	5,010	8 d	25 ppt test condition	[217]*

* References used in the risk characterization.

Table 25. Toxicity of atrazine (μg/L) to saltwater fish

Species	LC50	Duration	Comment	Reference
Leiostomus xanthurus	—	48 h	NOEC = 1,000	[218]
L. xanthurus	8,500	96 h	—	[215]*
Cyprinus carpio	—	6–24 h	10 μg/L caused change in serum and glucose activity	[219]
Cyprinodon variegatus	>16,000	96 h	Juvenile	[215]*
C. variegatus (larvae)	16,200	96 h	5 ppt	L.W. Hall, Jr., personal communication*
	2,300	96 h	15 ppt	
	2,000	96 h	25 ppt	
C. variegatus (embryo-juvenile)	—	28 d	Chronic value = 2,530	[215]

* References used in the risk characterization.

tests (5 to 7 d) with relatively sensitive species (i.e., several species of algae and duckweed). The slope of the regression through the LC5s is steeper than the line from the other data (Table 28) and reflects the selection of organisms with greater sensitivity. However, it is interesting to note that these data are protective of the larger data sets for both acute and chronic exposure. In the larger data set, considerable variation in results was observed between bioassays conducted by different investigators (data points shown on the same horizontal line in the graph). This probably reflects the difficulty of measuring endpoints in some species and variation among laboratories.

The sensitivity distributions of freshwater and saltwater species used in the assessment show little difference between freshwater and saltwater species (Fig. 20), although the regression line for saltwater species had slightly less slope. For the purposes of the risk assessments that follow, the data were pooled.

Plotting the acute LC50 values used in the assessment by class of organism (Fig. 21) clearly shows the relatively greater sensitivity of phytoplankton and lesser sensitivity of fish. Thus, it is unlikely that fish would be affected directly by atrazine. Data for benthic invertebrates and zooplankton were less numerous and exhibited a greater range of sensitivity as well as less slope. Once again, the EPA LC5 data were more conservative than the other data, but, as expected, the regression line was parallel to that for phytoplankton from the larger data set. These groupings, based on physiological sensitivity to atrazine, show that the organisms most likely to be impacted directly are the photosynthetic phytoplankton and, by extension, periphyton and possibly macrophytes. This specificity of action must be considered in the assessment of risk.

From these regressions, the atrazine concentration associated with any given percentile of sensitivity can be estimated. For the risk characterization that follows, the concentration corresponding to the 10th percentile of sensitivity was used as one factor for determining the margin of safety (MOS_{10}) and the probability that exposure concentrations would exceed this concentration. The 10th percentile for acute toxicity, based on all LC50 and EC50 values, was 37 μg/L. The 10th percentile for the EPA LC5 data was 5.4 μg/L.

Chronic toxicity. Sensitivity data for chronic exposures to atrazine were available (Tables 17 to 26), although not to the same extent as for acute sensitivity. As before, where multiple measurements were available, only the least value was included in the data set to present a conservative scenario. As for the acute data, the chronic data were analyzed as a distribution on the assumption that the data represented the universe of species, and percentiles were calculated as above. The results are shown in Figure 22. The 10th percentile for chronic sensitivity to atrazine was 3.7 μg/L. Interestingly, the 10th percentile for chronic endpoints was one-tenth of the 10th percentile for acute endpoints—analogous to the factor of 10 currently used by the EPA to estimate a threshold level of concern from the LC50.

Effects of atrazine in microcosms and mesocosms

As pointed out in the problem formulation section, a considerable gap exists between the endpoints measured in laboratory toxicity tests and the assessment endpoints that are the focus of this risk assessment. Microcosm and mesocosm studies with atrazine provide measurement endpoints that are closer to the assessment endpoints, for the following reasons:

Table 26. Toxicity of atrazine (μg/L) to saltwater benthos

Species	LC50	Duration	Comment	Reference
Crassostrea virginica	—	96 h	Juvenile; NOEC = 1,000	[218]
C. virginica	—	48 h	Embryo; EC50 = 30,000	[215]*
Cardium edule	>100,000	48 h	—	[220]*
Palaemonetes pugio	9,000	96 h	—	[215]*
Crangon crangon	10,000–30,000	48 h	—	[220]*
Penaeus aztecus	—		30% mortality after 48 h to 1,000 μg/L	[218]
	1,000	48 h	—	[211]*
Penaeus duorarum	6,900	96 h	—	[215]*
Mysidopsis bahia	920	96 h	Juvenile tested	
	—	28 d	Chronic value = 130	
Uca pugilator	>29,000	96 h	—	
U. pugilator	—	8 d	Chronic value = 9,970; NOEC = 1,000	[88]
Neopanope texana	>1,000,000	96 h	NOEC = 750,000	[221]*

* References used in the risk characterization.

Table 27. Estimation of the relative risk associated with phytotoxic metabolites of atrazine

Chemical	Relative toxicity (based on EC50 ratio)		Expected exposure ratio		Risk contribution
Atrazine	100*	×	1.0	=	100
DEA	28	×	0.28	=	7.8
DIA	14	×	0.15	=	2.1
			Total	=	109.9

* Arbitrary units.

Measurements of primary productivity in microcosms and mesocosms incorporate the aggregate responses of multiple species—often several dozen—in aquatic plant communities. Because plant species vary widely in their sensitivity to atrazine, the overall response of the plant community may be quite different from the responses of individual species as measured in laboratory toxicity tests.

Mesocosm and microcosm studies allow observation of population and community recovery from atrazine effects. Recovery can take place through several mechanisms, as described below.

Studies with microcosms and mesocosms allow measurement of indirect effects of herbicides on higher trophic levels. Indirect effects may result from changes in food supply, habitat, or water quality. Such effects may be inferred by extrapolation from laboratory toxicity data, but they can be measured directly only in multitrophic systems.

Microcosm and mesocosm studies can be designed to approximate realistic atrazine exposure regimes more closely than standard laboratory single-species toxicity tests. Most studies, especially those conducted in outdoor systems, incorporate partitioning, degradation, and dissipation—important factors in determining exposure. These factors are rarely accounted for in laboratory toxicity studies but may greatly influence the magnitude of ecological response.

Atrazine has been the subject of more than 20 mesocosm and microcosm studies addressing ponds, streams, lakes, wetlands, and salt marshes. Experiments have ranged from a few weeks to several years in duration, and exposure concentrations have ranged from 0.1 to 10,000 μg/L. Because atrazine concentrations greater than 50 μg/L are rare in surface waters, the following discussion focuses on the observations that have been made at concentrations less than 50 μg/L (summarized in Table 30).

Table 28. Regression data for atrazine toxicity distributions (lowest value used where multiple tests available)

	$y = ax + b$		Regression r^2
	a	b	
All acute EC50s/LC50s	0.78	2.50	0.97
EPA LC5s/EC5s	1.33	2.74	0.75
Freshwater acute LC50s/EC50s	0.75	2.48	0.89
Saltwater acute LC50s/EC50s	0.69	2.78	0.93
Benthos acute LC50s	0.81	1.83	0.90
Fish acute LC50s	1.92	−2.83	0.96
Phytoplankton acute LC50s/EC50s	1.23	2.60	0.85
Zooplankton acute LC50s	0.74	2.41	0.94
Chronic MACs/MATCs/NOECs*	0.77	3.28	0.93

* MAC = maximum algistatic concentration; MATC = maximum acceptable toxicant concentration; NOEC = no-observed-effect concentration.

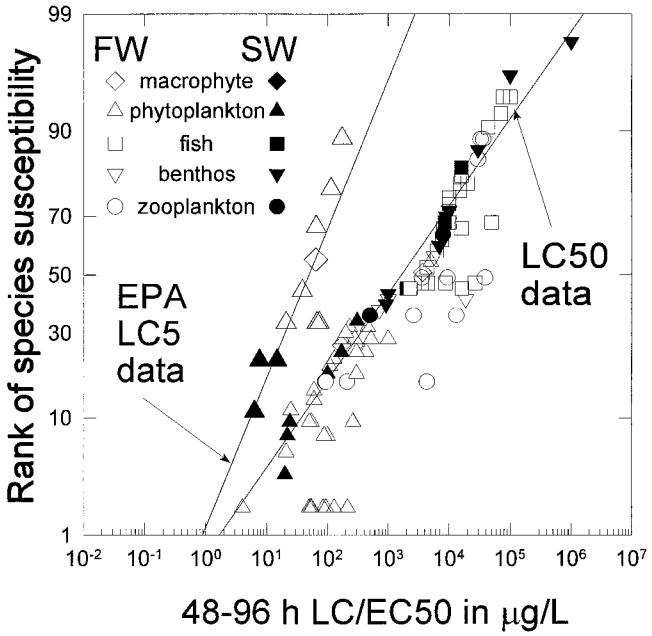

Fig. 19. Distribution of acute LC/EC5s (from EPA data) and LC/EC50s for atrazine in aquatic organisms. Points on the same horizontal line are for the same species, but from different sources/references. Measurement endpoints were selected from a larger data set and represent mortality, growth, and survival. Physiological and biochemical endpoints were excluded.

Phytoplankton. Primary production was measured in pond microcosms receiving continuous atrazine inputs at concentrations from 0.5 to 5,000 μg/L [78]. The least concentration causing a reduction in primary production was 50 μg/L; there was no effect at 5 μg/L. A concentration of 20 μg/L atrazine caused no adverse effects on productivity in mixed-flask culture microcosms derived from spring plankton communities in four Oregon lakes [79]. Similarly, no effect of 20 μg/L atrazine was found on phytoplankton in a compartmentalized pond [80].

A series of pond mesocosm studies with atrazine was conducted at the University of Kansas between 1979 and 1983 [81–86]. Phytoplankton biomass and production rates were unaffected by exposure to 20 μg/L atrazine. Phytoplankton biomass and photosynthesis were reduced on day 2 in the 20-μg/L ponds compared with control ponds [83]. However, data in Kettle [84] reveal that a phytoplankton bloom developed in one of the two control ponds, even though the biomass and photosynthetic rates of the control mesocosms were similar to those in ponds treated with 20 μg/L. Because of the variability among ponds, the inference of an atrazine effect at 20 μg/L cannot be supported. Chlorophyll concentrations were greater in ponds exposed to 20 μg/L than in control ponds. The increase in chlorophyll offset a decline in photosynthetic efficiency expressed as primary productivity per unit chlorophyll. An increase in chlorophyll per unit biomass has also been reported for vascular macrophytes exposed to atrazine [21]. Several authors (e.g., [21,87,88]) have noted the similarities between the effects of atrazine on primary producers and the effects of other factors, such as shading, that limit photosynthesis in other ways. Some phytoplankton species, notably the cryptomonads, became more abundant in the 20-

Table 29. Toxicity data from the U.S. EPA database [2] and other sources

Species	LC5 (μg/L)	LC50 (μg/L)	Slope	Reference
Isochrysis galbana	6	22	3.1	
Skeletonema costatum	8	24	3.3	
Selenastrum capricornutum	21	53	4.1	
Skeletonema costatum	15	55	2.9	[152]
Selenastrum capricornutum	68	120	6.6	
Microcystis aeruginosa	39	129	3.2	
Selenastrum capricornutum (TSCA protocol)	74	130	6.7	[149]
Lemna gibba	65	170	3.9	
Chlorella pyrenoidosa	115	282	4.2	
Porphyridium cruentum	66	308	2.4	
Dunaliella tertiolocta	176	431	4.2	

μg/L ponds than in the controls, although no species were measurably reduced.

The effects of 15 μg/L atrazine on lake phytoplankton in outdoor microcosms has been measured [89]. Total algal abundance was unaffected; green unicellular algae were less abundant, and diatoms were relatively more abundant. After 7 d of exposure, primary productivity was slightly less and chlorophyll concentrations were slightly greater in the microcosms treated with 15 μg/L atrazine than in the controls. This indicates a reduction in photosynthetic efficiency similar to that reported in the results of the Kansas studies [81,85]. There was no difference in chlorophyll or primary productivity between control and 15-μg/L microcosms after 14 d of exposure.

In two studies, small concentrations of atrazine were reported to affect natural phytoplankton communities. The abundance of phytoplankton in a field stream receiving agricultural tile drainage containing up to 1.9 μg/L atrazine has been determined [90]. Phytoplankton densities were less in the channel carrying tile flow than in the receiving stream and were less in the receiving stream downstream from the confluence with the tile drainage than at another station upstream of the confluence. However, many other water quality variables were different between the tile drainage and the receiving stream. Moreover, the cross-sectional area of the downstream station was less than half that of the upstream station, despite the addition of flow from the drainage channel, so the downstream current was at least twice as fast. Therefore, stream current may have been an influential factor. The lesser abundance of phytoplankton in the channel was to be expected regardless of any influence of atrazine because there was very little time for phytoplankton to become established after the water emerged from the drainage tile. In short, the evidence for an atrazine effect was inconclusive.

The other study reporting effects of small atrazine concentrations was that of Lampert et al. [91]. Lake phytoplankton communities enclosed in plastic tubes 1 m in diameter and 2.7 m long were treated with various concentrations of atrazine. Effects such as reductions in photosynthesis and chlorophyll were reported after exposure to as little as 0.1 μg/L. However, unlike virtually all the other mesocosm and microcosm studies reviewed, the effects in these enclosures did not appear until at

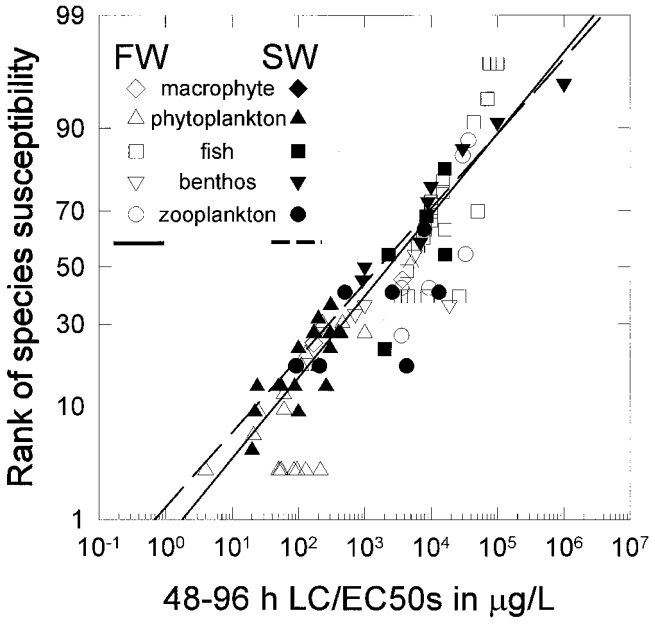

Fig. 20. Comparison of the distribution of acute sensitivity to atrazine in freshwater and saltwater species. Points on the same horizontal line are for the same species, but from different sources/references.

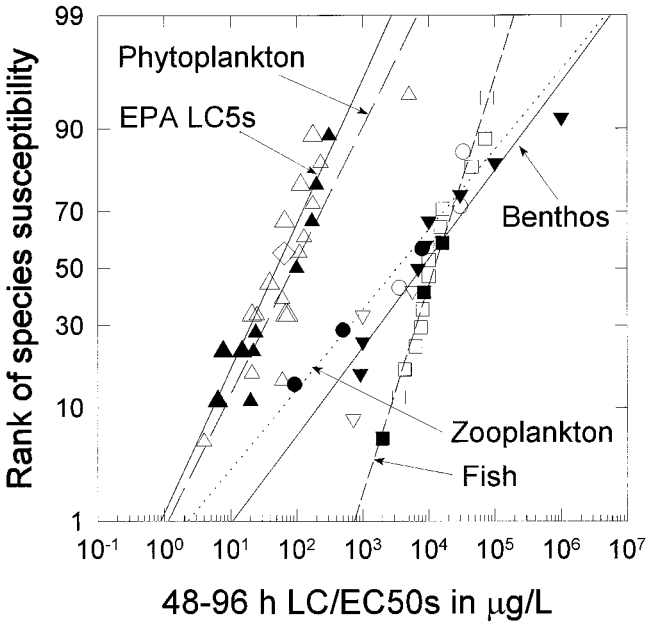

Fig. 21. Comparison of the distributions of acute sensitivity to atrazine in phytoplankton, zooplankton, benthos, and fish. Only the worst-case data (lowest reported values) are plotted. Endpoints and symbols as in Figure 20.

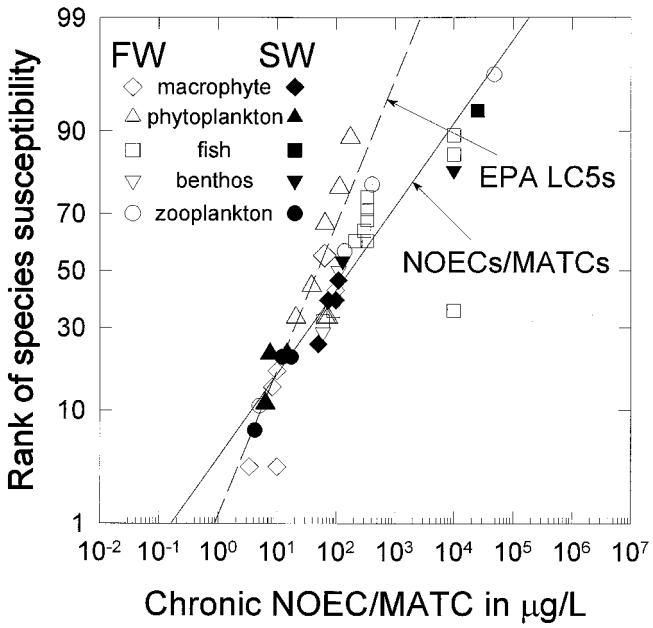

Fig. 22. Distribution of chronic sensitivity to atrazine in aquatic organisms. Endpoints included NOECs, MATCs, and MACs. Points on the same horizontal line are for the same species, but from different sources/references. Symbols as in Figure 19.

least a week, and sometimes as long as 18 d, after atrazine treatment. This trend is inconsistent with the pattern observed by other investigators, in which effects on productivity were greatest immediately after atrazine exposure. Because the descriptions of methods and results by Lampert et al. [91] are incomplete, their conclusions cannot be evaluated. In light of the discrepancies between this study and all other published reports, the occurrence of effects at 0.1 μg/L must be considered questionable.

Periphyton. The effects of atrazine on colonization of foam substrates by microbial communities, taxonomically typical of periphyton communities, in laboratory microcosms have been studied [92]. Atrazine concentrations ranging from 3.2 to 10 μg/L resulted in greater protein biomass and chlorophyll; however, these were less in the presence of 337 μg/L. Initially, dissolved oxygen concentration, as an indicator of primary productivity, was significantly less at 32 μg/L but greater after 21 d of exposure. The number of species in the community was greater at 32 μg/L or less and fewer at 110 μg/L or more. Overall, atrazine concentrations of 10 μg/L or less had no adverse effect on the community, and the only effect at 32 μg/L was a temporary reduction in dissolved oxygen.

When large model streams were treated with 25 μg/L atrazine for four 30-d periods separated by 60-d recovery periods [93], there were no significant effects on periphyton production or biomass at any season. In smaller laboratory streams exposed to 24 μg/L atrazine, Krieger et al. [94] measured a reduction in periphyton ash-free dry weight and chlorophyll at 25°C but not at 10°C; nutrient uptake rates were not affected.

When periphyton communities colonizing artificial streams in the presence of 10 μg/L atrazine were examined, two species of the diatom genus *Rhopalodia* were inhibited, whereas others (the blue-green alga *Chrorococcus* sp. and the diatom *Synedra delicatissima*) became more abundant in the treated streams than in the controls [95]. These taxonomic shifts had no effect on total periphyton biomass. Periphyton productivity was slightly less at 10 μg/L [96]. Total biomass was less only after subsequent exposure to 1,000 μg/L atrazine, a concentration at which some plants (notably *Cladophora*) were almost completely destroyed. Except at 10,000 μg/L, photosynthetic inhibition was incomplete and disappeared within a few days of treatment [96].

Macrophytes. In the University of Kansas studies [81–86], the effects of 20 μg/L atrazine on macrophytes were measured in 1979, 1981, and 1982. In 1979, visual estimates at the end of the season indicated a 60% reduction in macrophyte abundance in the 20-μg/L ponds compared with controls [84,85]. The actual abundances were not reported, but the brief mention of macrophytes by the authors in all of the published reports implies that macrophytes were sparse.

In the 1981 and 1982 studies, macrophytes in the Kansas ponds were investigated more intensely [86]. At the end of the 1981 growing season, total macrophyte abundance in ponds treated with 20 μg/L and 100 μg/L atrazine was not different from controls; however, there were fewer macrophytes at 200 μg/L. Early in 1982, four grass carp were added to each pond, and further effects of atrazine became indistinguishable from effects of grass carp (*Ctenopharyngodon idella*). However, a wire exclosure was constructed in each pond to exclude grass carp from about one-tenth of the pond area. Within these exclosures, macrophyte growth at 20 μg/L was similar to that in controls throughout the summer, but the plant community structure changed. The controls contained vascular plants, *Potamogeton* and *Najas,* in approximately equal abundance. In the 20-μg/L ponds, within the exclosures, *Potamogeton* was gradually replaced by the algal macrophyte *Chara* as the summer progressed. *Chara* was the only plant to survive in the enclosures when concentrations of atrazine were 100 or 200 μg/L.

In similar pond studies, Fairchild et al. [97] also observed a shift in macrophyte community structure—*Chara* replacing *Najas*—without a change in total macrophyte biomass or productivity, after exposure to 50 μg/L atrazine. A shift from the vascular plant *Myriophyllum* to *Chara* was reported in a reservoir treated with another herbicide [98].

Effects of atrazine in wetland ecosystems have also been reported. No difference in plant biomass of prairie wetland microcosms containing filamentous algae, duckweed, and macrophytes (*Potamogeton*) was observed after 6 weeks of exposure to 20 μg/L atrazine [99]. Dissolved oxygen concentrations were less, which implied a reduction in primary productivity. Primary production in prairie wetland microcosms was 23% less after 2 d of exposure to 10 μg/L atrazine and 32% less at 100 μg/L [100]. However, at both exposure concentrations, the effects on production disappeared before the next measurements were made at day 7.

The above studies support the conclusion that atrazine concentrations of 20 μg/L or less result in little or no adverse effects on the function of aquatic plant communities. Effects, when they occurred, were generally short-lived and did not reduce overall plant biomass and primary productivity; however, species composition was affected. At these exposure concentrations, the change in species composition had no discernable effect on other trophic levels. Other studies have shown that atrazine concentrations greater than 100 μg/L are more likely to affect plant communities. In his review of the ecotoxicological literature on atrazine, Huber [101] also concluded that 20 μg/L was an NOEC for aquatic ecosystems.

Recovery of plant communities. Plant communities recover readily from the inhibitory effects of atrazine. Recovery of primary production was observed in phytoplankton and periphyton

Table 30. Effects of atrazine (50 μg/L or less) on plant communities in microcosms and mesocosms

Concn.	Experimental system	Measurement endpoint	Effect	Recovery	Reference
0.5 μg/L	Pond microcosms	Phytoplankton productivity	No effect	—	[78]
2 μg/L	Stream enclosures	Periphyton biomass and abundance	No effect	—	[222]
3.2 μg/L	Pond microcosms	Periphyton biomass, chlorophyll, diversity, productivity	Biomass, chlorophyll, diversity increased; no effect on productivity	—	[92]
5 μg/L	Pond microcosms	Phytoplankton productivity	No effect	—	[78]
10 μg/L	Laboratory streams	Periphyton biomass and productivity; species abundance	No effect on biomass; productivity reduced; some diatom species reduced	3 wk	[95,96]
10 μg/L	Wetland microcosms	Macrophyte biomass and productivity	No effect on biomass; productivity reduced	7 d	[100]
10 μg/L	Pond microcosms	Periphyton biomass, chloro Ephyll, diversity, productivity	Biomass, chlorophyll, diversity increased; no effect on productivity	—	[92]
15 μg/L	Pond microcosms	Phytoplankton chlorophyll, productivity and abundance	Productivity reduced, chlorophyll increased; green unicells decreased, diatoms increased	14 d	[89]
20 μg/L	Pond mesocosms	Phytoplankton biomass, chlorophyll, productivity and abundance	No effect on biomass and productivity; chlorophyll increased; cryptoqmonads increased	—	[81–84,86]
20 μg/L	Pond mesocosms	Macrophyte biomass and abundance	No effect on biomass; *Potamogeton* replaced by *Chara*	—	[81,82,86]
20 μg/L	Pond mesocosms	Macrophyte biomass	Reduced	Not observed	[84,85]
20 μg/L	Naturally derived microcosms	Phytoplankton productivity	No effect	—	[79]
20 μg/L	Wetland microcosms	Macrophyte biomass and productivity	No effect on biomass; productivity reduced	6 wk	[99]
20 μg/L	Pond compartments	Phytoplankton biomass and productivity	No effect	—	[80]
24 μg/L	Laboratory streams	Periphyton biomass and chlorophyll	No effect at 10°C, reduced at 25°C	>12 d	[94]
25 μg/L	Laboratory streams	Periphyton biomass, productivity, and diversity	No effect	—	[122]
30 μg/L	Stream enclosures	Periphyton biomass and abundance	No effect	—	[222]
32 μg/L	Pond microcosms	Periphyton biomass, chlorophyll, diversity, productivity	No effect on biomass or chlorophyll; diversity increased; productivity reduced	>21 d	[92]
50 μg/L	Pond microcosms	Phytoplankton productivity	Reduced	1 d after input ceased	[78]
50 μg/L	Pond mesocosms	Macrophyte biomass, species abundance	No effect on biomass; *Najas* replaced by *Chara*	—	[97]

immediately after atrazine was removed from the water in flow-through laboratory microcosms [78,102]. This was most probably the result of the diffusion of atrazine from the plant tissues into the uncontaminated medium and removal from the site of action. Recovery of phytoplankton productivity after 7 d of exposure to 15 and 153 μg/L atrazine has also been reported [89]. In pond mesocosms, phytoplankton productivity was reduced by exposure to 100, 200, and 500 μg/L but recovered within 3 weeks [82,83]. Phytoplankton abundance and biomass in lake enclosures recovered from 100 μg/L atrazine by the spring following treatment [103]; periphyton biomass, however, did not recover within this time [87].

Evidence that phytoplankton communities can develop resistance to atrazine has been reported by several authors [80,82,83]. Communities exposed to atrazine were shown to be less affected by additional atrazine than were previously unexposed communities. This phenomenon was demonstrated by incubating mesocosm phytoplankton with various atrazine concentrations in the laboratory. Unfortunately, interpretation of these studies was complicated by carryover of atrazine in the mesocosm water. For example, adding 20 μg/L atrazine to phytoplankton and water from 500-μg/L mesocosms would not be expected to have as much impact as adding the same amount of atrazine to phytoplankton and water from control mesocosms. Better evidence of increased phytoplankton resistance came from comparison of effects over multiyear treatments [82]. For example, phytoplankton photosynthesis in mesocosms treated with 100 μg/L in 1981 was reduced but recovered within 3 weeks; in 1982, treatment of the same mesocosms with 100 μg/L had no effect on photosynthesis. Although not well studied, these observations are consistent with the observation that prior

exposure to atrazine confers resistance to subsequent exposures in terrestrial plants [25].

Other investigators have been unable to reproduce the results of deNoyelles et al. [81–83]. Laboratory streams treated with 10 μg/L atrazine for a 3-week colonization period and then assessed for the response of periphyton to additions of 100, 1,000, and 10,000 μg/L showed no difference between the pre-exposed periphyton communities and periphyton that had not been exposed during colonization [95,96]. Others also found that previous exposure to atrazine failed to reduce the impact of subsequent exposures [78,102].

Estuarine macrophytes can recover from the effects of atrazine even while exposure continues. *Potamogeton perfoliatus* photosynthesis was reduced by exposure to 130 μg/L atrazine but returned to pretreatment concentrations within 4 weeks, when 80 to 85% of the atrazine was still present in the water. Recovery was evident even at 1,200 μg/L [21]. *Myriophyllum spicatum* recovered from 100-μg/L exposures within 3 weeks [22]. These authors suggested two possible mechanisms for recovery: enzymatic detoxification, as has been found to occur in corn and other terrestrial plants [25], or inactivation of atrazine by sorption to colloidal material.

The ability of plant community productivity to recover from atrazine even after several weeks of exposure is pivotal for the ecological risk assessment. Brief exposure to inhibitory or algistatic atrazine concentrations may cause temporary inhibition, but, unless the concentrations are sustained for weeks, there is likely to be no lasting effect. For example, Kemp et al. [22] concluded that atrazine exposure in Chesapeake Bay tributaries (with measured concentrations reported as great as 50 μg/L after some runoff events) would cause less than 10% reduction in annual productivity of submersed plants.

Changes in community composition. In several studies, shifts observed in the relative abundance of plant taxa within the community have been reported. The replacement of vascular plants by macrophytic algae (*Chara*) was discussed earlier, and analogous community changes have been recorded in phytoplankton and periphyton studies. In the Kansas mesocosms, planktonic green algae and flagellates were reduced, whereas cryptophytes (especially *Cryptomonas*) and chrysophytes (especially *Mallomonas*) increased in abundance [83,84]. A replacement of green and blue-green algae by *Cryptomonas* and *Rhodomonas* has been observed [80]. Similarly, a reduction in green algae, diatom, and dinoflagellate numbers, no effect on chrysophytes, and a slight increase in cryptophyte numbers were observed in enclosed phytoplankton communities [103].

The relative sensitivity of different taxa is not consistent through all studies. Cryptophytes were resistant to atrazine in the studies mentioned above, cryptophytes as well as diatoms were reduced in laboratory streams exposed to 100 μg/L atrazine, and blue-green algae increased [100]. In another study, 100 μg/L eliminated bluegreen algae but had little effect on diatoms [87]. Sensitivity also varies within major algal groups. For example, among the periphytic green algae in lake enclosures treated with 100 μg/L atrazine, the relative abundances of *Bulbochaete* and *Oedogonium* were lower than in the controls, whereas the relative abundance of *Coleochaete* was greater than in the controls [87].

Although details of the specific taxonomic shifts vary with the situation, there is a tendency for resistant species to expand into niches vacated by sensitive species. This is particularly true for primary producers. Such redundancy is perhaps the most important mechanism for ecosystem recovery from atrazine effects. In terms of total biomass and primary productivity, an aquatic plant community is less sensitive to atrazine than is its most sensitive species.

Indirect effects. Direct effects of atrazine on primary productivity, plant biomass, and community composition can be expected to cause other changes within an aquatic ecosystem. Two general categories of indirect effects have been documented: changes in water quality and reductions in herbivore populations.

Many water quality parameters are influenced by photosynthesis and nutrient uptake. Dissolved oxygen is a very sensitive indicator of changes in photosynthetic rate, and reductions in oxygen concentrations almost invariably accompany atrazine-induced reductions in primary productivity in standing water. Other water quality changes include decreases in pH and increases in alkalinity and conductivity—all related to reduced photosynthetic uptake of bicarbonate. Several investigators have reported increases in inorganic nitrogen [78,89,104] or phosphorus [89,100] due to reductions in nutrient uptake rates. These water quality changes (other than dissolved oxygen) occur only at atrazine concentrations high enough (>100 μg/L) to cause severe effects on the plant community.

Indirect effects of atrazine on animal populations have been observed in a few microcosm and mesocosm studies. In the first of the Kansas mesocosm studies, abundance of the dominant zooplankton species (*Tropocyclops prasinus mexicanus*) was reduced by 75% in ponds treated with 500 μg/L atrazine [83]. In that study, in situ caged exposures of the cladoceran *Simocephalus serrulatus* and laboratory exposures of *Daphnia magna* revealed that reductions in growth and reproduction were related to reductions in phytoplankton abundance. In later studies in Kansas, no significant effects on zooplankton occurred [82]. Numbers of individual species of rotifers (*Conochilus*), cladocerans (*Bosmina*), and copepods (*Diaptomus*) were smaller in lake enclosures treated with 100 μg/L atrazine; however, the total numbers of rotifers, cladocerans, and copepods were not less in the treated compared to the reference enclosures [103]. *Bosmina* increased in outdoor microcosms treated with 15 μg/L atrazine but decreased in microcosms treated with 385 and 2,200 μg/L [89]; rotifers and copepods decreased at treatment concentrations of 153 μg/L and greater.

A variety of effects on macroinvertebrates, especially emergent insects, were reported in the Kansas mesocosms [105–107]. Overall, herbivores were affected more than predators. The changes in macroinvertebrates were attributed to destruction of the macrophyte community by a combination of atrazine and grass carp; effects of atrazine alone were not determined.

Elimination of macrophytes also caused indirect effects on bluegill sunfish in the Kansas mesocosms. Biomass of bluegill sunfish in ponds treated with 100 to 500 μg/L atrazine was 50 to 80% less than in controls and ponds treated with 20 μg/L [82]. The authors suggested that these effects were due to reduced food supply, such as macroinvertebrates associated with submersed vegetation and increased predation by adult bluegill sunfish on juveniles because of the loss of protective cover.

In an earlier study, bluegill sunfish recruitment was less in Kansas mesocosms treated with 20 and 500 μg/L atrazine than in controls [84]. Examination of the stomach contents of adult bluegill sunfish at the end of the season showed a decrease in the number and variety of food items in fish from atrazine-treated ponds [84,85]. Kettle explained these results as indirect effects of macrophyte loss, although macrophytes apparently

were not abundant even in the control mesocosms at that time. The measured reduction in bluegill sunfish recruitment in microcosms treated with 20 μg/L atrazine was not repeatable in later studies at the same concentration.

RISK CHARACTERIZATION

Risk characterization process

The final phase in the risk assessment process was risk characterization. Risk characterization was composed of two parts: risk estimation and risk description [3]. In the risk characterization, exposure and stressor–response profiles were combined to estimate the risk to assessment endpoints. Several approaches were taken to accomplish this. These ranged from comparison of single effect and exposure values through comparison of distributions of effects and exposure to the use of simulation modeling. Uncertainty in all phases of the risk assessment process was identified and quantified, where possible [4].

Risk estimation. The degree of overlap between the distributions of concentration and sensitivity may be used to estimate the risk to aquatic organisms. For example, if the concentration associated with the 10th percentile for acute sensitivity is equal to the 90th percentile for exposure, the overlap would be equivalent to a 0.01 (1%) risk (0.1 multiplied by 0.1) that, at any given time, some species may be exposed at or greater than their individual LC50s. For each exposure data set, the distribution of concentrations in water was compared with the distribution of acute and chronic sensitivities, and the margin of safety at the 10th and 90th percentiles (MOS_{10}) was calculated using the following equation:

$$MOS_{10} = \frac{\textit{sensitivity concentration}_{10}}{\textit{environmental concentration}_{90}}$$

where *sensitivity concentration*$_{10}$ is the 10th percentile for sensitivity distribution and *environmental concentration*$_{90}$ is the 90th percentile for the exposure distribution. For each of the selected exposure data sets, *environmental concentration*$_{90}$ was calculated either directly from the time-weighted data or from the equation for the distribution regression line (Table 31).

Examples of situations with MOS_{10} values greater than and less than 1 are presented in Figures 23 and 24. Each figure shows the distribution of exposure concentrations, the distribution of all acute toxicity values, and the distribution of the EPA LC5 data. For each toxicity data set (all acute values and EPA LC5s), a shaded rectangle was drawn with corners at the 10th percentile for toxicity and the 90th percentile for exposure concentrations. The horizontal width of the box indicates the MOS_{10}. In situations where the 90th percentile for exposure was greater than the 10th percentile for toxicity (e.g., Fig. 24), the margin of safety (Table 31) is less than 1. In addition, the intercept of the environmental concentration distribution with the 10th percentiles of the LC/EC50, EPA LC5, and chronic sensitivity distribution was used to estimate the probability that this concentration would be exceeded (Table 31). Because it is calculated from the 10th percentile of the sensitivity distribution, this probability is related to the MOS_{10} but gives a better estimate of the risk associated with the environmental concentration data set.

The characterization of risks in aquatic ecosystems based on the degree of overlap of distributions of sensitivity and environmental concentrations must consider the ecological relevance of the species most likely to respond. In addition, if these species have societal or commercial value, this should be taken into consideration in the regulatory decision. If these species do not have special societal or economic value and are not keystone species in the ecological community, effects on these organisms may be of minor concern.

Acute exposures. The distributions of species sensitivity were compared with distributions of exposure concentrations for a number of sites on major rivers, streams, and reservoirs in the U.S. and Canada. MOS_{10} values and probabilities of exceeding the *sensitivity concentration*$_{10}$ values were estimated from these data sets (Table 31 and Fig. 25).

When compared with the large data set of sensitivities based on the LC/EC50s (Fig. 25), only one of the high-risk Illinois reservoir sites showed cause for concern, with a MOS_{10} less than 1. As discussed above, these sites were selected for the likelihood of greater contamination, and the organisms most likely to be affected are the phytoplankton. The significance of these organisms in the structure and stability of the community must be assessed in an ecological context, and the impact of other confounding stressors must be taken into account (see below). None of the large rivers and other low-risk sites showed MOS_{10} values of less than 1 (Fig. 25). On this basis, atrazine concentrations are considered not to present an unacceptable risk to these ecosystems.

When compared to the distribution of the EPA LC5s, certain sites associated with smaller watersheds sampled only during the growing season (April to August) and small reservoirs showed MOS_{10} values less than 1 (Fig. 26). MOS_{10} values of less than 1 were associated primarily with high-exposure sites selected from a much larger sampling of Illinois water treatment plants [60], and they are not representative of these sites as a whole (see Fig. 14). Although the higher probability of concentrations greater than the *sensitivity concentration*$_{10}$ in some cases may be influenced by biased or nonrandom sampling during the use season only, it is clear that some of these sites show high probabilities for effects, at least toward the more sensitive phytoplankton/periphyton. The risks to the aquatic ecosystem at these sites should be assessed further with regard to the potential for recovery, responses observed in mesocosm studies, and confounding stressors.

With one exception, for large rivers in the U.S. and Canada and other low-risk sites, the MOS_{10} values with respect to the EPA LC5 data were greater than 1 (Fig. 26). This site on the Missouri River at St. Joseph, Missouri, is located downstream from an atrazine manufacture and formulation facility, and these concentrations more likely represent a point source of contamination than contamination from nonpoint agricultural uses [108]. Risks resulting from these acute exposures were judged not to be of concern.

Chronic exposure data. The 21-d time-weighted average concentrations for atrazine in surface waters in Ohio and Michigan were compared with the distribution of chronic toxicity NOECs, MATCs, and MACs (Fig. 22). Chronic exposure data were only available for a small subset of the monitoring sites (Midwest U.S. streams and rivers). MOS_{10} values were calculated as the difference between the 90th percentile of the concentration distribution and the 10th percentile of the chronic sensitivity distribution (Table 32 and Fig. 27). In addition, the probabilities of exceeding the *sensitivity concentration*$_{10}$ values were estimated from these data sets. The MOS_{10} values for seven sites were less than 1, suggesting that exposures of this duration (about 21 d) will occasionally present risks to some aquatic organisms. Consideration should be taken of the fact that, in this case, the assessment endpoint was the NOEC/MATC/MAC,

Table 31. Regression coefficients for the data distributions, MOS_{10} values, and probability of exposures exceeding the 10th percentile of the sensitivity distribution of acute responses derived from probabilistic risk assessments of acute exposures to atrazine in surface waters of North America[a]

Aquatic system	Location	Date	Ref.	$y = ax + b$		Reg.	Inter-cept	Margin of safety		Intercept with 10th percentile sensitivity line		Data type[a]
				a	*b*	r^2	90%	EPA LC5s	LC50s	EPA LC5s	LC50s	
All LC50s				0.77	2.49	0.96						
EPA LC5s				1.33	2.74	0.75						
Cedar River	Pallisades, IA	1990 (April–August)	[55]				2.14	2.5	17.3	2.5	<0.1	NP4d
Delaware River	Muscotah, KS	1990 (April–August)	[55]				5.8	0.9	6.4	10	<0.1	NP4d
Huron River	Milan, OH	1990 (April–August)	[55]				5.60	1	6.6	10	<0.1	NP4d
Iroquois River	Chebanse, IL	1990 (April–August)	[55]				3.40	1.6	10.9	3.5	<0.1	NP4d
Old Mans Creek	Iowa City, IA	1990 (April–August)	[55]				4.37	1.2	8.5	9	<0.1	NP4d
Roberts Creek	St Olaf, IA	1990 (April–August)	[55]				2.73	2	13.5	5.5	3	NP4d
Sangamon River	Monticello, IL	1990 (April–August)	[55]				0.56	9.6	66	0.1	<0.1	NP4d
Silver Creek	Freeburg, IL	1990 (April–August)	[55]				9.00	0.6	4.1	13	<0.1	NP4d
W. fork of Big Blue River	Dorchester, NE	1990 (April–August)	[55]				1.79	3	20.7	4.5	<0.1	NP4d
Surface water	Ontario/Quebec	1975–1991	[61–64]	1.32	5.89	0.97	2.0	2.8	18.9	3.2	0.2	PID
Illinois water supply reservoirs		1992–1993	[b]				2.86	1.9	12.9	2.3	<0.9	NPID
Selected Illinois water supply reservoirs	ADGPTV	1993–1994	[b]				14.0	0.4	2.6	33.8	<2.2	NPTW
	Carlinville	1993–1994	[b]				11.3	0.5	3.3	23	<2.8	NPTW
	Centralia	1993–1994	[b]				15.0	0.4	2.5	25.3	<1.9	NPTW
	Highland	1993–1994	[b]				26.0	0.2	1.4	43.6	<2.2	NPTW
	Hillsboro	1993–1994	[b]				11.0	0.5	3.4	34.5	<1.9	NPTW
	Nashville	1993–1994	[b]				14.0	0.4	2.6	50.1	2.5	NPTW
	Salem	1993–1994	[b]				48.0	0.1	0.8	25.6	14.8	NPTW
	SAVE	1993–1994	[b]				6.4	0.8	5.8	14.5	<2.2	NPTW
	Waverly	1993	[b]				7.7	0.7	4.8	6.3	<4.2	NPTW
	Wayne city	1993–1994	[b]				13.0	0.4	2.8	19.3	3.2	NPTW
Lost Creek	OH	1983–1993					1.60	3.4	23.1	4.5	0.1	NP4d
Maumee River	OH	1983–1993					3.40	1.6	10.9	6.2	<0.25	NP4d
Honey Creek	OH	1983–1993					4.22	1.3	8.8	8.1	0.03	NP4d
Rock Creek	OH	1983–1993					2.43	2.2	15.2	5.7	<0.03	NP4d
Sandusky River	OH	1983–1993					3.13	1.7	11.8	6.9	<0.03	NP4d
Mississippi River	Baton Rouge, LA	1991	[56]	1.79	5.62	0.98	2.3	2.3	16.1	2.7	<0.1	PID
	Clinton, IA	1991	[56]				0.38	14.2	97.3	<0.8	<0.8	NPTW
	Thebes, IL	1991	[56]				2.5	2.2	14.8	<0.8	<0.8	NPTW
	Hermann, MO	1991	[56]	1.55	5.21	0.93	4.9	1.1	7.5	9	0.5	PID
	Davenport, IA	1986	[c]	3.86	7.00	0.97	0.7	7.7	52.8	0.1	<0.1	PID
	Greenville, MS	1982–1984	[c]	2.03	5.53	0.91	2.3	2.3	16.1	2.2	<0.1	PID
	Helena, AR	1976	[c]	1.49	5.50	0.87	3.3	1.6	11.2	5.6	0.3	PID
	Memphis, TN	1975–1976	[c]	2.12	5.73	0.97	1.8	3.0	20.5	1.1	<0.1	PID
	Quincy, IL	1985	[c]	3.66	6.34	0.99	1.0	5.6	38.6	0.1	<0.1	PID
	St. Gabriel, MS	1975–1990	[c]	2.88	5.10	0.98	2.6	2.1	14.4	1.4	<0.1	PID
	St. Louis, MO	1975–1976	[c]	2.15	5.52	0.96	2.3	2.4	16.4	1.9	<0.1	PID
	Vicksburg, MS	1975–1989	[c]	2.33	5.71	0.99	1.8	3.0	20.9	0.8	<0.1	PID
	Venice, LA	1975–1979	[c]	2.77	5.24	0.98	2.4	2.3	15.6	1.2	<0.1	PID
Missouri River	Kansas City, MO	1976	[c]	1.91	5.32	0.96	3.2	1.7	11.7	4.3	<0.1	PID
	Lexington, MO	1986	[c]	2.35	5.20	0.97	2.9	1.9	12.8	2.8	<0.1	PID
	St. Charles, MO	1976–1985	[c]	2.42	5.40	0.98	2.3	2.3	16.1	1.5	<0.1	PID
	St. Joseph, MO	1975–1976	[c]	1.27	5.28	0.96	6.1	0.9	6.0	11.4	1.2	PID
Ohio River	Dam 53, IL	1991	[56]	1.82	6.08	0.93	1.3	4.2	28.6	0.8	<0.1	PID
	Junction with Mississippi	1975–1983	[c]	2.11	5.99	0.99	1.4	3.9	26.9	0.6	<0.1	PID
	Mount Vernon, IL	1986	[c]	2.27	5.53	0.98	2.1	2.5	17.3	1.5	<0.1	PID
Midwest reservoirs		1992 (April–May)	[56]				2.56	2.1	14.4	<1.3	<1.3	NPID
		1992 (June–July)	[56]				4.63	1.2	8.0	8.9	<1.3	NPID
		1992 (August–September)	[56]				2.71	2.0	13.6	<1.3	<1.3	NPID
		1992 (October–November)	[56]				2.5	2.2	14.8	<1.3	<1.3	NPID

Note: For all LC50s, 10% intercept = 37; for EPA LC5s, 10% intercept = 5.4.

[a] NP = 90th percentile determined nonparametrically, P = 90th percentile determined by regression, ID = instantaneous data were used in the analysis, TW = instantaneous data used with time-weighting, 4d = 4-d running averages used in the analysis.

[b] M. Cheung, personal communication.

[c] K. Balu, personal communication.

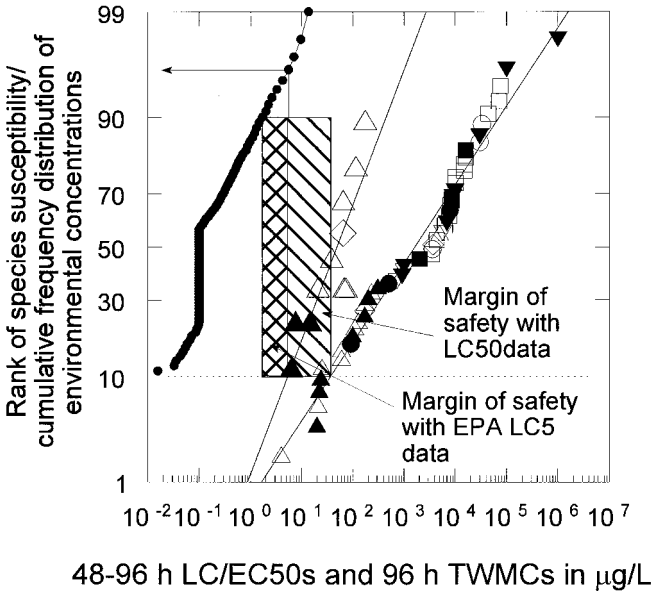

Fig. 23. Comparison between the distribution of atrazine concentrations in Lost Creek, Ohio and the acute sensitivity distribution to atrazine. The rectangles show the MOS_{10} values between the 90th percentile of the exposure distribution and the 10th percentiles of the acute LC/EC5 and LC/EC50 distributions. Only the worst-case data were used in the regression. The horizontal arrow shows the intercept of the 10th percentile of the sensitivity distribution (EPA LC5s) with the exposure distribution. Symbols as in Figure 22.

a concentration at which no responses would be expected. Unlike the LC50 data, some additional margin of safety would be conferred through the use of the NOEC/MATC.

Overlaps between the 21-d time-weighted average concentration and the EPA LC5 toxicity data set were observed at five sites. These tests were conducted with algae and duckweed and represent 5- to 7-d exposures. They are therefore similar to chronic exposures because the organisms undergo several growth cycles during the bioassay period. Thus, acute algal toxicity measurement endpoints are representative of chronic toxicity and the LC5 is representative of a chronic NOEC. This illustrates the conservatism that is conferred by the use of the EPA LC5 distribution but, again, must be interpreted in light of the ecological context and the impact of other confounding stressors.

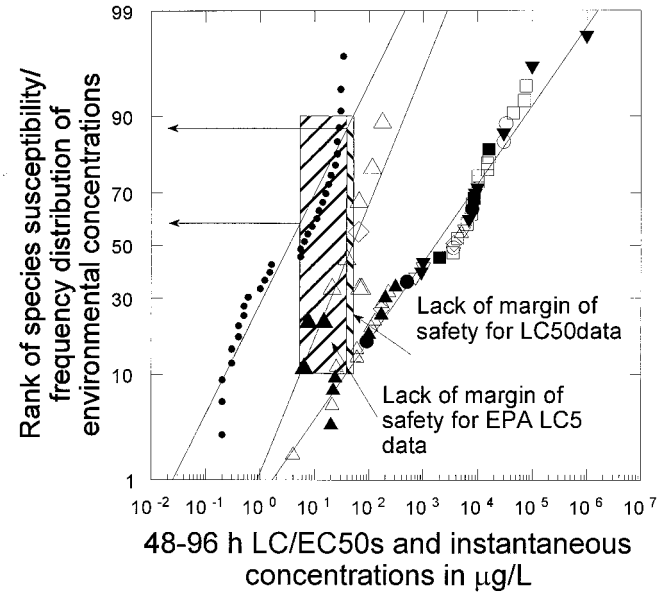

Fig. 24. Comparison between the distribution of atrazine concentrations in Highland, Illinois and the acute sensitivity distribution to atrazine. The rectangles show the lack of a MOS_{10} values between the 90th percentile of the exposure distribution and the 10th percentiles of the acute LC/EC5 and LC/EC50 distributions. Only the worst-case data were used in the regression. The horizontal arrows show the intercept of the 10th percentile of the sensitivity distribution (EPA LC5s and LC/EC50s) with the exposure distribution. Symbols as in Figure 22.

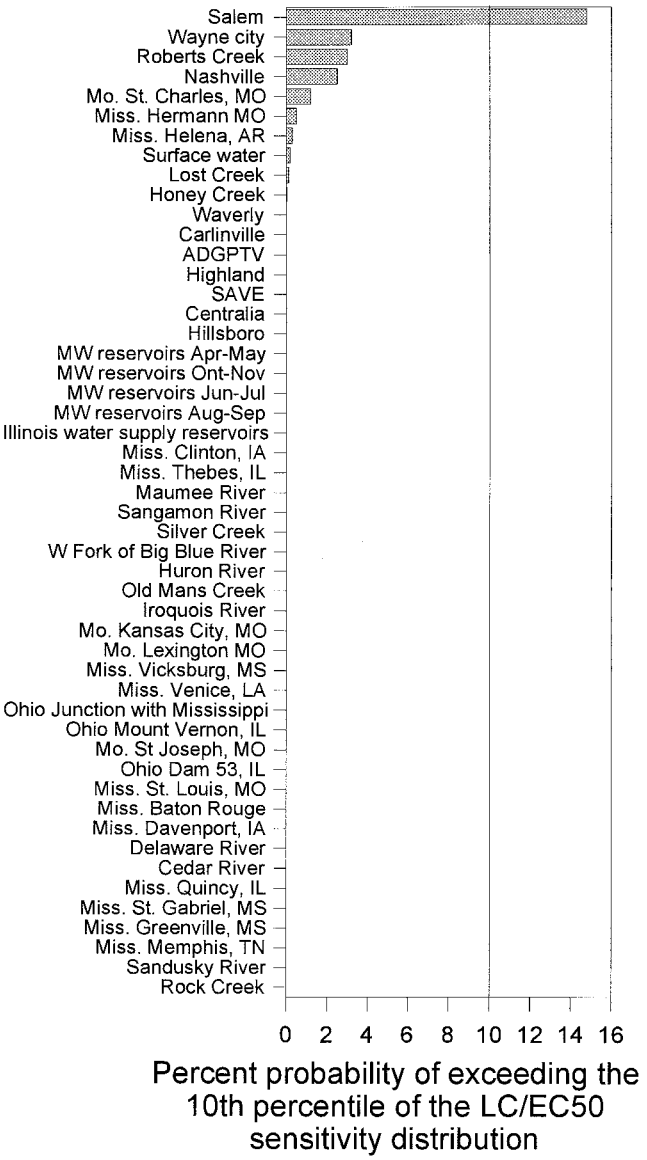

Fig. 25. Probability of exposure to atrazine at 96-h TWMC or instantaneous concentrations above the 10th percentile for acute sensitivity (LC/EC50s) to atrazine in aquatic organisms for selected high-risk sites and other locations in North America. The vertical line shows the point at which the 90th percentile of the exposure distribution would overlap with the 10th percentile of the sensitivity distribution, i.e., where $MOS_{10} = 1$.

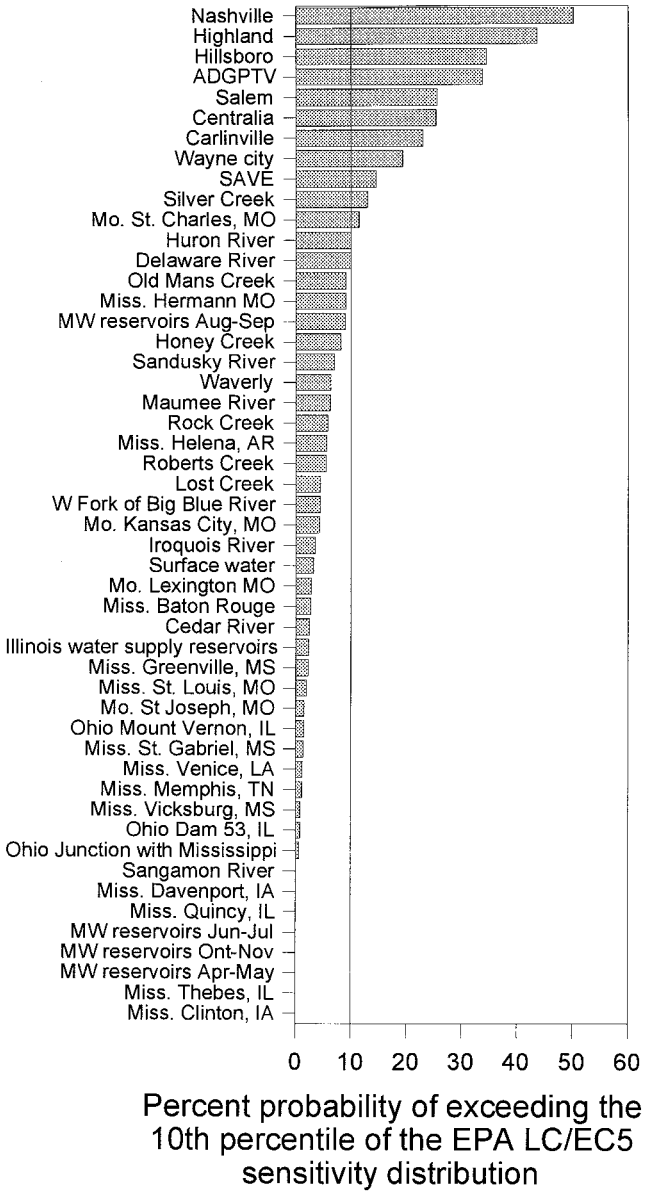

Fig. 26. Probability of exposure to atrazine at 96-h TWMC or instantaneous concentrations above the 10th percentile for acute sensitivity (EPA LC/EC5s) to atrazine in aquatic organisms for selected high-risk sites and other locations in North America. The vertical line shows the point at which the 90th percentile of the exposure distribution would overlap with the 10th percentile of the sensitivity distribution, i.e., where $MOS_{10} = 1$.

Microcosms and mesocosms. Microcosm and mesocosm studies provide measurement endpoints that are closer to assessment endpoints than do laboratory studies. Evaluation of these studies comprises the highest tier in this analysis. Our review of more than 20 microcosm and mesocosm studies showed that atrazine concentrations of 20 μg/L or less resulted in little or no adverse effect on the assessment endpoints, therefore we set this concentration as an approximate NOEC for ecosystem responses.

None of the 4- and 21-d 90th percentiles (Tables 31 and 32) exceeded this no-effect benchmark. Only two sites had 90th percentile instantaneous exposure estimates greater than 20 μg/L (Salem, 48 μg/L; Highland, 26 μg/L). Both of these were small reservoirs in Illinois that were among a high-risk subset ($N = 10$) of 114 community water supplies selected for intensive monitoring. The other eight reservoirs in the select group had 90th percentiles ranging from 6.4 to 15 μg/L (Table 32). The 90th percentile for all 114 sites during 1992 to 1993 was 2.86 μg/L.

Based on this analysis, it was concluded that margins of safety for aquatic ecosystems were adequate in rivers and streams in the Midwest. The Panel suggested, nonetheless, that it would be prudent to conduct site-specific risk assessments for some of the identified high-risk sites, such as the reservoirs. These evaluations should explore mitigation options and possibly include field surveys to assess the status of ecosystems and the likelihood that atrazine is an important factor in any apparent anthropogenic impacts observed.

CONSIDERATION OF UNCERTAINTY

Uncertainty analysis is important because it identifies and, to the extent possible, quantifies the uncertainty in the entire process of problem formulation, analysis, and risk characterization. In addition, an assessment of uncertainty may allow identification of ways in which uncertainty can be reduced. Uncertainties in risk assessment have three sources: (1) imperfect knowledge of things that should be known; (2) systematic errors such as computational, analytical, or data transformation errors (which can be addressed through quality control or, in the case of analytical errors, by use of a correction factor); and (3) nonsystematic errors, i.e., random or stochastic errors and variability that originate in the system being assessed. The latter group of errors can be described and quantified and are automatically considered in probabilistic approaches to risk assessment.

Characterization of uncertainty associated with exposure assessment

Error associated with sampling frequency. An optimized nonpoint-source pollutant sampling strategy that is independent of basin size or stream order has been suggested [109]. These authors concluded that, for storm runoff sampling, a sampling interval equal to 0.05 of the duration of storm flow was adequate to characterize concentration distributions in the runoff with an error less than 5%.

The sampling strategy employed by Richards and Baker [54] for rivers draining agricultural watersheds in northwestern Ohio involved maximum sampling rates of four evenly spaced samples per day during storm events. According to the sampling strategy described by Roman-Mas et al. [109], this sampling interval would have provided concentration distributions with error less than or equal to 5% if the storm runoff lasted more than 5 d. Hydrographs taken during these events (not shown) demonstrated that the majority of storm events resulted in runoff longer than 5 d. Hence, these data can be expected to provide estimates of pesticide distribution in rainfall runoff with errors less than or equal to 5%.

The sampling strategy employed by Klaine et al. [49] called for samples to be taken every 7.5 min for the first 2 h of runoff, every 15 min for the next 4 h of runoff, every 30 min for the next 8 h of runoff, and hourly thereafter. The longest events lasted approximately 1,000 min. Applying the sampling strategy of Roman-Mas et al. [109] to the data from Klaine et al. [49], an optimum sampling interval would have been every 20 min. Thus, for the first 6 h of the runoff sampling was more intense and for the next 8 h sampling was slightly less intense than needed to achieve the optimum rate. However, Klaine et al. [49]

Table 32. 90th percentiles of 21-d time-weighted mean concentrations, MOS_{10} values, and probability of exposures exceeding the 10th percentile of the sensitivity distribution of chronic responses derived from probabilistic risk assessments of acute exposures to atrazine in surface waters of North America; 10th percentile of chronic toxicity data (MATCs/NOECs) = 3.7 μg/L; 10th percentile of EPA LC5 values = 5.4 μg/L

					Margin of safety		Intercept with 10%ile of sensitivity line	
Aquatic system	Location	Date	Reference	Intercept 90%	EPA LC5	MATC/ NOEC	EPA LC5	MATC/ NOEC
Lost Creek	OH	1983–1993		2.71	2.0	1.4	5.3	6.1
Maumee River	OH	1983–1993		3.44	1.6	1.1	5.7	9.3
Honey Creek	OH	1983–1993		4.75	1.1	0.8	8.6	13.1
Rock Creek	OH	1983–1993		3.69	1.5	1.0	5.7	9.9
Sandusky River	OH	1983–1993		3.86	1.4	1.0	6.8	10.3
Cedar River	Pallisades, IA	1990 (April–August)	[55]	2.40	2.2	1.6	<0.1	3.5
Delaware River	Muscotah, KS	1990 (April–August)	[55]	6.07	0.9	0.6	12	17
Huron River	Milan, OH	1990 (April–August)	[55]	5.73	0.9	0.6	12	20
Iroquois River	Chebanse, IL	1990 (April–August)	[55]	3.52	1.5	1.1	<0.1	8
Old Mans Creek	Iowa City, IA	1990 (April–August)	[55]	7.19	0.7	0.5	12	14
Roberts Creek	St Olaf, IA	1990 (April–August)	[55]	9.17	0.6	0.4	11	12
Sangamon River	Monticello, IL	1990 (April–August)	[55]	0.52	10.4	7.2	<0.1	0.2
Silver Creek	Freeburg, IL	1990 (April–August)	[55]	7.71	0.7	0.5	18	21
W. fork of Big Blue River	Dorchester, NE	1990 (April–August)	[55]	2.29	2.4	1.6	5.3	7

also reported that atrazine migrated early in the storm event (first flush). Thus, the majority of the atrazine had migrated from the field during the first 300 min. Hence, these data can be expected to provide estimates of pesticide distribution in rainfall runoff with errors less than or equal to 5%. Details of sampling frequency were unavailable for the other data sets and it is not possible to judge the uncertainty associated with sample collection.

Error associated with chemical analysis. Chemical analysis error is dependent on atrazine concentrations. At lesser concentrations, recoveries of atrazine from water are more variable and quantitation techniques are subject to more interference from matrix effects. At concentrations greater than 5 μg/L, the data supplied by Baker and Richards (D.B. Baker, personal communication) were estimated to be precise within ±10%. For these data sets, errors for concentrations less than 5 μg/L are estimated to be ±50%. Because these errors are random, they are taken into account in the exposure distributions and, in any case, are unlikely to be in the region of overlap.

The data sets from Scribner et al. [55] were derived from ELISA as well as GC-MS methods for quantitation of atrazine. GC-MS was the only quantitative technique for 3% of the nearly 1,800 samples collected in 1990. Some samples (20%) were analyzed by both procedures, whereas the majority (80%) were analyzed by ELISA alone. From the quality assurance samples analyzed along with the field samples, average results for ELISA analysis for concentrations less than 5 μg/L were 180% of theoretical with a standard deviation (SD) of 140%. GC-MS results for sample analysis at the same atrazine concentrations were 130% (SD 40%). At atrazine concentrations greater than 5 μg/L, ELISA results were 88% (SD 45%) of theoretical and GC-MS results were 98% (SD 15%). The relatively few zero values in the data sets [55] may be explained by the overestimation of small values by the ELISA methods. This would bias the exposure distributions to slightly greater concentrations and more conservative estimates of risks. New generation ELISA tests are more precise and accurate [110].

Bias in data reduction. The Ciba analytical data and the Canadian data were corrected for recovery. The concentrations in the data sets of Baker and Richards (D.B. Baker, personal communication) and those of Scribner et al. [55] based on GC-MS methods were not fully corrected for recovery. In each case, quantification involved the use of an internal standard added after extraction. Thus, possible losses prior to or during extraction and some kinds of losses that might occur on-column during chromatography were not accounted for. Such biases would transfer to all forms of reduced or derived data reported here. General experience with analysis for atrazine using GC and GC/MS procedures suggests that unadjusted recoveries are in the range of 75 to 90%. Thus, data reported may be biased from 10 to 25% less than actual values. In the risk characterization, the MOS_{10} values for the sites in the Baker and Richards data set were all greater than 25%, even when assessed against the more conservative EPA EC5 toxicity data. Thus, these systematic errors would not alter the outcome of the risk characterization.

Samples in the Scribner et al. [55] study were filtered before extraction. Some atrazine can be lost due to adsorption to the particulates retained on the filter. In extreme cases, in samples with greater particulate matter and organic carbon content, nearly 50% of the atrazine may be adsorbed (Heidelberg College Water Quality Lab, unpublished data). Loss of adsorbed atrazine due to filtration probably represents an additional small bias in the study [55], the magnitude of which is unknown and presumably variable from sample to sample. Information presented in [55] is insufficient to evaluate possible biases in triazine results based on ELISA methods.

Using distributions of instantaneous measurements in assessments confers a degree of conservatism relative to 4- and 21-d time-weighted means because the latter smooth out short peaks of runoff amounts. This is particularly true if measurements were made only during the season of greater runoff and the season of lesser runoff was not sampled, or if measurements were made primarily during storm runoff events, when concentrations are typically greater. In these situations, the use of distributions of instantaneous measurements will result in a more conservative assessment but also will distort its representativeness.

The use of regressions to obtain a 90th percentile, as recommended in the ARAMDG report [4], can lead to substantial

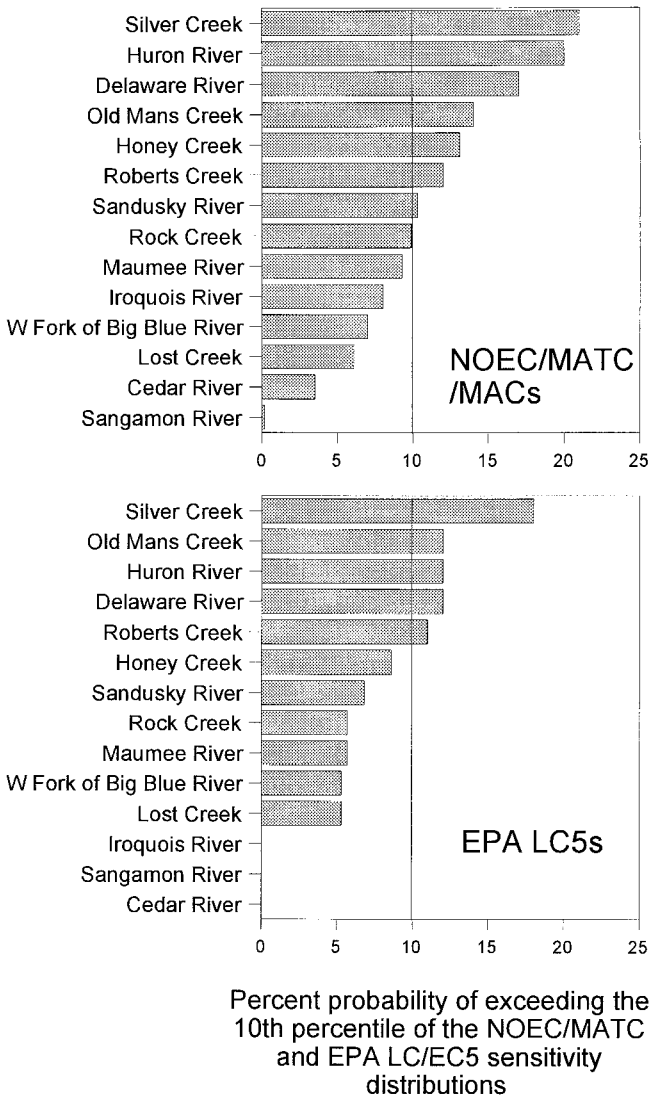

Fig. 27. Probability of exposure to atrazine at 21-d TWMC concentrations above the 10th percentile for chronic sensitivity (NOECs, MATCs, and MACs) and acute susceptibility (EPA LC/EC5s) to atrazine in aquatic organisms in Midwest and Ohio streams and rivers. The vertical line shows the point at which the 90th percentile of the exposure distribution would overlap with the 10th percentile of the sensitivity distribution, i.e., where $MOS_{10} = 1$.

errors, especially if the data are not log-normally distributed and are not uniformly distributed over the annual cycle. In these instances, the nonparametric, time-weighted 90th percentiles of the instantaneous concentrations are preferable to the 90th percentile based on regression.

Effects of annual climatic variability. Pesticide concentrations fluctuate greatly from year to year due to different weather patterns, particularly the amount and timing of rainfall [54]. The data sets of Baker and Richards and the Ontario and Quebec data used in this study span 10 years or more and thus are sufficiently long to encompass most of the range of annual fluctuation, although perhaps not long enough to avoid the influence of long-term climatic cycles. The data of Scribner et al. [55] span only a half year or a year and a half, depending on the station, and thus are potentially more subject to error due to nonrepresentative climatic conditions.

As discussed above, the change in the maximum recommended application rates that were implemented for the first time in 1993 and the increased use of atrazine in no-till situations will likely lead to reduced input into surface waters. The effect of this on the distribution of environmental concentrations will likely not be observable for several years and, even then, may be obscured by other factors, such as changes in rainfall patterns.

Uncertainty associated with ecological effects data

The relatively small number of taxa that can be routinely cultured and tested in laboratory toxicity studies leads to uncertainty when extrapolating these toxicity data to responses of natural populations. Uncertainty also increases when extrapolating from single-species tests to ecosystem assessment endpoints. Fortunately, the database for the toxicity of atrazine is rather robust compared with many other chemicals that have been evaluated in ecological risk assessments. Thus, the likelihood of underestimating risks to keystone species critical to the structure and function of the community or ecosystem was judged to be small.

Variability in the results of toxicity tests for a given species tested in different experiments or by different authors is a potential source of both random and systematic errors. In this assessment, the most conservative (smallest concentration) effect value was used where multiple studies were reported. The range of toxicity test results within and among trophic groups was relatively large in some cases. This was taken into consideration in developing the distribution of sensitivity. Where the range of values in a data set is large, distributions will have lesser slopes with greater probability of overlap.

In chronic studies, the range of test concentrations can influence the calculation of the NOEC, LOEC, and MATC values. It is difficult to correct for this type of uncertainty within these data sets. Poor selection of concentrations for these assays will result in greater values for the estimates of LOEC or MATC, and these systematic errors are thus more likely to be less conservative.

Uncertainty associated with the risk assessment

The probabilistic approach used in this risk assessment allows for a quantitative estimation of risks (analyzed as a distribution of sensitivity and exposure concentrations) and therefore incorporates many of the uncertainties associated with variability in the effects and exposure characterizations discussed above [4].

Considerations of biological uncertainty

In addition to uncertainty in the exposure and effects data, biological and ecological uncertainty must be considered. This includes consideration of the potential for organisms such as plants to recover from exposure, the effects of confounding stressors such as sediment loading, and ecological redundancy of the functions of the affected species.

Uncertainty as a result of recovery from exposure

As discussed above, the mechanism by which atrazine blocks the photosynthetic pathway is reversible. When exposure is reduced below the threshold of inhibition, photosynthesis returns to activities dictated by the concentration of atrazine present in the cell and simple competitive binding kinetics. The ultimate risk to plants thus depends on both the duration and magnitude of exposure, the rates of metabolism and detoxification, and the length of the recovery period. Exposure to atrazine in rivers and streams occurs in pulses related to rainfall events. These are separated by periods when exposure is absent or very small.

Table 33. Parameter values used in the algal biomass dynamics model

Variable	Description	Units	Value
μ_{max}	Maximum algal growth rate	/h	0.073
ρ	Algal respiration rate	/h	0.031
K_L	Michaelis–Menton half-saturation constant for light	cal/cm^2/min	0.50
K	Light extinction coefficient	/m	0.03
k_s	Extinction from suspended sediment	L/mg/m	0.004[a]
I*	Maximum incident solar radiation	cal/cm^2/min	1.0
z	Water depth	m	1.0
Θ_ρ	Empirical constant for ρ	—	1.047
Θ_μ	Empirical constant for μ	—	1.089

[a] See text for discussion of values for k_s.

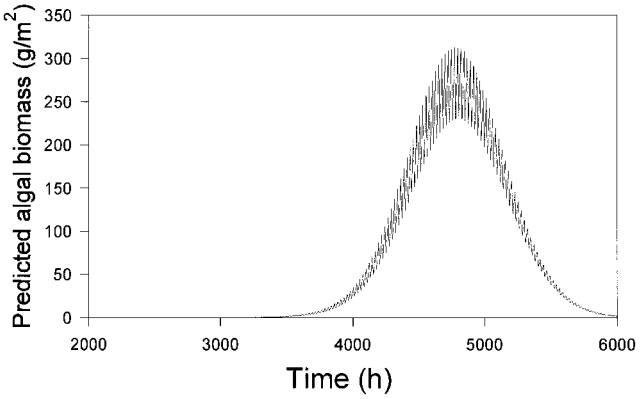

Fig. 28. Results of model calibration without atrazine or suspended sediment effects.

Thus, unless the plants die as a result of consuming all their energy reserves, full recovery may be possible.

Short-term (i.e., 5- to 7-d) toxicity tests with algae do not take into account this potential for rapid recovery when atrazine concentrations decline. Field studies have shown that inhibition of photosynthesis by atrazine exposures to 20 μg/L or less is short-lived and recovery normally occurs within 1 to 3 weeks. In some cases, recovery of photosynthesis (from the activity of atrazine-resistant species) has been observed even while atrazine was still present in the water.

Uncertainty resulting from confounding stressors

Models of algal growth show that the sediment loadings that accompany atrazine runoff events have a greater effect on photosynthesis than does atrazine itself. Thus, reductions in photosynthesis in periphyton as a result of runoff events from agricultural or other disturbed land will continue to occur, even in the absence of atrazine.

The Panel used a model of algal biomass dynamics to simulate the effects of atrazine and suspended sediment concentrations in a stream receiving surface runoff during a series of storm events. The algal species of interest were periphyton on the stream bottom. Because there were few laboratory data on photosynthesis in periphyton, we used data from laboratory experiments on a phytoplankton species, the green alga *S. capricornutum,* because of the sensitivity of this species to atrazine.

Simulation results and discussion. The model of algal biomass dynamics was calibrated without either atrazine or suspended sediment effects. The parameter values for the calibrated model are listed in Table 33. The algal respiration rate, *p,* was used to calibrate the model to yield a maximum net growth rate of 400 mg/m^2/h and a peak biomass of approximately 300 g/m^2 (Fig. 28). Limiting factors such as nutrients and grazing by zooplankton were not included in the model. The comparison between atrazine and suspended sediment effects, however, was not affected by these exclusions.

Forcing functions for atrazine and suspended sediment were developed using data for Rock Creek, provided by Baker and Richards. We used the data from Rock Creek for the initial forcing functions for the years 1983 to 1994. A cubic spline function was used to interpolate between data points. All negative values resulting from the interpolation were set to zero.

Effects of atrazine. The effects of atrazine were simulated for the years 1983 to 1994. Predicted effects of atrazine on algal biomass depend upon the frequency, magnitude, and duration of atrazine pulses and the time of the pulses relative to the growing season. Years with few or infrequent pulses (e.g., 1988) had little effect. Pulses that contained atrazine concentrations less than 20 μg/L, even during the peak growing season, reduced algal growth only slightly. Only pulses containing atrazine concentrations greater than 40 μg/L during the growing season (e.g., 1986, 1990, and 1991) caused reductions in algal growth that were judged to be ecologically important. Where pulse concentrations were great enough to cause an effect, those of short duration had less effect than those of longer duration. Pulses that came early or late in the growing season (e.g., 1984, 1987, and 1994) had little effect on algal growth.

These different scenarios of exposure and response to pulses of atrazine concentrations can be illustrated by comparing 1985 with 1990. In 1985 (Fig. 29), several small pulses between 2,000 and 3,600 h were too small and too early in the growing season to have an effect on algal growth. At 3,700 h, a pulse with a peak of about 35 μg/L reduced algal growth only slightly. Subsequent pulses throughout the rest of the growing season were too small to have any effect. In 1990 (Fig. 30) small pulses early in the growing season had little effect on algal production. Beginning at about 3,700 h, the same time as in 1985, a pulse occurred that exceeded the 1985 pulse in magnitude and du-

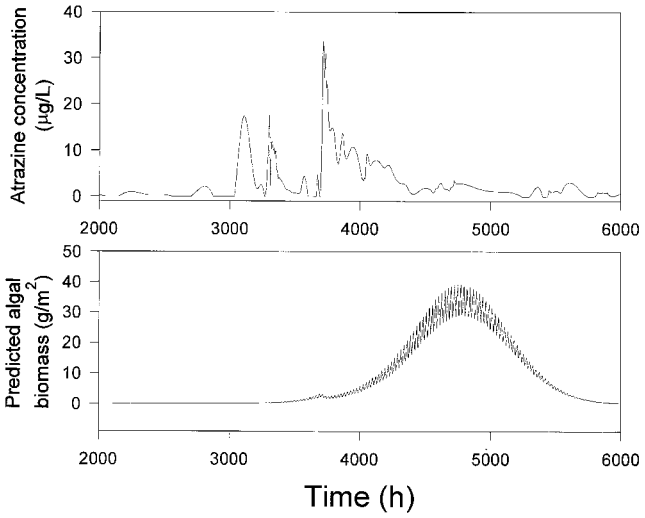

Fig. 29. Observed and predicted atrazine concentrations (μg/L) (top) and predicted algal concentrations (mg/m^2) (bottom) for Rock Creek in 1985.

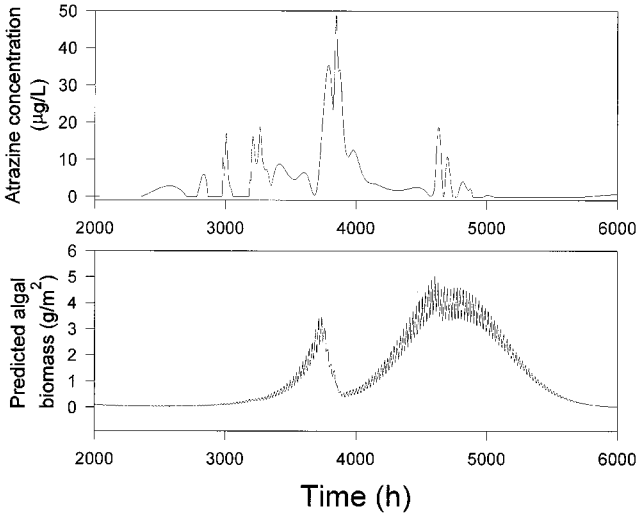

Fig. 30. Observed and predicted atrazine concentrations (μg/L) (top) and predicted algal concentrations (mg/m²) (bottom) for Rock Creek in 1990.

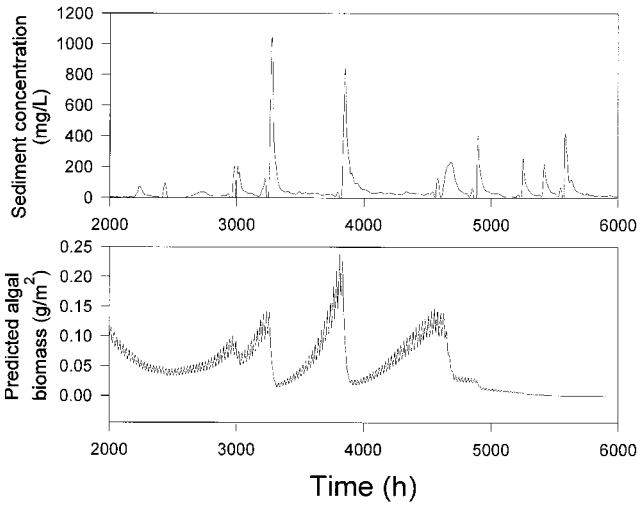

Fig. 31. Observed and predicted sediment concentrations (μg/L) (top) and predicted algal concentrations (mg/m²) (bottom) for Rock Creek in 1990.

ration. The result was a negative rate of net productivity and a decrease in biomass. After the pulse passed, the algal population recovered. A second series of lesser magnitude, short-duration pulses beginning at about 4,600 h caused only a slight reduction in biomass.

Effects of suspended sediment. We also simulated the effects of suspended sediment for the years 1983 to 1994. We used an initial value of 0.12 L/mg/m for k_s, the first-order extinction coefficient for effects of suspended sediment on light attenuation. These initial simulations predicted complete extinction of algae in all years. The value of 0.12 L/mg/m from Verduin [111] for k_s is typical of values found in the literature. Extinction coefficients including all factors (K) of approximately 2 to 3/m and suspended sediment concentrations of 14.6 to 29.8 mg/L have been measured in the tidal Potomac River [112]. These data would yield k_s values of 0.10 to 0.14 L/mg/m. Values of k_s ranging from 0.32 to 0.82 L/mg/m with a mean of 0.54 L/mg/m have been reported from six natural water bodies in Georgia, Mississippi, and Montana [113]. In a simulation of phytoplankton productivity in partially mixed estuaries, Peterson and Festa [114] used a k_s value of 0.10 L/mg/m. They also reported values of k_s from the literature ranging from 0.03 to 0.16 L/mg/m.

We set the value of k_s in the model to the smallest value reported in the literature (0.03 L/mg/m) to obtain a positive net growth rate. Whereas atrazine exposure usually occurs early in the growing season, suspended sediment can impact algal growth at any time during the growing season. When runoff of sediment occurs early and throughout the season, algal biomass is reduced to nearly zero, and although it increases later, it does not fully recover (e.g., 1984 to 1987, 1990, and 1992). Where there are long intervals between storms, algae may recover, but a heavy pulse of sediment will reduce algal biomass to zero (e.g., 1983, 1989, and 1993). Years with lesser sediment loads early and through the season (e.g., 1988, 1991, and 1994) allow algae to grow until the end of the growing season.

The years 1990 and 1991 illustrate the different responses to suspended sediment. In 1990, a series of pulses through the growing season reduced algal biomass to near zero (Fig. 31). In 1991, there were fewer pulses and they were of smaller magnitude than in 1990 (Fig. 32). They also occurred early in the growing season, which allowed a relatively great biomass to accumulate.

Combined atrazine and suspended sediment effects. Because suspended sediment had a greater impact on algal growth than did atrazine, the combined effects reflected those of suspended sediment alone. We made several assumptions concerning the model that led to a conservative simulation scenario relative to atrazine effects. (1) A sensitive species, *S. capricornutum,* was used to represent the biomass dynamics of periphyton. (2) A conservative function was used to represent the response of *S. capricornutum* to atrazine, even though the data suggested a stimulatory effect at lesser concentrations of atrazine. (3) An extremely small value of the coefficient of light extinction resulting from suspended sediment was used to yield a positive net rate of productivity.

A less conservative approach to assumptions 1 and 2 would reduce the predicted effects of atrazine and result in less biomass depression and shorter recovery times. A light extinction co-

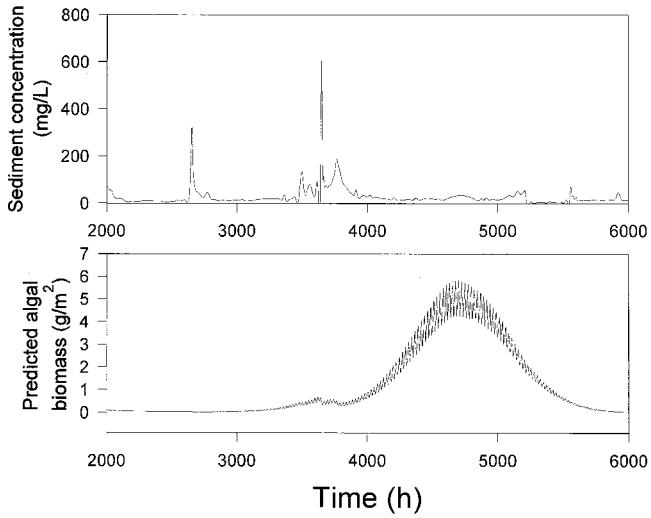

Fig. 32. Observed and predicted sediment concentrations (μg/L) (top) and predicted algal concentrations (mg/m²) (bottom) for Rock Creek in 1991.

efficient closer to more commonly observed values would result in greater effects of suspended sediment on predicted algal growth. Thus, other stressors, such as sediments present in these aquatic systems, probably have a greater effect on primary productivity of algae than does atrazine. It is also likely that, because these sediments attenuate light, they will actually protect chlorophyll from the combined effects of atrazine and light.

Other authors have noted the effects of confounding stressors on the potential effects of atrazine on aquatic algae. Water taken from Walnut Creek (IA) and Goodwater Creek (MO) was observed to have variable effects on *S. capricornutum* when assayed in the laboratory, causing stimulation of algal growth on some occasions and depression at other times [115]. Because the concentrations of herbicides were all less than 2 μg/L, this effect was ascribed to other causes. It has been suggested that the possible effects of atrazine and other herbicides in the Mississippi River on marine phytoplankton in the Gulf of Mexico would be confounded by other factors, such as sediment loading, nutrient addition, and changes in salinity [116].

Interactions with other chemicals present in surface waters may also influence the risk from atrazine. It is known that several other herbicides, including some of the triazines, are also found in surface waters [55]. Because of their physical properties, simazine and cyanazine would not be expected to be transported into surface waters and, indeed, are found less frequently and at smaller concentrations [55] (M. Cheung, personal communication). Because of their similar mechanism of action, the toxic effects of these compounds would be expected to be additive to atrazine and were judged to be of minor consequence. Alachlor and metolachlor are sometimes also found in surface waters but usually at smaller concentrations than atrazine. They have different mechanisms of action from atrazine and would not be expected to be additive or synergistic in their activity toward aquatic organisms. The possibilities of interactions with myriad other substances that may be present in surface waters are infinite. However, the probability that two or more potentially interactive chemicals will be present at the same time and in concentrations great enough to actually have interactive effects was judged to be small and of little consequence.

Uncertainty resulting from ecological redundancy

Although some phytoplankton were the most sensitive to atrazine of the bioassay organisms in the laboratory, there is a considerable range of sensitivities among and within the phytoplankton taxa, as well as within a single species. Field studies show that resistant taxa tend to replace more sensitive taxa in atrazine-stressed phytoplankton communities, reducing the impact on overall community productivity and biomass. Similar shifts are commonly observed in macrophyte communities. As a consequence, aquatic plant community functions, as a whole, are considerably less sensitive to the effects of atrazine than are the most sensitive plant species. The NOEC for plant biomass and productivity based on the microcosm and mesocosm studies is about 20 μg/L, which is more than five times greater than the 10th percentile for chronic toxicity (3.7 μg/L) and corresponds to about the 30th percentile for chronic toxicity. It is clear that the structure of aquatic communities may be changed by atrazine; however, the functions of the species affected are taken over by other organisms. Thus, these species are neither keystone species in the ecological sense nor socially or commercially valued. They would also be expected to recolonize rapidly from unexposed refugia, and any structural changes that occurred would be short-term in nature.

Interpretation of ecological significance

Human activities in the environment may result in a number of changes in the physical, chemical, and biological characteristics of the ecosystem. Interactions may be complex, with some stressors being additive or synergistic, whereas others may partially or wholly nullify each other. Although the probabilistic risk assessment procedure did identify some situations where greater than desired overlap between the distributions existed, data from mesocosm and field studies could be used to better understand the ecological significance of these observations. Organisms at greatest direct risk from atrazine exposures are the phytoplankton and, by implication, the periphyton and macrophytes (Fig. 21). This risk must be qualified by our knowledge of the mode of action of atrazine and its biological relevance. The following considerations apply:

Because the responses of plants to atrazine are reversible, effects will be transient. The probabilistic risk assessment was conservative because it was based on the responses of the more sensitive species exposed to a continuous, maximum concentration and did not consider recovery.

Resiliency has been demonstrated in the function of phytoplankton exposed to atrazine in ponds and microcosms. Atrazine-resistant species are able to maintain levels of primary productivity.

Field experiments indicate that laboratory toxicity tests overestimate the potential response of aquatic plant communities to atrazine. Through physiological adaptation and species shifts, plant communities can compensate for atrazine effects and reduce the impact on productivity and biomass. Although these compensating factors do not rule out the possibility for secondary effects on higher trophic levels, for example, by causing shifts in the diets of herbivores, such indirect effects have generally been observed only at relatively great concentrations of atrazine that cause drastic changes in aquatic plant communities. The results of microcosm and mesocosm studies are therefore inconsistent with the perception that atrazine and its breakdown products trigger deleterious effects at concentrations less than those predicted by laboratory studies.

Confounding stresses, such as sediment loading, that accompany greater atrazine concentrations associated with runoff events also inhibit photosynthesis and, in fact, may protect chlorophyll from atrazine-induced damage. Plants in the receiving ecosystems have evolved mechanisms to survive natural stresses such as these. These mechanisms also protect plants from possible effects of atrazine.

CONCLUSION OF THE ATRAZINE RISK ASSESSMENT

Our conclusion is that, in most situations, the probability that atrazine concentrations in surface waters in North America will exceed the 10th percentile of the sensitivity distribution is low and these concentrations do not present an ecologically significant risk to the aquatic environment. The probability of risk from the greatest atrazine exposures is higher in some small watersheds with extensive pesticide use and in reservoirs that receive drainage from these watersheds. In these situations, site-specific risk assessments should be conducted to assess the relevance of the likely effects of exposure, the use to which these particular ecosystems are put, and the effectiveness and cost–benefit aspect of any risk mitigation measure that may be applied.

The quality and quantity of monitoring and effects data available for atrazine were probably much greater than for any other pesticide. In spite of all of these data, recognizable areas for further investigation were identified. This ecological risk assessment focused on a compound that entered the aquatic ecosystem through nonpoint-source runoff. It was clear from the exposure analysis that aquatic organisms were subjected to episodic pulses of varying frequency and magnitude. Toxicity data for this type of exposure scenario were lacking. Research is needed, not only for atrazine but for any compound with similar episodic exposures, to characterize the response of aquatic organisms to pulsed exposures. This includes a need for characterizing both response to the exposure and recovery following exposure at environmentally relevant concentrations. As a part of this risk assessment, a model was developed to simulate algal response to episodic exposure. Strategically designed algal and macrophyte toxicity assessments with atrazine are needed to validate and parameterize this model.

During the exposure analysis, a major attempt was made to calibrate and validate surface runoff models to predict surface water atrazine concentrations in watersheds where monitoring data were absent or inadequate. This effort revealed a lack of correspondence between observed and predicted concentrations of atrazine in surface waters. Models that are routinely used in pesticide exposure assessment either overpredicted or underpredicted atrazine concentrations by as much as an order of magnitude. Research is needed to refine these models and to validate the refined models with independent data from freshwater and estuarine ecosystems. For the foreseeable future, models may be best used to identify geographic areas or specific use situations where additional field monitoring is needed but should not serve as a primary source of exposure data for risk assessment.

More research is needed to identify the most appropriate methods for calculating percentiles of environmental concentrations when data are seasonal in nature, not uniformly distributed in time, or depart significantly from the assumed lognormal distribution. Failure to use appropriate methods may lead to errors in estimation of higher percentiles that exceed an order of magnitude and errors in calculated margins of safety that exceed two orders of magnitude.

With sufficient continued monitoring, the overall trend toward reduced atrazine loading into aquatic systems caused by the combination of use rate reductions, use restrictions, and alterations of agricultural management practices may become discernable. Monitoring focused on certain key regions should be continued to better understand the effects of these mitigation methods.

Although the probabilistic risk assessment model is attractive for its relative simplicity and robustness to the addition of data, it needs to be validated in well-designed microcosm or field studies.

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