Atrazine
Chemical Summary

U.S. EPA, Toxicity and Exposure Assessment for Children's Health

This TEACH Chemical Summary is a compilation of information derived primarily from U.S. EPA and ATSDR resources, and the TEACH Database. The TEACH Database contains summaries of research studies pertaining to developmental exposure and/or health effects for each chemical or chemical group. TEACH does not perform any evaluation of the validity or quality of these research studies. Research studies that are specific for adults are not included in the TEACH Database, and typically are not described in the TEACH Chemical Summary.

I. INTRODUCTION

Atrazine (6-chloro-N-ethyl-N'-(1-methylethyl)-1,3,5-triazine-2,4-diamine) is a white, crystalline solid that is used as a synthetic herbicide to kill broadleaf weeds in agricultural and roadway applications (1, 2). Atrazine is one type of triazine herbicide, a chemical group that also includes simazine and cyanazine (2). Atrazine is the most commonly used herbicide in the United States, with application of approximately 76 million pounds active ingredient in 1997 (1). Atrazine is a Restricted Use Pesticide (RUP), which means that only registered professionals can apply atrazine, and it is not available to the general public (1-3). Atrazine is most commonly used on corn crops, with a large proportion of usage in the "Corn Belt" of the U.S., predominantly in the Midwest (1, 2). Atrazine is also used on sugarcane crops; and on roadway grasses, golf course turf, and residential lawns, predominantly in the Southeastern U.S. (1, 2).

The primary target of atrazine in humans and animals is the endocrine (hormonal) system (1, 2). Studies thus far suggest that atrazine is an endocrine disruptor; an agent that has been shown to alter the natural hormonal system in animals (1, 2). Implications of possible endocrine disruption for children's health are related to effects during pregnancy and during sexual development, though few studies are available. Increased risks for preterm delivery and intrauterine growth retardation have been associated with atrazine exposure (4, 5). Atrazine exposure has been shown to result in delays or changes in pubertal development in experimental animal studies (6-12). Recent studies of atrazine exposure of frogs suggest atrazine may impact sexual development, though the U.S. EPA concluded that data are currently insufficient to draw conclusions, and implications of these data for children's health remain unclear (1, 2). Studies of non-mammalian species are not included in the TEACH Database. There is minimal evidence suggesting possible carcinogenic effects of atrazine exposure (1, 2, 13). Chlorinated metabolites (break-down products) of atrazine are generated in animal tissues, soil, and water; and are considered to be equal in toxicity to atrazine (1).

Exposure to children can occur primarily from ingestion of contaminated drinking water, and from dermal contact or ingestion following agricultural and lawn applications (1-3).

II. EXPOSURE MEDIA AND POTENTIAL FOR CHILDREN'S EXPOSURE[1]

Exposure Media	Relative Potential for Children's Exposure[2, 3]	Basis[4]
Groundwater	Higher	Atrazine is a commonly reported groundwater contaminant, and degrades slowly once in water. Atrazine has been detected in groundwater at higher levels in some regions of the U.S. where atrazine is used on some crops and turf.
Surface Water	Higher	Atrazine contamination of surface water (lakes, rivers, and streams) is a concern to the U.S. EPA. Atrazine is the most commonly detected pesticide in U.S. surface waters, particularly in the U.S. Midwest Corn Belt region.
Drinking Water	Higher	Drinking water from a contaminated groundwater or surface water source can be a significant medium of exposure for children.
Diet	Medium	Atrazine is used on several crops, primarily on corn, sugarcane, and sorghum. Fruits, nuts, and grains are also treated. Exposure to the parent compound, atrazine, through food is considered lower, although exposure increases when chloro- and hydroxy-metabolites of atrazine in foods are considered.
Soil	Lower	Atrazine remains in soil for a matter of months and can migrate from soil to groundwater, but is not likely to be present in residential soil. The U.S. EPA requires that professional applicators water areas after application of atrazine to reduce the potential for exposure via residential soil.
Sediment	Lower	There is little data on the presence of atrazine in sediment. Existing data suggest little partitioning to sediment.
Ambient Air	Lower	Atrazine does not volatilize readily, making it a low concern for outdoor air.
Indoor Air	Lower	Atrazine is not used indoors and does not volatilize readily, making it a low concern for indoor air.

[1] For more information about child-specific exposure factors, please refer to the Child-Specific Exposure Factors Handbook (*http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=55145*).

[2] The Relative Potential for Children's Exposure category reflects a judgment by the TEACH Workgroup, U.S. EPA, that incorporates potential exposure pathways, frequency of exposure, level of exposure, and current state of knowledge. Site-specific conditions may vary and influence the relative potential for exposure. For more information on how these determinations were made, go to *http://www.epa.gov/teach/teachprotocols_chemsumm.html*.

[3] Childhood represents a lifestage rather than a subpopulation, the distinction being that a subpopulation refers to a portion of the population, whereas a lifestage is inclusive of the entire population.

[4] Information described in this column was derived from several resources (e.g., 1, 2) including studies listed in the TEACH Database (*http://www.epa.gov/teach*).

III. TOXICITY SUMMARY[5, 6]

Effects reported in adults (human and experimental animals) include shortening of estrous cycle length, attenuation of the LH (leutenizing hormone) surge, decreases in pituitary hormone levels, ovarian histopathology (changes in ovarian tissue), and liver effects including increased serum lipids and liver enzymes, and liver histopathology (2). Other effects on the central nervous system, immune system, and cardiovascular function have been reported in adults (2). Exposure to atrazine may be associated with some types of non-Hodgkin's lymphoma in adult humans (1, 2). Significantly increased risk of preterm delivery, intrauterine growth retardation, and decreased birth weight were significantly associated with atrazine concentrations in drinking water (4, 5).

Several experimental animal studies reported reproductive and endocrine effects following atrazine exposure (6-12, 14, 15). Reproductive studies with experimental animals (6, 8) reported increased incidence of preterm delivery and intrauterine growth retardation. Delayed onset of puberty occurred in young male (6, 7) and female (10-12, 16, 17) rats exposed to atrazine. Exposure to atrazine may be associated with mammary tumors in at least one strain of adult rats (17).

Chlorinated metabolites of atrazine (e.g., diethylatrazine (DEA), di-isopropyl-atrazine (DIA), and diaminochlorotriazine (DACT)) are generated in animal tissues, soil, and water, and are considered equal in toxicity to atrazine (1).

Carcinogenicity weight-of-evidence classification[7]: The U.S. EPA posted a draft Carcinogenicity Hazard Assessment and Characterization in 1999 (13), and an addendum to the most recent IRED (3) classified atrazine as not classifiable due to insufficient evidence for carcinogenicity. The World Health Organization International Agency for Research on Cancer (IARC) classifies atrazine as not classifiable (Group 3) as to carcinogenicity in humans (http://monographs.iarc.fr/ENG/Monographs/vol73/volume73.pdf).

[5] Please refer to research article summaries listed in the TEACH Database for details about study design considerations (e.g., dose, sample size, exposure measurements).

[6] This toxicity summary is likely to include information from workplace or other studies of mature (adult) humans or experimental animals if child-specific information is lacking for the chemical of interest. Summaries of articles focusing solely on adults are not listed in the TEACH Database because the TEACH Database contains summaries of articles pertaining to developing organisms.

[7] For recent information pertaining to carcinogen risk assessment during development, consult "Guidelines for Carcinogen Risk Assessment and Supplemental Guidance on Risks from Early Life Exposure" at *http://www.epa.gov/cancerguidelines*.

IV. EXPOSURE AND TOXICITY STUDIES FROM THE TEACH DATABASE

This section provides a brief description of human and animal studies listed in the TEACH Database. For more details about study design parameters, e.g., doses and exposure information, please refer to article summaries in the TEACH Database. Any consideration should include an understanding that exposure levels in animal studies, in many cases, are greater than exposure levels normally encountered by humans.

A. *HUMAN EXPOSURE AND EFFECTS*

- Studies of contaminants in ground water and well water indicate that atrazine is a common contaminant (18-21). The U.S. EPA National Survey of Pesticides in Drinking Water Wells found that atrazine was one of the most frequently detected herbicide or pesticide in domestic water wells (18, 20, 21).
- Atrazine was not detected in a New Zealand survey of infant formula and infant weaning foods (22). Testing a variety of foods in the U.S. diet by the U.S. EPA detected very low levels of atrazine (1). An exposure study in Minnesota detected atrazine in foods that comprise children's diet (23, 24). This Minnesota study also compared estimates of exposure predicted from parental responses to a questionnaire, with actual exposure levels of atrazine measured in air, foods, dust, soil, and in urine of 102 children (23, 24), and the authors concluded that questionnaire results underestimated atrazine exposure levels of the children.
- As a biomarker of exposure, concentrations of atrazine or an atrazine metabolite (atrazine mercapturate) have been measured in children's urine (23, 24). Atrazine mercapturate was detected in urine from 3 of 102 children tested in Minnesota in one study (24). The metabolite was not detected in children's urine in another Minnesota study (23) or in a national study (National Health and Nutrition Examination Survey (NHANES)) (25). It should be noted that atrazine mercapturate is detected in urine for only 24-48 hours after exposure, and thus is a measure of recent exposure only (2).
- There are very few studies of atrazine toxicity in children (2-5, 26). One study indicated that increased risk of preterm delivery and intrauterine growth retardation correlated with increasing levels of atrazine in maternal drinking water that contained a mixture of several pesticides, and an effect of the mixture could not be ruled out (4). Another study reported decreased birth weight was significantly associated with seasonal variations in atrazine concentrations in drinking water (5). One study of childhood cancers (bone and brain cancers, and lymphomas and leukemias) found increased incidence of these cancers was significantly associated with concentrations of three chemicals (atrazine, nitrates, and metachlor) together in drinking water, but not any of the three chemicals alone (26).

B. *EXPERIMENTAL ANIMAL EXPOSURE AND EFFECTS*

- Several animal studies suggest that atrazine affects endocrine and reproductive systems. Atrazine is thought to bind to the androgen receptor, and it may affect the neuroendocrine system by changing pituitary hormone levels such as leutenizing hormone and follicle stimulating hormone, both of which are critical for pregnancy (reviewed in 17).
- Atrazine exposure may affect germ cells (eggs and sperm). In one study, reduced sperm number and motility were observed in male rats injected with atrazine (8). In another study, decreased mating success (pregnancy) was observed following oral atrazine exposure of adult female, but not male rats; litter size was unaffected (27).
- Studies of embryotoxic effects following maternal atrazine exposure during pregnancy have yielded varied results. Maternal oral (28) or injection (29) exposure of rats to atrazine during pregnancy resulted in increased fetal death; the doses that caused increased fetal death, as well as the degree of fetal death at the higher doses, varied between genetically distinct strains of rats (28, 29). In another study, prenatal exposure of rats and rabbits via oral maternal exposure to atrazine resulted in embryotoxic effects only at doses that caused severe maternal toxicity (30).
- Delayed mammary gland development in female offspring at puberty was reported following exposure of female rats *in utero* and during lactation via gavage (tube-feeding) of their mothers (16). A subsequent study reported that exposure in utero or during lactation each alone resulted in delayed mammary gland development in female offspring (31).
- Effects on the immune system in exposed offspring have been studied. In one study, suppression of cellular immune responses and IgM antibody responses in adult male offspring, but not female offspring, was reported following *in utero* exposure via gavage of their mothers during pregnancy (32). In another study, enhanced T lymphocyte proliferation and cytolytic activity were observed in adult male, but not female, offspring who were exposed as fetuses and during lactation (33). Male rat offspring exposed to atrazine only during lactation via maternal breast milk from their orally-exposed mothers (exposed on postnatal days 1-4) exhibited increased inflammation of the prostate in adulthood, well after cessation of atrazine exposure (14).
- Onset of puberty in male rats was delayed following prepubertal oral exposure to atrazine (6) or atrazine metabolites (7). In these studies, atrazine exposure resulted in decreased food consumption and weight loss, and the authors concluded that decreased food consumption contributed to some (6) or all (7) of the observed effects of delayed onset of puberty in these specific studies. No such delays in the onset of puberty were observed in a separate study in rats (9).
- Onset of puberty in female rats was also delayed following prepubertal oral exposure to atrazine (10, 12) or atrazine metabolites (11). These delays were attributed to atrazine exposure and not to decreased food consumption (10).

V. CONSIDERATIONS FOR DECISION-MAKERS

This section contains information that may be useful to risk assessors, parents, caregivers, physicians, and other decision-makers who are interested in reducing the exposure and adverse health effects in children for this particular chemical. Information in this section focuses on ways to reduce exposure, assess possible exposure, and, for some chemicals, administer treatment.

- Atrazine is most commonly used on corn and sugarcane crops, and on residential lawns in Florida and other states in the Southeast. Heaviest atrazine uses per unit area, due primarily to agricultural use, occur in portions of Delaware, Iowa, Illinois, Indiana, Ohio, and Nebraska. Levels of atrazine in community water systems in these states, and in Kentucky, Louisiana, and Missouri, are of particular concern (1, 20).
- For individuals living in states of potential concern for atrazine, the U.S. EPA urges owners of private wells to consider testing their water (34); you can contact your local state EPA for information. Detailed information about concentrations of atrazine in drinking water from samples collected in several states is available (1, 18, 20, 21).
- The major metabolite of atrazine, atrazine mercapturate, was measured in urine as part of the second report of the ongoing national exposure assessment, National Health and Nutrition Examination Survey (NHANES), conducted by the U.S. Centers for Disease Control (25). Atrazine mercapturate was not detected in any samples collected from over 1,000 children between the ages of 6 and 19 years for NHANES in 1999-2000 (25). However, atrazine mercapturate is detected in urine for only 24-48 hours after exposure (2), so the lack of detection in these samples provides limited information about exposure.
- Chlorinated metabolites of atrazine (e.g., diethylatrazine (DEA), di-isopropyl-atrazine (DIA), and diaminochlorotriazine (DACT)) are considered equivalent in toxicity to atrazine, and exposure to metabolites are also of concern (1, 2).
- A risk assessment for atrazine, revised in 2003, is available from the U.S. EPA (1). Potential adverse effects on development were discussed, with remaining concerns that neuroendocrine consequences for children had not been previously addressed in toxicity testing. A cumulative risk assessment for three chlorinated triazine pesticides (atrazine, simazine, and propazine), which share a similar mechanism of action, is also available from the U.S. EPA (35).
- A drinking water atrazine exposure assessment (3) and risk assessment (36) were also performed by the U.S. EPA and the World Health Organization, respectively. In 1989, the World Health Organization set a guideline value of less than 2 µg/L atrazine in drinking water. This guideline value is similar to the U.S. EPA Maximum Contaminant Level (MCL) of 3 µg/L in drinking water (last reviewed in 2003).
- Caregivers may consider an alternate water supply, e.g. bottled water, where atrazine-contaminated ground water may be impacting drinking water.
- The U.S. EPA Office of Pesticide Programs (OPP) issued an Interim Reregistration Eligibility Document (IRED) for Atrazine in 2003 (1). The Atrazine IRED contains U.S. EPA's conclusions regarding atrazine exposure and potential human health and environmental risks of atrazine use. The IRED also includes U.S. EPA requirements for appropriate uses and labeling of the products. The interim status of the IRED was changed to Final status in 2006 (3).

- A detailed compilation and analysis of information pertaining to exposure and health effects of atrazine is also available in the U.S. ATSDR Toxicological Profile for Atrazine (2).
- The U.S. EPA IRED contains information about an agreement between U.S. EPA and Syngenta, a major producer of atrazine, to monitor 40 indicator U.S. watersheds in 10 states (Ohio, Indiana, Kentucky, Illinois, Iowa, Missouri, Nebraska, Minnesota, Tennessee, and Louisiana) for atrazine contamination (1).
- Consult "Child-Specific Exposure Factors Handbook," EPA-600-P-00-002B, for factors to assess children's drinking water consumption rates (37). An updated External Draft of the 2006 version of this handbook is available (38).

VI. TOXICITY REFERENCE VALUES

Atrazine

A. Oral/Ingestion

U.S. EPA Reference Dose (RfD) for Chronic Oral Exposure: 3.5E-2 (or 0.035) mg/kg-day, based on decreased body weight gain in adult rats, with supporting developmental studies in rats and rabbits (http://www.epa.gov/iris/subst/0209.htm, I.A.1) (39). Last agency verification date 9/93.

U.S. EPA Chronic Dietary Reference Dose (RfD): 0.018 mg/kg/day, based on attenuation of pre-ovulatory lutenizing hormone (LH) surge , as a biomarker indicative of hypothalamic funtion disruption (http://www.epa.gov/oppsrrd1/REDs/atrazine_ired.pdf) (40). Last revised 2003.

U.S. EPA Acute Dietary Reference Dose (RfD) for Females 13-50 Years Old: 0.1 mg/kg/day, based on delayed hardening of cranial bones in fetuses (http://www.epa.gov/oppsrrd1/REDs/atrazine_ired.pdf) (40). Last revised 2003.

U.S. EPA Maximum Contaminant Level (MCL) for Drinking Water: 0.003 mg/L, based on potential cardiovascular or reproductive toxicity (http://www.epa.gov/safewater/contaminants/index.html) (41). Last revised 6/03.

U.S. EPA Maximum Contaminant Level Goal (MCLG): 0.003 mg/L, based on potential cardiovascular or reproductive toxicity (http://www.epa.gov/safewater/contaminants/index.html) (41). Last revised 6/03.

U.S. ATSDR Minimal Risk Level (MRL): 0.01 mg/kg/day (acute oral), based on decreased body weight gain; and 0.003 mg/kg/day (intermediate oral), based on reproductive effects (http://www.atsdr.cdc.gov/mrls.html) (42). Last revised 9/03.

B. Inhalation

No toxicity reference values for inhalation are available at this time.

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Hydroxyatrazine

A. Oral/Ingestion

U.S. EPA Chronic Dietary Reference Dose (RfD): 0.01 mg/kg/day, based on histopathologic lesions of the kidneys (http://www.epa.gov/oppsrrd1/REDs/atrazine_ired.pdf) (40). Last revised 2003.

B. Inhalation

No toxicity reference values for inhalation are available at this time.

VII. U.S. FEDERAL REGULATORY INFORMATION

- In January, 2003, U.S. EPA issued an Interim Reregistration Eligibility Decision (IRED) for Atrazine (Case #0062), providing specific information to be used in risk assessments (1). Atrazine is a Restricted Use Pesticide (RUP), such that only registered professionals can use atrazine (1). An addendum to the IRED was added in October, 2003 (3). The interim status of the IRED was changed to Final status in 2006 (3).
- Atrazine was included on the list of chemicals to monitor in food under the Food Quality Protection Act (FQPA) of 1996 (43). This law sets standards for levels of pesticides in foods, and includes special consideration of children when setting those standards (43). Atrazine was re-evaluated by the FQPA Safety Committee in 2000 to take into account potential developmental toxicity, and current standards were found to be protective of these effects (44).
- The U.S. EPA requires reporting of quantities of certain chemicals that exceed a defined reportable quantity, and that quantity varies for different chemicals (45). Under the Emergency Planning and Community Right-to-Know Act (EPCRA) Section 313 “Toxic Chemicals,” quantities of atrazine greater than 25,000 pounds manufactured or processed, or greater than 10,000 pounds otherwise used, is required; under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), there are no reportable quantities for atrazine (45).

VIII. BACKGROUND ON CHEMICAL

A. CAS Number: 1912-24-9

B. Physicochemical Properties: Atrazine is a white crystalline solid that is moderately soluble in water. Go to the National Library of Medicine ChemID Web site (*http://chem.sis.nlm.nih.gov/chemidplus*) and search for atrazine.

C. Production: There are 12 states with a total of 24 production facilities, with the largest formulation facilities in Alabama, Mississippi, Missouri; and the largest atrazine processing volume in Louisiana with up to 49 million pounds active ingredient (2).

D. Uses: Atrazine is one of the most widely used herbicides in the U.S. (46). Total annual U.S. domestic use was estimated to be about 76 million pounds per year (active ingredient) between 1990-1997 (1, 2). The U.S. EPA Toxic Release Inventory (TRI) reported atrazine total releases and disposals of 504,187 pounds in 2005 (47); however, these reported releases do not include reports of all atrazine releases, such as atrazine applied to crops, a major use of atrazine (2). Atrazine is used on corn, sugarcane, and sorghum crops, and for forestry and turf maintenance (1, 2). Atrazine is used to treat approximately 75% of corn crops and 76% of sugarcane crops in the U.S. (1).

E. Environmental Fate: Atrazine persists in surface and groundwater with a half life of longer than 6 months, with some tendency to bind to sediment (2). Atrazine has a slight tendency to bioaccumulate in some aquatic organisms, including invertebrates and fish (2). Atrazine persistence in soil generally ranges from 14-109 days, though in some soils can persist to at least 4 years (1, 2). Atrazine can be found in the particulate and vapor phases of air following application, and can be transported up to 186 miles from the site of application (2). In air, atrazine can be degraded by reacting with hydroxyl radicals (2).

F. Synonyms and Trade Names: 6-Chloro-n-ethyl-n'-(1-methylethyl)-1,3,5-triazine-2,4-diamine; 2-Chloro-4-ethylamino-6-isopropylamine-s-triazine; Aatrex; Aktikon; Crisatina; Fenamin; Hungazin; Weedex A; Zeazine; and others (for a more complete list, go to *http://www.atsdr.cdc.gov/toxprofiles/tp153.pdf*, p. 140).

Additional information on atrazine is available in the TEACH Database for Atrazine and at the following Web sites:

http://www.epa.gov/safewater/dwh/c-soc/atrazine.html
http://extoxnet.orst.edu/pips/atrazine.htm
http://health.usgs.gov/dw_contaminants/domestic_wells/focazio_and_others_2006.pdf

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