

INFORMATION ON
CONTAMINANTS OF CONCERN

EAST KAPOLEI II PESTICIDE MIXING AND LOADING SITE
DEPARTMENT OF HAWAIIAN HOME LANDS
EWA, OAHU, HAWAII

This fact sheet answers the most frequently asked health questions (FAQs) about arsenic. For more information, call the ATSDR Information Center at 1-800-232-4636. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It is important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Exposure to higher than average levels of arsenic occur mostly in the workplace, near hazardous waste sites, or in areas with high natural levels. At high levels, inorganic arsenic can cause death. Exposure to lower levels for a long time can cause a discoloration of the skin and the appearance of small corns or warts. Arsenic has been found in at least 1,149 of the 1,684 National Priority List sites identified by the Environmental Protection Agency (EPA).

What is arsenic?

Arsenic is a naturally occurring element widely distributed in the earth's crust. In the environment, arsenic is combined with oxygen, chlorine, and sulfur to form inorganic arsenic compounds. Arsenic in animals and plants combines with carbon and hydrogen to form organic arsenic compounds.

Inorganic arsenic compounds are mainly used to preserve wood. Copper chromated arsenate (CCA) is used to make "pressure-treated" lumber. CCA is no longer used in the U.S. for residential uses; it is still used in industrial applications. Organic arsenic compounds are used as pesticides, primarily on cotton fields and orchards.

What happens to arsenic when it enters the environment?

- Arsenic occurs naturally in soil and minerals and may enter the air, water, and land from wind-blown dust and may get into water from runoff and leaching.
- Arsenic cannot be destroyed in the environment. It can only change its form.
- Rain and snow remove arsenic dust particles from the air.
- Many common arsenic compounds can dissolve in water. Most of the arsenic in water will ultimately end up in soil or sediment.
- Fish and shellfish can accumulate arsenic; most of this arsenic is in an organic form called arsenobetaine that is much less harmful.

How might I be exposed to arsenic?

- Ingesting small amounts present in your food and water or breathing air containing arsenic.
- Breathing sawdust or burning smoke from wood treated with arsenic.
- Living in areas with unusually high natural levels of arsenic in rock.
- Working in a job that involves arsenic production or use, such as copper or lead smelting, wood treating, or pesticide application.

How can arsenic affect my health?

Breathing high levels of inorganic arsenic can give you a sore throat or irritated lungs.

Ingesting very high levels of arsenic can result in death. Exposure to lower levels can cause nausea and vomiting, decreased production of red and white blood cells, abnormal heart rhythm, damage to blood vessels, and a sensation of "pins and needles" in hands and feet.

Ingesting or breathing low levels of inorganic arsenic for a long time can cause a darkening of the skin and the appearance of small "corns" or "warts" on the palms, soles, and torso.

Skin contact with inorganic arsenic may cause redness and swelling.

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Almost nothing is known regarding health effects of organic arsenic compounds in humans. Studies in animals show that some simple organic arsenic compounds are less toxic than inorganic forms. Ingestion of methyl and dimethyl compounds can cause diarrhea and damage to the kidneys

How likely is arsenic to cause cancer?

Several studies have shown that ingestion of inorganic arsenic can increase the risk of skin cancer and cancer in the liver, bladder, and lungs. Inhalation of inorganic arsenic can cause increased risk of lung cancer. The Department of Health and Human Services (DHHS) and the EPA have determined that inorganic arsenic is a known human carcinogen. The International Agency for Research on Cancer (IARC) has determined that inorganic arsenic is carcinogenic to humans.

How can arsenic affect children?

There is some evidence that long-term exposure to arsenic in children may result in lower IQ scores. There is also some evidence that exposure to arsenic in the womb and early childhood may increase mortality in young adults.

There is some evidence that inhaled or ingested arsenic can injure pregnant women or their unborn babies, although the studies are not definitive. Studies in animals show that large doses of arsenic that cause illness in pregnant females, can also cause low birth weight, fetal malformations, and even fetal death. Arsenic can cross the placenta and has been found in fetal tissues. Arsenic is found at low levels in breast milk.

How can families reduce the risks of exposure to arsenic?

If you use arsenic-treated wood in home projects, you should wear dust masks, gloves, and protective clothing to decrease exposure to sawdust.

- If you live in an area with high levels of arsenic in water or soil, you should use cleaner sources of water and limit contact with soil.
- If you work in a job that may expose you to arsenic, be aware that you may carry arsenic home on your clothing, skin, hair, or tools. Be sure to shower and change clothes before going home.

Is there a medical test to determine whether I've been exposed to arsenic?

There are tests available to measure arsenic in your blood, urine, hair, and fingernails. The urine test is the most reliable test for arsenic exposure within the last few days. Tests on hair and fingernails can measure exposure to high levels of arsenic over the past 6-12 months. These tests can determine if you have been exposed to above-average levels of arsenic. They cannot predict whether the arsenic levels in your body will affect your health.

Has the federal government made recommendations to protect human health?

The EPA has set limits on the amount of arsenic that industrial sources can release to the environment and has restricted or cancelled many of the uses of arsenic in pesticides. EPA has set a limit of 0.01 parts per million (ppm) for arsenic in drinking water.

The Occupational Safety and Health Administration (OSHA) has set a permissible exposure limit (PEL) of 10 micrograms of arsenic per cubic meter of workplace air ($10 \mu\text{g}/\text{m}^3$) for 8 hour shifts and 40 hour work weeks.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2007. Toxicological Profile for Arsenic (Update). Atlanta, GA: U.S. Department of Public Health and Human Services, Public Health Service.

Where can I get more information? For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology and Environmental Medicine, 1600 Clifton Road NE, Mailstop F-32, Atlanta, GA 30333. Phone: 1-800-232-4636, FAX: 770-488-4178. ToxFAQs Internet address via WWW is <http://www.atsdr.cdc.gov/toxfaq.html>. ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.



This fact sheet answers the most frequently asked health questions (FAQs) about atrazine. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It is important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: The general population is probably not exposed to atrazine. However, exposure to atrazine may occur at farms where it has been sprayed. Atrazine may affect pregnant women by causing their babies to grow more slowly than normal. Birth defects and liver, kidney, and heart damage has been seen in animals exposed to high levels of atrazine. This chemical has been found in at least 20 of the 1,636 National Priorities List sites identified by the Environmental Protection Agency (EPA).

What is atrazine?

Atrazine is an herbicide that does not occur naturally. Pure atrazine is an odorless, white powder that is not very volatile, reactive, or flammable and that will dissolve in water.

Atrazine is used to kill weeds, primarily on farms, but has also been used on highway and railroad rights-of-way. The EPA now restricts how atrazine can be used and applied; only trained people are allowed to spray it.

What happens to atrazine when it enters the environment?

- Atrazine enters the environment primarily through spraying on farm crops.
- In soil, atrazine is taken up by the plants growing in the soil or is broken down over a period of days to months.
- It may also wash from soil into streams or groundwater where it will stay for a long time, because breakdown of the chemical is slow in water.
- If atrazine enters the air, it may be broken down by reactions with chemicals in the air, or it may adhere to particles such as dust which eventually settle out of the air.
- Atrazine does not accumulate in living organisms such as algae, bacteria, clams, or fish.

How might I be exposed to atrazine?

- Most people are not exposed to atrazine on a regular basis.
- It is rarely found in food samples; when found, it is only at very low levels.
- Farm workers, chemical sprayers, and people who work in factories that make atrazine may be exposed.
- People may also be exposed to atrazine by digging in dirt that has atrazine in it.
- Individuals may also be exposed by drinking water from wells that are contaminated with the herbicide.

How can atrazine affect my health?

Liver, kidney, and heart damage has been observed in animals exposed to atrazine; we do not know if this would also occur in humans. Atrazine has also been shown to cause changes in blood hormone levels in animals that affected ovulation and the ability to reproduce. These effects are not expected to occur in humans because of specific biological differences between humans and these types of animals.

How likely is atrazine to cause cancer?

Available information is inadequate to definitely state whether atrazine causes cancer in humans. There are limited

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human and animal data that suggest that there may be a link between atrazine exposure and various types of cancer. A Cancer Assessment Review Committee (CARC) sponsored by EPA has classified atrazine as not likely to be carcinogenic to humans. The International Agency for Research on Cancer (IARC) has determined that atrazine is not classifiable as to its carcinogenicity to humans.

How can atrazine affect children?

Little information is available regarding the effects of atrazine in children. It is likely that the health effects seen in children should be similar to the effects seen in adults. We do not know whether children differ from adults in their susceptibility to atrazine.

A few studies are available that suggest that atrazine could affect pregnant women by causing their babies to grow more slowly than normal or by causing them to give birth early. However, the women in these studies were also exposed to other chemicals that may have caused or contributed to these effects. In pregnant animals, exposure to atrazine causes a decrease in fetal growth and birth defects. Exposure to high levels of atrazine during pregnancy caused reduced survival of fetuses. It is unclear whether or at what level of exposure this might occur in humans.

How can families reduce the risk of exposure to atrazine?

- The general population is not likely to be exposed to large amounts of atrazine. Populations living in the areas where atrazine is used on crops, however, may be exposed to greater amounts of atrazine. Therefore, staying away from fields that have been recently sprayed may reduce exposure.
- Atrazine may be washed from fields where it is sprayed into streams and rivers or may migrate into wells used for drinking and bathing. In areas of high atrazine use, individuals should avoid swimming in or drinking from

contaminated water sources and may desire to have personal well water tested for the presence of atrazine.

- Children should avoid playing in soils near uncontrolled hazardous waste sites where atrazine may have been discarded.

Is there a medical test to show whether I've been exposed to atrazine?

Atrazine can be detected in your blood and some other body tissues within 24 to 48 hours after your last exposure. These tests are not usually available at your doctor's office, but your doctor can send the samples to a laboratory that can perform the tests. None of these tests, however, can predict whether you will experience any health effects.

Has the federal government made recommendations to protect human health?

The EPA has set a maximum amount of atrazine in drinking water of 0.003 milligrams of atrazine per liter of drinking water (0.003 mg/L).

The Occupational Safety and Health Administration (OSHA) has set a limit of 5 milligrams of atrazine per cubic meter of workplace air (5 mg/m³) for an 8-hour workday and 40-hour work week.

The EPA has determined maximum levels allowed in foods of 0.02-15 parts atrazine per million parts of food (0.02-15 ppm).

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2003. Toxicological Profile for Atrazine. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

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This fact sheet answers the most frequently asked health questions (FAQs) about chlorinated dibenzo-p-dioxins (CDDs). For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It's important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Exposure to chlorinated dibenzo-p-dioxins (CDDs) (75 chemicals) occurs mainly from eating food that contains the chemicals. One chemical in this group, 2,3,7,8-tetrachlorodibenzo-p-dioxin or 2,3,7,8-TCDD, has been shown to be very toxic in animal studies. It causes effects on the skin and may cause cancer in people. This chemical has been found in at least 91 of 1,467 National Priorities List sites identified by the Environmental Protection Agency (EPA).

What are CDDs?

CDDs are a family of 75 chemically related compounds commonly known as chlorinated dioxins. One of these compounds is called 2,3,7,8-TCDD. It is one of the most toxic of the CDDs and is the one most studied.

In the pure form, CDDs are crystals or colorless solids. CDDs enter the environment as mixtures containing a number of individual components. 2,3,7,8-TCDD is odorless and the odors of the other CDDs are not known.

CDDs are not intentionally manufactured by industry except for research purposes. They (mainly 2,3,7,8-TCDD) may be formed during the chlorine bleaching process at pulp and paper mills. CDDs are also formed during chlorination by waste and drinking water treatment plants. They can occur as contaminants in the manufacture of certain organic chemicals. CDDs are released into the air in emissions from municipal solid waste and industrial incinerators.

What happens to CDDs when they enter the environment?

- When released into the air, some CDDs may be transported long distances, even around the globe.

- When released in waste waters, some CDDs are broken down by sunlight, some evaporate to air, but most attach to soil and settle to the bottom sediment in water.
- CDD concentrations may build up in the food chain, resulting in measurable levels in animals.

How might I be exposed to CDDs?

- Eating food, primarily meat, dairy products, and fish, makes up more than 90% of the intake of CDDs for the general population.
- Breathing low levels in air and drinking low levels in water.
- Skin contact with certain pesticides and herbicides.
- Living near an uncontrolled hazardous waste site containing CDDs or incinerators releasing CDDs.
- Working in industries involved in producing certain pesticides containing CDDs as impurities, working at paper and pulp mills, or operating incinerators.

How can CDDs affect my health?

The most noted health effect in people exposed to large amounts of 2,3,7,8-TCDD is chloracne. Chloracne is a severe skin disease with acne-like lesions that occur mainly on the face and upper body. Other skin effects noted in people exposed to high doses of 2,3,7,8-TCDD include skin rashes, dis-

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coloration, and excessive body hair. Changes in blood and urine that may indicate liver damage also are seen in people. Exposure to high concentrations of CDDs may induce long-term alterations in glucose metabolism and subtle changes in hormonal levels.

In certain animal species, 2,3,7,8-TCDD is especially harmful and can cause death after a single exposure. Exposure to lower levels can cause a variety of effects in animals, such as weight loss, liver damage, and disruption of the endocrine system. In many species of animals, 2,3,7,8-TCDD weakens the immune system and causes a decrease in the system's ability to fight bacteria and viruses. In other animal studies, exposure to 2,3,7,8-TCDD has caused reproductive damage and birth defects. Some animal species exposed to CDDs during pregnancy had miscarriages and the offspring of animals exposed to 2,3,7,8-TCDD during pregnancy often had severe birth defects including skeletal deformities, kidney defects, and weakened immune responses.

How likely are CDDs to cause cancer?

Several studies suggest that exposure to 2,3,7,8-TCDD increases the risk of several types of cancer in people. Animal studies have also shown an increased risk of cancer from exposure to 2,3,7,8-TCDD.

The World Health Organization (WHO) has determined that 2,3,7,8-TCDD is a human carcinogen.

The Department of Health and Human Services (DHHS) has determined that 2,3,7,8-TCDD may reasonably be anticipated to cause cancer.

How can CDDs affect children?

Very few studies have looked at the effects of CDDs on children. Chloracne has been seen in children exposed to high levels of CDDs. We don't know if CDDs affect the ability of people to have children or if it causes birth defects, but given the effects observed in animal studies, this cannot be ruled out.

How can families reduce the risk of exposure to CDDs?

- Children should avoid playing in soils near uncontrolled hazardous waste sites.
- Discourage children from eating dirt or putting toys or other objects in their mouths.
- Everyone should wash hands frequently if playing or working near uncontrolled hazardous waste sites.
- For new mothers and young children, restrict eating foods from the proximity of uncontrolled sites with known CDDs.

Is there a medical test to show whether I've been exposed to CDDs?

Tests are available to measure CDD levels in body fat, blood, and breast milk, but these tests are not routinely available. Most people have low levels of CDDs in their body fat and blood, and levels considerably above these levels indicate past exposure to above-normal levels of 2,3,7,8-TCDD. Although CDDs stay in body fat for a long time, tests cannot be used to determine when exposure occurred.

Has the federal government made recommendations to protect human health?

The EPA has set a limit of 0.00003 micrograms of 2,3,7,8-TCDD per liter of drinking water (0.00003 µg/L). Discharges, spills, or accidental releases of 1 pound or more of 2,3,7,8-TCDD must be reported to EPA. The Food and Drug Administration (FDA) recommends against eating fish and shellfish with levels of 2,3,7,8-TCDD greater than 50 parts per trillion (50 ppt).

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1998. Toxicological profile for chlorinated dibenzo-p-dioxins. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

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This fact sheet answers the most frequently asked health questions (FAQs) about pentachlorophenol. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It is important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Pentachlorophenol is a manufactured chemical which is a restricted use pesticide and is used industrially as a wood preservative for utility poles, railroad ties, and wharf pilings. Exposure to high levels of pentachlorophenol can cause increases in body temperature, liver effects, damage to the immune system, reproductive effects, and developmental effects. This substance has been found in at least 313 of the 1,585 National Priorities List sites identified by the Environmental Protection Agency (EPA).

What is pentachlorophenol?

Pentachlorophenol is a manufactured chemical that does not occur naturally. Pure pentachlorophenol exists as colorless crystals. Impure pentachlorophenol (the form usually found at hazardous waste sites) is dark gray to brown and exists as dust, beads, or flakes. Humans are usually exposed to impure pentachlorophenol (also called technical grade pentachlorophenol).

Pentachlorophenol was widely used as a pesticide and wood preservative. Since 1984, the purchase and use of pentachlorophenol has been restricted to certified applicators. It is no longer available to the general public. It is still used industrially as a wood preservative for utility poles, railroad ties, and wharf pilings.

What happens to pentachlorophenol when it enters the environment?

- Pentachlorophenol can be found in the air, water, and soil. It enters the environment through evaporation from treated wood surfaces, industrial spills, and disposal at uncontrolled hazardous waste sites.
- Pentachlorophenol is broken down by sunlight, other chemicals, and microorganisms to other chemicals within a couple of days to months.
- Pentachlorophenol is found in fish and other foods, but tissue levels are usually low.

How might I be exposed to pentachlorophenol?

- The general populations can be exposed to very low levels of pentachlorophenol in contaminated indoor and outdoor air, food, drinking water and soil.
- People who work or live near a wood treatment facility or in the production of utility poles, railroad ties, or wharf pilings may be exposed to pentachlorophenol in the air or by coming in contact with the treated wood.
- People living near hazardous waste sites may also be exposed to higher than usual levels of pentachlorophenol.

How can pentachlorophenol affect my health?

Studies in workers show that exposure to high levels of pentachlorophenol can cause the cells in the body to produce excess heat. When this occurs, a person may experience a very high fever, profuse sweating, and difficulty breathing. The body temperature can increase to dangerous levels, causing injury to various organs and tissues, and even death. Liver effects and damage to the immune system have also been observed in humans exposed to high levels of pentachlorophenol for a long time. Damage to the thyroid and reproductive system has been observed in laboratory animals exposed to high doses of pentachlorophenol. Some of the harmful effects of pentachlorophenol are caused by the other chemicals present in technical grade pentachlorophenol.

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How likely is pentachlorophenol to cause cancer?

Some studies have found an increase in cancer risk in workers exposed to high levels of technical grade pentachlorophenol for a long time, but other studies have not found this. Increases in liver, adrenal gland, and nasal tumors have been found in laboratory animals exposed to high doses of pentachlorophenol.

The EPA has determined that pentachlorophenol is a probable human carcinogen and the International Agency for Cancer Research (IARC) considers it possibly carcinogenic to humans.

How can pentachlorophenol affect children?

Infants who were exposed to diapers and bedding which was accidentally contaminated with pentachlorophenol had high fevers, a large amount of sweating, difficulty breathing, and harmful effects on the nervous system and liver, and some died. Although these effects are similar to effects seen in adults exposed to pentachlorophenol, we do not know whether children and adults differ in their susceptibility to pentachlorophenol.

We do not know if exposure to pentachlorophenol will result in birth defects or other developmental effects in people. Death, low body weights, decreased growth, and skeletal effects have been observed in laboratory animals exposed to high levels of pentachlorophenol during development.

How can families reduce the risk of exposure to pentachlorophenol?

Pentachlorophenol was a widely used pesticide for a long time. Today its use is restricted and it can only be used by certified applicators. You may have old containers of pesticides in your attic, basement, or garage that contain pentachlorophenol. Removing these old containers will reduce your family's risk of exposure to pentachlorophenol.

If you live near utility poles and railroad tracks, you should prevent your children from playing, climbing, or sitting on

them especially in the hot summer months.

Though pentachlorophenol has been found in some food, its levels are low. You can minimize the risk of your family's exposure by peeling and thoroughly washing fruits and vegetables before cooking.

Children should avoid playing in soils near hazardous waste sites where pentachlorophenol may have been discarded.

Is there a medical test to show whether I've been exposed to pentachlorophenol?

Tests are available to measure pentachlorophenol and its breakdown product in blood, urine, and body tissues. These tests cannot be performed in the doctor's office because they require the use of special equipment. Because pentachlorophenol leaves the body fairly quickly, these tests are best for finding exposures that occurred within the last several days. These tests do not tell you how much pentachlorophenol you have been exposed to and cannot be used to predict the occurrence, nature, or severity of toxic effects.

Has the federal government made recommendations to protect human health?

The EPA has set a limit for drinking water of 1 part of pentachlorophenol per billion parts of water (1 ppb).

The Occupational Safety and Health Administration (OSHA) has set a limit of 0.5 milligrams of pentachlorophenol per cubic meter of workplace air (0.5 mg/m³) for 8 hour shifts and 40 hour work weeks.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2001. Toxicological Profile for Pentachlorophenol Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

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EXTOXNET

Extension Toxicology Network

Pesticide Information Profiles

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

EXTOXNET primary files maintained and archived at Oregon State University

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AMETRYN

TRADE OR OTHER NAMES: Product names include Evik, Ametryne, Ametrex, Gesapax (48), G34162, Trinatox-D (a combination with 2,4-D), Crisazina-Crisatrina Kombi (a combination with atrazine)(49), Doruplant, Mebatryne, and Amephyt (50).

REGULATORY STATUS: Ametryn is an unrestricted or General Use Pesticide (GUP). In Florida and Texas, ametryn may be applied alone on grapefruit and orange trees. In Florida, it may be applied with simazine for common bermuda grass and annual grasses and broadleaf weeds. In Hawaii, a mixture with diuron may be used on sugarcane (51).

INTRODUCTION: Ametryn, a member of the Triazine chemical family, is a herbicide which inhibits photosynthesis and other enzymatic processes. It is used to control broadleaf weeds and annual grasses in pineapple, sugarcane and bananas. It is used on corn and potato crops for general weed control (48). It is also used as a vine desiccant on dry beans and potatoes (50). Ametryn is available as an emulsifiable concentrate, flowable wettable powder and a wettable powder. Products containing ametryn should bear the SIGNAL WORD: CAUTION. The EPA classifies it as Toxicity Class III, slightly toxic (49).

TOXICOLOGICAL EFFECTS

- **Acute Toxicity:** Ametryn is slightly toxic to humans. Symptoms of acute exposure to high doses include nausea, vomiting, diarrhea, muscle weakness, and salivation (52). Ametryn is moderately irritating to the eyes, skin, and respiratory tract. The LD50 is the dose of ametryn which is lethal to half of the test animals that ingest it. The oral LD50 of ametryn is 508 mg/kg for rats and 945 mg/kg for mice (42). The LC50 for rats that inhale ametryn for four hours is greater than 2.2 mg/l of air (51). The dermal LD50 is greater than 3,100 mg/kg for rats and 8,160 mg/kg for rabbits (42). Acute eye exposure in rabbits causes a temporary irritation (56).
- **Chronic Toxicity:** No information is currently available
- **Reproductive Effects:** No information is currently available.
- **Teratogenic Effects:** No information is currently available.

- **Mutagenic Effects:** Studies have shown that ametryn is not mutagenic (54).
- **Carcinogenic Effects:** There is not adequate data to determine if ametryn can increase the risk of cancer in humans (48,55).
- **Organ Toxicity:** Animal studies indicate that consuming large amounts of ametryn over a long period of time results in liver damage (48).
- **Fate in Humans and Animals:** Excretion of ametryn is rapid. In rats, all but 2 to 7% is eliminated in the urine and feces within 72 hours (58).

ECOLOGICAL EFFECTS

- **Effects on Birds:** Ametryn is only slightly toxic to birds. The dietary LC50 (8 day) is 30,000 mg/kg for bobwhite quail and 23,000 mg/kg for mallard ducks (50).
- **Effects on Aquatic Organisms:** Ametryn is moderately toxic to fish. The LC50 for rainbow trout exposed for 96 hours is 8.8 mg/l. The LC50 for bluegill is 4.1 mg/l and for goldfish it is 14.1 mg/l (49, 50). Ametryn is highly toxic to crustaceans and moderately to highly toxic to mollusks (8).
- **Effects on Other Animals (Nontarget species):** Ametryn is only slightly toxic to bees (49).

ENVIRONMENTAL FATE

- **Breakdown in Soil & Groundwater:** Ametryn's half-life in soils, the amount of time it takes to degrade to half of the original concentration, is 70 to 250 days, depending on the soil type and weather conditions. Loss from the soil is principally by microbial degradation (48, 50). Ametryn moves both vertically and laterally in soil due to its high water solubility (57). Because it is persistent, it may leach as a result of high rainfall, floods, and furrow irrigation (48). In a study of surface and groundwater contaminants in the U.S, ametryn was found in six states, in very few surface water samples and in 4% of the groundwater samples. The maximum concentration found was 0.1 micrograms/l in surface water and 450 micrograms/l in groundwater(54).
- **Breakdown in Vegetation:** Ametryn is broken down into non-toxic substances by tolerant plants and, to a lesser extent, by sensitive plants (50).

PHYSICAL PROPERTIES AND GUIDELINES:

Physical Properties:

- **Appearance:** White Powder
- **Chemical Name:** 2-(ethylamino)-4-isopropylamino-6-methyl-thio-s-triazine
- **CAS Number:** 834-12-8
- **Molecular Weight:** 227.3
- **Water Solubility:** 185 mg/l at 20 degrees C. It dissolves readily in organic solvents including hexane, toluene, methanol, and acetone (50, 54).
- **Solubility in Other Solvents:** Soluble in acetone (610), methanol(510), toluene (470), n-octanol (220), hexane (58)
- **Melting Point:** 84-85 degrees C (50)
- **Vapor Pressure:** 0.365 mPa
- **Partition Coefficient:** 676 (50).
- **Adsorption Coefficient:** Not Available

Exposure Guidelines:

- **HA:** 0.06 mg/l (lifetime); 8.6 mg/l (child) (57).
- **DWEL:** 0.3 mg/l (57).
- **LOEL:** 100mg/kg/day (53)
- **NOAEL:** 10 mg/kg/day (57)
- **RfD:** 0.0086 mg/kg/day (53, 57)
- **ADI:** Not Available
- **PEL:** Not Available
- **TLV:** Not Available
- **MCL:** Not Available

BASIC MANUFACTURER:

CIBA

Agricultural Division

P.O. Box 18300

Greensboro, NC 27419-8300

Telephone: 919-632-6000

REFERENCES

References for the information in this PIP can be found in Reference List [Number 8](#)

DISCLAIMER: The information in this profile does not in any way replace or supersede the information on the pesticide product label/ing or other regulatory requirements. Please refer to the pesticide product label/ing.

EXTOXNET**Extension Toxicology Network****Pesticide Information Profiles**

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

EXTOXNET primary files maintained and archived at Oregon State University

Revised June 1996

Atrazine

Trade and Other Names: Trade names include Aatrex, Aktikon, Alazine, Atred, Atranex, Atrataf, Atratol, Azinotox, Crisazina, Farmco Atrazine, G-30027, Gesaprim, Giffex 4L, Malermais, Primatol, Simazat, and Zeapos.

Regulatory Status: Atrazine has been classified as a Restricted Use Pesticide (RUP) due to its potential for groundwater contamination [2]. RUPs may be purchased and used only by certified applicators. Atrazine is toxicity class III - slightly toxic. In November, 1994, the EPA initiated a Special Review which could result in use restrictions or cancellation of atrazine if health data warrant such action. Products containing atrazine must the Signal Word CAUTION.

Chemical Class: triazine

Introduction: Atrazine is a selective triazine herbicide used to control broadleaf and grassy weeds in corn, sorghum, sugarcane, pineapple, christmas trees, and other crops, and in conifer reforestation plantings. It is also used as a nonselective herbicide on non-cropped industrial lands and on fallow lands. Over 64 million acres of cropland were treated with atrazine in the U.S. in 1990. It is available as dry flowable, flowable liquid, liquid, water dispersible granular, and wettable powder formulations.

Formulation: It is available as dry flowable, flowable liquid, liquid, water dispersible granular, and wettable powder formulations.

Toxicological Effects:

- **Acute toxicity:** Atrazine is slightly to moderately toxic to humans and other animals. It can be absorbed orally, dermally, and by inhalation. Symptoms of poisoning include abdominal pain, diarrhea and vomiting, eye irritation, irritation of mucous membranes, and skin reactions [3]. At very high doses, rats show excitation followed by depression, slowed breathing, incoordination, muscle spasms, and hypothermia [3]. After consuming a large oral dose, rats exhibit muscular weakness, hypoactivity, breathing difficulty,

prostration, convulsions, and death [16]. Atrazine is a mild skin irritant. Rashes associated with exposure have been reported. The oral LD50 for atrazine is 3090 mg/kg in rats, 1750 mg/kg in mice, 750 mg/kg in rabbits, and 1000 mg/kg in hamsters. The dermal LD50 in rabbits is 7500 mg/kg and greater than 3000 mg/kg in rats [15,16]. The 1-hour inhalation LC50 is greater than 0.7 mg/L in rats. The 4-hour inhalation LC50 is 5.2 mg/L in rats [3,6].

- **Chronic toxicity:** Some 40% of rats receiving oral doses of 20 mg/kg/day for 6 months died with signs of respiratory distress and paralysis of the limbs. Structural and chemical changes in the brain, heart, liver, lungs, kidney, ovaries, and endocrine organs were observed [3,16]. Rats fed 5 or 25 mg/kg/day of atrazine for 6 months exhibited growth retardation. In a 2-year study with dogs, 7.5 mg/kg/day caused decreased food intake and increased heart and liver weights. At 75 mg/kg/day, there were decreases in food intake and body weight gain, increased adrenal weight, lowered blood cell counts, and occasional tremors or stiffness in the rear limbs [3].
- **Reproductive effects:** Dietary doses of atrazine given to rats on days 3, 6 and 9 of gestation up to about 50 mg/kg/day caused no adverse reproductive effects [3].
- **Teratogenic effects:** Atrazine does not appear to be teratogenic. In mice, atrazine did not cause abnormalities in fetuses whose dams were given doses of 46.4 mg/kg/day during days 6 through 14 of gestation [3].
- **Mutagenic effects:** The weight of evidence from more than 50 studies indicates that atrazine is not mutagenic [3].
- **Carcinogenic effects:** Atrazine did not cause tumors when mice were given oral doses of 21.5 mg/kg/day from age 1 to 4 weeks, followed by dietary doses of 82 mg/kg for an additional 17 months. However, mammary tumors were observed in rats after lifetime administration of high doses of atrazine [3]. Thus, available data regarding atrazine's carcinogenic potential are inconclusive.
- **Organ toxicity:** Lethal doses of atrazine in test animals have caused congestion and/or hemorrhaging to the lungs, kidneys, liver, spleen, brain, and heart [3]. Long-term consumption of high levels of atrazine has caused tremors, changes in organ weights, and damage to the liver and heart [3].
- **Fate in humans and animals:** Atrazine is readily absorbed through the gastrointestinal tract. When a single dose of 0.53 mg atrazine was administered to rats by gavage, 20% of the dose was excreted in the feces within 72 hours. The other 80% was absorbed across the lining of the gastrointestinal tract into the bloodstream. After 72 hours, 65% was eliminated in the urine and 15% was retained in body tissues, mainly in the liver, kidneys, and lungs [3].

Ecological Effects:

- **Effects on birds:** Atrazine is practically nontoxic to birds. The LD50 is greater than 2000 mg/kg in mallard ducks. At dietary doses of 5000 ppm, no effect was observed in bobwhite quail and ring-necked pheasants [15,16].
- **Effects on aquatic organisms:** Atrazine is slightly toxic to fish and other aquatic life. Atrazine has a low level of bioaccumulation in fish. In whitefish, atrazine accumulates in the brain, gall bladder, liver, and gut [16].
- **Effects on other organisms:** Atrazine is not toxic to bees [16].

Environmental Fate:

- **Breakdown in soil and groundwater:** Atrazine is highly persistent in soil. Chemical hydrolysis, followed

by degradation by soil microorganisms, accounts for most of the breakdown of atrazine. Hydrolysis is rapid in acidic or basic environments, but is slower at neutral pHs. Addition of organic material increases the rate of hydrolysis. Atrazine can persist for longer than 1 year under dry or cold conditions [21]. Atrazine is moderately to highly mobile in soils with low clay or organic matter content. Because it does not adsorb strongly to soil particles and has a lengthy half-life (60 to >100 days), it has a high potential for groundwater contamination despite its moderate solubility in water [20]. Atrazine is the second most common pesticide found in private wells and in community wells [16]. Trace amounts have been found in drinking water samples and in groundwater samples in a number of states [23,21]. A 5-year survey of drinking water wells detected atrazine in an estimated 1.7% of community water systems and 0.7% of rural domestic wells nationwide. Levels detected in rural domestic wells sometimes exceeded the MCL [23]. The recently completed National Survey of Pesticides in Drinking Water found atrazine in nearly 1% of all of the wells tested [23].

- **Breakdown in water:** Atrazine is moderately soluble in water. Chemical hydrolysis, followed by biodegradation, may be the most important route of disappearance from aquatic environments. Hydrolysis is rapid under acidic or basic conditions, but is slower at neutral pHs. Atrazine is not expected to strongly adsorb to sediments. Bioconcentration and volatilization of atrazine are not environmentally important [21]. Atrazine has been detected in each of 146 water samples collected at 8 locations from the Mississippi, Ohio and Missouri Rivers and their tributaries. For several weeks, 27% of these samples contained atrazine concentrations above the EPA's maximum contaminant level (MCL) [24].
- **Breakdown in vegetation:** Atrazine is absorbed by plants mainly through the roots, but also through the foliage. Once absorbed, it is translocated upward and accumulates in the growing tips and the new leaves of the plant. In susceptible plant species, atrazine inhibits photosynthesis. In tolerant plants, it is metabolized [6]. Most crops can be planted 1 year after application of atrazine. Atrazine increases the uptake of arsenic by treated plants [16].

Physical Properties:

- **Appearance:** Atrazine is a white, crystalline solid [6].
- **Chemical Name:** 2-chloro-4-ethylamine-6-isopropylamino-S-triazine [6]
- **CAS Number:** 1912-24-9
- **Molecular Weight:** 215.69
- **Water Solubility:** 28 mg/L @ 20 C [6]
- **Solubility in Other Solvents:** chloroform v.s.; diethyl ether v.s.; dimethyl sulfoxide v.s. [6]
- **Melting Point:** 176 C [6]
- **Vapor Pressure:** 0.04 mPa @ 20 C [6]
- **Partition Coefficient:** 2.3404 [6]
- **Adsorption Coefficient:** 100 [20]

Exposure Guidelines:

- **ADI:** Not Available
- **MCL:** 0.003 mg/L [25]
- **RfD:** 0.035 mg/kg/day [26]
- **PEL:** Not Available
- **HA:** Not Available
- **TLV:** 5 mg/m³ (8-hour) [16]

Basic Manufacturer:

Ciba-Geigy Corp.
P.O. Box 18300
Greensboro, NC 27419-8300

- **Phone:** 800-334-9481
- **Emergency:** 800-888-8372

References:

References for the information in this PIP can be found in Reference List [Number 8](#)

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EXTOXNET

Extension Toxicology Network

Pesticide Information Profiles

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EXTOXNET primary files maintained and archived at Oregon State University

Revised June 1996

Pentachlorophenol (PCP)

Trade and Other Names: Trade names for pentachlorophenol include Dowicide, PCP, Penchlorol, Penta, Penta Plus, Pentachloral, Pentacon, Penwar, Priltox, Santobrite, Santophen, Sinituho, and Weedone.

Regulatory Status: Pentachlorophenol is a moderately toxic compound in EPA toxicity class II. It is a Restricted Use Pesticide (RUP) in its formulations as a wood preservative, but a General Use Pesticide (GUP) for other purposes; labels for products containing it must bear the Signal Word DANGER.

Chemical Class: chlorinated hydrocarbon

Introduction: Pentachlorophenol (PCP) is a chlorinated hydrocarbon insecticide and fungicide. It is primarily used to protect timber from fungal rot and wood-boring insects, but may also be used as a preharvest defoliant in cotton, a general pre-emergence herbicide, and as a biocide in industrial water systems. It is available in blocks, flakes, granules, liquid concentrates, wettable powders, or ready-to-use petroleum solutions.

Data presented in this profile are for technical grade pentachlorophenol, unless otherwise stated. Technical grade PCP has historically contained dioxins (e.g. tetra-, hexa- and octochlorodibenzo-p-dioxin) and hexachlorobenzene as manufacturing by-products. Technical grade PCP is typically about 86% pure. The discovery of these compounds in technical grade PCP may be one reason for its being phased out of use. Pentachlorophenol is also a major product of the metabolism of hexachlorobenzene in mammals.

Formulation: It is available in blocks, flakes, granules, liquid concentrates, wettable powders, or ready-to-use petroleum solutions.

Toxicological Effects:

- **Acute toxicity:** Pentachlorophenol is moderately toxic via the oral route, with reported oral LD50 values for various formulations ranging from 27 to 211 mg/kg in rats [69,70]. In mice the oral LD50 is 74 to 130 mg/kg, and in rabbits 70 to 300 mg/kg [69,70]. It is moderately toxic via inhalation as well, with a

reported inhalation LC50 of 0.2 to 2.1 mg/L in rats [17]. The time frame for this LC50 (e.g., 4-hour, 1-hour, etc.) was not given. Inhalation LD50 values (i.e., the median lethal doses, not concentrations, via the inhalation route) of 225 mg/kg in rats and 355 mg/kg in mice are reported, also without a time frame [70]. Another calculated LD50 in rats via the inhalation route is 11.7 mg/kg for 28 to 44 minutes of exposure, assuming a breathing rate of 80 mL/minute [69]. Pentachlorophenol causes irritation to the mucous membranes, skin, and eyes of test animals [9]. Via the dermal route, it is moderately toxic, with reported dermal LD50 values ranging from 96-330 mg/kg in the rat, and 40 to greater than 1000 mg/kg in the rabbit (depending on formulation) [69]. Skin penetration may be the most dangerous route of exposure, being responsible for about 50 known cases of PCP poisoning, 30 of which have resulted in death. Immersion of a human hand in a 0.4 percent PCP solution for 10 minutes caused pain and inflammation. Technical PCP resulted in chloracne on the ears of rabbits, and edema in chicks, but pure PCP did not [70]. High acute exposure to PCP can cause elevated temperature, profuse sweating, dehydration, loss of appetite, decreased body weight, nausea, and neurological effects such as tremors, uncoordinated movement, leg pain, muscle twitching, and coma [69,70]. Some of the symptoms may be due to the impurities in the formulation, rather than the pentachlorophenol itself [69].

- **Chronic toxicity:** Much research on PCP has been performed with poorly characterized technical material, and the chronic toxicity observed may depend in large measure on the proportion of chlorodibenzo-p-dioxins present in the mixture [69]. In a 90-day feeding trial in rats, 30 mg/kg/day produced depressed red blood cell and hemoglobin levels as well as liver degeneration, and even lower doses resulted in irregular blood chemistry and enzyme levels, along with increased liver and kidney weights [69,71]. Pure PCP, and also technical PCP without dioxin contamination, produced only slight enlargement of livers and kidneys [69]. Purified PCP also did not produce toxic effects such as liver damage and immune system alterations, which had previously been reported for the technical product [69,71]. In humans, the most common exposure to PCP is inhalation in the workplace. Abdominal pain, nausea, fever, and respiratory irritation, as well as eye, skin, and throat irritation, may result from such exposure [70], while very high levels may cause obstruction of the circulatory system in the lungs and cause heart failure [70]. Survivors of toxic exposures may suffer permanent visual and central nervous system damage [70]. Persons regularly exposed to PCP tend to tolerate higher levels of PCP vapors than persons having little contact with these vapors [70,71].
- **Reproductive effects:** Rats fed PCP at doses of 30 mg/kg/day for 62 days before mating and during lactation showed weight loss, but no decreases in fecundity and fertility [69]. Sperm of male mice given technical or purified PCP for 5 days at 50 mg/kg/day showed no abnormalities within 35 days of treatment [69]. The evidence indicates that PCP does not cause reproductive effects.
- **Teratogenic effects:** Offspring of rats fed PCP at doses of 30 mg/kg/day for 62 days before mating and during lactation showed lowered survival and growth rates [69]; 3 mg/kg/day did not have any effects [69]. Maternal doses of 5 mg/kg/day of technical PCP in rats produced toxicity to the fetus or embryo, and 50 mg/kg/day on days 6 to 15, 8 to 11 or 12 to 15 of gestation produced increases resorptions, swelling, dilated ureters, and skeletal anomalies [69]. It is unlikely that PCP has teratogenic effects in humans at normal exposure levels.
- **Mutagenic effects:** PCP is not mutagenic in bacteria or houseflies, but is weakly mutagenic in mice and may be mutagenic in yeast [71]. One study of chromosomal aberrations in occupationally exposed workers showed no increased incidence of sister-chromatid exchanges, while another did find increases [71]. Weak mutagenic effects were seen in human lymphocyte cultures exposed to PCP [71]. The evidence suggests that PCP is nonmutagenic or weakly mutagenic.
- **Carcinogenic effects:** Studies of two formulated PCP products (Dowcide and Penta) showed increases in cancers of the spleen, liver, and adrenal gland in test mice or rats at doses of about 17 to 18 mg/kg/day

[71]. These findings were not replicated for Dowcide in mice in a second study [71]. There have been reports of a possible association between occupational exposures to technical PCP and Hodgkin's disease, acute leukemia, and soft-tissue sarcoma, but confounding factors such as concurrent exposure to other substances makes interpretation of these data problematic [71]. No convincing evidence of PCP's carcinogenic effects in humans is available [71]. Current evidence is not sufficient to assess the potential of PCP to cause carcinogenic effects in humans.

- **Organ toxicity:** Data from animal studies indicate that the major target organs for PCP are the liver, kidneys, and central nervous system.
- **Fate in humans and animals:** PCP is rapidly absorbed through the gastrointestinal tract following ingestion [71]. Accumulation is not common, but if it does occur, the major sites are the liver, kidneys, plasma protein, brain, spleen, and fat [69,71]. Unless kidney and liver functions are impaired, PCP is rapidly eliminated from blood and tissues, and is excreted, mainly unchanged or in conjugated form, via the urine [71]. Single doses of PCP have half-lives in blood of 15 hours in rats, 78 hours in monkeys, and 30 to 50 hours in humans [69].

Ecological Effects:

- **Effects on birds:** The compound is slightly toxic to practically nontoxic to bird species. The reported 5-day dietary LC50 value in Japanese quail is greater than 5139 ppm [54]. Reported acute oral LD50 values for PCP are 380 mg/kg in mallard duck and 504 mg/kg in pheasant [55].
- **Effects on aquatic organisms:** PCP may be highly to very highly toxic to many species of fish; reported 96-hour LC50 values are 68 ug/L in chinook salmon, 52 ug/L in rainbow trout, 205 ug/L in fathead minnow, 68 ug/L in channel catfish, and 32 ug/L in bluegill sunfish [55]. Several species of fish, invertebrates, and algae have had levels of PCP that were significantly higher (up to 10,000 times) than the concentration in the surrounding waters [71]. Once absorbed by fish, pure PCP is rapidly excreted as is its metabolite, with a biological half-life of only 10 hours [71]. Biomagnification, that is the progressively higher concentration of a compound as it passes up the food chain, is not thought to be significant because of PCP's rapid break down in living organisms [70].
- **Effects on other organisms:** Cattle and other farm animals have ingested PCP by chewing and licking outdoor wood structures, or from being housed in wooden pens that were treated with PCP solutions. This has caused sickness and death in some of these animals [17].

Environmental Fate:

- **Breakdown in soil and groundwater:** PCP is moderately persistent in the soil environment, with a reported field half-life of 45 days [15]. PCP degrades most rapidly in flooded or anaerobic (airless) soils, at higher temperatures and in the presence of organic matter in the soil [12,15]. Breakdown is mainly by anaerobic biodegradation; breakdown by sunlight and hydrolysis do not appear to be significant processes [15]. It is poorly sorbed at neutral and alkaline conditions, and may be mobile in many soils [12,15]. Sorption will be slightly greater (and mobility slightly lesser) in soils with higher proportions of soil organic matter [12]. The compound has been found in groundwater in California, Oregon, and Minnesota at very low concentrations ranging from 0.06 ppt to 0.64 ppb [15].
- **Breakdown in water:** In the water environment, PCP is mainly bound to sediments and suspended particles in water [12]. PCP will dissociate by releasing a hydrogen ion and may then be more readily degraded by sunlight or microorganisms [12]. In water, biodegradation occurs, mainly at the surface, with

a half-life ranging from hours to days [12]. It does not evaporate to a significant degree. PCP has been detected at very low levels in rivers and streams (0.01 to 16 ug/L), surface water systems (1.3 to 12 ug/L), and seawater (0.02 to 11 ug/L) [12].

- **Breakdown in vegetation:** PCP may be taken up by plants; lettuce grown on soil containing PCP contained low levels of PCP residues [12]. Uptake and accumulation varies according to plant species. PCP is strongly toxic to plants [9].

Physical Properties:

- **Appearance:** At room temperature, pentachlorophenol is a colorless crystalline solid with a phenolic odor [9]. Color may vary from white to dark grayish brown, depending on the purity of the compound [9].
- **Chemical Name:** pentachlorophenol [9]
- **CAS Number:** 87-86-5
- **Molecular Weight:** 266.34
- **Water Solubility:** 80 mg/L @ 20 C [9]
- **Solubility in Other Solvents:** v.s. in acetone, alcohols, ether, and benzene; s. in petroleum ether, carbon tetrachloride, and paraffins [9]
- **Melting Point:** 191 C [9]
- **Vapor Pressure:** 16,000 mPa @ 20 C [9]
- **Partition Coefficient:** 5.12 [17]
- **Adsorption Coefficient:** 30 (at pH 7) (estimated) [15]

Exposure Guidelines:

- **ADI:** Not Available
- **MCL:** 0.001 mg/L [67]
- **RfD:** 0.03 mg/kg/day [8]
- **PEL:** 0.5 mg/m³ (8-hour) [28]
- **HA:** Not Available
- **TLV:** Not Available

Basic Manufacturer:

ISK Biosciences
5966 Heisley Road
P.O. Box 8000
Mentor, OH 44061-8000

- **Phone:** 216-357-4100
- **Emergency:** 216-357-7070

References:

References for the information in this PIP can be found in Reference List [Number 6](#)

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Simazine

Trade and Other Names: Trade names include Aquazine, Caliber, Cekusan, Cekusima, Framed, Gesatop, Primatol S, Princep, Simadex, Simanex, Sim-Trol, Tanzine and Totazine. This compound may also be found in formulations with other herbicides such as amitrole, paraquat dichloride, metolachlor, and atrazine.

Regulatory Status: Simazine is a General Use Pesticide (GUP). It is in EPA toxicity class IV - practically nontoxic. Products containing simazine bear the Signal Word CAUTION. In November 1994, the U.S. EPA began a Special Review of simazine which could result in use restrictions or even cancellation if data warrant such action.

Chemical Class: Triazine

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

Formulation: Simazine is available in wettable powder, water dispersible granule, liquid and granular formulations. It may be soil-applied.

Toxicological Effects:

- **Acute toxicity:** Simazine is slightly to practically nontoxic. The reported oral LD50 for technical simazine in rats and mice is >5000 mg/kg [6,15]; its dermal LD50 is 3100 mg/kg in rats and > 10,000 mg/kg in rabbits [6,15]. The 4-hour inhalation LC50 in rats is greater than 2 mg/L (6). The formulated products, in most cases, are less toxic via all routes [15]. Simazine is nonirritating to the skin and eyes of rabbits except at high doses [3]. Patch tests on humans have shown that simazine is not a skin irritant, fatiguing agent, or

sensitizer [3]. However, rashes and dermatitis from occupational exposure to simazine have occurred [3]. The triazine herbicides disturb energy metabolism (thiamin and riboflavin functions). Symptoms include difficulty in walking, tremor, convulsions, paralysis, cyanosis, slowed respiration, miosis (pinpoint pupils), gut pain, diarrhea, and impaired adrenal function [3]. No cases of poisoning in humans have been reported from ingestion of simazine [3]. Rats given an oral dose of 5000 mg/kg exhibited drowsiness and irregular breathing. In another study, a single oral dose of 4200 mg/kg produced anorexia, weight loss, and some deaths in rats within 4 to 10 days [26]. For unknown reasons, sheep and cattle are especially susceptible to poisoning by simazine. Doses of 500 mg/kg were fatal in sheep with death delayed for 5 to 16 days. Symptoms exhibited by poisoned sheep included lower food intake, higher water intake, incoordination, tremors, and weakness, especially in the hindquarters [3].

- **Chronic toxicity:** Some 90-day feeding studies showed reduced body weight at 67 to 100 mg/kg/day [10]. This same effect and kidney toxicity were seen in rats at doses of 150 mg/kg/day [10]. In 2-year chronic oral feeding studies in which rats were given daily dosages of 5 mg/kg/day of simazine in the diet, no gross or microscopic signs of toxicity were seen [3]. When rats were given repeated doses of 15 mg/kg/day, some liver cells degenerated during the first 3 days, but the condition did not progress. Instead, the liver adapted and the compound was metabolized [3]. Other effects observed in test animals include tremors, damage to the testes, kidneys, liver, and thyroid, disturbances in sperm production, and gene mutations [10].
- **Reproductive effects:** No adverse effects on reproductive capacity or development were observed in a three-generation study of rats fed 5 mg/kg/day simazine [10]. High rates of fetotoxicity and decreased birth weight were noted in the fetuses of pregnant rabbits fed 75 mg/kg/day [26]. Reproductive effects are not likely in humans under normal circumstances.
- **Teratogenic effects:** No dose-related teratogenic effects were observed when rabbits were given daily doses of 5, 75, or 200 mg/kg for days 7 through 19 of pregnancy [26]. Chronic inhalation of a cumulative dose of 0.3 mg/L for 8 days in pregnant rats resulted in no treatment-related developmental abnormalities [10]. Simazine does not appear to be teratogenic.
- **Mutagenic effects:** Simazine has shown negative results in a variety of mutagenicity tests on bacterial cultures [10]. Tests on human lung cell cultures have produced both positive and negative results [10]. When injected into adult male fruitflies, simazine increased the frequency of sex-linked lethal mutations, but failed to do so when fed to larvae. Other tests for mutagenicity in fruitflies were negative [3]. It is likely that simazine is either nonmutagenic or weakly mutagenic.
- **Carcinogenic effects:** Simazine was not tumorigenic in mice at the maximum tolerated dose of 215 mg/kg/day over an 18-month period [10]. In other studies, doses as low as 5 mg/kg/day produced excess tumors (thyroid and mammary) in female rats [3,10]. Because of inconsistencies in the data, it is not possible to determine simazine's carcinogenic status.
- **Organ toxicity:** Damage to the testes, kidneys, liver, and thyroid has been observed in test animals [3,10].
- **Fate in humans and animals:** Studies in rats, goats, and sheep reveal that 60 to 70% of the ingested dose may be absorbed into the system [10], with approximately 5 to 10% distributed systemically to tissues. The remainder is eliminated via urine within 24 hours [6]. Distribution led to detectable levels in red blood cells (highest), liver, kidney, fat, bone, and plasma [10]. When a cow was fed 5 ppm for 3 days, no simazine was found in the cow's milk during the next 3 days. It has been reported that simazine residues were present in the urine of sheep for up to 12 days after administration of a single oral dose. The maximum concentration in the urine occurred from 2 to 6 days after administration [16].

Ecological Effects:

- **Effects on birds:** Simazine is practically nontoxic to birds [6,16]. The reported LD50 values in mallard and Japanese quail are >4600 mg/kg and 1785 mg/kg, respectively [6]. The acute dietary LD50 values in hens and pigeons are both greater than 5000 ppm [2]. The 8-day dietary LC50 in bobwhite quail is >5260 ppm and in mallard ducks is >10,000 ppm [6,15].
- **Effects on aquatic organisms:** Simazine is slightly to practically nontoxic to aquatic species [6,15]. The 96-hour LC50 for simazine is >100 mg/L [46] in rainbow trout, 100 mg/L (wetable powder) in bluegill sunfish, 0.100 mg/L in fathead minnows [46], as well as carp [2]. It may be more toxic to Daphia and stoneflies [46]. A 96-hour LC50 of >3.7 mg/L is reported in oysters [15].
- **Effects on other organisms:** While many mammals may be insensitive to simazine [16], sheep and cattle are especially sensitive [3]. Simazine is nontoxic to bees [6,16]. A soil LC50 in earthworms of >1000 mg/kg has been reported [16].

Environmental Fate:

- **Breakdown in soil and groundwater:** Simazine is moderately persistent with an average field half-life of 60 days [20]. Soil half-lives of 28-149 days have been reported [20]. Residual activity may remain for a year after application (2 to 4 kg/ha) in high pH soils. Simazine is moderately to poorly bound to soils [20]. It does, however, adsorb to clays and mucks. Its low water solubility, however, makes it less mobile, limiting its leaching potential [15]. Simazine has little, if any, lateral movement in soil, but can be washed along with soil particles in runoff. Simazine is subject to decomposition by ultraviolet radiation, but this effect is small under normal field conditions. Loss from volatilization is also insignificant. In soils, microbial activity probably accounts for decomposition of a significant amount of simazine in high pH soils. In lower pH soils, hydrolysis will occur [15]. Simazine residues have been detected in groundwater in at least 16 states. The range was from 0.00002 mg/L to 0.0034 mg/L [23].
- **Breakdown in water:** The average half-life of simazine in ponds where it has been applied is 30 days, with the actual half-life dependent on the level of algae present, the degree of weed infestation, and other factors [15]. Simazine may undergo hydrolysis at lower pH. It does not readily undergo hydrolysis in water at pH = 7 [15].
- **Breakdown in vegetation:** Plants absorb simazine mainly through the roots, with little or no foliar penetration. From the roots, it is translocated upward to the stems, leaves, and growing shoots of the plant [6,15]. It acts to inhibit photosynthesis [6,15]. Resistant plants readily metabolize simazine. Plants that are sensitive to simazine accumulate it unchanged [6]. It is possible that livestock or wildlife grazing on these plants could be poisoned.

Physical Properties:

- **Appearance:** Simazine is a white or colorless crystalline solid [6].
- **Chemical Name:** 6-chloro-N2,N4-diethyl-1,3,5-triazine-2,4-diamine [6]
- **CAS Number:** 122-34-9
- **Molecular Weight:** 201.70
- **Water Solubility:** 5 mg/L @ 20 C [6]
- **Solubility in Other Solvents:** s. in methanol, chloroform, and diethyl ether [6]; s.s. in pentane [6]
- **Melting Point:** 225-227 C [6]
- **Vapor Pressure:** 0.000810 mPa @ 20 C [6]

- **Partition Coefficient:** 1.9600 [6]
- **Adsorption Coefficient:** 130 [20]

Exposure Guidelines:

- **ADI:** Not Available
- **MCL:** 0.004 mg/L [25]
- **RfD:** 0.005 mg/kg/day [26]
- **PEL:** Not Available
- **HA:** Not Available
- **TLV:** Not Available

Basic Manufacturer:

Ciba-Geigy Corporation
P.O. Box 18300
Greensboro, NC 27419-8300

- **Phone:** 800-334-9481
- **Emergency:** 800-888-8372

References:

References for the information in this PIP can be found in Reference List [Number 8](#)

DISCLAIMER: The information in this profile does not in any way replace or supersede the information on the pesticide product labeling or other regulatory requirements. Please refer to the pesticide product labeling.

EXTOXNET

Extension Toxicology Network

Pesticide Information Profiles

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

EXTOXNET primary files maintained and archived at Oregon State University

Revised June 1996

Trifluralin

Trade and Other Names: Trade names include Crisalin, Elancolan, Flurene SE, Ipersan, L-36352, M.T.F., Su Seguro Carpidor, TR-10, Trefanocide, Treficon, Treflan, Tri-4, Trifluralina 600, Triflurex Trim, and Trust. The compound may be found in formulations with other herbicides.

Regulatory Status: Products containing trifluralin bear the Signal Words CAUTION or WARNING, depending on the type of formulation. This compound is a General Use Pesticide (GUP) in toxicity class III - slightly toxic. N-nitrosamine contaminant levels in trifluralin are required to be below 0.5 ppm, a level which EPA believes will result in no toxic effects.

Chemical Class: dinitroaniline compound

Introduction: Trifluralin is a selective, pre-emergence dinitroaniline herbicide used to control many annual grasses and broadleaf weeds in a large variety of tree fruit, nut, vegetable, and grain crops, including soybeans, sunflowers, cotton, and alfalfa. Pre-emergence herbicides are applied before weed seedlings sprout. Trifluralin should be incorporated into the soil by mechanical means within 24 hours of application. Granular formulations may be incorporated by overhead irrigation. Trifluralin is available in granular and emulsifiable concentrate formulations. The technical material is approximately 96% pure and the emulsifiable concentrate is about 45% pure.

Formulation: Granular formulations may be incorporated by overhead irrigation. Trifluralin is available in granular and emulsifiable concentrate formulations. The technical material is approximately 96% pure and the emulsifiable concentrate is about 45% pure.

Toxicological Effects:

- **Acute toxicity:** Pure trifluralin is practically nontoxic to test animals by oral, dermal, or inhalation routes of exposure [138]. The oral LD50 for technical trifluralin in rats is greater than 10,000 mg/kg, in mice is greater than 5000 mg/kg, and in dogs, rabbits, and chickens, is greater than 2000 mg/kg. However,

certain formulated products that contain trifluralin may be more toxic than the technical material itself. For example, the oral LD50 for Treflan TR-10 in rats is greater than 500 mg/kg. The dermal LD50 for technical trifluralin in rabbits is greater than 2000 mg/kg. The 1-hour inhalation LC50 for technical trifluralin in rats is greater than 2.8 mg/L [58]. Nausea and severe gastrointestinal discomfort may occur after eating trifluralin. Trifluralin does not cause skin irritation. When applied to the eyes of rabbits, trifluralin produced slight irritation, which cleared within 7 days [8]. Skin sensitization (allergies) may occur in some individuals [8]. Inhalation may cause irritation of the lining of the mouth, throat, or lungs [8].

- **Chronic toxicity:** Prolonged or repeated skin contact with trifluralin may cause allergic dermatitis [8]. The administration of 25 mg/kg/day to dogs for 2 years resulted in no observed toxicity [58]. In another study of beagle dogs, toxic effects were observed at 18.75 mg/kg/day. These included decreased red blood cell counts and increases in methemoglobin, total serum lipids, triglycerides, and cholesterol [13]. Trifluralin has been shown to cause liver and kidney damage in other studies of chronic oral exposure in animals [139].
- **Reproductive effects:** The reproductive capacity of rats fed dietary concentrations of trifluralin as high as 10 mg/kg/day was unimpaired through four successive generations. Trifluralin administered to pregnant rabbits at doses as high as 100 mg/kg/day, and to rats at doses as high as 225 mg/kg/day, produced no adverse effect on either the mothers or offspring [58]. Loss of appetite and weight loss followed by miscarriages were observed when pregnant rabbits were fed high doses of 224 or 500 mg/kg/day. Fetal weight decreased and there was an increase in the number of fetal runts at the 500 mg/kg/day dosage [8]. It is unlikely effects on reproduction will be produced in humans at expected exposure levels.
- **Teratogenic effects:** No abnormalities were observed the offspring of rats fed doses as high as 10 mg/kg/day for four generations [58]. Studies in the rat and rabbit show no evidence that trifluralin is teratogenic. The highest doses tested in these studies were 1000 mg/kg/day in rats and 500 mg/kg/day in rabbits [138]. Trifluralin does not appear to be teratogenic.
- **Mutagenic effects:** No evidence of mutagenicity was observed when trifluralin was tested in live animals, and in assays using bacterial and mammalian cell cultures [138].
- **Carcinogenic effects:** In a 2-year study of rats fed 325 mg/kg/day, the highest dose tested, malignant tumors developed in the kidneys, bladder, and thyroid [138]. However, more data are needed to characterize its carcinogenicity.
- **Organ toxicity:** Liver, kidney, and thyroid damage appear to be the main toxic effects in chronic animal studies [139].
- **Fate in humans and animals:** Trifluralin is not readily absorbed into the bloodstream from the gastrointestinal tract; 80% of single oral doses administered to rats and dogs was excreted in the feces [8].

Ecological Effects:

- **Effects on birds:** Trifluralin is practically nontoxic to birds [63]. The LD50 in bobwhite quail is greater than 2000 mg/kg, as it is in female mallards and pheasants [63]. These values are for the technical product.
- **Effects on aquatic organisms:** Trifluralin is very highly toxic to fish and other aquatic organisms. The 96-hour LC50 is 0.02 to 0.06 mg/L in rainbow trout, and 0.05 to 0.07 mg/L in bluegill sunfish [37]. The 96-hour LC50 in channel catfish is approximately 1.4 to 3.4 mg/L [37]. Variables such as temperature, pH, life stage, or size may affect the toxicity of the compound. Trifluralin is highly toxic to *Daphnia*, a species of small freshwater crustacean, with a 48-hour LC50 of 0.5 to 0.6 mg/L [140]. The compound shows a moderate tendency to accumulate in aquatic organisms.

- **Effects on other organisms:** At exposure levels well above permissible application rates (100 mg/kg), trifluralin has been shown to be toxic to earthworms. However, permitted application rates will result in soil residues of approximately 1 ppm trifluralin, a level that had no adverse effects on earthworms [140]. It is nontoxic to bees [1].

Environmental Fate:

- **Breakdown in soil and groundwater:** Trifluralin is of moderate to high persistence in the soil environment, depending on conditions. Trifluralin is subject to degradation by soil microorganisms. Trifluralin remaining on the soil surface after application may be decomposed by UV light or may volatilize. Reported half-lives of trifluralin in the soil vary from 45 to 60 days [11] to 6 to 8 months [1]. After 6 months to 1 year, 80 to 90% of its activity will be gone [8]. It is strongly adsorbed on soils and nearly insoluble in water [11]. Because adsorption is highest in soils high in organic matter or clay content and adsorbed herbicide is inactive, higher application rates may be required for effective weed control on such soils [58,8]. Trifluralin has been detected in nearly 1% of the 5590 wells tested. However, it has been detected at very low concentrations, typically ranging from 0.002 ug/L to 15 ug/L [8].
- **Breakdown in water:** Trifluralin is nearly insoluble in water [1]. It will probably be found adsorbed to soil sediments and particulates in the water column.
- **Breakdown in vegetation:** Trifluralin inhibits the growth of roots and shoots when it is absorbed by newly germinated weed seedlings [58]. Trifluralin residues in crop plants will occur only in root tissues which are in direct contact with contaminated soil. Trifluralin is not translocated into the leaves, seeds, or fruit of most plants. On most crops, trifluralin applied to the leaves has no effect, but on certain crops, such as tobacco and summer squash, leaf distortion may occur [58].

Physical Properties:

- **Appearance:** Trifluralin is an odorless, yellow-orange crystalline solid [1].
- **Chemical Name:** a,a,a-trifluoro-2,6-dinitro-N,N-dipropyl-p-toluidine [1]
- **CAS Number:** 1582-09-8
- **Molecular Weight:** 335.50
- **Water Solubility:** <1 mg/L @ 27 C [1]
- **Solubility in Other Solvents:** s. in organic solvents such as acetone and xylene [1]
- **Melting Point:** 48.5-49 C [1]
- **Vapor Pressure:** 13.7 mPa @ 25 C [1]
- **Partition Coefficient:** 5.0719 @ pH 7 and 25 C [1]
- **Adsorption Coefficient:** 8000 [11]

Exposure Guidelines:

- **ADI:** Not Available
- **MCL:** Not Available
- **RfD:** 0.0075 mg/kg/day [13]
- **PEL:** Not Available
- **HA:** 0.005 mg/L (lifetime) [139]
- **TLV:** Not Available

Basic Manufacturer:

DowElanco
9330 Zionsville Road
Indianapolis, IN 46268-1054

- **Phone:** 317-337-7352
- **Emergency:** 800-258-3033

References:

References for the information in this PIP can be found in Reference List [Number 10](#)

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