
NPTN Technical Fact Sheets are designed to provide information that is technical in nature for individuals with a scientific background or familiarity with the regulation of pesticides by the U.S. Environmental Protection Agency (U.S. EPA). This document is intended to be helpful to professionals and to the general public for making decisions about pesticide use.

National
Pesticide
Telecommunications
Network

BORIC ACID

(Technical Fact Sheet)

For less technical information, please refer to the General Fact Sheet.

The Pesticide Label: Labels provide directions for the proper use of a pesticide product. *Be sure to read the entire label before using any product.* A signal word on each product label indicates the product's potential hazard.

CAUTION - low toxicity

WARNING - moderate toxicity

DANGER - high toxicity

What is boric acid?

- Boric acid and its sodium borate salts are active ingredients of pesticide products used against insects, spiders, mites, algae, molds, fungi, and weeds. They were first registered as pesticides in the United States in 1948 and reregistered in 1993. The borate salts include sodium tetraborate decahydrate, sodium tetraborate pentahydrate, sodium tetraborate (anhydrous borax), disodium octaborate tetrahydrate, disodium octaborate (anhydrous), and sodium metaborate (1). *Use of the term "boric acid" in this fact sheet refers to the acid and/or borate salts.*
- Boric acid is derived from boron, a naturally occurring element found in rocks, soil, and water. Boron is universal in the environment and typically is found in the form of borates or combined with other chemicals (2, 3, 4).
- Boric acid is generally a white solid, but it ranges from clear to opaque. It is odorless and generally stable under ambient conditions (1).
- Signal words for products containing boric acid range from Caution to Danger (5). The signal word reflects the combined toxicity of boric acid and other ingredients in each product. See the **Pesticide Label** box above.
- Boric acid products are used on a variety of sites including sewage systems, food and non-food crops, outdoor residential areas, and indoor sites such as homes, hospitals, and commercial buildings (1). Commercial formulations of the pesticide include aerosols, liquids (solutions, emulsifiable concentrates), granules, wettable powders, dusts, pellets/tablets, and impregnated materials (baits, stakes, etc.) (5).

How does boric acid work?

- Insects die by ingesting boric acid and borate salts. Borate salts are abrasive to the insect exoskeleton (1).
- Boron is an essential plant micronutrient, and some boric acid products are used to correct boron deficiencies in plants (1, 3). Plants require small amounts of boron but high concentrations are toxic (3). At high levels, boric acid is an herbicide that disrupts photosynthesis and causes plant desiccation (1).

- As a fungicide, boric acid inhibits the maturation of fungi by preventing spore formation (1).
- The mechanism of toxicity in animals is not known (6).

What are some products that contain boric acid?

- BORA-CARE®
- JECTA®
- NIBAN®
- NIBOR®
- Tim-Bor®

How toxic is boric acid?

Animals

- Boric acid is very low to low in toxicity when ingested. The acute oral LD50 in mice is 3450 mg/kg and for rats ranges from 2660-5140 mg/kg (2). See boxes on **Laboratory Testing**, **LD50/LC50**, and **Toxicity Category**.
- The LC50 values in mice for inhaled *boron* compounds range from 0.89-21.1 mg/L, indicating very low to low inhalation toxicity (2). The U.S. Environmental Protection Agency (EPA) has not required acute inhalation toxicity studies for boric acid (1).
- Boric acid is low in toxicity when applied to the skin (LD50 >2000 mg/kg) (1). It is poorly absorbed in rabbits across intact skin, but penetration is increased in damaged skin (7).
- The U.S. EPA classifies boric acid as low to very low in toxicity for skin irritation (1).
- Data are not available regarding skin sensitization (1).
- Boric acid is generally low in toxicity for eye irritation. An exception is sodium tetraborate (anhydrous borax), which is highly toxic to the eye (1).
- In a 90-day study, investigators fed dogs boric acid (males: 0, 3, 35, or 268 mg/kg/day; females: 0, 2, 22, or 192 mg/kg/day). At the highest dose (males: 268 mg/kg/day; females: 192 mg/kg/day), they noted altered blood chemistry, fat accumulation in select tissues, and toxicity to the testes. The no observed adverse effect level (NOAEL) was 35 mg/kg/day in males and 22 mg/kg/day in females (1).
- Researchers fed dogs boric acid in the diet for 2 years at doses of 0, 13, 26, or 77 mg/kg/day. The NOAEL was 77 mg/kg/day (1).
- Signs of toxicity in laboratory animals poisoned with boric acid include depression, muscle incoordination, vomiting, purple-red skin coloration, lowered body temperature, and central nervous system pain (6).

Exposure: Effects of boric acid on human health and the environment depend on how much boric acid is present and the length and frequency of exposure. Effects also depend on the health of a person and/or certain environmental factors.

Laboratory Testing: Before pesticides are registered by the U.S. EPA, they must undergo laboratory testing for short-term (acute) and long-term (chronic) health effects. Laboratory animals are purposely fed high enough doses to cause toxic effects. These tests help scientists judge how these chemicals might affect humans, domestic animals, and wildlife in cases of overexposure. When pesticide products are used according to the label directions, toxic effects are not likely to occur because the amount of pesticide that people and pets may be exposed to is low compared to the doses fed to laboratory animals.

Toxicity Category (Signal Word) (8)

	High Toxicity (Danger)	Moderate Toxicity (Warning)	Low Toxicity (Caution)	Very Low Toxicity (Caution)
Oral LD50	Less than 50 mg/kg	50 - 500 mg/kg	500 - 5000 mg/kg	Greater than 5000 mg/kg
Dermal LD50	Less than 200 mg/kg	200 - 2000 mg/kg	2000 - 5000 mg/kg	Greater than 5000 mg/kg
Inhalation LC50	Less than 0.05 mg/l	0.05 - 0.5 mg/l	0.5 - 2 mg/l	Greater than 2 mg/l
Eye Effects	Corrosive	Irritation persisting for 7 days	Irritation reversible within 7 days	Minimal effects, gone within 24 hrs
Skin Effects	Corrosive	Severe irritation at 72 hours	Moderate irritation at 72 hours	Mild or slight irritation

LD50/LC50: A common measure of acute toxicity is the lethal dose (LD50) or lethal concentration (LC50) that causes death (resulting from a single or limited exposure) in 50 percent of the treated animals. LD50 is generally expressed as the dose in milligrams (mg) of chemical per kilogram (kg) of body weight. LC50 is often expressed as mg of chemical per volume (e.g., liter (L)) of medium (i.e., air or water) the organism is exposed to. Chemicals are considered highly toxic when the LD50/LC50 is small and practically non-toxic when the value is large. However, the LD50/LC50 does not reflect any effects from long-term exposure (i.e., cancer, birth defects, or reproductive toxicity) that may occur at levels below those that cause death.

Humans

- Potential lethal boric acid doses are 3-6 g for infants and 15-20 g for adults (2).
- Two siblings, one 24 days old and the other 14 months, were inadvertently fed boric acid in their formula. The total amounts ingested were 2.6 g and 1.95 g, respectively. Symptoms included irritability, diarrhea, and localized redness in the groin area. Neither child developed severe toxicity, and both were asymptomatic one month after the incident (9).
- Investigators evaluated 784 cases of boric acid ingestion reported to poison control centers. No patients experienced severe toxicity and 692 (88.3%) were asymptomatic. In those patients experiencing symptoms, the most common complaints were vomiting, abdominal pain, and diarrhea. Lethargy, headaches, lightheadedness, and rashes were less frequent symptoms (10).

Is boric acid metabolized and excreted from the body?

Animals

- Absorption of boric acid by oral exposure is rapid and complete (approximately 95%). Metabolism of the chemical is limited in biological systems and the parent compound is eliminated primarily in the urine (2, 11).
- Rats fed boric acid in their diets did not accumulate the chemical in soft tissues. The highest level of retention occurred in bone in the form of boron (11).

Humans

- Six male volunteers ate single 750 mg oral doses of boric acid and excreted 94% of the chemical over a 96-hour period (12).
- In a study of nine patients ingesting boric acid, the elimination half-life ranged from 4.0-27.8 hours (10). See box on **Half-life**.

Half-life is the time required for half of the compound to degrade.

- 1 half-life = 50% degraded**
- 2 half-lives = 75% degraded**
- 3 half-lives = 88% degraded**
- 4 half-lives = 94% degraded**
- 5 half-lives = 97% degraded**

Remember that the amount of chemical remaining after a half-life will always depend on the amount of the chemical originally applied.

- Investigators evaluated infants 1.25-10 months old who received dermal applications of talcum powder containing 5% boric acid 7-10 times/day for at least 1 month for diaper rash. The calculated dose for the infants was 2.33 g boric acid/day. Only trace amounts of boric acid penetrated the skin (2).

Does boric acid cause reproductive or teratogenic effects?

Animals

- In a three-generation reproductive study, researchers fed rats boric acid at 0, 65, 154, or 515 mg/kg/day. At the highest dose (515 mg/kg/day), the rats did not reproduce, and researchers noted lower body weight gains in both sexes, decreased food efficiency in females, and testicular degeneration in males. The reproductive NOAEL was 154 mg/kg/day (1).
- In a two-generation reproductive study, scientists fed mice boric acid at 0, 150, 675, or 1350 mg/kg/day. They detected reproductive effects at the two highest doses (675 and 1350 mg/kg/day). At 675 mg/kg/day, scientists noted an increase in days between litters and a decrease in the number of females producing litters. At 1350 mg/kg/day, the mice did not produce litters, and the males had decreased sperm concentrations and motility. The reproductive NOAEL was 150 mg/kg/day (1).
- Researchers fed pregnant rats boric acid at approximate doses of 0, 78, 163, 330, or 539 mg/kg/day. They exposed the rats at the highest dose (539 mg/kg/day) on gestation days 6-15 and the other dose groups on gestation days 0-20. The maternal NOAEL was 78 mg/kg/day based on increased liver and kidney weights at higher doses. Scientists detected lower fetal body weights at all doses and increased incidences of rib and brain abnormalities at the three highest doses (163, 330, and 539 mg/kg/day) (13).
- In a developmental study, scientists exposed pregnant mice to boric acid on gestation days 0-17 at doses of 0, 248, 452, or 1003 mg/kg/day. At all doses, they noted kidney effects in mothers. The developmental NOAEL was 248 mg/kg/day based on lower fetal body weights at the two highest doses and morphological abnormalities at the highest dose (13).
- Laboratory workers orally exposed pregnant rabbits by gavage (stomach tube) to 0, 62.5, 125, or 250 mg/kg/day boric acid on gestation days 6-19. At the highest dose (250 mg/kg/day), workers observed increased fetal mortality and cardiovascular abnormalities. They also noted decreased maternal food intake and vaginal bleeding associated with pregnancy loss. The maternal and developmental NOAELs were 125 mg/kg/day (14).

Humans

- Investigators evaluated reproductive effects of boric acid on males employed at a mining and production facility. They did not detect any effects on male fertility (15).
- Data are not available from occupational exposure, accidental poisonings, or epidemiological studies regarding the developmental toxicity of boric acid.

Is boric acid a carcinogen?

Animals

- Laboratory workers fed mice boric acid for 2 years at approximate doses of 0, 450, or 1150 mg/kg body weight/day. They noted no evidence of carcinogenicity or clinical signs of toxicity in the study. Workers did observe in males increased numbers of some cell types and decreased size of the testes (1).
- Researchers fed rats boric acid for 2 years at doses of 0, 65, 154, or 515 mg/kg/day. They noted no evidence of carcinogenicity. Researchers did detect decreased body weight, possible anemia, and testicular effects at the highest dose (515 mg/kg/day). The NOAEL was 154 mg/kg/day (1).

- Researchers often use studies designed to test for mutagenicity to screen chemicals for carcinogenicity. Sufficient evidence exists to determine that boric acid does not have significant potential to cause mutagenicity (1, 2).

Cancer: The U.S. EPA has strict guidelines that require testing of pesticides for their potential to cause cancer. These studies involve feeding laboratory animals large *daily* doses of the pesticide over most of the lifetime of the animal. Based on these tests, and any other available information, EPA gives the pesticide a rating for its potential to cause cancer in humans. For example, if a pesticide does not cause cancer in animal tests at large doses, then the EPA considers it unlikely the pesticide will cause cancer in humans. Testing for cancer is not done on human subjects.

Humans

- The U.S. EPA currently classifies boric acid as a group E carcinogen (16). This means that boric acid is not considered a human carcinogen based on adequate evidence of non-carcinogenicity in laboratory animals. See box on **Cancer**.
- Data are not available from occupational exposures or epidemiological studies regarding the carcinogenicity of boric acid.

What is the environmental fate and behavior of boric acid?

- Boric acid naturally occurs in air, water (surface and ground water), soil, and plants, including food crops. It enters the environment through weathering of rocks, volatilization from seawater, and volcanic activity (2).
- Human activities and products that contribute to boric acid in the environment include agricultural chemicals, irrigation drainwater, laundry products, mining and processing, and coal burning (2, 4).
- Most boron compounds convert to boric acid in the environment, and boric acid is the boron compound of environmental significance (4).
- The relatively high water solubility of boric acid results in the chemical reaching aquatic environments (4).
- Boric acid is assumed to adsorb to soil particles and aluminum and iron minerals (4). Adsorption can be either reversible or irreversible, depending on soil characteristics (2). Boric acid is mobile in soil (1).

What effects does boric acid have on wildlife?

- Boric acid is practically nontoxic to fish (LC50 >1021 mg/L) and aquatic invertebrates (133-226 mg/L). Boric acid has a low bioaccumulation potential (1).
- Boric acid is practically nontoxic to birds acutely (LD50 >2510 mg/kg) (1). Boric acid may adversely affect development of young birds. In studies with mallards, the highest tested doses affected ducklings by increasing mortality, altering behavior, and decreasing growth and hatching success (17-19). The U.S. EPA does not anticipate adverse effects to birds from the use patterns of boric acid products (1).
- Boric acid is relatively nontoxic to bees. The contact LD50 is greater than 360 µg/g (1).

Date reviewed: August 24, 2001

For more information contact: NPTN

Oregon State University, 333 Weniger Hall, Corvallis, Oregon 97331-6502.

Phone: 1-800-858-7378 Fax: 1-541-737-0761 Email: nptn@ace.orst.edu

NPTN at <http://nptn.orst.edu/> EXTTOXNET at <http://ace.orst.edu/info/exttoxnet/>

References

1. *Reregistration Eligibility Decision Document: Boric Acid and its Sodium Salts*; EPA 738-R-93-017; U.S. Environmental Protection Agency, Office of Pesticide Programs, U.S. Government Printing Office: Washington, DC, September 1993.
2. World Health Organization. *Boron*, Environmental Health Criteria, 204; Geneva, Switzerland, 1998.
3. Woods, W. G. An Introduction to Boron: History, Sources, Users, and Chemistry. *Environ. Health Perspect.* **1994**, *102* (Suppl. 7), 5-11.
4. Eisler, R. Boron Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. *U.S. Fish and Wildlife Serv. Biol. Rep.* **1990**, *82*(1.20), 1-32.
5. *Pest-Bank Pesticide Product Data* [CD-ROM]; Purdue Research Foundation: West Lafayette, IN, 2000.
6. Clarkson, T. W. Inorganic and Organometal Pesticides. In *Handbook of Pesticide Toxicology*; Hayes, W. J. Jr., Laws, E. R. Jr., Eds.; Academic: San Diego, CA, 1991; Vol. 2, pp 497-583.
7. Draize, J. H.; Kelley, E. A. The Urinary Excretion of Boric Acid Preparations following Oral Administration and Topical Applications to Intact and Damaged Skin of Rabbits. *Toxicol. Appl. Pharmacol.* **1959**, *1*, 267-276.
8. U.S. Environmental Protection Agency, Office of Pesticide Programs, Washington, DC. Label Review Manual. <http://www.epa.gov/oppfead1/labeling/lrm/chap-07.htm> (accessed Dec 2000).
9. Baker, D. M.; Bogema S. C. Ingestion of Boric Acid by Infants. *Am. J. Emerg. Med.* **1986**, *4*, 358-361.
10. Litovitz, T. L.; Klein-Schwartz, W.; Oderda, G. M.; Schmitz, B. F. Clinical Manifestations of Toxicity in a Series of 784 Boric Acid Ingestions. *Am. J. Emerg. Med.* **1988**, *6*, 209-213.
11. Moseman, R. F. Chemical Disposition of Boron in Animals and Humans. *Environ. Health Perspect.* **1994**, *102*(Suppl 7), 113-117.
12. Schou, J. S.; Jansen, J. A.; Aggerbeck, B. Human Pharmacokinetics and Safety of Boric Acid. *Arch. Toxicol.* **1984**, *7*(Suppl), 232-235.
13. Heindel, J. J.; Price, C. J.; Field, E. A.; Marr, M. C.; Myers, C. B.; Morrissey, R. E.; Schwetz, B. A. Developmental Toxicity of Boric Acid in Mice and Rats. *Fundam. Appl. Toxicol.* **1992**, *18*, 266-277.
14. Price, C. J.; Marr, M. C.; Myers, C. B.; Seely, J. C.; Heindel, J. J.; Schwetz, B. A. The Developmental Toxicity of Boric Acid in Rabbits. *Fundam. Appl. Toxicol.* **1996**, *32*, 176-187.
15. Whorton, D. M.; Haas, J. L.; Trent, L.; Wong, O. Reproductive effects of sodium borates on male employees: birth rate assessment. *Occup. Environ. Med.* **1994**, *51*, 761-767.
16. *U.S. EPA Reference Dose Tracking Report*. U. S. Environmental Protection Agency, Office of Pesticide Programs, U.S. Government Printing Office: Washington, DC, 1997.
17. Hoffman, D. J.; Camardese, M. B.; Lecaptain, L. J.; Pendleton, G. W. Effects of Boron on Growth and Physiology in Mallard Ducklings. *Environ. Toxicol. Chem.* **1990**, *9*, 335-346.
18. Smith, G. J.; Anders, V. P. Toxic Effects of Boron on Mallard Reproduction. *Environ. Toxicol. Chem.* **1989**, *8*, 943-950.
19. Whitworth, M. R.; Pendleton, G. W.; Hoffman, D. J.; Camardese, M. B. Effects of Dietary Boron and Arsenic on the Behavior of Mallard Ducklings. *Environ. Toxicol. Chem.* **1991**, *10*, 911-916.

NPTN is sponsored cooperatively by Oregon State University and the U.S. Environmental Protection Agency. Data presented through NPTN documents are based on selected authoritative and peer-reviewed literature. The information in this profile does not in any way replace or supersede the restrictions, precautions, directions or other information on the pesticide label/ing or other regulatory requirements.
