This fact sheet was created in 2000; some of the information may be out-of-date. NPIC is not planning to update this fact sheet. More pesticide fact sheets are available here. Please call NPIC with any questions you have about pesticides at 800-858-7378, Monday through Friday, 7:30 am to 3:30 am PST.

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 (US EPA). This document is intended to be helpful to professionals and to the general public for making decisions about pesticide use.

DDT

(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 short-term toxicity.

CAUTION - low toxicity **WARNING - moderate toxicity** **DANGER - high toxicity**

What is DDT?

- DDT[1] is an organochlorine[2] insecticide that was first synthesized in 1874 by a chemist named Zeidler. Later another scientist, Mueller, discovered DDT's insecticidal properties in 1939 (1,2).
- DDT was a commonly-used pesticide for insect control in the United States until it was canceled in 1972 by the United States Environmental Protection Agency (U.S. EPA).
- Peak production of DDT in the U. S. was 188 million pounds in 1963. By the early 1970s, production decreased to 60 million pounds per year due to the ban (3).

Why was DDT used?

- DDT was initially used by the military in WW II for public health purposes (1). DDT was used to control malaria, typhus, body lice, and bubonic plague.
- DDT was a key element of malaria eradication in Italy and the United States. It was used to manage an epidemic of typhus in Italy and Germany during 1943-44 (1, 2). Cases of malaria decreased from 400,000 in 1946 to virtually none in 1950 (4).
- In addition to its public health uses, growers used DDT on a variety of food crops in the United States and worldwide. Some of the crops were beans, cotton, soybeans, sweet potatoes, peanuts, cabbage, tomatoes, cauliflower, brussel sprouts, corn, and other crops (3). DDT was also used in buildings for pest control.
- DDT is a versatile insecticide because it is effective, relatively inexpensive to manufacture, and persists in the environment (2).

Is DDT still used?

- DDT was canceled because of concern over carcinogenicity, bioaccumulation, and health effects on wildlife (5). In addition to these concerns, resistance to DDT occurs in some insects (like the house fly) that develop the ability to quickly metabolize DDT into the lower toxicity breakdown product DDE [3] (1).

Toxicity Category (***Signal Word***) (7)

	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
Inhalation LC50	Less than 0.2 mg/l	0.2 - 2 mg/l	2 - 20 mg/l	Greater than 20 mg/l
Dermal LD50	Less than 200 mg/kg	200 - 2000 mg/kg	2000 - 5000 mg/kg	Greater than 5000 mg/kg
Eye Effects	Corrosive	Irritation persisting for 7 days	Irritation reversible within 7 days	No irritation
Skin Effects	Corrosive	Severe irritation at 72 hours	Moderate irritation at 72 hours	Mild or slight irritation at 72 hours

What is the mechanism of action of DDT?

- DDT affects the nervous system by interfering with normal nerve impulses (2). DDT causes the nerve cells to repeatedly generate an impulse which accounts for the repetitive body tremors seen in exposed animals (2).

How toxic is DDT?

LD50/LC50: A common measure of toxicity is the lethal dose (LD50) or lethal concentration (LC50) which 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) which may occur at doses below those used in short-term studies.

Animals

- DDT is slightly to moderately acutely toxic to mammals, including humans, when ingested. See box on **Laboratory Testing**. The acute oral LD50 (rat) is 113 to 800 milligrams per kilogram of body weight or mg/kg (6). See boxes on **LD50** and **Toxicity Category**.

- One acute toxicity study found that DDT is slightly toxic if absorbed through the skin. The acute dermal LD50 (rat) is 2,500 to 3,000 mg/kg (6).

- DDT is poorly absorbed through mammalian skin, but it is easily absorbed through an insect's exoskeleton (2).

- Laboratory animals exposed to single or multiple doses of DDT develop hyperexcitability, tremors, incoordination, and convulsions. Death results from respiratory or heart failure (1).

- Animals given potentially fatal doses of DDT develop liver lesions (1). Mice and rats fed low doses (0 or 2 mg/kg for mice and 0 or 5 mg/kg for rats) of DDT over an extended period of time develop liver changes, including hepatocellular hypertrophy, margination, and formation of lipospheres. (1).

Humans

- People excessively exposed to DDT while working with the chemical or accidental exposure report a prickling sensation of the mouth, nausea, dizziness, confusion, headache, lethargy, incoordination, vomiting, fatigue, tremors in the extremities, anorexia, anemia, muscular weakness, hyperexcitability, anxiety, and nervous tension (2).

Does DDT cause reproductive or teratogenic effects?

Laboratory Testing: Before pesticides are registered by the U.S. EPA, they must undergo laboratory testing for short-term and long-term 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.

Animals

- In a three generational study, dogs fed DDT (technical) in low doses (up to 10 milligrams per kilogram or mg/kg) do not experience reproductive effects (1).
- DDE, one of DDT's breakdown products, fed to birds at a low dose (1 mg/kg) causes eggshell thinning (3). See section on Wildlife.
- Rats become sterile after 36 weeks when fed low doses (7.5 mg/kg) of DDT (6).
- Mice fed low levels of DDT have embryos that fail to attach to the uterus and irregular reproductive cycles (6). A six generational study of mice fed low to moderate doses (0, 25, 100 or 250 mg/kg) of DDT show reduced survival of the offspring at the two higher dose (1).

Humans

- Scientific data indicates that DDT causes no reproductive problems or teratogenic effects in humans (1).

Is DDT carcinogenic?

Animals

- Mammals exposed to moderate doses (500 mg/kg) of DDT (technical) have an increased risk of liver tumors (1).
- In a two generation study of mice fed low doses (2 mg/kg/day) of DDT for life, the males are twice as likely to develop liver tumors (1).

Humans

- The EPA has categorized DDT as a B2 carcinogen (8). This means that DDT has been shown to cause cancer in laboratory animals, but there is inadequate or no evidence that it may cause cancer in humans (1). See box on **Cancer**.
- Thirty-five workers employed at a DDT manufacturing facility were studied for 19 years; none of the workers developed cancer (1).
- To date, studies have not shown an increased risk of breast cancer in women exposed to DDT (9,10,11,12,13).

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, then the EPA considers it unlikely the pesticide will cause cancer in humans. Testing for cancer has not been done on human subjects.

Does DDT accumulate?

Fat Stores

- DDT is highly fat soluble (dissolves in fat easily), but is poorly soluble in water. Due to its 'fat-loving' nature it tends to accumulate in the fatty tissues of insects, wildlife, and people. DDT is stored and biomagnifies in fatty tissues, but produces no known toxic effects while it is stored (2).

- DDT is metabolized into various breakdown products in the body, including DDD (TDE)[4], DDE, and DDA[5] (6). Once there are no more exposures to DDT, the breakdown products are slowly excreted from the body at approximately 1% of the stored DDT per day (1).

- When fat stores are used during periods of starvation, the breakdown products of DDT are released into the blood where they are toxic to the liver and the nervous system (2).

- Once DDT is accumulated in the body, it can be excreted in the urine, feces or breast milk. Exposures to DDT can be measured in the blood and fat, where its presence would be expected. However, its concentration in breast milk is often used as a measurement of DDT exposure within a population. Because DDT stores in the fat, its presence would be expected in blood and breast milk samples in populations worldwide.

- There is no evidence to suggest that DDT stored in the body will affect human infant birth weight (1,14).

Biomagnification

- Because of DDT's chemical properties, it has the tendency to accumulate in animals. As animals lower on the food chain are eaten by other animals higher up, DDT becomes concentrated in the fatty tissues of the predators (3,15). This continues until reaching the primary predator of the food chain, who receives the highest dose of DDT, sometimes leading to adverse health effects. Once the use of DDT was discontinued in the U.S., its concentration in the environment and animals decreased.

- For six consecutive days, researchers measured the urine of workers at a DDT-formulating plant and analyzed it for the metabolite DDA[4]. DDA excretion occurred shortly after each work day during the first five days. It often peaked at midnight and then rapidly decreased. On the sixth day, when workers did not go to work, their DDA levels were low (1).

What is the environmental fate and behavior of DDT?

- DDT is highly persistent in the environment. The soil half-life for DDT is from 2 to 15 years (16). See box on **Half-life**. The half-life of DDT in an aquatic environment is about 150 years (17).

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.

What effects does DDT have on wildlife?

- DDT is slightly to moderately acutely toxic to birds when ingested (16). However, DDT causes reproductive problems in birds. DDE, a metabolite of DDT, causes eggshell thinning in birds which make the eggs more susceptible to fracturing (15). The exact mechanism of how DDE causes eggshell thinning in birds is not known.

- Predaceous birds are more sensitive to DDE than gallinaceous birds (15). In one study, American Kestrels (a predator) fed low doses of DDE (0, 0.3, 3, 6 or 10 mg/kg) produce thinly-shelled eggs (15). A study on Japanese quail (gallinaceous) fed moderate doses of 125 mg/kg DDE, DDT, and DDT (technical) shows no substantial eggshell thinning.

- DDT and its metabolites affect the reproductive rate of birds. Fifty percent of Japanese quail fed moderate doses (0, 100, 200 or 400 mg/kg) of DDT die after being exposed to the highest dose (15). The survivors exposed to the highest dose (400 mg/kg) exhibit decreased fertility and fewer hatchlings per clutch (15).

- DDT is highly acutely toxic to fish (15). DDT affects membrane function and enzyme systems. However, how it affects these systems in fish is not known (15).

- In one study, fish were not able to reliably detect DDT in water (15). This poor detection may increase the risk of exposure of fish to DDT in the environment.

- In a study designed to measure the effects of DDT on predation rates and feeding, Atlantic salmon eggs were exposed to water containing 5, 10, 50 or 100 µg/L of DDT (15). The hatched fry have balance problems and impaired behavioral development at 50 and 100 µg/L.

- DDT is highly acutely toxic to aquatic invertebrates at concentrations as low as 0.3 µg/L in water (15). DDT affects juvenile aquatic invertebrates more than adults (15). DDT causes reproductive, developmental, cardiovascular, and neurological changes in aquatic invertebrates (15).

- DDT is moderately acutely toxic to adult frogs when ingested. In an eight-week long study, no mortality was observed in adult frogs fed low doses (0.6 mg/kg) of DDT and allowed to consume a normal died twice a week (15). In the same study, 50% of frogs exposed to DDT in the same manner, but not allowed to consume a normal diet, died.

[1] DDT is dichlorodiphenyl**tri**chloro**ethane**
[2] Organochlorines are chemical compounds that contain hydrogen, carbon, chlorine, and, sometimes, other atoms.
[3] DDE is dichlorodiphenyl**di**chloro**ethylene**
[4] DDD (TDE) is dichlorodiphenyl**di**chloro**ethane**
[5] DDA is 2,2-bis(4-chlorophenyl)-acetic acid

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References

1. World Health Organization. *DDT and its derivatives. Environmental Health Criteria.* Geneva, Switzerland, 1979; Vol. 9.
2. *Casarett & Doull's toxicology. The basic science of poisons*, Fifth edition.; Klaassen, C. D., Amdur, M. O., Doull, J., Eds.; McGraw-Hill: New York, 1996.
3. *DDT. A review of scientific and economic aspects of the decision to ban its use as a pesticide*; EPA-540/1-75-022; U. S. Environmental Protection Agency, Office of Pesticide Programs, U. S. Government Printing Office: Washington, DC, 1975.
4. Casida, J. E.; Quistad, G. B. Golden age of insecticide research: past, present, or future? *Annu. Rev. Entomol.* **1998**, *43*, 1-16. http://biomedical.AnnualReviews.org/cgi/content/full/5/43/1.
5. *Suspended, canceled, and restricted use pesticides*; EPA-20T-1002; U. S. Environmental Protection Agency, Office of Pesticide Programs, U. S. Government Printing Office: Washington, DC, 1990.
6. *DDT*. Extension Toxicology Network (EXTOXNET); Oregon State University: Corvallis, Oregon, 1996. http://ace.orst.edu/info/extoxnet/pips/ddt.htm.
7. *Label Review Manual;* U.S. Environmental Protection Agency, Office of Pesticide Programs, U. S. Government Printing Office: Washington, DC, 1998. http://www.epa.gov/oppfead1/labeling/lrm/index.html.

8. *EPA Tracking Report;* U. S. Environmental Protection Agency: Washington, DC, 1997. http://ace.orst.edu/info/nptn/tracking/tracking.htm.
9. Safe, S. H. Interactions between hormones and chemicals in breast cancer. *Annu. Rev. Pharmacol. Toxciol.* **1998**, *38*, 121-158.
10. Hunter, D. J.; Hankinson, S. E.; Landen, F.; Colditz, G. A.; Manson, J. E.; Willett, W. C.; Speizer, F. E.; Wolff, M. S. Plasma organochlorine levels and the risk of breast cancer. *The New England Journal of Medicine* **1997**, *3337*(18), 1253-1258.
11. van't Veer, P.; Lobbezoo, I. E.; Martin-Moreno, J. M.; Guallar, E.; Gomez-Aracena, J.; Kardinal, A. F. M.; Kohlmeier, L.; Martin, B. C.; Strain, J. J.; Thamm, M.; van Zoonen, P.; Baumann, B. A.; Huttunen, J. K.; Kok, F. J. DDT (dicophane) and postmenopausal breast cancer in Europe: case-control study. *Br. Med. J.* **1997**, *315*, 81-85.
12. Lopez-Carillo, L.; Blair, A.; Lopez-Cervantes, M.; Cebrian, M.; Rueda, C.; Reyes, R.; Mohar, A.; Bravo, J. Dichlorodiphenyltrichloroethane serum levels and breast cancer risk: a case control study from Mexico. *Cancer Res* **1997**, *57*, 3728-3732.
13. Wolff, M. S.; Toniolo, P. G.; Lee, E. W.; Rivera, M.; Dubin, N. Blood levels of organochlorine residues and risk of breast cancer. *J. Natl. Cancer Inst.***1993**, 85(8), 648-652.
14. Hooper, K.; Petreas, M. X.; She, J.; Visita, P.; Winkler, J.; McKinney, M.; Mok, M.; Sy, F.; Garcha, J.; Gill, M.; Stephens, R. D.; Semenova, G.; Sharmanov, T.; Chuvakova, T. Analysis of breast milk to assess exposure to chlorinated contaminants in Kazakstan: PCBs and organochlorine pesticides in southern Kazakstan. *Environ Health Perspect* **1997**, *105*(11), 1250-1254.
15. World Health Organization. *DDT and its derivatives. Environmental aspects*. *Environmental Health Criteria.* Geneva, Switzerland, 1989; Vol. 83.
16. *Toxicology Profile for 4,4'-DDT, 4,4'-DDE, 4,4'-DDD (Update)*; U. S. Department of Human Health & Human Services, Agency for Toxic Substances and Disease Registry, 1994.
17. Water-related environmental fate of 129 priority pollutants. Callahan, M. A.; Slimak, M. W.; Gabel, N. W., Volume1. Washington, DC: United States Environmental Protection Agency, 1979. In: *The Hazardous Substances Data Bank (HSDB)* [CD-ROM]:U.S. National Library of Medicine; National Institutes of Health and Human Services: Bethesda, MD, 1998.