

insecticide

[Search](#) | [Index](#) | [Home](#) | [Glossary](#) | [Contact Us](#)

very low drinking water levels

September 2002

CONTENTS

Highlights

[What are aldrin and dieldrin?](#)

[What happens to aldrin and dieldrin when they enter the environment?](#)

[How might I be exposed to aldrin and dieldrin?](#)

[How can aldrin and dieldrin affect my health?](#)

[How likely are aldrin and dieldrin to cause cancer?](#)

[How can aldrin and dieldrin affect children?](#)

[How can families reduce their risk for exposure to aldrin and dieldrin?](#)

[Is there a medical test to show whether I've been exposed to aldrin and dieldrin?](#)

[Has the federal government made recommendations to protect human health?](#)

[References](#)

[Contact Information](#)

RELATED RESOURCES

ToxFAQ™

36k

ToxFAQ™ en Español

30k

Public Health Statement

131k

Public Health Statement

164k

Toxicological Profile

1.9MB

A-Z INDEX

A B C D E
F G H I J K
L M N O P
Q R S T U
V W X Y Z

ATSDR RESOURCES

ToxFAQs™

ToxFAQs™ en Español

Public Health Statements



SDMS DocID

2100493

ToxFAQs™

for

Aldrin/Dieldrin*(Aldrin/Dieldrin)*

CAS# Aldrin 309-00-2
Dieldrin 60-57-1

This fact sheet answers the most frequently asked health questions about aldrin and dieldrin. For more information, you may 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. This information is important 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 aldrin and dieldrin happens mostly from eating contaminated foods, such as root crops, fish, or seafood. Aldrin and dieldrin build up in the body after years of exposure and can affect the nervous system. Aldrin has been found in at least 207 of the 1,613 National Priorities List sites identified by the Environmental Protection Agency (EPA). Dieldrin has been found in at least 287 of the 1,613 sites.

What are aldrin and dieldrin?

Aldrin and dieldrin are insecticides with similar chemical structures. They are discussed together in this fact sheet because aldrin quickly breaks down to dieldrin in the body and in the environment. Pure aldrin and dieldrin are white powders with a mild chemical odor. The less pure commercial powders have a tan color. Neither substance occurs naturally in the environment.

From the 1950s until 1970, aldrin and dieldrin were widely used pesticides for crops like corn and cotton. Because of concerns about damage to the environment and potentially to human health, EPA banned all uses of aldrin and dieldrin in 1974, except to control termites. In 1987, EPA banned all uses.

[back to top](#)

What happens to aldrin and dieldrin when they enter the environment?

[Toxicological Profiles](#)[Minimum Risk Levels](#)[MMGs](#)[MHMI](#)[Interaction Profiles](#)[Priority List of Hazardous Substances](#)[Division of Toxicology](#)

- Sunlight and bacteria change aldrin to dieldrin so that we mostly find dieldrin in the environment.
- They bind tightly to soil and slowly evaporate to the air.
- Dieldrin in soil and water breaks down very slowly.
- Plants take in and store aldrin and dieldrin from the soil.
- Aldrin rapidly changes to dieldrin in plants and animals.
- Dieldrin is stored in the fat and leaves the body very slowly.

[back to top](#)

How might I be exposed to aldrin and dieldrin?

- Dieldrin is everywhere in the environment, but at very low levels.
- Eating food like fish or shellfish from lakes or streams contaminated with either chemical, or contaminated root crops, dairy products, or meats.
- Air, surface water, or soil near waste sites may contain higher levels.
- Living in homes that were once treated with aldrin or dieldrin to control termites.

[back to top](#)

How can aldrin and dieldrin affect my health?

People who intentionally or accidentally ingested large amounts of aldrin or dieldrin suffered convulsions and some died. Health effects may also occur after a longer period of exposure to smaller amounts because these chemicals build up in the body.

Some workers exposed to moderate levels in the air for a long time had headaches, dizziness, irritability, vomiting, and uncontrolled muscle movements. Workers removed from the source of exposure rapidly recovered from most of these effects.

Animals exposed to high amounts of aldrin or dieldrin also had nervous system effects. In animals, oral exposure to lower levels for a long period also affected the liver and decreased their ability to fight infections. We do not know whether aldrin or dieldrin affect the ability of people to fight disease.

Studies in animals have given conflicting results about whether aldrin and dieldrin affect reproduction in male animals and whether these chemicals may damage the sperm. We do not know whether aldrin or dieldrin affect reproduction in humans.

[back to top](#)

How likely are aldrin and dieldrin to cause cancer?

There is no conclusive evidence that aldrin or dieldrin cause cancer in humans. Aldrin and dieldrin have shown to cause liver cancer in mice. The International Agency for Research on Cancer (IARC) has determined that aldrin and dieldrin are not classifiable as to human carcinogenicity. The EPA has determined that aldrin and dieldrin are probable human carcinogens.

[back to top](#)

How can aldrin and dieldrin affect children?

Children can be exposed to aldrin and dieldrin in the same way as adults. There are no known unique exposure pathways for children. Children who swallowed amounts of aldrin or dieldrin much larger than those found in the environment suffered convulsions and some died, as occurred in adults. However, we do not know whether children are more susceptible than adults to the effects of aldrin or dieldrin.

We do not know whether aldrin or dieldrin cause birth defects in humans. Pregnant animals that ingested aldrin or dieldrin had some babies with low birth weight and some with alterations in the skeleton. Dieldrin has been found in human breast milk, therefore, it can be passed to suckling infants.

[back to top](#)

How can families reduce their risk for exposure to aldrin and dieldrin?

- Since aldrin and dieldrin are no longer produced or used, exposure to these compounds will occur only from past usage.
- Because aldrin and dieldrin were applied to the basement of some homes for termite protection, before buying a home families should investigate what, if any, pesticides have been used within the home.

[back to top](#)

Is there a medical test to show whether I've been exposed to aldrin and dieldrin?

There are laboratory tests that can measure aldrin and dieldrin in your blood, urine, and body tissues. Because aldrin changes to dieldrin fairly quickly in the body, the test has to be done shortly after you are exposed to aldrin. Since dieldrin can stay in the body for months, measurements of dieldrin can be made much longer after exposure to either aldrin or dieldrin. The tests cannot tell you whether harmful health effects will occur. These tests are not routinely available at the doctor's office because they require special equipment.

[back to top](#)

Has the federal government made recommendations to protect human health?

The EPA limits the amount of aldrin and dieldrin that may be present in drinking water to 0.001 and 0.002 milligrams per liter (mg/L) of water, respectively, for protection against health effects other than cancer. The EPA has determined that a concentration of aldrin and dieldrin of 0.0002 mg/L in drinking water limits the lifetime risk of developing cancer from exposure to each compound to 1 in 10,000.

The Occupational Safety and Health Administration (OSHA) sets a maximum average of 0.25 milligrams of aldrin and dieldrin per cubic meter of air (0.25 mg/m³) in the workplace during an 8-hour shift, 40 hour week. The National Institute for Occupational Safety and Health (NIOSH) also recommends a limit of 0.25 mg/m³ for both compounds for up to a 10-hour work day, 40-hour week.

The Food and Drug Administration (FDA) regulates the residues of aldrin and dieldrin in raw foods. The allowable range is from 0 to 0.1 ppm, depending on the type of food product.

[back to top](#)

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2002. Toxicological Profile for aldrin and dieldrin. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

[back to top](#)

Where can I get more information?

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.

For more information, contact:

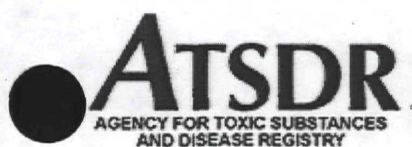
Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road NE, Mailstop F-32
Atlanta, GA 30333
Phone: 1-888-42-ATSDR (1-888-422-8737)
FAX: (770)-488-4178
Email: ATSDRIC@cdc.gov

[back to top](#)

ATSDR Information Center / ATSDRIC@cdc.gov / 1-888-422-8737

This page was updated on

[ATSDR Home](#) | [Search](#) | [Index](#) | [Glossary](#) | [Contact Us](#)
[About ATSDR](#) | [News Archive](#) | [ToxFAQs](#) | [HazDat](#) | [Public Health Assessments](#)
[Privacy Policy](#) | [External Links Disclaimer](#) | [Accessibility](#)
U.S. Department of Health and Human Services


[Search](#) | [Index](#) | [Home](#) | [Glossary](#) | [Contact Us](#)

September 2002

CONTENTS[Highlights](#)[What are DDT, DDE, and DDD?](#)[What happens to DDT, DDE, and DDD when they enter the environment?](#)[How might I be exposed to DDT, DDE, and DDD?](#)[How can DDT, DDE, and DDD affect my health?](#)[How likely are DDT, DDE, and DDD to cause cancer?](#)[How can DDT, DDE, and DDD affect children?](#)[How can families reduce the risk of exposure to DDT, DDE, and DDD?](#)[Is there a medical test to show whether I've been exposed to DDT, DDE, and DDD?](#)[Has the federal government made recommendations to protect human health?](#)[References](#)[Contact Information](#)**RELATED RESOURCES**[ToxFAQ™](#)  55k[ToxFAQ™ en Español](#)  30k[Public Health Statement](#)  163k[Public Health Statement en Español](#)  182k[Toxicological Profile](#)  2.6MB**A-Z INDEX**

A B C D E
F G H I J K
L M N O P
Q R S T U
V W X Y Z

ATSDR RESOURCES[ToxFAQs™](#)

ToxFAQs™
for
DDT, DDE, and DDD
(*DDT, DDE y DDD*)

CAS# DDT 50-29-3
DDE 72-55-9
DDD 72-54-8

This fact sheet answers the most frequently asked health questions about DDT, DDE, and DDD. For more information, you may 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. This information is important 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 DDT, DDE, and DDD occurs mostly from eating foods containing small amounts of these compounds, particularly meat, fish and poultry. **High levels of DDT can affect the nervous system causing excitability, tremors and seizures. In women, DDE can cause a reduction in the duration of lactation and an increased chance of having a premature baby.** DDT, DDE, and DDD have been found in at least 441 of the 1,613 National Priorities List sites identified by the Environmental Protection Agency (EPA).

What are DDT, DDE, and DDD?

DDT (dichlorodiphenyltrichloroethane) is a pesticide once widely used to control insects in agriculture and insects that carry diseases such as malaria. **DDT is a white, crystalline solid with no odor or taste. Its use in the U.S. was banned in 1972 because of damage to wildlife, but is still used in some countries.**

DDE (dichlorodiphenyldichloroethylene) and DDD (dichlorodiphenyldichloroethane) are chemicals similar to DDT that contaminate commercial DDT preparations. DDE has no commercial use. DDD was also used to kill pests, but its use has also been banned. One form of DDD has been used medically to treat cancer of the adrenal gland.

[back to top](#)

[ToxFAQs™ en Español](#)
[Public Health Statements](#)
[Toxicological Profiles](#)
[Minimum Risk Levels](#)
[MMGs](#)
[MHMIs](#)
[Interaction Profiles](#)
[Priority List of Hazardous Substances](#)
[Division of Toxicology](#)

What happens to DDT, DDE, and DDD when they enter the environment?

- DDT entered the environment when it was used as a pesticide; it still enters the environment due to current use in other countries.
- DDE enters the environment as contaminant or breakdown product of DDT; DDD also enters the environment as a breakdown product of DDT.
- DDT, DDE, and DDD in air are rapidly broken down by sunlight. Half of what's in air breaks down within 2 days.
- They stick strongly to soil; most DDT in soil is broken down slowly to DDE and DDD by microorganisms; half the DDT in soil will break down in 2-15 years, depending on the type of soil.
- Only a small amount will go through the soil into groundwater; they do not dissolve easily in water.
- DDT, and especially DDE, build up in plants and in fatty tissues of fish, birds, and other animals.

[back to top](#)

How might I be exposed to DDT, DDE, and DDD?

- Eating contaminated foods, such as root and leafy vegetable, fatty meat, fish, and poultry, but levels are very low.
- Eating contaminated imported foods from countries that still allow the use of DDT to control pests.
- Breathing contaminated air or drinking contaminated water near waste sites and landfills that may contain higher levels of these chemicals.
- Infants fed on breast milk from mothers who have been exposed.
- Breathing or swallowing soil particles near waste sites or landfills that contain these chemicals.

[back to top](#)

How can DDT, DDE, and DDD affect my health?

DDT affects the nervous system. People who accidentally swallowed large amounts of DDT became excitable and had tremors and seizures. These effects went away after the exposure stopped. No effects were seen in people who took small daily doses of DDT by capsule for 18 months.

A study in humans showed that women who had high amounts of a form of DDE in their breast milk were unable to breast feed their babies for as long as women who had little DDE in the breast milk. Another study in humans showed that women who had high amounts of DDE in breast milk had an increased chance of having premature babies.

In animals, short-term exposure to large amounts of DDT in food affected the nervous system, while long-term exposure to smaller amounts affected the liver. Also in animals, short-term oral

exposure to small amounts of DDT or its breakdown products may also have harmful effects on reproduction.

[back to top](#)

How likely are DDT, DDE, and DDD to cause cancer?

Studies in DDT-exposed workers did not show increases in cancer.

Studies in animals given DDT with the food have shown that DDT can cause liver cancer.

The Department of Health and Human Services (DHHS) determined that DDT may reasonably be anticipated to be a human carcinogen. The International Agency for Research on Cancer (IARC) determined that DDT may possibly cause cancer in humans. The EPA determined that DDT, DDE, and DDD are probable human carcinogens.

[back to top](#)

How can DDT, DDE, and DDD affect children?

There are no studies on the health effects of children exposed to DDT, DDE, or DDD. We can assume that children exposed to large amounts of DDT will have health effects similar to the effects seen in adults. However, we do not know whether children differ from adults in their susceptibility to these substances.

There is no evidence that DDT, DDE, or DDD cause birth defects in people. A study showed that teenage boys whose mothers had higher DDE amounts in the blood when they were pregnant were taller than those whose mothers had lower DDE levels. However, a different study found the opposite in preteen girls. The reason for the discrepancy between these studies is unknown.

Studies in rats have shown that DDT and DDE can mimic the action of natural hormones and in this way affect the development of the reproductive and nervous systems. Puberty was delayed in male rats given high amounts of DDE as juveniles. This could possibly happen in humans. A study in mice showed that exposure to DDT during the first weeks of life may cause neurobehavioral problems later in life.

[back to top](#)

How can families reduce the risk of exposure to DDT, DDE, and DDD?

- Most families will be exposed to DDT by eating food or drinking liquids contaminated with small amounts of DDT.
- Cooking will reduce the amount of DDT in fish.
- Washing fruit and vegetables will remove most DDT from their surface.
- Follow health advisories that tell you about consumption of fish and wildlife caught in contaminated areas.

[back to top](#)

Is there a medical test to show whether I've been exposed to DDT, DDE, and DDD?

Laboratory tests can detect DDT, DDE, and DDD in fat, blood, urine, semen, and breast milk. These tests may show low, moderate, or excessive exposure to these compounds, but cannot tell the exact amount you were exposed to, or whether you will experience adverse effects. These tests are not routinely available at the doctor's office because they require special equipment.

[back to top](#)

Has the federal government made recommendations to protect human health?

The Occupational Safety and Health Administration (OSHA) sets a limit of 1 milligram of DDT per cubic meter of air (1 mg/m³) in the workplace for an 8-hour shift, 40-hour workweek.

The Food and Drug Administration (FDA) has set limits for DDT, DDE, and DDD in foodstuff at or above which the agency will take legal action to remove the products from the market.

[back to top](#)

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2002. *Toxicological Profile for DDT, DDE, and DDD. Update*. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

[back to top](#)

Where can I get more information?

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.

For more information, contact:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road NE, Mailstop F-32
Atlanta, GA 30333
Phone: 1-888-42-ATSDR (1-888-422-8737)
FAX: (770)-488-4178
Email: ATSDRIC@cdc.gov

[back to top](#)

This page was updated on

[ATSDR Home](#) | [Search](#) | [Index](#) | [Glossary](#) | [Contact Us](#)
[About ATSDR](#) | [News Archive](#) | [ToxFAQs](#) | [HazDat](#) | [Public Health Assessments](#)
[Privacy Policy](#) | [External Links Disclaimer](#) | [Accessibility](#)
U.S. Department of Health and Human Services



U.S. Environmental Protection Agency

Ground Water & Drinking Water

[Recent Additions](#) | [Contact Us](#) | [Print Version](#) Search: **GO**

[EPA Home](#) > [Water](#) > [Ground Water & Drinking Water](#) > Consumer Factsheet on: LINDANE

Consumer Factsheet on: LINDANE

[Drinking Water and Health Basics](#)

[Frequently Asked Questions](#)

[Local Drinking Water Information](#)

[Drinking Water Standards](#)

[List of Contaminants & MCLs](#)

[Regulations & Guidance](#)

[Public Drinking Water Systems](#)

[Source Water Protection](#)

[Underground Injection Control](#)

[Data & Databases](#)

[Drinking Water Academy](#)

[Safe Drinking Water Act](#)

[National Drinking Water Advisory Council](#)

[Water Infrastructure Security](#)



List of Contaminants

As part of the Drinking Water and Health pages, this fact sheet is part of a larger publication:

National Primary Drinking Water Regulations

This is a factsheet about a chemical that may be found in some public or private drinking water supplies. It may cause health problems if found in amounts greater than the health standard set by the United States Environmental Protection Agency (EPA).

What is Lindane and how is it used?

Lindane is a white crystalline organic solid. Most uses being restricted in 1983, lindane is currently used primarily for treating wood-inhabiting beetles and seeds. It is also used as a dip for fleas and lice on pets, and livestock, for soil treatment, on the foliage of fruit and nut trees, vegetables, timber, ornamentals and for wood protection.

The list of trade names given below may help you find out whether you are using this chemical at home or work.

Trade Names and Synonyms:

gamma-Hexachlorocyclohexane
Exagamma
Forlin
Gallogamma
Gammaphex
Inexit
Kwell
Lindagranox
Lindaterra
Lovigram
Silvanol

Why is Lindane being Regulated?

In 1974, Congress passed the Safe Drinking Water Act. This law requires EPA to determine safe levels of chemicals in drinking water which do or may cause health problems. These non-enforceable levels, based solely on possible health risks and exposure, are called Maximum Contaminant Level Goals.

The MCLG for lindane has been set at 0.2 parts per billion (ppb) because EPA believes this level of protection would not cause any of the potential health problems described below.

Based on this MCLG, EPA has set an enforceable standard called a **Maximum Contaminant Level (MCL)**. MCLs are set as close to the MCLGs as possible, considering the ability of public water systems to detect and remove contaminants using suitable treatment technologies.

The MCL has been set at 0.2 ppb because EPA believes, given present technology and resources, this is the lowest level to which water systems can reasonably be required to remove this contaminant should it occur in drinking water.

These drinking water standards and the regulations for ensuring these standards are met, are called **National Primary Drinking Water Regulations**. All public water supplies must abide by these regulations.

What are the Health Effects?

Short-term: EPA has found lindane to potentially cause the following health effects when people are exposed to it at levels above the MCL for relatively short periods of time: **high body temperature and pulmonary edema**.

Long-term: Lindane has the potential to cause the following effects from a lifetime exposure at levels above the MCL: **liver and kidney damage**.

How much Lindane is produced and released to the environment?

Lindane enters surface water as a result of runoff from agricultural land and from home and garden applications where it is used as an insecticide.

From 1987 to 1993, according to EPA's Toxics Release Inventory, lindane releases to land and water totalled 1115 lbs.

What happens to Lindane when it is released to the environment?

When released to water, lindane is not broken down by microbes, but it is attacked by chemicals in basic waters. It is degraded by soil microbes, and may evaporate from the surface, or slowly leach to ground water. Lindane will accumulate slightly in fish and shellfish.

How will Lindane be Detected in and Removed from My Drinking Water?

The regulation for lindane became effective in 1992. Between 1993 and 1995, EPA required your water supplier to collect water samples every 3 months for one year and analyze them to find out if lindane is present above 0.02 ppb. If it is present above this level, the system must continue to monitor this contaminant.

If contaminant levels are found to be consistently above the MCL, your water supplier must take steps to reduce the amount of lindane so that it is consistently below that level. The following treatment methods have been approved by EPA for removing lindane: **Granular activated charcoal**.

How will I know if Lindane is in my drinking water?

If the levels of lindane exceed the MCL, 0.2 ppb, the system must notify the public via newspapers, radio, TV and other means. Additional actions, such as providing alternative drinking water supplies, may be required to prevent serious risks to public health.

Drinking Water Standards:

Mclg: 0.2 ppb

Mcl: 0.2 ppb

Learn more about your drinking water!

EPA strongly encourages people to learn more about their drinking water, and to support local efforts to protect and upgrade the supply of safe drinking water. Your water bill or telephone book's government listings are a good starting point.

Your local water supplier can give you a list of the chemicals they test for in your water, as well as how your water is treated.

Your state Department of Health/Environment is also a valuable source of information.

For help in locating these agencies or for information on drinking water in general, call: EPA's Safe Drinking Water Hotline: (800) 426-4791.

For additional information on the uses and releases of chemicals in your state, contact the: Community Right-to-Know Hotline: (800) 424-9346

List of Contaminants

[Safewater Home](#) | [About Our Office](#) | [Publications](#) | [Links](#) | [Office of Water](#) | [En Español](#) | [Questions and Answers](#)

[EPA Home](#) | [Privacy and Security Notice](#) | [Contact Us](#)

Last updated on Tuesday, November 28th, 2006
URL: http://www.epa.gov/safewater/contaminants/dw_contamfs/lindane.html

Lindane (BHC)

PESTICIDE NAME: Lindane (BHC)

Trade name(s): Isotox Seed Treater F, Gamma BHC

Manufacturer(s): Rhone-Poulence Agrochimie

14-20 rue Pierre Bouizet
Lyon 69009 France

I. Basic information

A. Molecular structure: C6H6Cl6

B. Chemical name: Gamma isomer of 1,2,3,4,5,6-hexachloro
cyclohexane

C. Derivatives: crude BHC metabolizes to beta, gamma, delta and
alpha isomers; gamma BHC metabolizes to gamma-pentachloro cyclohexene

D. Molecular weight: 290.8 g/mole

E. Solubility in water: 10 mg/l

F. Common physical appearance: colorless crystals

G. Oral LD50 (rat): 89-91 mg/kg

H. Pesticide classification: organochlorine insecticide

I. Restricted use list (N.Y.): yes

EPA priority pesticide list: no

J. Crop use: general pesticide on ornamentals

II. Text

Lindane is an immobile, long-lived organochlorine insecticide widely examined in the scientific literature. The persistence of lindane in soil is a matter of contention. Adsorption is considered to be reversible and can vary from 4 to 90% depending upon conditions. Organic matter is an important factor in adsorption of lindane; an increase in organic matter increases persistence whereas increasing

solubility of lindane results in an increase in mobility.

The literature contains substantial information concerning adsorption coefficients, degradation rates and leaching of lindane.

III. Soil information

A. Degradation and transformation

The degradation of lindane results in formation of the isomers alpha, beta, delta, and gamma BHC. These metabolites must be a consideration in discussion of the persistence of lindane. Individually, their persistence is beta>gamma>delta>alpha(13) and their dissipation in cultivated sandy soil has been reported to be alpha>gamma>delta>beta(17). The half-lives and recovery rates of these isomers as well as those of total lindane have been investigated. Values vary according to conditions: lindane added as crude BHC, at 43mo ca.50% remained as beta(13); half-life of lindane in silty clay = 10mo and sandy loam = 8mo(5); surface recovery of BHC in sandy loam at 6mo = 26.8-32.4% with subsoil recovery = 3.7-4.6% of applied(4). In uncultivated loam, no decrease in pesticide was found whereas in flooded sandy loam, there was a rapid decrease of BHC. In sandy loam cultivated twice, no decrease in alpha, gamma or delta isomers was seen and little decrease occurred in the beta isomer(17).

Degradation of lindane is influenced by several soil factors. In loam and sand increased organic matter resulted in an increase of lindane persistence(6). Loss is also pH dependent in that degradation under alkali conditions is higher than under normal conditions pointing to the influence of chemical factors. In sandy loam at pH8.2 and 9.5, the loss of BHC in 9mo was 28.6-33.4% of applied (pH8.2) and 41.7-45.4% of applied (pH9.5). Loss during the first 3mo was the highest, i.e., 12-13% (pH8.2) and 14-20% (pH9.5) (4). It has been shown that lindane degrades to a non-toxic residue which still responds to colorimetric analysis for BHC thus overestimation of lindane can result(4). Gamma BHC metabolizes to gamma-pentachlorocyclohexene with a toxicity 1/1000 that of lindane. In a mixture of muck, loam, sandy loam and clay loam, lindane was detoxified by an enzymatic process. Dechlorination occurs in moist acidic to neutral soils(20).

The following tables present data concerning degradation of lindane in soils. The reference is given in parentheses at the end of each title.

Concentration (ppm) of lindane in 3 Hawaiian soil materials 7yrs after application of the recommended dose(3)

Coral		Sandy loam		Clay	
applied	found	applied	found	applied	found
286	0.44	379	0.66	402	0.90

Residues of lindane isomers in sandy loam soil with pesticide applications from 1950-1953(16)

Isomer	Make-up of Tech.BHC	%iso. as total	%applied remain
		(1968)	(1968)
alpha	70	36	4
beta	6	36	44
gamma	12	16	10
delta	6	12	14

Residues of lindane (% of applied) in silt loam soil at 2 application rates (11)

Time (yrs) Applic. Rate (lb/A-6in) Residue lindane (%)

1	10	43
	100	55
11	10	0.50
	100	5.28
15	10	0.20
	100	0.17

B. Adsorption and transport

Lindane is readily adsorbed onto all types of soils. The rates of adsorption are related to soil adsorption capacity; soil bound residues are lower in sandy than loam soil (6). A study on adsorption of lindane on four soils found that equilibrium on mineral soils was reached in <2hrs whereas in muck the time was >10hrs. Adsorption percentages in this study were reported to be 97.74% (loamy sand), 98.07% (loam), 98.27% (sandy loam), and 99.89% (muck) of the total lindane applied. This equilibrium will be maintained until saturation is approached and the isotherm becomes non-linear (10). In sixteen Minnesota soils the lindane sorbed varied from 4-90%. The variability was not found to be mainly due to soil texture but to the organic carbon present (1,7). This was substantiated in a study on flooded soils which reported that sorption of lindane occurs almost entirely on organic matter (19). These authors reported that flooded soils adsorb less lindane than non-flooded especially under anaerobic, low organic matter conditions. Desorption is not affected by anaerobiosis as Fe³⁺ reduces to Fe²⁺ thereby decreasing inorganic surface area and resulting in interference with lindane adsorption (19).

The degree of desorption and leaching of lindane from soils is a subject of disagreement in the literature. A study on fine sandy loam and silty clay reported an application of 10cm water/mo to half the field plots whereas the other half were only irrigated when the pesticide was applied. Diffusion to untreated lower zones and to the surface where volatilization could occur was reported. Movement was greater in sandy loam soil than in the silty clay. In the second year of this study, lindane increased at the surface of both treatments. Lateral movement amounted to <15cm in 2yrs (5). Another study found lindane to be desorbed from a variety of soils in 2-4 washings (1).

If the solubility of lindane is increased, the mobility is increased (6). Volatilization increases with an increase in the vapor density of lindane; however, soil water content has no effect on the vapor density until the soil is dried to a monolayer of water (15). Lindane adsorption decreases (or desorption increases) as temperature increases (15).

The tables below present data concerning lindane adsorption in soils. The reference is given in parentheses at the end of each title.

Adsorption of lindane (percent of total) in organic and sandy loam soils (14)

soil	time	% adsorbed
organic	1min	55
	30min	70
	2hr	78

	24hr	79
sandy loam	1min	22
	30min	34

Adsorption coefficients on four soils(14)

	org.	sed	s.l.	s
l/n	0.98%	0.96	0.97	0.99
K	899	24	16	8

Residues of lindane (ug/g) in light sandy soil receiving regular lindane application over 15yr period (3 plots) (18)

Plot no.	Year	0-10	10-20	20-30	30-40	40-50	50-60 (cm)
B	1969	0.09	0.01	-	-	-	-
C	1969	0.34	0.07	-	-	-	-
	1973	0.03	0.02	-	-	-	-
D	1969	1.30	0.23	-	-	-	-
	1973	0.32	0.35	0.10	0.11	-	0.01

Distribution coefficient (P) of lindane in four soils(10)

Soil	P(ml/g)
sl	22.7
l	20.4
ls	17.3
muck	368

Amount of lindane not recovered as parent compound (original

application = 21kg/ha) (5)

	Placement	sandy clay(kg/ha)		fsl(kg/ha)	
	depth (cm)	1yr	2yr	1yr	2yr
irrigated	0-7.5	14.2	18.9	14.2	18.2
	7.5-15	12.0	17.1	16.0	18.6
non-irrig.	0-7.5	11.4	17.1	14.3	17.7
	7.5-15	10.6	16.3	9.5	18.2

Percent lindane recovered in four soils under two water regimes(7)

	12.7cm water added				25.4cm water added			
cm depth	c	sicl	l	ls	c	sicl	l	ls
0-3	79	82	47	60	77	68	31	37
3-6	1	4	24	17	4	12	38	28
6-9	T	T	1	2	T	1	6	4
9-12	0	0	0	T	T	T	1	1

Adsorption coefficients for beta and gamma isomers on varied

adsorbents: Ca-peaty muck, Ca-clay, Ca-bentonite, and silica gel in aqueous solutions where $k = \log x/m$ vs $\log c$ and $k' = \log x/m$ vs $\log c/c_0(12)$

Aq. gamma-BHC:		deg.C	k	k'	1/n
muck		10	377		.956
		20	331	2630	.969
		30	269	3350	.981
		40	197		.983
	clay	10	53.8		.866
		20	45.7	277	.841
		30	41.3	363	.845
		40	36.1		.846
	bentonite	20	2.92	19.5	.886
		30	2.71	28.2	.911
	gel	20	6.88	61.2	.938
		30	4.62	54.2	.957

Aq. beta-BHC:

muck		20	456	1170	.950
		30	437	1550	.990
		20	62.8	148	.861
		30	60.2	187	.883
	clay	19.8	3.96	9.33	.863
		30	4.46	13.9	.885
	bentonite	19.8	2.13	5.60	.971
		30	1.64	5.80	.985
	gel	20	456	1170	.950
		30	437	1550	.990
		20	62.8	148	.861
		30	60.2	187	.883

IV. References (*denotes key reference)

1. Adams, R.S., Jr. and P. Li. 1971. SSSAP. 35. 78-81.
2. Agnihotri, N.P., S.Y. Pandey, H.K. Jain and K.P. Srivastava. 1977. J.Ent.Res. 1. 89-91.
3. Bess, H.A. and J.W. Hylin. 1970. J.Econ.Ento. 63. 633-8.
- *4. Chawla, R.P. and S.L. Chopra. 1967. Punjab Ag.Univ.J.Res. 4. 96-103.
- *5. Cliath, M.M. and W.F. Spencer. 1971. SSSAP. 35. 791-95.
6. Fuhremann, T.W. and E.P. Lichtenstein. 1980. J.Ag.FoodChem 28. 446-52.
7. Guenzi, W.D. and W.E. Beard. 1967. SSSAP. 31. 644-7.
8. Huggenberger, F., J. Letey and W.J. Farmer. 1972. SSSAP. 36. 544-48.
9. Kahn, S.U. 1980. Pesticides in the Soil Environment. Amsterdam: Elsevier Press.
- *10. Kay, B.D. and D.E. Elrick. 1967. Soil Science. 104. 314-22.
11. Lichtenstein, E.P., T.W. Fuhremann and K.R. Schulz. 1971. J.Ag.FoodChem. 19. 718-21.

- *12. Mills, A.C. and J.W. Biggar. 1969. SSSAP. 33. 210-16.
13. Queensland. Tech. Comm. Bur. Sugar Exp. Sta. 1972. 43-44.
- *14. Sharom, M.S., J.R.W. Miles, C.R. Harris, F.L. McEwen. Water Res. 14. 1095-1100.
15. Spencer, W.F. and M.M. Cliath. 1970. SSSAP. 34. 574-8.
16. Stewart, D.K.R. and D. Chisholm. 1971. Can.J.SS. 51. 379-83.
- *17. Suzuki, M., Y. Yamato and T. Watanabe. 1975. Bull.Environ.Contam.Toxicol. 14. 520-9.
- *18. Voerman, S. and A.F.H. Besemer. 1975. Bull.Environ.Contam.Toxicol. 13. 501-5.
19. Wahid, P.A. and N. Sethunathan. 1980. J.Ag.FoodChem. 28. 623-25.
20. Yule, W.N., M.Chiba and A.V. Morley. 1967. J.Ag.FoodChem. 15. 1000-4.

Disclaimer: Please read the pesticide label prior to use. The information contained at this web site is not a substitute for a pesticide label. Trade names used herein are for convenience only; no endorsement of products is intended, nor is criticism of unnamed products implied. Most of this information is historical in nature and may no longer be applicable.



To Top

For more information relative to pesticides and their use, please contact the PMEP staff at:

5123 Comstock Hall
Cornell University
Ithaca, NY 14853-0901
(607)255-1866



Cornell University

Questions regarding the development of this web site should be directed to the [PMEP Webmaster](#)