DDT (dichlorodiphenyltrichloroethane)

**TRADE OR OTHER NAMES:** Trade or other names include Anofex, Cesarex, Chlorophenothane, Dedelo, p,p'-DDT, Dichlorodiphenyltrichloroethane, Dinocide, Didimac, Digmar, ENT 1506, Genitox, Guesapon, Guesarol, Gexarex, Gyron, Hildit, Ixodex, Kopsol, Neocid, OMS 16, Micro DDT 75, Pentachlorin, Rukseam, R50 and Zerdane (79,73).

**REGULATORY STATUS:** DDT is no longer registered for use in the United States, although it is still used in other (primarily tropical) countries. It is in EPA Toxicity Class II, moderately toxic (72). DDT was banned from use in the United States in 1972, and remains banned barring public health emergency (e.g., outbreak of malaria) (73).

**CHEMICAL CLASS:** Organochlorine

**INTRODUCTION:** DDT is an organochlorine insecticide used mainly to control mosquito-borne malaria; use on crops has generally been replaced by less persistent insecticides (79). It was extensively used during the Second World War among Allied troops and certain civilian populations to control insect typhus and malaria vectors, and was then extensively used as an agricultural insecticide after 1945 (73). DDT was banned for use in Sweden in 1970 and in the United States in 1972 (73). Many insect pests may have developed resistance to DDT (79). Unless otherwise specified, the toxicological, environmental effects and environmental fate and chemistry data presented here refer to the technical product DDT. Technical grade DDT is actually a mixture of three isomers of DDT, principally the p,p'-DDT isomer (ca. 85%), with the o,p'-DDT and o,o'-DDT isomers typically present in much lesser amounts (73).

**FORMULATION:** It is available in several different forms: aerosols, dustable powders, emulsifiable concentrates, granules and wettable powders (79, 72). It is reported to be compatible with many other pesticides and incompatible with alkaline substances (79).
**TOXICOLOGICAL EFFECTS**

- **Acute Toxicity:** DDT is moderately to slightly toxic to studied mammalian species via the oral route. Reported oral LD50s range from 113 to 800 mg/kg in rats (79,73); 150-300 mg/kg in mice (79); 300 mg/kg in guinea pigs (73); 400 mg/kg in rabbits (73); 500-750 mg/kg in dogs (79) and greater than 1,000 mg/kg in sheep and goats (79). Toxicity will vary according to formulation (79). DDT is readily absorbed through the gastrointestinal tract, with increased absorption in the presence of fats (73). One-time administration of DDT to rats at doses of 50 mg/kg led to decreased thyroid function and a single dose of 150 mg/kg led to increased blood levels of liver-produced enzymes and changes in the cellular chemistry in the central nervous system of monkeys (73). Single doses of 50-160 mg/kg produced tremors in rats, and single doses of 160 mg/kg produced hind leg paralysis in guinea pigs (73). Mice suffered convulsions following a one-time oral dose of 200 mg/kg. Single administrations of low doses to developing 10-day old mice are reported to have caused subtle effects on their neurological development (73). DDT is slightly to practically non-toxic to test animals via the dermal route, with reported dermal LD50s of 2,500-3,000 mg/kg in female rats (79, 73), 1000 in guinea pigs (73) and 300 in rabbits (73). It is not readily absorbed through the skin unless it is in solution (73). It is thought that inhalation exposure to DDT will not result in significant absorption through the lung alveoli (tiny gas-exchange sacs) but rather that it is probably trapped in mucous secretions and swallowed by exposed individuals following the tracheo-bronchial clearance of secretions by the cilia (73). Acute effects likely in humans due to low to moderate exposure may include nausea, diarrhea, increased liver enzyme activity, irritation (of the eyes, nose or throat), disturbed gait, malaise and excitability; at higher doses, tremors and convulsions are possible (73, 76). While adults appear to tolerate moderate to high ingested doses of up to 280 mg/kg, a case of fatal poisoning was seen in a child who ingested one ounce of a 5% DDT:kerosene solution (73).

- **Chronic Toxicity:** DDT has caused chronic effects on the nervous system, liver, kidneys, and immune systems in experimental animals (73, 74). Effects on the nervous system observed in test animals include: tremors in rats at doses of 16-32 mg/kg/day over 26 weeks; tremors in mice at doses of 6.5-13mg/kg/day over 80-140 weeks; changes in cellular chemistry in the central nervous system of monkeys at doses of 10 mg/kg/day over 100 days, and loss of equilibrium in monkeys at doses of 50 mg/kg/day for up to 6 months (73). The main effect on the liver seen in animal studies was localized liver damage. This effect was seen in rats given 3.75 mg/kg/day over 36 weeks, rats exposed to 5 mg/kg/day over 2 years and dogs at doses of 80 mg/kg/day over the course of 39 months (73). In many cases lower doses produced subtle changes in liver cell physiology, and in some cases higher doses produced more severe effects (73). In mice doses of 8.33 mg/kg/day over 28 days caused increased liver weight and increased liver enzyme activity (73). Liver enzymes are commonly involved in detoxification of foreign compounds, so it is unclear whether increased liver enzyme activity in itself would constitute an adverse effect. In some species (monkeys and hamsters), doses as high as 8-20 mg/kg/day caused no observed adverse effects over exposure periods as long as 3.5-7 years (73). Kidney effects observed in animal studies include adrenal gland hemorrhage in dogs at doses of 138.5 mg/kg/day over 10 days and adrenal gland damage at 50 mg/kg/day over 150 days in dogs (73). Kidney damage was also seen in rats at doses of 10 mg/kg/day over 27 months (73). Immunological effects observed in test animals include: reduced antibody formation in mice following administration of 13 mg/kg/day for 3-12 weeks and reduced levels of immune cells in rats at doses of 1 mg/kg/day (73). No immune system effects were observed in mice at doses of 6.5 mg/kg/day for 3-12 weeks (73). Dose levels at which effects were
observed in test animals are very much higher than those which may be typically encountered by humans (74). The most significant source of exposure to individuals in the United States is occupational, occurring only to those who work or worked in the production or formulation of DDT products for export (75). Analysis of U. S. market basket surveys showed approximately a 30-fold decrease in detected levels of DDT and metabolites in foodstuffs from 1969-1974, and another threefold drop from 1975-1981, with a final estimated daily dose of approximately 0.002 mg/person/day (73). Based on a standard 70-kg person, this results in a daily intake of approximately 0.00003 mg/kg/day. Due to the persistence of DDT and its metabolites in the environment, very low levels may continue to be detected in foodstuffs grown in some areas of prior use (73). It has been suggested that, depending on patterns of international DDT use and trade, it is possible that dietary exposure levels may actually increase over time (73). Persons eating fish contaminated with DDT or metabolites may also be exposed via bioaccumulation of the compound in fish (73). Even though current dietary levels are quite low, past and current exposures may result in measurable body burdens due to its persistence in the body (73). More information on the metabolism and storage of DDT and its metabolites in mammalian systems is provided below (Fate in Humans and Animals). Adverse effects on the liver, kidney and immune system due to DDT exposure have not been demonstrated in humans in any of the studies which have been conducted to date (73).

### Reproductive Effects:
There is evidence that DDT causes reproductive effects in test animals. No reproductive effects were observed in rats at doses of 38 mg/kg/day administered at days 15-19 of gestation (73). In another study in rats, oral doses of 7.5 mg/kg/day for 36 weeks resulted in sterility (73). In rabbits, doses of 1 mg/kg/day administered on gestation days 4-7 resulted in decreased fetal weights and 10 mg/kg/day on days 7-9 of gestation resulted in increased resorptions (73). In mice, doses of 1.67 mg/kg/day resulted in decreased embryo implantation and irregularities in the estrus cycle over 28 weeks (73). It is thought that many of these observed effects may be the result of disruptions in the endocrine (hormonal) system (73). Available epidemiological evidence from two studies does not indicate that reproductive effects have occurred in humans as a result of DDT exposure (73). No associations between maternal blood levels of DDT and miscarriage nor premature rupture of fetal membranes were observed in two separate studies (73, 77, 78). One study did report a significant association between maternal DDT blood levels and miscarriage, but the presence of other organochlorine chemicals (e.g., PCBs) in maternal blood which may have accounted for the effect make it impossible to attribute the effect to DDT and its metabolites (79).

### Teratogenic Effects:
There is evidence that DDT causes teratogenic effects in test animals as well. In mice, maternal doses of 26 mg/kg/day DDT from gestation through lactation resulted in impaired learning performance in maze tests (73). In a two-generational study of rats, 10 mg/kg/day resulted in abnormal tail development (73). Epidemiological evidence regarding the occurrence of teratogenic effects as a result of DDT exposure are unavailable (73). It seems unlikely that teratogenic effects will occur in humans due to DDT at likely exposure levels.

### Mutagenic Effects:
The evidence for mutagenicity and genotoxicity is contradictory. In only 1 out of 11 mutagenicity assays in various cell cultures and organisms did DDT show positive results (73). Results of in vitro and in vivo genotoxicity assays for chromosomal aberrations indicated that DDT was genotoxic in 8 out of 12 cases, and weakly genotoxic in 1 case (73). In humans, blood cell cultures of men occupationally exposed to DDT showed an increase in chromosomal damage. In a separate study, significant increases in chromosomal damage were reported in...
workers who had direct and indirect occupational exposure to DDT (73). Thus it appears that DDT may have the potential to cause genotoxic effects in humans, but does not appear to be strongly mutagenic. It is unclear whether these effects may occur at exposure levels likely to be encountered by most people.

- **Carcinogenic Effects**: The evidence regarding the carcinogenicity of DDT is equivocal. It has been shown to cause increased tumor production (mainly in the liver and lung) in test animals such as rats, mice and hamsters in some studies but not in others (73). In rats, liver tumors were induced in three separate studies at doses of 12.5 mg/kg/day over periods of 78 weeks to life, and thyroid tumors were induced at doses of 85 mg/kg/day over 78 weeks (73). In mice, lifetime doses of 0.4 mg/kg/day resulted in lung tumors in the second generation and leukemia in the third generation; liver tumors were induced at oral doses of 0.26 mg/kg/day in two separate studies over several generations. In hamsters, significant increases in adrenal gland tumors were seen at doses of 83 mg/kg/day in females (but not males), and in males (but not females) at doses of 40 mg/kg/day (73). In other studies, however, no carcinogenic activity was observed in rats at doses less than 25 mg/kg/day; no carcinogenic activity was seen in mice with at doses of 3-23 mg/kg/day over an unspecified period, and in other hamster studies there have been no indications of carcinogenic effects (73). The available epidemiological evidence regarding DDT’s carcinogenicity in humans, when taken as a whole, does not suggest that DDT and its metabolites are carcinogenic in humans at likely dose levels (73). In several epidemiological studies, no significant associations were seen between DDT exposure and disease, but in one other study, a weak association was observed (73, 80). In this latter study, which found a significant association between long-term, high DDT exposures and pancreatic cancers in chemical workers, there were questions raised as to the reliability of the medical records of a large proportion of the cancer cases (73,80).

- **Organ Toxicity**: Acute human exposure data and animal studies reveal that DDT can affect the nervous system, liver, kidney (73). Increased tumor production in the liver and lung has been observed in test animals (73). An association with pancreatic cancer was suggested in humans in one study (73, 80).

- **Fate in Humans & Animals**: DDT is very slowly transformed in animal systems (74). Initial degradates in mammalian systems are 1,1-dichloro-2,2-bis(p-dichlorodiphenyl)ethylene (DDE) and 1,1-dichloro-2,2-bis(p-chlorophenyl)ethane (DDD), which are very readily stored in fatty tissues (73). These compounds in turn are ultimately transformed into bis(dichlorodiphenyl) acetic acid (DDA) via other metabolites at a very slow rate (73). DDA, or conjugates of DDA, are readily excreted via the urine (73). Available data from analysis of human blood and fat tissue samples collected in the early 1970s showed detectable levels in all samples, but a downward trend in the levels over time (73). Later study of blood samples collected in the latter half of the 1970s showed that blood levels were declining further, but DDT or metabolites were still seen in a very high proportion of the samples (73). Levels of DDT or metabolites may occur in fatty tissues (e.g. fat cells, the brain, etc.) at levels of up to several hundred times that seen in the blood (73). DDT or metabolites may also be eliminated via mother’s milk by lactating women (73).

**ECOLOGICAL EFFECTS**

- **Effects on Birds**: DDT may be slightly toxic to practically non-toxic to birds. Reported dietary LD50s range from greater than 2,240 mg/kg in mallard, 841 mg/kg in Japanese quail and 1,334 mg/kg in pheasant (81). Other reported dietary LD50s in such species as bobwhite quail, California quail, red-winged blackbird, cardinal, house sparrow, blue jay, sandhill crane and
clapper rail also indicate slight toxicity both in acute 5-day trials and over longer periods of up to 100 days (82). In birds, exposure to DDT occurs mainly through the food web through predation on aquatic and/or terrestrial species having body burdens of DDT, such as fish, earthworms and other birds (82). There has been much concern over chronic exposure of bird species to DDT and effects on reproduction, especially eggshell thinning and embryo deaths (82). The mechanisms of eggshell thinning are not fully understood. It is thought that this may occur from the major metabolite, DDE, and that predator species of birds are the most sensitive to these effects (82). Laboratory studies on bird reproduction have demonstrated the potential of DDT and DDE to cause subtle effects on courtship behavior, delays in pairing and egg laying and decreases in egg weight in ring doves and Bengalese finches (82). The implications of these for long-term survival and reproduction of wild bird species is unclear. There is evidence that synergism may be possible between DDT’s metabolites and organophosphate (cholinesterase-inhibiting) pesticides to produce greater toxicity to the nervous system and higher mortality (82). Aroclor (polychlorinated biphenyls, or PCBs) may result in additive effects on eggshell thinning (82).

Effects on Aquatic Species: DDT is very highly toxic to many aquatic invertebrate species. Reported 96-hour LC50s in various aquatic invertebrates (e.g., stoneflies, midges, crayfish, sow bugs) range from 0.18 ug/L to 7.0 ug/L, and 48-hour LC50s are 4.7 ug/L for daphnids and 15 ug/L for sea shrimp (55). Other reported 96-hour LC50s for various aquatic invertebrate species are from 1.8 ug/L to 54 ug/L (82). Early developmental stages are more susceptible than adults to DDT’s effects (82). The reversibility of some effects, as well as the development of some resistance, may be possible in some aquatic invertebrates (55). DDT is very highly toxic to fish species as well. Reported 96-hour LC50s are less than 10 ug/L in coho salmon (4.0 ug/L), rainbow trout (8.7 ug/L), northern pike (2.7 ug/L), black bullhead (4.8 ug/L), bluegill sunfish (8.6 ug/L), largemouth bass (1.5 ug/L), and walleye (2.9 ug/L) (55). The reported 96-hour LC50s in fathead minnow and channel catfish are 21.5 ug/L and 12.2 ug/L respectively (55). Other reported 96-hour LC50s in largemouth bass and guppy were 1.5 ug/L and 56 ug/L respectively (82). Observed toxicity in coho and chinook salmon was greater in smaller fish than in larger (82). It is reported that DDT levels of 1 ng/L in Lake Michigan were sufficient to affect the hatching of coho salmon eggs (3). DDT may be moderately toxic to some amphibian species and larval stages are probably more susceptible than adults (81, 82). In addition to acute toxic effects, DDT may bioaccumulate significantly in fish and other aquatic species, leading to long-term exposure. This occurs mainly through uptake from sediment and water into aquatic flora and fauna, and also fish (82). Fish uptake of DDT from the water will be size-dependent with smaller fish taking up relatively more than larger fish (82). A half-time for elimination of DDT from rainbow trout was estimated to be 160 days (82). The reported bioconcentration factor for DDT is 1,000 to 1,000,000 in various aquatic species (83), and bioaccumulation may occur in some species at very low environmental concentrations (55). Bioaccumulation may also result in exposure to species which prey on fish or other aquatic organisms (e.g., birds of prey).

Effects on Other Animals (Nontarget species): Earthworms are not susceptible to acute effects of DDT and its metabolites at levels higher than those likely to be found in the environment, but they may serve as an exposure source to species that feed on them (82). DDT is non-toxic to bees; the reported topical LD50 for DDT in honeybees is 27 ug/bee (82). Laboratory studies indicate that bats may be affected by DDT released from stored body fat during long migratory periods (82).
ENVIRONMENTAL FATE

- **Breakdown in Soil and Groundwater:** DDT is very highly persistent in the environment, with a reported half-life of between 2-15 years (83, 84) and is immobile in most soils. Routes of loss and degradation include runoff, volatilization, photolysis and biodegradation (aerobic and anaerobic) (73). These processes generally occur only very slowly. Breakdown products in the soil environment are DDE and DDD, which are also highly persistent and have similar chemical and physical properties (82, 84). Due to its extremely low solubility in water, DDT will be retained to a greater degree by soils and soil fractions with higher proportions of soil organic matter (82). It may accumulate in the top soil layer in situations where heavy applications are (or were) made annually; e.g., for apples (72). Generally DDT is tightly sorbed by soil organic matter, but it (along with its metabolites) has been detected in many locations in soil and groundwater where it may be available to organisms (82, 83). This is probably due to its high persistence; although it is immobile or only very slightly mobile, over very long periods of time it may be able to eventually leach into groundwater, especially in soils with little soil organic matter. Residues at the surface of the soil are much more likely to be broken down or otherwise dissipated than those below several inches (3). Studies in Arizona have shown that volatilization losses may be significant and rapid in soils with very low organic matter content (desert soils) and high irradiance of sunlight, with volatilization losses reported as high as 50% in 5 months (85). In other soils (Hood River and Medford) this rate may be as low as 17-18% over 5 years (85). Volatilization loss will vary with the amount of DDT applied, proportion of soil organic matter, proximity to soil-air interface and the amount of sunlight (82).

- **Breakdown of Chemical in Surface Water:** DDT may reach surface waters primarily by runoff, atmospheric transport, drift, or by direct application (e.g. to control mosquito-borne malaria) (73). The reported half-life for DDT in the water environment is 56 days in lake water and approximately 28 days in river water (83). The main pathways for loss are volatilization, photodegradation, adsorption to water-borne particulates and sedimentation (73). Aquatic organisms, as noted above, also readily take up and store DDT and its metabolites. Field and laboratory studies in the United Kingdom demonstrated that very little breakdown of DDT occurred in estuary sediments over the course of 46 days (82). DDT has been widely detected in ambient surface water sampling in the United States at a median level of 1 ng/L (part per trillion) (73, 76).

- **Breakdown of Chemical in Vegetation:** DDT does not appear to be taken up or stored by plants to a great extent. It was not translocated into alfalfa or soybean plants, and only trace amounts of DDT or its metabolites were observed in carrots, radishes and turnips all grown in DDT-treated soils (82). Some accumulation was reported in grain, maize and riceplants, but little translocation occurred and residues were located primarily in the roots (73).

PHYSICAL PROPERTIES AND GUIDELINES

**Physical Properties:**

- **Appearance:** The physical appearance of technical product p,p'-DDT is a waxy solid, although in its pure form it consists of colorless crystals (79)
- **Chemical Name:** 1,1'-(2,2,2-trichloroethylidene)bis[4-chlorobenzene]; 1,1,1-trichloro-2,2-bis(4-chlorophenyl) ethane (79)
- **CAS Number:** 50-29-3 (79)
- **Molecular Weight:** 354.51 (79)
- **Water Solubility:** < 1 mg/L @ 20 degrees C (79)
- **Solubility in Other Solvents:** cyclohexanone v.s., dioxane v.s., benzene v.s., xylene v.s., trichloroethylene v.s., dichloromethane v.s., acetone v.s., chloroform v.s., diethyl ether v.s., ethanol s. and methanol s. (79).
- **Melting Point:** 108.5-109 degrees C (79)
- **Vapor Pressure:** 0.025 mPa @ 25 degrees C (79)
- **Partition Coefficient:** Not available
- **Adsorption Coefficient:** 100,000 (84)

**Exposure Guidelines:**
- **ADI:** 0.02 mg/kg/d (73)
- **MCL:** Not Available
- **RfD:** 0.0005 mg/kg/day (73)
- **PEL:** 1 mg/meters cubed (8-hour) (73)
- **HA:** Not Available
- **TLV:** Not Available

**BASIC MANUFACTURER**

No manufacturer review was available.

**REFERENCES**

References for the information in this PIP can be found in Reference List Number 6

**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.