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Guidelines for Interpretation of the Biological Effects of Selected Constituents in Biota, Water, and Sediment

DDT

Participating Agencies:

Bureau of Reclamation
U.S. Fish and Wildlife Service
U.S. Geological Survey
Bureau of Indian Affairs

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DDT

Description

DDT is a synthetic organochlorine compound, which has been used extensively for insect control throughout the world. Its technical name is dichlorodiphenyltrichloroethane or, more precisely, 2,2-*bis*(*p*-chlorophenyl)-1,1,1-trichloroethane. It has a molecular formula of $C_{14}H_9Cl_5$, a molecular weight of 354.5, and a melting point of 108 °C. It is insoluble in water but very soluble in ethanol and acetone. Technical grade DDT consists of a mixture of isomers, especially *p,p'*-DDT and *o,p'*-DDT (see figure 1); it is a cream-colored to gray powder with a faint fruitlike odor.

DDD and DDE (figure 1) are metabolites of DDT (Klaassen et al. 1986). These two breakdown products and DDT are often found together in the environment and are referred to collectively as total DDT.

Occurrence

DDT, DDD, and DDE are synthetic compounds and have no natural sources. DDT was synthesized as early as 1874, but its insecticidal properties were not discovered until 1939. DDT was patented for use in 1942 and was used during World War II to control lice and other insects on humans (Klaassen et al. 1986, EPA 1975). It was used most extensively during the 1950's and '60s, mainly to control insects on crops and to check vector-borne diseases, such as malaria, in humans. The domestic use of DDT peaked in 1959, but production did not peak until 1963. Since that time, exports of DDT have exceeded domestic use. DDT was banned in the United States in 1972, primarily due to its environmental effects (EPA 1975), but is very persistent in the environment and is still detected in many biochemical and geochemical surveys. In

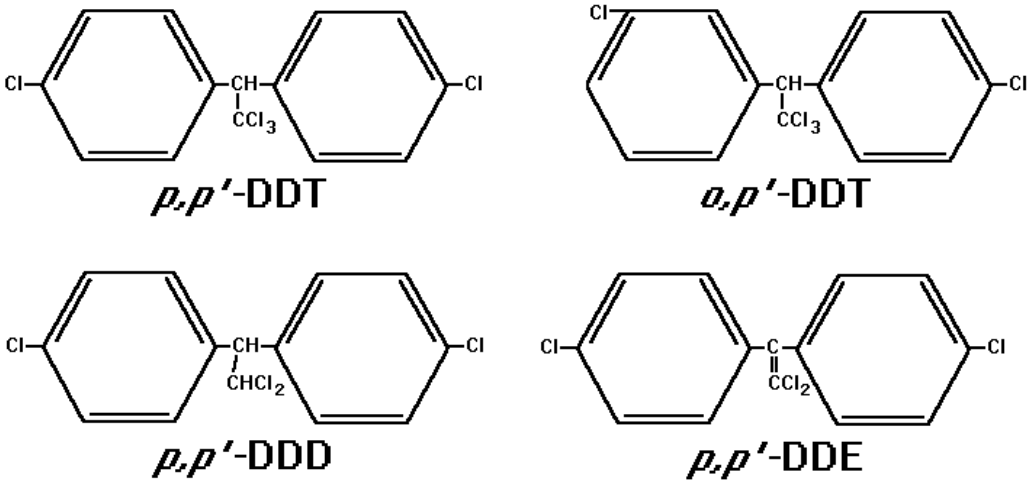

Figure 1.—Chemical structure of the two most common isomers of DDT and of the metabolites DDD and DDE.

addition, DDT is still in use in many parts of the world and is transported into the United States through animal migration and air and water movement.

Some DDT residues are derived from dicofol, an organochlorine pesticide with a structure similar to DDT, that has been used historically to treat mites in citrus orchards and cotton fields (Clark 1990). Studies conducted in 1982 found that dicofol products contained up to 15 percent total DDT. Although toxicity testing indicated that dicofol products were predominantly less toxic than DDT to fish, crustaceans, insects, birds, and mammals, EPA restricted the amount of total DDT contamination in dicofol to 2.5 percent, effective in 1986, and then reduced it further—to 0.1 percent—in 1989.

Summary of Effects

Background concentrations, effect levels, and criteria for the protection of fish and wildlife for DDD, DDE, and DDT are presented below for both physical media (surface water, sediment, and soil) and biotic media (aquatic organisms, terrestrial invertebrates, amphib-ians and reptiles, birds, and mammals). A summary of effect levels of DDT and its metabolites is presented in table 14. This summary is based on the available data, and ranges of effects were established using the following guidelines:

- No effect—studies reporting no observed effect levels or concentrations (NOELs and NOECs) and no observed adverse effect levels or concentrations (NOAELs and NOAECs)

- Level of concern—studies reporting some effect, including lowest observed effect and lowest observed adverse effect levels and concentrations (LOELs, LOECs, LOAELs, LOAECs), effective concentrations at which a certain percent of the test species showed an effect (e.g., EC25 or EC50), median effect concentrations where mortality was not the endpoint (e.g., MD25)

- Toxicity threshold—studies reporting mortality as the endpoint, such as lethal concentrations and doses (LC50s and LD50s)

In some cases, there is a gap or an overlap between levels of concern and toxicity thresholds. This may be the result of interspecies differences or of a lack of data reporting effects between the two ranges.

Field Cases

The persistence of organochlorine pesticides in the environment can be measured in different ways, including sampling and analysis of plants and wildlife (e.g., food items, predator species, or eggs) or abiotic media (e.g., surface water, sediment, or soil). The field studies described in this section include examples of these different measurements.

The persistence of DDT in surface water, sediment, and fish from the Yakima River drainage system in Washington was evaluated periodically from 1968 to 1982 by the U.S. Geological Survey and in recent studies by the State of Washington, Department of Ecology (Johnson et al. 1988). The primary source of DDT in the Yakima River was irrigation water runoff. At certain times of the year, irrigation water accounts for up to 80 percent of the water in the Yakima River.

The predominant compounds found during 1985 sampling of the Yakima River were as follows:

Table 14.— Summary of comprehensive effects: DDD, DDE, and DDT

["—" indicates that endpoints are not reported in available literature; dw = dry weight; ww = wet weight; bw, body weight. See Appendix II for explanation of other abbreviations and technical terms]

Medium	Compound	No effect	Level of concern	Toxicity threshold	Explanation
Water (µg/L)	Total DDT	—	—	0.013	For freshwater organisms (EPA 1996, ecotoxicity threshold).
		<0.3	0.3–800,000	—	For aquatic plants (ORNL 1996, lowest chronic value; EPA 1980, reduced growth).
	DDT	—	0.016	0.36	For *Daphnia* (see table 15).
	DDD	—	1.69–3.99	—	For fish (see table 15).[1]
	DDE	—	—	4,400	
	DDT	—	0.008–0.2	0.2	
Sediment (µg/kg dw)	DDD	—	8–110	6,000	Persaud et al. (1993), lowest (8) and severe (6,000) effect levels; ORNL 1996 screening criterion (110).
	DDE	—	2.2–27	19,000	Long et al. (1995) ERLs and ERMs; Persaud et al. (1993) severe effect levels.
	Total DDT	—	1.5–46	12,000	
Soil (mg/kg dw)	DDE	—	1.5	—	Toxic to *Lumbricus* (Cathey 1982).
Terrestrial inverte-brates (mg/kg dw)	Total DDT	—	32	—	Minimum hazardous level for birds (Beyer and Gish 1980).
Reptiles/amphibians	—	—	—	—	
Terrestrial birds, diet (mg/kg dw)	DDT	—	5–25	311	White-throated sparrow delayed development; LC50 for ring-necked pheasant (table 16).
	DDE	—	4	825	Bengalese finch impaired fledging; bobwhite quail LC 50 (table 16)
Raptor eggs (mg/kg ww)	DDE	—	3–16	—	Bald eagle eggshell thinning, reduced productivity (table 16).
Waterfowl diet (mg/kg dw, insectivores)	DDT	—	—	200	Mallard 95–100% lethality (Davison and Sell, 1974).
	DDE	—	10–30	3,572	Black duck eggshell thinning; mallard LC50 (table 16).
Waterfowl eggs (mg/kg ww, insectivores)	DDE	—	46–144	—	Black duck eggshell thinning (Longcore et al. 1971).
Waterfowl eggs (mg/kg ww, omnivores)	DDE	—	0.25–20	—	White-face ibis eggshell thinning (table 16).
Waterfowl eggs (mg/kg ww, piscivores)	DDE	—	0.62–66	—	Eggshell thinning, hooded merganser and brown pelican (table 16).
Mammals, diet (mg/kg bw/day)[1]	DDT	—	0.26–32.5	—	Mouse LOAELs (table 17).
		—	—	40	Bat LD50 (Clark and Stafford 1981)
		40	40–83	—	Hamster NOAEL & LOAEL (table 17)
		16	16–80	—	Dog NOAEL & LOAEL (Lehman 1965)
	DDD	107	—	—	Mouse NOAEL (NCI 1978).
Mammals, tissue (mg/kg dw)	DDT	—	—	210	LC50 for shrews (Blus 1978).

[1] Fish and mammalian effect levels vary widely depending on the endpoint and species. See tables 15 and 17 for species-specific toxicity information.

- Surface water
  River main stem: *p,p'*-DDE
  Tributaries: *p,p'*-DDE and *p,p'*-DDT

- Sediment: *p,p'*-DDT, *o,p'*-DDT, *p,p'*-DDE, and *p,p'*-DDD

- Fish: *p,p'*-DDE

The predominant metabolite found in surface water and fish collected from the main stem of the Yakima River was *p,p'*-DDE. In fish, it accounted for approximately 88 percent of the total DDT. These findings in fish and surface water from the main stem of the Yakima River indicate that DDT from historical releases has remained in the ecosystem for many years. In surface water collected from tributaries to the river and in sediment collected from both the main stem and from the tributaries, the concentrations of *p,p'*-DDT were equal to or greater than those of *o,p'*-DDT. In addition, *p,p'*-DDT to *o,p'*-DDT ratios in sediment were similar to those in technical-grade DDT (5:1). These ratios indicate that most of the DDT found in the Yakima River basin is under-graded material and suggest that DDT has a long half-life in these areas—perhaps near the upper end of the 4–30 year range proposed by Johnson et al. (1988).

Studies of other river drainages have also shown that levels of *p,p'*-DDT can remain high years after the last known usage. These include sediment from Puget Sound; surface water and sediments in Wisconsin streams; soils in New Mexico and Texas; and sediment, fish, and soil in California agricultural areas (Johnson et al. 1988). In addition, the occur-rence of *o,p'*-DDT and *p,p'*-DDT in ratios resembling technical-grade DDT indicates that *o,p'*-DDT is much more persistent than originally expected. Early studies of *o,p'*-DDT did not show it to be such a persistent com-pound because of difficulties in distinguishing it from other interfering compounds in the packed columns that were used in older analytical procedures (Johnson et al. 1988, Pham et al. 1993).

The occurrence and distribution of DDT in the St. Lawrence River in Quebec, Canada, also follows the trends observed in watersheds in the United States. Agricultural use of DDT in Canada peaked in 1969 but was banned during the 1970's. Restricted uses were still allowed until 1990 (Pham et al. 1993). Observed levels of *o,p'*-DDT in the St. Lawrence River indicated that *o,p'*-DDT was more persistent than *p,p'*-DDT. In addition, *p,p'*-DDT was found in the water column but not in the sediment. This may have been a result of dilution of suspended particulates carrying the *p,p'*-DDT as they settled through the water column or a result of the more rapid degradation of *p,p'*-DDT in sediment than in surface water. The pre-dominant degradation products of *p,p'*-DDT in sediment tend to be *p,p'*-DDE in the top aerobic layers and *p,p'*-DDD in the bottom anaerobic layers. It was hypothesized that *o,p'*-DDT would follow the same degradation processes as *p,p'*-DDT but at a slower rate (Pham et al. 1993).

The persistence of DDT in soil and earth-worms was studied for a 20-year period at the Patuxent Wildlife Research Center in Maryland (Beyer and Gish 1980, Beyer and Krynitsky 1989). DDT was sprayed on two replicate hay fields at a concentration of 9.0 kilograms per hectare. Concentrations in earthworms were measured several times throughout the 20-year period and half-lives (amount of time for the concentrations to decrease by 50 percent) were calculated for DDT and its metabolites in soil and earth-worms. Earthworms of various species and ages were collected from the test plots because presumably predators would not be selective in the type or age of the earthworms they fed on. Species collected included *Aporrectodea turgida*, *Aporrectodea trapezoides*, *Allolobophora chlorotica*, and *Lumbricus terrestris*. DDT metabolites found in soil 11 years after application included *p,p'*-DDT, *o,p'*-DDT, *p,p'*-DDE, and trace amounts of *p,p'*-DDD (Beyer and Gish 1980). DDT metabolites

found in earthworms 11 years after applica-tion included *p,p'*-DDT, *p,p'*-DDD, and *p,p'*-DDE. The half-life for total DDT in both soil and earthworms was 3.2 years (Beyer and Gish 1980). DDE was the most persistent DDT metabolite in earthworms (Beyer and Krynitsky 1989). DDE concentrations in earthworms increased during the first 3 years of the experiment to 13 mg/kg dry weight (dw) and then decreased through the remaining 17 years to 1.2 mg/kg dw, which was 9 percent of the peak concentration.

Abiotic Factors Affecting Bioavailability

Water

DDT and its metabolites are found in most surface water bodies, especially those that are downstream from tributaries draining urban and agricultural areas (EPA 1975). DDT can be transported to previously unimpacted water bodies through soil erosion, movement of suspended particulates, accumulated residues in free-swimming aquatic organisms, and rainfall carrying volatilized DDT.

Because of the low water solubility of DDT (1.2 µg/L), the water column primarily serves as a transfer mechanism between con-taminated sediments and aquatic organisms (EPA 1975). DDT present in the water column is directly available for uptake by aquatic plants and animals at all levels of the food web. However, for animals in the higher trophic levels, transfer of DDT through the water column is probably less important than food-chain transfer, which is discussed in later sections.

The toxicity of DDT in water can be affected by several factors, including temperature and pH. Studies using *Daphnia pulex* indicate that *p,p'*-DDT was significantly more toxic at 20 °C than at 17 °C (Smith et al. 1988). Lower temperatures may also increase toxicity. DDT was seven times more toxic to the scud and twice as toxic to *Daphnia* sp. at 5 °C than at 21 °C (EPA 1975).

Studies along the Yakima River in Washington and the St. Lawrence River in Quebec evaluated the persistence of DDT and its derivatives in water and in other media, as described in the "Field Cases" section, above.

Sediment

Sediments function as the primary sink for DDT and its metabolites (EPA 1975). In general, waterborne DDT concentrations in excess of 1.2 µg/L (water solubility) will either adsorb to or precipitate onto the bottom sediments. The DDT in the sediments is then available for direct contact or ingestion by bottom-dwelling organisms. The DDT can also be redissolved back into the water column whenever the water concentration falls below the saturation point. The per-sistence and degradation of DDT in sediment can be affected by several factors, including pH, organic carbon content, turbidity, and oxygen content.

Sediment quality guidelines developed by Long et al. (1995) were based on comparisons of effects levels for various organisms exposed to DDT-contaminated sediments and sedi-ment characteristics. These guidelines were developed for estuarine and marine sediments but are considered useful as screening levels for freshwater sediments. The distributions of effects data were evaluated to develop two guidelines: (1) an effects-range-low (ERL) guideline, the lower 10th percentile of the effects data for each chemical, and (2) an effects-range-median (ERM) guideline, the 50th percentile of the effects data. These two guidelines can be used to predict three levels of potential toxicity for aquatic organisms exposed to contaminated sediments. Adverse effects should be rare at concentrations below the ERL but may occur between the ERL and the ERM and are very likely above the ERM.

Concentrations below the ERL typically represent conditions where adverse effects would rarely occur. Concentrations between the ERL and the ERM represent conditions in which adverse effects may occur, and concentrations above the ERM represent conditions in which adverse effects are likely to occur. The ERL and ERM values for DDE and DDT are as follows:

Compound	ERL (µg/kg dw)	ERM (µg/kg dw)
p,p'-DDE	2.2	27
Total DDT	1.58	46.1

Oak Ridge National Laboratory in Tennessee developed sediment-screening criteria based on its own research (ORNL 1996). Its sedi-ment quality benchmarks are calculated using equilibrium partitioning with either secondary chronic water quality criteria or the lowest reported chronic values for fish or daphnids and assumes 1 percent organic carbon. The available values are as follows:

Compound	Secondary chronic value (µg/kg dw)	Lowest chronic values (µg/kg dw)	
		Fish	Daphnids
p,p'-DDD	110	16,865	—
DDT	343	19,280	422

The EPA's Office of Solid Waste and Emergency Response has selected benchmark values for use in screening contaminated sediments at sites being managed under the Comprehensive Environmental Response, Compensation, and Liability Act (EPA 1996). The value for DDT is 1.6 µg/kg dw, based on the ERL developed by Long et al. (1995).

Soil

The persistence of DDT in soil can be affected by several factors, including method of application, soil type, soil fertility, type of formulation, topography, climatic conditions, farming practices, soil pH, and organic carbon content (EPA 1975). The amount of time for concentrations of DDT to be reduced by 95 percent in soils ranged from 4 to 30 years, with an average time of 10 years (Edwards 1966).

DDT applied to the surface may be subject to volatilization, but as it becomes bound to the soil, its volatility decreases (Beyer and Gish 1980). DDT mixed with the soil is even less volatile. In addition, because of the low water solubility of DDT, it does not tend to leach through the soil.

Davis (1971) found that the accumulation of DDE and DDT in earthworms tends to decrease as the organic carbon content of the soil increases. The earthworm *Allolobophora caliginosa* accumulated the highest concentrations of DDT in soils with 2 percent organic carbon and showed low accumulation in soils with 21 percent organic carbon. The accumulation of DDE, however, increased as organic carbon increased up to 3.6 percent and then decreased with increasing organic carbon. The cause of the increased DDE accumulation up to 3.6 percent organic carbon was not determined, but it may have been due to other factors such as pH, soil density, and soil moisture, which can influence the feeding activity of the worms. The differences in accumulation between soils with low organic carbon (2 percent to 3.6 per-cent) and those with higher organic carbon (6.5 percent to 21 percent) were statistically significant for both DDE and DDT.

A 20-year study at the Patuxent Wildlife Research Center in Maryland, described above in the "Field Cases" section, examined the persistence of DDT in soil and earthworms.

Biotic Effects

Plants

Aquatic Plants.—Little toxicity information was found concerning toxicity of DDT to aquatic

plants. ORNL (1996) reported a lowest chronic value of 0.3 µg/L for aquatic plants but listed no specific species. EPA (1980, based on the work of Sodergren 1968) also reported a chronic value (for reduced growth and unusual morphology) of 0.3 µg/L in the green alga *Chlorella* sp. Other subacute effects of DDT reported by EPA (1980) include reduced growth in *Anacystis nidulans* at 800 µg/L and in *Scenedesmus quadricauda* at 100 µg/L, and inhibited photosynthesis in *Selenastrum capricornutum* at 3.6 µg/L.

Terrestrial Plants.—No toxicity information was found for effects of DDD, DDE, or DDT on terrestrial plants.

Aquatic Invertebrates

DDT is toxic to most aquatic organisms, and arthropods have been shown to be very sensitive to low levels of DDT. Studies using static and flow-through tests have established median lethal concentrations (48-h) for several freshwater arthropods, as shown in table 15.

Smith et al. (1988) conducted 48-hour static acute toxicity tests for several chemicals found in the Great Lakes. Chemical toxicity was ranked in comparison to that of *p,p'*-DDT, which had been found in fish tissues collected from the Great Lakes. The endpoint selected was an EC50 of immobilization or no movement when prodded. The EC50 for *Daphnia pulex* was 1.1 µg/L.

The marine crustacean *Artemia salina* has gained popularity for use in short-term toxicity testing. The toxicity of various chemicals to this species has been used to predict toxicity to *Daphnia magna* and marine copepods. Sanchez-Fortun et al. (1995) found that the relative order of toxicity of DDE, DDT, and other organochlorine pesticides (i.e., dieldrin and lindane) was the same for *A. salina* as for other crustaceans, but *A. salina* was much more tolerant to DDE and DDT than most other aquatic organisms. This tolerance can range from 7 to 27 times that of other marine decapods. Sanchez-Fortun et al. also found that sensitivity to DDE and DDT increased with increasing age of the *A. salina* larvae. Three-day-old larvae were twice as sensitive as 1-day-old larvae (table 15).

EPA's data base for *p,p'*-DDT contains more than 40 acute toxicity values for various aquatic organisms (EPA 1980). These range from 0.36 µg/L for *Daphnia pulex* to 1,230 µg/L for the planarian *Polycellis felina*. The AQUIRE data base for *p,p'*-DDD and *p,p'*-DDE has several acute and one chronic toxicity value for aquatic organisms (EPA 1984). For *p,p'*-DDD the acute toxicity values range from 1 µg/L for *Bosmina longirostris* to 2,360 µg/L for a flatworm. The chronic toxicity value reported for *p,p'*-DDD is 0.3 µg/L for *Nitocra spinipes*. For *p,p'*-DDE, the acute values range from 0.68 µg/L for the scud (*Gammarus* sp.) to 4,400 µg/L for the fathead minnow (*Pimephales promelas*).

Swartz et al. (1994) evaluated the sediment toxicity and abundance of amphipods at several sites along the Lauritzen Channel and in parts of Richmond Harbor in California. The property adjacent to these portions of the channel was historically used for the formulation and grinding of DDT. Although much of the contaminated sediment was removed, a concentration gradient still exists. Swartz et al. measured toxicity to the amphipod *Eohaustorius estuarius* and the field abundance of amphipods at several sites, and their results are shown in table 15. For comparison purposes, Swartz et al. also tested the toxicity of sediments collected at other sites in the United States. Table 15 includes LC50s for *Hyalella azteca* exposed to sediments from a freshwater stream in Alabama and for *Rhepoxynius abronius* exposed to sediments from the Palos Verdes Shelf, California.

Table 15.—DDD, DDE, and DDT effects on aquatic organisms

[See Appendix II for explanation of abbreviations and technical terms]

Species	Chemical	Concentration (µg/L in surface water, except as noted)	Effects	Reference
Aquatic plants				
Mixed macrophytes	DDT	0.3	Lowest chronic value	ORNL 1996
Aquatic invertebrates				
Daphnia pulex	DDT	0.36	48-h LC50	EPA 1975
	p,p'-DDT	1.1	48-h EC50 - immobilized	Smith et. al. 1988
Daphnia magna	DDT	4.4	48-h LC50	EPA 1975
	DDT-tech	1.1	48-h LC50	Randall et al. 1979
Daphnia sp.	DDT	0.016	Estimated lowest chronic value	ORNL 1996
Bosmina longirostris	*p,p'*-DDD	1	Acute LC50	EPA 1984
Marine crustacean *Artemia salina*	*p,p'*-DDE	159,000	24-h LC50 (1-day-old larvae)	Sanchez-Fortun et al. 1995 (DDE and DDT test solutions prepared by dissolving in DMSO and diluting in water)
		116,000	24-h LC50 (2-day-old larvae)	
		94,270	24-h LC50 (3-day-old larvae)	
	p,p'-DDT	43,010	24-h LC50 (1-day-old larvae)	
		17,040	24-h LC50 (2-day-old larvae)	
		16,400	24-h LC50 (3-day-old larvae)	
Scud (*Gammarus* sp.)	DDT	2.1	48-h LC50	EPA 1975
	p,p'-DDE	0.68	Acute LC50	EPA 1984
Caddisfly	DDT	3.4	48-h LC50	EPA 1975
Mayfly	DDT	0.3	48-h LC50	EPA 1975
Oyster	DDT	7	Reduced shell growth by 50%	EPA 1975
Amphipod *Hyalella azteca*	*p,p'*-DDD	0.19	10-d LC50	Phipps et al. 1995; Hoke et al. 1994
	p,p'-DDE	1.66	10-d LC50	
	p,p'-DDT	0.07	10-d LC50	
	p,p'-DDD	1.08 in pore water	10-d LC50	Hoke et al. 1994
	Total DDT	2,580 mg/kg C in sed.[1]	10-d LC50	Swartz et al. 1994
Amphipod *Eohaustorius estuarius*	Total DDT	2,500 mg/kg C in sed.[1] 300 mg/kg C in sed.[1] 100 mg/kg C in sed.[1]	10-d LC50 Toxic threshold Reduced abundance	Swartz et al. 1994
Amphipod *Rhepoxynius abronius*	Total DDT	1,040 mg/kg C in sed.[1]	10-d LC50	Swartz et al. 1994
Midge *Chironomus tentans*	*p,p'*-DDD	0.18	10-d LC50	Phipps et al. 1995
	p,p'-DDE	3		
	p,p'-DDT	1.23		
Oligochaete *Lumbriculus variegatus*	*p,p'*-DDE	>3.27	10-d LC50	Phipps et al. 1995
Planarian *Polycellis felina*	*p,p'*-DDT	1,230	Acute LC50	EPA 1980
Flatworm	*p,p'*-DDD	2,360	Acute LC50	EPA 1984
Nitocra spinipes	*p,p'*-DDD	0.3	Chronic toxicity	EPA 1984

[1] Milligrams of DDT per kilogram of organic carbon in sediment.

Table 15.—DDD, DDE, and DDT effects on aquatic organisms—Continued

Species	Chemical	Concentration (µg/L in surface water, except as noted)	Effects	Reference
Fish				
Mixed species	DDD	1.69	Lowest chronic value	ORNL 1996
		3.99	Estimated EC20	
		0.61	Population EC20	
	DDT	0.73	Lowest chronic value	
		0.35	Estimated EC20	
		0.008	Sensitive species EC20	
Bluegill sunfish (*Lepomis macrochirus*)	DDT-tech	3.4	96-h LC50	Randall et al. 1979
	DDT	0.2–1.0	96-h LC50	Ellgaard et al. 1977
		0.008	Increased locomotor activity	
	DDT	5.8	96-h LC50 at 7°C	Mayer and Ellersieck 1988
		1.6	96-h LC50 at 29°C	
Carp (*Catla catla*)	DDT	6,800	96-h LC50	Kulshrestha et al. 1986
		3,000–3,500	MATC	
Carp (*Cirrhinus mrigala*)	DDT	6,300	96-h LC50	Kulshrestha et al. 1986
		3,000–3,500	MATC	
Carp (*Labeo rohita*)	DDT	6,400	96-h LC50	Kulshrestha et al. 1986
		3,000–3,500	MATC	
Catfish *Heteropneustes fossilis*	DDT-tech	500	Decrease in white blood cell count	Mustafa and Murad 1984 (DDT dissolved in acetone and diluted with water)
		3,020	72-hr LC50	
		2,950	96-hr LC50	
		3,000	Erratic swimming, jerky movement	
		3,020	72-hr LC50	
		3,550	48-hr LC50	
Cichlid - Tilapia *Oreodhromis spilurus*	DDT	80	96-hr LC50,	Parkinson and Agius 1988
		190	72-hr LC50	
		250	48-hr LC50	
Fathead minnow *(Pimephales promelas)*	*p,p'*-DDE	4,400	Acute LC50	EPA 1984
	Total DDT	1.5 (water only)	Chronic exposure LC50	Jarvinen et al. 1977
		0.9 (+46 µg/g in food)	Chronic exposure LC50	
		0.9	MATC (DDT in water only)	
		0.4	MATC (DDT in water and food)	
		2	Reduced embryo hatchability	
Flounder (*Platichthys flesus*)	DDT	12.5 mg/kg bw in food	Hyperactivity; 20-fold increase in activity	Bengtsson and Larsson 1981
		1 mg/kg (in brain)	Hyperactivity	
Humpback salmon (*Oncorhynchus gorbuscha*)	DDT	1.32	Increased enzymatic activity	Andryushchenko and Khokhryakov 1982
Loach (*Misgurnus anguillicaudatus*)	DDT	350	24-hr LC50	Yang and Sun 1977
Sarotherodon mossambicus	DDT	1	Change in thyroid follicle organization and structure	Shukla and Pandey 1986

Effluent discharges from a DDT manufacturing company have affected both surface water and sediment in the Huntsville Spring Branch of the Indian Creek stream system in Alabama. Hoke et al. (1994) measured the toxicity of DDT and its metabolites to *Hyalella azteca* exposed to surface water.

Terrestrial Invertebrates

Historical studies of terrestrial invertebrates have found that earthworms are much more tolerant of organochlorine pesticides than arthropods (Davis 1971). Cathey (1982) estimated an effective toxic soil concentration of DDE to *Lumbricus*, at 1,500 μg/kg.

Laboratory studies on the uptake and accumulation of DDE and DDT were conducted on two earthworms (*Lumbricus terrestris* and *Allolobophora caliginosa*) that have different feeding patterns (Davis 1971). *L. terrestris* feeds on the soil surface and on vegetation, whereas *A. caliginosa* feeds primarily on soils below the surface. The uptake of DDT from soil by *L. terrestris* indicated that DDT concentrations in worms rose from 0.15 mg/kg (at 1 mg/kg soil residue) to 45 mg/kg (at 64 mg/kg soil residue). The accumulation of DDT increased with increasing soil concentration, and bioconcentration factors ranged from 0.15 to 0.7. DDE was the primary metabolite formed. The proportion of DDE to DDT was approximately 20 percent. The uptake and accumulation of DDT were also measured using apple leaves that had been sprayed with technical grade DDT. Results indicated that accumulation from treated leaves was significantly higher in *L. terrestris* than in *A. caliginosa*. Accumulation from treated leaves was also less than that from treated soil for both species. The differences in accumulation between *L. terrestris* and *A. caliginosa* for different types of DDT application were due to the differences in feeding activity and intake between the two species. When DDT was applied to the soil and cultivated in, A. caliginosa accumulated much higher levels than *L. terrestris* because it ingests relatively more soil and is more active below the surface; but, when DDT was applied to leaves at the soil surface, then *L. terrestris* accumulated higher levels because it is more active at the surface.

The accumulation of DDT in earthworms exposed to contaminated soil in the Rhine delta flood plains indicated that ratios of concentrations in earthworm fat and dry organic matter were independent of octanol-water partition coefficients (K_{ow}) (Hendriks et al. 1995). Earthworms collected from loca-tions along the Rhine delta accumulated DDT at 0.6 times and DDE at 2.3 times the soil concentrations measured at the same loca-tions. Previous studies had indicated that dry weight concentrations of DDE and DDT in field earthworms ranged from 1.8 to 9.2 times the soil concentrations (Ma 1985). When converted to a fat weight basis, this range increased to 2.9 to 15 times the soil concentration. The lower accumulation factors observed in the Rhine delta may have been the result of reduced bioavailability, non-equilibrium conditions, or biotransformation in local populations (Hendriks et al. 1995).

Beyer and Gish (1980) studied the accumulation of DDT in earthworms exposed to treated soils for 11 years. The storage ratios of total DDT in earthworms were calculated for DDE (6), DDD (0.27), *p,p'*-DDT (0.56), and total DDT (5.1). The storage of total DDT in earthworms can lead to harmful effects in higher trophic-level organisms, including birds and mammals. (See "Bioaccumulation".)

Fish

The toxicity and accumulation of DDT in fish are correlated with age, fat content, and body length. Signs of toxicity are similar to those exhibited by insects (Ellgaard et al. 1977). Exposure to lethal concentrations of DDT results in increasing levels of irritability or

excitability followed by muscular spasms, complete loss of equilibrium, convulsions, and eventually death. Toxic effect levels for various species of fish are presented in table 15.

Several studies have evaluated the toxicity and sublethal effects of DDT and its meta-bolites to various fish species (table 15). Ellgaard et al. (1977) found that exposure to DDT concentrations as low as 0.008 µg/L can affect locomotor rates of bluegill sunfish (*Lepomis macrochirus*). Behavioral changes, including erratic swimming, fast jerky movement, and convulsions, were observed in catfish (*Heteropneustes fossilis*) exposed to technical-grade DDT at concentrations greater than 3,000 µg/L (Mustafa and Murad 1984). Changes in thyroid follicle organization and structure were observed at DDT concentra-tions as low as 1.0 µg/L in *Sarotherodon mossambicus* (Shukla and Pandey 1986).

Hyperactivity and abnormal diurnal activity were observed in flounders (*Platichthys flesus*) that were force-fed DDT (Bengtsson and Larsson 1981). Dosages of DDT at 12.5 mg/kg bw resulted in a 20 percent increase in swimming activity, and extractable fat resi-dues in the brain greater than 1 mg/kg resulted in hyperactivity. Andryushchenko and Khokhryakov (1982) evaluated enzymatic changes in humpback (pink) salmon (*Oncorhynchus gorbuscha*) and found that concentrations of DDT at 1.32 µg/L resulted in increases of cytochrome P-450 by 30 percent and cytochrome b5 by 21 percent. In addition, benzpyrenhydroxylase activity was increased threefold.

Mayer and Ellersieck (1988) found a negative correlation between temperature and toxicity for DDD, DDT, dimethrin, methoxychlor, pyrethrins, and pyrethroids. This is opposite of the relationship observed for most other organic chemicals. The differences in toxicity due to temperature have been attributed to changes in respiration rates and chemical absorption, detoxification, and excretion.

Yang and Sun (1977) studied toxicity and rates of absorption of DDT in leaches (*Misgurnus anguillicaudatus*) and found that 96.5 percent of a predetermined concentration of DDT present in water was absorbed within 24 hours. The high rate of absorption is primarily due to the lipophilicity of DDT.

Jarvinen et al. (1977) tested the partial chronic toxicity of DDT to fathead minnows (*Pimephales promelas*). In separate tests, they administered the DDT in diet alone, in water alone, and in both diet and water. Fish fed DDT in the diet had lower survival rates than those fed clean food (table 15). High mortality was observed at two stages: (1) juveniles 45 to 73 days old and (2) spawning male fish. In addition, embryo residue levels and larval mortality rates for offspring of parent fish that were exposed to DDT in both water and food were two times higher than those for offspring of fish that were exposed to DDT only in the water. Tissue residues of DDT in adults were also higher for fish exposed to DDT in water alone than for fish exposed to DDT in diet alone, but results for fish exposed to DDT in both diet and water indicated that residue levels were additive based on the single-exposure studies. An equilibrium between tissue residues and concentrations in food was reached within 56 days. Bioconcentration factors were calculated for diet (1.2) and water (100,000).

In lake trout (*Salvelinus namaycush*) stocked in Cayuga Lake, New York, tissue residues of *p,p'*-DDE increased significantly with both the age of the fish and the fat content (Gutenmann et al. 1992). Fish collected in 1978 averaged 3.06 mg/kg dw for 6-year-old fish and 10 mg/kg dw for 12-year-old fish. Similarly, fish collected in 1991 averaged 0.89 mg/kg dw for 6-year old fish and 1.9 mg/kg dw for 12-year-old fish. In addition, the DDT concentrations in fish tissue showed a marked decrease in both the 6- and 12-year-old fish in the intervening 13 years.

Similar studies at another lake in New York found that residues of *p,p'*-DDE in lake trout were positively correlated to the length of fish (Young et al. 1994). Length accounted for 81 percent of the variation in residue levels. Length can also be used as an indicator of age and, hence, these results agree with those of Gutenmann et al. (1992).

Contrary to the results of the lake trout studies, Larsson et al. (1993) found that total DDT residue levels in northern pike (*Esox lucius*) taken from a eutrophic lake in southern Scandinavia were negatively correlated with age and with muscle or fat content. The decrease with increasing age was most pro-nounced in female pike, and this result was attributed to the seasonal use and elimination of lipids and, hence, of lipophilic pollutants during reproduction and release of eggs. In eggs and ovaries (roe), both the fat content and the contaminant levels were 10 times higher than in muscle. Male pike contained higher levels of contaminants than females
but have lower elimination via gonadal products. Germinal tissue can account for up to 15 percent of the body weight in females but only accounts for approximately 2 percent in males. Male germinal tissue also contains less fat than the ovaries in females. In addition, the largest fat deposits in pike are found in the germinal tissues; their muscle tissue has lower levels of fat (0.6 to 0.8 per-cent) than most other fish, including salmonids. These results differ from those
of the trout studies primarily because trout and other salmonids deposit more fat than pike do in the muscle, adipose, and visceral tissues, and their fat content increases with age. Other fish also tend to use fat deposits as an energy reserve. Pike are more lean, their percent fat content in muscle and germinal tissue is relatively constant throughout life, and they do not use fat deposits as energy reserves. Instead, they catabolize ordinary tissue when fasting. Overall, the uptake and elimination of persistent pollutants such as DDT can vary not only within a species because of sex, age, and size, but also between species because of differences in fat deposition.

Cullen and Connell (1992) studied whole-body residues of total DDT in fish collected from three rivers in New South Wales, Australia. The rivers selected had large plantations and a history of heavy use of chlorohydrocarbon pesticides. Fish species collected included whiting (*Sillago ciliata*), bream (*Acanthopagrus australis*), mullet (*Mugil cephalus*), and carp (*Cyprinus carpo*). At some point during their life history, whiting, bream, and mullet migrate from upstream impacted areas to downstream estuarine or open-sea areas with lower levels of contamination. The tissue levels in these fish increased with age in upstream juveniles but decreased after maturation and migration downstream. The levels of total DDT increased linearly with age for fish (carp) that did not migrate. The effects of migration included both exposure to lower environmental concentrations and the metabolism of fat reserves during migration and spawning. In addition, Cullen and Connell found that fish with higher fat content (mullet) contained significantly higher total DDT residues than fish with a lower fat content (bream and whiting), but variations occurred based on differences in movement patterns and exposure history.

Along the Yakima River of Washington, Johnson et al. (1988) studied the persistence of DDT and its metabolites in fish and in water and sediment, as described above in the "Field Cases" section.

Amphibians/Reptiles

Few studies were found that describe DDT's effects on amphibians and reptiles. Toxic effects can include uncoordinated behavior, loss of equilibrium, restricted development, weight loss, and death (Russell et al. 1995). Spring peepers (*Pseudacris crucifer*) collected from an area of historic DDT application were analyzed

for DDD, DDE, and DDT. All had elevated tissue concentrations (mean concen-trations in μg/kg ww: *p,p'*-DDD, 26.5; *p,p'*-DDE, 1,001; *p,p'*-DDT, 161). The high concentrations indicated that DDT application in the area may have accounted for local extinctions of three other species of frogs and toads (Russell et al. 1995).

Bishop et al. (1994) studied concentrations of DDE in the eggs of the common snapping turtle (*Chelydra serpentina serpentina*) to determine if higher concentrations of DDE were found in eggs produced by larger, older turtles or those with the largest clutch size or mass. The results of this study showed no correlation between the age or size of the female turtle and the amount of DDE found in the eggs. Relationships were previously found between body size of turtles and the concentrations of lipophilic compounds in fat or liver. The differences of DDE found in different clutches may simply reflect individual preferences in food and feeding locations. In addition, the lipids used for egg production may be derived from daily dietary intake shortly before egg production rather than from stored lipids in the female.

Bishop et al. (1995a) also found that concentrations of DDE varied widely in common snapping turtle eggs collected from the same clutch. The first five eggs laid in the nest contained the highest concentrations of DDE on both a wet-weight and a lipid-weight basis. The first-laid eggs of the snapping turtles tended to have greater lipid content and consequently greater organochlorine content than later eggs. This trend is opposite from what has been observed in some birds (e.g., herring gulls), in which eggs with the highest lipid and organochlorine content were those laid later.

Birds

The toxicity and accumulation of DDT and its metabolites are of primary concern in birds. During the early 1970's, DDT and its metabolites were probably present in the tissues of essentially all wild birds in the world (Fleming et al. 1983). Since then, however, many studies have shown that DDE residue levels in birds have been decreasing in many parts of the United States (Mora 1995). These chemicals can accumulate in fat after even brief, low-level exposures. In general, birds that feed on fish or other birds have greater tissue residues than those that feed on vegetation or seeds, and DDE is more common than either DDT or DDD in bird tissues (Stickel 1973, Blus 1996). Other adverse effects associated with DDT poisoning in birds include reproductive impairment, reduced fledging success, and eggshell thinning. Toxic effect levels for various types of birds are presented in table 16.

The storage of DDT in various tissues can be a function of the exposure concentrations (Stickel 1973). Continuous exposure to sublethal concentrations tends to result in tissue residues that are directly correlated to each other. The balance of tissue residues is broken when the exposure is to lethal concentrations or when stored tissue residues are metabolized and released back into the system at lethal concentrations. The residue level in the brain has been shown to be the best criterion for establishing that total DDT was the cause of death, and lethal levels of brain residues are similar for many different species of birds. Residue levels in the liver are a better indication of recent exposure and can be correlated either to an environmental dose or to metabolism of stored residues. Whole-body residue levels indicate the storage reserve and can be used to estimate the potential for adverse effects from metabolism to lethal levels or from normal metabolism and excretion (Stickel 1973).

Through their review of earlier studies, Noble and Elliot (1990) derived critical levels of DDE, resulting in acute toxicity, in the brains, livers, and eggs of several raptors. Critical levels in the brain and liver were 250 and 100 mg/kg

Table 16.—DDD, DDE, and DDT impacts to birds

[See Appendix II for explanation of abbreviations and technical terms]

Species	Chemical species	Concentration (mg/kg)[1]	Where measured	Effects	Reference
Raptors					
American kestrel	DDE	10 ww	Egg	Minimum critical levels (lowest levels at which productivity is affected), determined by review of available literature	Noble and Elliot 1990
Bald eagle		6 ww			
Golden eagle		10 ww			
Falcons[2]		10 ww			
Hawks[3]		10 ww			
Merlin		5 ww			
Northern harrier		10 ww			
Owls[4]		10 ww			
Osprey		4 ww			
Prairie falcon		1.2 ww			
Mixed species		250 ww	Brain		
		100 ww	Liver		
American kestrel	DDE	3 dw	Diet	13% eggshell thinning; reduced pipping	Lincer 1992
Bald eagle (*Haliaeetus leucocephalus*)	DDE	3.3 ww	Egg	8.8% thinner than pre-1947 eggs from Southern CA and Baja	Grubb et al. 1990
		3–5 ww		Depressed productivity	Wiemeyer et al. 1984
		5 ww		10% eggshell thinning	
		15 ww		No productivity	
		3.6–6.3 ww		50% reduction in productivity	Wiemeyer et al. 1993
		>6.3 ww		75% reduction in productivity	
		16 ww		15% eggshell thinning	
Osprey (*Pandion haliaetus*)	DDE	4 ww	Egg	15% eggshell thinning	Noble and Elliot 1990
		2 ww		10% eggshell thinning	Wiemeyer et al. 1988
		4.2 ww		15% eggshell thinning	
		8.7 ww		20% eggshell thinning	
Peregrine falcon (*Falco peregrinus*)	DDE	15 ww	Egg	Depressed productivity	Peakall et al. 1975
Terrestrial birds					
Bengalese finch (*Lonchrua striata*)	DDE	4 ww	Diet	Reduced fledging success	Jeffries 1971
	DDT	8 dw			
Bobwhite quai (*Colinus virginianus*)	DDE	825 dw	Diet	5-d LC50	Hill et al. 1975
	DDT	611 dw		5-d LC50	
	Total DDT	25–200 dw	Diet	3.1 mg/kg in brain; no effect	Hill et al. 1971
		400 dw		Weight loss	
		800 dw		7.5 mg/kg in brain; tail tremors, irregular head carriage	
		1,600 dw		Stumbling gait, tail tremors, head bobbing, loss of equilibrium, death	
		1,170-1,610 dw		5-d LC50	

Table 16.—DDD, DDE, and DDT impacts to birds—Continued

Species	Chemical species	Concentration (mg/kg)[1]	Where measured	Effects	Reference
Terrestrial birds—Continued					
Blue jay (*Cyanocitta cristata*)	Total DDT	415 dw	Diet	5-d LC50	Hill et al. 1971
	DDT	611 dw	Diet	5-d LC50	Heath et al. 1972
Brown-headed cowbird (*Molothrus ater*)	DDE	1,500 dw	Diet	300–400 mg/kg in brain residue; increased likelihood of death	Stickel et al. 1984
California quail (*Callipepla californica*)	DDT	595 bw	Oral dose	LD50 (single dose)	Hudson et al. 1984
Cardinal (*Richmondena cardinalis*)	Total DDT	535 dw	Diet	5-d LC50	Hill et al. 1971
Common grackle (*Quiscalus quiscula*)	DDE	1,500 dw	Diet	300–400 mg/kg in brain residue; increased likelihood of death	Stickel et al. 1984
Coturnix quail (*Coturnix japonica*)	DDE	1,355 dw	Diet	5-d LC50	Hill et al. 1975
	DDT	416 dw		5-d LC50	Hill and Camardese 1986
		568 dw		5-d LC50	Hill et al. 1975; Heath et al. 1972
		841 bw	Oral dose	LD50 (single dose)	Hudson et al. 1984
House sparrow (*Passer domesticus*)	Total DDT	415 dw	Diet	5-d LC50	Hill et al. 1971
Red-winged blackbird (*Agelaius phoeniceus*)	DDE	1,500 dw	Diet	300–400 mg/kg in brain residue; increased likelihood of death	Stickel et al. 1984
Ring-necked pheasant (*Phasianus colchicus*)	DDE	829 dw	Diet	5-d LC50	Hill et al. 1975
	DDT	311 dw		5-d LC50	
		1,334 bw	Oral dose	LD50 (single dose)	Hudson et al. 1984
Rock dove (*Columba livia*)	DDT	>4,000 bw	Oral dose	LD50 (single dose)	Hudson et al. 1984
Starling (*Sturnus vulgaris*)	DDE	1,500 dw	Diet	300–400 mg/kg in brain residue; increased likelihood of death	Stickel et al. 1984
White-throated sparrow (*Zonotrichia albicollis*)	DDE	5–25 ww	Diet(?)	Delayed development of migratory condition	Mahoney 1975
Waterfowl—Insectivores					
Black duck (*Anas rubripes*)	DDE	10 dw	Diet	Increased egg residues; 20% eggshell thinning (over 2 years)	Longcore and Stendell 1977
				Egg residues 46.3 mg/kg; 10% shell cracking	Longcore et al. 1971
		30 dw		Egg residues 144 mg/kg; 21% shell cracking	
		46.3 ww	Egg	Eggshell thinning of 18–29%	
		144 ww		Eggshell thinning of 24–38%	
Clapper rail (*Rallus longirostris*)	*p,p'*-DDT	1,612 dw	Diet	5-d LC50 (male)	Van Velzen and Kreitzer 1975
		1,896 dw		5-d LC50 (female)	
		30 ww	Brain	Lower lethal limit diagnostic of DDT-related death	

Table 16.—DDD, DDE, and DDT impacts to birds—Continued

Species	Chemical species	Concentration (mg/kg)[1]	Where measured	Effects	Reference
Waterfowl—Insectivores—Continued					
Common goldeneye (*Bucephala clangula*)	DDE	0.52 ww	Egg	Egg breakage; 15.4% eggshell thinning	Zicus et al. 1988
Mallard (*Anas platyrhynchos*)	DDT-tech	200 dw	Diet (12-week exposure)	95% lethality; 20% eggshell thinning	Davison and Sell 1974
	p,p'-DDT	200 dw		100% lethality after 343 d	
	p,p'-DDT	1,202 dw	Diet	20-d LC50 (5-d-old ducklings)	Friend and Trainer 1971
		1,622 dw		20-d LC50 (30-d-old ducklings)	
		1,419 dw		20-d LC50 (adults)	
	DDT	>2,240 bw	Oral dose	LD50 (single dose)	Hudson et al. 1984
	DDT	1,869 dw	Diet	5-d LC50	Hill et al. 1975
	DDE	3,572 dw	Diet	5-d LC50	
Waterfowl—Omnivores					
Black-crowned night-heron (*Nycticorax nycticorax*)	DDE	<1 ww	Egg	6.5% eggshell thinning	Findholt and Trost 1985
		1.01–4 ww		5.1% eggshell thinning	
		4.01–8 ww		10.2% eggshell thinning	
		>8 ww		15.6% eggshell thinning	
		8 ww		Reduced clutch size, decreased productivity, egg breakage	Henny et al. 1984, 1985
		8.62 ww		8–13% thinner than pre-1947 eggs	Ohlendorf and Marois 1990
		11–12 ww		36–39% hatching success; 14–17% eggshell thinning	Price 1977
		8–12 ww		27–58% decrease in nesting success	Blus 1984
		12 ww		Critical level for reproductive success based on field studies	
		25–50 ww		Total reproductive failure	
		36 ww		18% thinning based on regression analysis	
		54 ww		20% thinning; critical level for reproductive success based on regression analysis	
Great egret (*Casmerodius aalbus*)	DDE	24 ww	Egg	8–13% thinner than pre-1947 eggs	Ohlendorf and Marois 1990
Green-backed heron (*Butorides striatus*)	DDE	5–10 ww	Egg	Reduced hatching success	White et al. 1988
Red-necked grebe (*Podiceps grisegena*)	DDE	6.68 ww	Egg	Low egg viability; 6.5% eggshell thinning; reduced fledging success	De Smet 1987
Sandhill crane (*Grus canadensis*)	DDT	>1,200 bw	Single oral dose	LD50	Hudson et al. 1984
Snowy egret (*Egretta thula*)	DDE	5 ww	Egg	Reduced clutch size, decreased productivity, egg breakage	Henny et al. 1985

Table 16.—DDD, DDE, and DDT impacts to birds—Continued

Species	Chemical species	Concentration (mg/kg)[1]	Where measured	Effects	Reference
Waterfowl—Omnivores—Continued					
Western grebe (*Aechmophorus occidentalis*)	DDE	1 ww	Egg	1% thinning	Boellstorff et al. 1985
		5.4 ww		2.3% eggshell thinning; reduced productivity	Lindvall and Low 1980
White-face ibis (*Plegadis chihi*)	DDE	3 ww	Egg	Reduced clutch size, decreased productivity, egg breakage	Henny et al. 1985
		0.94 ww		3.2% eggshell thinning	King et al. 1980
		0.25 ww		4.5% eggshell thinning	
		4–8 ww		15% eggshell thinning	
		8–16 ww		17.4% eggshell thinning	Henny and Herron 1989
		16–20 ww		27.8% eggshell thinning	
Waterfowl—Piscivores					
American white pelican (*Pelicanus erythrorhynchos*)	DDE	2 ww	Egg	10–15% thinning in eggs from CA	Boellstorff et al. 1985
Black skimmer (*Rhyncops niger*)	DDE	3.2 ww	Egg	Decreased hatching and fledging success	Custer and Mitchell 1987
		3.4 ww		5% thinning, but no adverse effect on reproductive success	King et al. 1991
Brown pelican (*Pelicanus occidentalis*)	DDE	1 ww	Egg	5–10% shell thinning (FL, SC)	Blus et al. 1974, 1979
		2 ww		11% eggshell thinning (FL)	
		3 ww		16% eggshell thinning (SC)	
		5 ww		17% eggshell thinning (SC)	
		3 ww		Reduced productivity	King et al. 1985
		3.2 ww		11% thinner than normal	King et al. 1977
		2.6–3.0 ww		29–40% decrease in nesting success	Blus 1984
		3 ww		Critical level for reproductive success based on field studies	
		>3.7 ww		Total reproductive failure	
		5 ww		18% thinning based on regression analysis	
		8 ww		20% eggshell thinning	
		3 ww		18% eggshell thinning (BC)	Jehl 1973
		8 ww		26% eggshell thinning (BC)	
		25 ww		47% eggshell thinning (BC)	
		66 ww		46% eggshell thinning (BC)	
		59 ww		44% eggshell thinning (CA)	Risebrough 1972
Caspian tern (*Sterna caspia*)	DDE	9.3 ww	Egg	22% hatching failure; 4.6% died in hatching	Ohlendorf et al. 1985
Common tern (*Sterna hirundo*)	DDE	6.67 ww	Egg	17% thinning; hatching failure; embryo mortality	Fox 1976
Double crested cormorant	DDE	10 ww	Egg	20% eggshell thinning	Pearce et al. 1979

Table 16.—DDD, DDE, and DDT impacts to birds—Continued

Species	Chemical species	Concentration (mg/kg)[1]	Where measured	Effects	Reference
Waterfowl—Piscivores—Continued					
Elegant tern (*Sterna elegans*)	DDE	3.79 ww	Egg	Chick mortality during hatching	Ohlendorf et al. 1985
Forster's tern	DDE	1.6 ww	Egg	7% thinning, but no adverse effect on reproductive success	King et al. 1991
Hooded merganser (*Lophodytes cucullatus*)	DDE	0.62 ww	Egg	9.6% eggshell thinning; egg breakage	Zicus et al. 1988
Leach's storm petrel	DDE	12 ww	Egg	12% eggshell thinning	Noble and Elliot 1990
Northern gannet (*Sula bassanus*)	DDE	18.5 ww	Egg	17% eggshell thinning; low reproductive success	Elliott et al. 1988

[1] Weight basis: dw, dry weight; ww, wet weight; bw, dosage relative to body weight.
[2] Falcons include: peregrine falcon and gyrfalcon.
[3] Hawks include: Cooper's hawk, ferruginous hawk, northern goshawk, red-shouldered hawk, red-tailed hawk, rough-legged hawk, sharp-shinned hawk, and Swainson's hawk.
[4] Owls include: burrowing owl, great grey owl, great horned owl, long-eared owl, short-eared owl, and snowy owl.

ww, respectively. Critical levels of DDE in eggs were generally 10 mg/kg, but lower concentrations were found for prairie falcons (1.2 mg/kg), osprey (4 mg/kg), merlin (5 mg/ kg), and bald eagle (6 mg/kg).

Tissue concentrations in terrestrial birds have resulted in a number of adverse effects. Many workers have studied the toxicity of DDT to terrestrial birds. Table 16 shows a wide range of dietary LC50s and dose LD50s from Hill et al. (1975), Hill and Camardese (1986), and Hudson et al. (1984), among others.

Hill et al. (1971) studied the correlation between dietary exposure to DDT, brain residues of total DDT, and lethality for several species of birds; in particular, they correlated brain residues in bobwhite quail (*Colinus virginianus*) to dietary concentrations of DDT and to various toxic effects. Dietary concentrations of 400 mg/kg resulted in weight loss in the majority of the test birds
but no other signs of toxicity. Dietary concentrations of 800 mg/kg resulted in tail tremors and irregularities in head carriage. The brain residues of total DDT at this dose ranged from 7.5 to 30 mg/kg. Birds fed 1,600 mg/kg DDT had tail tremors, stumbling gait, head bobbing, loss of equilibrium, and death within 100 hours of dosing. Although other research had suggested a critical brain residue level of 30 mg/kg DDD+DDT, indicative of serious danger or death (Stickel et al. 1966), the results of this study indicate that 20 mg/kg in the brain would be a more appropriate critical level, especially as signs of intoxication were observed at levels as low as 7.5 mg/kg.

Mahoney (1975) conducted studies on the effects of DDT on migratory behavior. He found that DDT tissue concentrations of 5 to 25 mg/kg ww delayed the development of the migratory condition in white-throated sparrows (*Zonotrichia albicollis*).

Stickel et al. (1984) measured lethal brain residues in four wild birds exposed to DDE in the diet. In all species, brain residues of 300 to 400 mg/kg resulted in an increased likelihood of death. Stickel et al. (1984) also measured the loss rate of DDE in grackles (*Quiscalus quiscula*). Test animals were fed 1,500 mg/kg DDE in the diet for 7 days then given untreated food. A loss rate of 0.3 percent per

day was calculated at the end of 112 days. The estimated half-life for DDE was 229 days.

Mallards released to three experimental stations in Canada were evaluated for uptake of DDT as compared to controls to determine the feasibility of using the mallard as a sentinel species (Gebauer and Weseloh 1993). Two of the sites—a confined waste disposal facility and a sewage lagoon—were known to have sediment contaminant levels that exceeded guidelines of the Ontario Ministry of Environment (Persaud et al. 1993). The third location was a relatively clean natural marsh. All three sites were important resting and feeding areas for migratory and resident waterfowl. Mallards from each location were collected at specified intervals and were analyzed for DDE residues in breast muscle. Concentrations of DDE in muscle tissue prior to release were 3.3 μg/kg ww. For the birds released at the confined waste disposal facility, residues averaged 8.5 μg/kg ww after 10 days and 27.9 μg/kg ww after 115 days. Residues in birds released at the sewage lagoon had increased to 13.8 μg/kg ww after 10 days, 58.4 after 70-days, and 216.9 after 112 days. The tissue residues in birds collected from the sewage lagoon were significantly higher than the levels at the time of release at both 70 and 112 days. It was estimated from these results that the mean rate of accumulation of DDE at 30 days ranged from 0.09 micrograms per kilogram per day (μg/kg/d) at the waste disposal facility to 0.99 μg/kg/d at the sewage lagoon. The accumulation rate at the sewage lagoon after 112 days was 1.9 μg/kg/d and had not reached equilibrium.

Friend and Trainer (1971) found the toxicity of *p,p'*-DDT to be age dependent in mallards (*Anas platyrhynchos*), with 30-day-old ducklings having a higher LC50 than either adult birds or younger ducklings (table 16). In addition, the onset of mortality and the mean elapsed time until death were age related and were earlier for younger birds, indicating a dose-dependent mortality relationship. Moreover, the body weights of surviving birds were less than those of controls for both groups of ducklings, although the difference was not statistically significant. Brain residues of DDT were also measured in birds dying within each test group, and no correlation was found between the residue levels at time of death and the initial DDT dose. In addition, adult birds that died of DDT contained brain residues of DDD, DDE, and DDT that were 6 to 17 times greater than those in adult birds that survived the test. The average ratio of DDE, DDD, and DDT in the surviving birds was 2:5:3 (DDE: DDD: DDT).

Van Velzen and Kreitzer (1975) found that the toxicity of *p,p'*-DDT to clapper rails (*Rallus longirostris*) varies by sex. As shown in table 16, the 5-day LC50 was notably higher for females than for males. In addition, brain residues of DDD, DDE, DDT, and DDD+DDT were significantly higher in birds that died than in those that survived. Van Velzen and Kreitzer established a lower lethal limit of 30 mg/kg of DDD+DDT in the brain for diagnosing DDT as the cause of death. Research by Stickel et al. (1966) and Stickel and Stickel (1970) has shown that the lower limit of the lethal range for DDE is 250 mg/kg in the brain, but for the combined residues of DDE and DDT, 30 mg/kg in the brain is the practical separation point between birds that live and those that die in laboratory studies. Similarly, the combined residues of DDD and DDT in the brain are lethal at 20–30 mg/kg (Bernard 1963, Stickel et al. 1966, Hill et al. 1971).

Several investigators have studied DDT toxicity and accumulation in herons (Ohlendorf et al. 1981, Henny et al. 1984, 1985; Findholt and Trost 1985; Henny and Blus 1986; White et al. 1988; Custer and Custer 1995; Hothem et al. 1995). These studies indicate that herons are sensitive to DDT and that high DDT levels can be found in the tissues of birds throughout the United States.

Custer and Custer (1995) found that black-crowned night-heron chicks exposed to environmental concentrations of DDT have varying accumulation rates for DDT and its metabolites. Mean accumulation rates of p,p'-DDD, *p,p'*-DDE, and *p,p'*-DDT were 0.48, 42.9, and 0.2 mg/ kg/d, respectively. Ohlendorf et al. (1981) found that the level of DDE residues in the brain of a black-crowned night-heron from Nevada (230 mg/kg ww) was high enough to cause severe impairment, but death was probably not due to poisoning. Similarly, Call et al. (1976) found brain residues of 246 mg/kg and liver residues of 570 mg/kg ww of DDE in a great blue heron found dead in South Dakota. A great blue heron from North Carolina, which had 20 mg/kg ww of DDT in its brain, almost certainly died of DDT poisoning (Ohlendorf et al. 1981).

Adverse reproductive effects resulting from DDT poisoning in birds include reproductive impairment, reduced fledging success, and eggshell thinning. Through review of laboratory studies conducted with DDD, DDE, and DDT, Stickel (1973) observed that DDD did not produce significant eggshell thinning in mallards, but DDE produced significant eggshell thinning in three major groups of birds: the orders Strigiformes (screech owls, *Otus asio*), Falconiformes (American kestrels, *Falco sparverius*), and Anseriformes (mallards and black ducks, *Anas rubripes*). DDT resulted in eggshell thinning only after longer expo-sure duration, by which time some of the DDT may have been metabolized into DDE.

Several researchers have shown that while eggshell thinning of 5–7 percent is statistically significant, it is probably not biologically significant, and field studies have shown that an average thinning of 10 percent is seldom associated with egg breakage or population decline (King et al. 1980, 1991; Anderson et al. 1969; Blus 1970, 1982). In addition, the amount of thinning resulting from each incremental increase in DDE is greater at lower residue levels (Blus 1996). For example, 1 mg DDE/kg may result in 5–10 percent thinning, whereas 59 mg/kg results in 44 percent thinning in brown pelicans.

Most studies report eggshell thinning as the indicator of reproductive problems, but other factors are also important indicators, such as egg residue levels compared to the percentage of chick survival, the number of young pro-duced per nest, and eggshell strength (Blus 1996). Egg residue levels of DDD, DDE, and DDT and associated thinning or other repro-ductive effects are summarized in table 16.

Wiemeyer et al. (1984, 1993) studied repro-ductive activity in bald eagles (*Haliaeetus leucocephalus*) exposed to DDT. Eggshells were thinned by 10 percent at DDT levels of 5 mg/ kg (Wiemeyer et al. 1984) and by 15 percent at 16 mg/kg (Wiemeyer et al. 1993). The production of young was normal when eggs contained less than 3.6 mg/kg DDE but was reduced by nearly 50 percent when concentrations ranged between 3.6 and 6.3 mg/kg. When concentrations exceeded 6.3 mg/kg, reproduction was reduced by another 50 percent.

Similar results were observed by Grubb et al. (1990) for bald eagles in Arizona. Samples of eggshells collected from 1977 to 1985 had a mean DDE concentration of 3.3 mg/kg and were 8.8 percent thinner than pre-1947 eggs from southern California. However, pro-ductivity over the same period increased slightly, from an average of 0.8 young per occupied territory in 1975 to 1.13 young per occupied territory from 1985 to 1986. They speculated that although many factors other than decreasing environmental contamination could be contributing to the increase in population, the levels of DDE and eggshell thinning did not seem to be adversely affecting reproductive success.

Nesting failures for bald eagles in Oregon were associated with many factors, including exposure to DDT. Anthony et al. (1994)

surveyed 89 failed nests from 1980 to 1987 to determine the probable cause of failure. Nest condition, the presence of new nesting material, prey remains, unhatched eggs, and remains of dead young were evaluated. Based on results by Wiemeyer et al. (1984), nesting failures were attributed to DDE if egg concen-trations were >10 mg/kg ww or if eggshell thinning was >15 percent. DDE was attri-buted as the major cause of nesting failure in 32 percent of the nests.

Eggshells of American kestrels exposed to 3 mg/kg DDE in the diet were 13 percent thinner than those of controls, but none of the eggs broke (Lincer 1992). Of the eggs that were artificially incubated, only 30 percent pipped, but those that pipped generally hatched.

Clark et al. (1995) collected eggs of northern harriers (*Circus cyaneus*), great-tailed grackles (*Quiscalus mexicanus*), and black-necked stilts (*Himantopus mexicanus*) from several locations throughout California and Texas. Egg residue levels of DDT averaged 2.2 mg/kg ww for the grackle, 2.79 for the stilt, and 4.07 for the harrier. Although these concentrations were as high as the concentrations that had been reported as resulting in impaired repro-duction in bald eagles, black-crowned night-herons, and white-faced ibises, the researchers did not report observing any adverse effects in the birds from which eggs were collected.

For terrestrial birds, Jefferies (1971) found that tissue concentrations of 4 mg DDE/kg ww and 8 mg DDT/kg dw resulted in reduced fledging success of Bengalese finches (*Lonchura striata*).

Among the waterfowl, mallards have been studied extensively because even low concentrations of DDT can cause eggshell thinning in this species. Davison and Sell (1974) found that mallards that survived having 200 mg/kg of technical-grade DDT in their diet for 12 weeks produced eggs that had shells 20 percent thinner than normal and produced hatchlings with tremors.

Black ducks fed dietary concentrations of 10 mg DDE/kg dw for two breeding seasons (June through November in two successive years) and then clean food for 2 years continued to exhibit eggshell thinning at the end of the 2-year cleanup period (Longcore and Stendell 1977). Eggs collected during the two breeding seasons and the first year of clean diet were significantly thinner than controls (average 20 percent thinner). Eggs collected during the second year of clean diet were 10 percent thinner than controls. Residues in eggs increased significantly during the years of treatment (up to 64.9 mg/kg ww), then decreased during years of clean feed (down to 6.2 mg/kg ww). In addition, exposed hens continued to produce significantly fewer young than did controls after the second year of clean diet.

White et al. (1988) found that green-backed herons (*Butorides striatus*) and anhingas (*Anhinga anhinga*) had decreased hatching success and eggshell thinning in areas that had not been treated with DDT for more than 13 years. Concentrations of 5.1–10 mg/kg ww in eggs of green-backed herons were the threshold for reduced hatching success.

Studies of black-crowned night-herons in Idaho (Findholt and Trost 1985) indicated that relatively low concentrations of DDE resulted in eggshell thinning (table 16). Henny et al. (1984) studied black-crowned night-heron populations in Washington, Oregon, and Nevada between 1978 and 1980 to determine contaminant patterns and eggshell thinning. Eggs with residues greater than 8 mg/kg ww correlated with decreased clutch size, lower productivity, and an increased incidence of cracked eggs. Henny et al. (1982, 1984) also found that, with the exception of two locations along the Columbia River, there was a strong north-south DDE contaminant gradient. DDE residues in southern colonies were much

higher than those in northern colonies. This trend may contribute to increased exposure at wintering grounds to migrating waterfowl.

Henny and Blus (1986) found similar trends in DDE contamination for black-crowned night-herons from Idaho and Oregon that wintered in coastal Mexico, compared to those from Nevada that wintered in southern California. On a more local basis, Hothem et al. (1995) noted the same type of trend when they compared black-crowned night-herons within San Francisco Bay to those in the San Joaquin Valley.

Mora et al. (1987) evaluated the potential importance of the uptake of DDT and its metabolites from wintering grounds for northern pintail and gadwall, which migrate from northern California to southern California or Mexico. These results confirmed those for other pintails and mallards migrating through California and for black ducks that migrate to Texas. Pintails sampled from the Salton Sea, California, had higher whole-body DDE residues than those from the Lower Klamath National Wildlife Refuge, California, again suggesting a north-south organochlorine pesticide gradient in California. However, residues decreased in resident waterfowl (black-bellied and fulvous whistling ducks) collected farther south in Mexico. The researchers hypothesized that the increased levels found at the Salton Sea may have resulted from past heavy use of DDT, from current illegal use of DDT, or from DDE and DDT impurities in dicofol. They further speculated that the lower residues found in waterfowl from Mexico may have been due to variability in uptake between the species sampled and do not necessarily indicate lower levels of organochlorine pesticides in the Mexican wintering grounds.

Eggshell thinning in various species of grebes and northern gannets has also been reported for DDE. Eggshell thinning of 2.3 percent and reduced productivity was reported in western grebes (*Aechmophorus occidentalis*) from Utah with egg DDE concentrations of 5.4 mg/kg ww (Lindvall and Low 1980). Red-necked grebes (*Podiceps grisegena*) collected from Manitoba exhibited low egg viability, egg-shell thinning of 6.5 percent, and reduced fledging success at egg DDE concentrations of 6.68 mg/kg ww (De Smet 1987). Repro-ductive impairment, low reproductive success, and eggshell thinning (17 percent) in northern gannets (*Sula bassanus*) were associated with egg DDE concentrations of 18.5 mg/kg ww (Elliot et al. 1988 as cited in Forsyth et al. 1994).

Zicus et al. (1988) collected eggs from nests of hooded merganser (*Lophodytes cucullatus*) and common goldeneye (*Bucephala clangula*) where the hens had been found dead on the nest. Their analyses indicated that while organo-chlorine pesticides probably did not con-tribute to the death of the hens, eggshell thinning and egg breakage were probably the result of DDE. The mean egg concentration of DDE for mergansers was 0.62 mg/kg ww with an associated 9.6 percent thinning of eggshells. The mean egg concentration of DDE for goldeneye was 0.52 mg/kg ww, and the associated eggshell thinning was 15.4 per-cent. Breakage of eggs in successful nests was also greater for goldeneye than for mergansers.

Mammals

Studies of DDT toxicity to mammals have been generally limited to laboratory mammals. Liver, neurological, develop-mental, reproductive, and carcinogenic effects after exposure to DDT have also been noted for mice, rats, shrews, hamsters, monkeys, dogs, and bats (table 17). Laboratory studies with wild mammals have indicated that big brown bats (*Eptesicus fuscus*) are much more sensitive to DDT than other mammals (Stickel 1973).

Table 17.—DDD, DDE, and DDT effects on mammals

[See Appendix II for explanation of abbreviations and technical terms]

Species	Chemical species	Concentration (mg/kg bw)[1]	Exposure duration	Effects	Reference
Bat (*Eptesicus* sp)	DDT	40		LD50 (oral dose)	Clark and Stafford 1981
Bat (*Myotis* sp.)	DDE	600 (in tissue)		LC50	
Dog	DDT	16	160 weeks	NOAEL	Lehman 1965
		80		LOAEL, liver alterations	
	DDT-tech	1	2 generations	NOAEL	Ottoboni et al. 1977
		5		LOAEL, premature puberty	
	p,p'-DDT	12	14 months	LOAEL, maternal and fetal death	Deichmann et al 1971
Hamster	DDT-tech	40	Lifetime	NOAEL	Cabral et al. 1982
		41.5	128 weeks	LOAEL, necrosis	Rossi et al. 1983
		83		LOAEL, tremors	
Mouse	*p,p'*-DDD	107	78 weeks	NOAEL	NCI 1978
	p,p'-DDT	0.26	Lifetime	LOAEL, liver tumors	Tomatis et al. 1972
		6	78 weeks	NOAEL	NCI 1978
	DDT-tech	6.5	Lifetime + 5 generations	NOAEL	Turusov et al. 1973
		32.5		LOAEL, increase in preweening death	
Rat	DDT	0.5	2 years	LOAEL, liver lesions	Fitzhugh and Nelson 1948
		0.8	2 years	NOAEL	ORNL 1996
		1 mg/kg diet	—	NOAEL	Worthing 1987
	p,p'-DDT	1 mg/kg diet	27 weeks	NOAEL	Laug et al. 1950
		5 mg/kg diet		LOAEL, hepatocellular hypertrophy	
	DDT-tech	1	2 generations	NOAEL	Ottoboni 1969
		10		LOAEL, increased constricting rings of the tail	
	DDT-tech	12.5	Lifetime	LOAEL, liver tumors	Cabral et al. 1982
	DDT	260		LD50 (oral dose)	Gaines and Linder 1986
Rhesus monkey (*Macaca mulatta*)	DDT	8	7.5 years	NOAEL	Durham et al. 1963
Shrew (*Blarina* sp.)	total DDT	910		LC50	Blus 1978
		210 (in tissue)		LC50	

[1] Concentration is the daily dose, as milligrams per kilogram of body weight, unless stated otherwise.

Laboratory rats exposed to DDT in their feed for 27 weeks showed no effects on growth at dietary concentrations up to 50 mg/kg (Laug et al. 1950). However, at dietary concentra-tions of 5 mg/kg and above, they showed pathologic changes, including increased hepatocellular hypertrophy and cytoplasmic oxyphilia, as well as peripheral basophilic cytoplasmic granules. This study established the 5 mg/kg dietary level as the LOAEL and 1 mg/kg as the NOAEL. Fitzhugh and Nelson (1948) studied the long-term effects of relatively high DDT concentrations on rats. They observed liver lesions in rats exposed to 10–800 mg DDT/kg in the diet for 2 years and established an LOAEL of 0.5 mg/kg dw/day.

Although less research has been conducted on wild mammals than on birds, the distribution of tissue residues in mammals seems to be similar. Brain residue levels tend to provide the best indication of toxicity, and DDE is the predominant metabolite found (Stickel 1973).

Predatory and aquatic mammals tend to accumulate the highest residues (Stickel 1973). Mink accumulate higher levels than hares living in the same area. Similarly, shrews and other species in the order Insectivora accumu-late higher levels of total DDT than do mice and voles. Many small seed-eating mammals accumulate only low levels of total DDT even in areas with high environmental levels (Stickel 1973).

Accumulation differences between shrews, mice, voles, mink, and hares were observed in specimens collected over a 9-year period from a forest that had been treated with 1.12 kg DDT per hectare (Dimond and Sherburne 1969). The following residues were measured during the first and last years of the study:

Species	Total DDT, whole body (mg/kg)	
	Year of application	Nine years later
Shrews	15.6	1.18
Mice and voles	1.06	0.03
Mink	—	1.6
Hares	—	0.02

Throughout the study, residues in shrews were 10–36 times those in mice and voles. Similarly, mink collected from the same area contained residues 10–90 times those found in hares.

Analyses of small herbivorous and omniv-orous mammals collected from agricultural areas of Alabama, Arkansas, and Mississippi from 1965 to 1967 (U.S. Department of Agriculture 1969) indicated that rabbits, rice rats, and muskrats contained whole-body residues of total DDT less than the detectable limit; fox squirrels and chipmunks contained less than 0.1 mg/kg; and white-footed mice, cotton rats, and wood rats generally contained less than 0.5 mg/kg. However, harvest mice and house mice contained up to 3.94 mg/kg, and opossums contained up to 8.76 mg/kg.

Ranges of total DDT residues in fat from aquatic mammals and big game mammals collected from several Western States are presented in Stickel (1973). Residue levels in big game mammals include:

Species	Residue levels (mg/kg)
Bear	0.34
Elk	<0.06–29
Moose	0.17
Mountain goats	<0.09–0.9
Mule deer	<1.35–43
Pronghorn antelope	<0.17–0.23
White-tailed deer	<0.4–3

Bioaccumulation

Birds and other wildlife may become exposed to DDT through ingestion of contaminated prey species. Bioaccumulation factors for several species exposed to various environ-mental media are presented in table 18.

Jarvinen et al. (1977) tested fathead minnows for accumulation of DDT present in diet, water, and both diet and water. Tissue residues were greater in fish exposed to DDT in water than in the diet, and accumulation from both water and diet was additive.
Mean bioconcentration factors are shown in table 18. In addition, residue levels and mortality rates for embryos whose parents were exposed to DDT in both water and diet were approximately twice those of embryos whose parents were exposed to DDT only in water.

Table 18.—Bioconcentration factors for biota exposed to DDD, DDE, and DDT

Species	Uptake from:	Compound	Bioconcen-tration factor	Study time	Reference
Aquatic					
Brook trout	Diet	DDT	0.6	120 days	Macek and Korn 1970
Brown bullhead	Water	DDD	125,000	Not stated	Hunt and Bischoff 1960
Fathead minnow (*Pimephales promelas*)	Water	Total DDT	100,000	266 days	Jarvinen et al. 1977
	Diet		1.2		
Golden shiner (*Notemigonus crysoleucas*)	Water	DDT	100,000	15 days	Courtney and Reed 1972
Goldfish (*Carassius auratus*)	Diet	DDT	0.8		Grzenda et al. 1970
Rainbow trout (*Oncorhynchus mykiss*)	Water	DDT	34,900–91,000 (depending on conditions)	96 hours	Muir et al. 1994
Terrestrial					
Black duck (*Anas rubripes*)	Diet	DDE	4.63–4.8	Reproductive cycle	Longcore et al. 1971
Earthworm (*Lumbricus terrestris*)	Soil	DDT	0.71	4 weeks	Davis 1971
Mixed earthworms	Soil	DDD	0.27	11 years	Beyer and Gish 1980
		DDE	6		
		p,p'-DDT	0.56		
		Total DDT	5.1		
			1.8–9.2	Not stated	Ma 1985
Red-winged blackbird (*Agelaius phoeniceus*) eggs	Sediment	DDE	12.9–582.4	Not stated	Bishop et al. 1995b
Short-tailed shrew (*Blarina brevicauda*)	Prey	DDT	1–2.5	Not stated	Blus 1978
Tree swallow (*Tachycineta bicolor*) eggs	Sediment	DDE	16.2–868.6	Not stated	Bishop et al. 1995b
Tree swallow (*Tachycineta bicolor*) nestlings	Sediment	DDE	5–48.9	Not stated	Bishop et al. 1995b

Earthworms compose a large portion of the diets of some birds and reptiles, including woodcock (*Philohela minor*), robin (*Turdus migratorius*), red-bellied snake (*Storeria occipitomaculata*), and eastern garter snake (*Thamnophis sirtalis*). Earthworms are also eaten occasionally by mammals such as insectivores (shrews and moles), rodents, and carnivores (Mustelidae), and by other cluster flies (Beyer and Gish 1980). Based on toxicity studies for birds exposed to DDT, Beyer and Gish (1980) estimated that total DDT residues of 8 mg/kg ww or 32 mg/kg dw in earth-worms would constitute the minimum hazardous level for birds.

Bishop et al. (1995b) measured the bioconcentration of *p,p'*-DDE in red-winged blackbird eggs and in tree swallow (*Tachycineta bicolor*) eggs and nestlings as a function of lipid-normalized concentrations in biota and organic-carbon-normalized concentrations in sediment. In each case, they reported wide ranges of bioconcentration factors (table 18).

Interactions

Interactions between organochlorine pesticides were studied in the earthworm *Lumbricus terrestris* by Davis (1971). Measurements of the uptake of DDT and dieldrin indicated that while dieldrin accumulated more than DDT, the accumulation of either chemical did not affect the accumulation of the other.

Lincer (1992) examined the reproductive success of American kestrels exposed to DDE and Aroclor 1254 in the diet. Birds fed DDE alone and those fed both DDE (3 mg/kg) and Aroclor 1254 (10 mg/kg) had eggs with shells that were significantly thinner than controls. The thinning was 13 percent for birds fed DDE alone and 16 percent for birds fed both DDE and Aroclor 1254. Unexpectedly, birds fed Aroclor 1254 alone exhibited shells that were significantly thicker (6 percent) than controls. In addition, there was no egg breakage in the nests of birds fed either DDE or Aroclor 1254, but those that were fed a combination of DDE and Aroclor 1254 experienced some egg breakage in the all of the nests, and none of their eggs pipped. This study indicates that effects of DDE and Aroclor 1254 are synergistic.

Regulatory Standards

Federal

Ambient Water Quality.—EPA ambient water quality criteria (AWQC) for DDT and its metabolites have been developed for both freshwater and saltwater plants and animals (*Federal Register* 1980, 1992). These criteria are based on levels of DDT that would exceed Food and Drug Administration action levels for human consumption of fish (5 mg/kg; EPA 1996). In addition, screening ecotoxicity thresholds, based strictly on toxicity to ecological species, have been developed using methodology presented in the Great Lakes Water Quality Initiative—Tier II (40 CFR 9, 122 etc., 1995). These screening values represent concentrations above which adverse ecological effects could occur. The AWQCs and the Great Lakes screening ecotoxicity thresholds are listed in table 19.

State

For standards and criteria set by State agencies, contact those agencies directly. See Appendix I for a listing of water quality officials in the 17 Western States.

International

Quality standards for DDT have been established in the Netherlands and in Ontario, Canada. The Dutch quality standards for total

Table 19.—U.S. Environmental Protection Agency standards and criteria for DDT, DDD, and DDE

[See Appendix II for explanation of terms. Sources: EPA 1986, 1996; *Federal Register* 1980, 1982]

Status	Known carcinogen; EPA priority pollutant
Drinking water MCL	None established
Freshwater AWQC (DDT)	1.1 µg/L for acute exposure 0.001 µg/L for chronic exposure
Saltwater AWQC (DDT)	0.13 µg/L for acute exposure 0.001 µg/L for chronic exposure
Great Lakes Water Quality Initiative (DDT)	0.013 µg/L for acute exposure
Freshwater LOAEL (DDD)	0.6 µg/L for acute exposure
Freshwater LOAEL (DDE)	1,050 µg/L for acute exposure
1/1,000,000 cancer risk (water and organisms *or* organisms only)	DDT: 0.59 ng/L DDD: 0.83 ng/L DDE: 0.59 ng/L

DDT include limits of 10 µg/kg dw in sediment, 3 µg/kg dw in soil, and 500 µg/kg ww in wildlife food (Hendriks et al. 1995). The Ontario sediment quality guidelines (Persaud et al. 1993) are as follows:

Compound	Lowest effect level (mg/kg dw)	Severe effect level (mg/kg organic carbon)[1]
p,p'-DDD	0.008	6
p,p'-DDE	0.005	19
o,p'+*p,p'*-DDT	0.008	71
Total DDT	0.007	12

[1] Multiply times the total organic carbon content of a sample to find the bulk-sediment severe effect level for that sample.

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