Chapter 40

Chlorinated Hydrocarbon Insecticides

Synonyms

Organochlorines, OCs

Chlorinated hydrocarbon insecticides (OCs) are diverse synthetic chemicals that belong to several groups, based on chemical structure. DDT is the best known of these insecticides. First synthesized in 1874, DDT remained obscure until its insecticidal properties became known in 1939, a discovery that earned a Nobel Prize in 1948. The means of synthesizing the cyclodiene group, the most toxic of the OCs, was discovered in 1928 and resulted in a Nobel Prize in 1950. The insecticidal properties of cyclodienes, which include aldrin, dieldrin, and endrin (Table 40.1), were discovered about 1945. OCs became widely used in the United States following World War II. Their primary uses included broad spectrum applications for agricultural crops and forestry and, to a lesser extent, human health protection by spraying to destroy mosquitoes and other potential disease carriers. These compounds also became widely used to combat insect carriers of domestic animal diseases.

Cause

Chlorinated hydrocarbon insecticides are stored in body fat reserves or are lipophilic, and they remain in the environment for long periods of time after application. They bioaccumulate or are readily accumulated by animals through many exposure routes or repeated exposure and they tend to biomagnify or accumulate in higher concentrations in animals that are higher in the food chain. This combination of bioaccumulation and biomagnification can harm or kill wildlife, especially some species of birds. The highly toxic cyclodiene compounds cause direct mortality of birds as well as secondary poisoning, which results when birds prey on organisms dying from insecticide applications. Reproductive impairment is the primary effect of the less acutely toxic DDT and its metabolites, DDD and DDE. The cumulative storage of OC residues within body fat reserves presents an additional hazard for birds. Rapid use and depletion or mobilization of fat reserves during migration, food shortages, and other stressful conditions release OC residues into the blood. The residues are then carried to the brain, where they can reach toxic levels resulting in acute poisoning.

Species Affected

Acute mortality from exposure to OCs has been documented in many bird species (Table 40.1). However, the toxicity for birds of different types of these insecticides varies greatly (Tables 40.2 and 40.3). In general, birds that are higher in the food chain are more likely to be affected by OCs present in the environment than birds that are lower in the food chain. This is especially true for fish-eating birds and raptors (Fig. 40.1). Environmental biomagnification of these contaminants can be seen in the mortality of robins and other birds from DDT. Leaves from trees that were sprayed with DDT to control Dutch elm disease had high residues of DDT (174–273 parts per million) shortly after spray applications. When the leaves dropped in the fall, they still contained 20–28 parts per million of DDT. This leaf litter, along with spray residue that reached the ground, produced high DDT residues in the top levels of soil. Earthworms that fed in those soils concentrated the residues to a level high enough to kill birds that fed on them. Another hazard is OC seed dressings, which are used to prevent insect damage to agricultural crops, that may be ingested by waterfowl and other seed or grain-eating birds.

Distribution

Exposure to chlorinated hydrocarbon insecticides is global, and residues of these compounds are found in nearly every environment, even in Antarctica and the Arctic. Avian mortalities from OCs have been reported from Europe, Asia, North America, and South America. Poisoning may occur anywhere that birds are exposed to point sources of these chemicals or through bioaccumulation and biomagnification. Because of their environmental persistence and global movement, residues of chlorinated hydrocarbon insecticides impact bird health long after they become environmental contaminants and at locations far from the original application sites. For example, DDT compounds, polychlorinated biphenyls (PCBs), and dioxin-like compounds were recently found in black-footed albatross adults, chicks, and eggs on Midway Atoll in the Pacific.

Seasonality

Exposure of birds to OCs is most likely during spring and summer in countries where these compounds are still used to control insect pests during the growing season, but exposure may occur any time that residues are present in food sources. For example, waterfowl and other birds that fed on

Table 40.1 *Examples of avian mortality events caused by chlorinated hydrocarbon insecticides.*

Insecticide	Purpose of application	Means of bird exposure	Bird group affected	Principal species affected	Event location and time period
DDT	Spray application to control Dutch elm disease.	Biomagnification in terrestrial food chain.	Passerines	Robin and other small birds.	New England, Midwest; late 1940s to 1950s.
DDD	Spray application to control gnats.	Biomagnification in aquatic food chain.	Grebes	Western grebe	Clear Lake, California, 1950s.
Aldrin	Treatment of rice seed to combat agricultural pests.	Consumption of treated seeds, use and depletion of stored fat reserves during migration or periods of stress.	Waterfowl	Fulvous whistling duck, snow goose, blue-winged teal.	Texas, 1970s
Heptachlor	Treatment of wheat seed to control agricultural pests.	Consumption of treated seeds.	Waterfowl	Canada goose	Oregon, 1970s
Toxaphene	Spray application to control agricultural pests.	Direct contact with and consumption of contaminated food.	Waterfowl	Ducks and coot	California, 1960s
	Spray application for fisheries management.	Ingestion of contaminated food.	Waterfowl	Blue-winged teal, shoveler, mallard.	Nebraska, 1960s
Dieldrin	Spray application to control agricultural pests.	Biomagnification in food chain.	Raptors	Bald eagle, peregrine falcon	Nationwide, 1960s and 1970s.
Endrin	Spray application to control orchard rodents.	Direct contact with spray; consumption of contaminated food; biomagnification; use and depletion of fat reserves.	Gallina-ceous birds, raptors, geese	Quail, chukar partridge, goshawk, Cooper's hawk, barn owl, Canada goose.	Washington, 1960s 1970s, 1980s.
Chlordane	Dry formulations to control soil pests and termites.	Consumption of contaminated food; biomagnification.	Raptors, passerines	Great horned owl, American kestrel, Cooper's hawk, blue jay, robin, starling.	New York, Maryland, New Jersey, 1980s.

Table 40.2 *Toxicity for the mallard duck of some chlorinated hydrocarbon insecticides.*
[Modified from Heinz and others, 1979. LC_{50} is the insecticide concentration, in parts per million, in feed that is required to kill 50 percent of birds during a given period of time. LD_{50} is the insecticide amount, in milligrams per kilogram of body weight, in a single dose that is required to kill 50 percent of birds. ppm, parts per million; mg/kg, milligrams per kilogram; >, greater than; ≥, greater than or equal to. — no data available]

Insecticide	Subacute exposure LC_{50} (ppm)	Acute exposure LD_{50} (mg/kg)
Aldrin	155	520
Chlordane	858	1,200
DDT	1,869	>2,240
Dieldrin	169	381
Endosulfan	1,053	33
Endrin	22	5.6
Heptachlor	480	≥2,000
Lindane	—	>2,000
Mirex	>5,000	>2,400
Toxaphene	538	71

Table 40.3 *Relative acute toxicity of chlorinated hydrocarbon insecticides for birds.*
[Modified from Hudson and others, 1984. LD_{50} is the insecticide amount, in milligrams per kilogram of body weight, in a single dose that is required to kill 50 percent of birds. mg/kg, milligrams per kilogram; >, greater than; <, less than.]

	LD_{50} (mg/kg)								
Species	Aldrin	Chlordane	DDT	Dieldrin	Endosulfan	Endrin	Lindane	Mirex	Toxaphene
Canada goose				<141					
Mallard duck	520	1,200	>2,240	381	31–45	5.6	2,000	2,400	70.7
Fulvous whistling duck	29.2			100–200					99
Sandhill crane			>1,200						100–316
Pheasant	16.8	24–72	1,334	79	80 to >320	1.8		>2,000	40
Sharp-tailed grouse						1.1			19.9
Gray partridge				8.8					23.7
Chukar partridge				25.3					
Bobwhite quail	6.6								85.5
California quail		14.1	595	8.8		1.2			23.7
House sparrow				47.6					
Horned lark									581

endrin-treated winter wheat seed have died in the autumn, and raptors have died yearround. Reproductive effects are manifested during the breeding season, but the exposure that causes these effects can occur at any time of year.

Field Signs

Thin eggshells that often collapse under the weight of the nesting bird and eggs that break during incubation (Fig. 40.2) are classic signs of exposure to DDT and some other OCs. Clinically ill birds suffering from acute poisoning often exhibit signs of central nervous system disorders such as tremors, incoordination, and convulsions (Fig. 40.3). Other birds may be lethargic and exhibit additional behavioral changes (Table 40.4).

Gross Lesions

Birds dying of chronic exposure to OCs are often emaciated (Fig. 40.4). Those that die acutely usually exhibit no lesions. The pathological effects attributed to exposure to these compounds (Table 40.4) are not unique and, therefore, they cannot be used as the only basis for diagnosis.

Diagnosis

Residue analysis combined with necropsy findings, clinical signs, and an adequate field history are generally required for a diagnosis of chlorinated hydrocarbon insecticide poisoning. Brain is the tissue of choice for residue analysis because chemical concentrations that indicate poisoning in birds have been determined for several of these compounds. Take care not to contaminate tissues for residue analysis. Submit the entire carcass whenever possible, otherwise remove the head and send it intact to the laboratory. When it is necessary to remove the brain or other tissues for analysis, rinse the instruments with a solvent, such as acetone or hexane, to remove chlorinated hydrocarbon insecticide residues from them. Place the tissues in solvent-rinsed glass containers or wrap them in aluminum foil. The foil should not have been prepared by a manufacturer that uses oils made of animal fats. A "K" on the package label indicates that no animal fats were used in the manufacturing process.

Experimental studies have been done in an attempt to establish lethal brain levels for OCs in various species of birds (Fig. 40.5). DDE levels in the brains of bald eagles thought to have died from this contaminant have ranged from 212 to 385 parts per million (wet weight), and these levels are consistent with brain DDE levels of kestrels that died from experimental dosing studies (213–301 parts per million, wet weight). These findings are important for interpreting field data (Fig. 40.6). However, interpretation of residue values is complicated by the simultaneous occurrence of other contaminants that may combine with, interact with, or inhibit the toxic effects of any individual compound. Other factors, such as sex, age, and nutritional level also may affect toxicity.

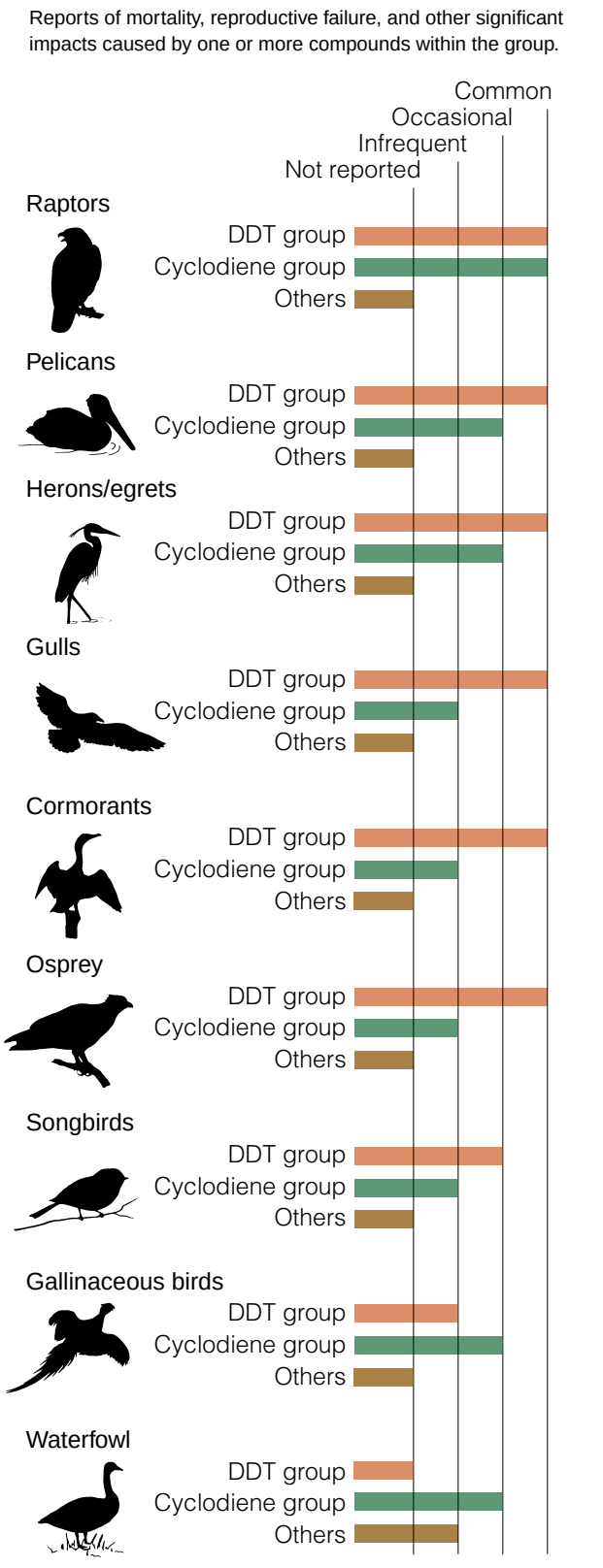

Figure 40.1 *Relative importance of health effects caused by chlorinated hydrocarbon insecticides in selected free-living birds.*

Photo by Milton Friend

Figure 40.2 *The flattened egg within this white-faced ibis clutch was caused by DDE.*

Photos by Milton Friend

Figure 40.3 *Damage to the central nervous system of birds by chlorinated hydrocarbon insecticides results in the type of aberrant posture seen in* ***(A)*** *this hen pheasant and* ***(B)*** *this mallard duck.*

Table 40.4 *Most commonly reported effects from chlorinated hydrocarbon insecticide exposures of birds.*

General effect	Specific effects
Behavioral	Lethargy, slowness, depression
	Locomotive and muscle incoordination (ataxia)
	Tremors and convulsions
	Reduced nest attentiveness and nest abandonment
	Violent wing beating
	Aberrant wing and body carriage
	Muscle spasms causing the body to bend backwards and become rigid (opisthotonos)
Reproductive	Embryo mortality
	Decreased egg hatchability
	Decreased egg production
	Eggshell thinning
	Egg breakage during incubation
Pathological	Emaciation; muscle wasting and absence of fat
	Congestion of the lungs, kidneys, and especially the liver have been reported in pheasants dying from dieldrin poisoning
	Increased liver weight
	Small spleens have been reported
	In general, pathological changes are not readily evident at the gross level, and microscopic changes are not diagnostic
Immunological	Increased susceptibility to infectious disease
Other	Disruption of salt gland function by DDE

Photo by James Runningen

Figure 40.4 *Chronic toxicity from chlorinated hydrocarbon insecticides can result in emaciation, demonstrated by the prominent keel and lack of subcutaneous fat in this black duck. In addition, emaciation caused by the rapid use and depletion of body fat stores due to stresses of migration, inadequate food supplies, and other causes can concentrate body residues of chlorinated hydrocarbons in the brain and cause acute toxicity.*

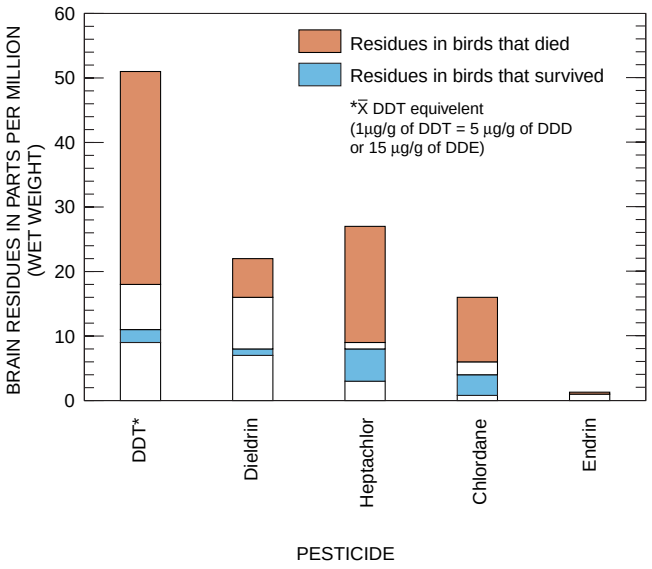

Figure 40.5 *Mean chlorinated hydrocarbon insecticide residues in brains of experimentally dosed passerines.*

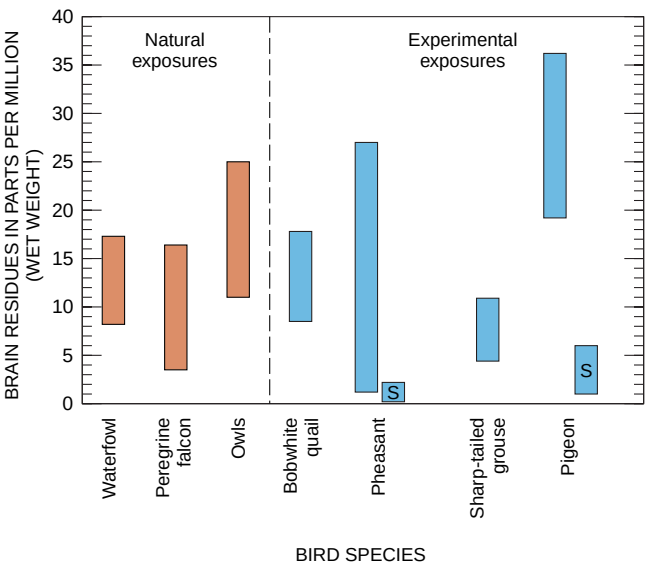

Figure 40.6 *Range of dieldrin residues in brains of selected avian species. [S is the range of concentration within which some of the birds survived.]*

Control

Because uses of most OCs have been banned or greatly curtailed in the United States, controlling wildlife exposure to these compounds depends largely on properly disposing of existing stores, preventing leakage into the environment, and preventing malicious use. The spreading of these compounds to environments where they are no longer used will continue until suitable alternative pest controls are found. Also, migratory wildlife that are exposed to these compounds in areas where they are still used may not exhibit effects until they reach other areas on their migratory route.

Human Health Considerations

As with many of the other toxins discussed in this section, residues of chlorinated hydrocarbons in birds are stored in tissues and are not transferred to humans through routine handling of carcasses. Exceptions include situations where a person could somehow come in contact with the pesticide, for example, in the stomach contents of a bird or on its skin or feathers. It is always wise to handle carcasses with disposable gloves, and to treat unknown mortalities as possible sources of infectious agents transferrable to humans.

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Supplementary Reading

Blus, L.J., 1995, Organochlorine pesticides, *in* Hoffman, D.J., and others, eds., Handbook of ecotoxicology: Boca Raton, Fla., Lewis Publishers, p. 275–300.

Blus, L.J., Wiemeyer, S. N., and Henny, C.J., 1996, Organochlorine pesticides, *in* Fairbrother, A., and others, eds., Noninfectious diseases of wildlife (2nd ed.): Ames, Iowa, Iowa State University Press, p. 61–70.

Heinz, G.H., Hill, E.F., Stickel, W.H., and Stickel, L.F., 1979, Environmental Contaminant studies by the Patuxent Wildlife Research Center, *in* Kenaga, ed., Avian and mammalian wildlife toxicology: Philadelphia, Penn., American Society for Testing and Materials, Special Technical Publication 693, p. 9–35

Hudson, R.H., Tucker, R.K., and Haegele, M.A., 1984, Handbook of toxicity of pesticides to wildlife, (2d ed.): Washington, D.C., U.S. Department of the Interior, Fish and Wildlife Service, Resources Publication 153, 90 p.

Peakall, D.B., 1996, Dieldrin and other cyclodiene pesticides in wildlife, *in* Beyer, W.N., and others, eds., Environmental contaminants in wildlife: interpreting tissue concentrations: Boca Raton, Fla., Lewis Publishers, p. 73–97.