12 DDT, DDD, and DDE in Birds

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12.1 INTRODUCTION

The organochlorine compound known as dichlorodiphenyltrichloroethane (DDT) was synthesized in 1874. Paul Müller discovered its insecticidal activity in 1939 and subsequently received the Nobel Prize for this discovery (Carson 1962). DDT was used extensively in human health operations during World War II. Agricultural applications started immediately after the war, and the amounts used increased exponentially after that time (Hayes 1991).

Concern about the effects on wildlife began almost immediately (Cottam and Higgins 1946). Early field studies with DDT were concerned with the short-term effects after heavy rates of application; for example, 5.6 kg of DDT per hectare (ha) resulted in immediate reductions in the populations of songbirds and invertebrates in an upland hardwood forest (Hotchkiss and Pough 1946). In contrast, 5.6 kg/ha had no effect on the eggs or nestlings of forest birds, but the DDT spray was limited to an area of only 0.09 m² around each nest (Mitchell 1946). DDT applied to a bottom land hardwood forest at a rate of 2.2 kg/ha had no effect on bird populations when applied for only 1 year (Stewart et al. 1946). We know now that the approximate cause-and-effect relation of DDT for mortality may occur immediately after application as well as many months or years after application.

The first important step in uncovering long-term relations was dependent on the development of precise and accurate analytical techniques that could detect DDT and its principal metabolites—DDD and DDE—in environmental samples. Heyroth (1950) summarized the early efforts to develop analytical methodology for detecting residues of DDT. Some of the early work was of limited value, because DDT and one or more metabolites were lumped together or DDE was not measured. Residue

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analysis improved with time, vastly progressing with the development of electron-capture gas chromatography, and was essentially perfected with the development of mass spectrometry. With these advances, the residues of DDT and its metabolites in wildlife could be related to lethal as well as sublethal effects, especially eggshell thinning and reduced reproductive success. Technical DDT, the insecticidal formulation applied in the field, consists of several compounds that may be changed or broken down by a number of physical or biological factors in the environment. Of these compounds, only *p,p'*-DDT (DDT), *p,p'*-DDD (DDD), and *p,p'*-DDE (DDE) have been related to adverse environmental effects. The residue data reported here are on a wet-weight basis unless otherwise indicated.

The purposes of this chapter are to summarize the residue levels of these three compounds in birds that are diagnostic for or are associated with mortality and important sublethal effects and to suggest improvements in the design of contemporary field studies that will result in maximum usefulness in interpreting residue data.

12.2 INTERPRETING LETHAL RESIDUES

12.2.1 BRAIN

The first experimental attempts to measure lethal levels in animals fed DDT-contaminated diets included analyses of several tissues, including the brain. With the limitations of analytical methodology, some of the studies combined DDT with DDD, or DDE was not detected. Considering DDT and DDD combined, Bernard (1963), Stickel et al. (1966), and Stickel and Stickel (1969) concluded that 30 µg/g in the brain were a useful approximation of the lower level representing serious danger and possible death. Most measurements of DDT + DDD in the brains of birds dying from DDT were above this level, but lethal levels were as low as 25 µg/g in house sparrows used in experiments (*Passer domesticus*; Bernard 1963) and 17 µg/g in wild American robins (*Turdus migratorius*) dying with tremors (Hunt 1968). Stickel et al. (1966) concluded that the relative importance of DDT and DDD was not apparent from these data.

With improvements in analytical methodology, residues of DDT, DDD, and DDE were determined in the brains of experimental and wild animals killed by DDT, and more definitive evaluations of the contribution of the individual compounds to lethality were established. Weighting was necessary, because residues in the brains at death ranged from nearly all DDD to nearly all DDT (Stickel et al. 1970). It was also necessary to evaluate residues in the brains of apparently normal animals exposed to DDT and euthanized at periods when others were dying from accumulated dosage and to evaluate the effects of exposure routes, time to death, age, sex, and various stresses on lethal levels in the brain. It was concluded by Stickel et al. (1970) that there is little or no postmortem breakdown of DDT to DDD. Measurements of the lethal levels of DDE and DDD in animals exposed to these individual compounds also helped in evaluating the relative contributions of each toxicant. By considering the levels of DDT and its major metabolites in the brains of animals on DDT dosage that either died or were euthanized, an excellent relation of residues to lethality was established (Table 12.1). This separation was possible because residues in the brain increase rapidly shortly before death, and concentrations uniformly meet or exceed the lower lethal limit in relation to exposure routes, time to death, and the other variables mentioned above (Stickel et al. 1970). There was variation in the means of DDT and the major metabolites in the brain at death; for example, DDT ranged from 15 µg/g in American robins (Wurster et al. 1965) to 40 µg/g in brown-headed cowbirds (*Molothrus ater*; Stickel and Stickel 1969), and DDD ranged from 2 µg/g in northern bobwhite quail (*Colinus virginianus*; Hill et al. 1971) to 99 µg/g in brown-headed cowbirds (Stickel and Stickel 1969). Because of these variations, Stickel et al. (1970) developed the concept of a DDT equivalent, wherein 1 µg/g of DDT equals 5 µg/g of DDD or 15 µg/g of DDE. Using this weighting system, Stickel et al. (1970) indicated that 10 DDT equivalents in the brain constitute an approximate lower lethal limit. American robins that died in tremors had as little as 10 DDT equivalents in their brains. In Table 12.1, all the mean DDT equivalents in the brains of animals that died were

TABLE 12.1
Residues of DDT, DDD, and DDE in Brains of Birds that Died or Were Euthanized While on Experimental DDT Dietary Dosage
and in Brains of Wild American Robins that Died in Tremors

Species	Sex/Fate ^a	µg/g ww						Mean DDT Equivalent	Reference
		DDT		DDD		DDE			
		Mean	Range	Mean	Range	Mean	Range		
Brown-headed cowbird	M/D	39	27–90	59	29–99	7	5–12	51	Stickel and Stickel 1969
	M/E	7	3–19	9	4–17	1	<1–2	9	Stickel and Stickel 1969
	F/D	40	27–77	50	27–71	8	6–10	51	Stickel and Stickel 1969
	F/E	9	6–21	9	6–17	1	<1–3	11	Stickel and Stickel 1969
House sparrow	B/D	28	18–38	16	8–29	9	5–18	35	Hill et al. 1971
Northern bobwhite									
Wild	B/D	23	17–29	8	6–14	11	9–13	25	Hill et al. 1971
Game farm	B/D	25	19–32	3	2–4	9	8–11	26	Hill et al. 1971
Northern cardinal	B/D	19	17–24	8	6–10	3	2–3	21	Hill et al. 1971
(<i>Cardinalis cardinalis</i>)									
Blue jay	B/D	16	12–20	7	6–9	3	2–4	18	Hill et al. 1971
(<i>Cyanocitta cristata</i>)									
American robin	B/D	15	NL	39	NL	57	NL	27	Wurster et al. 1965
Clapper rail	M/D	25	19–31	18	13–27	4	3–7	29	Van Velzen and Kreitzer 1975
(<i>Rallus</i>)	F/D	26	20–31	19	15–23	4	3–5	30	Van Velzen and Kreitzer 1975
(<i>longirostris</i>)	M/E	6	2–10	8	4–13	1	<1–2	8	Van Velzen and Kreitzer 1975

^a B, both sexes; D, died; E, euthanized, appeared normal.
NL, not listed.

≥18, and the mean DDT equivalents in the brains of brown-headed cowbirds that were euthanized on DDT dosage ranged from 8 to 11. Stickel et al. (1970) indicated that the DDT equivalent system was approximate; a 50% margin of error was estimated when all series of data were included. The DDT equivalent weighting system remains a valuable interpretive tool, although the equivalent for DDE probably should be raised to 20 or 25 to reflect the lethal level of DDE alone.

With two notable exceptions, DDE residues in the brains of animals dying from DDT ranged from <1 to 28 µg/g (Table 12.1). Wurster et al. (1965) reported 57 µg/g of DDE in the brains of wild American robins that died from DDT sprayed for Dutch elm disease; DDE exceeded DDT and DDD in these birds. Another more striking exception was the high mean levels of DDE in the brains of cockerels (*Gallus gallus*) that died after being fed DDT; DDE equaled or exceeded the levels of DDT + DDD in these birds with a high of 227 µg/g in a series of birds fed 250 µg of DDT per day (Ecobichon and Saschenbrecker 1968). In comparison with the results of other studies, these high levels of DDE seem anomalous, possibly because of problems in analytical methodology or species differences in the metabolism of DDT.

In animals given diets containing DDE during experiments, residues of only DDE were detected in their brains (Table 12.2). The mean lethal residue of DDE was 499 µg/g in four species of passerine birds, with the lowest individual level of 250 µg/g in a brown-headed cowbird. Stickel et al. (1984) concluded that, for all species tested, residues of DDE in the brain were clearly diagnostic; there was a strong likelihood for death with residues ≥300 µg/g. DDE residues ranged from 52 to 400 µg/g in the brains of birds that were euthanized while receiving dietary levels of DDE that were lethal to other birds. The DDE residue level in the brain of only one euthanized bird overlapped the levels in birds that died (Stickel et al. 1970, 1984).

Regarding DDT equivalents in the brains of birds receiving DDE dosage, the means ranged from 35 to 39 for those that died and from 9 to 10 for those that were euthanized (Table 12.2). Few possible cases of lethal levels of DDE in the brains of wild birds in the United States exist; these include a bald eagle (*Haliaeetus leucocephalus*) with 385 µg/g (Belisle et al. 1972), a great blue heron (*Ardea herodias*) with 246 µg/g (Call et al. 1976), and a black-crowned night heron (*Nycticorax nycticorax*) with 230 µg/g (Ohlendorf et al. 1981). Also, two experimental American kestrels (*Falco sparverius*) that died after a long period on a low dietary dosage of DDE (2.8 ppm) had 213 and 301 µg/g of DDE in their brains (Porter and Wiemeyer 1972). Three other American kestrels died several days

TABLE 12.2
Residues of DDE and DDD in Brains of Birds that Died or Were Euthanized after
Experimental Dietary Exposure to DDE or DDD

Species	Sex/Fate ^a	µg/g ww		Mean DDT Equivalent	Reference
		Mean	Range		
DDE					
Brown-headed cowbird	M/D	499	250–660	39	Stickel et al. 1970
	M/E	152	67–400	10	Stickel et al. 1970
Passerines ^b (4 species)	B/D	499	305–694	39	Stickel et al. 1984
	B/E	137	52–219	9	Stickel et al. 1984
DDD					
Brown-headed cowbird	M/D	172	86–358	34	Stickel et al. 1970
	M/E	42	19–105	8	Stickel et al. 1970

^a B, both sexes; D, died; E, euthanized, appeared normal.

^b Combined data for brown-headed cowbird, common grackle (*Quiscalus quiscula*), red-winged blackbird (*Agelaius phoeniceus*), and European starling (*Sturnus vulgaris*).

after receiving diets containing 160–250 ppm of DDE; their brains contained from 230 to 280 µg/g of DDE (Henny and Meeker 1981). Although the lower lethal limit of 300 µg/g seems to provide a reliable criterion, there is some evidence that lower levels occasionally prove lethal.

Concerning lethal residues of DDD in the brain, birds dying on DDT dosage had mean levels that varied from 3 to 59 µg/g, with individual levels as high as 151 µg/g (Table 12.1). The brains of birds on experimental dietary dosages of DDD contained an average of 172 µg/g of DDD at death, with an individual lower level of 86 µg/g (Table 12.2). Birds on DDD dosage that were euthanized had 19–105 µg/g in their brains. Stickel et al. (1970) concluded that brain concentrations ≥ 65 µg/g indicate an increasing likelihood that death was due to poisoning from DDD. DDT equivalents were 34 in those dying on DDD dosage and 8 in those euthanized on that same dosage (Table 12.2). The only confirmed instance of DDD poisoning in a wild animal was that of a common loon (*Gavia immer*). Its brain contained 200 µg/g of DDD, 130 µg/g of DDE, and 2 µg/g of DDT; DDT equivalents totaled 41 (Prouty et al. 1975). The most vivid example of the effects of DDD occurred when Clear Lake in California was treated with DDD for several years. Although DDD almost certainly adversely affected western grebe (*Aechmophorus occidentalis*) survival and reproductive success, the only two brains analyzed (both females found moribund and euthanized) had DDD residues of 46 and 48 µg/g (Rudd and Herman 1972); these levels were less than the lowest individual residues of birds dying on DDD dosage but were slightly greater than the mean level found in birds euthanized (Table 12.2).

One problem with interpreting residues of the DDT group is that other contaminants including organochlorines are frequently present in the eggs or tissues of wild birds. Regarding organochlorines in the brain, Sileo et al. (1977) assumed straightforward additivity of the toxic effects and developed an “organochlorine index” based on the addition of the proportions of lower lethal levels for all compounds; for example, one half of a lower lethal level contributes 0.5 to the index on the basis of 1 indicating lethality. This index has received little use, one reason being that the lower lethal level of 150 µg/g of DDE ascribed by Sileo et al. (1977) is nearly 100 µg/g less than the accepted lower lethal limit (Table 12.2). Another reason is that the lethal limits of individual organochlorines in wild birds are usually distinct from one another. Finally, the assumed additivity of organochlorines related to lethality in birds has received little verification from experimental studies, although additivity seems the most common joint action (Smyth et al. 1969).

12.2.2 LIVER

Residues in the livers of animals dying during experiments while receiving dietary dosages of DDT differ from residues in their brains in that DDD constitutes the bulk of the residues (Table 12.3). Mean levels of DDT ranged from 1 to 35 µg/g, with a range in individual values from <1 to 254 µg/g. Residues of DDE were relatively low in livers except in American robins, where the residues exceeded those of DDD (Wurster et al. 1965); this is the same series that had exceptionally high residues of DDE in their brains. Cockerels dying while receiving a DDT dietary dosage also had levels of DDE in their livers that exceeded the levels of DDD and DDT combined; the same relation held for residues in their brains as previously mentioned (Ecobichon and Saschenbrecker 1968).

Mean residues of DDE in the livers of brown-headed cowbirds receiving dietary DDE (Table 12.4) were 3883 µg/g (range, 460–11725 µg/g) in those that died and 523 µg/g (range, 266–1560 µg/g) in those that were euthanized (Stickel et al. 1970). In Great Britain, Newton et al. (1992) reported that 23 Eurasian kestrels (*Falco tinnunculus*) and 10 Eurasian sparrowhawks (*Accipiter nisus*) died with lethal levels of DDE in their livers, but the lower lethal limit of 100 µg/g was based on correlative field evidence related to DDT and metabolites (Cooke et al. 1982). According to experimentally derived lethal levels (Stickel et al. 1970), DDE residues in Eurasian sparrowhawk livers (140–254 µg/g) were too low to ascribe lethality to DDE, but DDE residues in the livers of at least three Eurasian kestrels (812, 1474, and 1500 µg/g) were within the lethal range. In the United States, the

TABLE 12.3

Residues of DDT, DDD, and DDE in Livers of Birds that Died or Were Euthanized after Experimental Dietary Exposure to DDT and in Wild American Robins that Died in Tremors

Species	Sex/ Fate ^a	µg/g ww						Reference
		DDT		DDD		DDE		
		Mean	Range	Mean	Range	Mean	Range	
Brown-headed cowbird	M/D	34	3–254	768	215–1,640	55	25–104	Stickel and Stickel 1969
	M/E	5	1–20	58	30–115	3	2–6	Stickel and Stickel 1969
	F/D	35	9–161	552	292–1,063	53	32–88	Stickel and Stickel 1969
	F/E	8	4–16	72	61–107	4	2–8	Stickel and Stickel 1969
American robin	B/D	1	NL	139	NL	165	NL	Wurster et al. 1965
Clapper rail	M/D	3	<1–5	308	75–938	24	7–47	Van Velzen and Kreitzer 1975
	F/D	4	<1–10	229	130–337	19	13–27	Van Velzen and Kreitzer 1975
	M/E	3	<1–7	157	38–352	11	2–36	Van Velzen and Kreitzer 1975

^a B, both sexes; D, died; E, euthanized, appeared normal.

NL, not listed.

TABLE 12.4

Residues of DDE and DDD in Livers of Male Brown-Headed Cowbirds that Died or Were Euthanized after Exposure to DDE or DDD in Their Diets (after Stickel et al. 1970)

Fate ^a	$\mu\text{g/g ww}$	
	Mean	Range
DDE		
D	3,883	460–11,725
E	523	266–1,560
DDD		
D	1,219	79–5,300
E	521	104–2,854

^a D, died; E, euthanized, appeared normal.

only liver analysis from a wild bird that apparently died from DDE was that of a great blue heron that had DDE residues of 246 $\mu\text{g/g}$ in the brain and 570 $\mu\text{g/g}$ in the liver (Call et al. 1976).

The mean residue levels of DDD in the livers of birds receiving dietary dosages of DDD were 1219 $\mu\text{g/g}$ (range, 79–5300 $\mu\text{g/g}$) in those that died and 521 $\mu\text{g/g}$ (range, 104–2854 $\mu\text{g/g}$) in those that were euthanized (Stickel et al. 1970).

To interpret the lethal levels of DDT and its metabolites in the liver, researchers should devise a weighting system, such as that developed for the brain, to determine the relative contribution of each of the compounds. Also, Stickel et al. (1970) indicated that, for birds killed by DDT, residue levels of

DDT and its metabolites in the livers of wild birds were lower than those of laboratory birds. Cooke et al. (1982) indicated that starvation of birds dying from organochlorine pesticides complicated the interpretation of lethal levels in the liver. Bernard (1963) concluded that residues in the brain were more consistent than those in the liver with regard to the interpretation of lethal residues.

12.2.3 OTHER TISSUES

A number of tissues including blood plasma, carcass remainder, kidney, heart, breast muscle, intestinal tract, skin, and fat have been analyzed in birds that were dying or euthanized while receiving a dosage of DDT (Ecobichon and Saschenbrecker 1968, Stickel et al. 1970). Although residues in these tissues have not received the same scrutiny as those in the brain during the assessment of diagnostic lethal levels, Stickel et al. (1970) concluded that residue levels of DDD + DDT in the carcasses of birds dying from DDT increased with the time on dietary dosage and that residue levels in those that were euthanized were essentially indistinguishable from those that died on dosage. In contrast, DDE residues, expressed on a lipid basis, in the carcasses of brown-headed cowbirds sacrificed while receiving DDE dietary dosage differed markedly from those that died on that dosage (Stickel et al. 1984). The same authors concluded that residues in carcass lipids accurately predicted lethal brain residues.

12.3 INTERPRETING SUBLETHAL RESIDUES

12.3.1 EGGS

12.3.1.1 Eggshell Thinning

The classic paper by Ratcliffe (1967a) described eggshell thinning in eggs of peregrine falcons (*Falco peregrinus*) and Eurasian sparrowhawks in Great Britain that occurred following the introduction of DDT.

Soon thereafter in the United States, eggshell thinning was documented in several species of raptorial and fish-eating birds, and the inverse relation between DDE residues in eggs and shell thickness was first established (Hickey and Anderson 1968). Also, Hickey and Anderson (1968) were the first to document decreases in the mean eggshell thickness over a period of years in relation to population declines. Heath et al. (1969) first documented eggshell thinning and associated lowered reproductive success of experimental birds on DDE diets. Subsequently, there have been a substantial number of experimental and field studies that document eggshell thinning and a smaller number that relate residues in eggs to thinning (Tables 12.5 and 12.6).

Birds in experiments on dietary dosages of DDE laid eggs that had considerably thinner shells than did birds on “clean” diets without the compound (Table 12.5). Barn owls (*Tyto alba*) exhibited

TABLE 12.5
Relation of DDE Residues in Eggs to Eggshell Thinning in Birds on Experimental Dietary Dosages of DDE

Species	Thinning (%)		Residues (µg/g ww)		Reference
	Mean	Range	Mean	Range	
Black duck	18	12–29	46	34–63	Longcore et al. 1971
	24	12–32	144	96–219	Longcore et al. 1971
American kestrel	10	1–18	32	17–44	Wiemeyer and Porter 1970
Barn owl	20	NL	12	NL	Mendenhall et al. 1983
	28	NL	41	NL	Mendenhall et al. 1983

NL, not listed.

TABLE 12.6
DDE Residues in Eggs Associated with Eggshell Thinning in Wild Birds

Species	Area ^a	Mean		Reference
		µg/g ww	% Thinning	
Brown pelican	CA	59 ^b	44	Risebrough 1972
	BC	66 ^b	46	Jehl 1973
	BC	25 ^{b,c}	47	Jehl 1973
	BC	8 ^b	26	Jehl 1973
	BC	3 ^b	18	Jehl 1973
	SC	5	17	Blus et al. 1974, 1979
	SC	3	16	Blus et al. 1974, 1979
	SC	1	10	Blus et al. 1974, 1979
	FL	1	5	Blus et al. 1974, 1979
	FL	2	11	Blus et al. 1974, 1979
	TX	3	11	King et al. 1977
American white pelican (<i>Pelecanus erythrorhynchos</i>)	CA	2	15	Boellstorff et al. 1985
	CA	2	10	Boellstorff et al. 1985
Western grebe	CA	1	1	Boellstorff et al. 1985
Great blue heron	WA	4	10	Fitzner et al. 1988
	WA	5	13	Fitzner et al. 1988
Peregrine falcon	AK	2	3 ^d	White 1973
	AK	7	8 ^d	White 1973
	AK	4	7 ^d	White 1973
	AK	44	22 ^d	Cade et al. 1971
	AK	34	17 ^d	Cade et al. 1971
	AK	8	8 ^d	Cade et al. 1971
	AU	18	20 ^d	Pruett-Jones et al. 1988
Northern gannet (<i>Sula bassanus</i>)	QU	19	17	Elliott et al. 1988
Double-crested cormorant	CA ^{b,e}	32	11	Gress et al. 1973
	BC ^{b,e}	24	30	Gress et al. 1973
	ON	24	15	Weseloh et al. 1983
Snowy egret	NV	2	12	Henny et al. 1985
	NV	1	3	Henny et al. 1985
White-faced ibis	NV	2	12	Henny et al. 1985
	NV	1	8	Henny et al. 1985
	TX	1	3	King et al. 1980
	TX	3	14	King et al. 1980
White-tailed eagle (<i>Haliaeetus albicilla</i>)	FI	30	15	Koivusaari et al. 1980
Black-crowned night heron	CO, WY	4	9	McEwen et al. 1984
	QU	2	<1 ^d	Tremblay and Ellison 1980
	MA	4	4	Custer et al. 1983
	MA	2	0	Custer et al. 1983
	RI	4	6	Custer et al. 1983
	RI	1	0	Custer et al. 1983
Gray heron (<i>Ardea cinerea</i>)	GB	6	12 ^d	Cooke et al. 1976
	GB	3	19 ^d	Cooke et al. 1976
Eurasian sparrowhawk	GB	7	18 ^d	Newton and Bogan 1974
Black skimmer (<i>Rynchops niger</i>)	TX	12	12	White et al. 1984
	TX	3	0	White et al. 1984
Osprey	CT	9	15	Wiemeyer et al. 1975

TABLE 12.6 (continued)
DDE Residues in Eggs Associated with Eggshell Thinning in Wild Birds

Species	Area ^a	Mean		Reference
		µg/g ww	% Thinning	
	MD	2	12	Wiemeyer et al. 1975
Bald eagle	OR, WA	10	10	Anthony et al. 1993
Golden eagle	GB	0.1	7 ^d	Newton and Galbraith 1991
(<i>Aquila chrysaetos</i>)	GB	0.1	1 ^d	Newton and Galbraith 1991
	GB	0.2	3 ^d	Newton and Galbraith 1991
	GB	0.3	4 ^d	Newton and Galbraith 1991
	GB	0.3	5 ^d	Newton and Galbraith 1991

^a CA, California; BC, Baja California; SC, South Carolina; FL, Florida; TX, Texas; WA, Washington; AK, Alaska; AU, Australia; QU, Quebec; ON, Ontario; NV, Nevada; FI, Finland; CO, Colorado; WY, Wyoming; MA, Massachusetts; RI, Rhode Islands; GB, Great Britain; CT, Connecticut; MD, Maryland; OR, Oregon.

^b Approximate value converted from lipid basis.

^c Authors suspected residues were too low because of analytical errors.

^d Percentage of thinning based on thickness index (Ratcliffe 1967a); all others based on eggshell thickness.

^e Intact eggs used only for eggshell thickness and residue analysis; the mean eggshell thinning of both crushed and intact eggs was 29% in CA and 38% in BC.

20% eggshell thinning when eggs contained 12 µg/g of DDE (Mendenhall et al. 1983), and black ducks (*Anas rubripes*) exhibited 18% thinning when eggs contained 46 µg/g (Longcore et al. 1971). Although there were several studies of eggshell thinning of birds that were given diets containing technical DDT, most did not list residues in the eggs, and DDE—not DDT—comprises most of the dietary exposure of wild birds with significant eggshell thinning (Stickel 1973). Eggs and tissues of ring-necked pheasants (*Phasianus colchicus*) accumulated high levels of DDT, about 5 times greater than DDE, in areas of intense application of technical DDT (Hunt and Keith 1963). Domestic chickens given diets containing 300 µg/g of technical DDT showed no effects on eggshell thickness compared with controls, even though the eggs of dosed birds contained mean levels of 10 µg/g of DDE and 87 µg/g of DDT (approximate conversion from egg yolk basis at 14 days of incubation; Waibel et al. 1972). Results of the various studies of DDE relations to eggshell thickness in wild birds indicated extreme species differences in sensitivity. Brown pelicans (*Pelecanus occidentalis*) in California and Baja California (Risebrough 1972, Jehl 1973) displayed extreme eggshell thinning and high residues of DDE in 1969 and the early 1970s, with nearly all eggs breaking in the most heavily contaminated colonies. In South Carolina, Florida, and Texas, much lower residues still resulted in mean eggshell thinning of 5–17% (Blus et al. 1974, 1979, King et al. 1977). While there was a statistically significant relation between DDE and eggshell thickness or the thickness index, there were some marked intraspecific differences in response. For example, when considering means (Table 12.6), the peregrine falcon in Alaska showed 22% thinning at 44 µg/g of DDE (Cade et al. 1971) compared with 20% thinning at 18 µg/g of DDE in Australia (Pruett-Jones et al. 1980). Intact eggs of the double-crested cormorant (*Phalacrocorax auritus*) in California exhibited 11% shell thinning at 32 µg/g compared with 24% thinning at 30 µg/g in Baja California (Gress et al. 1973). Many of the cormorant eggs were crushed in both colonies; overall thinning in collections containing both crushed and intact eggs reached 29% in California and 38% in Baja California.

Eggshell thinning is based on either eggshell thickness or the thickness index. In comparisons of the thickness index with eggshell thickness using museum specimens, the index indicated ≥% thinning 76% of the time (Anderson and Hickey 1972) with extreme differences of 10% for each measurement. Thus, it is obvious that either of these measurements represents an accurate indication of

eggshell thinning, but thickness is probably the measurement of choice in most instances, particularly when shells are cut because of loss of fragments.

The rate of thinning per microgram per gram of DDE is much greater at lower residues. Using the brown pelican as an example, there is 5–10% thinning at 1 µg/g of DDE compared with 44% at 59 µg/g (Table 12.6). While there is evidence that certain other contaminants and physiological conditions may induce eggshell thinning, the burden of proof overwhelmingly indicates that DDE is the major cause of the eggshell thinning syndrome. There have been attempts to relate DDE residues in the egg to a level of eggshell thinning that is associated with population decline if such thinning persists over a period of years. Initially, Hickey and Anderson (1968) concluded that ≥18% thinning was associated with declining populations; Anderson and Hickey (1972) modified this to “above 15–20% for a period of years.” Thus, some have taken 15% as an effect level; however, with few exceptions, 18% is probably a more accurate indicator.

One notable exception was that of a declining Eurasian sparrowhawk population in the Netherlands that had poor production, 18% eggshell thinning, and mean DDE residues of 25 µg/g. Even though there was no significant relation between DDE and eggshell thickness, Koeman et al. (1972) suggested that DDE was responsible for the reproductive problems of Eurasian sparrowhawks. Also, Wiemeyer et al. (1972) found a poor correlation between eggshell thinning and DDE concentrations in bald eagle eggs, but there was a significant relation established when additional data were accumulated (Wiemeyer et al. 1988). One of the problems in these relations is that a greater effect per microgram per gram of DDE occurs at lower levels and, if residues are clumped, particularly on the high side, statistical relations are more difficult to establish. Ideally, a wide spread in residues is optimal for detecting effects on eggshell thickness.

When regression analysis was used to relate DDE levels to 20% eggshell thinning (Table 12.7), the critical estimates have ranged from 5 µg/g for the California condor (*Gymnogyps californianus*; Kiff et al. 1979) to 60 µg/g (fresh eggs) to 110 µg/g (failed eggs) for the bald eagle (Wiemeyer et al. 1993). Estimates in Table 12.7 are of value, but they must be interpreted with some caution. For example, the regression equation listed by Cade et al. (1971) indicated that 20% eggshell thinning was associated with 22 µg/g of DDE, whereas their tabular data indicated 17% thinning at 34 µg/g and 22% thinning at 44 µg/g (Tables 12.6 and 12.7). Blus (1984) reported that most of the error was related to extending the regression line beyond the range of the data. Thus, the critical level of 19 µg/g for the great blue heron is much too low, and that of 54 µg/g for the black-crowned night heron is much too high. There are wide disparities in the estimates for the common loon, osprey (*Pandion haliaetus*), and the peregrine falcon. While the California condor is listed as the most sensitive to DDE-induced thinning, the estimated critical level is based on the measurement of eggshell fragments and extraction of DDE from eggshell membranes and then the calculation of residues in the entire egg from these measurements. From work on the extraction of DDE from membranes of intact peregrine falcon eggshells in museums, lower residues are associated with a far greater degree of thinning than are those of intact eggs collected from the field (Peakall and Kiff 1979). Therefore, the accuracy of this technique requires experimental verification. Although Fox (1979) indicated that the measurement of eggshell thickness was a reliable indicator of the DDE content of the egg in some populations, Blus (1984) concluded that the DDE-thickness relation is not tight enough to do this for individual eggs and that residue analysis is essential for interpretation.

The calculated no-effect level for DDE in eggs related to the effects on eggshell thickness ranged from 0.1 µg/g for the brown pelican (Blus 1984) to 2 µg/g for the peregrine falcon (Cade et al. 1971). An earlier estimate for the brown pelican was 0.5 µg/g (Blus et al. 1974), but the sample size and range in residues were much smaller than in the subsequent study.

12.3.1.2 Eggshell Strength

The strength of eggshells, as determined by various mechanical devices, is related to eggshell thickness and, therefore, to DDE residues in the egg. Shell strength decreased more than eggshell thickness per unit of DDE; for example, 8–16 µg/g in sample eggs of the white-faced ibis were associated

TABLE 12.7
Estimated Residues of DDE in Eggs Associated with Eggshell Thinning of
20% in Wild Birds

Species	DDE ($\mu\text{g/g ww}$)	Reference
Common loon	14	Price 1977
	47	Fox et al. 1980
California condor	5 ^a	Kiff et al. 1979
Peregrine falcon	15–20 ^b	Peakall et al. 1975
	22 ^b	Cade et al. 1971
	18 ^b	Pruett-Jones et al. 1980
	20	Enderson and Wrege 1973
Brown pelican	8	Blus 1984
Black-crowned night heron	54	Blus 1984, Henny et al. 1984
Prairie falcon	7	Enderson and Wrege 1973
Great blue heron	19	Blus 1984
Osprey	9	Wiemeyer et al. 1988
	29 ^c	Wiemeyer et al. 1988
	41 ^c	Spitzer et al. 1978
Eurasian sparrowhawk	10 ^c	Newton et al. 1986
Merlin	16 ^c	Newton et al. 1982
White-faced ibis	7	Henny and Herron 1989
Bald eagle	60–110	Wiemeyer et al. 1993

^a Based on thickness of eggshell fragments and DDE content of shell membranes.

^b Percentage of thinning based on thickness index (Ratcliffe 1967a); all others based on eggshell thickness.

^c Eggs collected after nest failure; all other studies except that of Kiff et al. (1979) included at least some eggs collected while nests were active.

with a decrease of 16% in thickness and 37% in strength. In addition, productivity of the young was related to DDE, shell thickness, and shell strength. Although shell strength may provide a more sensitive indicator of potential egg failure due to DDE, simple thickness measurements have served very well in that regard, and there are fewer logistical and financial constraints than are required to measure strength.

12.3.1.3 Productivity

DDT, primarily through its major metabolite DDE, also affects the reproductive success of birds. Eggshell thinning is an important, but not exclusive, factor related to reproductive problems (Blus 1984). Unfortunately, most experimental studies of the reproductive effects of DDE or DDT did not present residues. This was a loss for interpreting the effects of residues on reproductive success in field studies.

There are several methods of expressing reproductive success relative to residues in birds. One method relates the overall reproductive success of a colony or other breeding group, such as a pen of experimental birds, to the mean residue content in their eggs (Table 12.8). Black ducks (*A. rubripes*) receiving dietary dosages of 10 or 30 ppm of DDE had a significantly reduced survival of embryonated eggs or hatchlings to 3 weeks posthatch in relation to controls; DDE averaged 46 and 144 $\mu\text{g/g}$ in eggs of treated birds (Longcore et al. 1971). Barn owls on a diet containing 3 ppm of DDE had hatchling and fledging rates that were reduced about 75% from control values over 2 years when eggs contained an average of 12 $\mu\text{g/g}$ the first year and 41 $\mu\text{g/g}$ the second year (Mendenhall et al. 1983). The response was approximately the same each year, even though residues in eggs of the barn owls were much higher the second year. Although these studies document the effects from DDE,

TABLE 12.8

Residues of DDE in Eggs Related to the Reproductive Success of Birds on Experimental Dietary Dosages of DDE

					Reproductive Success (% of Survival to 3 Weeks Posthatch)	
Species	Reference	Treatment	Residues (µg/g ww)		Hatchlings	Embryonated Eggs
			Mean	Range		
Black duck	Longcore et al. 1971	Control	0.28	0.14–0.67	91	38
		10 ppm	46	34–63	64 ^a	23 ^a
		30 ppm	144	96–219	50 ^a	9 ^a
					Mean per Pair	
					Eggs Hatched	Young Fledged
Barn owl	Mendenhall et al. 1983	Control (1st year)	0.25	NL	3.2	2.9
		Control (2nd year)	0.40	NL	3.7	3.1
		3 ppm (1st year)	12	NL	1.1 ^a	0.7 ^a
		3 ppm (2nd year)	41	NL	0.9 ^a	0.7 ^a

^a Significantly different ($p \leq .05$) from controls.

NL, not listed.

TABLE 12.9

Mean Residues of DDE in Eggs Related to Mean Reproductive Success of Wild Birds

Species	Area ^a	Residues ($\mu\text{g/g ww}$)	Young Produced per Active Nest	Reference
Double-crested cormorant	CA	32 ^b	0.0	Gress et al. 1973
	BC	24 ^b	<0.1	Gress et al. 1973
	ON	14–16	0–0.1 ^c	Weseloh et al. 1983
	ON	5	0.3	Weseloh et al. 1983
Black-crowned night heron	QU	2	2.4	Tremblay and Ellison 1980
Common loon	SA	6	0.7	Fox et al. 1980
Brown pelican	CA	59 ^d	<0.1	Risebrough 1972
	BC	66 ^d	0.0	Jehl 1973
	BC	25 ^{d,e}	<0.1	Jehl 1973
	BC	8 ^d	0.1	Jehl 1973
	BC	3 ^d	–0.8	Jehl 1973
Bald eagle	OR, WA	10	0.6	Anthony et al. 1993

^a CA, California; BC, Baja California; ON, Ontario; QU, Quebec; SA, Saskatchewan; OR, Oregon; WA, Washington.

^b Approximate conversion from dry weight basis.

^c Five colonies.

^d Approximate conversion from lipid weight basis.

^e Authors suspected residues were too low because of analytical errors.

a narrow part of the relation is presented; for example, there is no indication of the dietary levels or residues in eggs at which problems first appear or where they initially become serious.

Some of the field data on reproductive success also follow the average residue-average effect design (Table 12.9). A method that gives more insight into the effects of residues on reproductive

success is the sample egg technique (Table 12.10), whereby one egg is taken from a nest and analyzed, the nest is marked, its fate is monitored through periodic visits, and the residues in eggs are related to nest success (Blus 1984). This is particularly valuable in the field, where many factors may influence reproductive outcome. Blus (1984) lists advantages and disadvantages of this method. Where nest predation is a problem and where clutch size permits, one egg may be collected for residue analysis and another taken and placed in an incubator.

There are several variations to the sample egg technique as shown in Table 12.10. One involves work with threatened or endangered species or other special situations where the sample egg is not collected until the fate of the marked nest is determined (Spitzer et al. 1978). The major bias with collecting eggs after the fact is that those with thin eggshells and high DDE residues have a greater chance of being crushed or cracked and therefore lost from the population. Other variations relate to statistical analysis of the data, regardless of the time of egg collections. One method ranks young fledged versus residues (Fyfe et al. 1976, Spitzer et al. 1978), and another method ranks residues or a range of residues versus young fledged (Blus et al. 1980, Blus 1982, Henny et al. 1984, 1985, Henny and Herron 1989, Ambrose et al. 1988, Wiemeyer et al. 1993). Of these two methods, I recommend the second, because

TABLE 12.10
Residues of DDE in Sample Eggs of Wild Birds Related to Reproductive Success

Species	Area ^a	Residues (µg/g ww)	Young Produced per Active Nest ^b	Reference
Osprey	CT, NY	23 ^{c,d,e}	0.0	Spitzer et al. 1978
	CT, NY	12 ^{c,d,e}	1.0	Spitzer et al. 1978
	CT, NY	6 ^{c,d,e}	2.1	Spitzer et al. 1978
	ID	14	0.0	Johnson et al. 1975
	ID	6	1.6	Johnson et al. 1975
Peregrine falcon	AK	≤15	1.8	Ambrose et al. 1988
	AK	15–30	2.0	Ambrose et al. 1988
	AK	>30	1.0	Ambrose et al. 1988
Snowy egret	NV	≤1	2.2	Henny et al. 1985
	NV	1–5	2.4	Henny et al. 1985
	NV	5–10	1.0	Henny et al. 1985
	NV	10–20	1.0	Henny et al. 1985
Prairie falcon	AB	2 ^c	0.0	Fyfe et al. 1976
	AB	2 ^c	1.0	Fyfe et al. 1976
	AB	2 ^c	2.0	Fyfe et al. 1976
	AB	2 ^c	3.0	Fyfe et al. 1976
	AB	1 ^c	4.0	Fyfe et al. 1976
Merlin	AB	11 ^c	0.0	Fyfe et al. 1976
	AB	11 ^c	1.0	Fyfe et al. 1976
	AB	6 ^c	2.0	Fyfe et al. 1976
	AB	5 ^c	3.0	Fyfe et al. 1976
	AB	6 ^c	4.0	Fyfe et al. 1976
Brown pelican	SC ^f	≤1.5	0.6 (FL), ^g 0.8 (EM)	Blus 1982
	SC ^f	1.5–3	0.6 (FL), 0.8 (EM)	Blus 1982
	SC ^f	>3	0.0 (FL), 0.6 (EM)	Blus 1982
Bald eagle	U.S. ^e	<2.2	1.0	Wiemeyer et al. 1993
	U.S. ^e	2.2–3.5	1.0	Wiemeyer et al. 1993
	U.S. ^e	3.6–6.2	0.5	Wiemeyer et al. 1993
	U.S. ^e	6.3–11.9	0.3	Wiemeyer et al. 1993
	U.S. ^e	≥12	0.2	Wiemeyer et al. 1993

continued

TABLE 12.10 (continued)
Residues of DDE in Sample Eggs of Wild Birds Related to Reproductive Success

Species	Area ^a	Residues (µg/g ww)	Young Produced per	Reference
			Active Nest ^b	
Black-crowned night heron	U.S.	≤1	2.0	Henny et al. 1984
	U.S.	1–4	1.7	Henny et al. 1984
	U.S.	4–8	1.5	Henny et al. 1984
	U.S.	8–12	1.1	Henny et al. 1984
	U.S.	12–16	1.0	Henny et al. 1984
	U.S.	16–25	0.8	Henny et al. 1984
	U.S.	25–50	0.4	Henny et al. 1984
White-faced ibis	NV	≤1	1.8	Henny and Herron 1989
	NV	1–4	1.8	Henny and Herron 1989
	NV	4–8	1.3	Henny and Herron 1989
	NV	8–16	0.8	Henny and Herron 1989
	NV	>16	0.6	Henny and Herron 1989
Great blue heron	OR, WA	3	1.7–2.0	Blus et al. 1980

^a CT, Connecticut; NY, New York; ID, Idaho; AK, Alaska; NV, Nevada; AB, Alberta and nearby areas; SC, South Carolina; U.S., various locations within the U.S.

^b Young produced not adjusted for sample egg collected.

^c Approximate adjustment from dry weight basis.

^d Elevated levels (17–29 µg/g) of polychlorinated biphenyls also present.

^e All or most eggs were collected after the fate of marked nests was determined. Production of young at each nest is based on a 5-year mean.

^f Sample egg either freshly laid or embryonated when collected.

^g FL, freshly laid; EM, embryonated.

it seems to more closely approximate the dependent variable-independent variable relation; however, it should be recognized that these methods have not been subjected to rigid statistical testing.

Problems with comparing the young fledged per nest with the residue content of sample eggs seemed evident in a study of prairie falcons (*Falco mexicanus*) in Alberta, Canada, and surrounding areas, where the mean DDE levels of 1–2 µg/g were said to adversely affect fledging success (Table 12.10; Fyfe et al. 1976). However, differences in the mean residue content were not statistically different, and the egg with the highest level of 11 µg/g was from a nest that fledged five young (Fyfe et al. 1976). In merlins (*Falco columbarius*), fledging success was significantly related to DDE residues in sample eggs with an effect level of near 10 µg/g but, again, high levels of about 31 and 26 µg/g were found in eggs from nests that fledged one and five young, respectively (Fyfe et al. 1976). On the basis of addled or deserted eggs collected from merlin nests in Great Britain, Newton et al. (1982) indicated a positive correlation between fledging success and DDE residues, with zero young fledged when eggs contained 5 µg/g increasing to four young fledged at 8 µg/g. Although the lower critical level of DDE that adversely affects the reproductive success of peregrine falcons was considered to be 15–20 µg/g (Peakall 1976), nest success in Great Britain seemed unaffected by DDE, with the highest residues of 25 and 31 µg/g being detected in sample eggs from successful nests (Ratcliffe 1967b). More recent, albeit limited, evidence from peregrine falcons in Alaska indicated that the effects on nest success occur only when residues exceed 30 µg/g (Ambrose et al. 1988).

Considering DDE residues versus the young produced per nest, declines in the productivity of brown pelicans (Blus 1982), bald eagles (Wiemeyer et al. 1993), black-crowned night herons (Henny et al. 1984), snowy egrets (*Egretta thula*; Henny et al. 1985), and the white-faced ibis (*Plegadis chihi*; Henny and Herron 1989) are obvious (Table 12.10). One can determine the level at which

residues first begin having an adverse effect on the number of young produced, for example, 4–8 $\mu\text{g/g}$ in the white-faced ibis (Henny and Herron 1989), and at which few or no young are produced, for example, ≥ 15 $\mu\text{g/g}$ in the bald eagle. Black-crowned night herons demonstrate an impressive gradual decline in productivity with an increase in residues; however, a few young are produced even at levels > 25 $\mu\text{g/g}$ (Henny et al. 1984). In the brown pelican, Blus (1982) indicated a dramatic effect above 3 $\mu\text{g/g}$, with no young produced in sample eggs that were freshly laid when collected and a 25% reduction in those that were embryonated when collected (Table 12.10). The brown pelican is apparently the most sensitive avian species to DDE, with reproductive failure when residues in eggs exceed 3.7 $\mu\text{g/g}$ (Blus 1982). In South Carolina, a combination of effects from DDE, including eggshell thinning, seemed to adversely affect reproductive success, whereas in California and Baja California nearly every egg collapsed from extreme eggshell thinning in 1969 and several subsequent years (Risebrough 1972, Jehl 1973). To interpret what a DDE-related reduction in the number of young fledged means in terms of population reduction, one has to know a great deal about the population, for example, an approximate recruitment standard (number of young that must be fledged per pair of breeding age to maintain a stable population) and adult mortality compensating mechanisms, including renesting and other factors. Recruitment standards are 1.2–1.5 young for the brown pelican (Henny 1972), 1.0 young for the bald eagle (Wiemeyer et al. 1993), 1–1.3 young (Henny and Wight 1969) and 0.8 young (Spitzer et al. 1983) for the osprey, 1.9 young for the great blue heron (Henny 1972), and 2–2.1 young for the black-crowned night heron (Henny 1972). Variations in the recruitment standard for the osprey probably result from whether active nests (Spitzer et al. 1983) or pairs of breeding age (Henny and Wight 1969) are used in the calculations.

In Table 12.10, no compensation in the young produced was made for collection of the sample egg. Therefore, most of these productivity data are probably biased low, compared with nests without an egg collected. To measure this bias, Henny and Herron (1989) compared production in white-faced ibis nests with an egg collected to that without an egg collected; sample egg collection was associated with a 30% reduction in the young produced per active nest. The percentage of the reduction, of course, would be influenced by clutch size in the species of interest.

Although DDE was responsible for most reproductive failure in birds, very high levels of DDT in ring-necked pheasant eggs in California may have caused reproductive problems, such as crippling and mortality of young; however, the link between them was never clearly established (Hunt and Keith 1963).

Domestic chickens given diets containing 300 $\mu\text{g/g}$ of technical DDT showed no significant effects on reproduction compared with controls, even though their eggs contained mean levels of 10 $\mu\text{g/g}$ of DDE and 87 $\mu\text{g/g}$ of DDT (approximate conversion from egg yolk basis at 14 days of incubation; Waibel et al. 1972).

12.4 FOOD

The lowest dietary concentration of DDE that resulted in critical eggshell thinning and decreased production in the peregrine falcon was estimated at 1 $\mu\text{g/g}$ (Enderson et al. 1982). A more recent study used 3 $\mu\text{g/g}$ as a critical level, but this was based on dietary levels given to experimental raptors that experienced serious reproductive problems and even adult mortality (DeWeese et al. 1986). For the brown pelican, the lower critical dietary level of DDE was estimated at about 0.1 $\mu\text{g/g}$ on the basis of $31 \times$ biomagnification from fish to pelican egg; however, because the chief prey fish also contained DDT at one-half the amount of DDE, the 0.1- $\mu\text{g/g}$ level probably should be raised slightly to account for metabolism from DDT to DDE (Blus et al. 1977). These examples included a highly sensitive species and a moderately sensitive species, so higher estimates for less sensitive species are expected based on experimental studies of the domestic chicken, one of the least sensitive species (Waibel et al. 1972); but lower estimates are unlikely. Because of the lipophilicity and bioaccumulativeness of all three compounds, the highest residues and, depending on species sensitivity, the most extreme effects are found in species at the highest trophic levels, as is evident in most of the studies summarized in this review.

12.5 OTHER TISSUES

There are other measurements of the sublethal effects related to residues of DDT and its metabolites in other tissues of animals used in experiments, but most of these data are fragmentary, and few of these measurements have proven useful in field studies.

Summary

Although technical DDT was initially hailed as a tremendous tool in pest control, the environmental problems soon outweighed the positive aspects. As a result, this compound was banned over much of the world; however, use of DDT continues in some countries, especially for control of insects that are disease vectors.

One of the first findings related to the use of technical DDT was the mortality of wildlife after heavy applications, but suitable analytical techniques were required to detect residues because many adverse effects occurred sometime after exposure. Residues in tissues, particularly the brain, have proven to be diagnostic of lethality in animals on dietary dosages of DDT, DDD, and DDE in experiments. When used in field investigations, this technique made possible the interpretation of lethality when DDT equivalents (weighting system where an equivalent equals 1 $\mu\text{g/g}$ of DDT, 5 $\mu\text{g/g}$ of DDD, or 15 $\mu\text{g/g}$ of DDE) are as low as 10 in brains; however, most birds or mammals that die from DDT have DDT equivalents >20 . Few dead wild birds have been found with lethal levels of DDE or DDD in their brains. Residues in livers also have been used to establish the lethality of DDT in wild animals, but a system for the weighting of the three compounds has not been developed.

Residues in the eggs of birds are a reliable indicator of eggshell thinning and reproductive success. Of the three compounds reviewed in this chapter, evidence overwhelmingly indicates that DDE is responsible for most eggshell thinning, reproductive problems, and population reductions. There is a tremendous variation in species sensitivity to these compounds. The brown pelican is the most sensitive, with eggshell thinning and depressed productivity occurring at 3.0 $\mu\text{g/g}$ of DDE in the egg and total reproductive failure when residues exceed 3.7 $\mu\text{g/g}$. In contrast, adverse effects on the reproductive success of peregrine falcons first occur when DDE residues in the egg are about 10-fold higher, that is, 30 $\mu\text{g/g}$. Black-crowned night herons demonstrate a different pattern involving a gradual decline in productivity with increasing residues. A few young are still produced at levels >25 $\mu\text{g/g}$. The domestic chicken is very tolerant of high dietary exposure to technical DDT. By efficient use of the sample egg technique, the effects induced by DDE residues within one colony or breeding area, or compared with a reference colony or area where residues are low, can be quantified and related to the adverse effects on the individual and the population. Techniques for quantifying the relation between residues of DDT and its metabolites and the effects on the biota have been successful, but the process required much time, effort, and financial outlay. Many contemporary field studies are designed inefficiently with regard to quantifying residues, with little or no consideration given to establishing the effects, or less commonly, establishing the effects without evidence from residues. In addition, few experimental studies are directly applicable to the field. Results of experimental and field studies could be made more pertinent to interpretation of field data by changes in the experimental design, so that efficient use can be made of the establishing and measuring of effects induced by residues.

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