ENVIRONMENTAL CONTAMINANTS in WILDLIFE

Interpreting Tissue Concentrations

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SETAC Special Publications Series

Publication sponsored by the Society of Environmental Toxicology and Chemistry (SETAC) and the SETAC Foundation for Environmental Education

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LEWIS PUBLISHERS

Boca Raton New York London Tokyo

The frontispiece of the Eastern screech owl was drawn by Sabra Niebur.

Library of Congress Cataloging-in-Publication Data

Environmental contaminants in wildlife : interpreting tissue concentrations / edited by W. Nelson Beyer, Gary H. Heinz, Amy W. Redmon-Norwood.
p. cm. -- (SETAC special publications series)
"Publication sponsored by the Society of Environmental Toxicology and Chemistry (SETAC) Foundation for Environmental Education."
ISBN 1-56670-071-X (alk. paper)
1. Biological monitoring. 2. Histology, Pathological. 3. Pollution--Environmental aspects. I. Beyer, W. Nelson. II. Heinz, Gary H. III. Redmon-Norwood, Amy W. IV. SETAC (Society) V. SETAC Foundation for Environmental Education. VI. Series.
RA1223.B54E57 1996
615.9'02--dc20 95-45493
CIP

Lewis Publishers is an imprint of CRC Press

No claim to original U.S. Government works
International Standard Book Number 1-56670-071-X
Library of Congress Card Number 95-45493
Printed in the United States of America 1 2 3 4 5 6 7 8 9 0

CHAPTER 2

DDT, DDD, and DDE in Birds

Lawrence J. Blus

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 m^2 around each nest (Mitchell, 1946). DDT applied to a bottomland 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 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

1-56670-071-X/96/$0.00+$.50

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.

INTERPRETING LETHAL RESIDUES

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 *(Passer domesticus)* used in experiments (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 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

Table 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

		μg/g, Wet weight						Mean	
	Sex/	DDT		DDD		DDE		DDT	
Species	fate[a]	Mean	Range	Mean	Range	Mean	Range	equivalent	Source[b]
Brown-headed cowbird	M/D	39	27-90	59	29-99	7	5-12	51	1
	M/E	7	3-19	9	4-17	1	<1-2	9	1
	F/D	40	27-77	50	27-71	8	6-10	51	1
	F/E	9	6-21	9	6-17	1	<1-3	11	1
House sparrow	B/D	28	18-38	16	8-29	9	5-18	35	2
Northern bobwhite									
Wild	B/D	23	17-29	8	6-14	11	9-13	25	2
Game farm	B/D	25	19-32	3	2-4	9	8-11	26	2
Northern cardinal *(Cardinalis cardinalis)*	B/D	19	17-24	8	6-10	3	2-3	21	2
Blue jay *(Cyanocitta cristata)*	B/D	16	12-20	7	6-9	3	2-4	18	2
American robin	B/D	15	NL[c]	39	NL	57	NL	27	3
Clapper rail	M/D	25	19-31	18	13-27	4	3-7	29	4
(Rallus longirostris)	F/D	26	20-31	19	15-23	4	3-5	30	4
	M/E	6	2-10	8	4-13	1	<1-2	8	4

[a] B, both sexes; D, died; E, euthanized, appeared normal.
[b] 1, Stickel and Stickel (1969); 2, Hill et al. (1971); 3, Wurster et al. (1965); 4, Van Velzen and Kreitzer (1975).
[c] NL, not listed.

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 1, all the mean DDT equivalents in the brains of animals that died were ≥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 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 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; Stickel et al., 1984).

Table 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, Wet weight Mean	Range	Mean DDT equivalent	Source[b]
		DDE			
Brown-headed cowbird	M/D	499	250-660	39	1
	M/E	152	67-400	10	1
Passerines[c] (4 species)	B/D	499	305-694	39	2
	B/E	137	52-219	9	2
		DDD			
Brown-headed cowbird	M/D	172	86-358	34	1
	M/E	42	19-105	8	1

[a] B, both sexes; D, died; E, euthanized, appeared normal.
[b] 1, Stickel et al. (1970); 2, Stickel et al. (1984).
[c] Combined data for brown-headed cowbird, common grackle *(Quiscalus quiscula)*, red-winged blackbird *(Agelaius phoeniceus)*, and European starling *(Sturnus vulgaris)*.

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 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 after receiving diets containing 160 to 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 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 2). Birds on DDD dosage that were euthanized had 19 to 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 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 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 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).

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 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).

Table 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

		μg/g, Wet weight						
	Sex/	DDT		DDD		DDE		
Species	fate[a]	Mean	Range	Mean	Range	Mean	Range	Source[b]
Brown-headed cowbird	M/D	34	3-254	768	215-1,640	55	25-104	1
	M/E	5	1-20	58	30-115	3	2-6	1
	F/D	35	9-161	552	292-1,063	53	32-88	1
	F/E	8	4-16	72	61-107	4	2-8	1
American robin	B/D	1	NL[c]	139	NL	165	NL	2
Clapper rail	M/D	3	<1-5	308	75-938	24	7-47	3
	F/D	4	<1-10	229	130-337	19	13-27	3
	M/E	3	<1-7	157	38-352	11	2-36	3

[a] B, both sexes; D, died; E, euthanized, appeared normal.
[b] 1, Stickel and Stickel (1969); 2, Wurster et al. (1965); 3, Van Velzen and Kreitzer (1975).
[c] NL, not listed.

Mean residues of DDE in the livers of brown-headed cowbirds receiving dietary DDE (Table 4) were 3883 μg/g (range, 460 to 11,725 μg/g) in those that died and 523 μg/g (range, 266 to 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 to 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 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 μg/g in the brain and 570 μg/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 μg/g (range, 79 to 5300 μg/g) in those that died and 521 μg/g (range, 104 to 2854 μg/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

Table 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]	μg/g, Wet weight 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.

in the brain were more consistent than those in the liver with regard to the interpretation of lethal residues.

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.

INTERPRETING SUBLETHAL RESIDUES

EGGS

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 U.S., 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 5 and 6).

Table 5 Relation of DDE Residues in Eggs to Eggshell Thinning in Birds on Experimental Dietary Dosages of DDE

Species	Thinning (%)		Residues (μg/g, wet weight)		Source[a]
	Mean	Range	Mean	Range	
Black duck	18	12-29	46	34-63	1
	24	12-32	144	96-219	1
American kestrel	10	1-18	32	17-44	2
Barn owl	20	NL[b]	12	NL	3
	28	NL	41	NL	3

[a] 1, Longcore et al. (1971); 2, Wiemeyer and Porter (1970); 3, Mendenhall et al. (1983).
[b] NL, not listed.

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 5). Barn owls *(Tyto alba)* exhibited 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 interspecific 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 to 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 6), the

Table 6 DDE Residues in Eggs Associated with Eggshell Thinning in Wild Birds

Species	Area[a]	Mean µg/g, Wet weight	Mean % Thinning	Source[b]
Brown pelican	CA	59[c]	44	1
	BC	66[c]	46	2
	BC	25[c,d]	47	2
	BC	8[c]	26	2
	BC	3[c]	18	2
	SC	5	17	3,4
	SC	3	16	3,4
	SC	1	10	3,4
	FL	1	5	3,4
	FL	2	11	3,4
	TX	3	11	5
American white pelican	CA	2	15	6
(Pelecanus erythrorhynchos)	CA	2	10	6
Western grebe	CA	1	1	6
Great blue heron	WA	4	10	7
	WA	5	13	7
Peregrine falcon	AK	2	3[e]	8
	AK	7	8[e]	8
	AK	4	7[e]	8
	AK	44	22[e]	9
	AK	34	17[e]	9
	AK	8	8[e]	9
	AU	18	20[e]	10
Northern gannet *(Sula bassanus)*	QU	19	17	11
Double-crested cormorant	CA[c,f]	32	11	12
	BC[c,f]	24	30	12
	ON	24	15	13
Snowy egret	NV	2	12	14
	NV	1	3	14
White-faced ibis	NV	2	12	14
	NV	1	8	14
	TX	1	3	15
	TX	3	14	15
White-tailed eagle *(Haliaeetus albicilla)*	FI	30	15	16
Black-crowned night heron	CO, WY	4	9	17
	QU	2	<1[e]	18
	MA	4	4	19
	MA	2	0	19
	RI	4	6	19
	RI	1	0	19
Gray heron *(Ardea cinerea)*	GB	6	12[e]	20
	GB	3	9[e]	20
Eurasian sparrowhawk	GB	7	18[e]	21
Black skimmer *(Rynchops niger)*	TX	12	12	22
	TX	3	0	22

Table 6 (continued) DDE Residues in Eggs Associated with Eggshell Thinning in Wild Birds

Species	Area[a]	Mean µg/g, Wet weight	% Thinning	Source[b]
Osprey	CT	9	15	23
	MD	2	12	23
Bald eagle	OR, WA	10	10	24
Golden eagle	GB	0.1	7[e]	25
(Aquila chrysaetos)	GB	0.1	1[e]	25
	GB	0.2	3[e]	25
	GB	0.3	4[e]	25
	GB	0.3	5[e]	25

[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 Island; GB, Great Britain; CT, Connecticut; MD, Maryland; OR, Oregon.

[b] 1, Risebrough (1972); 2, Jehl (1973); 3, Blus et al. (1974); 4, Blus et al. (1979); 5, King et al. (1977); 6, Boellstorff et al. (1985); 7, Fitzner et al. (1988); 8, White et al. (1973); 9, Cade et al. (1971); 10, Pruett-Jones et al. (1980); 11, Elliott et al. (1988); 12, Gress et al. (1973); 13, Weseloh et al. (1983); 14, Henny et al. (1985); 15, King et al. (1980); 16, Koivusaari et al. (1980); 17, McEwen et al. (1984); 18, Tremblay and Ellison (1980); 19, Custer et al. (1983); 20, Cooke et al. (1976); 21, Newton and Bogan (1974); 22, White et al. (1984); 23, Wiemeyer et al. (1975); 24, Anthony et al. (1993); 25, Newton and Galbraith (1991).

[c] Approximate value converted from lipid basis.

[d] Authors suspected residues were too low because of analytical errors.

[e] Percentage of thinning based on thickness index (Ratcliffe, 1967a); all others based on eggshell thickness.

[f] 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.

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 µg/g of DDE is much greater at lower residues. Using the brown pelican as an example, there is 5 to 10% thinning at 1 µg/g of DDE compared with 44% at 59 µg/g (Table 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 to 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 μg/g 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 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 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 6 and 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

Table 7 Estimated Residues of DDE in Eggs Associated with Eggshell Thinning of 20% in Wild Birds

Species	DDE (μg/g, Wet weight)	Source[a]
Common loon	14	1
	47	2
California condor	5[b]	3
Peregrine falcon	15-20[c]	4
	22[c]	5
	18[c]	6
	20	7
Brown pelican	8	8
Black-crowned night heron	54	8,9
Prairie falcon	7	7
Great blue heron	19	8
Osprey	9	10
	29[d]	10
	41[d]	11
Eurasian sparrowhawk	10[d]	12
Merlin	16[d]	13
White-faced ibis	7	14
Bald eagle	60-110	15

[a] 1, Price (1977); 2, Fox et al. (1980); 3, Kiff et al. (1979); 4, Peakall et al. (1975); 5, Cade et al. (1971); 6, Pruett-Jones et al. (1980); 7, Enderson and Wrege (1973); 8, Blus (1984); 9, Henny et al. (1984); 10, Wiemeyer et al. (1988); 11, Spitzer et al. (1978); 12, Newton et al. (1986); 13, Newton et al. (1982); 14, Henny and Herron (1989); 15, Wiemeyer et al. (1993).

[b] Based on thickness of eggshell fragments and DDE content of shell membranes.

[c] Percentage of thinning based on thickness index (Ratcliffe, 1967); all others based on eggshell thickness.

[d] 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.

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.

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 to 16 μg/g in sample eggs of the white-faced ibis were associated with a decrease of 16% in thickness and 37% in strength (Henny and Bennett, 1990). 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.

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 8). Black ducks *(Anas 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 μg/g in eggs of treated birds (Longcore et al., 1971). Barn owls on a diet containing 3 ppm of DDE had hatchling and fledgling rates that were reduced about 75% from control values over 2 years when eggs contained an average of 12 μg/g the first year and 41 μg/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, 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.

Table 8 Residues of DDE in Eggs Related to the Reproductive Success of Birds on Experimental Dietary Dosages of DDE

			Residues (μg/g, wet weight)		Reproductive success (% of survival to 3 weeks posthatch)	
Species	Source[a]	Treatment	Mean	Range	Hatchlings	Embryonated eggs
Black duck	1	Control	0.28	0.14-0.67	91	38
		10 ppm	46	34-63	64[b]	23[b]
		30 ppm	144	96-219	50[b]	9[b]
					Mean per pair	
					Eggs hatched	Young fledged
Barn owl	2	Control (1st year)	0.25	NL[c]	3.2	2.9
		Control (2nd year)	0.40	NL	3.7	3.1
		3 ppm (1st year)	12	NL	1.1[b]	0.7[b]
		3 ppm (2nd year)	41	NL	0.9[b]	0.7[b]

[a] 1, Longcore et al. (1971); 2, Mendenhall et al. (1983).
[b] Significantly different ($P \leq 0.05$) from controls.
[c] NL, not listed.

Table 9 Mean Residues of DDE in Eggs Related to Mean Reproductive Success of Wild Birds

Species	Area[a]	Residues (μg/g, wet weight)	Young produced per active nest	Source[b]
Double-crested cormorant	CA	32[c]	0.0	1
	BC	24[c]	<0.1	1
	ON	14-16	0 to 0.1[d]	2
	ON	5	0.3	2
Black-crowned night heron	QU	2	2.4	3
Common loon	SA	6	0.7	4
Brown pelican	CA	59[e]	<0.1	5
	BC	66[e]	0.0	6
	BC	25[e,f]	<0.1	6
	BC	8[e]	0.1	6
	BC	3[e]	~0.8	6
Bald eagle	OR, WA	10	0.6	7

[a] CA, California; BC, Baja California; ON, Ontario; QU, Quebec; SA, Saskatchewan; OR, Oregon; WA, Washington.
[b] 1, Gress et al. (1973); 2, Weseloh et al. (1983); 3, Tremblay and Ellison (1980); 4, Fox et al. (1980); 5, Risebrough (1972); 6, Jehl (1973); 7, Anthony et al. (1993).
[c] Approximate conversion from dry weight basis.
[d] Five colonies.
[e] Approximate conversion from lipid weight basis.
[f] Authors suspected residues were too low because of analytical errors.

Some of the field data on reproductive success also follow the average residue-average effect design (Table 9). A method that gives more insight into the effects of residues on reproductive success is the sample egg technique (Table 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 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; Wiemeyer et al., 1984). 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 vs. residues (Fyfe et al., 1976; Spitzer et al., 1978), and another method ranks residues or a range of residues vs. young fledged (Blus et al., 1980, 1982; Henny et al., 1984, 1985, 1989; Ambrose et al., 1988; Wiemeyer et al., 1993). Of these two methods, I recommend the second, because 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.

Table 10 Residues of DDE in Sample Eggs of Wild Birds Related to Reproductive Success

Species	Area[a]	Residues (μg/g, wet weight)	Young produced per active nest[b]	Source[c]
Osprey	CT, NY	23[d,f]	0.0	1
	CT, NY	12[d,f]	1.0	1
	CT, NY	6[d,f]	2.1	1
	ID	14	0.0	2
	ID	6	1.6	2
Peregrine falcon	AK	≤15	1.8	3
	AK	15-30	2.0	3
	AK	>30	1.0	3
Snowy egret	NV	≤1	2.2	4
	NV	1-5	2.4	4
	NV	5-10	1.0	4
	NV	10-20	1.0	4
Prairie falcon	AB	2[d]	0.0	5
	AB	2[d]	1.0	5
	AB	2[d]	2.0	5
	AB	2[d]	3.0	5
	AB	1[d]	4.0	5
Merlin	AB	11[d]	0.0	5
	AB	11[d]	1.0	5
	AB	6[d]	2.0	5
	AB	5[d]	3.0	5
	AB	6[d]	4.0	5
Brown pelican	SC[g]	≤1.5	0.6 (FL),[h] 0.8 (EM)	6
	SC[g]	1.5-3	0.6 (FL), 0.8 (EM)	6
	SC[g]	>3	0.0 (FL), 0.6 (EM)	6
Bald eagle	US[f]	<2.2	1.0	7
	US[f]	2.2-3.5	1.0	7
	US[f]	3.6-6.2	0.5	7
	US[f]	6.3-11.9	0.3	7
	US[f]	≥12	0.2	7
Black-crowned night heron	US	≤1	2.0	8
	US	1-4	1.7	8
	US	4-8	1.5	8
	US	8-12	1.1	8
	US	12-16	1.0	8
	US	16-25	0.8	8
	US	25-50	0.4	8
White-faced ibis	NV	≤1	1.8	9
	NV	1-4	1.8	9
	NV	4-8	1.3	9
	NV	8-16	0.8	9
	NV	>16	0.6	9
Great blue heron	OR, WA	3	1.7-2.0	10

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

[b] Young produced not adjusted for sample egg collected.

[c] 1, Spitzer et al. (1978); 2, Johnson et al. (1975); 3, Ambrose et al. (1988); 4, Henny et al. (1985); 5, Fyfe et al. (1976); 6, Blus (1982); 7, Wiemeyer et al. (1993); 8, Henny et al. (1984); 9, Henny and Herron (1989); 10, Blus et al. (1980).

Table 10 (continued) Residues of DDE in Sample Eggs of Wild Birds Related to Reproductive Success

[d] Approximate adjustment from dry weight basis.
[e] Elevated levels (17 to 29 μg/g) of polychlorinated biphenyls also present.
[f] 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.
[g] Sample egg either freshly laid or embryonated when collected.
[h] FL, freshly laid; EM, embryonated.

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 to 2 μg/g were said to adversely affect fledging success (Table 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 to 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 vs. 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 10). One can determine the level at which residues first begin having an adverse effect on the number of young produced, for example, 4 to 8 μg/g in the white-faced ibis (Henny and Herron, 1989), and at which few or no young are produced, for example, ≥15 μg/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 μg/g (Henny et al., 1984). In the brown pelican, Blus (1982) indicated a dramatic effect above 3 μg/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 10). The brown pelican is apparently the most sensitive avian species to DDE, with reproductive failure when residues in eggs exceed 3.7 μg/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). In order 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, e.g., an approximate recruitment standard (number of young that must be fledged per pair of breeding age in order to maintain a stable population) and adult mortality compensating mechanisms, including renesting and other factors. Recruitment standards are 1.2 to 1.5 young for the brown pelican (Henny, 1972), 1.0 young for the bald eagle (Wiemeyer et al., 1993), 1 to 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 to 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 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 μg/g of technical DDT showed no significant effects on reproduction compared with controls, even though their eggs 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).

FOOD

The lowest dietary concentration of DDE that resulted in critical eggshell thinning and decreased production in the peregrine falcon was estimated at 1 μg/g (Enderson et al., 1982). A more recent study used 3 μg/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 μg/g on the basis of 31× 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-μg/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.

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 some time 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 μg/g of DDT, 5 μg/g of DDD, or 15 μg/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 paper, 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 μg/g of DDE in the egg and total reproductive failure when residues exceed 3.7 μg/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 μg/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 μg/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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