Arch. Environ. Contam. Toxicol. 13, 7–13 (1984)

Archives of Environmental Contamination and Toxicology

Aroclor 1254® Residues in Birds: Lethal Levels and Loss Rates

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Abstract. Lethal residues of polychlorinated biphenyls (PCBs) were determined experimentally in four species of wild birds given dietary dosage of 1,500 ppm of Aroclor 1254® until one-half had died, sacrificing the survivors, chemically analyzing the tissues, and comparing results in dead birds and survivors. For all species, residues of 310 ppm or higher in the brain showed increasing likelihood of death from PCB poisoning. Residues in dead birds did not differ among species except for starlings (*Sturnus vulgaris*), which averaged slightly lower than the others. However, the species differed in the length of time to 50% mortality and in the levels of PCBs in brains at sacrifice.

Concentrations in bodies and livers were not diagnostic when expressed on a wet weight basis. On a lipid basis, however, concentrations of PCBs in bodies of dead birds were higher than in sacrificed birds, but in both groups residues increased with time, suggesting that overlapping values could be expected.

Loss rates were followed in grackles (*Quiscalus quiscula*) fed 1,500 ppm PCBs for 8 days, then given untreated feed and sacrificed at intervals of 7, 28, 56, 112, and 224 days. PCB residues were lost from bodies at somewhat irregular rates; overall, the rate was estimated at 0.77% per day (half-life 89 days). Residues in brains generally were related to the percentage of body fat, but also showed a somewhat irregular pattern.

Residues of PCBs have been prevalent in the environment and in the bodies of wild animals for many years, and remain so despite regulatory actions designed to restrict their use (Kaiser *et al.* 1980; Ohlendorf *et al.* 1979). Toxicity and toxic effects differ greatly among species (Peakall 1972; Stendell 1976; Combs and Scott 1977); the most pronounced appeared in mink (Aulerich and Ringer 1977).

Knowledge of residues that could be considered lethal and the rates of loss of PCBs from wild bird species, however, is minimal. Our experiments were undertaken to evaluate lethal brain residues in several species and to estimate depletion rates from high levels of PCBs.

Methods and Materials

Test Organisms

Birds were captured on the Patuxent Wildlife Research Center and cage-conditioned for several weeks before studies were begun. They were kept in roofed outdoor cages; food (turkey starter crumbles) and water were provided *ad libitum*.

Experimental Procedures

Aroclor 1254® (obtained by open market purchase) was dissolved in Wesson oil and the solution was mixed into the diet to produce a dry-weight concentration of 1,500 ppm. Oil constituted 2% of both treated and untreated diets. Aroclor 1254® was selected for the study because chromatographic patterns of PCBs in wild birds resemble those of this mixture (or of Aroclor 1260®) most closely, and are used as the basis of quantification of residues in specimens taken from the wild.

Lethal residues were determined for four species: immature male common grackles (*Quiscalus quiscula*), immature female red-winged blackbirds (*Agelaius phoeniceus*), adult male brown-headed cowbirds (*Molothrus ater*) and immature female starlings (*Sturnus vulgaris*). Accumulation of PCBs on dosage and its loss after discontinuation of dosage were measured in grackles.

Dietary dosage of 1,500 ppm of Aroclor 1254® was given to 84 grackles beginning 16 November 1970. Four apparently healthy birds were sacrificed after 3, 5, and 8 days of dosage, the latter

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being the 50% mortality point among a designated monitor group of 42 birds from which no sacrifices were made. At that time, survivors were restored to an untreated diet. Birds from this group were sacrificed in sets of four after 7, 28, 56, and 112 days; eight were sacrificed after 224 days.

Dietary dosage of 1,500 ppm also was given to red-winged blackbirds, cowbirds, and starlings in groups of 15 birds each, beginning on 16 November. Ten birds in each group were the designated monitor group; when five of these birds had died, the five remaining birds were sacrificed.

Chemical Analysis

Brains, livers, and bodies (whole body less brain, liver, beak, feet, wings, skin, and gastrointestinal tract) were preserved in 10% chemically pure formalin until analysis at WARF Institute, Inc. (now Hazleton Raltech, Inc.), Madison, WI. Entire brains and livers and 15 to 20 g subsamples of homogenized bodies were dried with sodium sulfate and extracted for 16 hr on a Soxhlet® extractor, using approximately 200 ml of a solvent mixture consisting of ethyl ether:petroleum ether (7:17). The entire volume of formalin solution containing a brain or liver sample was extracted three times with a hexane:acetone mixture (41:59); the extracts were combined with the tissue extract, concentrated to 1–2 ml, and made up to 100 ml in hexane. The same method was followed for body samples except that the volume of formalin extracted was proportional to the weight of the tissue subsample taken for analysis. Because of the high concentrations of PCBs in the body samples, no cleanup procedure was used; an aliquot was diluted and injected directly. It was found necessary, however, to subject a small number of brain and liver samples to clean-up on a Florisil® column before analysis.

Gas chromatographic analysis employed a Barber Coleman Model 5000, with instrument conditions as follows: temperature 225°C (column), 255°C (injector), and 270°C (detector); column 8 ft by 2 mm glass packed with 2% Apiezon L and 0.02% Epon 1001 resin on 100/120 Gas Chrom Q; nitrogen carrier gas, pressure 30 lb/sq in. Quantification followed the method used by Fries *et al.* (1977) in a study of retention and excretion of PCBs by hens. In this method, the PCBs that were fed were used as the standard and quantities were calculated by comparing the total area under all the peaks of the samples with the total area under all the peaks of the standard. Fries *et al.* (1977) stated that this method was subject to some error because of differences in electron capture response of different components but that error would be systematic and not change major conclusions.

Results were not corrected for recoveries, which were 98 and 102% for two duplicate portions of a control sample fortified with Aroclor 1254® in hexane and allowed to equilibrate for four days. Reproducibility was checked by duplicate analysis of three brain samples, three liver samples, and five carcass samples. Coefficients of variation (standard deviation/mean) of residue determinations in duplicate samples averaged 3.3% (0.5–6.0%) for brain samples, 5.8% (1.5–11.2%) for liver samples, and 3.1% (1.8–6.7%) for body samples. PCBs in four pretreatment samples (one for each species) ranged from 0.9 to 2.1 ppm wet weight.

Lipids were determined from a 25 ml aliquot of the extract, which was placed in a tared vial; the solvent was evaporated and the vial containing the lipid was placed in a 40°C oven overnight; the vial was allowed to cool in a dessicator, then weighed, and the amount of lipid calculated. Lipid percentages had coefficients of variation of 3.7% (0.5–6.4%) for brain samples, 3.2% (3.0–3.4%) for liver samples, and 3.9% (0.7–10.4%) for body samples.

Results

Toxicity

The 50% mortality point for starlings was reached in 4 days, red-winged blackbirds in 6 days, cowbirds in 7 days, and grackles in 8 days. The only delayed mortality was one grackle that died 4 days after untreated feed was restored.

Signs of PCB poisoning began with birds becoming inactive and showing slight intermittent tremors. Tremors later became continuous and of moderate amplitude. Frequently the beak would be pointed upward in different degrees of opisthotonos. Some birds fell to the floor, had trouble walking, and sat fluffed and blinking. In two birds, the head was drawn to the right. At necropsy, the liver frequently had hemorrhagic areas. The gastrointestinal tract often had blackish fluid in all or part of its length, from the mouth back. These features were not consistent, however, and so would be of only confirmatory value in diagnosis.

Lethal Residues

PCB residues in brains of birds that died were distinct from those in sacrificed survivors, providing suitably diagnostic criteria (Figure 1). PCB residues (ppm wet weight) ranged from 349 to 763 ppm in brains of dead birds and from 54 to 301 ppm in sacrificed birds. An appropriate break point for high probability of PCB-induced mortality would be around 310 ppm (three standard deviations below the mean). PCBs in brains of cowbirds, red-winged blackbirds, and grackles did not differ significantly from each other; combined, they averaged 579 ppm (s.d. 117.7). Residues in starlings (367–469 ppm; mean 439; s.d. 42.8), although encompassed within the range of the others, averaged significantly lower than those in red-winged blackbirds and grackles, but did not differ significantly from those in cowbirds. PCB residues measured 593 ppm in the brain of the one grackle that died four days after untreated food was restored, a level near the middle of those in birds that died on dosage.

PCBs in brains of birds sacrificed at the 50% mortality point differed among species and was unrelated to the different lengths of time on dosage. At the 50% mortality points, PCB residues averaged

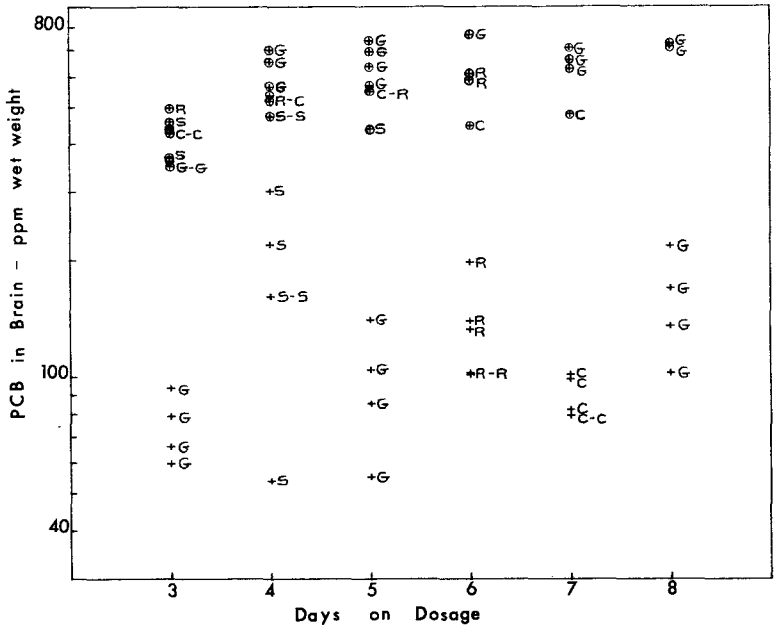

Fig. 1. PCB residues in brains of birds that died on dietary dosage of 1,500 ppm Aroclor 1254® (⊕, above) and in sacrificed survivors (+, below). Species symbols: starlings, S; red-winged blackbirds, R; cowbirds, C, and grackles, G

179 ppm (s.d. 90.6) in starlings, 134 ppm (s.d. 39.1) in red-winged blackbirds, 88 ppm (s.d. 10.7) in cowbirds, and 156 ppm (s.d. 49.1) in grackles.

PCB residues (ppm wet weight) in livers of grackles increased with time on dosage; at any given point in time, residues in dead birds were higher than in sacrificed birds, but they overlapped broadly overall (dead birds, 513 to 3,770 ppm; sacrificed birds, 144 to 1,970 ppm) and would be difficult to employ diagnostically. Equations for the regression of residues on dosage time were (1) for grackles that died: $\log_e Y = 5.849 + 0.258(\log_e X)$, $R^2 = 0.81$; and (2) for sacrificed birds: $\log_e Y = 4.159 + 0.344(\log_e X)$, $R^2 = 0.99$ (means) and 0.80 (individual values).

Ratios of brain residues to liver residues varied from 18.9 to 70.0% (mean 46.9 ± 3.6%) among grackles that died on dosage and 8.6 to 45.8% (mean 28.1 ± 3.8%) among sacrificed survivors; the ratio was 12.2% in the one bird that died four days after dosage was discontinued.

PCB residues (ppm wet weight) in bodies (including livers) increased with time on dosage. Residues in sacrificed birds generally were higher than in birds that died, although levels overlapped even at the same time points (dead birds 172 to 1,120, sacrificed birds 367 to 1,510). Equations for the regression of residues on dosage time were (1) for grackles that died: $\log_e Y = 4.465 + 0.314(\log_e X)$, $R^2 = 0.80$; and (2) for sacrificed birds: $\log_e Y = 5.527 + 0.211(\log_e X)$, $R^2 = 0.97$ (means) and 0.87 (individual values).

PCB residues in bodies on a lipid basis, however, were more clearly separate, although the increase of residues with time suggest that overlapping values could be expected (Figure 2). Residues ranged from 22,600 to 98,600 in birds that died and from 6,690 to 22,500 ppm in sacrificed birds. It follows that the ppm (lipid base) in the body is correlated with the ppm (wet weight) in the brain. The relationships, however, are expressed by different regressions for dead and sacrificed birds, and development of curves to provide dependable prediction of one from the other would require data beyond that available. Equations for the regression of ppm wet weight in the brain on ppm lipid weight in the body were (1) for dead birds: $\log_e Y = 1.778 + 0.426(\log_e X)$, $R^2 = 0.70$; and (2) for sacrificed birds: $\log_e Y = -3.163 + 0.836(\log_e X)$, $R^2 = 0.94$.

Loss Rates

PCB residues declined slowly, from a level of 1,300 ppm, on the day clean food was restored, to 169 ppm 224 days later (Figure 3). The rate of decline was irregular, however, and apparently was influenced by gain and loss of body fat. A linear regression of PCB concentration on days off dosage estimates a loss rate of 0.77% per day (half-life of 89 days) from day 0 to day 224.

This approximation to loss rates is influenced by differences in different compounds of the PCB mixture as well as by fat changes. In particular, the

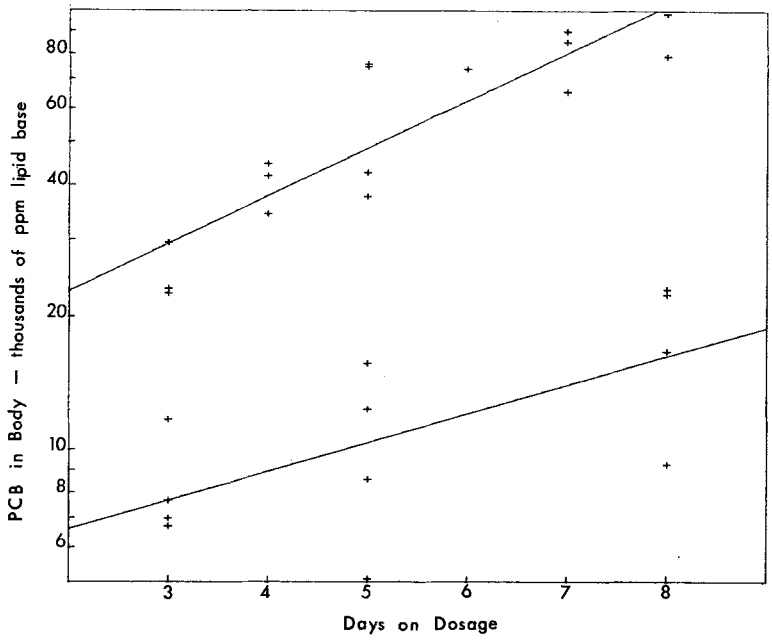

Fig. 2. PCB residues (ppm lipid) in bodies of grackles that died on dosage of Aroclor 1254® (upper curve) and in sacrificed survivors (lower curve). Regression equations for (1) birds that died: $\log_e Y = 9.536 + 0.250(\log_e X)$, $R^2 = 0.78$; and (2) sacrificed survivors: $\log_e Y = 8.491 + 0.151(\log_e X)$, $R^2 = 0.97$ (means), 0.44 (individual values)

conspicuous drop in concentrations between days 7 and 21 may have resulted from the more rapid loss of some of the compounds, as has been reported by others (Grant *et al.* 1974; Bush *et al.* 1974; Lincer and Peakall 1973). Gas chromatograms of our samples showed pattern changes of that sort, but quantitative evaluations were not feasible. In contrast, the increase in concentrations between days 56 and 112 evidently was the result of decreased body weight produced by pronounced loss of fat in that period.

PCB residues in the brain tended to decrease as body fat increased during the first 112 days, although the 28-day sample was an exception. Between 112 and 224 days, PCB residues were lost steadily from the brain as both body burden and percentage fat declined.

Discussion

Lethal Residues

PCB residues in birds that died in our study correspond well with those in most studies of other species:

Pheasants that died from sequential capsule-dosage of Aroclor 1254® had residues in the brain that ranged from 320 to 770 ppm (mean 520 ppm, s.d. 110) (Dahlgren *et al.* 1972). Nine sacrificed birds had 280 to 500 ppm (mean 140 ppm, s.d. 53). Three that died had residues below 400 ppm; these birds died first (1.3 to 1.9 days). Fifty % of the monitor group had died at 3.7 days. Three sacrificed birds had residues above 400 ppm. PCB residues in birds that died, therefore, were much the same as in our study, but residues in sacrificed pheasants were higher, almost all of them reaching levels that were lethal in the species we studied. Although the elevated levels in sacrificed pheasants suggest that they may already have received lethal doses, interpretation of levels below 400 ppm in pheasants should be cautious and carefully consider necropsy findings.

PCBs averaged 291 ppm wet weight in brains of five ring doves that were starved to death after 105 days of dietary dosage of Aroclor 1254® at 10 ppm (Lincer and Peakall 1973). PCBs in muscle tissue of these same birds averaged 172 ppm wet weight and 31,300 ppm lipid base.

Young cockerels (day-old at start) that died on dosage of 600 ppm Aroclor 1260® contained 270 and 420 ppm of PCBs in the brain (Vos and Koeman 1970), levels similar to those in our study. Dosage with other formulations that also contained 60% chlorine resulted in more variable residues. Residues in brains of five birds that died on Phenoclor® dosage ranged from 70 to 700 ppm and in four birds that died on Clophen® dosage ranged from 120 to 380 ppm. These other formulations also were more toxic and produced pathological signs that were absent from the birds fed Aroclor 1254®. Both of these formulations proved to be contaminated with chlorinated dibenzofurans (Vos *et al.* 1970).

Cormorants dosed experimentally with Clophen A60® also died with significantly lower brain resi-

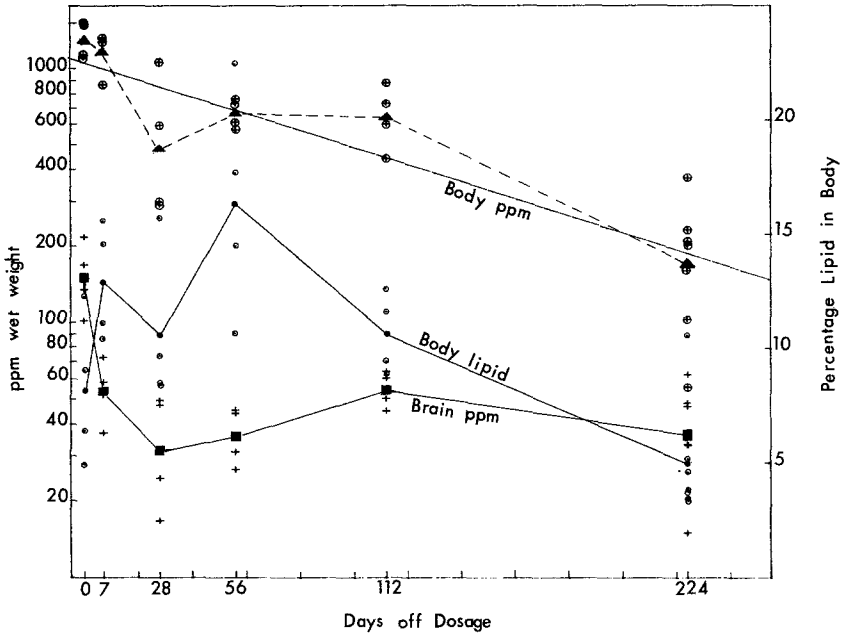

Fig. 3. Loss of PCB residues from bodies and brains of grackles following discontinuation of dosage, in relation to changes in percentage of body fat. Symbols: wet weight ppm in body, ⊕ (individual values), and ▲ (means); wet weight ppm in brain, + (individual values) and ■ (means); percentage lipid in body, ○ (individual values), and ● (means). Equation for the regression of ppm wet weight in the body on days off dosage: $\log_e Y = 6.966 - 0.008(\log_e X)$, $R^2 = 0.80$ (means), 0.70 (individual values)

dues (76, 115, 120, 160, and 180 ppm) (Koeman 1973; Koeman *et al.* 1973) than the grackles in our study, possibly the result of the toxic action of the dibenzofurans. It is possible also, however, that cormorants are especially sensitive to PCBs, because three herons that died on Clophen® dosage contained 420, 430, and 445 ppm of PCBs in the brain (Koeman 1973). The pathological signs reported for young cockerels were not present in either cormorants or herons.

In contrast, in another study with day-old cockerels, fed 500 ppm Aroclor 1254®, birds died with brain residues from about 80 to about 190 ppm and residues in liver and muscle also were low; all increased sharply with time on dosage (Platonow et al. 1973). Pathological examination revealed lesions and severe edema that did not occur in our experimental birds, suggesting that mortality was the result of different pathological factors. The signs, in fact, closely resembled those in birds dosed with PCB formulations that contained dibenzofurans as contaminants.

Corresponding to our results for grackles, PCBs in livers of Bengalese finches that died as a result of Aroclor 1254® dosage were 70 to 697 ppm, which did not provide diagnostic differences from the residues of 3 to 634 ppm in survivors (Prestt *et al.* 1970). PCB residues in brains were high in relation to those in livers of dead birds (83.7 ± 7.4%) and lower in survivors (26.4 ± 6.1%). The brain:liver ratio, however, did not provide helpful diagnostic criteria in our study.

In the field, PCBs were the probable cause of mortality of many ring-billed gulls that died in southern Ontario in late summer and early fall of 1969 and 1973 (Sileo *et al.* 1977). Among 54 gulls for which no disease-related cause of death could be determined, residues of PCBs in the brain exceeded 300 ppm (310 to 1,110) in 33 specimens. Although dieldrin and DDE were also present in most samples, DDE residues in all but one were well below lethal levels; dieldrin levels were 5 ppm or higher in six specimens. PCB residues were above 200 ppm in an additional 16 specimens, of which two also contained dieldrin residues of 5 ppm. Although additivity or other interaction among the chemicals appears to be implicated in mortality of some of the gulls, as suggested by Sileo, the levels of PCBs alone in most samples were sufficiently high to have caused mortality.

Loss Rates

PCBs were lost slowly from the bodies of grackles when untreated food was restored, but the rate of decline was irregular, apparently influenced both by differences in different compounds in the PCB mixture and by changes in body fat of the birds. The over-all loss rate of 0.77% per day (half-life of 89 days), calculated for the entire 224-day period, provides a generalized approximation.

Weight changes also affected disappearance rates of PCBs from fat and blood of growing lambs and pigs fed Aroclor 1254® (Borchard *et al.* 1976). Adjusting for weight gain, PCBs were lost more rapidly

from fat and blood of growing pigs fed Aroclor 1254® than from growing lambs. Estimated rates of loss from fat over a period of 70 days were 2.7% per day for pigs and 1.0% per day for lambs. Species differences were evident in relation to individual components as well as to total PCBs.

PCB levels in muscle of 5-day-old chicks that obtained the chemical from the egg were reduced only slowly in relation to the time that dosage of the hen had been discontinued (Bush *et al.* 1974); from the published graph, the rate appears to be about 0.5% per day. Loss of PCB residues from fat or eggs of laying hens fed Aroclor 1254® proceeded more rapidly, usually at a rate greater than 1% per day (Platonow and Reinhart 1973; Tumasonis *et al.* 1973; Teske *et al.* 1974; Fries *et al.* 1977).

Following single-capsule dosage of 50 mg Aroclor 1254®, and after the initial rapid clearance period, residues in muscle tissue of pheasants declined from 2.3 to 1.9 ppm between 7 and 28 days (approximately 0.9% per day) (Dahlgren *et al.* 1971).

PCBs were lost more rapidly from heart muscle of rats than from fat and more rapidly from higher initial concentrations than from lower ones (Grant *et al.* 1974). The half-life of PCBs during 182 days following a 62-day dosage of Aroclor 1254® at 100 ppm averaged 63 days in heart muscle and 125 days in fat. Concentration in muscle when dosage ceased was 5.98 ppm. When dosage (20 ppm) and initial concentrations (1.59 ppm) were lower, the half life was 115 days. Rats given 500 ppm of Aroclor 1254® for 4 weeks followed by untreated feed for 26 weeks had a residue half-life of 56 days in the fat tissue.

PCBs in dairy cows exposed to Aroclor 1254® in the diet were lost from milk at rates nearly identical to those for DDE (Fries *et al.* 1972). In dairy cows fed Aroclor 1254® for 60 days followed by uncontaminated feed for 60 days, PCBs declined slowly in body fat, with an average half-life of about 69 days. PCBs in milk fat dropped 50% in 15 days, then continued to be lost at approximately the same rate as in body fat (1.0% per day) (Fries *et al.* 1973).

Loss rates of PCBs in grackles in our study, however, were considerably more rapid and less uniform than loss of DDE in a parallel study conducted at the same time and with the same species, in which the half-life of DDE was estimated to be about 229 days (Stickel *et al.* 1984).

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Manuscript received March 21, 1983 and in revised form June 22, 1983.