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## PCB PHARMACODYNAMICS IN THE RING DOVE AND EARLY GAS CHROMATOGRAPHIC PEAK DIMINUTION

JEFFREY L. LINCER\* & DAVID B. PEAKALL

Section of Ecology & Systematics, Langmuir Laboratory, Cornell University,  
Ithaca, New York 14850, U.S.A

### ABSTRACT

*Ring Doves (Streptopelia risoria) were maintained on a daily dietary dose of 10 ppm of the polychlorinated biphenyl (PCB), Aroclor 1254. Sequential sampling and residue analysis of eggs indicated a linear increase in PCB levels until approximately 105 days, after which an asymptote was reached at 80 ppm, oven dried weight (OD). At equilibrium PCB residues (ppm, OD) were as follows: fat 197, liver 51.8, muscle 28.1, brain 25.4, faeces 2.27. At death, following starvation stress, residue levels increased by the following factors: liver 78, muscle 25, brain 56, faeces 5.5. No differential toxicity attributable to PCB mobilisation was observed in experimentals compared with controls. Experimental birds tended to lose relatively more weight than the controls. The high levels in the livers of our starvation-stressed birds compared with those found in the livers of seabirds involved in the Irish Sea kill suggests that PCB was not the primary factor in their death. Early gas-liquid chromatography peaks showed considerable diminution in all samples compared with Aroclor 1254 standard and this was particularly marked in muscle and fat samples.*

### INTRODUCTION

The widespread distribution of polychlorinated biphenyls (PCB) is well known. Their properties, uses, distribution, toxicity and the means of detection and quantification have been reviewed previously (Peakall & Lincer, 1970; Reynolds, 1971; Risebrough, 1971; Lincer, 1971; Peakall, in press). One of the most important properties of organochlorines is their high lipid-low water solubility. This property, combined with their considerable stability, governs their movements both within the

\* Present address: Mote Marine Laboratory, 9501, Blind Pass Road, Sarasota, Florida 33581, USA.

individual and the ecosystem. Within the individual this means that the bulk of the organochlorine is to be found in the adipose tissue. While stored the PCB is comparatively inert, but if the fat is mobilised then the organochlorine is released into circulation. Studies with organochlorine pesticides have shown that release of DDT (Bernard, 1966), dieldrin (Stickel *et al.*, 1969) and heptachlor (Stickel *et al.*, 1965) caused by reduced food intake can be fatal to birds. PCB, like the organochlorine pesticides, is able to accumulate up food chains (see Peakall & Lincer, 1970; Peakall, in press, for reviews). Holdgate (1971) considered that one of the most striking findings in the sea birds found dead in the Irish Sea area in the autumn of 1969 was the unusually high levels of PCB in their livers.

This paper is concerned with the distribution, metabolic transformation, storage and excretion of Aroclor 1254 (a PCB supplied by Monsanto) in the Ring Dove. In view of the coincidence of high PCB levels and bird die-offs such as that in the Irish Sea (Holdgate, 1971), of particular comparative interest are the PCB tissue residues resulting from known dosage and the possible lethal effects contributed by mobilisation of these stored poisons upon starvation stress. Because many metabolites of pesticides are known to be biologically active (e.g. parathion-paraoxon; DDT-DDE) and PCB undergo *in vivo* metabolic transformation (Koeman *et al.*, 1969a; Grant *et al.*, 1971; Nimmo *et al.*, 1971) this facet of the problem is also considered.

#### METHODOLOGY

For the first experiment, single pairs of doves were maintained in cages 115 × 134 × 229 cm, on 14 h light, 10 h dark photoperiod and at a temperature of  $20 \pm 1^\circ\text{C}$ . Aroclor 1254 was given in pellets of turkey grower mash (Agway) at a dosage of 10 ppm.

For the starvation experiment which followed, five doves were maintained per cage. The birds were fed between 15 and 50 g of food per cage on alternate days which led to a weight loss of approximately 10% per week. After three weeks of this regimen the birds were completely starved until death occurred. The objective was to mimic likely field conditions wherein birds obtained an inadequate amount of food and were finally too weak to search for food. The analytical procedure used was a modification by WARF (Wisconsin Alumni Research Foundation) of an earlier one (US Dept. of Health, Education & Welfare, 1970). Specifically, samples were dried for 48 h at 40–45°C, ground with a coarse anhydrous  $\text{Na}_2\text{SO}_4$ , placed in Soxhlet extractors and extracted for 8 h with a 1:3 mixture of ethyl ether and petroleum ether. The extracts were concentrated to 50 ml and a 5 ml aliquot was evaporated to dryness at 40–45°C for 2 h to arrive at an extractable fat estimate. Sample clean-up was accomplished with a Florisil column (USDHEW, 1970). As the second (15% ethyl ether in petroleum ether) fraction did not contain any PCB peaks, only the first (6% ethyl ether) fraction was analysed. Determination was

carried out on a Varian Aerograph model 2100 gas chromatograph equipped with a  $^{63}\text{Ni}$  electron-capture detector. Two 4 mm (id)  $\times$  2 m glass columns were used, the liquid phases and solid supports were: 1% QF-1 on Chrom. G A/W DMCS  $^{70}/_{80}$ ; 2% SE-30 on Chrom. G A/W DMCS  $^{70}/_{80}$ . Nitrogen gas flow rates of 35 and 150 ml/min were used, respectively. Injector, column and detector temperatures were 225°, 200° and 285°C, respectively. Although the dangers of varied detector response depending on degree of chlorination (Rote & Murphy, 1971) are recognised, it was felt that, for within-group comparison, quantification based on the combined height of only a percentage of the total number of peaks would be quite adequate. Therefore, peaks 7 and 10 on the QF-1 column and peaks 7 and 8 on the SE-30 column (Fig. 1) were used.

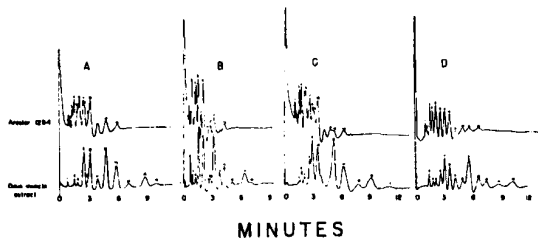


Fig. 1. Comparison of chromatograms of Aroclor 1254 (above) and extract of muscle from Aroclor 1254-dosed dove (below). (A) 1% QF-1 on Chrom. G A/W DMCS  $^{70}/_{80}$ , 4 mm (id)  $\times$  2 m, injector—238°C, column—203°C, detector—281°C, nitrogen flow—30 ml/min. (B) 1% silicone GE XE-60 on Varaport #30 100/120, 2 mm  $\times$  1.56 m, injector—238°C, column—203°C, detector—281°C, nitrogen—25 ml/min. (C) 4% SE-30 + 6% QF-1, Chrom. G  $^{60}/_{80}$ , 2 mm  $\times$  2 m, injector—250°C, column—225°C, detector—281°C, nitrogen—90 ml/min. (D) 2% SE-30 on Chrom. G A/W DMCS  $^{70}/_{80}$ , 4 mm  $\times$  2 m, injector—250°C, column—225°C, detector—281°C, nitrogen—90 ml/min.

In an attempt to elucidate PCB metabolism as reflected by chromatographic profiles of various tissues, egg and faeces samples, the relative diminution of the first two major peaks of QF-1 (Fig. 1A) was compared between sample types. A relative diminution value was calculated for each sample as follows:  $100 - [(100 \times (A/B))]$ ; where  $A$  is the ratio of the combined heights of peaks (4 + 6)/(7 + 10) in the sample and  $B$  is the same ratio in a similarly-concentrated standard of Aroclor 1254. Since there is evidence for a change in peak ratios over time (Grant *et al.*, 1971), peak diminution data are based on tissues presumed to be at equilibrium.

Except in the peak diminution experiment, samples were routinely measured on both columns and the lower of the two results chosen. Results agreed within

approximately 15%. Recoveries made in this laboratory of common organochlorines (e.g. p,p'-DDE, -TDE and -DDT) exceeded 88%. WARE's recovery values for Aroclor 1254 using this technique ranged between 85% and 90% (personal communication: D. L. Hughes). No corrections were made for recovery.

# RESULTS

Doves fed 10 ppm Aroclor 1254 usually produced a pair of eggs every ten days if eggs were removed as laid. The sequential sampling and analysis of these pooled eggs indicated a linear increase in residue levels until approximately 105 days after

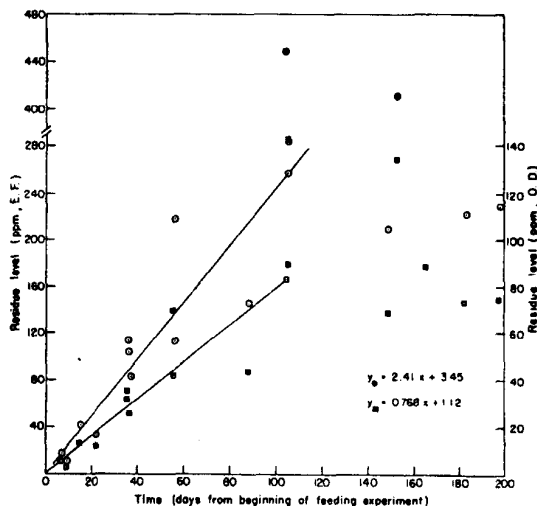


Fig. 2. Aroclor 1254 residues (ppm oven dry weight basis (dotted square) and extractable fat weight basis (dotted circle)) in dove eggs versus time (each point represents one residue value for two pooled eggs of a clutch; two points shown as (crossed square) or (crossed circle) were not considered in curve-fitting respective curves because they came from a single female which showed predisposition to laying eggs containing inordinately high residues). In the equation  $y =$  residues;  $x =$  time.

the onset of the experiment. The equations for that increase, based on the oven dry and extractable fat egg sample weights (OD and EF, respectively), refer only to the initial linear portions of the curves. After that time an asymptote was reached at approximately 80 ppm (OD) or 240 ppm (EF) (Fig. 2). Extractable fat in eggs was much more consistent within egg samples from the same female than between females. There was no apparent trend in extractable fat from the beginning of the experiment to 200 days, the average  $\pm$  standard deviation being  $31.7 \pm 1.18\%$  for 13 samples (two pooled eggs/sample).

Residue levels in tissues, eggs and faeces sampled from doves at this time of equilibrium and after starvation stress are shown in Fig. 3. PCB equilibrium residues (ppm, OD) were as follows: fat—797; eggs—73.7; liver—51.8; muscle—28.1;

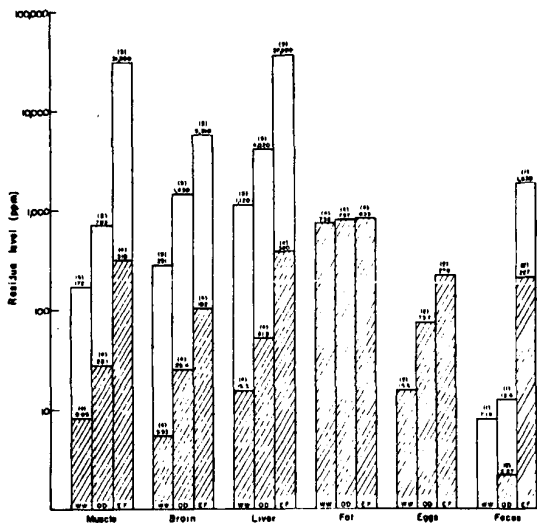


Fig. 3. Tissue egg and faeces residue levels of Aroclor 1254 at equilibrium (hatched sections) and after period of starvation (clear sections). Figures are in ppm based on wet weight (WW), oven dry weight (OD) and extractable fat (EF) of respective tissues. (Number of samples in parentheses, ppm, wet weight, for eggs estimated assuming 79% water.)

TABLE I  
AROCLOL 1254 RESIDUES IN TISSUES AND FAECES AT EQUILIBRIUM (A) AFTER STARVATION (B) AND THE RATIO OF N/A (C)

| Sample         | Muscle |                 |       | Brain |                 |      | Liver |                 |       | Fat |                 |     | Faeces |                 |      |
|----------------|--------|-----------------|-------|-------|-----------------|------|-------|-----------------|-------|-----|-----------------|-----|--------|-----------------|------|
|                | WW     | OD              | EF    | WW    | OD              | EF   | WW    | OD              | EF    | WW  | OD              | EF  | WW     | OD              | EF   |
| ppm based on   |        |                 |       |       |                 |      |       |                 |       |     |                 |     |        |                 |      |
| A              |        |                 |       |       |                 |      |       |                 |       |     |                 |     |        |                 |      |
| mean           | 8.05   | 28.1            | 318   | 5.53  | 25.4            | 102  | 15.3  | 51.8            | 380   | 736 | 797             | 833 | —      | 2.27            | 324  |
| std. dev.      | 2.72   | 6.04            | 121   | 1.85  | 8.88            | 36.5 | 9.91  | 35.9            | 774   | 253 | 273             | 296 | —      | 0.61            | 7.78 |
| std. error     | 1.36   | 4.52            | 60.3  | 0.92  | 4.44            | 18.2 | 4.96  | 17.5            | 137   | 126 | 136             | 148 | —      | 0.00            | 5.50 |
| EF $\pm$ s.d.† |        | 9.18 $\pm$ 2.91 |       |       | 25.5 $\pm$ 3.14 |      |       | 13.4 $\pm$ 0.17 |       |     | 96.1 $\pm$ 1.39 |     |        | 1.02 $\pm$ 0.03 |      |
| B              |        |                 |       |       |                 |      |       |                 |       |     |                 |     |        |                 |      |
| mean           | 172    | 703             | 31300 | 263   | 1430            | 5910 | 1120  | 4020            | 37200 | *   | *               | *   | 7.19   | 12.4            | 1830 |
| std. dev.      | 43     | 177             | 19900 | 31.5  | 125             | 900  | 422   | 1370            | 6450  | *   | *               | *   | —      | —               | —    |
| std. error     | 19     | 79.3            | 8500  | 14.1  | 15.8            | 402  | 188   | 613             | 3780  | *   | *               | *   | —      | —               | —    |
| EF $\pm$ s.d.† |        | 2.91 $\pm$ 1.34 |       |       | 24.5 $\pm$ 2.20 |      |       | 10.7 $\pm$ 2.48 |       |     |                 |     |        | 1.52            |      |
| C: B/A††       | 21.4   | 25.0            | 98.4  | 53.0  | 56.3            | 57.9 | 73.2  | 77.6            | 97.9  | *   | *               | *   | —      | 5.49            | 8.20 |

N = 4 for tissues at equilibrium; 5 for tissues after starvation period; 2 for faeces at equilibrium; 1 for faeces after starvation.

WW = wet weight; OD = oven dry weight; EF = extractable fat weight.

\* No fat available for analysis.

† Extractable fat (mean)  $\pm$  standard deviation.

†† Ratio of starvation equilibrium residue levels.

brain—25.4; faeces—2.27. Although the relationships of residue levels in various samples varied, depending partially on the amount of extractable fat, the above descending order remained, regardless of basis of ppm calculation. Extractable fat decreased in stressed birds to varying degrees, depending on tissue. There was no fatty tissue remaining in stressed birds. In terms of per cent of original extractable fat, there was 32% in muscle tissues of stressed birds, 80% in livers and 97% in brain tissues. These decreases tended to exaggerate the increase in residue levels in muscle tissue and, to a lesser extent, livers based on extractable fat. At death, following starvation stress, residue levels increased by the following factors (ratios based on ppm, OD): liver—78; muscle—25; brain—56; faeces—5.5 (Table 1).

With the exception of one control, all birds died within the same six-day period and gave no suggestion of differential mortality between groups. These was, however, an indication that the experimental group lost, on the average, more weight relative to their own original weight (Table 2).

TABLE 2  
WEIGHT CHANGE IN DOVES\* IN STARVATION EXPERIMENT  
FIGURES ARE MEAN  $\pm$  STANDARD DEVIATION (STANDARD ERROR)

|                                   | Controls              | PCB-fed               | Significance    |
|-----------------------------------|-----------------------|-----------------------|-----------------|
| Initial weight (g)                | 171 $\pm$ 9.78(4.37)  | 162 $\pm$ 7.65(3.42)  | ns at 0:10      |
| Weight at death (g)               | 86.8 $\pm$ 5.77(2.58) | 83.1 $\pm$ 8.13(3.64) | ns at 0:10      |
| Weight at death (% of initial wt) | 52.4 $\pm$ 2.18(0.97) | 47.3 $\pm$ 4.45(1.99) | signif. at 0:10 |

\* N = 5 for each group.

Until recently, the analytical picture concerning PCB has been clouded not only by the past lack of synthesising and characterising the individual peaks (Hutzinger *et al.*, 1971) but also the apparent differential metabolism of early-arising gas chromatographic peaks (Koeman *et al.*, 1969a; Heath *et al.*, in press; Grant *et al.*, 1971; Nimmo *et al.*, 1971). Although chromatogram profiles from dove sample extracts resembled each other superficially, the relative early peak diminution varied considerably, depending on type of sample (Table 3). Concurrent with the reduction or removal of the first three peaks came the appearance of three new peaks following what would normally be the last Aroclor 1254 standard peak. This same pattern emerged with each of the four different columns used (Fig. 1).

#### CONCLUSIONS AND DISCUSSION

PCB residues increased to, and levelled off at, 80 ppm (OD) or 17 ppm wet weight (WW), in eggs from doves fed 10 ppm Aroclor 1254 (Fig. 2). Scott *et al.* (1971) noted a similar levelling off of PCB residues at approximately 3 ppm (WW) in the eggs of chickens fed 10 ppm Aroclor 1248. A comparison of egg to fat residue levels

leads to the following ratios: 1:3.8 (EF); 1:11 (OD); 1:47 (WW). For comparison, Scott's group found the same ratio to be 1:12 (WW). Further comparison is difficult due to differences in both species and Aroclor used.

With regard to wild bird populations, these egg residue levels appear comparable to those reported by Jensen *et al.* (1969) for some marine birds and Prestt *et al.* (1970) for a variety of wild birds from many families. Dove liver residue levels (15 ppm, WW) were close to liver residue levels reported in birds of prey (Risebrough *et al.*, 1968; Koeman *et al.*, 1969b) but somewhat lower than those found in great crested grebe livers (Prestt & Jefferies, 1969).

The starvation stress experiment conducted on the doves and the subsequent mobilisation of stored fat resulted in an increase in liver PCB residues of almost two orders of magnitude (*i.e.* 15.3 to 1120 ppm, WW). Since there was no apparent differential toxicity between controls and experimentals and the liver levels of experimentals were roughly twenty times higher than those in livers from birds found dead after the Irish Sea wreck (*i.e.* average = 56 ppm; Holdgate, 1971) it appears that PCB was probably not the major cause of death in those wild birds. Due caution should be applied, however, as wide differences have been observed in the (1) toxic biological effects between different PCB (Street *et al.*, 1969; Ville-neuve *et al.*, 1971; Heath *et al.*, in press; Nagai *et al.*, 1971); (2) species sensitivities to toxicants (Heath *et al.*, in press); (3) ability of various organs to reflect the cause of death (Stickel *et al.*, 1966; Prestt *et al.*, 1970); and (4) analytical and quantitation techniques used for PCB (Peakall & Lincer, 1970; Lincer, 1971), the last of which alone can lead to a difference of over 100% (Rote & Murphy, 1971). In addition, the possibility of: synergistic or additive action of PCB and other environmental pollutants (Lichenstein *et al.*, 1969) or infectious diseases (Friend & Trainer, 1970); interactions affecting the retention of body residue burdens (Street *et al.*, 1966); gross reproductive effects (Dahlgren & Linder, 1971; Scott *et al.* 1971) and defects in proper embryonic development (Hays & Risebrough, 1972) stemming possibly from aberrant chromosome formation (Peakall *et al.*, 1972) must be considered very carefully before classifying PCB as innocuous.

The diminution of early-arising gas chromatographic peaks occurred in all tissues sampled, as well as eggs and faeces. The presence of late-arising 'metabolic' peaks from the various samples (Fig. 1) and the similarity of peak-diminution (Table 3) in the faeces (compared with other sample types) suggests that an *in vivo* transformation, possibly microsomal (Lincer & Peakall, 1970) dechlorination or hydroxylation, is taking place and not a differential excretion of those PCB components associated with early peaks. It is, of course, difficult to say whether later peaks are increasing, early peaks decreasing or a combination of the two. Early peak diminution was particularly acute in muscle (68.8%) and fat (70.8%) samples. Although one should be especially cautious in comparing chromatograms originating from different laboratories, there appears to be a basic similarity between those from the rat (Grant *et al.*, 1971) and ours from the dove (Fig. 1C) with some

TABLE 3  
EARLY PEAK DIMINUTION\* OF PCB'S (AROCLOL 1254) IN DOVE TISSUES, TOGIS AND FAECES. (EXPRESSED AS % CHANGE RELATIVE TO EQUIVALENT STANDARD; SAMPLE SIZES IN PARENTHESES.)

| Parameter | Muscle(4) | Brain(4) | Liver(5) | Fat(4) | Egg(4) | Faeces(2) |
|-----------|-----------|----------|----------|--------|--------|-----------|
| $\bar{x}$ | -68.8     | -58.3    | -57.4    | -70.8  | -54.0  | -54.5     |
| s.d.      | 6.85      | 3.86     | 3.13     | 2.63   | 9.49   | 0.71      |
| s.e.      | 3.42      | 1.93     | 1.40     | 1.31   | 4.74   | 0.50      |

\* See 'Methodology' for calculation of values in table and operating parameters.

notable differences (*i.e.* the lack of their prominent peak 2 in ours, and *vice versa* the lack of our peak 13 in theirs). The fact that this kind of early peak diminution has also been described in fish and invertebrate (shrimp) tissues (Nimmo *et al.*, 1971) suggests that this is a widespread mechanism among the animal kingdom. Considering the possibility of differential toxicity of individual components (Vos *et al.*, 1970; Yoshimura & Oshima, 1971) the need and desirability of investigations on the toxicity of individual components in PCB parent compounds, and especially metabolically-derived compounds, is evident. It would indeed be unfortunate if, after evolving the necessary apparatus to break down foreign compounds, an organism produced more toxic compounds, and thereby was killed.

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