been known that the inhibition of nuclear DNA synthesis by certain chemicals invariably blocks mitosis (DuPraw, 1968). Charnetski et al. (1973) reported a cessation of mitotic divisions in the apical meristems of treated pea roots resulting in enlarged cells with multilobed nuclei. Certainly a reduction of DNA synthesis and subsequent inhibition of mitosis could explain the stunted, dormant character of roots exposed to lindane. On the other hand, the reduction of DNA synthesis and mitotic abnormalities due to lindane may be related to disruptions of membrane or energy supply systems. Lichtenstein et al. (1962) found that lindane decreased the respiration rate in corn and oat root tips. Evidence of membrane abnormalities were found both in our study and that of Charnetski et al. (1973).

Data described above indicate that the insecticide lindane affected the synthesis of DNA in corn roots, possibly disrupting normal cell mitosis. This then seems to explain findings by others (Charnetski et al., 1973) who used microscopy and electron microscopy and showed the presence of enlarged root cells with greatly enlarged and lobed nuclei.

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Effects of Graded Levels of Toxaphene on Poultry Residue Accumulation, Egg Production, Shell Quality, and Hatchability in White Leghorns

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Graded levels of toxaphene (0, 0.5, 5, 50, and 100 ppm) were added to the diets of female (White Leghorn) chicks from 1 day of age. Each treatment consisted of 90 randomly selected birds (30 birds in each of three replicates). On the sixth week, 20 birds in each replicate were transferred to floor pens and continued into the egg laying phase. Two sexually mature White Leghorn roosters were placed in each pen during the 23rd week. The equations which best describe the dissipation of toxaphene from adipose and egg tissue are $\hat{Y} = e^{-0.0346 \times +4.70}$ and $\hat{Y} = e^{-0.0441 \times +2.498}$, respectively ($\hat{Y}$ = toxaphene level in ppm and X = withdrawal period in days). Birds fed 100 ppm toxaphene in their diet did not have significantly weaker shells as measured by Instron stress analysis. Birds fed 5, 50, and 100 ppm toxaphene exhibited sternal or keel deformation at 30 weeks of age. Histopathological examination revealed renal lesions in birds fed toxaphene at 50 and 100 ppm. Toxaphene at 100 ppm did not significantly alter egg production, hatchability, or fertility.

Toxaphene has been extensively used for insect pest control on crops and livestock for the past 15–20 years. In 1971, 37 million pounds of toxaphene (Andrilenas, 1970) was used by agriculture for control of insect pests. Despite the fact that toxaphene is used in larger amounts than other chlorinated hydrocarbon insecticides in the United States (Andrilenas, 1970), there is little information on several aspects of the chemistry, persistence, and environmental fate of this group of compounds.

With the banning of DDT and dieldrin, toxaphene–methyl parathion combinations will be used much more extensively. Toxaphene is registered for the use on agronomic commodities such as alfalfa, barley, corn, cotton, cow peas, sorghum, rice, rye, soybeans, wheat, and a variety of horticultural crops (EPA Summary, 1976). Since

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cottonseed meal, corn, rye, soybeans, soybean meal, and peanut meals are important commodities in the feeding of livestock and poultry, it is essential to identify the effects, if any, toxaphene may exert as it enters the food chain.

Studies to determine the accumulation or storage of toxaphene in cattle show that dermally absorbed or orally ingested toxaphene is stored in the fat in proportion to the amount administered (Bateman et al., 1953; Zweig et al., 1963). In contrast to their inability to eliminate other chlorinated hydrocarbons, domestic livestock appear to be able to eliminate toxaphene as soon as the toxaphene intake ceases (Bateman et al., 1953; Zweig et al., 1963). Since poultry concentrate chlorinated insecticide residues in organs and tissues to an extent several times that of cattle, hogs, and sheep (Gannon et al., 1959), a problem associated with exposure to chemical agents such as toxaphene is apt to first appear in the avian species. No information is available concerning the accumulation or distribution of toxaphene in the tissues of poultry. The specific objectives of these studies were: (1) to determine the extent of accumulation of toxaphene residues in the adipose tissues of young White Leghorn chickens; (2) to evaluate the effect of toxaphene upon egg production and hatchability of eggs; (3) to determine the extent of transmission of toxaphene into eggs; (4) to determine the depletion rate of toxaphene residues in the organs and tissues of egg-laying chickens.

MATERIALS AND METHODS

Feeding Trials. A total of 450 female, White Leghorn chickens were individually identified with numbered wing bands and weighed preceding the experimental trial and at weekly intervals thereafter for the first 8 weeks. The birds received continuous incandescent light. Feed and water were supplied ad libitum.

The birds were randomly assigned to five groups, 90 birds per group (30 birds in each of three replicates). The birds within each group received diets, from day old until 34 weeks, containing 0, 0.5, 5, 50, or 100 ppm of toxaphene. Toxaphene was incorporated into corn oil and this in turn was incorporated into the test feed (Table I).

The feed used for the mixing of the treated feed was analyzed for initial pesticide residue content and found to be free of contamination. The pesticide level in each lot of feed was determined by analysis to be within 10% of the desired value. Toxaphene as a formulated material (Tox-sol-6; 59.0% active ingredient, Woolfolk Chemical Works, Inc., Fort Valley, Ga.) was used to spike the feed.

Tissue Sampling. On weeks 4 and 8, three birds from each of the three replicate groups were sacrificed by cervical dislocation, and liver, kidney, heart, skeletal muscle, and abdominal fat tissues were collected for residue analysis. Tissues were quick frozen and stored until residue analysis could be completed. In addition, tissues (i.e., liver, heart, lung, kidney, brain, pancreas, adrenal glands, small intestine, and gonads) were collected and fixed for histopathological evaluation.

Egg Production. The remaining birds were transferred to floor pens and continued into the egg laying phase. Egg production ordinarily begins at about 20–22 weeks of age. During the 23rd week, two healthy White Leghorn roosters were placed in each pen (one rooster per ten hens). An Instron Quasistatic loader (Instron Corporation, Canton, Mass.) which measures the force (expressed as kilograms of breaking force) required to break the shell was used to measure breaking strength.

The effect of toxaphene upon hatchability was determined by collecting and setting a hundred eggs from each of the five treatment groups on Monday of 6 consecutive weeks commencing with the 28th week.

Table I. Grower, Layer Rations Calculated Analysis (Formulated)[b]

	Grower	Layer
Metabolizable energy, kcal/lb	1256.00	1349.00
Protein, %	14.60	16.71
Fat, %	5.02	4.40
Fiber, %	8.02	3.20
Ca, %	1.01	2.90
P (available), %	0.396	0.392
Vitamin A (IU), added	2000.00	2000.00
Vitamin D_3, ICU, added	400.00	400.00
Riboflavin, mg, total	2.93	2.97
Pantothenic acid, mg, total	8.86	7.33
Niacin, mg, total	28.04	29.14
Choline, mg, total	588.10	587.10
Vitamin B_{12}, mcg, added	3.00	3.00
Methionine, %	0.255	0.300

Ingredients	Grower, %	Layer-Breeder, %
Ground yellow corn	32.525	67.525
Ground oats	50.00	
Soybean meal (dehulled)	5.00	15.00
Alfalfa meal (17% dehy.)	3.75	3.75
Meat and bone meal (50)	6.00	6.00
Poultry fat	1.00	1.00
Salt (NaCl)	0.50	0.50
Defluorinated phosphate	0.25	0.25
Ground limestone	0.625	5.625
Vitamin premix[a]	0.25	0.25
Trace mineral mix[a]	0.10	0.10
	100.00	100.00

[a] Trace mineral mix contains: 6% Mn, 0.04% Co, 2.5% Fe, 0.2% Ca, 2.0% Zn, 0.12% I. [b] Vitamin premix provides (per lb of feed): 2000 IU of vitamin A, 400 ICU of vitamin D, 5 IU of vitamin E, 2 mg of riboflavin, 4 mg of pantothenic acid, 20 mg of nicotinic acid, 100 mg of choline Cl, 1 mg of vitamin B_6, 1 mg of menadione S.B., 1 mg of thiamine, 3 mcg of vitamin B_{12}, 250 mcg of folic acid, 50 mcg of biotin, 57 mg of ethoxyquin.

Withdrawal Study. During week 32, 12 eggs were collected from each of the replicate groups to determine the transmission, if any, of toxaphene or its major components. During week 34, toxaphene was removed from the diet of all birds. Eggs were collected weekly for 8 weeks to determine the depletion rate of toxaphene and its metabolites. Three birds per replicate were also sacrificed weekly for the collection of tissues for residue determination.

Sample Cleanup. Toxaphene levels in the test feed were determined by transferring 5 g of uniformly mixed feed into a 500-mL Erlenmeyer flask fitted with a ground glass stopper. One hundred milliliters of 15% ether in hexane was added and the flask was shaken on a reciprocating shaker for 15–20 min. The supernatant was transferred to 100-mL centrifuge tubes and centrifuged (Model EXD International Centrifuge equipped with heads to accommodate 15- or 100-mL centrifuge tubes) for 15 min at half speed. The resulting supernatant was transferred to 250-mL boiling flasks and taken to dryness on a rotary evaporator (40 °C). The extract residue was made to 10 mL with gel permeation chromatography (GPC) solvent (75:25, ethyl acetate–toluene) and the fat was removed by GPC (Johnson et al., 1976).

Adipose tissue was heated and a 1-g sample of the liquified fat was transferred to a graduated culture tube and made to a total volume of 10 mL with ethyl acetate–toluene (3:1). The tubes were well mixed with a vortex mixer and then centrifuged for 15 min (half speed) to

Table II. Recovery of Toxaphene from Various Tissues Analyzed[a]

Type of sample	Fortification, ppm	Recovery %
Feed	3.6	68.3
Eggs	0.9	73.4
Breast muscle	3.6	71.3
Leg muscle	3.6	67.2
Fat	18.0	88.3
Spiking solution (GPC clean up only)	2.0	93.0

[a] Samples were spiked with 1 mL of hexane containing 20 μg of toxaphene-^{36}Cl (specific activity, 98 dpm/μg). The tissues were extracted as previously described in this paper and radioactivity determined by liquid scintillation counting. Correction was made for quenching by addition of an internal standard.

remove insoluble materials. Fat was removed by GPC (Johnson et al., 1976).

After thawing, 5 g of muscle tissue was weighed into a high-speed blender jar. Approximately 50 g of Na_2SO_4 and 300 mL of ethyl acetate were added, and the sample was blended for 5–10 min. The homogenate was filtered with suction through a 9-cm Buchner funnel fitted with Reeves Angel glass filter paper into a 500-mL suction flask. The filtrate was transferred to a boiling flask and taken to dryness using a rotary evaporator (50 °C). The extract residue was made to 10 mL with ethyl acetate–toluene (75:25) and the fat was removed by GPC (Johnson et al., 1976).

Three to six eggs from each replication were broken and beaten together. A 20-g aliquot of thoroughly mixed yolks and whites was transferred into a high-speed blender jar, and 200 mL of ethyl acetate and 50 g of Na_2SO_4 were added. The eggs were blended for 5 min, and the homogenate was filtered with suction through a 9-cm Buchner funnel fitted with Reeves Angel glass fiber filter paper into a 500-mL suction flask. The filtrate was transferred to a boiling flask and taken to dryness using a rotary evaporator (50 °C). The extract residue was made to 10 mL with ethyl acetate–toluene (75:25), and the fat was removed by GPC (Johnson et al., 1976).

GPC Preparation. An automated GPC AutoPrep 1001 (Analytical Biochemistry Laboratories, Inc., Columbia, Mo.) equipped with a 25 × 270 mm column containing 40 g of 100 to 200 mesh Bio-Beads SX-3 was used for cleanup of tissue extracts. The GPC AutoPrep 1001 was operated under the following conditions: ethyl acetate–toluene (3:1); solvent flow rate, 4.5 mL/min; dump time, 20 min; collect time, 20 min; wash time, 10 min.

Gas-Liquid Chromatography. Gas-liquid chromatography (GLC) analysis was conducted with a Tracor Model 220 Gas Chromatograph which was equipped with a ^{63}Ni electron-capture detector, meeting AOAC 29.018 sensitivity requirements. Operating conditions used for the column were as follows: 1.5% OV-17, 1.95% OV-210 on Chromosorb W (100–200 mesh); column temperature, 200 °C; carrier flow rate, 70–80 cm^3/min; detector temperature, 230 °C.

Toxaphene levels were determined by comparison of peak height of sample chromatograms to those of an analytical toxaphene standard. The average of the three peak heights (RRT aldrin = 3.40, 4.11, 5.64) was taken as the toxaphene response (Gaul et al., 1966).

A reagent blank and a spiked sample were included with each set of analyses, and values were corrected for percent recovery. Samples spiked with radioactive toxaphene-^{36}Cl at the time of homogenization gave average percentage recovery of greater than 70% for feed, muscle, egg, and fat samples (Table II).

The data were evaluated using analysis of variance with significant differences between treatment means determined by the multiple range test of Duncan (1955). The level of significance chosen was $P \leq 0.05$.

RESULTS AND DISCUSSION

Feeding toxaphene at levels up to 100 ppm did not produce toxicity symptoms in White Leghorn chickens throughout the 50-week study. Mortality was minimal, with all groups including the control group, having less than 5% mortality. Body weight at 6 and 30 weeks was significantly decreased when the birds were fed 100 ppm toxaphene in their diet (Table III). There were no significant treatment-related changes in heart, liver, gizzard, or kidney weight at 4 or 8 weeks.

Necropsy of 31-week old birds fed 5, 50, and 100 ppm toxaphene in their diet revealed sternal deformation resembling osteomalacia. Occasional keel deformation involving the cartilaginous tissue as well as an apparent increase in the growth of cartilage was found in birds fed 0.5 ppm toxaphene in their diet. No keel deformation was observed in the control birds. Dietary Ca and P were analyzed and found to be 3% Ca and 0.6% P, respectively, in both treated and control rations.

Deformation of the cartilaginous region of the keel was of particular interest in the birds of this study. It has been reported in fish that an exposure to toxaphene results in the reduction of backbone collagen and an increase in the calcium and phosphorus concentrations (Mehrle and Mayer, 1975a, 1975b). The concentration of hydroxyproline as well as seven other amino acids was significantly decreased in collagen from the backbone of fish exposed to toxaphene. Radiographic examination of the fish indicated that toxaphene induced a weakened, fragile backbone. Future studies are planned to determine whether or not the backbone of the chicken as well as other skeletal structures are affected by toxaphene.

Histopathological examination of organs from 31-week old birds revealed mild nephrosis of the kidney of birds fed toxaphene at 5, 50, and 100 ppm. Kidneys were found to contain cysts resulting from expansion of the glomerular space. Occasional renal tubules contained cellular casts. There was also an accumulation of a brown granular

Table III. Effect of Graded Levels of Toxaphene on Body Weight of White Leghorn Chickens[a]

Dietary toxaphene, ppm	3 weeks (g)		6 weeks (g)		30 weeks (g)	
	$\bar{X}$	α	$\bar{X}$	α	$\bar{X}$	α
0	180.2	15.2 a	445	48.0 a,b	1854	184 a
0.5	182.9	15.8 a	447	36.0 a	1833	158 a,b
5	182.5	45.6 a	428	37.0 c,d	1727	245 b
50	166	16.0 a	434	34.8 a,b,c	1805	136 a,b
100	168	17.5 a	420	38.4 d	1759	172 b

[a] Dosages of 0, 0.5, 5, 50, and 100 ppm toxaphene were added to the diet of 1-day old birds. Treatment means in a column not followed by the same letter are significantly different at the 0.05 level according to Duncan's multiple range test.

Table IV. Effect of Toxaphene in the Diet on Egg Production, Egg Tissue Residues, Hatchability, and Shell Strength[a]

Dietary toxaphene, ppm	Residues in eggs, ppm	Egg production, eggs per hen/day	% hatchability	Shell strength[b]
0	<0.2	0.786 a	90.7 a,b	2.79 b
0.5	<0.2	0.836 a	88.7 a,b	2.67 a,b
5	0.7	0.732 a	94.0 b	2.48 a
50	5.0	0.759 a	84.9 a	2.75 b
100	13.9	0.772 a	90.2 a,b	2.73 b

[a] White Leghorns were continuously fed graded levels of toxaphene for 32 weeks. Starting with the 26th week, daily egg production records were kept and 100 eggs per treatment per week for 6 weeks were set to determine percent hatchability. Treatment means in a column not followed by the same letter are significantly different at the 0.05 level according to Duncan's multiple range test. [b] Expressed in kilograms of breaking force.

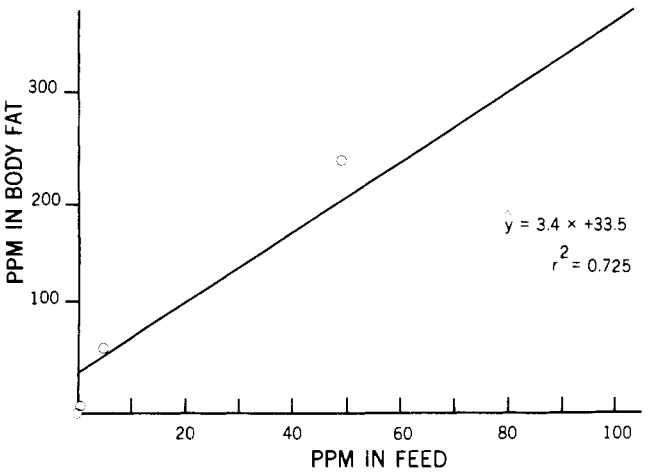

Figure 1. Toxaphene accumulation in adipose tissue of 8-week old White Leghorns as a function of toxaphene in the feed; the graded levels (0.5, 5, 50, and 100 ppm) of toxaphene were fed for 8 weeks.

pigment in the cytoplasm of some tubular epithelial cells. All other organs examined appeared normal.

In agreement with the work of Arscott et al. (1976), toxaphene at 100 ppm did not significantly alter egg production or hatchability of fertile embryos (Table IV). Shell strength at the 100 ppm feeding level was not altered (Table IV).

The level of toxaphene in the excisable adipose tissue of 8-week old birds increased with increasing dietary toxaphene as described by the equation: residue = $a + bx$ where x is the level of toxaphene in the feed (Figure 1). Assuming 2.5 lb of feed per pound of weight gain, a 1 lb White Leghorn would have consumed 2.5 lb of feed containing 5 ppm toxaphene or 5681.6 μg. If an 8-week old bird contains approximately 10% fat (Edwards et al., 1973) then a residue level of 125 ppm would be expected in adipose tissue. Birds fed 5 ppm toxaphene actually contain 62 ppm toxaphene. Thus only 50% of the estimated toxaphene that was ingested can be accounted for in the adipose tissue of the birds (Figure 1). Toxaphene accumulates in adipose tissue to a concentration of three to four times over that in the feed. However, this still accounts for only 41% of the toxaphene consumed as compared with 100% for HCB (unpublished results) and 50% of the PCB (Britton and Charles, 1974).

The accumulation of toxaphene in the tissues of older birds (i.e., 32 weeks of age) which were fed the chemical

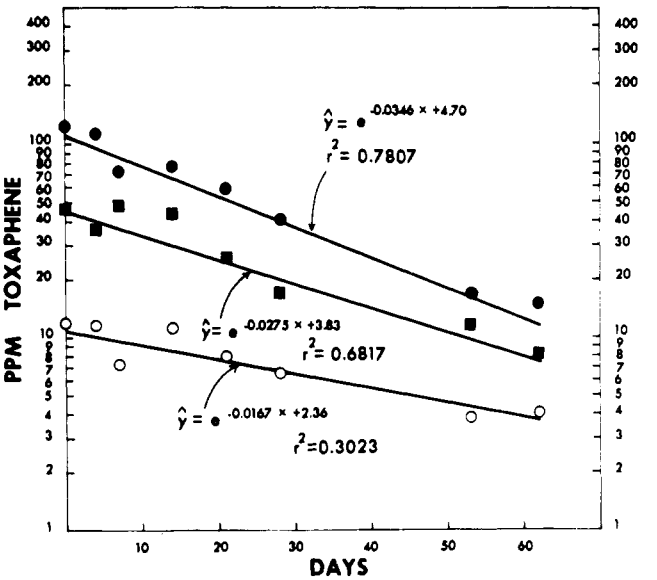

Figure 2. Depletion or elimination of toxaphene from adipose tissue of 32-week old White Leghorns that had been fed a continuous level of toxaphene. At the end of 32 weeks the birds were transferred to feed free of toxaphene. (Toxaphene levels in medicated feed: 100 ppm, 50 ppm, 5 ppm, 0.5 ppm.)

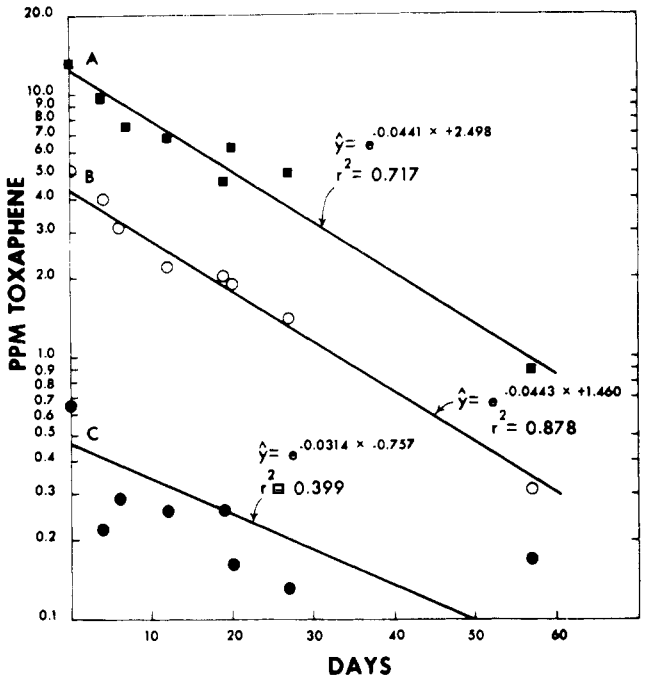

Figure 3. Toxaphene levels in eggs from White Leghorn chickens placed on untreated feed after 32 weeks, the birds were transferred to feed free of toxaphene; levels of the chemical in the eggs were monitored periodically. (Toxaphene levels in medicated feed prior to withdrawal: 100, 50, 5, 0.5 ppm.)

throughout their life span was much less than in young birds. Thirty-two week old birds accumulated toxaphene in the adipose tissues at a level approximately equal to the level of toxaphene in the feed at time zero (Figure 2). These results suggest that mature White Leghorns have a mechanism for metabolism and/or excretion which facilitates the degradation and elimination of toxaphene at a rate comparable with dietary intake. Although the role of the microsomal enzyme system in the degradation of toxaphene in birds is unknown, it may be that microsomal enzyme activity in the young bird is undeveloped which could account for greater accumulation of toxaphene

residues compared with the older birds. Significant residues were detected in eggs from birds fed 5, 50, and 100 ppm following the continuous feeding of toxaphene (Figure 3). At a sensitivity of 0.2 ppm, no residues of toxaphene could be detected in eggs from birds fed 0.5 ppm.

Cummings et al. (1966), Smith et al. (1970), and Herrick et al. (1969) found that there is a direct proportion between the peak or plateau level of lindane, heptachlor, dieldrin, and endrin residues in the eggs and the level of corresponding pesticide in the feed. The plateau levels of dieldrin and heptachlor epoxide in eggs approximate that in the feed. In contrast, however, toxaphene levels in the egg were approximately 12% of the level in the feed (Table IV) which suggests that toxaphene is not as readily transmitted into the egg as other chlorinated hydrocarbons.

To determine the length of time necessary for depletion of chlorinated residues from abdominal fat and eggs contaminated with insecticide residues from feeding of toxaphene on a continuous basis, 12–20 birds from each treatment were transferred to a basal diet containing no detectable toxaphene. The half-life of toxaphene in adipose tissue was found to be 20.0, 25.2, and 41.5 days (Figure 2) for birds fed 100, 50, and 5 ppm toxaphene, respectively. Half-life values of 15.7, 15.6, and 20.0 days were found for the depletion of toxaphene residues in eggs (Figure 3).

When the withdrawal data are plotted using a linear regression on the natural logarithm of the adipose residue level characteristic of drug depletion or withdrawal, the equation of the line $y = e^{ax + b}$, "a" is a measure of the rate of decrease of pesticide residue. Upon calculating values of "a" for withdrawal in adipose residues from the data of Stadelman et al. (1965), the following values are obtained: lindane, –0.0028; dieldrin, –0.00975; heptachlor, –0.0156; DDT and metabolites, –0.0147. Thus, the values of –0.0167 to –0.0346 for toxaphene (Figure 2) indicate that toxaphene depletion from poultry tissue is faster than for the other chlorinated pesticides.

Calculation of "a" for pesticide dissipation in eggs when hens are placed on clean feed from the data of Cummings et al. (1966) revealed values as follows: dieldrin, –0.0043; heptachlor epoxide, –0.0017; endrin, –0.005; lindane, –0.056; and DDT, –0.0033. The value of –0.040 obtained for toxaphene in this study indicates that toxaphene depletion from the egg occurs at a faster rate than DDT, dieldrin, heptachlor epoxide, or endrin, but not as rapidly as lindane.

The results of the present experiments, therefore, suggest that poultry have a mechanism for metabolism and/or excretion of toxaphene. A possible mechanism for toxaphene metabolism has been shown in rat liver microsomes containing P-450. These preparations have been shown to dechlorinate toxaphene to form more polar hydroxytoxaphene (Khalifa et al., 1976; Conley, 1952) which may in turn be excreted as the free alcohol, glucuronide conjugates, or sulfate conjugates (Ohsawa et al., 1975). The cytochrome P-450 system responsible for the dechlorination of DDT is stimulated by the presence of chlorinated pesticides; inasmuch as toxaphene is a chlorinated compound, it may also stimulate this enzyme system and may in turn be dehalogenated by this system.

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