Dieldrin Poisoning of Chickens During Severe Dietary Restriction[1]

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Since chlorinated hydrocarbon insecticides such as dieldrin are very persistent, trace amounts of these insecticides are now commonly found in our environment. These insecticides are found in the body fat of many domestic and wild animals. The knowledge that insecticides are present in body fat has led to conjecture that starvation of birds brought about by inclement weather or migration would cause metabolism of body fat with simultaneous release of the stored insecticide, thereby creating poisoning. Similarly, a potential hazard might exist in people, particularly those employed in insecticide manufacture or application. Should large residues of chlorinated hydrocarbon insecticides accumulate in their body fat, any sudden reduction in body weight might prove hazardous.

Bernard (1) reported that sparrows fed a diet containing 300 ppm DDT died within 17 hours after feed was removed, whereas none of the sparrows fed a control diet died in that period of time. Seifert et al. (2) also found that chickens fed diets containing 20, 40, or 80 ppm dieldrin died sooner than controls during prolonged fasting. Feed consumption was not controlled in either of these studies. However, it was observed that chickens fed diets containing more than 20 ppm dieldrin drastically reduced their feed intake and they lost weight (2). Assuming that the sparrows may have reacted to the high level of dietary DDT in a similar manner to the dieldrin-fed chickens, it becomes obvious that any relationship established between death during fasting and the insecticide residues accumulated in body tissue would be confounded with stresses imposed by reduced feed consumption before fasting.

Although mobilization of insecticide residues is often suspected in causing the deaths of wild birds, the chicken, as a manageable domestic bird, was used to study the accumulation of dieldrin in body fat and to determine the toxicity of the accumulated dieldrin under severe diet restriction.

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Methods

Experiment 1. Forty-eight white Leghorn hens were divided into four equal groups and fed individually the following diets for 12 weeks: (a) commercial ration ad libitum, (b) commercial ration restricted to intake of rations c and d, (c) commercial ration containing 10 ppm dieldrin,[2] and (d) commercial ration containing 20 ppm dieldrin.[2] Hens fed diets b, c, and d were arranged in 12 sets; and twice weekly feed intake was adjusted so that the three hens within a set were eating at a rate established by the lowest consumer of the set during the preceding period. Therefore, at least one hen in the set was eating ad libitum for a 3- or 4-day period and feed consumption could fluctuate.

Biopsies of abdominal fat were taken at 0, 4, 8, and 12 weeks from the beginning of the experiment. Feed was removed at the end of 12 weeks and 1-ml blood samples were then taken at weekly intervals from the heart of survivors. The carcasses were ground and lyophilized.

Experiment 2. Sixty white Leghorn hens were randomly alloted to five groups of 12 hens each and fed diets containing 0, 1, 5, 10, and 20 ppm of dieldrin.[2] They were fed individually 75 g of feed per day for 12 weeks and then the feed was removed. At death, the carcasses were ground and lyophilized.

Water was supplied ad libitum during all phases of both experiments.

Analytical. Dieldrin and fat were extracted from the lyophilized fat and carcasses with petroleum ether on a Soxhlet extractor. Portions of this extract were then cleaned up by the florisil method of Johnson (3). Eluting efficiency of dieldrin from the florisil column was checked on all samples by adding a small amount of dieldrin-^{14}C to the extract before cleanup, and the effluent from florisil was corrected to 100% recovery for the radioactivity. Portions of the florisil eluate were dried under a stream of nitrogen at 37°C and the residue dissolved in hexane. Dieldrin in this hexane solution was determined by electron capture gas chromatography.

Dieldrin in blood was determined directly by the method of Crosby and Archer (4). Dry matter in fat and carcasses was determined by loss in weight after lyophilization, and fat was determined by loss in weight after extraction with petroleum ether.

Statistical. The data were analyzed statistically by least squares analysis of variance. Dieldrin levels were treated as discrete effects, and time was treated as a continuous variable. Data not statistically significant at probabilities of 5% or less are not discussed as treatment related.

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[2] Supplied as technical dieldrin, but enough was used to supply 1, 5, 10, or 20 ppm of the active ingredient 1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1,4-endo-exo-5,8-dimethanonaphthalene.

TABLE 1

Feed consumption, body weights, survival, egg production, carcass composition, and dieldrin residues of hens in experiment 1[1]

Dietary regime	Ad libitum	Equalized intake		
		Control	Dieldrin 10 ppm	Dieldrin 20 ppm
Feed per hen per day, g	86.9	77.8	78.0	77.7
Average body weights, g				
Initial	1610	1561	1629	1597
Week 4	1727	1589	1671	1555
Week 8	1650	1503	1586	1417
Week 12	1732	1561	1654	1527
At death	716[a]	702[a]	871[b]	906[c]
Survival after feed removal, days	38.8[a]	33.0[b]	28.3[b]	18.9[c]
(range)	(27-52)	(14-42)	(8-40)	(3-36)
Eggs produced per hen, no.	15.9	15.8	13.0	14.0
Dry matter in carcasses,[2] %	31.1	32.5	34.0	35.7
Fat in carcass dry matter,[2] %	13.4[a]	13.1[a]	22.7[b]	23.1[b]
Dieldrin in carcass dry matter,[2] mcg/g	0.4[a]	0.5[a]	127.0[b]	261.4[c]
(range)	(0.1-0.8)	(0.0-1.8)	(81-186)	(156-408)
Total dieldrin per carcass,[2] mg	0.1	0.1	37.6	84.5

[1] Values with different superscripts are significantly different, $P < 0.05$.
[2] Data pertain to carcasses of hens which died during fasting.

Results

Experiment 1. Tabular data from the first experiment are given in Table 1. Hens fed ad libitum consumed 9 g more feed per day than hens on the controlled feeding regimes. However, significant changes in body weight did not occur among treatments during the feeding phase of the experiment. Similarly, number of eggs produced was not significantly affected by treatment.

Accumulation of dieldrin in body fat is shown in Figure 1. Dieldrin accumulation was directly related to amount of dieldrin consumed. The rate of accumulation for each level of dieldrin fed diminished with time. With the loss in body weight following removal of feed, the amount of dieldrin in body fat was concentrated, so that the level of dieldrin remaining in body fat at death was markedly higher than the level of dieldrin in abdominal fat at the start of fasting (week 12).

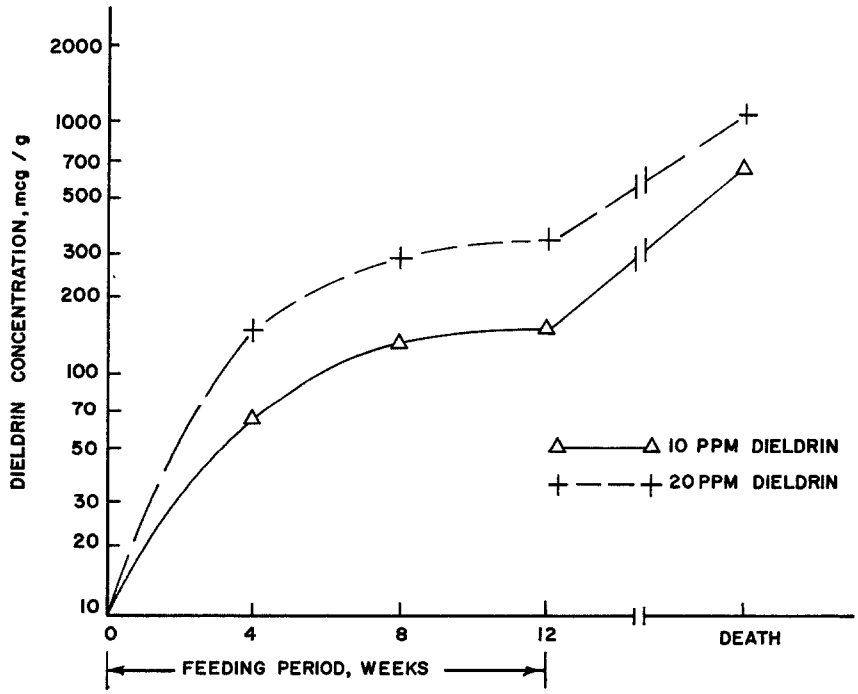

Figure 1. The accumulation of dieldrin in the abdominal fat of chickens during the feeding phase of experiment 1, and the residues remaining in the extractable lipid at death.

Five hens died during the experiment for reasons other than those induced during fasting. One death attributed to dieldrin toxicity occurred on the 68th day in the group of hens consuming the diet containing 20 ppm dieldrin. Three deaths, occurring at weeks 4, 8, and 12, were attributed to the surgery involved in obtaining fat tissue. One death was caused by hemorrhage following collection of a blood sample at the end of the first week of fasting.

Chickens that had been fed dieldrin died sooner and at heavier weights, with their carcasses containing a higher percentage of fat than did controls. Dieldrin concentration per carcass and total dieldrin per carcass were increased with each level of dieldrin fed.

Given in Figure 2 are the changes in level of dieldrin in the blood from the time feed was removed to death. All data are plotted along with the least squares equation which best fit it. The level of dieldrin in blood declined during the first 2 weeks, and then it increased with prolonged fasting. The initial decline was probably caused by cessation of dieldrin intake, while the subsequent increase was probably caused by mobilization of dieldrin from residues in body fat.

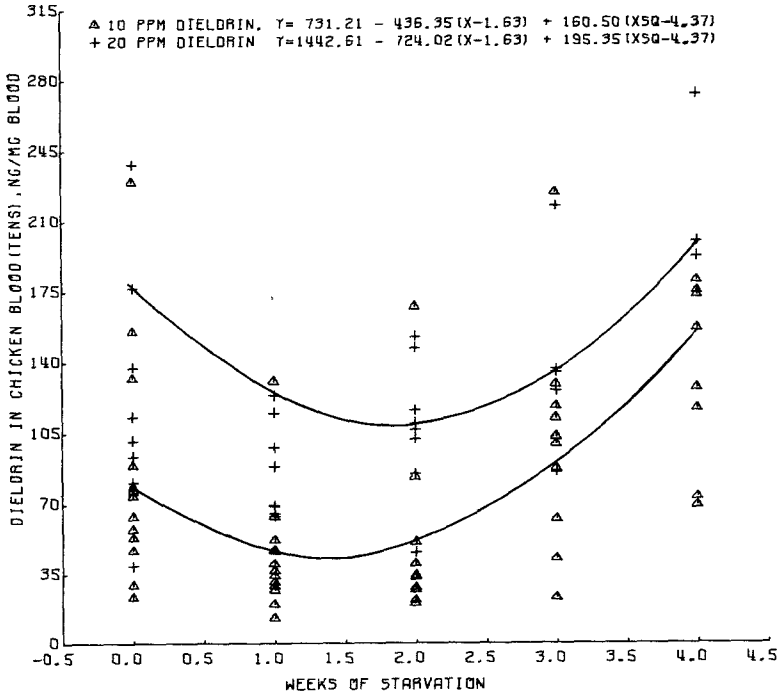

Figure 2. Changes in dieldrin concentration in the blood of chickens of experiment 1 following withdrawl of feed and dieldrin.

TABLE 2

Body weights, survival, egg production, carcass composition, and dieldrin residues of hens in experiment 2[1]

Dietary regime	Control	Dieldrin 1 ppm	Dieldrin 5 ppm	Dieldrin 10 ppm	Dieldrin 20 ppm
Average body weights, g[2]					
Initial	1450	1395	1533	1452	1430
Week 4	1357	1376	1437	1407	1406
Week 8	1377	1369	1416	1393	1386
Week 12	1430	1401	1473	1405	1387
At death	608[a]	672[ab]	735[ab]	778[b]	940[c]
Survival after feed removal, days	29.3[a]	28.7[a]	26.3[a]	21.2[ab]	15.8[b]
(range)	(23-38)	(20-37)	(13-39)	(13-31)	(7-34)
Eggs produced per hen, no.	41.8	34.2	36.8	35.4	28.8
Dry matter in carcasses,[3] %	31.0[a]	34.9[b]	31.6[ab]	33.0[ab]	33.5[ab]
Fat in carcass dry matter,[3] %	4.8[a]	12.6[b]	6.9[ab]	9.6[ab]	12.1[b]
Dieldrin in carcass dry matter,[3] mcg/g	0.0[a]	7.3[b]	31.2[c]	72.3[d]	132.9[e]
(range)	(0)	(2.9-14.6)	(8.8-53.6)	(35-110)	(52-215)
Total dieldrin per carcass,[3] mg	0.0	1.5	6.2	16.5	36.8

[1] Values with different superscripts are significantly different, $P < 0.01$.
[2] Significant quadratic response of body weight with time to week 12, $P < 0.01$.
[3] Data pertain to carcasses of hens which died during fasting.

Experiment 2. Results of experiment 2 are tabulated in Table 2. Death of a hen fed a diet containing 20 ppm dieldrin occurred on the 75th day. This death was attributed to the dieldrin poisoning.

Body weight was not measurably affected by any level of dieldrin fed, and egg production was not significantly affected. Body weight at death was higher in hens fed 10 and 20 ppm dieldrin, while survival at these levels was reduced following removal of the feed.

Carcass dry matter and fat were generally higher in hens that had been fed dieldrin than in controls. As observed in experiment 1, dieldrin residues in the carcasses were directly related to the amount of dieldrin consumed.

Discussion

Since egg production was not to be a major objective in this research, low producing hens were selected. This accounts for the relatively low rate of production observed.

In a previous study (2), it was observed that 20 ppm was the maximum concentration of dieldrin that could be tolerated in the diet of hens for a 12-week period. This observation apparently was correct, since one death attributed to dieldrin poisoning occurred in hens fed diets containing 20 ppm dieldrin during each of these experiments. Considerable stress was imposed on the hens in experiment 1 by the tissue and blood sampling procedures, as evidenced by the 4 deaths attributed to these procedures. Tissue and blood samplings were not done during the second experiment specifically to avoid these stresses.

Hens poisoned with diets containing 20 ppm dieldrin exhibited intermittent convulsions. These often began days before death, regardless of whether death occurred during the feeding or fasting phases of the experiment. Some hens fed diets containing 10 ppm dieldrin convulsed intermittently during the fasting phase of the experiments. Hens fed diets containing 0, 1, or 5 ppm dieldrin convulsed shortly before death, only. It became quite obvious that hens fed the two higher levels of dieldrin were poisoned by this substance when they were fasted.

The droppings of all hens, including controls, became whitish upon fasting, and the feathers surrounding the anal area became matted. Droppings of hens that survived many days of fasting became greenish, and the gizzard and intestines contained a greenish material, presumably of biliary origin.

In experiments conducted with quail (5, 6), pigeons (6), or sparrows (1), emaciation, whitish droppings, and matted feathers were listed among the signs of poisoning with dieldrin or DDT. In these studies dieldrin was fed at graded levels to 250 ppm, and DDT was fed to 300 ppm, but feed consumption was not reported. These observations are similar to those made during the fasting phase of our experiments, regardless of the level of dieldrin fed. It is more likely that these were signs of starvation of the quail, pigeons, and sparrows rather than signs of dieldrin

or DDT poisoning per se. It has been shown previously that chickens fed diets containing 20 to 80 ppm dieldrin voluntarily (2) ate less feed than controls, with a resultant loss in body weight.

While the LD_{50} of the chlorinated hydrocarbon insecticides varies with the insecticide and species of animal (birds are generally more easily poisoned than mammals), the signs of acute poisoning are quite similar for the different insecticides among several species of animals (7, 8). For this reason, it is believed that the information reported here for dieldrin may be used as a general guide for predicting the effects of other chlorinated hydrocarbon insecticides. In special circumstances, birds in their natural environment could be starved as suddenly and as totally as was done here with hens. However, the more difficult point to determine is whether or not sufficient residues occur in birds for poisoning in this manner to be a frequent problem.

A recent nationwide survey (9) showed that dieldrin residues in wings from mallard and black ducks ranged from 0 to 2.97 ppm (wet weight basis). In another survey (10), dieldrin residues in whole starlings averaged less than 1.5 ppm (wet weight basis). These values are in the same range as those obtained by us from hens fed 1 ppm dieldrin (corrected to a wet weight basis).

Other chlorinated hydrocarbons measured in the surveys included DDT and its metabolites, heptachlor epoxide, lindane, and benzene hexachloride. DDT and its metabolites were the most frequent and abundant, although these residues were generally less than 3 ppm, wet basis. Stickel (11) has summarized data from selected areas showing that birds may accumulate DDT and metabolites in much higher concentrations than those reported in the surveys, while dieldrin accumulation was within the range reported above.

While it is apparent that chlorinated hydrocarbon insecticide residues in body tissues may become poisonous during starvation, we conclude that such an occurrence would be rare. Dale et al. (12) have previously expressed an opinion that poisoning of man in this manner would be unlikely, whether starvation was created by deprivation, dieting, or disease.

Summary

Two experiments were conducted to establish the effect of dieldrin on survival of chickens during severe dietary restriction. White Leghorn hens were fed rations containing 0, 1, 5, 10, and 20 ppm of dieldrin for 12 weeks. At this time, dieldrin had accumulated in the body fat in direct proportion to the amount fed. During subsequent feed restriction, survival of hens fed 10 or 20 ppm dieldrin was significantly shorter than that of hens fed 0, 1, or 5 ppm dieldrin. Dieldrin concentration in blood declined during the first and second weeks following feed removal, then it increased. Blood sampling was terminated at the end of 4 weeks because nearly all hens had died, but dieldrin concentration in the blood had not increased appreciably over the concentration observed in blood at the time that feed was removed.

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