Journal of the Minnesota Academy of Science

Volume 44 | Number 1 Article 13

1978

The Effects Of Dieldrin On Chickens

Sondra Severson
Hallock High School, Hallock, Minnesota

The Effects Of Dieldrin On Chickens

Sondra Severson*

ABSTRACT: In this study, insecticide residues in chicken eggs, livers, and fats were monitored in a flock of chickens for seventy-nine days. The test group was fed dieldrin in their drinking water for eighteen days and then was returned to normal water for forty-two more days. The sacrificed birds of the test group showed a 0.39 ppm average increase in dieldrin residue in the eggs, 0.14 ppm in the livers and 9.54ppm in the fats when compared with the control eggs, livers and fats respectively. The t-test calculations showed these results to be significant.

Observations of the embryos from the incubated eggs of each group showed abnormalities in the test birds' embryos compared to normal development in the control embryos. There were no significant differences in the eggshell thicknesses in either the test or control groups.

Behavioral changes were noted in the test birds after the dieldrin feedings that were not present in the control birds activities. They had become very nervous and leg reflex excitability was evident.

Of all the man-created hazards to wildlife, few have caused more concern than the widespread contamination of the environment by toxic chemicals. It is now well documented in scientific literature that pesticidal contamination of ecosystems can alter the status of animal populations through diverse, often complex methods of action (11). Extensive studies have been done on the effects of dieldrin on quail (3), pheasants (10), starlings (15), and rats (4), although little or no research has been done on the relation of the pesticide to embryonic development. In the chicken, the oral LD_{50} for adults has been reported to be between 20 and 30 mg/kg while other studies indicate that 44 mg/kg caused no mortality (9).

Dieldrin, octahydro-endo-exo-demethanonaphthalene, residues found in nature are second in frequency only to those of the DDT group (12). Dieldrin is a breakdown product of the chlorinated hydrocarbon aldrin (7). Through microbiological degradation, an epoxy group is added, making the two substances chemically different (1). Dieldrin has more stability, although it is not known for its outstanding inertness. Because of these properties, dieldrin has been a matter of great controversy.

This paper shows observations made on the studies conducted on mortality rate, embryo development, eggshell thickness, and parts per million contained in eggs, liver and fat samples. This procedure was done in four consecutive phases.

Material and Methods.

In Phase 1, twelve adult, egg-laying hens, 1-3 years old and three roosters were procured randomly for testing. These birds were divided into two groups, red representing the control birds, and blue, representing the test birds. Each bird was numbered and tagged. The birds were then grouped into seperate, similar areas. Roosts and nests were available to both.

Every day, the weight of each bird, daily water and approximate food consumption was recorded. The test and control groups were fed egg-laying mash and oats, giving them free choice as to feed. Pilot brand oyster shells were mixed in with gravel in a seperate feeder. Food consumption could not be measured accurately, since feed was scattered by birds and became mixed with litter, straw, and droppings.

This phase was observed for 18 days. One dozen eggs were selected at random for incubation and three were stored for G.C. analysis from each group.

In Phase 2, the total volume of water consumed by the birds of each group during Phase 1 was determined. 2.5 grams of dieldrin, the LD_{50} for quail (3), was added to 10 gallons.

Dieldrin is virtually insoluable in water so the solution was mixed thoroughly every day.

Phase 2 was run until the total solution was used by the test chickens; 18 days. The same data was recorded as previously in Phase 1. Eggs were stored for G.C. analysis and incubation.

The procedure for Phase 3 was conducted exactly the same as Phase 1 for a period of 18 days. No dieldrin was added to food or water. Eggs were kept for G. C. analysis and incubation.

Phase 4 also followed the same procedure as Phase 1 and was conducted for 18 days. Eggs were kept for G.C. analysis and incubation.

The birds were allowed to live for eight days after Phase 4. During this time they were not upset by daily weighing, but all other measurements were taken as before. Upon cervical dislocation, fat and liver samples were removed and stored for G.C. analysis.

The method for G.C. analysis followed in this experiment was the procedure described by Chet Netivinyoo (8).

Results.

During Phase 1, the average number of eggs collected per day in the test group was 92% of the control average. As Phase 2 and Phase 3 averages were taken, the test averages became significantly greater than the control. It was noted that the number of eggs collected after Phase 4 reduced in number for both groups. However, the test range was 300% larger than the control (Table 1).

There appeared to be no regularities in the increases or decreases in weights between the control birds. Although, the weights of the test group showed that a greater percent had gained weight as compared to the control.

No notable observations were made during Phase 1. But, when the dieldrin was added to the test's water in Phase 2, noticeable changes occured. The test bird's appetites had increased, while no alteration was seen in the control. After the first few days of having the chemical in their diet, the test appeared to be much more relaxed than usual. By the end of Phase 2, both groups were very difficult to catch and weigh. The test birds were moving much faster and more protective of themselves.

In Phase 3, further increases in the eating habits of the test group were noted. They were eating more egglaying mash and oyster shells than the control. The test birds appeared to be very healthy, and their feathers were much fluffier and shinier in contrast to the control.
None of the test birds died throughout the experiment, but at the middle of Phase 4, some of the test chickens had a habit of shaking their legs and appeared to be nervous.

During Phase 1, there were no abnormalities noted in the embryos of either group. Although, abnormalities were apparent in the test embryos four days after the start of dieldrin feeding. These abnormalities were observed through the end of the experiment in the test group, while the control group had normal development throughout the experiment. Common abnormalities were: organs protruding on the right side; no visible wing or feet development; bald spots on the head, dorsal and ventral sides on the anterior and posterior ends; buttery substances on the yolk; irregular shapes and bruised and discolored skins. These deformities had become increasingly more severe by the end of the last phase.

The amounts of residues contained in the eggs, liver and fat samples are shown in Tables 2, 3, and 4 respectively.

Table 1 EGG COLLECTIONS – TOTAL AVERAGES (18 Days/Phase)

		CONTROL	TEST
Phase 1	Total Eggs	38	35
	Average Eggs/Day	2.1 ± .5	1.7 ± .7
	Total Eggs	51	56
	Average Eggs/Day	2.8 ± .52	3.1 ± .81
	Range	2	2
Phase 3	Total Eggs	58	67
	Average Eggs/Day	3.2 ± .98	3.7 ± 1.8
	Range	3	3
Phase 4	Total Eggs	53	53
	Average Eggs/Day	2.9 ± .58	2.9 ± .53
	Range	2	2
Last 8 days	Total Eggs	19	21
	Average Eggs/Day	2.4 ± .48	2.6 ± .73
	Range	1	3

FINAL AVERAGE FOR EGG COLLECTIONS

	CONTROL	TEST
Total Eggs	222	235
Average Eggs/Day	2.8 ± .63	2.9 ± .94
Range	2.2	2.4

Table 2 Dieldrin Residues in the Eggs

PHASE	CONTROL Average Egg wt. (g)	Dieldrin Amount (mg)	ppm* (mg/kg)
Phase 1	42.2	$.70 \times 10^{-3} \pm .14$	.02 ± .003
Phase 2	46.7 46.7	$.95 \times 10^{-3} \pm .19$	$.02^{3}$-.004
Phase 3	43.3	$.65 \times 10^{-3} \pm .2$	.02 ± .003
Phase 4	55.4	$2.75 \times 10^{-3} \pm 0.5$	$.05^{3}$-.01

PHASE	TEST Average Egg wt. (g)	Dieldrin Amount (mg)	ppm* (mg/kg)
Phase 1	55.4	$1.45 \times 10^{-3} \pm 0.3$	0.3 ± .005
Phase 2	54.6	$7.25 \times 10^{-3} \pm 1.4$	1.33 ± .26
Phase 3	49.0	$8.5 \times 10^{-3} \pm 1.7$	.17 ± .034
Phase 4	51.5	$9.0 \times 10^{-3} \pm 1.8$	.17 ± .034

* t = 3

AVERAGE RESIDUES IN EGGS

GROUP	Average Egg Wt. (g)	Dieldrin Amount (mg)	ppm (mg/kg)
CONTROL	46.9	$1.26 \times 10^{-3} \pm .24$	.03 ± .005
TEST	52.6	$6.55 \times 10^{-3} \pm 1.3$	.42 ± .085

Table 3 Dieldrin Residues in the Liver

Bird (Hen No.)	CONTROL Average Liver Wt. (g)	Dieldren Amount (mg)	ppm (mg/kg)
L – 1	28.63	$1.85 \times 10^{-3} \pm 0.4$	.07 ± .013
L – 2	24.7	$.38 \times 10^{-3} \pm 0.3$	.02 ± .003
L – 3	28.4	$.20 \times 10^{-3} \pm 0.3$	.05 ± .010
L – 4	______	______	______
L – 5	24.9	$1.30 \times 10^{-3} \pm 0.3$	.05 ± .010
L – 6	35.4	$1.33 \times 10^{-3} \pm 0.3$	.04 ± .008

BIRD (Hen No.)	TEST Average Liver Wt. (g)	Dieldren Amount (mg)	ppm (mg/kg)
L – 1	49.5	$12.25 \times 10^{-3} \pm 2.0$	.25 ± .050
L – 2	37.0	$2.75 \times 10^{-3} \pm .05$	.07 ± .015
L – 3	45.6	$13.5 \times 10^{-3} \pm 3.0$	.30 ± .059
L – 4	64.7	$5.75 \times 10^{-3} \pm 1.1$	.09 ± .018
L – 5	40.5	$9.75 \times 10^{-3} \pm 1.9$	.24 ± .048
L – 6	38.8	$6.00 \times 10^{-3} \pm 1.2$	.16 ± .031

AVERAGE RESIDUES IN LIVER

GROUP	Average Liver Wt. (g)	Dieldrin Amount (mg)	ppm (mg/kg)
CONTROL	28.4	$1.01 \times 10^{-3} \pm .22$	.04 ± .007
TEST	46.0	$8.33 \times 10^{-3} \pm 1.5$	.18 ± .037

Table 4 Dieldrin Residues in the Fat

BIRD NO.	CONTROL Sample Wt. (g)	Dieldrin Amount (mg)	ppm (mg/kg)
1	12	$2.9 \times 10^{-3} \pm 0.6$	.24 ± .04
2	12	$1.9 \times 10^{-3} \pm 0.4$	.16 ± .03
3	12	$2.4 \times 10^{-3} \pm .5$	.20 ± .04

BIRD NO.	TEST Sample Wt. (g)	Dieldrin Amount (mg)	ppm (mg/kg)
1	12	$120 \times 10^{-3} \pm 30.0$	10.0 ± 2.0
2	12	$160 \times 10^{-3} \pm 30.0$	13.3 ± 2.66
3	12	$71 \times 10^{-3} \pm 15.0$	5.92 ± 1.18

AVERAGE FAT RESIDUES

GROUP	Sample Wt. (g)	Dieldrin Amount (mg)	ppm (mg/kg)
CONTROL	12	$2.4 \times 10^{-3} \pm .5$	.2 ± .037
TEST	12	$117 \times 10^{-3} \pm 25.0$	9.74 ± 1.95

Discussion.

There were no significant differences noted between the control birds and the dieldrin fed birds when comparing eggshell thicknesses, egg collections, water consumption, and weights. Small variances were noted and can possibly be attributed to physiological differences of the birds. The water amounts consumed varied with the temperature and the humidity.

Ninety-four percent of the eggshell is made of calcium (13). Thus, a calcium deficient diet could cause eggshell thinning.

The test birds had been eating more mash and oyster shells than the control. The increase in the intake of the amounts of oyster shells alone could have replaced the calcium ions had there been a deficiency caused by the dieldrin feeding.

Dieldrin, though, had definite effects on embryo growth. These visible abnormalities could have been caused by changes in the egg development as early as germ layer formations. The ectoderm layer determines the growth and development of external coverings, sense organs, and the nervous system. The embryos from the test chicken showed evidence of abnormalities in the lack of feather formation and organ placement. Another germ layer possibly affected could be the mesoderm. After forming mesenchyme, it determines the development of the skeleton, circulatory system, and the dermis. Lack of supportive and connective tissues, and the ventral skin covering seems to point towards this conjecture.

The chemical that goes to the yolk represents a large part of the intake of the hen (6). These residues are possibly procured in the female's follicles when the yolk layers are accumulated, since the amount of dieldrin found in the blood is also a reflection of the amounts found in the eggs (2).

Due to these high yolk concentrations a mortality syndrome characterized by an abrupt appearance of abnormal symptoms, is notable especially during the last stages as the yolk is used up. The small dieldrin amounts in the control eggs suggest that there was some other source of dieldrin contamination present.

During Phase 2, when the dieldrin was added to the water, the test egg residues had risen a value of 66.5 times greater than the control. In phase 3 and 4, when the test group was again drinking ordinary water, the test residues were only 3 to 11 times greater than that of the control.

There were other notable characteristics that distinguished the test birds from the control. Symptoms of acute dieldrin poisoning seemed to be occuring (14). Hyperexcitability, fluffed feathers and leg reflex excitability were observed in the test birds. Ataxia, another commonly acute symptom, was not apparent in the chickens fed dieldrin. Actually, the opposite seemed to be occuring. The test birds were very swift moving and reacted quickly to a stimulus. The roosters were more susceptible to the toxic effects of the insectide orally.

These changes in behavioral activities may also be correlated to thyroid changes. In Phase 2, dieldrin acted as a depressant. Perhaps this could be attributed to mixedema (5). But by the end of Phase 3, thyrotoxicosis possibly was occurring.

The liver ppm were considerably lower than those of the fat. The mean fat ppm was 9.74 as compared to the mean of 0.18 ppm in the test livers. The parts per million contained in the various tissues is dependent upon the lipid levels present in that tissue.

In conclusion, dieldrin should only be used for insect controlling purposes if the need would be of greater importance than the effects the insecticide has on the environment.

Acknowledgment.
The author wishes to express appreciation to Dr. Thomas Boates, Mr. Philip Peterson, and Mr. Jim Matthiae for their advice, encouragement and assistance in the preparation of this paper.

LITERATURE CITED

American Chemical Society, Washington, D.C. 1969

BUGG, J.C. *et al.* 1967. Chlorinated pesticide levels in the eastern oyster (*Crassostrea virginica)* from selected areas of the south Atlantic and Gulf of Mexico. Pesticide Monitor J. 1:9.

DeWITT, J.B. 1955. Chronic toxicity to quail and pheasants of some chlorinated insecticides. J. Agricul. Food Chem. 3:672

Executive Office of the President, Office of Science and Technology. June 1971. Ecological effects of pesticides on nontarget species. pp. 33-35.

FISHBEIN, MORRIS, *et al.* The new illustrative medical and health encyclopedia. 5:682-684.

LINDER, R.L., and DONALD W. LAMB. 1967. Relationship of dieldrin and penned pheasants. Farm & Home Research. Volume XVIII, No. 2, Spring 1967.

LOFTGREN, J.A., D.M. NOETZEL, P.K. HAREIN, and L.K. CUTKOMP. 1974. Insecticides and their use in Minnesota. p. 11.

NETIVINYOO, CHETH. 1974. Chlorinated pesticide residues in eggs from three caspian tern colonies in the great lakes. Graduate Research paper, Bemidji State University.

the great lakes. Graduate Research paper, Bemidji State University.

Shell Chemical Co. An assessment of the environment and their relation to registered uses. Sect. 6.

SKRYPEK, J. and J. B. MOYLE. 1972. Pesticides and wildlife in Minnesota. Special Publ. No. 96. pp. 3-11.

STICKEL, L.F. 1968. Organochlorines in the environment. U.S. Bureau of Sport Fisheries & Wildlife, Special Sci. Rpt., Wildlife No. 119. xii † 32.

STICKEL, WILLIAM H., *et al.* 1969. Tissue residues of dieldrin in relation to mortality in birds and mammals. Chem. Fallout: Current Research on Persistent Pesticides. pp. 176-183.

STURKIE, P.D. 1954. Avian physiology. Comstock Publishing Assoc., New York.

TUCKER, RICHARDK. and D. GLEN CRABTREE. 1970. Handbook of pesticides to wildlife. Resource Publ. No. 84, March. p. 47.

WHITE, DONALD H. 1974. Nationwide residues of organchorines in starlings.

*SONDRA SEVERSON is a senior at Hallock High School, Hallock, Minnesota. She is active in sports, music, F.H.A. and theater. She has won awards at regional, state and international science fairs. Her esearch project resulted in the opportunity to summer work at the U.S.D.A. Research Center in Beltsville, Maryland under Dr. Richard W. Miller.