ENDRIN POISONING IN CHICKENS

E. W. Morin, A. Robertson and M. Beauregard*

Introduction

ENDRIN is one of the cyclodiene group of organochlorine pesticides; others in the group include chlordane, aldrin and dieldrin. It has been used as an insecticide and rodenticide for the protection of crops and pastures and is available to the public through feed and seed outlets. The indiscriminate use of products containing endrin could constitute a hazard to livestock and poultry.

To the authors' knowledge, clinical cases of endrin toxicity in chickens have not been reported in the literature. Reports of accidental poisonings have been cited by Rudd and Genelby (4) in pheasants, and in geese and pigeons by Keith (2). Kelly (3) observed losses in chickens which had access to orchards treated with endrin as a rodenticide against field mice.

Sherman and Rosenberg (5) in their experimental studies with endrin reported nervous system involvement in chicks prior to death. DeWitt *et al.* (1) compared the relative toxicities of DDT, aldrin and endrin in rats, bobwhite quail, pheasants and mallard ducks; endrin was the most toxic. Pheasants were the most sensitive, while rats were the most resistant.

This paper reports the occurrence and describes the toxic effects of endrin in chickens and discusses various aspects of outbreaks in several farm flocks in the Ottawa Valley.

Observations on Field Outbreaks

Within a period of one month during the summer of 1966, chickens from seven flocks were submitted to the diagnostic laboratory with histories and/or symptoms suggesting acute poisoning. Only small flocks ranging in age from five to 12 weeks were involved. All became affected within a few days of feeding a fresh supply of feed which had been obtained at about the same time from a single retail outlet. The feed was delivered to the farms in the owners' vehicles. There were no complaints involving feed delivered from the company mill, located elsewhere.

Symptoms in all flocks were similar. The chickens appeared to be hungry but were reluctant to eat. Birds developed incoordination followed by fits of aimless flying, convulsions and death. Losses varied from 17 to 100%.

Necropsies performed on 15 chickens from the seven flocks failed to reveal any significant gross pathological lesion nor were pathogenic microorganisms isolated by routine bacteriological techniques.

Microscopic examination of brain sections showed evidence of softening in the medullary and Purkinje cell layers of the cerebellum and, to a lesser extent, in the medulla oblongata. An edematous reaction, the severity of which varied from bird to bird, was also observed in heart, liver and kidney tissues. Other organs appeared normal.

In consideration of the history and clinical findings, feed samples were examined and found negative for heavy metals. Subsequent examinations for pesticides were performed by Dr. C. V. Marshall, Analytical Control Laboratory, Plant Products Division, Production and Marketing Branch, Canada Department of Agriculture, Ottawa.

The technique used for analysis may be briefly described as follows: a Beckman GC2A Gas Chromatograph with hydrogen flame detector was used. The column was stainless steel, 0.25 in diameter and 6 ft long, and was filled with 100–120 mesh Gas Chrom Q coated by 5% Versilube F50. Column temperature was 210°C, injection temperature was 240°C. Helium carrier gas flow rate was 100 ml/min.

Table I summarizes clinical and toxicological findings in the seven chicken flocks which had received the suspected feeds. Endrin was detected in all the rations analyzed and levels varied from 1.8 to 35 ppm. Symptoms appeared earlier and losses were greater in those flocks which received the more heavily contaminated rations. Losses of 74, 80 and 90 per cent occurred in flocks fed rations contaminated with 19, 32 and 35 ppm of endrin respectively.

Experimental Trials

1. *Suspect Feed Trials* Prior to chemical analyses of the feeds, an attempt was made to

*Animal Pathology Division, Health of Animals Branch, Canada Department of Agriculture, Animal Diseases Research Institute, P.O. Box 1400, Hull, Quebec.

Present address of E. W. Morin – Veterinary Medicine Division, Bureau of Scientific Advisory Service, Food & Drug Directorate, Dept. of National Health and Welfare, Tunney's Pasture, Ottawa, Ontario.

Present address of M. Beauregard – Faculté de Médecine vétérinaire de l'Université de Montréal, St-Hyacinthe, Québec.

TABLE I

CLINICAL AND TOXICOLOGICAL FINDINGS IN SEVEN CHICKEN FLOCKS

Flock	Number of birds	Age in weeks	Onset of symptoms (days)	Losses (%)	Endrin in feed (ppm)
A	103	8	21	22	1.8
B	125	12	21	17	2.2
C	100	5	4	66	n/a*
D	190	8	4	74	19.0
E	100	12	2	80	32.0
F	200	11	2	90	35.0
G	75	9	2	100	n/a

*Not available.

TABLE II

TOXICITY TESTS OF TWO FEED SAMPLES OBTAINED FROM AFFECTED PREMISES

Feed	Endrin (ppm)	Mortality during feeding period (days) 3	4	5	6	7	8	9*	Died/survived
Control	0	0	0	0	0	0	0	0	0/8
E	32	1	2	1	1	0	1	0	6/2
F	35	0	0	2	1	1	0	1	5/3

*Birds placed on normal feed on 9th day.

TABLE III

EFFECT OF ENDRIN ON FEED CONSUMPTION AND BODY WEIGHT DURING A NINE-DAY TEST PERIOD

Feed	Endrin (ppm)	Average per bird in lbs. Weight change*	Feed consumed
Control	0	+0.73	2.13
E	32	−0.2	1.1
F	35	−0.5	1.4

*Initial average weight of test birds—1.5 lbs.

duplicate the condition as it occurred in the field. Three groups of eight meat-type chickens, five weeks of age, were banded, weighed and placed in batteries. After fasting for 20 hours, one group was given a normal ration and the other two groups, the suspect feeds which were subsequently shown to contain 32 and 35 ppm of endrin. These latter groups were identified as E and F. All birds were examined several times a day and any abnormalities recorded. On the ninth day groups E and F were placed on the normal ration.

Clinical symptoms were not observed during the first two days of the experiment. There was, however, a severe reduction in feed consumption in the two groups which received the suspect feed. On the morning of the third day, a few birds from groups E and F appeared dull and their feathers were ruffled. By afternoon symptoms had progressed to irritability, incoordination and fits of aimless flying, during which one bird died. Some of the birds originally showing fits apparently recovered but developed subsequent attacks and died during the next 24 to 48 hours. Birds which either died late or survived were those which apparently consumed less feed.

The pattern of mortality is presented in Table II. In contrast to no losses in the control group, mortalities in groups E and F were 75 and 62.5% respectively. In general, only a few hours elapsed between the onset of symptoms and death.

Data concerning average weight of the chickens at the start of the experiment, average weight changes and amount of feed consumed are summarized in Table III. The chickens on the normal ration ate almost twice as much feed as those in each of the test groups. Over a period of nine days, an average weight

TABLE IV

MORTALITY RESULTING FROM FEEDING EXPERIMENTAL RATIONS CONTAINING VARIOUS LEVELS OF ENDRIN OVER A PERIOD OF 50 DAYS

Endrin (ppm)	Deaths during feeding period (days) 7–13	14–21	22–50	Died/survived
0	1*	0	0	1/7
1.0	0	0	0	0/8
15.0	1	2	2	5/3
30.0	6	2	0	8/0
30.0†	8	0	0	8/0
32.0 (ration E)	7	1	0	8/0

*Bird sacrificed: Marek's disease.
†Endrin emulsion.

gain of 0.73 lb was observed in the control groups in contrast to weight losses in groups E and F.

On post-mortem examination, none of the chickens showed significant gross lesions. These findings concurred with those seen in earlier field specimens. However, the chickens in groups E and F showed histopathological changes which were similar to those seen in the field cases.

2. *Effect of rations containing known levels of endrin* At the termination of the suspect feed trial, analyses showed that the commercial feeds contained endrin. An experiment was then undertaken to determine the toxic levels for chickens when the chemical was added to feed.

Endrin technical dry powder 99% (Shell) and endrin emulsion (Marquette 20) were obtained from the Feed, Fertilizer and Pesticide Division, Canada Department of Agriculture, Ottawa. Rations containing 1, 15 and 30 ppm were prepared using the technical powder while one containing 30 ppm was made using the endrin emulsion. In addition, the commercial feed designated E used earlier and now known to contain 32 ppm and one without endrin were included.

Six groups of eight chickens similar to those used in the feed trial were fasted for 24 hours, placed on these rations and observed during the 50-day experiment.

Symptoms or losses attributable to endrin toxicity did not occur in groups which were fed rations free or containing 1 ppm of endrin. Nervous symptoms similar to those previously described first appeared on the fourth day in the groups which received higher levels. The pattern of mortality is presented in Table IV. Losses occurred on the seventh day in the groups fed rations containing 30 ppm or more of endrin, leading to 100% mortality by the end of 21 days. In the group of birds fed a ration containing 15 ppm of endrin losses occurred later and totalled 62.5%.

Post mortem examination on all chickens, either at the time of death or at the end of the experiment failed to reveal the presence of significant gross lesions.

3. *Determination of the LD_{50} endrin level in chickens* Broiler chickens, five weeks of age, were divided into five groups of five birds each, identified and fasted for 24 hours. Endrin in corn oil was then administered per os as a single dose to these groups at levels of 0, 1, 2, 4 and 8 mg per kilogram of body weight respectively. The duration of the experiment was 10 days.

All chickens which received 8 mg/kg of endrin died within one hour showing the symptoms described previously. In the group given 4 mg/kg of endrin there were two losses within 12 hours, two the following day and the last on the third day. Two of the five birds which received 2 mg/kg of endrin died on the second day. The control group and the one dosed with 1 mg/kg of endrin appeared normal.

This data did not permit an accurate determination of the LD_{50} since dose levels between 2 mg/kg (40% mortality) and 4 mg/kg (100% mortality) were not employed. However, the findings indicate the LD_{50} would be in the range between 2 and 4 mg/kg.

DISCUSSION

Endrin has been reported to be one of the most toxic organochlorines to mammalian and avian species. In comparing the toxicities of endrin and aldrin with DDT, DeWitt *et al.* (1) found endrin to be 65, 400 and 900 times more toxic than DDT in mallard ducks, bobwhite quail and pheasants respectively.

Young chickens and pheasants seemed to be particularly sensitive to endrin. The LD_{50} of 3 mg/kg in pheasants as reported by DeWitt *et*

al. (1) may be compared with the 2–4 mg/kg range obtained in the five-week-old broiler chickens used in this trial.

The results now being reported also appear to be in close agreement with those of Sherman and Rosenberg (5) who, having fed chickens rations containing 12 and 1.5 ppm of endrin for 84 days, obtained survival rates of 5 and 100%. The present study showed that rations containing levels of 15 and 1 ppm administered for 50 days resulted in 37 and 100% survivors.

The reduced feed consumption and weight loss of the chickens was attributed to unpalatability of the feed rather than the toxicity. The birds appeared anxious to eat but were reluctant to continue after tasting it. This view is supported by the observation that the survivors of trial 1 commenced to eat well when they discovered that the suspect feed had been replaced by a normal ration.

It was not established how or where the contamination of the commercial feed occurred despite intensive investigation. Company supplies of the batch of feed or its ingredients were no longer available when contamination was suspected. However, there were no complaints from poultrymen whose feed had been delivered in bulk from the mill. Owners of the affected flocks obtained their feed in bags at the company retail store and transported it in their own vehicles. This and the wide variation of endrin levels found in the various bags of feed suggested that the contamination occurred at the store where endrin, in the form of an emulsion, was available.

Summary

Accidental endrin poisoning was encountered in seven commercial chicken flocks. Both endrin contaminated feeds and endrin compounded rations proved to be toxic for chickens. Losses were proportional to the level of the pesticide in the feed.

Résumé

Sept empoisonnements accidentels dus à l'endrin se sont produits dans autant de troupeaux de poulets. Les moulées contaminées ainsi que celles auxquelles nous avons incorporé de l'endrin se sont avérées toxiques pour les poulets, le taux de mortalité étant proportionnel à la concentration de l'élément toxique dans la ration.

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ABSTRACT

Nicoletti, P. L., Quinn, B. R. & Minor, P. W. (1967) Canine to human transmission of brucellosis.—New York State J. Med. 67. 2886–2887 (U.S. Dep. Agric. Albany, New York).

A woman developed a febrile illness accompanied by a brucella agglutinin titre of 1:2,240. Her bitch, which had aborted five months previously, also gave a positive agglutination test, at 1:800. The dog was killed and *Br. suis* type I was isolated from pharyngeal and supramammary lymph nodes. Pigs on a neighbouring farm were also infected with the same brucella.

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