Susceptibility of Chickens Fed p,p'-DDT to Histomoniasis

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DDT (1,1,1-trichloro-2,2-bis-[p-chlorophenyl] ethane) accumulates in high amounts in tissues and incites adverse physiological disturbances in certain animals (1). Although DDT has low toxicity for mammals, harmful effects have been observed on birds, such as eggshell thinning and lowered reproductive success (2). Moore (3) discussed the problem of persistent pesticides and focused attention on the potentially great ecological importance of sublethal effects of these pesticides. Sublethal poisoning in nature could lead to serious consequences such as an increase in the susceptibility of wild animals to disease and/or parasitism. Recently Friend and Trainer (2,4) reported that polychlorinated biphenyl (PCB), dieldrin, or DDT rendered mallard ducklings more susceptible to duck hepatitis virus.

Histomoniasis (blackhead; infectious enterohepatitis), an infectious disease affecting primarily the cecae and livers of wild and domestic gallinaceous birds, is caused by the protozoan Histomonas meleagridis which is transmitted by the nematode Heterakis gallinarum (5,6). Susceptibility to this disease differs among species, breeds, and ages of birds. Domestic turkeys are highly susceptible and the infection causes clinical signs, gross lesions, and death. Domestic chickens, however, are more resistant and mild cecal involvement usually disappears 21 to 28 days following exposure (6).

Chickens tolerate high amounts of DDT without exhibiting clinical signs (7). The role played by sublethal amounts of DDT with respect to disease susceptibility is not known. The present report deals with the relationship of sublethal doses of p,p'-DDT to the susceptibility of chickens to histomoniasis. The data reported herein are from the second of two experiments; a preliminary experiment using smaller numbers of animals gave similar results.

Florida Agricultural Experiment Station Journal Series No. 4071.

Bulletin of Environmental Contamination & Toxicology, Vol. 8, No. 3, 1972, © by Springer-Verlag New York Inc.

METHODS AND MATERIALS

Six groups of 20 one-day-old male white leghorn chickens were raised in brooder pens and fed commercial chicken starter mash (free of coccidiostats or antibiotics) and distilled water provided ad libitum. p,p'-DDT* was dissolved in olive oil at the rate of 10 mg/ml and administered orally in gelatin capsules to three groups in dosages of 10, 30, and 50 mg/kg body weight on alternate days from 8 to 38 days of age. These dosages were several orders of magnitude less than those known to produce clinical signs (7). Similarly, two groups of chickens were given comparable amounts of olive oil and distilled water, respectively. A control group received no treatment. Starter mash, olive oil, and gelatin capsules were determined to contain no detectable DDT. At two weeks of age, six days following the first DDT dosage, each bird of the first five groups was given 425 embryonated eggs of H. gallinarum orally. The eggs were taken from a pool which had been tested previously for viability and pathogenicity in turkeys. Thirty days after infection the surviving chickens (44 days of age) were killed and examined for lesions of histomoniasis. Birds which died during the course of the experiment were also examined. Gross cecal lesions were scored on a zero to four scale of increasing severity similar to the method used for studies on coccidiosis (8). Cecal worms were fixed in warm glycerine-alcohol and counted. Cecal and liver tissues were fixed in 10% buffered formalin, sectioned, and stained with Haematoxylin-Eosin or Periodic Acid Schiff reagent for histopathologic studies. Samples of brain, liver, and cecum were collected from each bird, pooled according to treatment, and frozen until analysed for DDT residues. Ten grams of each sample was extracted with petroleum ether in a soxhlet apparatus following the procedure of Reichel and Addy (9). A Varian-Aerograph 2100 gas chromatograph equipped with a 6' x 1/4" glass column packed with 3% OV-17 on 100/120 Chromosorb W and an electron capture detector was used for analysis. Operating parameters were: injection port, 210°C, column, 200°C and detector 210°C with a nitrogen flow of 45 ml/min. The retention time of p,p'-DDT was about 20 minutes.

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* M.W. 345.49; M.P. 108-109°C, Aldrich Chemical Company, Milwaukee, Wisconsin.

RESULTS AND DISCUSSION

Nine of 60 birds which received DDT and eggs of H. gallinarum died during the experimental period. There was no correlation between deaths and DDT concentration. All the dead birds had cecal lesions typical of histomoniasis. At necropsy all surviving birds which received 30 and 50 mg DDT/kg and 94% of the birds which received 10 mg DDT/kg, showed pathognomonic lesions of histomoniasis. Histopathologic examination of cecal and liver tissues confirmed the presence of H. meleagridis; no evidence of infection by coccidia was observed in cecal tissues. No birds in the control group or in the group given H. gallinarum eggs, but no DDT, contained lesions (Table 1).

There was a direct correlation between the administered doses of p,p'-DDT and residues of p,p'-DDT and its metabolites (p,p'-DDD and p,p'-DDE) in the brain (Fig. 1). Total residue in the liver also increased with higher doses, but most of the residue was in the form of DDD and DDE. Residue levels in the ceca (Fig. 1) and the amount of tissue abnormality (Table 1) were similar in chickens fed DDT regardless of dosage. At the levels of parasitic infection utilized in this experiment, the threshold effect of DDT on the susceptibility of chickens to histomoniasis was less than 10 mg/kg. Further work could show what minimal levels of DDT in conjunction with the etiologic agents of histomoniasis produce pathologic effects.

Several possible mechanisms could be suggested to explain the results of this experiment. DDT-treated birds may have had cecal lesions at day 30, while control birds did not, because DDT retarded development of Heterakis larvae and subsequently the release of histomonads. Lesions in control birds may have regressed by day 30. Secondly, DDT may have affected the development of immunity to histomoniasis resulting in persistent lesions. Lastly, it is possible that, since certain species of bacteria must be present along with Histomonas in order to produce the disease syndrome of histomoniasis (5), DDT may have caused an effect on the bacterial flora which resulted in observed changes in susceptibility of the chicken to this disease.

The results of this study support the idea (2) that synergism between sublethal levels of persistent insecticides and various disease agents may have broad ecological significance.

TABLE 1

Histomoniasis lesions and numbers of adult Heterakis gallinarum in cecae of chickens exposed to 425 H. gallinarum eggs and fed p,p'-DDT over a 30-day period. The control group did not receive either DDT or H. gallinarum eggs.

Treatment	p,p'-DDT (mg/kg wt.)	Percentage of chickens with histomoniasis cecal lesions	Mean cecal lesion score	Mean number worms in cecae
DDT + H. gallinarum eggs	50	100.0	2.3	24.6
DDT + H. gallinarum eggs	30	100.0	2.1	29.9
DDT + H. gallinarum eggs	10	94.4	2.0	26.3
Olive oil + H. gallinarum eggs	0	5.0	0.1	22.0
Distilled water + H. gallinarum eggs	0	0.0	0.0	26.1
Control	0	0.0	0.0	0.0

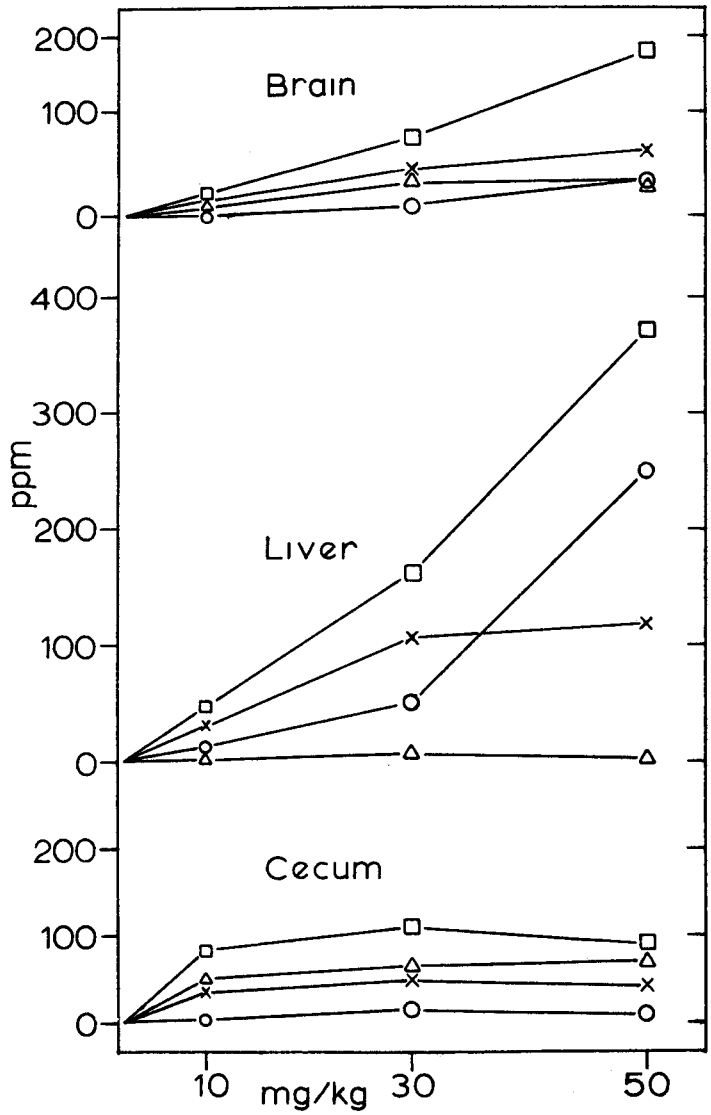

FIG. 1. Residues in brain, liver, and cecal tissues of chickens fed p,p'-DDT and infected with Histomonas meleagridis. Squares represent total DDT, circles DDD, triangles DDT, crosses DDE.

ACKNOWLEDGMENTS

We thank Mr. Doyle C. Golden of the Pesticide Residue Section, Dept. of Agriculture, Tallahassee, Florida for certain residue analyses. Mr. Edward Balducci, NSF secondary school student, helped in the care and feeding of the chickens and Dr. R. E. Bradley provided certain facilities. We also thank Dr. K. P. C. Nair for help in histopathology and Dr. C. F. Simpson for providing the p,p'-DDT and for advice in the preparation of this manuscript.

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