Some Effects of Sublethal Levels of Insecticides on Vertebrates

Authors: FRIEND, MILTON, and TRAINER, DANIEL O.

Source: Journal of Wildlife Diseases, 6(4) : 335-342

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-6.4.335

Some Effects of Sublethal Levels of Insecticides on Vertebrates

MILTON FRIEND and DANIEL O. TRAINER

Department of Veterinary Science,
University of Wisconsin, Madison, Wisconsin 53706

Sublethal levels of persistant insecticides are available for wildlife acquisition from many sources and are not limited to direct exposure from spraying operations. The widespread application of persistant insecticides, such as DDT and dieldrin, is common knowledge and transportation of these and other pollutants through the ecosystem has been shown to take place by both physical and biological means.[40,42] Organochlorine residues have been detected in air, snow, inland and oceanic water, soil, plants and agricultural crops, as well as in a variety of invertebrates and vertebrates.[40,42] Residues have been detected even from such remote areas as Antarctica.[18,43] Because of the intimate dependency of wildlife on the environment, opportunity for exposure to insecticides by contact or through food chains is great.

The effects of insecticides on vertebrate populations are most dramatic when acute toxicity results in readily observable manifestations such as trembling and dying robins or extensive fish kills. Such observations were catalysts in stimulating interest in the total concept of environmental pollution. Many of the less obvious, but probably more significant, aspects of insecticide intoxication are not always attributed to insecticides because of a delay in response that only becomes evident following biological interactions.

It is the aim of this paper to focus attention on some of the delayed responses which result from sublethal levels of insecticides, such as effects on reproduction, behavior and susceptibility to infectious disease.

Sublethal levels are implied in the following discussion and are defined as levels that alone are insufficient to cause mortality by acute toxicity or through chronic exposure.

Reproduction

Reproductive failures of wild and captive salmonoid fish, a variety of raptors, and fish eating bird populations, stand out as dramatic illustrations of the delayed effects of insecticides.

The declining lake trout *(Salvelinus namaycush)* fishery of Lake George, New York has been correlated with reproductive failures in this species resulting from DDT.[10] High levels of this insecticide were detected in adult females and their eggs; and during 1955-1959, nearly the entire production of eggs stripped from adult females captured in that lake died at the fry stage. Subsequent investigations revealed that fry suffered heavy mortality when DDT levels reached 2.95 ppm or greater in the eggs.

Declining production of Sebago Lake in Maine has also been correlated with DDT contamination of the watershed. This lake has been the site of an important landlock salmon, *Salmo salar,* (Girard) sport fishery since the late 1800's. Sport fishing remained satisfactory until 1956 when a decline in fishing success began. This

Approved by the Wisconsin Experiment Station Director and published as Veterinary Science Paper No. 659.

decline was later associated with the increasing practice of large scale applications of DDT to shoreline properties and State campsites.[5]

Laboratory studies have documented the detrimental effects of DDT on reproduction of salmonoid fish, including: lake trout,[10] cutthroat trout, *(Salmo clarkii lewisi),*[1] and brook trout *(Salvelinus fontinalis).*[29] These investigators reported that, while numbers and survival of eggs produced are not affected, significantly higher mortality occurs among yolk-sac fry as levels of DDT increase in the eggs. Studies with other fish have disclosed that several chlorinated hydrocarbons caused abortions in pregnant mosquito fish *(Gambusia affinis);*[9] endrin concentrations as low as 0.0005 ppm prevented reproduction in guppies *(Lebistes reticulatus);*[31] and DDT caused death at birth or within several hours among progeny of guppies exposed to DDT-treated water.[26]

Dramatic declines of avian populations, not attributable to catastrophic die-offs of existing populations or to destruction of habitat, have been recorded for a number of species in both Europe[36] and North America.[21] Common characteristics of these declining species are their position in the food chain (all are at top trophic levels), existence of high levels of chlorinated hydrocarbon residues in their tissues and eggs, and a significant reduction in their egg shell thickness.

The pending extermination of peregrine falcon *(Falco peregrinus)* nesting populations in northwestern Europe and the loss of nesting populations of this species in the eastern half of the United States has been well documented.[20] Chlorinated hydrocarbons, especially DDE (a product of DDT metabolism), have been associated with the decline of the peregrine as well as other avian species, including the sparrow hawk *(Accipiter nisus),*[36] golden eagle *(Aguila chrysaetos)*[28] and shag *(Phalacrocorax aristotelis)*[35] in Europe; the osprey *(Pandion haliaetus),*[21] bald eagle *(Haliaetus leucocephalus),*[21] double-crested cormorant *(Phalacrocorax auritus),*[2] and brown pelican *(Pelecanus occidentalis)*[25] in the United States; and the Bermuda petrel *(Pterodroma cahow),*[48] a pelagic species which feeds far at sea.

Field studies of these population declines have disclosed thinning of egg shells, considerable egg breakage, eating of broken eggs by the parents, delayed breeding, failure to lay eggs or failure to renest, and at times, high mortality of embryos and fledglings.[33] Comparison of egg shell measurements prior to and after 1946 when widespread applications of persistent insecticides came into use disclosed a correlation between decreasing shell thickness in affected populations and insecticide use.[33] The extreme in eggshell thinning was recently observed in a colony of brown pelicans off the coast of California where the eggs could not be picked up without denting the shell.[24]

Reproduction abnormalities have been produced in the laboratory using low levels of DDE, dieldrin and DDT either alone or in combination in the American sparrow hawk *(Falco sparvericus),*[34] mallard duck *(Anas platyrhnchos),*[19,27] prairie falcon *(Falco mexicanus),*[14] coturnix quail *(Coturnix coturnix)*[8] and ringed turtle dove *(Streptoplelia risoria).*[33]

Thinning and breakage of eggshells is hypothesized to be a result of the inhibition by DDT and its metabolites of carbonic anhydrase (an enzyme that plays an important role in making calcium available to the eggshell in the oviduct). Other reproductive abnormalities in birds (such as failure to lay eggs) are hypothesized to result from depression of estrogen levels which might be caused by induction of liver enzymes.[33]

An ecological complication of delay in ovulation caused by DDT could arise if second broods of double-brooded birds or late breeding birds get out of phase with suitable climatic conditions and food supplies, thus reducing their potential for survival.[23]

Not all avian species experiencing thinning of eggshells since the widespread use of insecticides began are suffering detectable population declines, nor have all laboratory studies with a specific organochlorine insecticide produced similar reproductive results in the same species. There is, however, sufficient scientific evidence to clearly illustrate that sublethal levels of insecticides, such as DDT and dieldrin, not only can disrupt reproduction in fish and birds under laboratory conditions but are at least partially responsible for similar situations in nature.

Reproductive failure due to insecticides is often correlated with position in the food chain and type of food web involved. For the most part, those species whose diets consist mainly of various stages of vegetation have not suffered the types of reproductive abnormalities noted among raptors and fish eating birds. Exceptions among game birds include reports of reduced reproduction of bobwhite quail *(Colinus virginianus)* and wild turkey *(Meleagris gallopava)* in the south following applications of heptachlor and dieldrin for fire ant control,[12] and suppression of woodcock *(Philohela minor)* reproduction in new Brunswick, Canada.[47]

Behavior

Behavioral changes in fish after exposure to sublethal concentrations of insecticides may result from peripheral and/or central nervous systems alterations.[4] Exposure of brook trout to DDT greatly amplifies the response of the lateral line system to physical disturbances (hyperexcitability) in the acquatic environment and this effect is more intense at lower temperatures.[3]

Warner *et al.*[44] reported that behavioral changes represent the final integrated results of a diversity of biochemical and physiological processes. They concluded that single behavioral changes generally were more comprehensive than individual physiological or biochemical changes and could be used as a quantitative assay for sublethal levels of chlorinated hydrocarbons in fish. In their studies, gold fish *(Carassius auratus)* were exposed to toxaphene contaminated water for 96 hours after which gross behavioral changes to combinations of light, shock and noise were evident.

The optimal water temperature selected by brook trout and other salmonoids is altered by DDT;[32] low doses lowering the selected temperature and higher doses raising it.[4] This interference with the thermal acclimation mechanism could influence survival by altering habitat selection and spawning behavior. DDT also prevented brook trout from learning to avoid a preferred light condition while untreated trout became conditioned within 30 trials. When half the conditioned trout were treated with DDT and retrained to avoid the same light condition, it took about the same number of trials to become retrained, while untreated controls were retrained in only 11 trials.[4]

Changes of conditioned reflexes in animals exposed to insecticides reflect the response of the nervous system and are characteristic of specific types of toxicants.[30] Low levels of DDT or aldrin, insufficient to cause changes in the strength of conditioned reflexes in animals, may render the host susceptible to reactions suggestive of hypersensitivity. For example, house cats *(Felix domesticus)* exposed to aldrin at a level well below the threshold dose and then re-exposed ten days later at the threshold dose had an exaggerated response, including a total cessation of the conditioned reflexes and unconditioned food and orientation reflexes. These responses did not return to normal for 6-8 days.[30] No evidence was presented for or against an immunological basis for these responses.

Impairment of these types of reflex conditions might greatly alter chances of survival in wild animals. For example, prey-predator relationships may be enhanced to favor either party. The quail or cottontail failing to respond quickly and correctly to the shadow of an approaching raptor is more likely to become prey than a more alert and responsive individual. Failure of the predator to respond swiftly and adeptly to the presence of prey might sufficiently alter the ratio of energy expended to that gained per meal to a point where the predator can no longer maintain itself. Rudd[40] reports several examples of wild fish and rodents exposing themselves to predation following applications of chemicals to their environment.

Depression due to a decrease in motor activities is often an initial manifestation of changing behavior during early stages of poisoning by many toxicants.[30] This type of response was noticeable in some of our studies with mallard ducks maintained on dieldrin contaminated feed.* The birds appeared dull and indifferent to human presence and attempts to capture single birds in the treatment pens failed to arouse any escape response in other birds. The ducks assumed a rather passive attitude or wandered aimlessly about. The response of mallards maintained on DDT diets, however, was generally in marked contrast: these birds appearing to be in a state of hyperexcitability. When the pens were entered the birds immediately attempted to escape and often piled up three to four birds deep in the furthest corner of the pen. Some individual birds exhibited aggressive behavior.

These examples of behavioral alterations and the hypothesis regarding their significance are not intended to be all inclusive, but rather serve to illustrate another type of effect of sublethal levels of chemical toxicants. It is easy to postulate situations where some of the changes in behavior due to insecticides in the laboratory could have catastrophic effects on wild populations. Among game species, reduced wariness, decreased mobility, or delayed migratory movements might result in increased vulnerability to the gun, predation or physical factors of the environment (such as climate and food supply). However, we hasten to point out that extrapolations of laboratory findings to field situations cannot be made directly, but serve only as a basis for hypotheses to be tested.

*These studies were supported in part by the Patuxent Wildlife Research Center, Bureau of Sport Fisheries and Wildlife.

Disease

According to Durham:[18]

> "The unifying concept which underlies many of the interactions of pesticides with other factors involves the drug detoxifying enzyme systems in the liver microsomes . . . The interactions of pesticides with factors such as species, sex, age and nutritional status are at least partly mediated through the microsomal enzymes."

Certain diseases, such as obstructive jaundice, untreated alloxan-induced diabetes, hepatic tumors and partial hepatectomy, because of their known effect on the activity of liver microsomal enzymes might also be expected to interact with pesticides.[13]

Criteria used to measure the effects of interactions vary with the insecticide and site of action, and encompass an entire spectrum from changes in a single enzyme to death of the host. These responses may be classified as additive, synergistic or antagonistic. Additive responses describe situations in which the total response exceeds that expected from any single agent but not the combination of all agents involved, while synergism is restricted to response levels in excess of simple additive effects. Antagonism implies protection or a reduction in response to one agent through the action of another.

The problem of pesticide interactions is usually considered from a viewpoint of changes in the toxicity threshold levels of the host. Another aspect of the problem, pesticides as a predisposing factor for infectious disease, is seldom considered. The major emphasis of pesticides-disease studies has been an exploration of the carcinogenic potential of these chemicals.[7] More recently, effects on the immune response[45] and studies of interactions with biologically active agents have been undertaken.[17]

Investigation of the effects of organochloride insecticides on body defense systems in the rat suggested that DDT may significantly reduce antibody production by inhibitory action upon antibody producing cells. It was also reported that normal adrenal hypertrophy observed in surgically traumatized control rats was inhibited in DDT treated rats subjected to similar treatments.[45]

Studies in cell culture with organochlorine, organophosphorus and dinitrophenol insecticides disclosed alterations in the physiology of the insecticide-treated cells which influenced cell response to polio and vaccinia viruses and diphtheria toxin. Both antagonism and synergism were observed. The increased susceptibility of insecticide-treated cell cultures to polio-virus was thought to have resulted from the inhibition of interferon activity. In addition to changes in the pathogenesis of polio and vaccinia viruses within *in vitro* systems, possible activation of a latent cytopathogenic agent in the cell cultures was observed.[17]

Studies of possible interactions between insecticides and infectious disease in wildlife are essentially lacking. We have been investigating this problm utilizing the mallard duck as the experimental host. The approach has been to study changes in susceptibility to infectious disease rather than decreased host thresholds of toxicity. The results of these studies using mortality as an end-point, have varied from antagonism to synergism. An example of synergism is illustrated by Figure 1. In this experiment, 30-day old mallard ducklings that had sublethal levels of DDT or dieldrin were challenged intravenously with duck hepatitis virus. The virus alone produced six percent mortality while three to nine fold increases in mortality occurred among groups pretreated with these insecticides. No direct extrapolation of these findings to natural situations can be made. However, the possibility of such alterations in susceptibility has now been illustrated and must be considered.

Based on these results, it is interesting to speculate on the possible role of insecticides in the epizootiology of fowl cholera in waterfowl. California has been an enzootic area for this disease since the late 1940's.[37] The first cases were noted in 1944 when approximately 1,000 coot died[38] and the first major epizootic (involving some 40,000 swans, geese, ducks and coot) occurred during the winter of 1948-49.[38,30] Since then periodic winter outbreaks of the disease have occurred in California, with no species or population density dependency noted. Most of these outbreaks have started near the confluence of the Sacramento and San Joaquin Rivers in the general area known as the delta. A popular hypothesis to explain these outbreaks is that carriers among the migrating waterfowl are stressed by hunting and severe winter weather until they succumb to the disease and provide a focus of infection for the concentrated flocks of susceptible waterfowl in the immediate area.[37]

An equally tenable hypothesis is that the inducing or stressing factor is a chemical pollutant that either causes a "break" in the carriers or interacts in some manner with the host and the causative agent *(Pasteurella multocida)*, or both, to produce overt disease. The chronology of the reported history of the disease in wild waterfowl (1946 marks the beginning of widespread use of insecticides), the widespread use of agricultural chemicals in California (20 percent of the national total of pesticides consumed were used in California during 1960),[46] and the delta origin of many of the outbreaks (discharge of pesticide residues from the Central Valley of California into San Francisco Bay is estimated at almost 2 tons/year)[6] may all be interpreted as supporting data for this hypothesis.

The recent occurrence of type E botulism in fish-eating water birds on Lake Michigan is another interesting area for speculation. Late fall and winter mortality among gulls *(Larus sp)*, loons *(Gavia sp)*, and grebes *(Colymbus sp)* has been noted annually since the first diagnosed epizootic in 1963.[15] High levels of organochlorine insecticide residues are known to occur in this ecosystem,[22] but their relationship, if any, to disease in water birds of that ecosystem is unknown.

Direct mortality resulting from insecticide use is too often the only type of response considered; it represents the crudest type of end-point being characterized by an all or none response: it is easily measured and readily observable but conveys a minimum amount of information. Less obvious endpoints such as antibody production and enzyme induction are not grossly observable, more difficult to measure and interpret, but may be of greater importance. For example, suppression of the immune response by insecticides might lead to increased disease susceptibility and also to the activation of latent viral infections. An analgous situation occurs in guinea pigs and man where cytomegalovirus infections are often associated with immunosuppressive treatment.[16]

Interpretation of the effects of suppression of the immune response is further complicated by a consideration of the type of disease agent involved. Antibody formation is of little protective value in cases of primary infection with many viral agents but is of considerable importance in most bacterial infections.[11,10]

Many other situations of possible interaction between insecticides and disease could be suggested. The examples cited are sufficient to focus attention on the possibility of insecticides acting as synergists in multiple agent relationships.

Interpretation of an interaction between multiple etiological agents is difficult Each agent may act independently of the other; each may enhance the action of the other at similar or dissimilar target locations and times; the relationship between agents and the host may involve well defined dose-effect curves; or one or more of the agents may exhibit a dose-independent relationship (all or none response).

Conclusion

Sublethal represents an artificial category of the scheme in which pesticide effects are classified. By definition, levels causing acute and chronic toxicity with resulting mortality are separated from those insufficient to cause mortality by themselves.

In the preceding discussion, we have illustrated that sublethal levels of insecticides may induce biological interactions resulting in death. In some cases, the consequences of sublethal levels may be far more devastating to a population than acute toxicity levels (reproductive failure). Also, an animal that dies as a result of increased susceptibility to disease or from predation due to a decrease in conditioned reflexes is as much a casualty of insecticides as the fish killed by acute toxicity or the robin dying from chemicals that have reached toxic levels via biological magnification within its food web.

It is essential to recognize the complexity of interactions that might take place. The biological integrity of the cell, the animal, the population, the ecosystem, and the biosphere are all related, and effects at any level may ultimately cause changes in other levels. Classification of the effects of pesticides mainly serves as an area of reference within which a simplified presentation of complex and interwoven interactions may be discussed.

Literature Cited

1. ALLISON, D., B. J. KALLMAN, O. B. COPE, and C. C. VAN VALIN. 1964. Some chronic effects of DDT on cutthroat trout. U.S. Bureau of Sport Fisheries and Wildlife. Research Report 64, p. 30.
2. ANDERSON, D. W., J. J. HICKEY, R. W. RISEBROUGH, D. F. HUGHES, and R. E. CHRISTENSEN. 1969. Significance of chlorinated hydrocarbon residues to breeding pelicans and cormorants. Canad. Field-Nat. 83: 91-112.
3. ANDERSON, J. M. 1968. Effect of sublethal DDT on the lateral line of brook trout, *Salvelinus fontinalis*. J. Fish Res. Bd. Canada. 25: 2677-2682.
4. ———— and M. R. PETERSON. 1969. DDT: Sublethal effects on brook trout nervous system. Science 164: 440-441.
5. ANDERSON, R. B. and W. H. EVERHART. 1966. Concentrations of DDT in landlocked salmon *(Salmo salar)* at Sebago Lake, Maine. Trans. Amer. Fish. Soc. 95: 160-164.

6. BAILEY, T. and J. HANNUM. 1967. Distribution of pesticides in California. J. Sanitary Eng. Div., Soc. Civil Engineers, Proc. 551: 27-43 (Cited by Rudd, R. L. 1969).

7. BARNES, J. M. 1966. Carcinogenic hazards from pesticide residues. Residue Reviews 13: 69-82.

8. BITMAN, J., H. C. CECIL, S. J. HARRIS, and G. F. FRIES. 1969. DDT induces a decrease in eggshell calcium. Nature 222: 44-46.

9. BOYD, C. E. 1964. Insecticides cause mosquito fish to abort. Progressive Fish Cult. 26: 138.

10. BURDICK, G. E., E. J. HARRIS, H. J. DEAN, T. M. WALKER, J. SKEA, and D. COLBY. 1964. The accumulation of DDT in laketrout and the effect on reproduction. Trans. Am. Fish. Soc. 93: 127-136.

11. DAVIS, B. D., R. DULBECCO, H. N. EISEN, M. D. GINSBERG, and W. BARRY WOOD, JR. 1968. Microbiology. Harper and Row, New York, p. 1464.

12. DEWITT, J. B., and J. L. GEORGE. 1960. Pesticide — Wildlife Review. 1959. Bureau of Sport Fisheries and Wildlife Circ. 84, rev., p. 36. (Cited by Rudd, R. L., 1966).

13. DURHAM, W. F. 1967. The interaction of pesticides with other factors. Residue Reviews 18: 22-103.

14. ENDERSON, J. H., and D. D. BERGER. 1970. Pesticides: Eggshell thinning and lowered production of young in prairie falcons. Bioscience 20: 355-356.

15. FAY, L. D. 1966. Type E Botulism in Great Lakes Waterbirds. Trans. 31st N. Amer. Wildl. Conf. 139-149.

16. FENNER, F. 1968. The biology of animal viruses. Vol. II. The pathogenesis and ecology of viral infections. Academic Press, New York. p. 370.

17. GABLIKS, J., and L. FRIEDMAN. 1969. Effects of insecticides on mammalian cells and virus infections. In: Biological effects of pesticides in mammalian systems. Annals N.Y. Academy of Sciences. 160: 254-271.

18. GEORGE, J. L., and D. E. H. FREAR. 1966. Pesticides in the Antarctic. J. Appl. Ecol. 3 (suppl.): 155-167.

19. HEATH, R. G., J. W. SPANN, and J. F. KREITZER. 1969. Marked DDE impairment of mallard reproduction in controlled studies. Nature 224: 47-48.

20. HICKEY, J. J. 1969. Peregrine falcon populations. Their biology and decline. U. of Wis. Press, Madison. p. 596.

21. ———— and D. W. ANDERSON. 1968. Chlorinated hydrocarbons and eggshell changes in raptorial and fish-eating birds. Science 162: 271-273.

22. ————, J. A. KEITH, and F. B. COON. 1966. An exploration of pesticides in a Lake Michigan ecosystem. Jour. Appl. Ecol. 3 (Suppl.): 141-154.

23. JEFFERIES, D. J. 1967. The delay in ovulation produced by pp'-DDT and its possible significance in the field. Ibis. 109: 226-272.

24. JEHL, J. R., JR. 1969. The brown pelican, a vanishing American. Environmental Southwest. 418: 4.

25. KEITH, J. O. 1970. Reproductive failure in brown pelicans. Trans. 35th N. Amer. Wildl. Conf. In press.

26. KING, S. F. 1962. Some effects of DDT on the guppy and the brown trout. U.S. Fish and Wildlife Service, Special Scientific Report — Fisheries. No. 399, p. 22.

27. LEHNER, P. N., and A. EGBERT. 1969. Dieldrin and eggshell thickness in ducks. Nature 224: 1218-1219.

28. LOCKIE, J. D., and D. A. RATCLIFFE. 1964. Insecticides and Scottish golden eagles. British Birds 57: 89-102.

29. MACEK, K. J. 1968. Reproduction in brook trout *(Salvelinus fontinalis)* fed sublethal concentrations of DDT. J. Fish Res. Bd. Canada 25: 1787-1796.

30. MEDVED, L. I., E. I. SPYNU, and S. KAGAN. 1964. The method of conditioned reflexes in toxicology and its application for determining the toxicity of small quantities of pesticides. Residue Reviews. 6: 42-74.

31. MOUNT, D. I. 1962. Chronic effects of endrin on bluntnose minnows and guppies. U.S. Bureau of Sport Fisheries and Wildlife. Research Report 58 p. 38 (Cited by Stickel, 1968).

32. OGILVIE, D. M., and J. M. ANDERSON. 1965. Effect of DDT on temperature selection by young Atlantic Salmon *(Salmo salar)*. J. Fish Res. Bd. Canada, 22: 503-512.

33. PEAKALL, D. B. 1970. Pesticides and the reproduction of birds. Scientific Amer. 222: 72-78.

34. PORTER, R. D., and S. N. WIEMEYER. 1969. Dieldrin and DDT effects on sparrow hawk eggshells and reproduction. Science 165: 199-200.

35. POTTS, G. R. 1968. Success of eggs of the shag on the Farne Islands, Northumberland, in relation to their content of dieldrin and pp' DDE. Nature 217: 1282-1284.

36. RATCLIFFE, D. A. 1967. Decrease in eggshell weight in certain birds of prey. Nature 215: 208-210.

37. ROSEN, M. N. 1970. Avian Cholera. In J. W. Davis [Ed.] Diseases of wild birds. Iowa State University Press (In press).

38. ROSEN, M. N., and A. I. BISCHOFF. 1949. The 1948-49 outbreak of fowl cholera in birds in the San Francisco Bay Area and surrounding counties. Calif. Fish and Game. 35: 185-192.

39. ——— 1950. The epidemiology of fowl cholera as it occurs in the wild. Trans. 15th N. Amer. Wildl. Conf. 147-154.

40. RUDD, R. L. 1966. Pesticides and the living landscape. Univ. of Wisconsin Press, Madison. p. 320.

41. RUDD, R. L. 1966. Pesticide kinetics in ecosystems disturbed by man. 76-87. In: J. Y. Wang [ed] Man and his environment: Interaction and interdependence, Environmental Science Institute, San Jose, California.

42. STICKEL, L. F. 1968. Organochlorine pesticides in the environment. U.S. Bureau of Sport Fisheries and Wildlife, Special Scientific Report—Wildlife No. 119. p. 32.

43. TATTON, J. O'G., and J. H. A. RUZICKA. 1967. Organochlorine pesticides in Antarctica. Nature 215: 346-348.

44. WARNER, R. E., K. K. PETERSON, and L. BORGMAN. 1966. Behavioral pathology in fish: A quantitative study of sublethal pesticide toxication. J. Appl. Ecol. 3 (Suppl.) 223-247.

45. WASSERMANN, M., D. WASSERMANN, Z. GERSHON, and L. ZELLERMAYER. 1969. Effects of organochlorine insecticides on body defense systems. In: Biological effects of pesticides in mammalian systems. Annals N.Y. Academy of Sciences 160: 393-401.

46. WIESNER, J. B. 1964. Use of pesticides: A report. The President's Science Advisory Committee (J. B. Riesner, Chairman) Residue Reviews 6: 1-22.

47. WRIGHT, B. S. 1965. Some effects of heptachlor and DDT on New Brunswick woodcocks. J. Wildl. Mgmt. 29: 172-185.

48. WURSTER, C. F., JR., and D. B. WINGATE. 1968. DDT residues and declining reproduction in the Bermuda petrel. Science 159: 979-981.