RESEARCH ARTICLE

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Effect of Pesticides on Reproductive Ability of Birds in Special Context of Biomagnification and Bioaccumulation of DDT

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ABSTRACT

Effect of pesticides is of immense importance when talked about humans. Every researcher goes to explore effect on human beings or similar mammals model. The other side of picture is less touched as it is also of very importance, the major components of ecosystem like birds. This article highlights the effect of pesticides on birds which are of great importance to the ecological point of view. The DDT is in special context for ready reference.
Key words: *Pesticides, Reproductive Ability of Birds, Biomagnification, Bioaccumulation, DDT*

INTRODUCTION

In order for biomagnification to occur, the pollutant must be:

1. long-lived
2. mobile
3. soluble in fats
4. biologically active

If a pollutant is not active biologically, it may biomagnify, but we really don't worry about it much, since it probably won't cause any problems. A pollutant is short-lived, it will be broken down before it can become dangerous. If it is not mobile, it will stay in one place and is unlikely to be taken up by organisms. If the pollutant is soluble in water it will be excreted by the organism. Pollutants that dissolve in fats, however, may be retained for a long time.

CLASSIC EXAMPLE: DDT

DDT stands for dichloro, diphenyltrichloroethane. It is a chlorinated hydrocarbon, a class of chemicals which often fit the characteristics necessary for biomagnification. DDT has a half-life of 15 years, which means if you use 100 kg of DDT, it will break down as follows-

Year	Amount Remaining
0	100 kg
15	50 kg
30	25 kg
45	12.5 kg
60	6.25 kg
75	3.13 kg
90	1.56 kg
105	0.78 kg
120	0.39 kg

This means that after 100 years, there will still be over a pound of DDT in the environment. If it does bioaccumulate and biomagnify, much of the DDT will be in the bodies of organisms. DDT actually has rather low toxicity to humans (but high toxicity to

insects, hence its use as an insecticide). Because it could be safely handled by humans, it was extensively used shortly after its discovery just before WW II. After the war, DDT became popular not only to protect humans from insect-borne diseases, but to protect crops as well. As the first of the modern pesticides, it was overused, and soon led to the discovery of the phenomena of insect resistance to pesticides, bioaccumulation, and biomagnification (Gilman, *et al.* 1977).

Birds played a major role in creating awareness of pollution problems. Indeed, many people consider the modern environmental movement to have started with the publication in 1962 of Rachel Carson's classic Silent Spring, which described the results of the misuse of DDT and other pesticides. In the fable that began that volume, she wrote: "It was a spring without voices. On the mornings that had once throbbed with the dawn chorus of robins, catbirds, doves, jays, wrens, and scores of other bird voices there was now no sound; only silence lay over the fields and woods and marsh." Silent Spring was heavily attacked by the pesticide industry and by narrowly trained entomologists, but its scientific foundation has stood the test of time. Misuse of pesticides is now widely recognized to threaten not only bird communities but human communities as well (Bennett, *et al.* 1991).

But DDT, its breakdown products, and the other chlorinated hydrocarbon pesticides (and nonpesticide chlorinated hydrocarbons such as PCBs) posed a more insidious threat to birds. Because these poisons are persistent they tend to concentrate as they move through the feeding sequences in communities that ecologists call "food chains." For example, in most marine communities, the living weight (biomass) of fish-eating birds is less than that of the fishes they eat. However, because chlorinated hydrocarbons accumulate in fatty tissues, when a ton of contaminated fishes is turned into 200 pounds of seabirds, most of the DDT from the numerous fishes ends up in a relatively few birds. As a result, the birds have a higher level of contamination per pound than the fishes. If Peregrine Falcons feed on the seabirds, the concentration becomes higher still. With several concentrating steps in the food chain below the level of fishes (for instance, tiny aquatic plants crustacea small fishes), very slight environmental contamination can be turned into a heavy pesticide load in birds at the top of the food chain. In one Long Island estuary, concentrations of less than a tenth of a part per million (PPM) of DDT in aquatic plants and plankton resulted in concentrations of 3-25 PPM in gulls, terns, cormorants, mergansers, herons, and ospreys (Peakall, 1948).

The term "Bioconcentration" of pesticides in avian fauna high on food chains occurs not only because there is usually reduced biomass at each step in those chains, but also because predatory birds tend to live a long time.

Egg shell-thinning is very important and resulted in the decimation of the Brown Pelican populations in much of North America and the extermination the Peregrine Falcon in the eastern United States and southeastern Canada. Shell-thinning caused lesser declines in populations of Golden and Bald Eagles and White Pelicans, among others. Similar declines took place in the British Isles. Fortunately, the cause of the breeding failures was identified in time, and the use of DDT was banned almost totally in the United States in 1972 (Ohlendorf, *et al.* 1986; Fox and Tom, 1980)).

Environmental contamination by agricultural chemicals and industrial waste disposal results in adverse effects on reproduction of exposed birds. The diversity of pollutants results in physiological effects at several levels, including direct effects on breeding adults as well as developmental effects on embryos. The effects on embryos include mortality or reduced hatchability, failure of chicks to thrive (wasting syndrome), and teratological effects producing skeletal abnormalities and impaired differentiation of the reproductive and nervous systems through mechanisms of hormonal mimicking of estrogens. The range of chemical effects on adult birds covers acute mortality, sublethal stress, reduced fertility, suppression of egg formation, eggshell thinning, and impaired incubation and chick rearing behaviors. The types of pollutants shown to cause reproductive effects include organochlorine pesticides and industrial pollutants, organophosphate pesticides,

petroleum hydrocarbons, heavy metals, and in a fewer number of reports, herbicides, and fungicides. o,p'-DDT, polychlorinated biphenyls (PCBs), and mixtures of organochlorines have been identified as environmental estrogens affecting populations of gulls breeding in polluted "hot spots" in southern California, the Great Lakes, and Puget Sound. Estrogenic organochlorines represent an important class of toxicants to birds because differentiation of the avian reproductive system is estrogen dependent (Bishop *et al.*, 1991).

Wild birds are very conspicuous in the landscape. Injuries to populations of birdsfrom environmental pollutants and pesticidesare obvious indicators of environmentaldamage. Rachel Carson's Silent Spring identified the urban use of pesticides (primarily DDT) as the cause of a notice able decline of birds singing in the eastern United States and also the cause of mass songbird mortalities. While direct exposure to DDT is not highly toxic to birds, heavy and repetitive use of the pesticide is. DDT was used aggressively to kill the beedes that spread Dutch Elm disease; this resulted inthe bioaccumulation of DDE in non target species of earthworms. The levels were high enough that robins and other song birds which ingested the earthworms received lethal doses of the pesticide, resulting in large losses of urban birds (Holmes, 1982).

Behavior impairment has also been correlated with organochlorine exposure to ring doves and merlins. Parathion has been correlated with altered incubation behavior in experimentally exposed mallards and laughing gulls. The effects of pollutants on reproduction are mediated at many different physiological levels. The diversity and extent of effects have been impossible to predict because many of the biochemical mechanisms of the side effects of agricultural chemicals are unrelated to the specific mechanisms of action of the designed use. The unexpected side effects, such as eggshell thinning by DDE or estrogenic effects of op'-DDT, could not be predicted behavior impairment has also been correlated with organochlorine exposure to ring doves and merlins. Parathion has been correlated with altered incubation behavior in experimentally exposed mallards and laughing gulls. The effects of pollutants on reproduction are mediated at many different physiological levels. The diversity and extent of effects have been impossible to predict because many of the biochemical mechanisms of the side effects of agricultural chemicals are unrelated to the specific mechanisms of action of the designed use (Gilman, *et al.* 1977).

The urban and agricultural use of DDT and other organochlorine pesticides resulted in localized high-residue levels in soils and plants; runoff resulted insignificant aquatic residues in estuaries, resulting in bioaccumulation by fish and predatory birds. For example, reproductive failure of grebes, ospreys, peregrine falcons, and bald eagles was caused primarily by urban and agricultural insect control measures. Point source pollution from manufacturing plants was responsible for reproductive failure of cormorants and pelicans in southern California. Breeding failures and high organochlorine levels in aquatic birds nesting in the Great Lakes implicated DDE, polychlorinated biphenyls (PCBs), dioxins, and dibenzofurans. Eggs hell thinning and breeding failure of raptors and sea birds were documented with DDT and its metabolites. DDT, however, was not the only pesticide or pollutant to affect reproduction in birds. Other persistent organochlorine pesticides with documented effects included dieldrin, endrin, aldrin, mirex, kepone, chlordane, toxaphene, hexachlorobenzene, and lindane. Most ofthe organochlorines contributed only in a minor way compared to DDT and dieldrin, and most were banned from use in the United States in the early 1970s. Many continue to be used in Asia, Africa, and South America. In estuaries, the environmental buildup of industrial chemicals such as PCBs and dioxins also occurred during the 1950s and1960s. Documented exposures to birds, especially in the Great Lakes, Long Island Sound, Puget Sound, and San Francisco and San Diego Bays, were correlated with declines in populations of fish-eating birds. In these cases, the causative agent(s) was difficult to pinpoint because several organochlorines and heavy metals (copper, mercury, selenium, tributyltin) were often found in complex mixtures (Rudd and Herman, 1972).

EFFECT ON BIRD REPRODUCTIVE HORMONES AND FUNCTIONING

Avian embryos are particularly at risk from metabolite activation because the metabolite products are not excreted from the egg but remain in the blood circulation throughout incubation. The normal nitrogenous metabolic wastes are sequestered in the allantois as a semisolid slurry of urates, and water-soluble metabolites of xenobiotics will remain in the circulation. Hydroxylated PCB congeners also have been identified as estrogenic, with a large variation in potency between hydroxylated metabolites. In experimental studies the enzymatic hydroxylation can be manipulated by the dosing regimen, with small initial doses of PCBs causing the induction of liver microsomal enzymes and subsequent larger doses being rapidly hydroxylated to active estrogens. Most of the hydroxylated metabolites formed are more water soluble than the parent congeners, and most were thought to be excreted in the urine of mammals. However, retention of 13 hydroxylated PCB metabolites at high levels has recently been shown in humans, rats, and seals, indicating both that hydroxylation is a widespread metabolic pathway and that hydroxylated metabolites may be retained in the circulation or fat (Tanabe, *et al.*, 1987; Hunt and Hunt 1977).

EFFECT OF SOME LESS POPULAR XENOBIOTICS ON HORMONAL SYSTEM

Estrogens Alkyl phenols, widely used as wetting agents, surfactants, and industrial chemical additives, are also estrogenic and have been demonstrated to have adverse effects on fish downstream of municipal wastewater discharges. In response to alkyl phenol exposure, male fish are reported to synthesize vitellogenin, an estrogenic protein synthesis by the liver and normally expressed only in females. No studies to date have implicated alkyl phenols as being estrogenic in birds. Many plants synthesize isoflavanoid phytoestrogens, which may have either estrogenic agonist or antiestrogenic effects on estrogen receptor binding (56). Most do not have significant effects on avian reproduction but may be used as chemical cues to modify reproduction. Variations in levels of isoflavanoids in clover and other legumes have been implicated in affecting reproduction in wild quail. The extent to which animals are at risk from estrogenic xenobiotics is difficult to estimate. Animals have been exposed to phytoestrogens for many generations and have apparently developed metabolic pathways to adjust to this exposure from natural sources. Most hydroxylated metabolites of organochlorines are weak estrogens, they are unlikely to be bioaccumulated because of their water solubility, and they will be excreted in the urine of adult animals before reaching concentrations high enough to adversely affect adult reproductive function. Embryonic animals, however, are sensitive to permanent developmental effects of estrogens, and the risk of exposure to hydroxylated organochlorines is largely unknown. The binding constants for organochlorines to AFP may be very different from binding constants for steroidal estrogens, and exposure of the fetus will be a complex function of maternal or fetal liver hydroxylation, estrogen receptor binding, AFP sequestration, and maternal urinary excretion. The exposure risk for avian embryos is also very different from that of mammals (Spitzer, *et al.*, 1978; Cade, *et al.*, 1971).

TERATOGENIC EFFECTS

Mortality of birds is not a specific reproductive effect, but on a population level, reproduction is impaired due to decreased numbers of breeding birds and decreased fitness of remaining adults. Sublethal exposures may adversely affect reproduction through nonspecific morbidity or increased stress, which results in cessation of lay, interruption of incubation, or reduced care of chicks. Petroleum oil exposure to breeding birds, either by exposure of the plumage or by ingestion of oil, causes increased stress with elevated circulating corticosterone and apparent feedback down regulation of reproduction at the pituitary level. Oil exposure will cause cessation of egg yolk formation, which results in reduced lay or abandonment of breeding. Laboratory exposure studies have been reviewed by Albers, demonstrating considerable variability in

sensitivity to hydrocarbon induced hemolytic anemia and induction of liver mixed-function oxidases effects that appear to contribute to increased stress and reduced breeding success. Field studies have shown that exposure to 0.1- to 2.0-ml weathered crude oil is sufficient to prevent or impair egg formation and egg laying, incubation, and stability of the pair bond (Hoffman, *et al.*1990).

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