Pesticide Poisoning in Raptors in the US

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The term pesticide includes herbicides, fungicides, rodenticides, avicides, and insecticides. There is little information on the toxicity of herbicides, fungicides, and avicides to raptors. Some of the newer, more potent rodenticides like brodifacoum have been reported to cause owl toxicity, but still little has been published.[16] The U.S. Environmental Protection Agency (EPA) registers 45,000 pesticide products containing 600 active ingredients and 1200 inert ingredients.[1] Most of the pesticide problems reported in raptors are due to the insecticides.[2,6,14,17,18,22,26,27,29,30,33,34,40,44,45,46,47]

There are four main chemical classes of insecticides: organochlorines (also called chlorinated hydrocarbons), organophosphates, carbamates, and pyrethroids. Only the pyrethroids are relatively nontoxic to terrestrial vertebrates.[5] The majority of the insecticides are used in agriculture or around the home and are sprayed or otherwise deposited in the environment. Raptors are end-stage predators and are at prime risk for coming into contact with these products either by being sprayed directly or by eating contaminated prey such as insects and small vertebrates.

Insecticides can affect free-living wildlife in five basic ways by:

1. causing direct mortality and/or behavioral abnormalities;
2. causing decreased reproduction;
3. other sublethal effects;
4. altering the habitat; and
5. altering or decreasing the food supply.

Most of the reported affects on raptors have been limited to direct mortality, behavioral abnormalities, and decreased reproduction.[14,18,22,30]

The organochlorines were the first insecticides to be widely used, starting in the 1940's with DDT. DDD and DDE are the main breakdown products of DDT.[40] DDE is more prevalent because it is more resistant to degradation in animals and nature.[40] DDE caused egg shell thinning and reproductive failure in a wide variety of avian species including the osprey (*Pandion haliaetus*), peregrine falcon (*Falco peregrinus*), and the bald eagle (*Haliaetus leucocephalus*). The general use of DDT was severely restricted in 1972 by the EPA and the population of osprey and bald eagles have increased since then.[15,39]

Unfortunately, DDT is still a threat to U.S. wildlife because of its persistence in the environment and its use in parts of Central and South America where many U.S. raptors and their prey spend the winter. Other organochlorines were or are still in use including dicofol, methoxychlor, endrin, heptachlor, lindane, and chlordane. Organochlorine insecticides are stored in body fat and birds exposed sublethally may accumulate large amounts which can then be mobilized during times of stress and result in poisoning.[40]

A number of studies have demonstrated that U.S. raptors still contain organochlorines in their bodies.[27,34,50,51,52,53,54] Some of these species are migratory, but others such as the great horned owl (*Bubo virginianus*) are not. At the Wildlife Center of Virginia, "acute" organochlorine toxicity has been diagnosed annually since 1987. The affected birds rarely have injuries and are found on the ground, away from roads and buildings. They are all emaciated and exhibit tonic-clonic convulsions, especially when stimulated. Their pupillary light reflex is present, but the pupils also dilate and constrict independently. The birds appear blind, exhibit abnormal posture, and usually cannot stand. The complete blood count (CBC) is unremarkable except for low packed cell volumes (<15%) and very low total solid values (<1.0 gm/dL).

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These birds are treated with intra-osseous or intra-venous fluids, intra-osseous blood transfusions, oral hyperalimentation formulas, vitamin B complex, and other supportive measures. Some authors recommend diazepam or barbiturates to control the convulsions.[11] There is no specific antidote for organochlorine toxicity. Few of the birds survive, with most dying in two to three days. It is uncertain whether the cause of death is due to metabolic problems related to emaciation or the chlorinated hydrocarbons.

The definitive diagnosis of organochlorine poisoning is made post mortem. Necropsy findings are nonspecific except for emaciation. Histopathology is unremarkable. Tissues must be submitted for analysis by gas chromatography. Specific residue concentrations in the brain are diagnostic.[40] In the "normal" bird the highest levels will be found in body fat, although blood can also be analyzed.[54]

Interpretation of the levels found may be difficult as toxic levels vary among species and there is some synergysm between different chemicals.[40] DDT and its degradation products DDD and DDE are best assessed together using the formula ppm DDE/15 + ppm DDD/5 + ppm DDT = DDT equivalents (ppm are in wet weight). Lethally poisoned birds have values exceeding 20 DDT equivalents.[40,42] Chlordane poisoning should be suspected whenever the sum of the brain levels of heptachlor epoxide and oxychlordane exceed 4.0 ppm.[40] Brains of heptachlor poisoned birds contain heptachlor epoxide levels exceeding 9.0 ppm.[40] Lethal levels of chlordane begin at 5.0 ppm.[40] Residues of 0.8 ppm of endrin in the brain causes death.[43] Dieldrin is considered toxic when brain levels exceed 3 to 4 ppm.[40]

In the dead bird brain and body fat, if present, are taken for analysis. The tissues are separately wrapped in aluminum foil, labeled, and frozen. The tissues are submitted for analysis by gas chromatography. The method used by our lab is that of Bertuzzi et al. Raptors tested at the Wildlife Center of Virginia have been found to have detectable levels of nine different organochlorines, most in small amounts. The highest levels are found in the great horned owls (*Bubo virginianus*). One of our great horned owls had 54.31 ppm of DDE in its fat after two years in captivity on a diet of laboratory rodents.

As organochlorines were phased out, they were replaced by the organophosphorous pesticides. These neurotoxicants inactivate the enzyme acetylcholinesterase (AChE), which is responsible for the breakdown of acetylcholine, the neurotransmitter at cholinergic nerve endings and myoneural junctions.[28] This enzyme inhibition allows the buildup of acetylcholine which results in continued stimulation and fatigue of cholinergic end-organs and muscles.[28] Death is due to respiratory failure. While organophosphates are not as persistent in the host or the environment as organochlorines, they are more acutely toxic to mammals and especially birds.[19,27,34,35,45] (see table 1)

As of June 1992, there were 36 different organophosphates contained in 2,972 different products registered by the EPA. These products have uses ranging from nematicides to external parasiticides. They come as sprays, powders, and granules. The granules are readily available and often toxic to songbirds which die and are then eaten by raptors.[2] The effects of organophospates on raptors have been well documented.[14,17,18,19,24,27,29,33,34,35,46,47] The clinical signs seen depend on the specific compound, dose, time period from intoxication to discovery, and the specie affected. They often differ from the signs described for mammals, which ressemble overstimulation of the parasympathetic nervous system.[28,33] In acutely poisoned bald eagles seen at the Wildlife Center of Virginia the clinical signs exhibited were ataxia, inability to stand, opisthotonus, and a spastic nictitans. Other less obvious signs seen in red tailed hawks (*Buteo jamaicensis*) include a detached attitude, inability to fly unrelated to any injury, and a crop full of dead songbirds. Most of the affected birds are in good flesh, but this depends on when and where they are found. Some of these birds have other more obvious injuries such as fractures and the organophosphate toxicity easily may be overlooked. Clinical signs observed by others include rigid paralysis with tightly clenched talons, rapid respirations, salivation, muscle twitching, alternating miosis and mydriasis, and the absence of a pupillary light reflex.[2,17,18,37]

Some organophosphates such as EPN and leptophos cause a delayed neuropathy syndrome which takes eight days to develop. In these cases a secondary metabolite is produced which affects peripheral axons and myelin sheaths resulting in sensory and motor neuropathy.[28] This syndrome has been produced experimentally in chickens, but has not been reported in raptors.

Treatment is often initiated based on history and clinical signs and prior to a definitive diagnosis which involves finding depressed plasma cholinesterase levels.[19,20,26,28,49] Specific treatment includes atropine sulfate at 0.5 mg/kg, which may be given intramuscularly or one fourth the dose given intravenously and the rest IM.[28,33] Atropine has no effect on the insecticide enzyme bond, but blocks the muscarinic and some central nervous system effects at the nerve endings.[28] If some reduction in the severity of clinical signs is not seen in 15 minutes the atropine dose or the diagnosis should be reviewed.[28] Diphenhydramine at 4 mg/kg IM or orally has been shown to block the effects of the nicotinic receptor overstimulation and appears to enhance nerve function and prevent receptor paralysis.[28] Its usefulness in birds has not yet been reported.

The use of pralidoxime chloride is controversial. This drug given at 20 mg/kg IM will break the organophosphate-acetylcholinesterase bond, but is most effective when administered within 24 hours of intoxication and excess drug may itself inhibit the enzyme.[13,28] This drug was used to successfully treat a golden eagle (*Aquila chrysaetos*) at the WCV.

Supportive therapy is also very important including oral, intra-osseous, or intravenous fluids as needed. If the crop is full it should be emptied. The birds should be kept in a warm, quiet place and given nutritional support until they start eating on their own. If the bird had been sprayed with the insecticide it should be washed with a dilute dishwashing detergent. These birds may show signs repeatedly after treatment if all the insecticide is not removed. Birds that are treated usually survive although their return to the wild may be prolonged. The affected golden eagle noted above was released in two weeks while an affected bald eagle needed ten months to recuperate.

Organophosphate poisoning can be definitively diagnosed in the live bird either by measuring plasma or serum cholinesterase or by identfying the pesticide in the crop or gastrointestinal contents by gas chromatography.[19,33,34] Little has been published on normal values in raptors.[20,33,34] Also sample collection, storage, and the laboratory method used will affect the results.[12,19,21] Hemolysis, icterus, and lipemia in the sample will all result in elevated values.[21] Adding to the confusion is the fact that birds have butrylcholinesterease in addition to acetylcholinesterase, which may be in higher amounts and may not be measured by the testing procedure used. Tests which measure true and psuedocholinesterase will measure both chemicals. Diagnostic kits that require a colorimeter are available but the results are reported in different units than those reported by reference laboratories. A new slide cholinesterase test became available in early 1992. This test is used with the Kodak Ektachem DT60 and DTSC dry blood chemistry analyzers. The test requires only 10 microliters of plasma and the results are reported in five minutes. Using this machine, plasma cholinesterase levels below 1 u/ml are considered depressed in red-tailed hawks, eagles, and great horned owls at the Wildlife Center of Virginia.[34] Other species appear to have higher normal values. In the absence of known normals, a sample from another member of the same specie may be run for comparison. Plasma cholinesterase levels typically return to normal within 72 hours. Brain cholinesterase levels take longer to return to normal, which explains the prolonged recovery time often seen.

Other factors may depress cholinesterase levels including repeated freezing and thawing, end-stage liver disease, and certain drugs with which wild raptors would not come into contact.[12,21] However, plasma cholinesterase is stable if kept frozen.[21]

The diagnosis of organophosphate poisoning in dead birds requires a relatively fresh carcass.[19,20] Crop and gastrointestinal contents can be analyzed by gas chromatography for specific chemicals or more commonly the brain is analyzed for cholinesterase levels. Normal values are not readily available for many species so normal controls are often run at the same time.[19,20] Brain cholinesterase levels depressed by 20% indicate exposure. Levels less than 50% of normal are diagnostic, but most poisoned birds have levels depressed by 70%.[12,19,20]

The carbamate insecticides also inhibit the enzyme, acetylcholinesterase, resulting in the build-up of acetylcholine and the continued stimulation and fatigue of choliergic end-organs and muscles.[28] The major difference from organophosphates is that the carbamate may separate from the enzyme resulting in reactivation.[28] The clinical signs, diagnosis, and treatment are the same as for organophosphate toxicity, except that pralidoxime chloride may not be useful.[28] The EPA registered 1,626 different products containing 46 different carbamate compounds as of July, 1992 and their toxicity to birds varies.

Carbofuran, a commonly used carbamate, is extremely toxic to birds. The state of Virginia banned the use of granular carbofuran in 1991. The EPA then ordered a five year gradual phaseout of the use of this product in the U.S. (EPA, Office of Pesticides and Toxic Substances, 1989). This product has been linked to deaths in the recovering bald eagle population in the Chesapeake Bay. A USDI Endangered Species Impact response states that the recovery of this population will be slowed by the impact of eagle deaths due to this product. An EPA special review team report states that granular carbofuran may be responsible for up to 2,000,000 bird deaths annually in the US. Most of these deaths involve songbirds, but secondary poisoning has been seen in many species of raptor including the bald eagle.

Research from Saskatchewan has shown that aerial application of carbofuran to control grasshoppers resulted in a 54% decrease in the number of burrowing owl young per nest and a 50% decrease in the proportion of pairs that raised one or more young if applied within 400 meters of the nest burrow.[22] This impact is thought to be a result of alterations in the parental behavior of the adult burrowing owls.

The indisputable fact is that birds of prey are still suffering mortalities and other effects from pesticides. The drastic effects seen on raptor reproduction due to the organochlorines have not been observed with the today's commonly used organophosphates and carbamates. However, direct mortalites have been seen due to these cholinesterase inhibitors. Most of the affected individuals die in the wild. Those found can be successfully diagnosed, treated, and returned to the wild.

The diagnosis of pesticide intoxication requires a complete medical workup. There are many causes of neurological signs in wild raptors including trauma, parasites, infectious agents, and other toxins. A thorough history including where and under what circumstances the bird was found is very helpful in making a diagnosis.

While there are a variety of diagnostic tests available to help substantiate a diagnosis of pesticide intoxication, it may be difficult to find a suitable laboratory to run the tests. Also, these tests tend to be expensive. Tissue pesticide screens often cost over one hundred dollars.

Another problem is the scarcity of normal tissue and blood levels of pesticides and affected enzymes in raptors. Most of the research has been conducted on mallards (*Anas platyrhynchos*) and quail (*Cotinus virginianus*).

We also know very little about the sublethal effects on these pesticides. Another question is what, if any, are the effects of the inert ingredients in these chemicals? Researchers are just starting to explore this in humans.

More research is necessary if the answers to any of these questions are to be found. Also what we have not yet discovered may be even more significant.

Table 1

Acute oral toxicities of carbamate and organophosphorous pesticides in the Mallard (*Anas platyrhynchos*)[9]

Chemical	LD50 (mg/kg)
Aldicarb	3.4
Baygon	11.9
Carbaryl	>2179.0
Carbofuran	0.4
DDVP	7.8
Demeton	7.2
Diazinon	3.5
Dicrotophos	4.2
Ethoprop	12.6
Disulfoton	6.5
Famphur	9.9
Fenthion	5.9
Fensulfothion	0.7
Fonophos	16.9
Malathion	1485
Methamidophos	8.5
Methidathion	23.6
Methiocarb	12.8
Methyl Parathion	10.0
Mevinphos	4.6
Monocrotophos	4.8
Parathion	2.4
Phosmet	1830
Phorate	2.6
Phosphamidon	3.8
Trichlorfon	36.8

An LD50 < 20 mg/kg is considered highly toxic

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