Toxins in Wildlife: A Retrospective Study

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ABSTRACT

The "Toxins in Wildlife" project was conducted as a reconnaissance study to investigate the incidence and the trends in the distribution of intoxication in wild animals in Massachusetts. After reviewing approximately 1732 animals over a 2 1/2 year period, the study confirmed the existence of death by intoxication in a wide variety of both wild mammals and birds throughout southeastern Massachusetts. It seems likely that 33 animals in 11 different case studies died as the result of intoxication by one of the following classes of compounds: chlorinated hydrocarbons, organophosphates, or polychlorinated byphenyls. Because of the manner in which animals are referred to the Wildlife Center, and because animals are never euthanized to gain data, it is likely that this represents only acute severe intoxication. It is likely that many instances, especially chronic cases, have been undetected by this study.

These intoxications were caused by people intentionally feeding poisons to animals because they were considered pests (17 animals, two cases), by people accidentally misusing toxins such as lawn pesticides (10 animals, three cases), and by people disposing of toxins without being aware of or without caring about the danger posed to the environment (six animals, six cases).

The poisonings do not occur in one easily defined habitat, geographic area or biological grouping. Passerines, waterfowl, sea birds, and rodents were found to be intoxicated. Intoxication was found in animals collected in cities, suburban areas, and in rural farmlands. Animals that frequent estuaries, fresh water, lawn, wood lots, and meadows were found to be intoxicated. Clearly this is not a simple problem.

INTRODUCTION

Evaluating the incidence of intoxication by man-made substances is best approached if only a limited number of precise compounds are studied. The reasons for this are multiple. First, the number of man-made compounds that can be toxic ranges in the thousands. Second, the flow of these compounds in the environment can be complex and third, manifestation of intoxication is highly variable and often species specific.

To sort out some of these variables the New England Wildlife Center focused the study on the incidences of three classes of compounds. These were the chlorinated hydrocarbons, the organophosphates and the polychlorinated byphenyls. The rationale for choosing these three classes of compounds lay in the fact that all three were known to cause intoxication in animals, that all three were currently or historically used or disposed of in a cavalier manner in a variety of habitats, and that commercial laboratories were able to test for these compounds and to derive a quantitative value for the level of these compounds in animal tissues.

All of these factors are important, but the last especially so. When intoxication is suspected it is not sufficient to submit the tissue and ask which compound killed this animal. The laboratory screening process is significantly more precise. It is one that requires that a particular compound be asked for. A test for that substance is then run. The result is read out as "No, the compound is not present", or "Yes, it is found at this concentration in this sample". So it is necessary to know what compound or compounds are suspected before a tissue sample is submitted.

The process for suspecting a particular toxin is complex and to some degree subjective. Issues that must be considered include the history of how the compound is used, the behavior of the compound in the environment, and the degree to which clinical symptoms suggest intoxication.

Each class of compounds considered in this study is comprised of a series of specific substances that were designed to perform a particular function or is the by-product of a substance that was designed to perform a particular function. Below is a brief description of the history, behavior and clinical symptoms of each of the classes of compounds considered in the project.

ORGANOPHOSPHATES

Organophosphates were developed in the 1930's in Germany as part of experiments to find sources of chemical warfare. They act as neural inhibitors - their presence in animal tissues interrupts normal nerve function by preventing the repolarization of a nerve cell after it has been fired. Paralysis and other neurological symptoms result. Because of their ability to kill quickly, and because the effects are dose/animal size related, organophosphates were developed as pesticides.

Intoxication is dose related. Virtually all animals are affected, but small animals more readily because of their small size. A light, uniform dusting is a high enough concentration to kill an insect-sized animal, but this amount has a smaller impact on a human-sized animal.

Organophosphates are commonly used to control insect pests, usually on farms and in gardens, although they are sometimes applied across wide geographic areas as was done by the State of Massachusetts in September, 1990 across Bristol and Plymouth Counties in an effort to control mosquito populations that were feared to be carrying high levels of Eastern Equine Encephalitis. In this instance malathion was applied from airplane carriers that sprayed a relatively uniform dusting across a variety of habitats.

The most common organophosphates include Diazinon, Dursban (Chlopyrifos), Parathion, and Malathion. Garden centers stock pesticides containing these substances and lawn care professionals commonly use Diazinon and Dursban to control grubs, nematodes and other grass feeding animals considered pests.

Diazinon was commonly used on golf courses to control these problems until it was banned from such large-scale use in the late 1980's because it was linked to deaths of Canada Geese that fed on the treated grass. It is believed to be the first pesticide banned solely because of its danger to birds. Parathion is one of the most toxic organophosphate pesticides, and numerous human deaths have been attributed to its careless use and disposal. Federal regulations restricting its use are now being put into place with little opposition from the pesticide industry. Malathion is a much less toxic substance. By way of comparison it takes approximately 600 times as much Malathion by weight to kill a Mallard as it does Parathion.

In cases where more than one animal died in a group with an acute onset, for no other apparent cause, then organophosphate poisoning was suspected.

CHLORINATED HYDROCARBONS

Chlorinated hydrocarbons were the pesticides of choice before organophosphates came into use. They were discovered in 1939 and were used from the 1940's into the 1960's. Towards the end of this period resistance by the target insects to these compounds started to develop and concurrently the impact on wildlife began to be understood. Use of these pesticides, which include DDT and Chlordane, were severely restricted in the 1970's. Many of these pesticides persist in the soil for years, so even though they are no longer regularly used, birds can still be exposed to them. Not only do the chemicals persist in the environment, but they also stay in the body tissue of the animal and are not metabolized; so they will accumulate in an animal over time. If this animal falls prey to a predator the full load of pesticide is passed on to the predator where it will persist. In this fashion the concentration of the pesticide will be magnified with each predator up the food chain.

Although chlorinated hydrocarbons are severely restricted in the United States, at this point in time a variety of third world countries still utilize them. It is suspected that long distance migratory birds may be currently carrying these toxins northward from Latin America when they migrate.

While songbirds that eat poisoned insects are the most likely type of bird to be first exposed to these pesticides, it is often raptors that suffer permanent visible damage. DDT, the best known of the chlorinated hydrocarbons, works at the cell membrane level inhibiting the flow of electrolytes in and out of nerve cells. Often in songbirds the pesticide will be just high enough to debilitate the bird to the point of allowing it to be captured by a bird of prey. As a result it is likely that these toxins are preferentially introduced into the food chain.

In general, acute toxicity is less commonly seen in animals than chronic toxicity that is a result of biomagnification. At necropsy, in acute cases there is evidence of edema of the lungs, heart and nerve tissues. In chronic exposure there are changes in liver and fat tissue. Different species of animals will manifest different signs. One of the most commonly recognized signs of chlorinated hydrocarbons is the fact that they cause egg shell thinning.

Signs of neurological impairment, especially in an animal that is high on the food chain raises the index of suspicion that this animal is suffering from chlorinated hydrocarbon intoxication.

POLYCHLORINATED BIPHENYLS

Polychlorinated biphenyls (PCB's) were discovered in the 1800's and have since been used in insulation material for electrical devises and are found in hydraulic fluid, lubricants, fire retardants, plasticizers and paints. They also are a byproduct of the bleaching of wood pulp procedures used by the paper industry.

PCB's are extremely stable compounds and are similar in structure and share many of the physical properties of chlorinated hydrocarbons. PCB's are found in dumps, industrial discharge, leakage from transformers and capacitors and fluids used on unpaved roads for dust control. PCB's are heavier than water and when disposed of in aquatic settings sink to the bottom of ponds, lakes or harbors forming a sludge with other sediments. This sludge can persist in the environment for decades and continues as a source of poison as long as it remains.

Little is known about how PCB's act in the body, but it is likely they interfere with the flow of water in and out of cells. The most pathological sign of PCB intoxication is tissue edema, often in relation to cardiac tissue. Any prominent edema qualified an animal as a PCB suspect. Exposure can be acute or chronic. Diagnosis can be confirmed by presence of PCB's in liver or body fat tissues.

Because many former PCB disposal sites have been identified historically, birds suspected of toxin exposure coming from these areas were considered for research purposes in the current study as having potentially been exposed to PCB's. These birds were often at the top of the food chain and as a result were suspected of potential exposure to both PCB's and chlorinated hydrocarbons. This combination is so common that Hazelton Laboratories, one of the commercial laboratories to which we submitted samples, combines both tests under one screening.

MATERIALS AND METHODS

Only dead animals were considered in this study. All animals admitted together, having suffered similar events, were considered as one case. As a result one animal may comprise a case or multiple animals may comprise a case.

Reviewing cases consisted of determining the likelihood that death was caused by intoxication. That process followed a typical medical rule-out protocol. In this procedure, evidence to support the cause of death was reviewed against the likelihood that the disease process was one of trauma, infection, neoplasia, developmental abnormalities, or intoxication. The categories with the least clinical support were ruled out as unlikely candidates. As new data are gathered, support for one cause of death generally becomes prominent. Data for the rule-out process were gathered by conducting thorough histories, clinical evaluations, gross necropsies, histopathology, and finally, chemical screenings in cases of high suspicion.

As complete a history as possible was obtained. In many instances, as is typical in wildlife medicine, little was known about the signs and symptoms preceding patient presentation. The history often was relegated to the little bit of information that the carrier of the animal provided. In cases where the history or the clinical signs indicated intoxication early in the review process, a field investigation was conducted by a NEWC staff member. Figure 1 is a work sheet developed for this purpose to help sort out the salient features of these suspect cases. As the study progressed, it became apparent that there were several patterns of clinical signs or death that raised suspicions of intoxication. Any indication of multiple animals dying acutely within close proximity to one another or instances of reported multiple deaths within one species strongly suggested a common cause of disease. Intoxication is often the principal rule-out in these circumstances because it is rare that infection, neoplasia, physiological or developmental disease can work so synchronously.

A high degree of suspicion was also assigned to cases of neurological dysfunction. The chlorinated hydrocarbons and organophosphates in cases of acute poisoning cause prominent abnormal neurological signs. It is also possible to see neurological signs caused by polychlorinated biphenyls, although it is less commonly seen.

Gross necropsies were perhaps the most reliable method of gathering information. Unfortunately acute intoxication often manifests with little significant change in gross tissue features. It was sometimes possible to find white chalky material or crystalline material in the stomachs of animals poisoned with organophosphates. The polychlorinted byphenyls and the chlorinated hydrocarbons are not so readily detected. The value of the gross necropsies lay in the fact that many bacterial, some viral and most parasitic infections will show readily apparent tissue changes. These items were weeded from the sample.

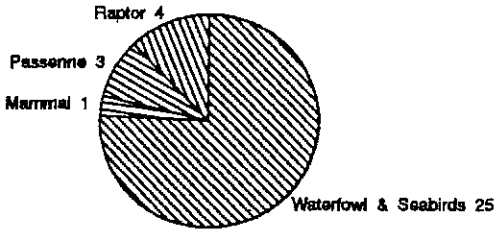
MATERIALS AND METHODS (continued)

Tissue samples that were to be screened for toxins were stored frozen in glass bottles until a group was shipped to one of two commercial laboratories. These two were the Laboratory of Veterinary Diagnostic Medicine at the University of Illinois at Urbana-Champaign, Illinois, and the Hazelton Laboratories of America, Inc. in Madison, Wisconsin. These two laboratories were chosen because of their work in veterinary toxicology. Both laboratories provided similar date. One lab was chosen over another according to work and time schedules of the participating parties.

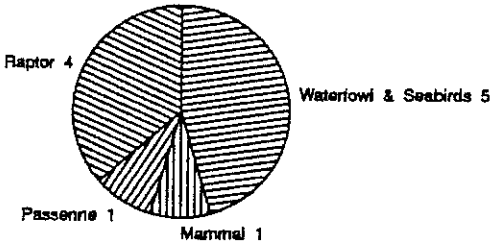
RESULTS

Approximately 2,000 animals died or were admitted dead on arrival to the Center during the duration of the study from Jan. 1, 1989 to June 1, 1991. Of these, 31 cases consisting of 59 animals were considered after case reviews to have a high enough index of suspicion of intoxication to warrant chemical screening. Chart 1 on Page 19 describes all the animals whose tissues were submitted for chemical screening. Of these, 11 cases, consisting of 33 animals, are considered positive for a likely cause of death by intoxication. Chart 2 on Page 15 summarizes the animals believed to have died as a result of intoxication.

TYPES OF ANIMALS POISONED



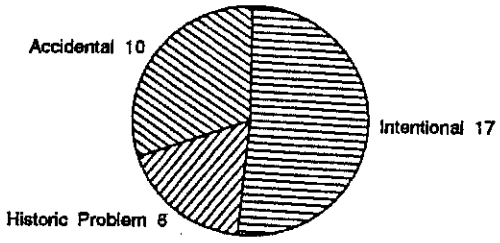
By Individual



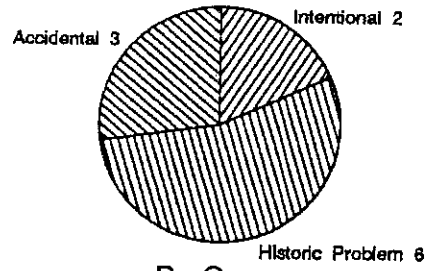
By Case

Of the 33 animals, 25 of them (75%) were waterfowl and seabirds, 4 (12%) were raptors, 3 were perching birds (9%) and one was a squirrel (3%). Of the suspected poisonings, there were three multiple cases. Two of these multiple cases were waterfowl. Waterfowl tend to get poisoned in large numbers because they travel as a group. It is sometimes better to consider the cases of bird poisonings as opposed to looking at the wider dual numbers. For example, while only 13% of the poisoned animals were raptors, that group made up 37% of the 11 cases.

POISONING METHOD



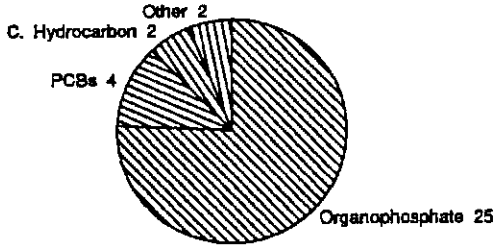
By Animal



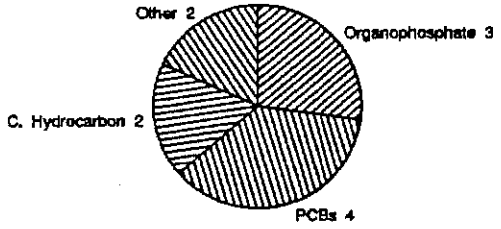
By Case

The rationale of poisoning was divided into three categories: 17(51%) were intentional, 6 (18%) were caused by historical environmental problems and 10 (30%) by misuse of pesticide. Once again, it's important to consider the case numbers because multiple cases with high numbers can skew the perception. For example, while 51% of the animals were poisoned intentionally, that was only two cases (18%). Six cases (54%) were poisoned because of historical environmental problems and 3 cases (27%) by misuse of pesticide. By considering bird numbers alone, it would appear that historical environmental causes were not a big concern because only 19% of the total birds considered died by the means. But the fact that more than half the cases were related to these problems shows they are widespread. By the nature of biomagnification and the insidious nature of chlorinated hydrocarbon intoxication it is possible that the study results are skewed because one sick animal is not often detected whereas groups of dying animals are.

TYPE OF POISON



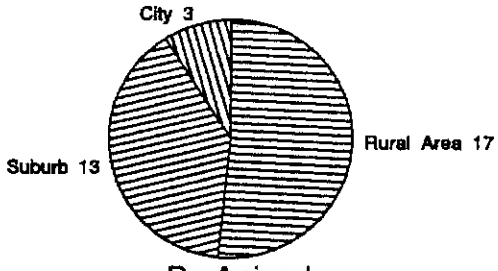
Number of Animals



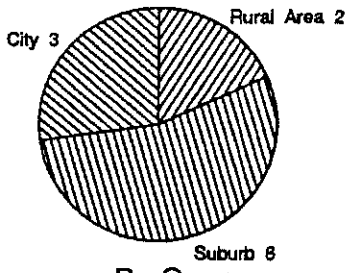
Number of Cases

By design, three classes of toxins were considered in this study: 25 birds (75%) were poisoned by organophosphates, 2 birds (6%) by other intoxicants. Again, the number of cases differs from that of the total number of animals. Three cases (27%) were killed by organophosphates, 2 cases (18%) by chlorinated hydrocarbons, 4 cases (36%) by PCB's and 2 cases (18%) by other means. While just considering the animal numbers show a large majority of cases of organophosphate contamination, the case numbers show it is more evenly distributed among the three types of toxins considered.

WHERE POISONING OCCURRED



By Animal



By Case

Of the animals, 17 (51%) were found in rural areas, 13 (39%) in suburban communities and 3 (9%) in a city. While those figures make it look like most poisonings occurred in rural areas, the case numbers show 6 cases (54%) occurred in suburban communities while 2 cases (18%) occurred in rural areas. Three cases (27%) occurred in urban settings.

CONCLUSION

There are several important findings in this study. The first, and probably the most important, is that there are wild animals dying as a result of intoxication in Massachusetts. They come from widely different habitats, geographic areas and vertebrate taxa. This study, although limited to only three classes of compounds, indicates that toxins in the environment have a widespread and complicated impact on wildlife. More study at a variety of levels is urgently needed if these phenomena are to be understood. The importance of a better understanding is not only directly related to the health of wildlife species but also to the human community, because all species ultimately share the whole environment.

Perhaps the most important result of this study lies in the use of these data in our educational endeavors. Already the toxin project has been the subject of several in-house medical seminars for our interns and volunteers. It also has been presented to the pubic in our New England Wildlife Lecture Series. We plan to submit a non-technical manuscript to one of the regional nature magazines like SANCTUARY or MASSACHUSETTS WILDLIFE for review.

The results of this study are also being used in our clinical approach to wildlife patients. Because of the finding that many species are affected over a wide range of habitats and geographic areas, these results have not allowed us to lower our guard for any incoming patients. Intoxication must be considered as a potential rule out for any wildlife admission.

It is also important to note that this study has served as a planning guide for further research into the incidence and distribution of man-made poisons in wildlife. Now that intoxication in Massachusetts' wildlife is confirmed to be a broad and disparate problem further investigation is needed on the specifics of its occurrence.

One other table use of the data came as a result of two legal suits brought against the AVX Corporation of New Bedford, MA. One action was brought by the United States Environmental Protection Agency and the other by the commonwealth of Massachusetts Attorney General's Office. In both cases, results from tissue samples from two intoxicated common terns were utilized in developing cases against the defendant for the improper disposal of hazardous waste. The case settled before trial but the data provided by the Center figured in the final disposition.

The New England Wildlife Center is now prepared to investigate this problem from one of several possible perspectives. It is feasible to study the problem in a small geographic area like a specific harbor or wetlands in which it is now known that toxins occur. Equally valid is a study that traces the distribution and movement of a particular toxin in a specific well-defined habitat like a salt marsh or harbor basin. Perhaps the most appealing approach from the Center's view point is the study of the effects of a specific toxin in a particular taxa like diving ducks or passerines.

In any event the impact of toxins in wildlife in Massachusetts requires further investigation if we are successfully to safeguard environmental quality against further degradation.

Cases of Suspected Intoxication

Case No.	Species	Town	Screened	Poisoned	Source	Comments
1	Mallard Duck	Weymouth	Yes	Unlikely		Some diazinon, feet paralyzed, no spray in 3 years
2	Blue Jay	E. Weymouth	Yes	No		Hit window, report states: "Not poisoned"
3	American Robins	N. Dighton	Yes	No		3 birds submitted, report says: "No poison indicated"
4	Yellow-shafted Flicker	Norwell	Yes	Unlikely	Lawn Pe..	
5	Common Grackle	Scituate	Yes	?-Likley	Pesticide	Many birds affected, regional pest. appli., low levels
6	Red-tailed Hawk	Danvers	Yes	Unlikely		No stated suspicion, low PC, Clor. Hy. levels
7	Blue Jay	Pembroke	Yes	Unlikely		Incomplete report, screen showed low OP
8	American Robin	Braintree	Yes	?-Likely		Body position indicates poison, low OP levels
9	American Crow	Westwood	Yes	Unlikely	Pesticide	Elevated Chlor. Hu., but below poison guidelines
10	American Crow	S. Weymouth	Yes	?-Likely		Found paralyzed, diarrhea, low OP levels
11	Blue Jay	Bedford	Yes	Unlikely		Report states: Unlikely toxin related"
12	Canada Goose	Norton	Yes	Likely	Pesticide	High diazinon levels - likely poisoned by pesticide
13	American Kestrel	Boston	Yes	Likely	Pesticide	Possible rat poisoned, high Chlor. Hy. levels
14	Great Horned Owl	Weston	Yes	Likely		Tremors, suspect chlordane poisoning
15	Common Tern	Marion	Yes	Poisoned	Haz Waste	One of two, believed poisoned by PCB's in bay
16	American Kestrel	Somerville	Yes	Likely	Haz Waste	Found at chemical plant, smelled of oil, some levels
17	Mallard Ducks	Pembroke	Yes	Unlikely		No indications of poisoning, elevated diazinon levels
18	Eastern Screech-Owl	Manomet	Yes	Unlikely		Paralyzed, no sign of trauma, low chemical levels
19	Common Grackle	Milford	Yes	Likely	Pesticide	Many dead found, toxins nearby, high diazinon levels
20	Blue Jay	Dracut	Yes	Unlikely		Spasms, body position indicate CNS, no chem found
21	Slate-colored Junco	Dracut	Yes	Unlikely		Found dead with 54, no OP
22	Red-breasted Merganser	Weymouth	Yes	Likely	Haz Waste	Screen on stomach, brain, liver showed high PCB's
23	Cooper's Hawk	Marion	Yes	Likely	Haz Waste	Spasms, can't fly, near terns, high Chlor. Hy., PCBs
24	Roseate Tern	Marion	Yes	Unlikely		Screening revealed no toxins
25	Great-horned Owls	Topsfield	Yes	Unlikely		Two submitted, low Chlor. Hy. levels
26	House Finch	Hingham	Yes	?-Likely	Pesticide	Suspect malathion, no stomach for OP screen
27	Common Tern	Marion	Yes	Poisoned	Haz Waste	One of two terns believed died from PCB in bay
28	Must Swan	Scituate	Yes	Unlikely		Bird couldn't stand, no evidence of OP in screen
29	Barred Owl	Halifax	Yes	Unlikely		No visible injury, doesn't move, low Chlor, Hy. levels
30	Canade Goose	Franklin	Yes	Likely	Poisoned	16 birds, screen revealed high ethyl parathion levels
31	Brown Trout	N-W Boston	Yes	Unlikely		Suspect malathion, liver screen showed low OP levels
32	Tree Swallow	Westport	Yes	?-Likely		Several found, bayberry seeds, no stomach for OP

Intoxication Confirmed by Screening

Case No.	Species	No. of Animals	Type of Animals	Type of Oxins	Poison Method	Location
1	Canada Goose	16	Waterfowl	Organophosphate	Intentional	Rural, farm
2	Cooper's Hawk	1	Raptor	PCB's	Haz. Waste	Rural, seaside
3	Red-breasted Merganser	1	Waterfowl	PCB's	Haz. Waste	Suburbs
4	Common Grackle	3	Passerine	Organophosphate	Accidental	Suburbs
5	Gray Squirrel	1	Mammal	?	Intentional	City
6	American Kestrel	1	Raptor	Petroleum	Accidental	City
7	Common Tern	2	Waterfowl	PCB's	Haz. Waste	City, seashore
8	Great Horned Owl	1	Raptor	Chlorinated Hy.	Haz. Waste	Suburbs
9	American Kestrel	1	Raptor	Chlorinated Hy.	Haz. Waste	City
10	Canada Goose	6	Waterfowl	Organophosphate	Accidental	Suburbs