

US EPA ARCHIVE DOCUMENT

59901 famphur
aldicarb
~~monocrotophos~~ phorate

OCT [1] 1987

RECENT PESTICIDE-RELATED MORTALITIES OF EAGLES IN IDAHO

Presented by Special Agent Michael Sutton,
U.S. Fish and Wildlife Service at the Annual Meeting of the
Raptor Research Foundation, Boise, Idaho, October 1987

Introduction

I'd like to talk first this afternoon about poisoning as a cause of eagle mortality, then discuss recent trends in the use of agricultural pesticides in the West. I'll present several case histories of pesticide poisonings of eagles in Idaho, and finally offer some recommendations as to how we can address and perhaps control the pesticide-related mortality of eagles.

As most of you are probably aware, the bald eagle is protected by three different federal laws and the golden eagle by two of these statutes. In addition, eagles are protected by various state laws throughout the country. The first federal law covering eagles was the Eagle Protection Act, passed in 1940. At that time, only the bald eagle was protected by the Act; the golden eagle was added in 1962. The second federal statute which protects eagles is the Migratory Bird Treaty Act. Eagles and other raptors were brought under the umbrella of this Act in 1972. And finally, the bald eagle is also protected by the Endangered Species Act. This species was listed in 1978 as endangered in 43 states and threatened in 5, including Oregon and Washington.

History

Under these federal statutes, the Fish and Wildlife Service has the responsibility to investigate eagles mortalities from all causes throughout the United States. Over the past 25 years, bald eagles found dead have been submitted to the National Wildlife Health Research Center in Madison, Wisconsin for necropsy. During the period 1963 through 1984, more than 1,400 bald eagles were necropsied at the Madison lab. The results of these analyses allow us to recognize what have come to be known as the "Big Five" causes of eagle mortalities in the United States: Shootings, impact injuries resulting in trauma, poisonings, electrocutions, and trapping. Note that poisonings have traditionally ranked third in importance as a cause of bald eagle mortalities, accounting for between 11.1% and 12.6% of all mortalities. This proportion varied among the states, and was substantially greater in some western states. About half of the poisoning cases involved heavy metal toxicosis, such as lead and mercury poisoning. Twenty-two percent of the poisoning mortalities involved the so-called "economic" poisons such as strychnine, 1080, and cyanide. Pesticides, primarily organochlorines such as DDT and dieldrin accounted for the remaining 25% of poisoning mortalities. However, it is important to note that the National Wildlife Health Research Center did not begin screening for all types of pesticides on a regular basis until two years ago, in 1985.

Since 1972, when the insecticide DDT was banned in the U.S. because of its persistent environmental effects, farmers and ranchers have been shifting away from the use of organochlorines such as DDT

towards the use of organophosphate and carbamate pesticides, particularly in the western states. In contrast to the organochlorines, the organophosphates and carbamates degrade quickly in the environment but are much more acutely toxic, particularly to birds. These pesticides act by interfering with the parasympathetic nervous system through inhibition of brain enzyme action. A 50% or greater depression of the enzyme acetylcholinesterase in the brain is indicative of death due to organophosphate or carbamate poisoning.

Case Histories

Before I get into the case histories of recent eagle poisonings in Idaho, let me explain what happens when an eagle is found dead or injured and pesticide poisoning is suspected. First, the local wildlife biologist, conservation officer, or Fish & Wildlife Agent is notified, and the bird is located and identified. A primary field investigation is conducted, involving photography and collection of the carcass in a safe manner. Preliminary cause of death is determined, if possible, and the site is detoxified, usually by burying any remaining toxicant. The eagle is then sent to the National Wildlife Health Research Center in Madison for necropsy. The lab determines the probable cause of death and identifies the pesticide to family by measuring enzyme activity in the brain tissue. Samples from the gut tract of the eagle and samples of any toxicant found at the scene are then submitted for identification to the Patuxent Wildlife Research Center or a local toxicology lab. As soon as the toxicology report is received, a secondary investigation is

begun. A number of techniques are employed, ranging from interviews to undercover work and surveillance in order to determine whether the poisoning was intentional, and if violations of any laws have occurred. Prosecutions are initiated against individuals or corporations responsible for intentional misuse of a pesticide which results in the death of an eagle. The pesticide manufacturer is also usually notified of an eagle mortality at the hands of their product, and negotiations are begun which may result in the alteration of labelling or eventual removal of a particular formulation from the market.

Six bald eagles and three golden eagles were found poisoned by a variety of pesticides in Idaho between 1985 and 1987. Five of these eagles died in two incidents in southeast Idaho during 1987 involving intentional poisonings by sheep ranchers. These case histories will illustrate the different types of pesticide poisonings we have experienced and give you some insight into the nature of these investigations.

Probably the most common example of accidental pesticide poisoning of eagles in the western states involves an organophosphate known as famphur, brand name Warbex, a pour-on insecticide use to control cattle grubs. Recently, for example, 26 bald eagles were killed near Nanaimo, on Vancouver Island, British Columbia by feeding on one cow carcass which had been treated with Warbex. In February, 1985 a bald eagle was found dead under a roost tree on the Pruitt ranch in Elmore County, Idaho. The carcass was submitted to the National Wildlife Health Research Center for necropsy. The mouth,

esophagus, and stomach were found to contain red meat fragments and fine gray hairs, as if the eagle had been feeding on cattle carrion. Brain enzyme activity was depressed by 81%. Stomach contents were sent to the Patuxent Wildlife Research Center for toxicology, which showed famphur in the gut tract. We concluded the eagle was poisoned by famphur due to feeding on a livestock carcass recently treated with Warbex.

Similarly, in March, 1986 a bald eagle was found dead on an island in the South Fork of the Snake River in Bonneville County, Idaho. Necropsy showed brain enzyme activity inhibition and the remains of a large blackbird in the stomach contents. Toxicology found famphur in the stomach contents of the eagle, and in the crop contents of the blackbird. Investigation revealed widespread use of Warbex by cattle ranchers in the vicinity. We concluded that the eagle died due to ingestion of a blackbird which had been poisoned by feeding on or near cattle treated with Warbex.

Of greater concern than these accidental poisonings is a marked rise in the intentional misuse of pesticides to kill eagles. For example, in February, 1987 an adult female bald eagle and an immature raven were found dead at a sheep carcass on federal land in southeastern Idaho. The site receives heavy sheep grazing pressure during the winter, and the allotments are managed by the Bureau of Land Management. The sheep carcass was found to be laced with small dark gray granules. The eagle and raven were submitted for necropsy within ten days. Both birds were in good body condition, and both contained recently ingested fine white wavy hairs, indicating they

had been feeding on the sheep carcass. Brain enzyme activity was depressed consistent with poisoning by a carbamate pesticide. Aldicarb, brand name Temik was found in the eagle esophagus and stomach contents, and in the raven stomach contents. Samples from the sheep carcass tested positive for aldicarb. Additional laced sheep carcasses were found nearby which also tested positive for the pesticide. We concluded the eagle and raven were poisoned by the intentional misuse of Temik for predator control.

About the same time in February, 1987 a bald eagle and three golden eagles were found dead at the remains of a sheep carcass near the Wyoming-Idaho line, several hundred miles south of the previous incident. The eagles were found to have recently ingested meat and small dark gray granules. Brain enzyme activity was depressed approximately 50% in each eagle. Aldicarb was detected in the stomach and esophageal contents of all eagles, and was also found in samples of the sheep carcass submitted for analysis. As in the previous incident, we concluded the eagles were poisoned by the intentional misuse of Temik for predator control.

Later the same month, an adult female bald eagle was found dead underneath a roost tree in southeastern Idaho. The bird had recently ingested fatty tissue containing fine white wavy hairs. Brain enzyme activity was severely depressed at 90% inhibition. Thimet, an organophosphate insecticide, was detected in the stomach and esophageal contents of this eagle. We concluded that the eagle was probably poisoned by the intentional misuse of Thimet for predator control.

~~organophosphate~~
phosphate

Conclusion

Thimet, Temik, Warbex, and other hazardous insecticides are available in farm chemical stores throughout Idaho and the west. The manufacture, distribution, and application of these chemicals is controlled by the Environmental Protection Agency and the Food and Drug Administration. Each pesticide is registered with the EPA and labelled according to its approved use. Most of these pesticides are designated "restricted-use", and supposedly can only be purchased and use by applicators who are licensed by the states. However, in many states including Idaho, farmers need only attend a half-day training session to be licensed as applicators. Federal and state pesticide statutes prohibit the purchase of restricted-use chemical by unlicensed individuals, and the use of any pesticide in a manner inconsistent with its labelling.

In conclusion, these findings lead us to believe that the poisoning of bald and golden eagles by the misapplication of acutely-hazardous, restricted-use pesticides may be more widespread than we realize or have been able to document. With this in mind, I offer the following recommendations:

- 1) Eagle mortalities must be fully investigated, with timely notification of state agencies and the Fish and Wildlife Service. Operating procedures to be followed in cases of eagle mortalities should be established in each state.
- 2) Facilities must be available for rapid toxicology screening. Cases of intentional pesticide poisoning should be given first

priority at the Patuxent Wildlife Research Center and at the new National Wildlife Forensic Laboratory, under construction in Ashland, Oregon. This is essential for the timely investigation of eagle and other raptor mortalities related to toxicants.

- 3) Raptor rehabilitators and veterinarians must be familiarized with the treatment of sublethal poisonings by organophosphate and carbamate pesticides. The administration of medications must be immediate if a bird is to survive.
- 4) State and federal pesticide statutes and regulations, such as applicator licensing requirements, must be stiffened and enforced. States should give serious consideration to following the lead of California, where some restricted-use pesticides are dispensed only by county extension agents.
- 5) The use of less hazardous insecticides such as pyrethroids should be encouraged or required, and the use of extremely toxic pesticides should be curtailed or banned in areas where endangered species, including bald eagles, have been affected. The EPA has recently proposed this type of ban, but it is meeting with stiff opposition in the farm lobby and Congress.
- 6) And finally, the manufacture and use of alternative, less toxic formulations of hazardous pesticides should be encouraged, even though these changes may be costly and time-consuming. Only with the full cooperation of state agencies, chemical companies, farmers, and ranchers can we keep these pesticide poisonings from becoming a real problem for wildlife conservation.