Pg 2

ALBUQUERQUE F.O.	
	Peterson
	Donahoo
	Burton
	Hanson
	McDonald
	Mullins
	O'Brien
	Roehm
	Roy 3/19
	Lovato
	Montoya
	File

UNITED STATES
DEPARTMENT OF THE INTERIOR
FISH AND WILDLIFE SERVICE
Ecological Services
Suite D, 3530 Pan American Highway, NE
Albuquerque, New Mexico 87107

March 19, 1990

Memorandum

To: Dan Marshall, Special Agent, Division of Law Enforcement, U.S. Fish and Wildlife Service, Albuquerque, New Mexico

From: Field Supervisor, U.S. Fish and Wildlife Service, Ecological Services, Albuquerque, New Mexico

Subject: Final Report, Albuquerque Ecological Services, interpretation of cause of death of 3 golden eagles and 1 bald eagle Case # NM-A-1, NM-A-2, NM-1-3, NM-A-5

Attached is the report prepared by my staff Environmental Contaminant Specialists on the cause of death of the 3 golden eagles and 1 bald eagle found near Folsom, New Mexico.

This report interprets the results of the analysis conducted by the Service National Wildlife Health Research Center at Madison, Wisconsin (Madison) and the Patuxent Analytical Control Facility (PACF) at Patuxent, Maryland. The specialists concluded that the eagles were killed by carbofuran for the following reasons: a) Madison determined that brain cholinesterase (ChE) levels were reduced; b) the PACF confirmed the presence of the pesticide carbofuran; c) the dosage of carbofuran detected in the stomach contents of the eagles have been shown to be acutely toxic to other bird species; d) the ChE activity was depressed to a level considered to be diagnostic of the cause of death by either an organophosphate or carbamate compound and; e) a carbamate compound was further indicated by the percent of ChE recovery.

If you have any questions, please call Tom O'Brien or Richard Roy at (505) 883-7877 or FTS 474-7877.

Sincerely yours,

John C. Peterson

Attachment

cc: (w/cy att)
Regional Director, U.S. Fish and Wildlife Service, Fish and Wildlife Enhancement, Attn: Charlie Sanchez, Albuquerque, New Mexico

Introduction:

On February 1, 1990, U.S. Fish and Wildlife Service (Service) Special Agents collected three golden eagles (Aquila chrysaetos) and one bald eagle (haliaeetus leucocephalus) found dead near bait stations in northeastern, New Mexico. The eagles were transported to the Albuquerque law enforcement office. The golden eagles and the bald eagle were sent to the Fish and Wildlife Service National Wildlife Health Research Center in Madison, (Madison) Wisconsin for necropsy and brain cholinesterase (ChE) inhibition analysis. Digestive tracts from the eagles were sent to the U.S. Fish and Wildlife Service Patuxent Analytical Control Facility (PACF) at the Patuxent Wildlife Research Center in Maryland for pesticide confirmation.

Methods:

The confiscated eagles were frozen within several hours after being obtained and were shipped packed on dry ice. They remained frozen until they were received by Madison on February 7, 1990. Cholinesterase was determined through the Ellman method described by Hill and Fleming (1982). Cholinesterase levels returned to normal after 18 hours of incubation. This is indicative of a carbamate poison. The values were then compared to ChE levels from "normal" eagles (Hill, 1988; Windingstad 1990 pers. comm). A complete necropsy was performed by Madison according to their established protocols. The digestive tracts and their contents remained frozen and were shipped to PACF from Madison.

The PACF conducted organophosphorus/carbamate compound analysis. This method involves homogenization of the sample (gut content) followed by mixing with acetone and methylene chloride to separate the pesticides from the tissue. The organic extract is filtered and adjusted to volume prior to gas chromatography (GC) using a flame photometric detector for organophosphorus determinations and a nitrogen phosphorous detector for carbamate determinations. Megabore capillary columns are used for the GC separations. Results of the analysis were confirmed using GC/MS confirmation. These analysis were performed by the PACF in conformance with the Environmental Contaminants Research Branch Quality Assurance Program. The accuracy and precision of all analysis, spiked samples and duplicates was acceptable.

Results and Discussion:

The results from Madison indicated that ChE was reduced between 79 percent to 90 percent in the four eagles, and carbofuran (furadan) was detected by the PACF in all samples of gut contents found in the eagles (Table 1). According to Ludke et al. (1975) brain ChE depression of > 50 percent is diagnostic of the cause of death from anticholinesterase compounds, and that 20 percent depression is indicative of exposure to these compounds.

Cholinesterase is responsible for "controlling" the electrical impulses through the nerve synapse. Choline is the chemical responsible for the

electrical impulse that travels from nerve cell to nerve cell (Murphy 1986). When ChE levels are depressed, electrical impulse are not controlled and the nerves continuously "fire". This results in a short circuit of the nervous system. Carbofuran (furadan) is carbamate pesticide used primarily on corn crops, and kills by inhibiting ChE. This pesticide has a high acute toxicity and is a Class I toxicant (Extremely toxic, LD50< 40 mg/kg) (Hill, et al. 1975). Carbofuran as with other carbamate pesticides, have reversible affects unlike organophosphate pesticides. However, carbofuran fed to birds (Japanese quail) in experimental situations evoked almost immediate response and some died within 30 minutes of ingestion (Hill, 1989).

Table 1

	Sample Identification Number			
	NM-A-1	NM-A-2	NM-A-3	NM-A-5
Carbofuran concentration in gut content (PPM wet weight)	9.3	7.8	40.0	11.0
Measured ChE activity (umols/g/min)	2.7	3.5	1.9	3.1
% of normal ChE activity	84	79	90	80
Normal ChE activity (umols/g/min)	14(n=16) (Hill, 1988) 16.42(n=25) (Windingstad, 1990)	14(n=16) (Hill, 1988) 16.42(n=25) (Windingstad, 1990)	14(n=16) (Hill, 1988) 16.42(N=25) (Windingstad, 1990)	16(n=6) (Hill, 1988) 16.42(n=25) (Windingstad, 1990)
Estimated lethal dosage of carbofuran (mg carbofuran/Kg eagle)[1]	.057	.469	0.80	0.65
Known LD50 for several bird species (mg/kg)[2]	0.238 to 12	0.238 to 12	0.238 to 12	0.238 to 12

1) $\frac{\text{Feed eaten (g)}}{\text{weight of bird (g)}}$ x pesticide residue (ppm) = mg carbofuran/kg of eagle

2) Smith (1987)

Secondary poisoning of red-shouldered hawks by carbofuran has been documented by Balcomb (1983). He found two red-shouldered hawks that had eaten rodents and birds that had died as a result of carbofuran poisoning. One hawk with 96.6 ug/g carbofuran in the digestive tract, was found severely paralyzed and was sacrificed. The other bird had less severe symptoms and was released within 24 hours.

Acute symptoms of carbofuran poisoning in birds include loss of muscular coordination, wings crossed over the back, head nodding, vocal sounds, salivation of brown fluids, tears, diarrhea, immobility with wings spread, labored breathing, eye pupil construction, arching of back, and arching of head over back; death may be as sudden as five minutes after ingestion (Tucker and Crabtree, 1970; Balcomb, 1983).

Conclusion:

There is conclusive evidence that the three golden eagles and the one bald eagle died as a result of eating meat that had been tainted with carbofuran and/or by secondary poisoning from consuming tissue from animals that had died after feeding on the cow carcasses. We can make that determination from the ChE inhibition and the presence of carbofuran in the gut. In addition, the estimated dosage for each eagle is within the range of the LD50 (lethal dose to 50 percent of the test organism) for several bird species (Table 1). There is a strong indication that eagles may be more sensitive and have a lower LD50 dose response. The amount of carbofuran ingested may also have been greater because it is quickly absorbed by birds, through the digestive tract. The rate of absorbation cannot be determined in this situation.

LITERATURE CITED

Balcomb, R. 1983. Secondary poisoning of red-shouldered hawks with carbofuran. J. Wildl. Manage. 47: 1129-1132.

Hill, E.F., R.G. Heath, J.W. Spann, and J.D. Williams. 1975. Lethal dietary toxicities of environmental pollutants to birds. U.S. Fish and Wildlife, Spec. Sci. Rep. - Wildl. 191. 61pp.

Hill, E.F., and W.J. Fleming. 1982. Anticholinesterase poisoning of birds. Field Monitoring and Diagnosis of Acute Poisoning. Envir. Tox. and Chem. 1:27-38.

Hill E.F. 1988. Brain Cholinesterase activity of apparently normal wild birds. Journ. Wildl. Dis. 24(1): 51-61.

Hill, E.F. 1989. Divergent effects of postmorten ambient temperature on organophosphorous-and carbamate-inhibited brain cholinesterase activity in birds. Pest. Biochem. and phys. 33, 264-275.

Ludke, J.L., E.F. Hill, and M.P. Dieter. 1975. Cholinesterase (ChE) response and related mortality among birds fed ChE inhibators. Arch. Environ. Contam. Toxicol. 3:1-21.

Murphy, S.D. 1986. Toxic effects of pesticides in Toxicology: The Basic Science of Poisons. pp 519-581. Eds. C.D. Klaasen, M.O. Amdor, and J. Doull. McMallin Publishing Co. New York. Third ed. p.974

Smith, G.J. 1987. Pesticide use and toxicology in relation to wildlifeL: organophosphorous and carbamate compounds. U.S. Fish and Wildl. Serv. Spec. Resour. Rep. 170. pp171.

Tucker, R.K. and D.G. Crabtree. 1970. Handbook of toxicity of pesticides to wildlife. U.S. Fish and Wildl. Serv. Resour. Rep. 84. 131pp.

Windingstad, R. 1990. Personal Communication. U.S. Fish and Wildlife Service, National Wildlife Health Research Center - Madison, Wisconsin.