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SHORT COMMUNICATIONS

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DDE Poisoning in an Adult Bald Eagle

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ABSTRACT: A 12-year-old female bald eagle (*Haliaeetus leucocephalus*) was found in May 1993 on Santa Catalina Island, California (USA), in a debilitated condition, exhibiting ataxia and tremors; it died within hours. On necropsy, the bird was emaciated but had no evidence of disease or physical injury. Chemical analyses were negative for organophosphorus pesticides and lead poisoning. High concentrations of DDE (wet weight basis) were found in the brain (212 ppm), liver (838 ppm), and serum (53 ppm). Mobilization of DDE, from depleted fat deposits, probably resulted in the lethal concentration in the eagle's brain.

Key words: DDE, poisoning, organochlorine, bald eagle, *Haliaeetus leucocephalus*, mortality, California.

The organochlorine compound *p,p'*-DDE [2,2-bis(*p*-chlorophenyl)-1,1-dichloroethylene] (DDE), a metabolite of the agricultural pesticide DDT [1,1,1-trichloro-2,2-bis(*p*-chlorophenyl)ethane], has been closely associated with reproductive impairment in bald eagles (*Haliaeetus leucocephalus*) (Wiemeyer et al., 1993). After the 1972 ban on use of DDT in the United States, bald eagle populations recovered in most areas of the United States and Canada (Henny and Anthony, 1989), but bald eagle reproduction in some areas continues to be affected because of the long persistence of DDE in the environment (Anthony et al., 1993). While DDT was never used on Santa Catalina Island, DDE transported in the tissues of seabirds and marine mammals feeding in contaminated waters near mainland California is the likely pathway of DDE to the eagles (Garcelon et al., 1989).

There is little published evidence of potential lethal effects of DDE on post-embryonic bald eagles. Stickel et al. (1966) conducted experiments determining DDT toxicity in captive bald eagles, but no similar dose/response experiments have been conducted for DDE in bald eagles. In this paper we report on a wild adult bald eagle which probably died due to elevated levels of DDE in the brain.

On 28 May 1993, a banded 12-yr-old female bald eagle was recovered on the east end of Santa Catalina Island, California (USA) (33°18'N 118°18'W). On the day of recovery, two eagles were observed in a confrontation on the ground, with one bird on top of the other. The altercation stopped when the birds were approached, and one eagle flew away. The remaining eagle was recovered and examined by a veterinarian. The eagle had signs of a central nervous system disorder, including positional nystagmus, ataxia, body feathers held erect, and uncontrolled tremors. Muscle and fat on the sternum were depleted. The bird had no obvious injuries other than minor facial lacerations. Eleven ml of blood were drawn from the brachial vein; 2 ml were placed in a tube containing anticoagulant for analysis to detect organophosphorus pesticides and lead, and 9 ml were placed in a tube with no anticoagulant and centrifuged to obtain serum for analysis of DDT and metabolites and polychlorinated biphenyls (PCBs). The blood samples were kept frozen until laboratory analyses were performed.

The eagle was infused intravenously with 10 mg of the corticosteroid dexamethasone sodium phosphate (Victor Medical Co., Irvine, California) in 55 ml lactated Ringers Solution (LRS) over approximately 3 min. Approximately 3 hr later 150 mg of calcium ethylenediaminetetracetate (Abbott Laboratories, North Chicago, Illinois, USA), a chelating agent used to treat lead poisoning, was given intravenously with an additional 25 ml LRS. The bird was placed in a large cardboard box and kept in a warm dark area. The bird was found dead the following morning.

The carcass was stored at −10 C and later sent to the National Wildlife Health Center (NWHC) in Madison, Wisconsin (USA), where a necropsy was performed. Samples of lung, liver, kidney, spleen, and heart were fixed in 10% buffered formalin, and embedded in paraffin; 5 μm sections were stained with hematoxylin and eosin for light microscopic examination. Samples of lung and liver were inoculated onto 5% sheep blood agar plates (Difco Laboratories, Detroit, Michigan, USA) for bacterial isolation. Virus isolation from lung and liver tissue was attempted by the method of Docherty and Solta (1988). Brain, liver, and skeletal muscle were collected for chemical analysis and stored at −20 C.

Blood was analyzed for a variety of environmental contaminants. Analyses of serum for organophosphorus and carbamate pesticides and lead were conducted at the University of California Veterinary Diagnostic Laboratory (UCVDL) in San Bernardino, California. Cholinesterase (ChE) activity was tested using the methods described by Tor et al. (1994), with a detection limit of 0.1 μM/ml/minute. Blood was screened for 42 organophorphorus insecticides using methods described by Holstege et al. (1994). Blood lead concentration was examined using atomic absorption spectrometry with sensitivity of 0.05 to 0.06 ppm.

Post-mortem testing for brain ChE activity and lead concentration in the liver was conducted at the NWHC. Brain ChE activity was analyzed according to Ellman et al. (1961) and as later modified by Dieter and Ludke (1975) and Hill and Fleming (1982). The ChE activity was compared with normal published values (Hill, 1988) or control values determined by the NWHC (Smith et al., 1995). Liver lead residue was determined according to Boyer (1984), with a detection limit of 0.25 ppm, on a wet weight basis.

Analyses of tissues for DDTs and PCBs were conducted at the Geochemical and Environmental Research Group (Texas A & M, College Station, Texas, USA). Tissue samples were macerated in 100 ml methylene chloride and 30 to 50 g of sodium sulfate. The eluate was dried with sodium sulfate and purified using alumina to remove matrix interferences. A silica gel/alumina column and a Hewlett Packard Liquid Chromatography or Gel Permeation Chromatograph (ABC Laboratories, Columbia, Missouri, USA) were used if further purification was required. Chlorinated hydrocarbons concentrations were determined using high resolution capillary gas chromatography with electron capture detection (GC/ECD). The GC/ECD utilized 30-m × 0.25-mm fused silica capillary column with DB-5 and DB-17 bonded phase columns (J & W Scientific, Folsom, California). Detection limits for DDTs and PCBs were 0.0001 μg/g. Concentrations of all contaminants were reported in parts per million (ppm) on a wet weight basis. Total PCB values were reported as the sum of concentrations for all congeners detected. For comparative purposes, concentrations of contaminants in serum (collected in this study) were considered equal to plasma concentrations, and both serum and plasma concentrations were considered twice that of whole blood (Wiemeyer et al., 1989).

On necropsy, the bird had a complete lack of subcutaneous, abdominal, coronary, and perirenal fat. Fat within the stifle joints had undergone serious atrophy and the pectoral muscles were moderately atrophied. No evidence of disease or debilitating injury were found on gross or microscopic examination. No bacteria or viruses were isolated. Concentrations of DDE in the brain, liver, serum, and skeletal muscle were highly elevated (Table 1); PCBs also occurred in the tissues sampled (Table 1).

TABLE 1. Concentrations of contaminants (ppm of wet weight) found in the tissues of an adult female bald eagle recovered on Santa Catalina Island, California, May 1993.

Tissue	Contaminant concentration (ppm, wet weight)				Percent lipid	Percent water
	p,p'-DDE	p,p'-DDD	p,p'-DDT	PCBs[a]		
Serum	53.0	0.14	0.11	26.0	0.43	97.7
Brain	212.5	ND[b]	0.1	58.6	1.5	80.7
Liver	838.3	ND	0.1	294.0	2.1	71.4
Skeletal muscle	317.5	ND	0.02	3.9	3.0	57.9

[a] Total PCBs; sum of 45 PCB congeners.
[b] ND = not detected.

The ChE activity in brain and blood samples was not inhibited (brain = 14.7 μM/ml/min; blood = 0.11 μM/ml/min); thus there was no evidence for organophosphorus or carbamate poisoning. None of 42 organophosphorus insecticides included in the analysis were found at detectable levels and lead was not detected in blood or liver.

Of 737 bald eagles analyzed for contaminant residues between 1964 and 1981 (Reichel et al., 1969, 1984; Mulhern et al., 1970; Belisle et al., 1972; Cromartie et al., 1975; Prouty et al., 1977; Kaiser et al., 1980), only one had a brain DDE concentration higher (385 ppm) than the Santa Catalina Island eagle; cause of death for that bird was attributed to DDE poisoning with a possible contribution of PCBs (Belisle et al., 1972). American kestrels (*Falco sparverius*) experimentally fed diets containing DDE, died with brain concentrations of DDE ranging from 213 to 301 ppm (Porter and Wiemeyer, 1972; Henny and Meeker, 1981).

Concentration of DDE in the serum was an order of magnitude higher than concentrations previously reported in whole blood or plasma of adult bald eagles (Wiemeyer et al., 1989), and to our knowledge is the highest reported for an avian species. Concentrations of PCBs in the brain were lower than previously associated with mortality in birds (Sileo et al., 1977).

Concentrations of DDE in the brain has been linked to the amount of body fat present (Stickel et al., 1984). As body fat deposits harboring DDE were metabolized, the level of the DDE in the brain was increased (Van Velzen et al., 1972). The redistribution of DDE to the brain occurs because brain lipid levels remain stable even after lipid levels in other tissues have sharply declined (Bogan and Newton, 1977). Birds, including raptors, that have died after being experimentally fed diets containing DDE, typically have reduced body fat and increased levels of DDE in the brain (Porter and Wiemeyer, 1972; Stickel et al., 1984). While DDE concentrations in blood were not reported in those studies, they likely increased during lipid catabolism, as blood is the pathway by which contaminants are redistributed to lipid in the brain.

Six of seven bald eagles experimentally dosed with DDT had tremors prior to death (Chura and Stewart, 1967) which resembled tremors seen in the Santa Catalina eagle. Young et al. (1979) reported similar tremors in seabirds suspected of being poisoned by DDE.

Storage of DDE residues in body fat provides a physiologic mechanism for shunting immediate toxic effects. Disease, migration, reproduction, molt or cold weather, which tend to accelerate mobilization of body fat, may result in the death of birds long after exposure to the chemical, or at dosages that are not immediately lethal (Van Velzen et al., 1972). The brain DDE concentration in the bald eagle reported here exceeded all but one reported value for eagles obtained from the wild, and was within the range of values reported to cause death in experimentally dosed American kestrels. The physical symptoms exhibited by the bird prior to death were congruent with those shown in raptors and other birds experimentally dosed with lethal concentrations of DDE. As no other diseases or abnormalities were detected in the bird it is highly probable that this eagle died from DDE poisoning.

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