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Authors: Stendell, Rey C., Beyer, W. Nelson, and Stehn, Robert A.

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## Accumulation of Lead and Organochlorine Residues in Captive American Kestrels Fed Pine Voles from Apple Orchards

**Rey C. Stendell,** W. Nelson Beyer, and Robert A. Stehn, 1 U.S. Fish and Wildlife Service, Northern Prairie Wildlife Research Center, Jamestown, North Dakota 58402, USA; 2 U.S. Fish and Wildlife Service, Patuxent Wildlife Research Center, Laurel, Maryland 20708, USA; 3 U.S. Fish and Wildlife Service, Alaska Fish and Wildlife Research Center, Anchorage, Alaska 99503, USA

ABSTRACT: Pine voles (Microtus pinetorum) were collected from pesticide-treated orchards in New York (USA) and fed to three captive American kestrels (Falco sparverius) for 60 days to evaluate potential hazards from soil-borne persistent insecticides. Three control kestrels were fed uncontaminated laboratory mice (Mus musculus). The pine voles contained an average of 38 ppm lead, 48 ppm DDE and 1.2 ppm dieldrin (wet weight). The kestrels accumulated sublethal amounts of lead (1 ppm lead wet weight) in their livers. In contrast, DDE and dieldrin accumulated in the tissues and brains of kestrels to toxicologically significant concentrations. Control kestrels remained healthy and accumulated insignificant concentrations of the contaminants. The results indicated raptors may not be significantly at risk from lead residues in soil and biota following field applications of lead arsenate. However, sublethal effects may be expected from the level of contamination by organochlorine pesticides.

Key words: American kestrel, Falco sparverius, pesticide exposure, lead poisoning, organochlorine concentration, pine voles, Microtus pinetorum, experimental study.

Raptors are especially susceptible to poisoning from certain cumulative environmental contaminants. Organochlorine insecticides have been responsible for deaths of individual raptors and population declines of several species (Newton, 1979). Feeding trials with captive raptors have demonstrated lethal and sublethal effects at relatively low dietary concentrations (Stickel, 1973). We generally do not expect lead to biomagnify in food chains as one might typically associate with organochlorine insecticides. It appears, however, that elevated concentrations of lead in certain biota can increase exposure to organisms at a higher trophic level. This study reports on the potential hazards to raptors from lead and organochlorine residues in soil and biota resulting from the use of lead arsenate and organochlorine compounds as insecticides. Potential prey of raptors were collected from treated orchards and fed to captive raptors.

Apple orchards of the Hudson Valley of New York (USA) received heavy applications of lead arsenate from the early 1900's and organochlorine pesticides from the late 1940's (Johnson et al., 1976; Elfving et al., 1978). Elevated concentrations of DDT and dieldrin were reported in soil, earthworms, and robins (Turdus migratorius) (Johnson et al., 1976), and elevated concentrations of lead were reported in meadow voles (Microtus pennsylvanicus) and pine voles (Microtus pinetorum) (Elfving et al., 1978). Chemical analyses of raptors and ground-feeding insectivorous birds collected from 1982 to 1986 in New York show that organochlorine pesticides continued to poison birds years after the use of most of these chemicals has been discontinued (Stone and Okoniewski, 1988).

Pine voles were trapped from the Hudson Valley Region (41°44.9'N, 74°05.1'W) between November 1976 and January 1977, and were frozen and shipped to the Patuxent Wildlife Research Center (Laurel, Maryland 20708, USA) to be fed to captive American kestrels (Falco sparverius). Six 6-mo-old female American kestrels raised in captivity at Patuxent were placed in individual outdoor pens (1.7 ×  $1.8 \times 1.8$  m) equipped with a food tray, water pan and several perches. Three kestrels were fed a contaminated pine vole early in the morning each day for 60 days (7 December 1976 through 4 February 1977). Controls comprised three other kestrels kept under the same conditions as the treated birds but fed laboratory mice

(Mus musculus) of approximately the same weight as the pine voles. Laboratory mice were obtained as surplus animals from several research facilities in the Washington, D.C. area. These animals served as a staple diet for captive birds at the Patuxent Wildlife Research Center. All kestrels were fed an additional laboratory mouse, ad libitum, in the afternoon after the first vole or mouse had been consumed. Samples of the pine voles and control laboratory mice were saved for chemical analysis.

One kestrel on the vole diet died after 31 days. The other two kestrels on the vole diet and the three control kestrels were killed by euthansia with carbon dioxide after 60 days. Livers and tibiae were removed and sent to the Environmental Trace Substances Center (Route 3, Columbia, Missouri 65201, USA) to be analyzed for lead by atomic absorption spectrometry (Perkin-Elmer Model 1403 atomic absorption spectrophotometer. Norwalk. Connecticut 06859, USA). Brains and carcasses were analyzed by gas chromatography (Hewlett Packard 5840 gas chromatograph equipped with electron capture detector, Rockville, Maryland 20850, USA) for p,p'-DDE, p,p'-DDD, p,p'-DDT, dieldrin, heptachlor epoxide, mirex, endrin, oxychlordane, cis-chlordane and/or transnonachlor, cis-nonachlor, hexachlorobenzene, and polychlorinated biphenyls. Procedures followed those described by Cromartie et al. (1975). Concentrations were expressed as ppm, wet weight, with a lower limit of sensitivity of 0.1 ppm for carcasses and 0.05 ppm for brains. Eight pine voles and eight laboratory mice were eviscerated and analyzed for lead and organochlorine residues by the above methods. Fourteen composited samples of regurgitated pellets from the test kestrels and 12 from the control kestrels were analyzed for lead.

Pine voles contained  $38 \pm 6$  (SE) ppm lead,  $48 \pm 9$  ppm DDE,  $4.5 \pm 1.0$  ppm DDD,  $14.1 \pm 3.8$  ppm DDT,  $1.2 \pm 0.4$  ppm dieldrin,  $0.4 \pm 0.16$  ppm endrin, and  $0.14 \pm 0.00$  ppm PCB's. Control mice con-

tained  $0.06 \pm 0.01$  ppm lead and concentrations of organochlorine insecticides below detection limits. Regurgitated pellets from kestrels fed pine voles contained 130  $\pm$  15 ppm lead, and those from control birds contained 2.4  $\pm$  0.4 ppm lead.

The kestrel that died (on day 31) lost 27% of its body weight, although it continued to eat until its death. No gross effects were noted in this bird other than emaciation. The two other kestrels fed pine voles lost more weight than did the control kestrels (Table 1).

The lead residues detected in the pine voles were typical of the values reported in small mammals from other polluted sites, such as mining sites (Roberts and Johnson, 1978) and near major highways (Quarles et al., 1974; Clark, 1979). Only at heavily contaminated sites, such as near Kellogg, Idaho (Blus et al., 1987), are kestrels likely to be exposed to substantially higher concentrations of lead in their diets. However, the average of approximately 1 ppm lead detected in both bones and livers demonstrates that kestrels accumulated little of the ingested lead. A hepatic lead concentration of 1 ppm is considered elevated above normal levels, but well below the lethal range for eastern screech owls (Otus asio) (Beyer et al., 1988). Livers of these owls contained from 8 to 61 ppm (wet weight) when exposed to a dietary dosage that killed half of the birds. Moreover, Custer et al. (1984) did not find any lesions in kestrels fed chicks containing as much as 448 ppm lead for 60 days. Accordingly, it appears that kestrels are not likely to be killed by lead from feeding on contaminated small mammals from lead-contaminated orchards. When wild raptors have been poisoned by lead, it has probably been from lead shot ingested with their prey (Custer et al., 1984).

The concentrations of DDE in the carcasses of the three kestrels fed pine voles were higher than the average reported in 13 male kestrels chronically fed 10 ppm DDE (wet weight, Porter and Wiemeyer, 1972); females on the same diet laid eggs

	Residues in and	weights of captive	kestreis ted voie	es from appie orcha	irds or control mice for 60	
days.						

	Treated			
	Died at 31 days $(n = 1)$	Sacrificed at 60 days (n = 2)	Control $(n = 3)$	
Change in body weight (%)	-27	$-12.5 \pm 2.5$	$-3.6 \pm 3.9$	
Lipids/carcass weight (%)	2.5	$17.8 \pm 0.4$	$21.7 \pm 1.9$	
Lead (ppm wet wt)				
Liver	0.92	$1.05 \pm 0.05$	$0.06 \pm 0.00$	
Tibiae	0.70	$0.70 \pm 0.20$	$0.27~\pm~0.05$	
Dieldrin (ppm wet wt)				
Carcass	2.8	$5.9 \pm 0.6$	$0.18 \pm 0.02$	
Brain	2.2	$0.42 \pm 0.10$	$0.02 \pm 0.00$	
DDE (ppm wet wt)				
Carcass	147	$232 \pm 9$	$1.18 \pm 0.47$	
Brain	63	$10.4 \pm 0.6$	$0.04 \pm 0.02$	

with abnormally thin eggshells (Wiemeyer and Porter, 1970). However, the 63 ppm DDE in the brain of the kestrel that died in this study is below the 212 ppm and 301 ppm detected in brains of kestrels known to have died from DDE poisoning (Porter and Wiemeyer, 1972). Consequently, we consider the 63 ppm DDE to be a toxicologically significant, but sublethal, concentration. The 2.2 ppm dieldrin in the brain of the kestrel that died also should be considered significant, but by itself it was probably not lethal. Heinz and Johnson (1982) found an average concentration of 16 ppm dieldrin in brains of cowbirds that died from a chronic dietary exposure, and an average of 6.8 ppm in brains of cowbirds when they ceased feeding prior to death. None of the other organochlorine insecticides analyzed were detected at greater than trace concentrations in brains of any of the six kestrels. Concentrations of PCB's in brains were low also ( $\leq 1$  ppm).

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