

A RETROSPECTIVE STUDY OF POSTMORTEM FINDINGS IN RED-TAILED HAWKS

J. CHRISTIAN FRANSON, NANCY J. THOMAS, MILTON R. SMITH,
ALISON H. ROBBINS,¹ SCOTT NEWMAN² AND PAUL C. MCCARTIN³
*U.S. National Biological Service, National Wildlife Health Center, 6006 Schroeder Road,
Madison, WI 53711-6223 U.S.A.*

ABSTRACT.—We studied necropsy results from carcasses of 163 red-tailed hawks (*Buteo jamaicensis*) submitted to the National Wildlife Health Center from 1975 through 1992. The most frequent post-mortem finding was emaciation of unknown etiology, diagnosed in 33 (20%) carcasses. Proportionally more juveniles than adults were emaciated. Evidence of non-gunshot trauma, often suggestive of collision with vehicles or structures near roadways, was found in 29 (18%) birds. Of 25 (15%) toxicoses, 20 were attributed to agricultural pesticides, including famphur (4), fenthion (3), carbofuran (2), phosphamidon (2), endrin (1), and unidentified organophosphorus compounds (8). Lead and strychnine poisoning were diagnosed in two birds each, and selenium poisoning in one. Diseases, including aspergillosis, tuberculosis, pasteurellosis, and pox, were found in 21 (13%) hawks. Gunshot and electrocution were each diagnosed in six (4%) birds, one (0.6%) was trapped, miscellaneous conditions were found in 10 (6%), and no diagnosis could be determined for 32 (19%) of the carcasses.

KEY WORDS: *Buteo jamaicensis*; diseases; mortality; pesticides; poisoning; red-tailed hawk; trauma.

Un estudio retrospectivo de encuentros postmortem en *Buteo jamaicensis*

RESUMEN.—Estudiamos los resultados de la necropsia de restos de 163 individuos de la especie *Buteo jamaicensis* sometidos al National Wildlife Health Center desde 1975 hasta 1992. El encuentro post-mortem más frecuente fue adelgazamiento de etiología desconocida, diagnosticado en 33 (20%) restos. Proporcionalmente, más juveniles que adultos estaban adelgazados. Traumas no causados por armas de fuego, sino que por posibles colisiones con vehículos o estructuras cerca de las carreteras, fueron encontrados en 29 (18%) aves. De 25 (15%) individuos intoxicados, 20 fueron atribuidos a pesticidas de uso agrícola, incluyendo "famphur" (4), "fenthion" (3), "carbofuran" (2), "phosphamidon" (2), "endrin" (1) y compuestos organofosforados no identificados (8). Envenenamiento por plomo y estricnina fueron diagnosticados en dos aves, respectivamente, envenenamiento por selenio, sólo en una. Enfermedades, incluyendo aspergilosis, tuberculosis, pasteurellosis y viruela, fueron encontradas en 21 individuos (13%). Disparos y electrocución como causa de muerte fue diagnosticada en seis aves (4%), una fue atrapada, condiciones miscelaneas fueron encontradas en 10 (6%). No se pudo emitir un diagnóstico para 32 (19%) restos.

[Traducción de Ivan Lazo]

The red-tailed hawk (*Buteo jamaicensis*) is a frequent breeder in North America and the most abundant hawk in the United States and southern Canada during cold-weather months (Johnsgard 1990). Despite its wide range and abundance, little is known about factors that influence the health of the red-tailed hawk. Occasional case reports of diagnostic

findings, primarily infectious diseases and toxicoses, have been published but most of these accounts involve only one or several birds. Some of the diseases previously reported from individual sick and dead red-tailed hawks include avian pox (Halliwell 1972, Fitzner et al. 1985, Chubb 1987, Roszkopf et al. 1987), tuberculosis (Sykes 1982, Mollhoff 1983, Clark 1986), erysipelas (Pace et al. 1987), and oral capillariasis (Santiago et al. 1985). Poisonings documented in red-tailed hawks have often been the result of their exposure to pest-control chemicals. Henny et al. (1985, 1987) reported mortality in red-tailed hawks from exposure to famphur, an insecticide topically applied to livestock for parasite control, and Hooper et al. (1989) described intoxication

Present addresses: ¹Wildlife Clinic, Tufts University, School of Veterinary Medicine, 200 Westboro Road, North Grafton, MA 01536 U.S.A.; ²Wildlife Health Center, School of Veterinary Medicine, University of California, Davis, CA 95616 U.S.A.; ³New England Wildlife Center, 19 Fort Hill Street, Hingham, MA 02043 U.S.A.

Table 1. Postmortem findings in 163 red-tailed hawks.

CATEGORY	<i>N</i>	%
Emaciation	33	20
Undetermined ^a	32	19
Trauma	29	18
Toxicoses	25	15
Diseases	21	13
Miscellaneous ^b	10	6
Gunshot	6	4
Electrocution	6	4
Trapped	1	<1

^a Postmortem examination revealed no cause of death or significant findings.

^b Peritonitis, visceral gout, parasitism, hepatitis, endocarditis, and other nonspecific infections based on gross observations only, etiologies unknown.

of red-tailed hawks by organophosphorus pesticides applied to orchards. Cyclodiene organochlorine insecticides persistent in soil in urban and suburban areas have been implicated in recent poisonings of red-tailed hawks (Okoniewski and Novesky 1993).

In an effort to evaluate the scope of the causes of illness and death in red-tailed hawks, we studied diagnostic findings from 163 carcasses examined at the National Wildlife Health Center (NWHC) over nearly 20 yr. Because of the nonrandom nature of carcass collection and submission, this summary does not necessarily reflect the actual proportional distribution of causes of illness and mortality in the red-tailed hawk population as a whole. However, our findings identify some of the major factors that cause or contribute to the death of these hawks.

METHODS

We reviewed and summarized records of 163 red-tailed hawk carcasses that were submitted for cause of death determination to the NWHC, a U.S. Department of Interior facility that provides diagnostic services to natural resource managers throughout the United States and its territories. Most of these birds had been found dead in the field and were collected in 26 states from 1975 through 1992. Some birds were captured alive and later died or were euthanized because of the extent of their injuries or illness at the time of capture, but no birds undergoing extended rehabilitation were included in the review. A gross examination, including a description of wounds and abnormalities and an evaluation of overall body condition with respect to fat reserves and pectoral muscular development, was conducted on each carcass. Samples of organs were tested for a variety of microorganisms, parasites, and toxins as indicated by gross findings and field information provided by the collector. Tissues for histopathology were fixed in 10% buffered formalin, embedded in paraffin,

sectioned for light microscopy and stained with hematoxylin and eosin for routine examination, Ziehl-Neelsen acid-fast for mycobacteria, and/or Grocott silver for fungi. Bacteria were isolated by inoculation of tissues onto 5% sheep red-blood agar and eosin-methylene blue plates (DIFCO Laboratories, Detroit, MI U.S.A.), incubated at 37° C for 72 hr, and then characterized with the API-20E system (Analytab Products, Plainview, NY U.S.A.). Tissues for virus isolation attempts were processed according to Docherty and Slota (1988) and Senne (1989).

When agricultural pesticide poisoning was suspected by those who submitted the carcasses, or when the pathologist found stomach contents of animal remains consistent with possible secondary poisoning, brains were screened for cholinesterase activity. Cholinesterase assays were according to Ellman et al. (1961) and as later modified by Dieter and Ludke (1975) and Hill and Fleming (1982), including incubation (18 hr at 37°C) and retesting of samples with initially low enzyme activities. Cholinesterase inhibition was calculated by comparison with normal published values (Hill 1988) or control values determined by the NWHC (Smith et al. 1995). In most cases where inhibition of brain cholinesterase activity was noted, stomach contents were analyzed at the Patuxent Wildlife Research Center, Laurel, Maryland for 24 organophosphorus and six carbamate compounds according to Belisle and Swineford (1988) (Patuxent Analytical Control Facility standard operating procedure 0–25.00). Brains were analyzed for residues of chlorinated hydrocarbons by gas-liquid chromatography (Cromartie et al. 1975, Blus et al. 1989), liver lead residues were determined according to Boyer (1984), and methods for selenium analysis of liver followed Krynitsky (1987). Analysis of stomach contents for strychnine was according to Feldstein (1960). Animal material found in esophagi and stomachs of the hawks were described, but not identified to species. Chi-square and analysis of variance (Zar 1984) were used for age and gender comparisons of diagnostic findings.

RESULTS AND DISCUSSION

General Findings. Ninety-seven (60%) of the 163 red-tailed hawks were females, 59 (36%) were males, and seven (4%) were of undetermined gender. The age distribution was 83 (51%) juveniles, 74 (45%) adults, and 6 (4%) undetermined. The gender distribution by age, of the 155 birds for which both were determined, was 51 juvenile females, 31 juvenile males, 46 adult females, and 27 adult males. The most frequent state of origin was California ($N = 54$, 33%), followed by Wisconsin ($N = 27$, 16%) and Illinois ($N = 17$, 10%). Factors contributing to or directly causing the death of the bird were determined for 131 carcasses. The most frequent findings were emaciation, trauma, toxicoses, and diseases (Table 1). Less common causes of death included gunshot, electrocution, trapping, and a variety of miscellaneous conditions. We found no differences in the proportional distribution of gender or age

among the various categories of diagnostic findings. However, there was a significant ($\chi^2 = 5.26$, $df = 1$, $P = 0.02$) difference in the distribution of juveniles and adults when emaciated birds were compared with all other groups combined. Proportionally more juveniles than adults were emaciated.

Because a diagnosis of emaciation was applied subjectively, based on visual observation of fat reserves and muscle mass, we sought to verify this determination by comparing the mass of emaciated red-tailed hawks with the mass of birds diagnosed with gunshot, other trauma, and electrocution. The latter three groups were selected for comparison because we expected hawks that died such acute deaths would have body mass representative of the normal population. In fact, the mass of birds in this acute death group (Table 2) agreed closely with those reported by Dunning (1984). Regardless of gender or age, the average mass of emaciated birds was significantly ($F = 78.21$, $df = 1,46$, $P = 0.0001$) less than birds in other mortality categories (Table 2).

Emaciation. Diagnosed in 33 birds, emaciation was the most frequent finding, but examination of the carcasses failed to reveal its causes in individual birds or an explanation for the high rate of occurrence. These hawks may have died of starvation because of scarcity of prey or perhaps had unapparent injuries or diseases that led to their emaciated condition. However, chronic poisonings cannot be ruled out because tissues were not analyzed for the presence of toxins. As for the finding that more juveniles than adults suffered from emaciation, we speculate that juveniles, because of their more restricted diet (Craighead and Craighead 1956), may be less successful in obtaining food during periods of adverse environmental conditions, reduced prey populations, or dispersal into new territories.

Trauma. Trauma, exclusive of gunshot, was the second most commonly identified finding and was noted in 29 red-tailed hawk carcasses. Frequent traumatic injuries included fractures of long bones, vertebrae, and of the skull. These injuries and the evaluation of information, when provided, regarding the locations where the carcasses were found suggested that at least 10 of these birds probably collided with vehicles or structures near roadways. The fact that human observers spend a great deal of time on or near roadways may have contributed to the high frequency of traumatic injuries found among the hawks that were submitted.

Gunshot trauma was diagnosed in six birds. The

Table 2. Mass (g) of emaciated red-tailed hawks and of those that died acute deaths.

SEX	MEAN MASS (SD, N)	
	EMACIATION	ACUTE DEATH ^a
Male		
Adult	680 (94, 6) ^b	1031 (72, 4)
Juvenile	747 (118, 7)	1025 (167, 3)
Female		
Adult	1040 (85, 2)	1236 (173, 11)
Juvenile	777 (105, 12)	1192 (131, 9)

^a Trauma, gunshot, and electrocution.

^b Regardless of sex or age, weights of emaciated hawks were significantly ($F = 78.21$, $df = 1,46$, $P = 0.0001$) less than weights of birds in the acute death category.

appearance of the wounds and characteristics of projectiles, or portions thereof, that were recovered led pathologists to conclude that four of the hawks were shot with rifles and two with shotguns.

Toxicoses. Of the 25 poisoning cases in red-tailed hawks, 20 were attributed to agricultural pesticides. A single case of poisoning by endrin, a chlorinated hydrocarbon pesticide, was diagnosed in a bird of very poor body condition collected in 1982. This hawk was among 11 species of Falconiformes that died of endrin poisoning in or near orchards in central Washington (Blus et al. 1989). Organophosphorus or carbamate compounds were responsible for the remaining 19 agricultural chemical poisonings, collected from 1987 through 1992, based on brain cholinesterase activity or analytical identification of a pesticide in stomach contents. Carbofuran, phosphamidon, fenthion, and famphur were responsible for 11 of the mortalities (Table 3). Stomach contents in 10 of these hawks were of avian origin and the other contained the remains of a small mammal, consistent with secondary pesticide poisoning from consuming intoxicated prey (Stone et al. 1984, Henny et al. 1985, 1987, Hunt et al. 1992). Brain cholinesterase activity was inhibited by 77–97%, except for one case of carbofuran poisoning in which the initial activity was inhibited by only 39%, probably because of partial postmortem reactivation (Hill and Fleming 1982). After incubation and retesting, the cholinesterase activity of this sample increased to 113% of normal, a characteristic response to carbamate exposure (Smith et al. 1995) and the carbofuran concentration in stomach contents was

Table 3. Organophosphorus and carbamate pesticide poisonings in 11 red-tailed hawks.

PESTICIDE	STATE YEAR (N)	CONCEN- TRATION IN STOMACH CONTENTS ^a	BRAIN ChE ^b INHIBI- TION (%)
Carbofuran	MD, 1988 (1)	150	39
	MD, 1988 (1)	38	87
Phosphamidon	KS, 1990 (2)	3.5, 9.7	94, 97
Fenthion	IL, 1992 (2)	0.5, 2.8	83, 87
	MO, 1992 (1)	13	96
Famphur	UT, 1987 (1)	0.2	84
	UT, 1991 (2)	0.9, 0.5	78, 77
	WA, 1989 (1)	1.5	89

^a ppm, wet weight; stomach contents consisted of avian remains in all instances, except that parts of a small mammal were found in the first carbofuran (150 ppm) case listed.

^b Cholinesterase.

150 ppm wet weight. The relatively low level of brain cholinesterase inhibition, but high carbofuran residues, are in agreement with Greig-Smith (1991) who reported that a correlation between brain cholinesterase activity and carbamate pesticide residues is lacking.

In eight birds, brain cholinesterase activity was inhibited by 48–99%, without reversal after incubation of the sample, but chemical analysis of stomach contents was not done ($N = 5$) or failed to reveal the presence of any of the compounds included in the assay ($N = 3$). The magnitude of cholinesterase inhibition and the lack of reversal is consistent with exposure to an organophosphorus compound (Ludke et al. 1975, Hill and Fleming 1982, Smith et al. 1995), and we describe these as cases of suspected poisoning by an organophosphorus compound. In fact, three of these birds were found during mortality events where stomach contents from other red-tailed hawks were positive for famphur or phosphamidon. At least three possible explanations exist for the situation in which brain cholinesterase results and field circumstances point to organophosphate poisoning, but analytical results from stomach contents are negative: (1) Brain cholinesterase activity can remain inhibited several days after exposure to organophosphorus pesticides (Fleming and Bradbury 1981), and it is possible that by the time these birds died the concentration of the responsible chemical in stomach contents was below detectable limits as a result of degradation, absorption, or regurgitation;

(2) A loss of brain cholinesterase activity might result from advanced decomposition, although Prijono and Leighton (1991) found this enzyme to be quite stable for up to 8 d at 25°C; or, (3) The birds may have been poisoned by a compound not included in the testing procedure.

The earliest of the organophosphorus and carbamate poisonings in this group of birds occurred in 1987, well after secondary famphur poisoning was documented in a red-tailed hawk in 1982 (Henny et al. 1985). This may reflect nonrandom sample bias, expressed by an increased interest in these types of poisonings during the mid-1980s, and the fact that screening for brain cholinesterase activity was not common practice at the National Wildlife Health Center until about 1985. Our finding of a 12% (19 of 163) frequency of organophosphorus and carbamate poisonings in red-tailed hawks is similar to that (17%) reported in a 1975–88 survey in The Netherlands of eight raptor species (Lumeij et al. 1993), but higher than that found in some other studies. A 30-yr summary of over 4300 bald eagle (*Haliaeetus leucocephalus*) and golden eagle (*Aquila chrysaetos*) mortalities that began in the early 1960s indicated an overall frequency of anticholinesterase poisonings of about 3% (Franson et al. 1995). Most of those cases occurred after the early 1980s. Gremillion-Smith and Woolf (1993) tested brain cholinesterase activity of 105 raptor carcasses of five species collected in Illinois in 1985–87, and reported that 6% were anticholinesterase poisoning suspects.

Lead toxicosis was diagnosed in one red-tailed hawk collected in Illinois in 1981 and one found in California in 1985. Both birds were emaciated and the liver-lead concentrations were 4.3 and 10 ppm wet weight, respectively, but no ingested lead was found. In raptors, liver-lead residues of 2–4 ppm wet weight indicate unusual exposure to lead, while residues greater than 5 ppm wet weight are compatible with death when pathology consistent with lead poisoning is present (Franson 1996). The liver-lead concentration of 4.3 ppm wet weight in the first hawk reflects lead exposure in the toxic range but, because emaciation was the only lesion noted, this concentration is not itself conclusive for a definitive diagnosis of fatal lead poisoning. We can merely infer that this bird was affected by lead toxicosis. The liver-lead concentration of 10 ppm wet weight and the emaciated condition of the second hawk are compatible with a lead-poisoning diagnosis. This second case is complicated by the presence of pas-

teurellosis (*Pasteurella multocida*) and aspergillosis (*Aspergillus fumigatus*) in the bird's respiratory tract. Debilitation and immunosuppression, subsequent to lead exposure, may have facilitated the invasion of these organisms. Immunosuppressive effects of lead are well-known from studies of laboratory animals but less understood in wildlife (Franson 1986), although reduced numbers of immunologic cells in mallards (*Anas platyrhynchos*) exposed to lead has been reported (Rocke and Samuel 1991).

One hawk found in Minnesota in 1983 and another in California in 1990 died of strychnine poisoning, with concentrations of strychnine in stomach contents of 16 and 330 ppm wet weight, respectively. The death of a hawk collected in 1987, from an area in California with a previous history of selenium contamination (Ohlendorf et al. 1990, Schuler et al. 1990), was attributed to selenium toxicosis. The carcass was emaciated and had selenium residues of 19 ppm wet weight in its liver. Background residues of selenium in avian liver are generally less than about 5 ppm wet weight (White et al. 1980, Hutton 1981). Mallard ducklings that died after experimental selenium exposure had liver selenium residues of 7 and 18 ppm wet weight (Heinz et al. 1988). Eastern screech-owls (*Otus asio*) euthanized after receiving selenomethionine in their diet for more than 3 mo were thin and had a mean liver selenium concentration of 17 ppm wet weight (S.N. Wiemeyer pers. comm.).

Diseases. Evidence of infectious disease was the primary finding in 21 of the red-tailed hawk carcasses. Aspergillosis was the most frequent of these, occurring in nine birds. Gross observations were consistent with chronic disease, characterized by the presence of fungal mats, caseous nodules, or plaques within the respiratory system, and *Aspergillus fumigatus* was isolated from tissues. Aspergillosis is frequently associated with other conditions that weaken birds and predispose them to infection (O'Meara and Witter 1971). Concurrent diseases, two cases of pasteurellosis and one of mycobacteriosis (*Mycobacterium* sp.) were found in three of these hawks, but no other infectious agents or contributory causes were identified in the other six birds with aspergillosis. Aspergillosis is also common in birds undergoing rehabilitation, but four of the hawks were found dead in the field and the others were in captivity for less than 7 d.

Pasteurellosis was found in five of these red-tailed hawks, including two cases where it was the primary

finding, one case associated with lead poisoning and aspergillosis, and two concurrent with aspergillosis only. Of the two birds with primary pasteurellosis, one had a swollen foot and yellow fibrinous material in the pericardial sac, both of which yielded cultures of *P. multocida*. The only lesion seen in the second hawk was congestion of the lungs, but *P. multocida* was isolated from liver, lung, and heart. No lesions suggestive of pasteurellosis were seen in the lead-poisoned hawk, but *P. multocida* was isolated from air sacs. Gross lesions in the birds with concurrent aspergillosis/pasteurellosis included thickened air sacs and fibrinous adhesions within the body cavity, and *P. multocida* was isolated from the lungs and air sacs. All five carcasses were collected in California, one each in January and November of 1978, two in January of 1985, and one in January of 1988. Pasteurellosis, or avian cholera, is an endemic disease of wintering waterfowl in California, where yearly losses of ducks and geese numbering in the tens of thousands are commonplace (Botzler 1991). Pasteurellosis was reported from a red-tailed hawk collected in California in 1982 (Brogden and Rhoades 1983). Reports in raptors are infrequent, although they are often exposed to the organism when scavenging on carcasses of waterfowl that have died of avian cholera.

Mycobacteriosis (tuberculosis) was diagnosed in four hawks, including three in which tuberculosis was the primary finding. Tuberculosis is caused by bacteria of the genus *Mycobacterium*, including several species with a predilection for birds. The disease can be acquired by carnivorous birds that ingest the bacteria in tissues of infected prey, or may be spread among ground-feeding birds through fecal contamination of soil or food. The characteristic lesions of avian tuberculosis are nodules located along the gastrointestinal tract or in various organs. The affected red-tailed hawks were found in Wisconsin in 1976 and 1978, Illinois in 1977, and California in 1985. Typical nodular lesions were found in liver and lung, liver, spleen, and sternum; liver and spleen; and proventriculus, respectively. In all cases, characteristic acid-fast bacteria were seen on histopathology and in two cases tissues were sent to the National Veterinary Services Laboratories, Ames, Iowa, where *Mycobacterium avium* was isolated.

Avian pox, characterized by proliferative lesions on unfeathered skin of the feet and face, was noted in three birds. These hawks were found in Wisconsin in 1985, in Utah in 1987, and in Nebraska in 1988.

Intracytoplasmic inclusions consistent with pox were seen in tissues of all three hawks, and pox virus was isolated from lesions of one bird. Viral enteritis was diagnosed in one bird and protozoal enteritis in another by histologic examination of tissues, but attempts to further characterize these agents were unsuccessful. One hawk had splenitis and severe pneumonia, with consolidation of part of one lung and thickening of the air sacs. *Klebsiella pneumoniae* was isolated from the spleen and *Pseudomonas* sp. was isolated from the spleen, liver, and air sac.

Other Diagnoses. Electrocution was diagnosed in six carcasses with burn marks on feathers or skin, four of which were found in close proximity to utility lines. One red-tailed hawk had injuries to a foot and wing as the result of having been caught in a leg-hold trap. Miscellaneous conditions ($N = 10$) of uncertain etiology included visceral gout (2), peritonitis (2), verminous enteritis (2), pneumonia (1), sinus abscess (1), and air sacculitis (1). These diagnoses were based on gross observations only. Histopathology revealed hepatitis of unknown origin in one bird.

Summary. Because of biases inherent in the non-random collection and submission of carcasses, the true relative significance of these causes of mortality on red-tailed hawk populations cannot be determined. However, these cumulative findings provide baseline data on a variety of factors that cause mortality in red-tailed hawks, against which future diagnostic surveillance can be compared. Of the 131 carcasses for which diagnoses were determined, 39 (30%) died as a result of anthropogenic causes (toxicooses, gunshot, electrocution, and trapping). In addition, a portion of the 29 trauma cases were apparently the result of collisions with man-made structures. Steps can be taken to reduce these mortalities by providing education to increase public awareness of the impact of human activities on red-tailed hawks. Future research might be designed to identify significant temporal and geographic patterns of anthropogenic causes of mortality in red-tailed hawks, monitor pesticide poisonings, identify causes of undetermined mortality and emaciation, and to evaluate population effects of all mortality factors.

ACKNOWLEDGMENTS

We thank the many field biologists who submitted these carcasses, members of the Resource Health Team at the National Wildlife Health Center who consulted with field personnel, and those who conducted necropsies, including

C. Brand, P. Gullett, S. Kerr, J. Langenberg, L. Locke, H. McAllister, C. Meteyer, T. Roffe, S. Schmeling, L. Sileo, R. Stroud, and P. Whiteley. Laboratory support was provided by B. Campbell, R. Cole, and B. Tuggle for parasitology, R. Duncan for microbiology, and D. Docherty for virology. Chemists at the Patuxent Wildlife Research Center conducted pesticide and selenium analyses; staff at the Wisconsin Central Animal Health Laboratory provided additional toxicology support; G. Colgrove, E. Himes, and C. Thoen of the National Veterinary Services Laboratories identified mycobacteria; and M. Samuel and D. Xiang consulted on statistical treatment of data. L. Blus, L. Locke, C. Preston, and S. Wiemeyer provided helpful comments on the manuscript.

LITERATURE CITED

- BELISLE, A.A. AND D.M. SWINEFORD. 1988. Simple, specific analysis of organophosphorus and carbamate pesticides in sediments using column extraction and gas chromatography. *Environ. Toxicol. Chem.* 7:749-752.
- BLUS, L.J., C.J. HENNY AND R.A. GROVE. 1989. Rise and fall of endrin usage in Washington state fruit orchards: effects on wildlife. *Environ. Pollut.* 60:331-349.
- BOTZLER, R.G. 1991. Epizootiology of avian cholera in wildfowl. *J. Wildl. Dis.* 27:367-395.
- BOYER, K.W. 1984. Metals and other elements at trace levels in foods. Pages 444-476 in S. Williams [Ed.], *Official methods of analysis of the Association of Official Analytical Chemists*, 14th edition. Assoc. Official Analytical Chemists, Inc., Arlington, VA U.S.A.
- BROGDEN, K.A. AND K.R. RHOADES. 1983. Prevalence of serologic types of *Pasteurella multocida* from 57 species of birds and mammals in the United States. *J. Wildl. Dis.* 19:315-320.
- CHUBB, K. 1987. Avian pox in a red-tailed hawk. *Ont. Bird Banding* 19:35.
- CLARK, F.D. 1986. Mycobacteriosis in a red-tailed hawk (*Buteo jamaicensis*). *Southwest. Vet.* 37:200-201.
- CRAIGHEAD, J.J. AND F.C. CRAIGHEAD, JR. 1956. *Hawks, owls and wildlife*. Stackpole Co., Harrisburg, PA U.S.A.
- CROMARTIE, E., W.L. REICHEL, L.N. LOCKE, A.A. BELISLE, T.E. KAISER, T.G. LAMONT, B.M. MULHERN, R.M. PROUTY AND D.M. SWINEFORD. 1975. Residues of organochlorine pesticides and polychlorinated biphenyls and autopsy data for bald eagles, 1971-1972. *Pestic. Monit. J.* 9:11-14.
- DIETER, M.P. AND J.L. LUDKE. 1975. Studies on combined effects of organophosphates and heavy metals in birds. I. Plasma and brain cholinesterase in coturnix quail fed methyl mercury and orally dosed with parathion. *Bull. Environ. Contam. Toxicol.* 13:257-262.
- DOCHERTY, D.E. AND P.G. SLOTA. 1988. Use of muscovy duck embryo fibroblasts for the isolation of viruses from wild birds. *J. Tiss. Cult. Meth.* 11:165-170.
- DUNNING, J.B., JR. 1984. Body weights of 686 species

- of North American birds. *West. Bird Banding Assoc. Monogr. No. 1.*
- ELLMAN, G.L., K.D. COURTNEY, V. ANDRES, JR. AND R.M. FEATHERSTONE. 1961. A new and rapid colorimetric determination of acetylcholinesterase activity. *Biochem. Pharmacol.* 7:88-95.
- FELDSTEIN, M. 1960. Strychnine. Pages 498-499 in C.P. Stewart and A. Stolman [EDS.], *Toxicology—mechanisms and analytical methods. Vol. I.* Academic Press, New York, NY U.S.A.
- FITZNER, R.E., R.A. MILLER, C.A. PIERCE AND S.E. ROWE. 1985. Avian pox in a red-tailed hawk (*Buteo jamaicensis*). *J. Wildl. Dis.* 21:298-301.
- FLEMING, W.J. AND S.P. BRADBURY. 1981. Recovery of cholinesterase activity in mallard ducklings administered organophosphorus pesticides. *J. Toxicol. Environ. Health* 8:885-897.
- FRANSON, J.C. 1986. Immunosuppressive effects of lead. Pages 106-109 in J.S. Feierabend and A.B. Russell [EDS.], *Lead poisoning in wild waterfowl—a workshop.* Natl. Wildl. Fed., Washington, DC U.S.A.
- . 1996. Interpretation of tissue lead residues in birds other than waterfowl. In W.N. Beyer, G.H. Heinz and A.W. Redmon [EDS.], *Environmental contaminants in wildlife: interpreting tissue concentrations.* Lewis Publishers, Boca Raton, FL U.S.A. In Press.
- , L. SILEO AND N.J. THOMAS. 1995. Causes of eagle deaths. Page 68 in E.T. LaRoe, G.S. Farris, C.E. Puckett, P.D. Doran and M.J. Mac [EDS.], *Our living resources: a report to the nation on the distribution, abundance, and health of U.S. plants, animals, and ecosystems.* Natl. Biol. Serv., Washington, DC U.S.A.
- GREIG-SMITH, P.W. 1991. Use of cholinesterase measurements in surveillance of wildlife poisoning in farmland. Pages 127-150 in P. Mineau [ED.], *Cholinesterase-inhibiting insecticides—their impact on wildlife and the environment. Vol. 2,* Elsevier, Amsterdam, The Netherlands.
- GREMILLION-SMITH, C. AND A. WOOLF. 1993. Screening for anticholinesterase pesticide poisoning in Illinois raptors. *Trans. Ill. State Acad. Sci.* 86:63-69.
- HALLIWELL, W.H. 1972. Avian pox in an immature red-tailed hawk. *J. Wildl. Dis.* 8:104-105.
- HEINZ, G.H., D.J. HOFFMAN AND L.G. GOLD. 1988. Toxicity of organic and inorganic selenium to mallard ducklings. *Arch. Environ. Contam. Toxicol.* 17:561-568.
- HENNY, C.J., L.J. BLUS, E.J. KOLBE AND R.E. FITZNER. 1985. Organophosphate insecticide (famphur) topically applied to cattle kills magpies and hawks. *J. Wildl. Manage.* 49:648-658.
- , E.J. KOLBE, E.F. HILL AND L.J. BLUS. 1987. Case histories of bald eagles and other raptors killed by organophosphorus insecticides topically applied to livestock. *J. Wildl. Dis.* 23:292-295.
- HILL, E.F. 1988. Brain cholinesterase activity of apparently normal wild birds. *J. Wildl. Dis.* 24:51-61.
- AND W.J. FLEMING. 1982. Anticholinesterase poisoning of birds: field monitoring and diagnosis of acute poisoning. *Environ. Toxicol. Chem.* 1:27-38.
- HOOPER, M.J., P.J. DETRICH, C.P. WEISSKOPF AND B.W. WILSON. 1989. Organophosphorus insecticide exposure in hawks inhabiting orchards during winter dormant-spraying. *Bull. Environ. Contam. Toxicol.* 42: 651-659.
- HUNT, K.A., D.M. BIRD, P. MINEAU AND L. SHUTT. 1992. Selective predation of organophosphate-exposed prey by American kestrels. *Anim. Behav.* 43:971-976.
- HUTTON, M. 1981. Accumulation of heavy metals and selenium in three seabird species from the United Kingdom. *Environ. Pollut.* 26A:129-145.
- JOHNSGARD, P.A. 1990. *Hawks, eagles, and falcons of North America.* Smithsonian Inst. Press, Washington, DC U.S.A.
- KRYNITSKY, A.J. 1987. Preparation of biological tissue for determination of arsenic and selenium by graphite furnace atomic absorption spectrometry. *Anal. Chem.* 59:1884-1886.
- LUDKE, J.L., E.F. HILL AND M.P. DIETER. 1975. Cholinesterase (ChE) response and related mortality among birds fed ChE inhibitors. *Arch. Environ. Contam. Toxicol.* 3:1-21.
- LUMEIJ, J.T., I. WESTERHOF, T. SMIT AND T.J. SPIERENBURG. 1993. Diagnosis and treatment of poisoning in raptors from the Netherlands: clinical case reports and review of 2,750 postmortem cases, 1975-1988. Pages 233-238 in P.T. Redig, J.E. Cooper, J.D. Remple, D.B. Hunter and T. Hahn [EDS.], *Raptor biomedicine.* Univ. Minnesota Press, Minneapolis, MN U.S.A.
- MOLLHOFF, W.J. 1983. Avian tuberculosis in a red-tailed hawk. *Nebr. Bird Rev.* 51:92-93.
- OHLENDORF, H.M., R.L. HOTHEM, C.M. BUNCK AND K.C. MAROIS. 1990. Bioaccumulation of selenium in birds at Kesterson Reservoir, California. *Arch. Environ. Contam. Toxicol.* 19:495-507.
- OKONIEWSKI, J.C. AND E. NOVESKY. 1993. Bird poisonings with cyclodienes in suburbia: links to historic use on turf. *J. Wildl. Manage.* 57:630-639.
- O'MEARA, D.C. AND J.F. WITTER. 1971. Aspergillosis. Pages 153-162 in J.W. Davis, R.C. Anderson, L. Karstad and D.O. Trainer [EDS.], *Infectious and parasitic diseases of wild birds.* Iowa State Univ. Press, Ames, IA U.S.A.
- PACE, L.W., M.M. CHENGAPPA, S. GREER AND C. ALDERSON. 1987. Isolation of *Erysipelothrix rhusiopathiae* from a red-tailed hawk (*Buteo jamaicensis*) with a concurrent pox virus infection. *J. Wildl. Dis.* 23:671-673.
- PRIJONO, W.B. AND F.A. LEIGHTON. 1991. Parallel measurement of brain acetylcholinesterase and the muscarinic cholinergic receptor in the diagnosis of acute,

- lethal poisoning by anti-cholinesterase pesticides. *J. Wildl. Dis.* 27:110-115.
- ROCKE, T.E. AND M.D. SAMUEL. 1991. Effects of lead shot ingestion on selected cells of the mallard immune system. *J. Wildl. Dis.* 27:1-9.
- ROSSKOPF, W.J., JR., D. VAN DE WATER AND E.B. HOWARD. 1987. Response to autogenous vaccination and recovery by a red-tailed hawk (*Buteo jamaicensis*) from avian pox virus infection. *Compan. Anim. Pract.* 1:39-42.
- SANTIAGO, C., P.A. MILLS AND C.E. KIRKPATRICK. 1985. Oral capillariasis in a red-tailed hawk: treatment with fenbendazole. *J. Amer. Vet. Med. Assoc.* 187:1205-1206.
- SCHULER, C.A., R.G. ANTHONY AND H.M. OHLENDORF. 1990. Selenium in wetlands and waterfowl foods at Kesterson Reservoir, California, 1984. *Arch. Environ. Contam. Toxicol.* 19:845-853.
- SENNE, D.A. 1989. Virus propagation in embryonating eggs. Pages 176-181 in H.G. Purchase, L.H. Arp, C.H. Domermuth and J.E. Pearson [EDS.], A laboratory manual for the isolation and identification of avian pathogens. Kendall/Hunt Publ. Co., Dubuque, IA U.S.A.
- SMITH, M.R., N.J. THOMAS AND C. HULSE. 1995. Application of brain cholinesterase reactivation to differentiate between organophosphorus and carbamate pesticide exposure in wild birds. *J. Wildl. Dis.* 31:263-267.
- STONE, W.B., S.R. OVERMANN AND J.C. OKONIEWSKI. 1984. Intentional poisoning of birds with parathion. *Condor* 86:333-336.
- SYKES, G.P. 1982. Tuberculosis in a red-tailed hawk (*Buteo jamaicensis*). *J. Wildl. Dis.* 18:495-499.
- WHITE, D.A., K.A. KING AND R.M. PROUTY. 1980. Significance of organochlorine and heavy metal residues in wintering shorebirds at Corpus Christi, Texas, 1976-77. *Pestic. Monit. J.* 14:58-63.
- ZAR, J.H. 1984. Biostatistical analysis. Prentice Hall, Englewood Cliffs, NJ U.S.A.

Received 31 May 1995; accepted 21 September 1995