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EFFECTS OF PESTICIDES ON OWLS IN NORTH AMERICA

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ABSTRACT.—A literature review of the effects of pesticides on owls in North America showed that relatively few studies have been undertaken. Owls used in experiments seem as sensitive to organochlorine pesticides (OCs) as other birds of prey, but wild owls experienced few serious problems, primarily because they were exposed to lower residues in their predominately mammalian or invertebrate prey. For example, the great horned owl (*Bubo virginianus*) and the common barn-owl (*Tyto alba*) neither experienced marked changes in mortality or recruitment rates nor was there any evidence of population decreases even during the maximum period of OC pesticide use. Also, eggshell thinning was not a widespread problem. There were adverse effects on individual owls including verified records of 74 owls of six species that died from secondary or tertiary poisoning related to strychnine, organochlorines, anticholinesterases (antiChEs) and anticoagulants in 16 states within the U.S. and one province in Canada. Most of the pesticide-related deaths occurred during the 1980s, although this probably does not represent a true temporal distribution. Verified mortalities of owls probably represent a small fraction of the actual number that died from pesticides. Incidence of mortality seems biased geographically toward areas such as New York that have active ecotoxicological programs. Burrowing owl (*Speotyto cunicularia*) populations currently are decreasing throughout much of the range in the U.S. and Canada. Studies in Canada indicate that antiChE pesticides, particularly carbofuran, were responsible for the declines there.

KEY WORDS: *Owls; pesticides; mortality; secondary poisoning; reproductive effects; population decline; North America.*

Efectos de pesticidas sobre búhos en Norteamérica

RESUMEN.—Una revisión de la literatura sobre efectos de pesticidas en búhos de Norteamérica, muestra que relativamente pocos estudios han sido hechos. Búhos usados en experimentos parecen tan sensibles a pesticidas organoclorados (OC) como otras aves rapaces, en cambio búhos silvestres han sostenido pocos problemas serios, primariamente porque ellos estuvieron expuestos a residuos menores en sus presas predominantes de mamíferos e invertebrados. Por ejemplo, ni *Bubo virginianus* y tampoco *Tyto alba* experimentaron cambios marcados en la tasa de mortalidad o reclutamiento o hubo alguna evidencia de disminución poblacional durante el período de máximo uso de los pesticidas OC. El adelgazamiento de la cubierta calcárea de los huevos tampoco fue un problema importante. Hubo efectos adversos sobre individuos, que incluyeron registros verificados de 74 búhos de seis especies, que murieron por envenenamiento secundario y terciario relacionado con stricnina, organoclorados, anticolinesterasas (antiChEs) y anticoagulantes, en 16 estados de los Estados Unidos y una provincia en Canadá. La mayoría de las muertes relacionadas con pesticidas ocurrieron durante la década de 1980, aunque probablemente esto no representa una distribución temporal real. Las mortalidades verificadas de búhos probablemente representan una pequeña fracción del actual número que muere por pesticidas. La incidencia de mortalidad parece sesgada geográficamente hacia áreas tales como New York que tienen programas ecotoxicológicos activos. Actualmente las poblaciones de *Speotyto cunicularia* han decrecido en la mayor parte de su rango de distribución en los Estados Unidos y Canadá. Estudios realizados en Canadá indican que pesticidas antiChEs, particularmente carbonofurano, fue responsable de estas declinaciones.

[Traducción de Ivan Lazo]

Owls occupy upper trophic levels in various food chains. Therefore, one would expect that these birds would be as susceptible to effects of lipid-soluble organochlorine (OC) pesticides as other birds of prey. Nevertheless, there have been few intensive studies and no reviews of the effects of pesticides on owls in North America. In this review, I summarize and discuss effects of pesticides on

North American owls and briefly compare these findings with experimental studies. All residues listed are on a wet-weight basis unless otherwise stated.

EFFECTS ON REPRODUCTION

Organochlorines. Few data are available that relate effects of pesticides on reproduction of wild (free-ranging) owls. Regarding eggshell thickness, Hickey and Anderson (1968) found no shell thinning in a stationary population of great horned owls (*Bubo virginianus*) in California from 1948–50; they considered that ≥18% thinning could affect productivity immediately and could result in population declines if it occurred over several years. A more thorough study of shell thickness of great horned owl eggs, conducted in six areas in the U.S. and Canada from 1947–68, revealed no significant difference from pre-1947 norms except for 11–19% thinning in Florida (Anderson and Hickey 1972). Other studies of eggshell thickness and DDE residues in great horned owls reported 10% shell thinning associated with 5 μg/g in three eggs from New York in 1972 (Lincer and Clark 1978), no shell thinning associated with 0.7 μg/g in three eggs from Montana in 1967 (Seidensticker and Reynolds 1971) and 1.6% shell thinning associated with 3 μg/g in a study from Ohio in 1974–77 (Springer 1980).

Shell thinning of 7.4% in seven eggs of the western screech-owl (*Otus kennicottii*) collected in Oregon from 1978–81 was significantly correlated with residues of DDE in eggs; but eggshell thicknesses (N = 4–6) of the burrowing owl (*Speotyto cunicularia*), short-eared owl (*Asio flammeus*) and northern saw-whet owl (*Aegolius acadicus*) essentially were unchanged from the pre-1947 norms (Henny et al. 1984). Coincidentally, there was no evidence of adverse effects of OCs on reproductive success of five species of owls in Oregon and residues were relatively low (Henny et al. 1984). A study of great horned owls in Montana showed that low levels of DDE and other OCs had little apparent effect on the number of young fledged per nest (Seidensticker and Reynolds 1971). Klaas and Swineford (1976) found no effect of OCs on eggshell thickness or reproduction of the eastern screech-owl (*Otus asio*) in Ohio in the 1970s. A breeding study of common barn-owls (*Tyto alba*) on Chesapeake Bay, Maryland found that OC residues in eggs were associated with a slight change in eggshell thickness (5.5% thinning) but little or no decrease in fledged young (Klaas et al. 1978).

Eggs of the great horned owl, snowy owl (*Nyctea scandiaca*), burrowing owl, long-eared owl (*Asio otus*) and short-eared owl collected from Canada in 1965–72 and the great gray owl (*Strix nebulosa*) collected in Minnesota in 1980–88 were analyzed for residues of OCs (Noble and Elliott 1990). Except for elevated residues of DDE (3–16 μg/g) in a few eggs of the great horned owl, short-eared owl, long-eared owl and snowy owl, residues of OCs were low; however, there were no measurements of reproductive success or eggshell thinning (Noble and Elliott 1990).

Experiments with eastern screech-owls receiving 2.8 μg/g of DDE in the diet for two breeding seasons demonstrated no significant eggshell thinning the first breeding season but 13% thinning the second year (McLane and Hall 1972). Common barn-owls given diets containing 2.8 μg/g DDE alone or 2.8 μg/g of DDE and 0.6 μg/g of dieldrin experienced eggshell thinning of 20–28%, decreases of 22–76% in young fledged per nest and mortality of several adults on dosage (Mendenhall et al. 1983). Common barn-owls receiving just 0.5 μg/g of dieldrin in the diet experienced no effects on number of young fledged, a slight decrease in eggshell thickness and deaths of several adults. Effects of DDE were similar each year even though residues in eggs averaged 12 μg/g the first year and about 40 μg/g the second year. On the 0.5 μg/g dieldrin diet, dieldrin residues in eggs of common barn-owls doubled by the second year (4–8 μg/g), but there was still no effect on the number of young fledged (Mendenhall et al. 1983).

Experimental eastern screech-owls given dicolfol (Kelthone®)-contaminated diets (10 mg/kg containing either 0 or 3.4% DDT-related compounds) exhibited no effects on productivity but mean eggshell thickness was reduced 8–11% (Wiemeyer et al. 1989). In contrast, eastern screech-owls given a diet containing 0.75 mg/kg endrin produced 43% fewer fledged owlets than controls and there was no eggshell thinning (Fleming et al. 1982).

Anticholinesterases. James and Fox (1987) reported that carbofuran, but not carbaryl, was detrimental to productivity of burrowing owls in Canada. Nest success (≥1 young observed above ground) was reduced from 74% when there was no insecticide exposure within 400 m of nesting burrows to 50% and 38% when carbofuran was sprayed within 400 and 50 m, respectively. Coinci-

Table 1. Mortality of owls from organochlorine pesticides in North America.

PESTICIDE	YEAR	AREA	NUMBER OF DEATHS	SOURCE[a]
		Great Horned Owl (*Bubo virginianus*)		
Chlordane	1980	Oregon	1	1
	1986–90[b]	New York and Maryland	9	2
	1982–86	New York	4	3
Dieldrin	1986–90	New York and Maryland	2	2
	1982–86	New York	1	3
	1974–81	Illinois	1	4
	1985–89	Colorado	3	5
Endrin	1981	Washington	1	6
Dieldrin + Chlordane	1982–86	New York	2	3
Several[c]	1981	New York	1	7
Several[c]	1982–86	New York	6	3
Aldrin[d]	1968 or 1970	Texas	1	8
		Common Barn-Owl (*Tyto alba*)		
Chlordane	1986–90	New York and Maryland	1	2
Endrin	1981–83	Washington	4	6
Dieldrin	1982–86	New York	1	3
		Eastern Screech-Owl (*Otus asio*)		
Chlordane	1986–90	New York and Maryland	3	2
Dieldrin	1982–86	New York	1	3
Several[c]	1987	Ontario	1	9
		Species Not Listed		
Several[c]	1980	Ontario	1	9

[a] 1 = Blus et al. (1983); 2 = Okoniewski and Novesky (1993); 3 = Stone and Okoniewski (1988); 4 = Havera and Duzan (1986); 5 = Fordham and Reagan (1993); 6 = Blus et al. (1989); 7 = Stone and Okoniewski (1983); 8 = Flickinger and King (1972); 9 = Frank and Braun (1990).

[b] Brains of 2 additional great horned owls, 1 common barn-owl, 1 eastern screech-owl and 1 barred owl (*Strix varia*) contained near-lethal levels of chlordane components (heptachlor epoxide and oxychlordane).

[c] Deaths apparently related to combined effects of DDE, dieldrin, heptachlor epoxide and oxychlordane as well as polychlorinated biphenyls.

[d] Aldrin rapidly breaks down to dieldrin after application; therefore, animals dying as a result of aldrin use have lethal levels of dieldrin in their brains. The owl from Texas died in an area where aldrin was used.

dentally, the average maximum number of young observed per nest declined from 3.8 to 1.8 (James and Fox 1987).

PESTICIDE-RELATED MORTALITY

Organochlorines. Food chain effects (biomagnification) are important in accumulation of OC pesticides in tissues of owls. Death in the field is verified by comparing diagnostic lethal residues of OCs in tissues (preferably the brain, but the liver is sometimes used) of experimental animals exposed to one or more pesticides (Beyer et al. 1996). OCs are stored in lipids throughout the body. Residues of OCs are mobilized into the blood along with lipids during periods of food shortage or other stress and are then transported to the brain or other compartments where they may induce mortality or serious sublethal effects (Van Velzen et al. 1972).

Deaths of 44 owls, including 32 great horned owls, 6 common barn-owls, 5 eastern screech-owls and one unidentified species, were attributed to OC pesticides (Table 1). Chlordane was involved in most of the deaths acting either as the sole toxicant (N = 18), combined with dieldrin (N = 2) or with a mixture of other OCs (N = 9). Nine deaths from dieldrin, five from endrin and one from aldrin were recorded from seven states and one province in Canada, but most occurred in New York. I found no records of organochlorine pesti-

cide-related deaths of owls in Mexico. After spraying of DDT for Dutch elm disease in Michigan, Bernard and Wallace (1967) reported that an eastern screech-owl that died in tremors had no residues of total DDT in its brain even though other tissues contained high concentrations. I suspect that this owl probably died from DDT poisoning, as did many other birds in the area and that absence of residues in its brain was probably due to an analytical error. Ferguson (1964) reported that a barred owl (*Strix varia*) and a number of other birds apparently died in 1960 from heptachlor after this compound was applied to a pasture in Mississippi, but tissues were not analyzed for residues.

Anticholinesterases. The relatively short-lived anticholinesterase (antiChE) pesticides include organophosphorus (OP) and carbamate compounds. These pesticides are not lipophilic and have a short half-life in the body as well as in the environment. The effects of these pesticides on the central nervous system occur shortly after exposure, particularly for carbamates. Death in the field is verified by assaying the brain for cholinesterase (ChE) activity; inhibition of ≥50% compared to control birds is used as presumptive evidence that death was related to an antiChE compound (Hill and Fleming 1982). Analysis of the contents of the upper gastrointestinal tract or gizzard is used to verify the antiChE compound involved, although residues are not always detected, particularly when death occurs several days after exposure.

Five species of owls (18 individuals) and one unlisted species (one individual) from 11 states were reported killed by antiChE compounds including the OPs phorate, famphur and fenthion; the carbamate carbofuran and an unknown compound (Table 2). The record from Oregon (Henny et al. 1987) was unique because of tertiary poisoning when a great horned owl died after consuming a red-tailed hawk (*Buteo jamaicensis*) that ingested a black-billed magpie (*Pica pica*) that was probably exposed to famphur poured on the backs of cattle. Unraveling this chain of events was only possible because famphur is very toxic and the birds died or were killed shortly after exposure. The famphur study (Henny et al. 1987) was instigated because of a report that a captive great horned owl died after it was mistakenly fed black-billed magpies that were apparently killed by famphur. Most records of mortality I found were from unpublished reports with incomplete data relating to ChE assays or residue analyses.

Table 2. Mortality of owls from anticholinesterase pesticides in North America.

PESTICIDE	YEAR	AREA	NUMBER OF DEATHS	SOURCE[a]
Great Horned Owl (*Bubo virginianus*)				
Phorate	1982	South Dakota	4	1, 2
Famphur	1985	Oregon	1	3
Carbofuran	1987	Virginia	1	4
	1989	Delaware	1	2, 4
	1990	Iowa	1	4
Fenthion	1993	Missouri	1	2
	1996	Washington	1	5
Unknown[b]	1986–87	Illinois	2	6
	1991	Utah	1	2
Common Barn-Owl (*Tyto alba*)				
Phorate	1989	Wisconsin	1	1
Short-eared Owl (*Asio flammeus*)				
Carbofuran	1982	Utah	1	4
Fenthion	1989	Washington	1	5
Eastern Screech-Owl (*Otus asio*)				
Unknown[b]	1986–87	Illinois	1	6
Snowy Owl (*Nyctea scandiaca*)				
Fenthion	1988	Illinois	1	5
Species Not Listed				
Carbofuran	1990	New Mexico	1	4

[a] 1 = J. Spinks, Jr., U.S. Fish and Wildlife Service (unpubl. data); 2 = Franson and Little (1996); 3 = Henny et al. (1987); 4 = L. Lyon, U.S. Fish and Wildlife Service (unpubl. data); 5 = M. Marsh, U.S. Environmental Protection Agency (unpubl. data); 6 = Gremillion-Smith and Woolf (1993).

[b] Deaths from anticholinesterases based on cholinesterase activity in the brain.

There were no records of owl deaths from antiChE poisoning in Mexico or Canada. The seriously declining burrowing owl population in Canada (Dundas 1995) has been attributed to antiChE pesticides, particularly carbofuran (James and Fox 1987, Fox et al. 1989). Populations of burrowing owls are also declining in the U.S., but no studies have been conducted on pesticide involvement (Fuller et al. 1995).

Deaths of experimental eastern screech-owls occurred after they were given capsule doses of individual antiChE compounds including the OPs EPN, fenthion and monocrotophos and the carbamate carbofuran. They were unusually tolerant of the OP EPN in comparison to other species (Wiemeyer and Sparling 1991).

Several captive eastern screech-owls died after being fed carbofuran or fenthion mixed in "meatballs" (N. Vyas pers. comm.); however, the owls were more tolerant of these antiChEs than reported by Wiemeyer and Sparling (1991).

Anticoagulants. Death from anticoagulants is more difficult to document than most other classes of compounds but hemorrhaging in tissues is usually a good indicator. Residues in tissues may be present, but they are not meaningful in a diagnostic sense (Hegdal and Colvin 1988).

Field experiments with radio-marked eastern-screech owls, common barn-owls, great horned owls and long-eared owls in Virginia showed that six eastern screech-owls and one long-eared owl apparently were killed by the rodenticide brodifacoum (on the basis of presence of residues or extensive hemorrhaging) in study areas where baits were applied (Hegdal and Colvin 1988). In experimental studies where owls were fed rodents killed by anticoagulants, two great horned owls and a northern saw-whet owl died from diphacinone and common barn-owls died from bromadiolone (one bird) and brodifacoum (five birds; Mendenhall and Pank 1980).

Cyanide. Several eastern screech-owls died after being administered gelatin capsules containing sodium cyanide (Wiemeyer et al. 1986); however, I located no records of mortality of wild owls from cyanide.

Strychnine. Strychnine is an extremely toxic, fast-acting pesticide that is used to control avian and mammalian pests. Death occurs after ingestion of treated grain or animal carcasses. Residues of strychnine in ingesta are used as an indicator of death, particularly when they equal or exceed an amount considered lethal (Redig et al. 1982).

After strychnine-treated grain was used in a rock dove (*Columba livia*) eradication program in Minnesota, three snowy owls and a great horned owl were found dead along with a number of rock doves and other birds (Table 3, Redig et al. 1982). Residues of strychnine were found in ingesta (primarily remains of rock doves) of all four owls and levels were considered lethal in two of the snowy owls and probably lethal in the other two birds (Redig et al. 1982). Secondary poisoning was not reported in other field studies (summarized by Colvin et al. 1988), and the secondary hazard from strychnine was classified as minimal.

Table 3. Mortality of owls from strychnine and anticoagulant pesticides (brodifacoum) in North America.

PESTICIDE	YEAR	AREA	NUMBER OF DEATHS	SOURCE[a]
Eastern Screech-Owl (*Otus asio*)				
Brodifacoum	1981–82	Virginia	6	1
Long-eared Owl (*Asio otus*)				
Brodifacoum	1981–82	Virginia	1	1
Snowy-Owl (*Nyctea scandiaca*)				
Strychnine	1981	Minnesota	3	2
Great Horned Owl (*Bubo virginianus*)				
Strychnine	1981	Minnesota	1	2

[a] 1 = Hegdal and Colvin (1988); 2 = Redig et al. (1982).

OTHER REALIZED OR OTHER POTENTIAL SUBLETHAL EFFECTS

Organochlorines. Several studies of OC residues in owls indicated no obvious effects. For example, Sundlof et al. (1986) analyzed brains of 30 owls (17 eastern screech-owls, 10 barred owls and three great horned owls found dead in Florida in the 1970s) for residues of OCs. Levels were below the lethal range but several contained elevated residues that may have induced other adverse effects. Generally low residues of OCs were detected in brains and other tissues of eight common barn-owls, seven great horned owls, three burrowing owls, one long-eared owl and three short-eared owls in Canada (Noble and Elliott 1990).

Anticholinesterases. Not all birds exposed to antiChEs become intoxicated and not all intoxicated birds die. For example, a radio-telemetry study of great horned owls in agricultural areas in Iowa in 1987–88 concluded that OPs had no apparent effects on survival of owls and there was slight inhibition of ChE activity in their brains (Buck et al. 1996). Common barn-owls secondarily exposed to low doses of the OP famphur in poisoned prey for 10 days experienced no overt signs of intoxication; however, there was a reduction of 34–67% in mean ChE activity in their brains (Hill and Mendenhall 1980).

Strychnine. Two captive great horned owls exhibited significant changes in behavior after they consumed mice that died after ingesting strychnine (Cheney et al. 1987); however, Wiemeyer (1989) questioned the validity of these findings be-

cause of the small sample size, no controls and repeated use of the same owl in different tests.

Anticoagulants. Field experiments in New Jersey with radio-marked barred owls (Hegdal and Colvin 1988) and common barn-owls (Hegdal and Blaskiewicz 1984) indicated that the survival of either species and productivity of common barn-owls apparently was unaffected in areas where brodifacoum baits were applied. Two of the three common barn-owls that died after treatment exhibited signs of trauma and none had quantifiable residues of brodifacoum in their tissues.

A field experiment with eastern screech-owls and other raptors in a fruit orchard in Virginia where brodifacoum pellets were applied revealed that no quantifiable residues were detected in the mutilated carcass of one owl found dead. Two owls were euthanized several weeks after application of pellets. One owl appeared normal but the other had extensive areas of hemorrhaging and low residues of brodifacoum (Merson et al. 1984).

Zinc phosphide. Zinc phosphide is used as a rodenticide and treated grain or pellet formulatives are used. Although the baits are extremely toxic when ingested, there is little or no chance of secondary poisoning because the material breaks down rapidly. For example, captive great horned owls showed no signs of intoxication after multiple feedings on black-tailed jack-rabbits (*Lepus californicus*) that had died after ingesting zinc phosphide (Evans et al. 1970).

Mercury. Mercury was formerly used as a fungicide (seed treatment) on planted grain. Residues of mercury were recorded in eggs and livers of several species of owls in Canada in the 1960s and 1970s, but most residues were below known effect levels (Fimreite et al. 1970, Noble and Elliott 1990).

Arsenic. A pair of common barn-owls nested on a Superfund site in Texas that contained high levels of arsenic and other contaminants (S. Sheffield pers. comm.). Concentrations of arsenic were detected in tissues and prey of the barn owls. Productivity may have been reduced to some extent (10 fledged young from five nesting attempts from 1988–90) and the owls apparently ingested more invertebrate prey than usual, but effects were not linked to arsenic or other contaminants (S. Sheffield pers. comm.).

DISCUSSION

In comparison to other birds of prey, there are relatively few studies on the effects of contaminants on wild owls. There was little indication that OC pesticides exerted adverse effects on productivity or mortality of owls in North America; this was related primarily to lower residues in predominantly mammalian and invertebrate prey of owls in comparison with avian prey (Henny 1972, Noble and Elliott 1990). Sundlof et al. (1986) presented residue data for owls found dead or moribund that were arbitrarily categorized as insectivorous (eastern screech-owl) or omnivorous (barred owl and great horned owl). None of the owls had lethal levels in their brains and there were no apparent differences in residues between the two groups. Only one instance of a significant relationship between DDE residues in the egg and shell thinning was located (Henny et al. 1984). Also, only one instance of eggshell thinning of ≥18% (level associated with population declines when occurring over several years) was found (Anderson and Hickey 1972). Most studies of owls reported few effects of OCs on eggshell thinning, productivity, mortality or population trends even during the peak of OC use (Seidensticker and Reynolds 1971, Henny 1972, Klaas and Swineford 1976, Klaas et al. 1978, Lincer and Clark 1978, Springer 1980, Henny et al. 1984, Noble and Elliott 1990). Population declines of common barn-owls in the midwestern United States from the 1950s to the 1980s were thought related to habitat deterioration rather than pesticides (Colvin 1985); however, no contaminant-related studies were conducted. Coincidentally, populations of common barn-owls were stable or increasing in most of its range in the U.S. (Stewart 1980, Fuller et al. 1995).

Mortality from OCs was low with only 44 owls of three species reported killed and most of these were records from New York State in the 1980s. The temporal and geographical data seem biased toward periods and areas with active ecotoxicological programs. Most owl mortality from OCs probably occurred in the 1950s and 1960s when there was greatest use of the most toxic compounds such as DDT, heptachlor, aldrin, dieldrin and endrin. Deaths of most owls from OCs probably occurred from agricultural applications, but the three great horned owls that died from dieldrin on the Rocky Mountain Arsenal in Colorado (Fordham and Reagan 1993) apparently were exposed to high levels of OCs originating from former pesticide manufacturing plants. Further research with owls and OCs is continuing at the Rocky Mountain Arsenal (Vander Lee et al. 1995).

Experimental owls exposed to OCs seemed as sensitive as other birds of prey as they experienced mortality, reproductive problems and eggshell thinning; however, field studies revealed that owls infrequently accumulated residues sufficient to induce serious problems.

In relation to effects of antiChE compounds on owls, only 19 deaths were recorded in North America. Several of the mortalities of great horned owls from carbofuran were related to illegal deployment of laced carcasses or bait to deliberately kill wildlife (L. Lyon unpubl. data). Burrowing owls in Canada (James and Fox 1987, Fox et al. 1989) showed highly significant correlations between carbofuran spraying near nest burrows and immediate effects on reproductive success; but mortality from carbofuran was not established and there were no reports of ChE data from blood or brain samples. Other important questions regarding burrowing owls pertain to age-related sensitivity and whether their invertebrate prey base was reduced. The impacts of mercury, arsenic, zinc phosphide and cyanide on North American owls appear minimal; a few owls died from anticoagulants ($N = 7$) in field experiments and strychnine ($N = 4$).

In conclusion, it appears that owls in North America have tolerated pesticides relatively well. Only one field study indicated reproductive problems, there was little evidence of eggshell thinning, relatively few pesticide-related mortalities were reported and there was little hard evidence for pesticide-related population declines. Nevertheless, there are many relations of pesticides and owls that have received little attention. For example, limited evidence indicates that captive common barn-owls are more susceptible to anticoagulants during molting (Newton et al. 1994). More information is needed on effects of sublethal exposure on long-term survival and fecundity as well as interspecific and intraspecific differences in response to pesticides. From a contaminant standpoint, the burrowing owl is a top candidate for further study due to continued population declines in most of its range in the U.S. and Canada.

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LITERATURE CITED

ANDERSON, D.W. AND J.J. HICKEY. 1972. Eggshell changes in certain North American birds. *Proc. Int. Ornithol. Congr.* 15:514–540.

BERNARD, R.F. AND C.J. WALLACE. 1967. DDT in Michigan birds. *Jack-Pine Warbler* 45:11–17.

BEYER, W.N., G.H. HEINZ AND A.W. REDMON-NORWOOD [EDS.]. 1996. Environmental contaminants in wildlife–interpreting tissue concentrations. Lewis Publishers, Boca Raton, FL U.S.A.

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.

———, O.H. PATTEE, C.J. HENNY AND R.M. PROUTY. 1983. First records of chlordane-related mortality in wild birds. *J. Wildl. Manage.* 47:196–198.

BUCK, J.A., L.W. BREWER, M.J. HOOPER, G.P. COBB AND R.J. KENDALL. 1996. Monitoring great horned owls for organophosphorus pesticide exposure in agricultural areas of southcentral Iowa. *J. Wildl. Manage.* 60:321–331.

CHENEY, C.D., S.B. VANDER WALL AND R.J. POEHLMANN. 1987. Effects of strychnine on the behavior of great horned owls and red-tailed hawks. *J. Raptor Res.* 21: 103–110.

COLVIN, B.A. 1985. Common barn-owl population decline in Ohio and the relationship to agricultural trends. *J. Field Ornithol.* 56:224–235.

———, P.L. HEGDAL AND W.B. JACKSON. 1988. Review of non-target hazards associated with rodenticide use in the U.S.A. *EPPO Bull.* 18:301–308.

DUNDAS, H. 1995. Burrowing owl status and conservation programs. *Bird Trends* 4:21–22.

EVANS, J., P.L. HEGDAL AND R.E. GRIFFITH. 1970. Methods of controlling jack rabbits. Pages 109–116 *in* R.A. Dana [ED.], Proc. 4th Vertebrate Pest. Conf., Sacramento, CA U.S.A.

FERGUSON, D.E. 1964. Some ecological effects of heptachlor on birds. *J. Wildl. Manage.* 28:158–162.

FIMREITE, N., R.W. FYFE AND J.A. KEITH. 1970. Mercury contamination of Canadian prairie seed eaters and their avian predators. *Can. Field-Nat.* 84:269–276.

FLEMING, W.J., M.A.R. MCLANE AND E. CROMARTIE. 1982. Endrin decreases screech owl productivity. *J. Wildl. Manage.* 46:462–468.

FLICKINGER, E.L. AND K.A. KING. 1972. Some effects of aldrin-treated rice on Gulf Coast wildlife. *J. Wildl. Manage.* 36:706–727.

FORDHAM, C.L. AND E.P. REAGAN. 1993. Ecological risk assessment case study: assessing ecological risk at Rocky Mountain Arsenal. Pages 8–1 to 8–39 *in* W.V.D. Schalie, R. Landy and D.C. Menzie [EDS.], A review of ecological case studies from a risk assessment perspective. EPA/630/R-92/005, Washington, DC U.S.A.

FOX, G.A., P. MINEAU, B. COLLINS AND P.C. JAMES. 1989. The impact of the insecticide carbofuran (Furadan 480F) on the burrowing owl in Canada. Technical Rep. Series No. 72, Canadian Wildlife Service, Ottawa, Canada.

FRANK, R. AND H.E. BRAUN. 1990. Organochlorine resi-

dues in bird species collected dead in Ontario 1972–1988. *Bull. Environ. Contam. Toxicol.* 44:932–939.

FRANSON, J.C. AND S.E. LITTLE. 1996. Diagnostic findings in 132 great horned owls. *J. Raptor Res.* 30:1–6.

FULLER, M.R., C.J. HENNY AND P.H. WOOD. 1995. Raptors. Pages 53–55 *in* Our living resources 1994: a report to the nation on the distribution, abundance and health of U.S. plants, animals, and ecosystems. National Biological Service, Washington, DC U.S.A.

GREMILLION-SMITH, C. AND A. WOOLF. 1993. Screening for anticholinesterase pesticide poisoning in Illinois raptors. *Trans. Ill. Acad. Sci.* 86:63–69.

HAVERA, S.P. AND R.E. DUZAN. 1986. Organochlorine and PCB residues in tissues of raptors from Illinois. 1966–81. *Bull. Environ. Contam. Toxicol.* 36:23–32.

HEGDAL, P.L. AND R.W. BLASKIEWICZ. 1984. Evaluation of the potential hazard to barn owls of Talon (brodifacoum bait) used to control rats and house mice. *Environ. Toxicol. Chem.* 3:167–179.

——— AND B.A. COLVIN. 1988. Potential hazard to eastern screech-owls and other raptors of brodifacoum bait used for vole control in orchards. *Environ. Toxicol. Chem.* 7:245–260.

HENNY, C.J. 1972. An analysis of the population dynamics of selected avian species–with special reference to changes during the modern pesticide era. Wildlife Res. Rept. 1, Bureau of Sport Fisheries and Wildlife, Washington, DC U.S.A.

———, L.J. BLUS AND T.E. KAISER. 1984. Heptachlor seed treatment contaminates hawks, owls, and eagles of Columbia Basin, Oregon. *Raptor Res.* 18:41–48.

———, 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.

HICKEY, J.J. AND D.W. ANDERSON. 1968. Chlorinated hydrocarbons and eggshell changes in raptorial and fish-eating birds. *Science* 162:271–273.

HILL, E.F. AND W.J. FLEMING. 1982. Anticholinesterase poisoning in birds: field biomonitoring and diagnosis of acute poisoning. *Environ. Toxicol. Chem.* 1:27–38.

——— AND V.M. MENDENHALL. 1980. Secondary poisoning of barn owls with famphur, an organophosphate insecticide. *J. Wildl. Manage.* 44:676–681.

JAMES, P.C. AND G.A. FOX. 1987. Effects of some insecticides on productivity of burrowing owls. *Blue Jay* 45: 65–71.

KLAAS, E.E. AND D.M. SWINEFORD. 1976. Chemical residue content and hatchability of screech owl eggs. *Wilson Bull.* 88:421–426.

———, S.N. WIEMEYER, H.M. OHLENDORF AND D.M. SWINEFORD. 1978. Organochlorine residues, eggshell thickness, and nest success in barn owls from the Chesapeake Bay. *Estuaries* 1:46–53.

LINCER, J.L. AND R.J. CLARK. 1978. Organochlorine residues in raptor eggs in the Cayuga Lake Basin. *New York Fish Game J.* 25:121–128.

MCLANE, M.A. AND L.C. HALL. 1972. DDE thins screech owl eggs. *Bull. Environ. Contam. Toxicol.* 8:65–68.

MENDENHALL, V.M. AND L.F. PANK. 1980. Secondary poisoning of owls by anticoagulant rodenticides. *Wildl. Soc. Bull.* 8:311–315.

———, E.E. KLAAS AND M.A. MCLANE. 1983. Breeding success of barn owls (*Tyto alba*) fed low levels of DDE and dieldrin. *Arch. Environ. Contam. Toxicol.* 12:235–240.

MERSON, M.H., R.E. BYERS AND D.E. KAUKEINEN. 1984. Residues of the rodenticide brodifacoum in voles and raptors after orchard treatment. *J. Wildl. Manage.* 48: 212–216.

NEWTON, I., I. WYLLIE, A. GRAY AND C.V. EADSFORTH. 1994. The toxicity of the rodenticide flocoumafen to barn owls and its elimination via pellets. *Pestic. Sci.* 41: 187–193.

NOBLE, D.G. AND J.E. ELLIOT. 1990. Levels of contaminants in Canadian raptors, 1966 to 1988; effects and temporal trends. *Can. Field-Nat.* 104:222–243.

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.

REDIG, P.T., C.M. STOWE, T.D. ARENDT AND D.H. DUNCAN. 1982. Relay toxicity of strychnine in raptors in relation to a pigeon eradication program. *Vet. Hum. Toxicol.* 24:335–336.

SEIDENSTICKER, J.C., IV AND H.V. REYNOLDS, III. 1971. The nesting, reproductive performance, and chlorinated hydrocarbon residues in the red-tailed hawk and great horned owl in south-central Montana. *Wilson Bull.* 83:408–418.

SPRINGER, A.S. 1980. Pesticide levels, egg and eggshell parameters of great horned owls. *Ohio J. Sci.* 80:184–187.

STEWART, P.A. 1980. Population trends of barn owls in North America. *Am. Birds* 34:698–700.

STONE, W.B. AND J.C. OKONIEWSKI. 1983. Organochlorine toxicants in great horned owls from New York, 1981–82. *Northeast. Environ. Sci.* 2:1–7.

———. 1988. Organochlorine-pesticide related mortalities of raptors and other birds in New York, 1982–1986. Pages 429–438 *in* T.J. Cade, J.H. Enderson, C.G. Thelander and C.M. White [EDS.] Peregrine falcon populations: their management and recovery. The Peregrine Fund, Boise, ID U.S.A.

SUNDLOF, S.F., D.J. FORRESTER, N.P. THOMPSON AND M.W. COLLOPY. 1986. Residues of chlorinated hydrocarbons in tissues of raptors in Florida. *J. Wildl. Dis.* 22: 71–82.

VAN VELZEN, A.C., W.B. STILES AND L.F. STICKEL. 1972. Lethal mobilization of DDT by cowbirds. *J. Wildl. Manage.* 36:733–739.

VANDER LEE, B.A., R.A. FRANK AND R.S. LUTZ. 1995. Comparison of great horned owls and black-billed magpies as monitors of environmental contamination at the Rocky Mountain Arsenal, Colorado (Ab-

stract). The Wildl. Soc. 2nd Annual Conf. Portland, OR U.S.A.

WIEMEYER, S.N. 1989. Effects of strychnine on raptors. *J. Raptor Res.* 23:18–19.

———, E.F. HILL, J.W. CARPENTER AND A.J. KRYNITSKY. 1986. Acute oral toxicity of sodium cyanide in birds. *J. Wildl. Dis.* 23:538–546.

———, J.W. SPANN, C.M. BUNCK AND A.J. KRYNITSKY. 1989. Effects of Kelthane® on reproduction of captive eastern screech-owls. *Environ. Toxicol. Chem.* 8:903–913.

——— AND D.W. SPARLING. 1991. Acute toxicity of four anticholinesterase insecticides to American kestrels, eastern screech-owls and northern bobwhites. *Environ. Toxicol. Chem.* 10:1139–1148.

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