Owls as Biomonitors of Environmental Contamination

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Abstract.—Much like the caged canary used by miners, a plethora of wildlife species have been promoted as biomonitors of environmental contamination. These species provide an "early warning system" for toxic contaminants in the environment. Species promoted as useful biomonitors share many common life history characters, such as wide distribution, territorial, non-migratory behavior, high trophic status, and high reproductive rates. Raptor species generally possess these characteristics. The Bald Eagle (*Haliaeetus leucocephalus*) and Osprey (*Pandion haliaetus*) have been widely used as biomonitors of aquatic contamination. However, few higher order consumers have been studied in detail or proposed for use in terrestrial systems. Exposure and effects of environmental contaminants on owls has been largely understudied. The studies done to date on owls and environmental contaminants have been conducted on both captive and wild owls, and have focused on a few selected species. Most of the captive studies have been conducted using Eastern Screech-owl (*Otus asio*) and Barn Owl (*Tyto alba*) colonies at the USFWS Patuxent Wildlife Research Center, Laurel, MD. The relatively few studies conducted on wild owls have included many different species, but have concentrated on the Great Horned Owl (*Bubo virginianus*), Barn Owl, and Eastern Screech-owl, and have focused heavily on analyzing contaminant levels (residue analyses) and post-mortem examination for cause of death of individual owls found dead. As higher order consumers, owls are susceptible to secondary poisoning and can bioconcentrate many different environmental contaminants through their prey. Owls have proven to be sensitive to a wide variety of toxic compounds, including pesticides, PCB's, metals, and fluoride, and are highly susceptible to secondary poisoning from consuming pesticide-poisoned prey. Endpoints examined include reproductive effects, eggshell thickness, residue analyses, cholinesterase inhibition, and induction of liver detoxifying enzymes. Much more work remains to be done using owls as biomonitors of environmental contamination, particularly with captive populations, salvaged individuals, raptor rehabilitation center birds, and with wild populations in areas around hazardous waste sites, smelters, landfills, agricultural croplands, and other major sources of environmental contamination.

In the field of wildlife toxicology, a plethora of wildlife species have been promoted as biomonitors, bioindicators, or sentinels, of environmental contamination (National Research Council 1991, Sheffield and Kendall, in press). Much like the caged canary used by miners, wildlife species today are being increasingly utilized as biomonitors of environmental health and overall quality. These species may provide an "early warning system" for toxic contaminants in the environment. Among the most attractive species for this purpose are the top predators. These species are positioned at the top of food chains and are in a position to be negatively impacted by secondary poisoning and bioaccumulation of contaminants in the

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environment. Within the birds of prey, species such as the Bald Eagle (*Haliaeetus leucocephalus*) and Osprey (*Pandion haliaetus*) have been closely studied, particularly with regard to their reproductive failure and subsequent population declines due to eggshell thinning. However, these species generally feed in aquatic environments. It is generally considered that birds are the most sensitive taxa to contaminants in terrestrial environments (Grue *et al.* 1983, Hoffman 1995). Few birds of prey have been studied in detail or proposed as a sentinel species for use in terrestrial systems. One notable exception to this is the Peregrine Falcon (*Falco peregrinus*), which has been studied extensively due to its dramatic populations declines and endangered status following reproductive failure from exposure to DDT and other organochlorine (OC) insecticides. However, peregrine populations have greatly increased in North America following the ban on DDT and other OCs, and the focus on Peregrine Falcons and contaminants has diminished to a large extent. Currently, owl species, as higher order consumers that take a wide variety of prey species, potentially are of great value as wildlife biomonitor species in terrestrial systems. Yet, owls have been relatively neglected in terms of wildlife toxicology studies (Blus 1996, Wiemeyer 1991). The studies done to date on owls and environmental contaminants have been conducted on both captive and wild owls, and have focused on a few selected species. Most of the captive studies have been conducted using Eastern Screech-owl (*Otus asio*) and Barn Owl (*Tyto alba*) colonies at the USFWS Patuxent Wildlife Research Center, Laurel, MD. The relatively few studies conducted on wild owls have included many different species, but have concentrated on the Great Horned Owl (*Bubo virginianus*), Barn Owl, and Eastern Screech-owl, and have focused heavily on analyzing contaminant levels (residue analyses) and post-mortem examination for cause of death of individual owls found dead. Secondary poisoning of owls that consumed prey tainted with anti-cholinesterase (anti-ChE) insecticides, anti-coagulant rodenticides, or other environmental contaminants is considered a significant route of exposure and can contribute significantly to owl mortality as well as to impaired reproduction and other sublethal effects (Grue *et al.* 1983, Blus 1996).

The rationale for using owls as biomonitors of environmental contamination is that they possess many of the life history characteristics that are desirable of a good biomonitor species. These life history characteristics include: (1) high trophic level status (secondary consumer), (2) wide distribution, (3) territorial, generally non-migratory behavior, (4) high reproductive rates, (5) relatively easy to capture, handle, enumerate, (6) their biology is relatively well known, and (7) they are sensitive to a wide variety of environmental contaminants.

The objectives of this paper are to review and synthesize studies dealing with exposure and effects of environmental contaminants on owls worldwide, the use of owls as biomonitor (sentinel) species in wildlife toxicology studies, the possible role of environmental contaminants in the decline of owls, and to suggest future directions for research in these areas.

CATEGORIZATION OF STUDIES

This paper is divided into two categories, captive studies and field studies. These two categories are then further divided into more specific types of studies, including residue analysis studies, mortality (secondary poisoning), and effects.

Captive Studies

A vast majority of the captive studies on owls have been completed at the U.S. Fish and Wildlife Service's Patuxent Wildlife Research Center in Laurel, Maryland. Here, studies have been conducted examining exposure and possible effects of environmental contaminants, including secondary poisoning, in owls for over two decades. These studies have focused primarily on two species of owls, the Eastern Screech-owl and the common Barn Owl.

Studies on Secondary Poisoning in Captive Owls

Secondary poisoning studies have focused on examining anti-coagulant rodenticides and highly toxic anti-ChE pesticides that are illegally broadcast to control predators (table 1). Generally, it has been found that owls are highly susceptible to secondary poisoning by anti-ChE insecticides and anti-coagulant rodenticides through the consumption of contaminated prey items. Effects found in these studies include both lethal and sublethal effects. Lethal effects were seen in owls exposed to organophosphate (OP) insecticides

Table 1.—*Studies examining secondary poisoning in captive owls.*

Species[1]	Compound	Major findings	Author(s)
TYAL	famphur	found signif. secondary poisoning, signif. plasma and brain ChE inhibition	Hill and Mendenhall (1980)
TYAL, BUVI, AEAC	six rodenticides	demonstrated potential secondary hazards of 4 of 6 rodenticides, sublethal effects seen (regurgitated blood, internal bleeding)	Mendenhall and Pank (1980)
STAL	warfarin	sublethal effects seen (dec. levels of plasma prothrombin), concludes that lethal effects would be unlikely	Townsend *et al*. (1981)
NYSC	Pb	mortality seen following feeding on prey containing lead shot	MacDonald *et al*. (1983)
TYAL	flocoumafen	20% (1 of 5) dosed birds died in 6 days	Newton *et al*. (1994)
TYAL	brodifacoum, flocoumafen	4 of 6 dosed owls died in 1 day trial with brodifacoum, 1 of 5 died in 6 day trial with flocoumafen	Wyllie (1995)
OTAS	carbofuran, fenthion	mortality, signif. depression in brain ChE activity	Vyas *et al*. (unpubl. data, pers. comm.)

[1] See Appendix 1.

(Hill and Mendenhall 1980, N. Vyas pers. comm.), rodenticides (Mendenhall and Pank 1980, Newton *et al.* 1994, Wyllie 1995), and lead shot (MacDonald *et al.* 1983). Sublethal effects seen included regurgitated blood and internal bleeding (Mendenhall and Pank 1980) and decreased levels of plasma prothrombin (Townsend *et al.* 1981) from anti-coagulant rodenticides, and plasma and brain ChE inhibition (Hill and Mendenhall 1980, N. Vyas pers. comm.) for anti-ChE insecticides.

Studies on Effects in Captive Owls

Captive studies on possible effects of environmental contaminants have focused on the OC insecticides (e.g., endrin, kelthane, DDE), PCBs, and fluoride; however, OP (EPN, fenthion, monocrotophos) and carbamate (carbofuran) insecticides, cyanide, lead, and selenium have also been studied (table 2). Dietary exposure to relatively low levels of the OC insecticides DDE and kelthane have been shown to cause severe reproductive effects such as eggshell thinning, egg breakage, embryonic mortality, and reduced reproductive productivity (McLane and Hall 1972, Mendenhall *et al.* 1983). Dietary exposure to low levels of the OC insecticide endrin was not found to result in eggshell thinning, but was found to cause a significant decrease in hatching success (Fleming *et al.* 1982). Although low dietary levels of a PCB congener were found to result in no apparent reproductive effects (McLane and Hughes 1980), an interperitoneal (i.p.) exposure to a PCB congener resulted in induction of the mixed-function oxidases (MFOs) system (cytochrome P450) as well as liver hemorrhages and hepatomegaly (Rinsky and Perry 1981). Dietary fluoride was found to result in elevated fluoride concentrations in bone and eggshells, significantly decreased hatching success and an overall impairment of reproduction, morphological (shorter tibiotarsus and radius-ulna lengths) and biochemical (plasma phosphorus levels) abnormalities, and significantly decreased egg and hatchling weights (Hoffman *et al.* 1985, Pattee *et al.* 1988). Cyanide was found to be acutely lethal to owls at extremely low doses, and elevated levels of cyanide in blood allowed for easier detection of cyanide poisoning than other tissues (Wiemeyer *et al.* 1986). Selenium at levels known to occur in small mammals at Kesterson NWR in California was found to cause morphological abnormalities (decreased femur lengths), biochemical changes in the liver (activation of the glutathione system,

Table 2.—*Studies examining effects of environmental contaminants on captive owls.*

Species[1]	Compound	Major findings	Author(s)
OTAS	DDE	signif. eggshell thinning (13%) from diet of 10 ppm dry wt.	McLane and Hall (1972)
BUBU, 26 other indiv. (species not given)	dieldrin	28 owls died mysteriously; dieldrin-treated lumber used for shavings in cages of rodents fed to owls linked to at least 20 of the deaths	Jones *et al*. (1978)
OTAS	PCBs	Aroclor 1,248 fed (3 mg/kg) to owls - no effects on eggshell thickness, no. of eggs laid, young hatched or fledged	McLane and Hughes (1980)
TYAL	PCBs	injection of 30 mg/kg Aroclor 1254 resulted in induction of MFO system (cytochrome P450), liver hemorrhages and hepatomegaly seen	Rinsky and Perry (1981)
OTAS	endrin	owls fed 0.75 ppm endrin produced 43% fewer fledged owlets than controls; hatching success appeared to be main repro. variable affected; no eggshell thinning seen	Fleming *et al*. (1982)
TYAL	DDE, dieldrin	owls fed diet containing 3.0 ppm DDE, 0.5 ppm dieldrin, or both; DDE caused signif. eggshell thinning, egg breakage, embryonic mortality, reduced repro. productivity; dieldrin caused slight (but signif. eggshell thinning, no signif. reduction in breeding success	Mendenhall *et al*. (1983)
OTAS	fluoride	owls fed diet containing 0, 40, or 200 ppm fluoride; at 40 ppm, signif. smaller egg volume, shorter tibiotarsus length and higher plasma P seen, at 200 ppm, signif. lower egg wts., lengths, shorter tibiotarsus and radius-ulna lengths seen, day 1 hatchling weight about 10% less than controls; overall signif. repro. impairment seen	Hoffman *et al*. (1985)
OTAS	sodium cyanide	owls given 6, 12, 24, or 48 mg/kg sodium cyanide in gelatin capsule placed in proventriculus; LD50 8.6 mg/kg, elevated blood cyanide levels found, blood superior to liver as tissue of choice for detecting cyanide exposure	Wiemeyer *et al*. (1986)
OTAS	fluoride	owls fed 0, 40, or 200 mg/kg; hatching success neg. impacted at 200 mg/kg, eggshell thickness not affected, fluoride concs. elevated in bone and eggshells, large variations among indivs.	Pattee *et al*. (1988)
OTAS	kelthane	owls fed diet containing 10 ppm kelthane (with or without DDT-related contams.); eggshell wt. and thickness index signif. lower for both dosed groups, eggshell thickness signif. lower for kelthane w/o DDT impurities than for controls, signif. dec. in % of eggs hatching for owl pairs w/no nesting experience	Wiemeyer *et al*. (1989)
OTAS	EPN, fenthion, carbofuran, monocrotophos	acute toxicity high (LD50's 1.5-3.9 mg/kg) for all compounds, brain ChE activity depressed >65% for all compounds in owls that died within 24 hrs.	Wiemeyer and Sparling (1991)
OTAS	selenium	owls fed diets containing 0. 4.4, or 13.2 ppm (wet wt.) Se; at 4.4 ppm, no malformed nestlings, but femur lengths of young signif. dec., liver biochem. (glutathione/lipid peroxidation) neg. affected in 5 day old nestlings; at 13.2 ppm, adult mass and repro. success dec. signif.	Wiemeyer and Hoffman (1996)

[1] See Appendix 1.

increased liver peroxidation), and a significant decrease in adult mass and reproductive success (Wiemeyer and Hoffman 1996).

Field Studies

Residue Analyses in Wild Owl Tissues

Residue analysis studies have been relatively numerous and wide-ranging in owls (table 3). A vast majority of these studies have been conducted in North America (US, Canada), but studies in Europe (The Netherlands, Norway, Spain, UK) and Africa (South Africa) have also been done. Many different owl species have been used in these studies—everything from small owls (*Otus asio*) to large owls (*Bubo bubo*). The largest data bases found for contaminant residues in owls are for *Bubo virginianus* (11 studies), *Tyto alba* (10 studies), *Asio otus* (six

Table 3.—*Studies examining contaminant residues in wild owls.*

Species[1]	Contaminants	Location	Author(s)
ASOT, TYAL	OCs/PCBs	The Netherlands	Koeman and van Genderen (1966)
ASFL, BUVI, SPCU	Hg	Canada (Alb., Sask.)	Fimreite *et al*. (1970)
BUVI	OCs/PCBs	Canada (Ontario)	Postupalsky (1970)
STAL	OCs/PCBs	Denmark	Karlog *et al*. (1971)
BUVI (eggs, juv., adults)	OCs/PCBs	US (Montana)	Seidensticker and Reynolds (1971)
ASOT, TYAL	OCs/PCBs	The Netherlands	Fuchs *et al*. (1972)
BUVI	OCs/PCBs	US (Texas)	Flickinger and King (1972)
OTAS (eggs)	OCs/PCBs	US (Ohio)	Klaas and Swineford (1976)
STAL, TYAL	Hg	United Kingdom	Stanley and Elliott (1976)
BUVI, OTAS (eggs)	OCs	US (New York)	Lincer and Clark (1978)
TYAL	OCs/PCBs	US (Maryland)	Klaas *et al*. (1978)
ASCA, ASFL, BUAF, BUVI, TYAL, TYCA	OCs/PCBs	Canada, South Africa	Peakall and Kemp (1980)
BUVI	OCs/PCBs	US (Ohio)	Springer (1980)
BUVI	OCs	US (New York)	Stone and Okoniewski (1983)
OTKE	heptachlor	US (Oregon)	Henny *et al*. (1984)
AEAC, ASFL, ASOT, BUVI, OTAS, STVA	OCs/PCBs	US (Illinois)	Havera and Duzan (1986)
OTAS, BUVI, STVA, TYAL	OCs/PCBs	US (Florida)	Sundlof *et al*. (1986)
AEFU, ASFL, ASOT BUBU, GLPA, STAL, SUUL	Hg, OCs/PCBs	Norway	Froslie *et al*. (1986)

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Species[1]	Contaminants	Location	Author(s)
TYAL	OCs	Spain	Sierra and Santiago (1987)
ASFL, ASOT, BUVI, NYSC, SPCU, STNE	Hg, OCs/PCBs	Canada	Noble and Elliott (1990)
OTAS	OCs/PCBs	Canada (Ontario)	Frank and Braun (1990)
TYAL	Cd, Cu, Pb, Zn	The Netherlands	Denneman and Douben (1993)
ASOT, BUBU, OTSC STAL, TYAL	Pb	France	Pain and Amiard-Triquet (1993)
BUVI, OTKE	Pb, Cd	US (Idaho)	Henny *et al.* (1994)
TYAL	Cd, Cu, Pb, Mn, Fe	The Netherlands	Esselink *et al*. (1995)
TYAL	As	US (Texas)	Sheffield and McClure (in review)
BUVI	dieldrin	US (Colorado)	Hoff (pers. comm.)

[1] See Appendix 1.

studies), *Asio flammeus* (five studies), and *Otus asio* (five studies). Contaminants studied in owls include metals (mostly Hg, Pb, and Cd), metalloids (As, Se), OC insecticides, and PCBs. It is not possible to generalize the patterns of contaminants found in owls, but relatively high levels of contaminants have been found in owl eggs and tissues in many different contaminated areas. Recently, several studies have successfully used non-lethal methods to examine exposure to contaminants on owls, including residue analysis of primary feathers for assessing detectable metal burdens in owls from contaminated sites (Denneman and Douben 1993, Esselink *et al.* 1995, Sheffield and McClure, unpubl. data) and blood and fecal samples for analyzing OP insecticide exposure and plasma ChE activity (Buck *et al.* 1996).

Accounts of Mortality (Secondary Poisoning) in Wild Owls

There have been a number of accounts of mortality through secondary poisoning in wild owls (table 4). Post-mortem examinations of individual owls have found that insecticides and rodenticides have been responsible for many deaths, and it should be kept in mind that there may be a multitude of undetected owl mortality incidents for every one observed. The rodenticide thallium sulfate was found to cause secondary poisoning in owls in Germany (Steininger 1952) and Denmark (Clausen and Karlog 1977). Several anti-ChE insecticides (OPs and carbamates) have been implicated in mortalities of wild owl species. In Israel, a mass mortality of raptors, including *Tyto alba*, *Asio otus*, and *Asio flammeus*, occurred from feeding on contaminated prey following application of the OP insecticide monocrotophos (Mendelssohn and Paz 1977). The OP insecticide famphur, used on cattle, was found to cause tertiary mortality in a Great Horned Owl in Oregon (Henny *et al.* 1987). A number of Great Horned Owls throughout the U.S. were found to have been poisoned by anti-ChE insecticides, including phorate, fenthion, and carbofuran (Franson and Little 1996). The major component of the avicide Rid-a-Bird, the OP insecticide fenthion, has been found to cause mortality in many species of owls in North America, including Snowy Owls (*Nyctea scandiaca*), Short-eared Owls (*Asio flammeus*), and Great Horned Owls (*Bubo virginianus*). In Kenya, Africa, Keith and Bruggers (in press) report on raptor mortalities from fenthion poisoning used to control Quelea (*Quelea quelea*) colonies. Owls found to have died from fenthion poisoning include Cape Eagle Owls

Table 4.—*Accounts of mortalities (secondary poisoning) in wild owl populations.*

Species[1]	Compound	Major findings	Location	Author(s)
ATNO, TYAL	thallium sulfate	dead ATNO and TYAL found following thallium use to control rodents	Germany	Steininger (1952)
STVA	heptachlor	one indiv. found dead in pasture sprayed with heptachlor	US (Mississippi)	Ferguson (1964)
ASOT, TYAL	Hg, dieldrin, aldrin, heptachlor	several dead indivs. found	The Netherlands	Koeman *et al*. (1969)
BUVI	aldrin	one indiv. found dead in aldrin-treated rice field	US (Texas)	Flickinger and King (1972)
ASFL, ASOT, TYAL	monocrotophos	dead or dying (ASFL 5, ASOT 2, TYAL 22), and paralyzed but recovered (ASFL 4, ASOT 2, TYAL 10) owls found	Israel	Mendelssohn and Paz (1977)
STAL	thallium sulfate	dead STAL found following thallium use to control rodents	Denmark	Clausen and Karlog (1977)
NYSC, BUVI	strychnine	dead NYSC(3) and BUVI(1) found having fed on pigeons that fed on strychnine-laced corn	US (Minnesota)	Redig *et al*. (1982)
BUVI	chlordane	BUVI(1) found dead	US (Oregon)	Blus *et al*. (1983)
TYAL	brodifacoum	no mortality found, only one TYAL with residues after use of brodifacoum on farms to control rats and mice; little exposure due to TYAL prey choice (meadow voles)	US (New Jersey)	Hedgal and Blaskiewicz (1984)
OTAS, TYAL	brodifacoum	dead OTAS (1) found, owls and voles contained signif. brodifacoum residues, OTAS (1) found with sublethal clotting	US (Virginia)	Merson *et al*. (1984)
TYAL	brodifacoum	mass mortalities in palm-oil plantations after TYAL fed on rodents	Malaysia	Duckett (1984)
BUVI	dieldrin	BUVI(1) found to have lethal levels (1974-81)	US (Illinois)	Havera and Duzan (1986)
BUVI	famphur	one indiv. found dead near cattle feedlot, brain ChE activity depressed 85%, suspected tertiary poisoning - owl ate Red-tailed Hawk that ate magpie	US (Oregon)	Henny *et al.* (1987)
ASOT, BUVI, OTAS, STVA	brodifacoum	owl species living in vicinity of apple orchard impacted by brodifacoum; 32 of 38 OTAS exposed, 6 OTAS mortalities, one ASOT mortality	US (Virginia)	Hedgal and Colvin (1988)
BUVI, OTAS, TYAL	OC insecticides	BUVI found to have died from exposure to chlordane (4), dieldrin (1), dieldrin and chlordane (2), and mixture of OCs (6); TYAL(1) and OTAS(1) died from dieldrin exposure (1982-1986)	US (New York)	Stone and Okoniewski (1988)

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Species[1]	Compound	Major findings	Location	Author(s)
BUVI, TYAL	endrin	BUVI(1) and TYAL(4) found dead	US (Washington)	Blus *et al*. (1989)
SPCU	carbofuran	carbofuran sprayed to control grasshoppers had signif. impact on Burrowing Owl survival and repro. success; 27% dec. in repro. success in nests sprayed within 50 m	Canada	Fox *et al*. (1989)
OTAS	OC insecticides	OTAS(1) found with lethal levels of a mixture of OC insecticides	Canada (Ontario)	Frank and Braun (1990)
TYAL	brodifacoum, difenacoum	145 owl carcasses examined (1983-1989); 10% found with rodenticide residues (one or the other or both)	UK	Newton *et al*. (1990)
TYAL	aldrin/dieldrin	627 owls autopsied, poisoning found in 8.8% of owls (up to 40% of all mortalities in some agricultural areas from 1963-1977)	UK	Newton *et al*. (1991)
BUVI, OTAS, STVA	OP, carbamate insecticides	105 owl carcass examined for cause of death and brain ChE activity; 5.7% found to have died from anti-ChE insecticides	US (Illinois)	Gremillion-Smith and Woolf (1993)
BUVI, OTAS, TYAL	chlordane, dieldrin	BUVI(9), TYAL(1), and OTAS(3) found to have died from exposure to chlordane, BUVI(2) died from dieldrin (1986-1990)	US (New York, Maryland)	Okoniewski and Novesky (1993)
TYAL	rodenticides (4)	353 owl carcasses examined (1990-1994); only 1.4% died from rodenticide poisoning, but 32% of carcasses contained residues	UK	Wyllie (1995)
BUVI	OC, OP insecticides, H_2S	132 owl carcasses examined for cause of death; 8% found to have died from exposure to toxic chemicals	US (24 states)	Franson and Little (1996)
BUVI	dieldrin	subacute exposure to dieldrin found to kill a large but unknown no. of juv. and adult owls from 1994-1996 (residues found in blood, brain, liver)	US (Colorado)	D. Hoff (pers. comm.)
ASFL	carbofuran	ASFL(1) found dead	US (Utah)	L. Lyon (unpubl. data)
BUVI	carbofuran	BUVI(1 each) found dead in VA(1987), DE (1989), IA (1990)	US (Virginia, Iowa, Delaware)	L. Lyon (unpubl. data)
ASFL	fenthion	ASFL(1) found dead	US (Washington)	M. Marsh (pers. comm.)
BUVI	fenthion	BUVI(1) found dead (1996)	US (Washington)	M. Marsh (pers. comm.)
NYSC	fenthion	NYSC(1) found dead	US (Illinois)	M. Marsh (pers. comm.)
TYAL	phorate	TYAL(1) found dead (1989)	US (Wisconsin)	J. Spinks (unpubl. data)
ASOT, BUVI OTAS, TYAL	OC insecticides, PCBs	lethal levels found in numerous BUVI, ASOT, OTAS, and TYAL (OC mixtures and PCBs) in late 1980s	US (New York)	W. Stone (pers. comm.)
NYSC	fenthion	NYSC(at least 1) found dead at airport	US (Virginia)	N. Vyas (pers. comm.)

[1] See Appendix 1.

(*Bubo capensis*), Giant Eagle Owls (*Bubo bubo*), and a Pearl-spotted Owlet (*Glaucidium perlatum*). In New York, many individual Great Horned Owls, Barn Owls, and Eastern Screech-owls have been found to have died from OC insecticide exposure, including DDE, dieldrin, chlordane, heptachlor, and PCBs, since the early 1980's (Stone and Okoniewski 1983, Okoniewski and Novesky 1993, W.B. Stone pers. comm.). At the Rocky Mountain Arsenal in Colorado, numerous juvenile and adult Great Horned Owls were found to have died from exposure to high levels of the OC insecticide dieldrin, a soil contaminant at the site.

Several studies have examined cause of death in owls through post-mortem necropsy of owl carcasses accumulated over a number of years. In the U.S., an examination of 132 Great Horned Owl carcasses revealed that about 8 percent of them were killed by toxic chemical poisoning, mainly insecticides (Franson and Little 1996). Also in the U.S., 105 owl carcasses (Great Horned Owl, screech owl, Barred Owl (*Strix varia*)) from central and southern Illinois were examined for cause of death and brain ChE activity (Gremillion-Smith and Woolf 1993). They determined that at least six (5.7 percent) of the owls may have died from anti-ChE insecticide poisoning, including two adult Great Horned Owls whose brain ChE activities were depressed 53 percent and 69 percent of normal activities and one subadult screech owl whose brain activity was depressed 60 percent of normal activity.

In the UK, secondary poisoning of Barn Owls by anti-coagulant rodenticides has been closely examined. Newton *et al.* (1990) examined brodifacoum and difenacoum exposure in Barn Owls from the UK, and of the 145 owls tested from 1983-1989, 10 percent were found to have rodenticide residues (one or the other or both). A study of 627 Barn Owl carcasses from the UK revealed that about 9 percent of these owls were poisoned by the OC insecticide aldrin/dieldrin, although up to 40 percent of all mortalities in some agricultural areas resulted from aldrin/dieldrin poisoning (Newton *et al.* 1991). Wyllie (1995) analyzed 353 carcasses of Barn Owls from 1990-1994, and found that, although only 1.4 percent of owls died from poisoning, 32 percent of the owls contained rodenticide residues.

Studies of Secondary Poisoning in Wild Owls

Several studies have been carried out specifically to test the secondary poisoning hazards of trial rodenticides on non-target species. The anti-coagulant rodenticide brodifacoum has been the focus of several studies in wild owls. Hedgal and Blaskiewicz (1984) found that Talon (50 ppm brodifacoum), used to control house mice and rats, did not cause mortality in Barn Owls from New Jersey. Residues were found in only one Barn Owl. However, as these Barn Owls fed mostly on meadow voles (*Microtus pennsylvanicus*) and did not spend much time hunting in and around farms, it is not surprising that no mortality was seen. Merson *et al.* (1984) examined brodifacoum exposure to owls from its use in controlling voles in a Virginia apple orchard. Three screech owls and one Barn Owl inhabiting the orchard area were fit with radio-transmitters. One screech owl died, one screech owl had large subcutaneous blood clot on left side of breast, voles contained significant brodifacoum residues, and two screech owls contained brodifacoum residues, indicating that secondary poisoning of the screech owl population in vicinity of orchard occurred. In a similar but more extensive study, Hedgal and Colvin (1988) used radiotelemetry to examine brodifacoum (10 ppm) exposure in Eastern Screech-owls (38), Barred Owls (five), Great Horned Owls (two), and Long-eared Owls (two) living in the vicinity of an apple orchard. They found that 32 screech owls were exposed to brodifacoum, six died from the exposure, four of the six live screech owls contained brodifacoum residues, and one Long-eared Owl died from brodifacoum poisoning.

Studies on Sublethal Effects in Wild Owls

Few studies have examined exposure and possible sublethal effects of environmental contaminants in wild owl populations (table 5). Eggshell thinning due to exposure to OC insecticides has been examined in owls in the U.S. (Hickey and Anderson 1968, Klaas and Swineford 1976, Springer 1980) and Australia (Olsen *et al.* 1993). In California, Hickey and Anderson (1968) found no significant changes in eggshell weights in Great Horned Owl eggs collected from 1886-1936 and 1948-1950. Springer (1980) compared addled and viable Great Horned Owl eggs from Ohio for pesticide

Table 5.—*Studies of sublethal effects of contaminants in wild owl populations.*

Species[1]	Compounds	Major findings	Author(s)
BUVI	OCs	no signif. eggshell thinning (small "n")	Hickey and Anderson (1968)
BUVI	OCs	eggshells showed slight inc. in thickness and weight (n=3)	Seidensticker and Reynolds (1971)
TYAL	OCs	reproductive success	Klaas *et al.* (1978)
SPCU	carbofuran, carbaryl	reproductive success	James and Fox (1987), Fox *et al.* (1989)
TYAL, TYLO TYNO, TYTE, NIRU, NIST, NICO	OCs	signif. eggshell thinning found in TYNO and NIST	Olsen *et al.* (1993)
BUVI	OP insecticides	plasma ChE activity and fecal urates measured, no signif. exposure in corn crops, non-treated habitat and wide diversity of prey limited exposure	Buck *et al.* (1996)
TYAL	arsenic	altered feeding habits (insects preferred); dec. repro. success (#eggs hatched/nest, # young fledged/nest)	Sheffield and McClure (unpubl. data)

[1] See Appendix 1.

levels and eggshell parameters, and found that addled eggs contained consistently higher pesticide levels and were an average of 5 percent thinner. No differences in eggshell thickness was found in screech owl eggs from pre-1947 and from 1973 (Klaas and Swineford 1976). In Montana, Great Horned Owl eggs were found to have relatively low levels of OC insecticide residues and no significant changes in eggshell weight and thickness were found between pre-1946 and 1967 samples (Seidensticker and Reynolds 1971). In New York, relatively high DDE residues were found in screech owl and Great Horned Owl eggs, and eggshell thickness of Great Horned Owl eggs was found to be significantly less than that of pre-DDT era eggshells (Lincer and Clark 1978). In Australia, Olsen *et al.* (1993) found that average eggshell thickness significantly decreased (by 6.3 percent) in the Southern Boobook Owl (*Ninox novaeseelandiae*), and strongly decreased in the Powerful Owl (*Ninox strenua*), after the introduction of DDT to Australia. Eggshell thickness for six other owl species was not found to differ significantly, but sample sizes for most species were small.

Beyond eggshell thinning, few studies have attempted to assess exposure and potential effects of environmental contaminants on wild owl populations. Klaas *et al.* (1978) examined OC insecticide residues and reproductive success in Barn Owls from Chesapeake Bay, Maryland. In 18 nests, they found relatively high levels of DDE, PCBs and dieldrin in eggs and found significant eggshell thinning (5.5 percent) when compared to eggshells from pre-DDT times. Eggshell thickness was found to be inversely correlated with concentrations of DDE, DDD, and dieldrin residues. Reproductive productivity of these Barn Owls was found to be lower than that needed to maintain a stable population, and it was calculated that at least 15 percent of the Barn Owl population had contaminant residue burdens high enough to be detrimental to their reproduction. Following the finding of OP insecticide residues in *Peromyscus* spp. in Iowa cornfields, Buck *et al.* (1995) examined potential exposure of Great Horned Owls to OP insecticides. They used radio-telemetry and non-lethal sampling techniques, analyzing blood plasma for ChE activity and fecal samples for fecal urates (OP metabolites). Of the 27 individual owls followed, three had plasma ChE activities that were significantly less than those of controls. However, they concluded that the large proportion of non-treated habitat within owl home ranges

and the diversity of prey consumed limited OP insecticide exposure in the Great Horned Owls monitored. James and Fox (1987) and Fox *et al.* (1989) found that the anti-ChE insecticide carbofuran, applied to control grasshoppers, significantly impacted Burrowing Owl (*Speotyto cunicularia*) survival and reproductive success when sprayed over nest burrows. In addition, they found a 27 percent decrease in reproductive success in nests where carbofuran was sprayed within 50 m of the nest. The results suggested that the negative impacts were a result of toxicity rather than food removal. Sheffield and McClure (unpubl. data) found that Barn Owls living on an arsenic-contaminated hazardous waste site in Texas showed altered prey selection (preferring katydids over mammals) and decreased reproductive success when compared to clean sites in Texas. Although the number of eggs/clutch was not significantly different, the number of eggs hatched and the number of young fledged per nest were significantly lower than Barn Owls from clean sites and were closer to the averages found in Barn Owls at a DDE-contaminated site (Klaas *et al.* 1978).

DISCUSSION

Many different types of contaminants have been studied in owls, including OC insecticides (DDT and its metabolites, dieldrin, endrin, kelthane, etc.), OP insecticides (chlorpyrifos, terbufos), PCB's, heavy metals (Hg, Pb) metalloids (As, Se), fluoride, and a number of rodenticides (warfarin, brodifacoum, etc.). In the studies outlined above, many different endpoints of contamination have been studied in owls, including bioaccumulation (residue analysis), secondary poisoning, biochemical (ChE inhibition, MFO induction), reproductive (eggshell thinning, productivity), and ecological (dietary changes, etc.). Owls have proven to be among the most sensitive avian species to a number of different environmental contaminants, but have been underused as sentinel species to this point. Owl species should serve as key sentinel species in any evaluation of exposure and possible effects of environmental contamination.

Declines of Owls and Possible Role of Contaminants

It is possible that owl population declines over the last several decades are linked directly to the increased rate of pesticide use over that time, but few studies have examined this issue. Therefore, this is an area that needs further study currently. Several species of owls around the world have been in slow or rapid decline since the mid-1900's when pesticide use dramatically increased. Among these are the Barn Owl in the United States, whose populations have drastically decreased in the agroecosystems of the Great Lakes and midwestern states (Colvin 1985, Marti 1992). Populations of the Burrowing Owl currently are facing problems in North America due mainly to habitat destruction and alteration, although no studies have been conducted to examine a possible role of pesticides in the declines. From the study by Fox *et al.* (1989) in the Prairie Provinces of Canada, it is likely that pesticides are at least partly responsible for the decline. High levels of OC insecticides, PCBs and Hg have been found in eggs and tissues of arctic owls (e.g., Snowy Owl, Great Gray Owl (*Strix nebulosa*), northern populations of Great Horned Owl), who tend to move great distances between seasons and whose populations fluctuate greatly (Noble and Elliott 1990). However, recent residue data for arctic owls is not available and a possible connection between contaminants and population health, fluctuations, and declines in these owls has not been studied. In addition to this, several other North American owls are declining including the Elf Owl, Ferruginous Pygmy-owl, Spotted Owl, and Short-eared Owl (White 1994). While habitat destruction and alteration may serve as the major factor in these declines, the role of widespread pesticide use must not be discounted and should be investigated.

Future Research Directions and Conclusions

There are several different directions for future research on owls and environmental contaminants to follow. Among these are:

1. Continued captive studies are required. Captive studies are relatively expensive, and few facilities are equipped to conduct studies such as this. It is important to attempt to determine the extent of exposure and possible hazard of rodenticides and insecticides in wild owls using captive animals.

2. It is important to systematically monitor for dead owls and to salvage dead owls for examination. This is a common practice in parts of Europe (e.g., The Netherlands, UK), but a greater effort needs to be put forth in other parts of world.

3. There is a need for greater use of radiotelemetry in following the fate of wild owls and their exposure and effects of contaminants.

4. The continued use and development of biomarkers of exposure in owls and their relation to impending biological effects is of great importance.

5. Greater use of non-lethal techniques, such as the use of feathers, eggshells, and blood and fecal samples, to monitor exposure to environmental contaminants should be considered important and will allow continued monitoring of the same live individuals over time while not impacting owl populations.

6. Raptor rehabilitation facilities hold a lot of promise for monitoring exposure and effects of contaminants in owls as well as other raptors. Injured and dead owls are brought to these facilities routinely, and monitoring exposure to contaminants will allow us some insight into potential environmental problems.

7. It is of importance to monitor wild populations of owls at such locations as hazardous waste sites, industrial areas, agricultural areas, landfills, mining areas, and other potentially contaminated sites. As relatively sensitive sentinel species, owls can provide an early warning to potential environmental health hazards.

8. We need to expand international efforts of monitoring contaminants in owls. As many hazardous pesticides and other chemicals are currently being used around the world, mass dieoffs of birds are still occurring in some locations (e.g., Dickcissels (*Spiza americana*) in Venezuela, Swainson's Hawks (*Buteo swainsoni*) in Argentina, Mississippi Kites (*Ictinia mississipiensis*) in Columbia). Continental monitoring, identifying areas of high probability of exposure, is important and should be implemented.

9. Ultimately, the question of whether or not owls (as sensitive, non-target, predatory species) are adequately protected by regulations on toxic chemicals in the environment needs to be answered.

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Appendix 1.—*Listing of common and scientific names of owls used in this paper.*

Aegolius acadicus—Northern Saw-whet Owl (AEAC)
Aegolius funereus—Boreal (nearctic) or Tengmalm's (palaearctic) Owl (AEFU)
Asio capensis—Marsh Owl (ASCA)
Asio otus—Long-eared Owl (ASOT)
Asio flammeus—Short-eared Owl (ASFL)
Athene noctua—Little Owl (ATNO)
Bubo africanus—Spotted Eagle Owl (BUAF)
Bubo bubo—Eagle Owl (BUBU)
Bubo bubo bengalensis—Indian Eagle Owl (BBBE)
Bubo capensis—Cape Eagle Owl
Bubo virginianus—Great Horned Owl (BUVI)
Glaucidium passerinum—Pygmy Owl (GLPA)
Glaucidium perlatum—Pearl-spotted Owlet (GLPE)
Ninox rufa—Rufous Owl (NIRU)
Ninox strenua—Powerful Owl (NIST)
Ninox connivens—Barking Owl (NICO)
Ninox novaeseelandiae—Southern Boobook Owl (NINO)
Nyctea scandiaca—Snowy Owl (NYSC)
Otus asio—Eastern Screech-owl (OTAS)
Otus kennicottii—Western Screech-owl (OTKE)
Otus scops—Scops Owl (OTSC)
Strix varia—Barred Owl (STVA)
Strix nebulosa—Great Gray Owl (STNE)
Strix aluco—Tawny Owl (STAL)
Speotyto (Athene) cunicularia—Burrowing Owl (SPCU)
Surnia ulula—Northern Hawk Owl (SUUL)
Tyto alba—Barn Owl (TYAL)
Tyto capensis—Grass Owl (TYCA)
Tyto longimembris—Eastern Grass Owl (TYLO)
Tyto novaehollandiae—Masked Owl (TYNO)
Tyto tenebricosa—Sooty Owl (TYTE)