Toxicology

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INTRODUCTION

In earlier years (1947–1985), many contaminant-related problems concerning raptors were related to chlorinated hydrocarbon (CH) insecticides, such as DDT, dieldrin, heptachlor, and chlordane, most of which now have been banned in the U.S. and elsewhere. Other contaminants mentioned in the first edition of this manual (Peakall 1987) included mercury, lead, polychlorinated biphenyls (PCBs), and acid deposition, the latter impacting fish populations in poorly buffered lakes and, therefore, adversely affecting Ospreys (*Pandion haliaetus*) and Bald Eagles (*Haliaeetus leucocephalus*). Secondary poisoning of raptors by anticoagulant rodenticides and organophosphorus (OP) pesticides was beginning to be evaluated. The extirpation of the Peregrine Falcon (*Falco peregrinus*) from the eastern U.S. by 1964, and major reductions in numbers elsewhere around the world, was due primarily to DDT, and, perhaps, other CHs. The recovery of the peregrine in the U.S., following the 1972 ban on the widespread use of DDT and much effort in reintroducing the species, and its eventual delisting in 1999 from being an Endangered Species, was recently told in *Return of the Peregrine* (Cade and Burnham 2003).

Overall, the relative importance of specific contaminant issues today is not the same as discussed in the first edition of this manual, and new issues have emerged. That said persistent CHs still adversely influence some species at selected locations (e.g., DDE-reduced nesting success and significantly thinned the eggshells of some Ospreys breeding along the lower Columbia River in 1997–98, even though the population was increasing at the time [Henny et al. 2004]).

This chapter is subdivided into different classes of environmental contaminants that may adversely affect raptor populations. For each class of contaminants, we present: (1) structure and chemistry (what they are), (2) sources and use patterns (where and how they are used), (3) fate and transport (how mobile they are in the environment), (4) toxicology (what their basic mode[s] of action are), (5) effects criteria (what residue concentration and biochemical response in which tissues should be investigated; Table 1), and (6) techniques for studying field exposure and effects (Table 2).

As a note of caution, residue concentrations in the literature may be presented in several ways, which can be confusing (e.g., wet weight [ww], dry weight [dw], lipid weight [lw]). Sometimes the methods section of a paper must be read carefully to determine which value was used; it is critical to understand this terminology because reported concentrations vary tremendously depending upon how data are presented, as well as with the percent moisture and percent lipid in the tissue examined. Concentrations (C) readily can be converted (e.g., $C_{dry} = C_{wet}$ 100/100 - % moisture).

Table 1. Selection of estimated toxicity threshold values for contaminants in raptor species[a].

Species	Chemical [b]	Tissue	Effect [c]	Value w.w. (units)	Ref. [d]
Bald Eagle (*Haliaeetus leucocephalus*)	DDE	Egg	15% reduction in shell thickness	16 mg/kg	1
		Egg	Corresponds to 0.7 young/occupied territory	5.9 mg/kg	2
		Egg	Significant reduction in productivity	12 mg/kg	3
		Egg	Embryo lethality	5.5 mg/kg	2
		Plasma	Corresponds to 5.9 mg/kg in eggs	41 µg/kg	2
		Brain	Lowest value poisoned adult	212 mg/kg	4
	Dieldrin	Brain	Lowest value poisoned adult	3.6 mg/kg	5
	ΣPCBs	Egg	Reduced probability of producing young	20 mg/kg	2
		Plasma	Corresponds to 20 mg/kg in eggs	189 µg/kg	2
	TCDD TEQs	Egg	NOAEL hatching	303 ng/kg	2
		Egg	NOAEL CYP1A induction	135 ng/kg	2
		Egg	LOAEL CYP1A induction	400 ng/kg	2
White-tailed Eagle (*H. albicilla*)	DDE	Egg	LOAEL productivity	6.0 mg/kg	6
		Egg	Strong reduction in desiccation index	8.5 mg/kg	6
		Egg	Corresponds to 0.7 young/occupied territory	10.5 mg/kg	6
	ΣPCBs	Egg	LOAEL for productivity	25 mg/kg	6
	TCDD TEQs	Egg	LOAEL for embryo mortality	320 ng/kg	6
Osprey (*Pandion haliaetus*)	DDE	Egg	Corresponds to 0.8 young/occupied nest	4.2 mg/kg	7
	TCDD TEQs	Egg	NOAEL for productivity	162 ng/kg	8
		Egg	NOAEL for hatching	136 ng/kg	9
		Egg	NOAEL for CYP1A induction	36 ng/kg	9
		Egg	LOAEL for CYP1A induction	130 ng/kg	9
American Kestrel (*Falco sparverius*)	PCB 126	Egg	Embryonic LD_{50}	65 µg/kg	10
		Egg	Significant increase in malformations and edema	2.3 µg/kg	10
	PCB 77	Egg	Embryonic LD_{50}	688 µg/kg	10
	ΣPCBs	Egg	Effects on reproductive and endocrine endpoints	34 mg/kg	11,12
	HE	Egg	Reduced productivity	1.5 mg/kg	13
Peregrine Falcon (*F. peregrinus*)	DDE	Egg	Reduced productivity	15–20 mg/kg	14
	ΣPCBs	Egg	Reduced productivity	40 mg/kg	14
Common Kestrel (*F. tinnunculus*)	MeHg	Brain	Mortality	25–33 mg/kg	15
	MeHg	Liver	Mortality	50–120 mg/kg	15
Red-tailed Hawk (*Buteo jamaicensis*)	MeHg	Liver	Mortality	20 mg/kg	16

[a] Sensitivity to most contaminants is species-specific. Much additional information on non-raptorial species is available (see Beyer et al. [1996]).

[b] DDE = p,p'-dichlorodiphenyl-dichloroethylene; ΣPCBs = sum polychlorinated biphenyl congeners; TCDD TEQs = 2,3,7,8-tetrachlorodibenzo-p-dioxin toxic equivalents; PCB 126 = 3,3',4,4',5-penta-CB (one of the most toxic PCB congeners); PCB 77 = 3,3',4,4'-tetra-CB (one of the most toxic PCB congeners); HE = heptachlor epoxide; MeHg = methylmercury.

[c] NOAEL = no-observed-adverse-effect-level; LOAEL = lowest-observed-adverse-effect-level; LD_{50} = acute oral median lethal dosage.

[d] References: 1, Wiemeyer et al. 1993; 2, Elliott and Harris 2002; 3, Nisbet and Risebrough 1994; 4, Garcelon and Thomas 1997; 5, Prouty et al. 1977; 6, Helander et al. 2002; 7, Wiemeyer et al. 1988; 8, Woodford et al. 1998; 9, Elliott et al. 2001; 10, Hoffman et al. 1998; 11, Fernie et al. 2001; 12, Smits et al. 2002; 13, Henny et al. 1983; 14, Peakall et al. 1990; 15, Koeman et al. 1971; 16, Fimreite and Karstad 1971.

Table 2. Examples of studies using recommended techniques in raptor field ecotoxicology.

Chemical(s) of concern	Sampling matrix	Technique(s)	Reference[a]
Persistent organic pollutants	Egg	**Salvage unhatched eggs or fragments for chemistry** (combined with productivity and other measurements)	1,2,3
	Egg	**Sample egg technique** (collection of eggs for chemistry) (combined with productivity and other measurements)	4,5,6
	Egg	**Laboratory incubation of fresh eggs** (chemical analysis of the yolk sacs or of a sibling egg; morphology, histology, biochemistry of organs)	7,8
	Egg	**Egg swap experiments** (can be combined with collection of eggs and behavioral observations; always combined with productivity, and potentially, other measurements)	9,10
	Major organs: liver, kidney, brain	**Mortality monitoring**: collection of dead and moribund birds for necropsy and chemistry, biochemistry, and histology	3,11
	Blood	**Capture**: migrant or breeding birds, residues	12,13
Mercury	Egg	**Sample egg technique** (see above)	14
	Blood	**Nestlings**: residues	14
	Feathers	**Adults and nestlings**: residues	14
	Liver, kidney, brain	**Nestlings**: residues	14
Lead	Blood	**Nestlings, adults**: residues, ALAD, protoporphyrin, hemoglobin	15,16
Anti-cholinesterase insecticides	Brain	**Cholinesterase activity**: dead or moribund birds	17,18
	Blood	**Cholinesterase activity**: dead or moribund birds	19
	Blood	**Cholinesterase activity**: captured birds	20
	Crop contents	**Chemical residues**: dead birds or surgically removed from live,9 poisoned birds	18,21

Note: ALAD = delta-aminolevulinic acid dehydratase.

[a] References: 1, Ratcliffe 1970; 2, Newton and Galbraith 1991; 3, Newton 1988; 4, Blus 1984; 5, Henny et al. 1983; 6, Henny et al. 2004; 7, Elliott et al. 1996a; 8, Elliott et al. 2001; 9, Wiemeyer et al. 1975; 10, Woodford et al. 1998; 11, Prouty et al. 1977; 12, Henny et al. 1996; 13, Court et al. 1990; 14, DesGranges et al. 1998; 15, Henny et al. 1991; 16, Henny et al. 1994; 17, Henny et al. 1987; 18, Elliott et al. 1996b; 19, Elliott et al. 1997b; 20, Hooper et al. 1989; 21, Henny et al. 1985.

CHLORINATED HYDROCARBON (CH) INSECTICIDES

Chemistry and Toxicology

Matsumura (1985) characterized these synthetic organic insecticides by (1) the presence of carbon, chlorine, hydrogen and sometimes oxygen atoms, including C-Cl bonds, (2) the presence of cyclic carbon chains (including benzene rings), (3) whether or not they were preferentially lipid-soluble, and (4) their stability in the environment. These compounds generally persist in the environment and biomagnify in food chains (some more than others), with raptors at the top of food chains and, especially bird-eating and fish-eating species, being particularly vulnerable. Generally, there are three kinds of CH insecticides: (1) DDT and its analogs (including methoxychlor and dicofol [kelthane]), (2) benzene hexachloride (BHC) isomers including lindane, and (3) cyclodiene compounds (including chlordane, heptachlor, aldrin, dieldrin (HEOD), endrin, toxaphene, mirex, kepone, endosulfan and telodrin. All are neuroactive agents whose modes of action include effects on ion permeability (DDT group) or effects as agents for nerve receptors (BHC and cyclodienes).

These compounds were first introduced in the late 1940s and early 1950s, and, with few exceptions, were banned in most industrialized countries during the 1970s or shortly thereafter. Their principal uses were in agriculture and for disease-vector control. Effects of persistent CH insecticides on raptor populations were widely documented and were catastrophic for some species. Continuing concern reflects their persistence, biomagnification and continued public health use for mosquito control in some countries. In theory, raptors may become exposed to CHs at great distances from application sites due to: (1) atmospheric transport (e.g., elevated concentrations in the Canadian arctic [Barrie et al. 1992]), (2) migratory prey species transporting material from distant sources, or (3) migratory raptors themselves transporting material from distant sources (Henny et al. 1982).

Criteria and Techniques

DDT (and its breakdown product DDE), heptachlor, dieldrin, and perhaps other CH insecticides can cause reduced productivity (Lockie et al. 1969, Ratcliffe 1970, Henny et al. 1983). Unhatched (failed) eggs have been and continue to be analyzed to determine the contaminants causing reduced reproductive success (e.g., Wegner et al. 2005). A nonviable Peregrine Falcon egg analyzed in 1960 represents the earliest study of pollutant-related effects on raptors (Moore and Ratcliffe 1962). As Peakall (1987) pointed out, examining eggs is advantageous because it directly examines the target (i.e., the nonviable egg). CH residue concentrations in the egg are directly related to levels in the adult female (Norstrom et al. 1985), which is not necessarily true for other classes of pollutants. CH concentrations reported from nonviable eggs remaining in the nest, after the expected hatch date (a non-random sample), are usually biased towards higher values, if CHs adversely influenced hatchability. Scientists prefer residues from a randomly collected single "sample egg" (Blus 1984) (1 to 2 weeks into incubation) from a series of nests to evaluate possible effects of CHs on success of eggs remaining in the clutch and to document contaminant levels in populations. Collecting a sample egg can cause nest abandonment for some raptor species, such as Bald Eagles (Grier 1969), which negatively influences productivity, whereas for other species, including Ospreys, nests are rarely abandoned after a short visit for egg collection. The reduction for each Osprey egg collected (usually from a three-egg clutch), for example, was only 0.28 young fledged per active nest (Henny et al. 2004; Fig. 1). The sensitivity of eggs to this group of insecticides is species-specific, and as such, no single diagnostic egg concentration can be used for all species, e.g., DDE adversely influences Osprey reproductive success above 4.2 mg/kg (ww) (Wiemeyer et al. 1988), Bald Eagle above 5.9 mg/kg (ww) (Elliott and Harris 2002), and Peregrine Falcon above 15–20 mg/kg (ww) (Peakall et al. 1990). As expected, the degree of eggshell thinning caused by a given egg concentration of DDE, the only CH insecticide known to thin eggshells except for the structurally similar dicofol (Bennett et al. 1990), also varies among families of raptors and even among species within the same family (Peakall 1975). Usually, shell thickness is compared to pre-DDT era norms based upon eggshells in museums.

CH insecticides, especially the cyclodienes, also kill birds, and when dead raptors are found and these insecticides suspected, the brain should be analyzed and residues compared to diagnostic concentrations based on laboratory studies (see criteria in Beyer et al. 1996). Peakall (1996) reviewed the causes of death of Bald Eagles found dead in the U.S. by a network of federal, state, and private investigators from 1966 to 1983. The

Figure 1. Increasing Osprey (*Pandion haliaetus*) populations have pioneered back into some potentially contaminated industrial sites, e.g., Seattle Harbor, U.S.A., where they are again being used as an indicator species to evaluate numerous contaminants. Note Opsrey nest with two eggs in the foreground *(Photo by J. Kaiser, USGS).*

percentage of deaths attributed to dieldrin decreased following a ban on its use (i.e., 13% in 1966–70, 6.5% in 1971–74, 3.0% in 1975–77, and 1.7% in 1978–83). Decreases in mortality and increases in natality in the late 1970s and 1980s were followed by population increases in those species adversely affected in earlier years. The best evidence of the impact of dieldrin poisoning is the long-term study of Eurasian Sparrowhawks (*Accipiter nisus*) in Britain (Newton et al. 1986). A recent re-analysis of these data shows that at least 29% of the sparrowhawks in the area of high cyclodiene use died directly from dieldrin poisoning, which led to a population decline (Sibley et al. 2000). Comparison of temporal trends in populations of Sharp-shinned Hawks (*A. striatus*) with both egg residues and usage patterns of dieldrin and DDT in North America support the possibility that dieldrin poisoning also may have impacted North American accipiters (Elliott and Martin 1994). Chlordane, persisting from earlier efforts to control turf pests in parks and gardens, recently poisoned songbirds and raptors, particularly Cooper's Hawks (*A. cooperii*) (Stansley and Roscoe 1999).

Blood plasma can be used to monitor long-term CH residue trends in raptor populations and to evaluate local exposure (Henny and Meeker 1981, Court et al. 1990, Elliott and Shutt 1993, Jarman et al. 1994). Migratory species (both raptors and their prey) often are exposed elsewhere during their travels. Based upon DDE measured in blood plasma of migratory Peregrine Falcons captured on the Texas coast as they departed and returned to the U.S. during migration, Henny et al. (1982) concluded DDE at that time was largely accumulated during winter in Latin America. This study continued for long-term monitoring purposes and, with the use of satellite telemetry to locate breeding and wintering localities, documented the decrease of DDE in arctic-breeding peregrines from the late 1970s to 1994 (Henny et al. 1996).

Two general types of CH studies continue: (1) long-term monitoring of the productivity and population sizes of species previously in trouble, often with egg or blood-plasma collections for residue analyses, and (2) evaluations of potentially sensitive species based upon diet (i.e., fish or bird-eaters) or at locations with limited information.

POLYCHLORINATED BIPHENYLS (PCBS), POLYCHLORINATED DIBENZO-P-DIOXINS (PCDDS), AND POLYCHLORINATED DIBENZOFURANS (PCDFS)

Chemistry and Toxicology

These related chemicals are released to the environment mainly from industrial and commercial chemical sources. Being relatively persistent and volatile, they have dispersed throughout the global environment where they biomagnify, particularly in aquatic food chains. Some of the highest PCB concentrations in biota have been reported in eagle and falcon species, and thus have been investigated as potential factors in populations of raptors with chronic low productivity. Exposure and effects on wildlife, including raptors, have been

reviewed by Hoffman et al. (1996) and Rice et al. (2003).

PCBs were used for a variety of purposes including manufacture of electrical transformers, and formulation of lubricating and cutting oils, pesticides, plastics, paints, etc. More than a billion kilograms were produced worldwide, with a third having been released into the environment (Tanabe 1988). PCB use has been banned or heavily restricted in most countries since the late 1970s.

Neither PCDDs nor PCDFs are deliberately produced commercially, but they are formed either as by-products during synthesis of other chemicals, such as chlorophenolic herbicides, or during combustion of chlorine-containing materials. Incineration of municipal and industrial wastes is the major global source of dioxins, which can be transported long distances and deposited in soils and sediments (Czuczwa et al. 1984).

The number and position of chlorine atoms determines the chemical and biological attributes of each dioxin, furan, or PCB isomer (Fig. 2). More chlorine atoms generally lead to greater fat solubility and resistance to degradation. The most toxic isomers have chlorines at the 2,3,7,8 (PCDD/Ds) or 3, 3',4,4' (PCBs) positions. Those congeners are more planar in shape and readily bind a cellular protein known as the *Ah* or arylhydrocarbon receptor, which leads to a variety of biological responses.

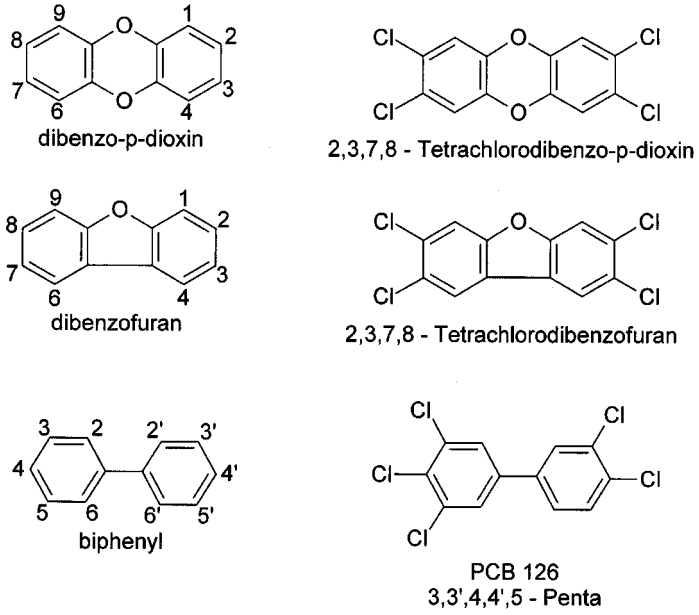

Figure 2. Structure of PCDDs, PCDFs and PCBs.

Toxicity relative to 2,3,7,8-TCDD can be compared using TCDD Toxic Equivalents (TEQs) (Van den Berg et al. 1998). Embryos and growing nestlings are at most risk to TCDD toxicity (Peterson et al. 1993). In laboratory studies, birds of prey including kestrels were less sensitive to PCBs than were quail and chickens (Elliott et al. 1990, 1991, 1997a), but more sensitive than Common Terns (*Sterna hirundo*) (Hoffman et al. 1998). Feeding an environmentally relevant concentration of PCBs to American Kestrels (*F. sparverius*) caused reproductive effects, as well as altered immune and endocrine endpoints (Fernie et al. 2001, Smits et al. 2002). Less persistent congeners, which were less likely encountered in the field, appeared more toxic than the persistent ones.

Toxic effects have been well studied in wild Great Lakes colonial waterbirds. A set of toxic symptoms referred to as GLEMEDS (Great Lakes embryo mortality, edema, and deformities syndrome) has been attributed to exposure to dioxin-like chemicals in gull, tern and cormorant populations (Gilbertson et al. 1991). Bowerman et al. (1994) reported bill deformities in Bald Eagles, but no quantitative relationship between incidence and contaminant exposure. However, kestrel embryos exhibited malformations and edema when eggs were injected with concentrations of PCB-126 at considerably lower levels than measured in Great Lakes Bald Eagles, supporting the contention for dioxin-like chemicals as the cause of observed defects in Bald Eagle chicks (Hoffman et al. 1998).

Despite high concentrations in eggs, it has proved difficult to link PCB concentrations with significant reproductive effects in raptor populations, including peregrines, Ospreys, and accipiters (Newton et al. 1986, Wiemeyer et al. 1988, Peakall et al.1990, Elliott et al. 2001). Statistical associations between productivity and concentrations of PCBs in eggs were found for Bald Eagles. However, the strong intercorrelation with DDE, which showed a greater effect on productivity (Wiemeyer et al. 1984, 1993), was a confounding factor in that and other studies. A more recent analysis of available data for Bald Eagles showed significant associations between productivity and DDE, but not PCBs (Elliott and Harris 2002). In a long-term study of White-tailed Eagles (*H. albicilla*) in Sweden, Helander et al. (2002) found a correlation for PCBs and the incidence of embryo mortality, but not with productivity. Data from Helander et al. (2002), supported by laboratory evidence (Hoffman et al. 1998, Fernie et al. 2001), indicate that PCBs have affected *Haliaeetus* populations in

Figure 3. Osprey (*Pandion haliaetus*) nest on an easily accessible (with U.S. Coast Guard approval) channel marker near a paper mill on lower Columbia River (River Mile 44) downstream of Portland, Oregon. Ospreys nest at regular intervals along some major river systems and bays which can result in strategic or random sampling of eggs or blood for contaminant evaluation *(Photo by J. Kaiser, USGS).*

areas of high exposure; however, effects are difficult to separate not only from DDE, but also from ecological factors such as food supply (Dykstra et al. 1998, Elliott and Norstrom 1998, Elliott et al. 1998, Gill and Elliott 2003, Elliott et al. 2005a).

Sublethal effects of dioxin-like chemicals have been reported in some raptors. In a study of Ospreys breeding on a river in Wisconsin, nestlings grew more slowly at a site contaminated with 2,3,7,8-TCDD from a pulp mill than at uncontaminated sites (Woodford et al. 1998; Fig. 3). Induction of cytochrome P450 liver enzymes (CYP1A) of a type responsive to exposure to 2,3,7,8-TCDD toxic equivalents has been reported in embryos of both Ospreys and Bald Eagles breeding near bleached kraft pulp mills (Elliott et al. 1996a, 2001).

Criteria and Techniques

Collection and analysis of eggs is still the preferred method for investigating exposure of raptors to PCBs and related chemicals. The pros and cons of collecting fresh versus unviable eggs, and the use of the sample egg technique discussed in the section on chlorinated hydrocarbon pesticides apply equally here. Concentrations of PCBs in eggs and related compounds diagnostic of effects, such as embryo survival or overall nest success, have not been defined clearly for most species. Threshold levels for PCBs in eggs were estimated using older analytical methods, such as 40 mg/kg (ww) for peregrines (Peakall et al. 1990). Based on a review and re-analysis of existing data for Bald Eagles, Elliott and Harris (2002) suggested that the reproductive effect threshold was at least 20 mg/kg (ww) total PCBs for Bald Eagles. Helander et al. (2002) determined the lowest observable effect level of 25 mg/kg (ww) (500 mg/kg [lw]) for PCB effects on productivity of White-tailed Eagles. Combining an egg swap design (for more details see Peakall [1987:325] and the discussion below) with regular measurements of chick growth rates, Woodford et al. (1998) suggested a no-observable-adverse-effect-level (NOAEL) of at least 136 ng/kg (ww) for 2,3,7,8-TCCD for the hatchability of Osprey eggs.

Effects of dioxin-like chemicals in birds also can be studied using the technique of laboratory incubation of wild eggs. This approach separates egg-intrinsic effects from adult behavior (egg-extrinsic) and also permits measurement of biomarkers in hatchlings. Using this approach, Elliott et al. (2001) determined a no-effect level for TEQs in Osprey nestlings and a lowest-observable-adverse-effect level of 130 ng/kg (ww) TEQs for hepatic CYP1A induction. Effects of dioxin-like chemicals were studied in laboratory-incubated Bald Eagle eggs (Elliott et al. 1996a) and critical values subsequently recalculated using updated toxic equivalence factors (Elliott and Harris 2002). The results indicate a NOAEL of 135 ng/kg (ww) and lowest-observed-effect-level (LOAEL) of 400 ng/kg for CYP1A induction, and, for embryo toxicity, a NOAEL of 303 ng/kg.

The possible role of dioxin-like chemicals in instances of chronic low reproductive success can be investigated by experimental manipulation of eggs in the field. The logistics of such experiments are complex, given factors such as potential nest abandonment, and the lack of synchronicity in timing of breeding. Embryonic mortality can be caused not only by toxicants within the egg (an intrinsic factor), but also by inadequate parental care caused by the pollutant load (an extrinsic factor), or by a combination of both. These factors can be separated by an egg-exchange experiment between clean and contaminated sites. Adult:Egg combinations in such an experiment (and expected results) include: clean, clean (normal reproduction); clean, contaminated (intrinsic only); contaminated, clean (extrinsic only); and contaminated, contaminated (both intrinsic and extrinsic). For this type of research to be successful, clean and contaminated sites must be

near identical from an ecological perspective, including food availability. Swapping of eggs between treatment and reference sites has provided valuable information in contaminant studies of Ospreys (Wiemeyer et al. 1975), particularly when combined with intensive observation of nesting behavior (Woodford et al. 1998). Nest surveillance, whether directly by an observer or by use of video recording technology, has proved useful in factoring contaminant and ecological variables (Dykstra et al. 1998, Elliott et al. 1998, Gill and Elliott 2003).

Measurement of contaminant levels in blood samples of nestling raptors provides a non-destructive approach, particularly for threatened populations (Elliott and Norstrom 1998, Olsson et al. 2000, Bowerman et al. 2003). Adults also can be trapped either at the nest (Court et al. 1990, Newson et al. 2000), or during migration (Elliott and Shutt 1993), and their blood sampled to assess exposure to PCBs. Diagnostic values for plasma generally are not available, but a value of 189 µg/kg (ww) total PCBs in nestling plasma was suggested as being correlated with 20 mg/kg (ww) in eggs of Bald Eagles (Elliott and Harris 2002).

LEAD

Chemistry and Toxicology

Sources of lead include lead mining, smelting and refining activities, battery-recycling plants, areas of high vehicular traffic, urban and industrial areas, sewage and spoil-disposal areas, dredging sites, and areas with heavy hunting pressure (Eisler 2000). Most of these sources are local, but until recently, lead exposure from spent shotgun pellets and vehicular traffic were much more widespread. Amounts of lead in roadside soils increased as a direct result of the combustion of gasoline containing organo-lead additives. After about a two-decade phase-out, lead additives in gasoline were totally banned in 1996 for on-road vehicles in the U.S. Since 1998, similar regulations were approved in the European Union, progressively restricting and finally banning the use of leaded gasoline in vehicles.

Lead concentrations in livers of Common Kestrels (*F. tinnunculus*) from both rural and city regions of southeastern Spain decreased significantly between 1995–97 and 2001 (Garcia-Fernandez et al. 2005). The U.S. banned the use of lead shot to hunt waterbirds in 1991. Lead shot was similarly banned in the 1990s in Canada, Denmark, Finland, The Netherlands, and Norway, and in portions of Australia and Sweden (see country policies in Miller et al. [2002]). Thus, two widespread sources of lead were eliminated or were in the process of being reduced in many countries, although lead from the earlier use remains in the environment, and lead shot and bullet use continue for other types of hunting in most countries.

Lead modifies the function and structure of kidney, bone, the central nervous system, and the hematopoietic system, and produces adverse biochemical, histopathological, neuropsychological, fetotoxic, teratogenic, and reproductive effects (Eisler 2000). Lead poisoning in raptors has been fairly well documented since the 1970s. Secondary poisoning from consumption of lead-poisoned or shot waterfowl is believed to be the predominant source of lead exposure for wintering Bald Eagles and Golden Eagles (*Aquila chrysaetos*) (Feierabend and Myers 1984). Upland-foraging raptors and scavengers that typically include game birds and mammals in their diet are also at risk for lead poisoning (Kim et al. 1999, Clark and Scheuhammer 2003, Fry 2003, Wayland et al. 2003).

Criteria and Techniques

Depending on its severity, lead poisoning causes specific clinical signs including depression, foul-smelling breath, lime green feces, nonregenerative anemia, vomiting, diarrhea, ataxia, blindness, and epileptiform seizures (Gilsleider and Oehme 1982). Subclinical or chronic lead exposure usually decreases the ability to hunt and predisposes raptors to injury from environmental hazards such as vehicles, power lines, etc., which could partially explain why many raptors were admitted to rehabilitation centers with miscellaneous trauma (Kramer and Redig 1997). Blood-lead concentrations between 0.2–0.6 mg/kg (ww) were classified as subclinical lead exposure and birds with concentrations between 0.61–1.2 mg/kg classified as clinical (treatable) lead poisoning. Blood-lead concentrations >1.2 mg/kg were invariably associated with death (Kramer and Redig 1997). Blood parameters (g-aminolevulinic acid dehydrase [ALAD], hematocrit, proporphyrin, hemoglobin) have been used in field studies. ALAD inhibition of 80% is often associated with decreased hemoglobin and hematocrits (see references in Henny 2003). Lead-poisoning categories in livers based upon Pain (1996) include: <2 mg/kg (ww) (background), 2–5.9 mg/kg (subclinical), 6–15 mg/kg (clinical) and >15 mg/kg (severe clinical).

Lead poisoning has been documented in at least 14 species of raptors that eat or scavenge prey containing lead shot or bullets (including hunter-wounded birds and mammals). These include California Condor (*Gymnogyps californianus*), Andean Condor (*Vultur gryphus*), King Vulture (*Sarcoramphus papa*), European Honey Buzzard (*Pernis apivorus*), Bald Eagle, White-tailed Eagle, Steller's Sea Eagle (*H. pelagicus*), Western Marsh Harrier (*Circus aeruginosus*), Red-tailed Hawk (*Buteo jamaicensis*), Roughleg (*B. lagopus*), Golden Eagle, Prairie Falcon (*F. mexicanus*), Peregrine Falcon and Great Horned Owl (*Bubo virginianus*) (Locke and Friend 1992, Pain et al. 1994, Kim et al. 1999, Eisler 2000, Clark and Scheuhammer 2003). Most available information was reported from U.S., Canada, Europe and Japan. That most raptors regurgitate pellets (i.e., undigested bones, fur, feathers, and often lead shot) definitely reduces their exposure to lead. Shot has been reported in field-collected pellets, and a laboratory study with five Bald Eagles showed that of 196 shot ingested, only 18 were retained at death, with a median retention time of 2 days (Pattee et al. 1981). Based on these and other findings, Henny (1990) concluded that without pellet casting, the Bald Eagle probably would have become extirpated because of lead poisoning in portions of its range a hundred years ago, long before the lead problem was understood. Thus, lead poisoning could have been much more serious for raptors than it has been.

To avoid various threats, particularly lead poisoning, all remaining California Condors were brought into captivity in 1987. Release of captive-propagated condors began in 1992 and, despite extensive efforts to reduce incidental exposure of condors to lead ammunition fragments, they continue to suffer from acute lead poisoning (Meretsky et al. 2000, Fry 2003). California Condors feed mainly on soft tissues, rarely ingesting bones, hair or feathers (Snyder and Snyder 2000), and thus not only reduced the need to cast pellets, but also increased exposure to ingested lead fragments. Lead is a problem not only in the U.S., but also worldwide. During the winter of 1998–1999 in Hokkaido, Japan, 16 Steller's Sea Eagles and 9 White-tailed Eagles died of lead poisoning after consuming sika deer (*Cervus nippon*) remains containing lead-bullet fragments (Kurosawa 2000).

Lead poisoning of raptors from mining sources has been studied at the Coeur d'Alene (CDA) lead mining and smelting complex in northern Idaho, U.S.A. (Henny et al. 1991, 1994; Henny 2003). Waterfowl were most affected, due to their consumption of sediment (Beyer et al. 2000). Raptors do not ingest sediment, and most raptors do not digest bones of prey species (a major storage area in vertebrates for lead), thus it became clear why Ospreys, hawks and owls in the CDA basin were less contaminated with lead from mining sources than were waterfowl.

MERCURY

Chemistry and Toxicology

Toxicity of mercury to birds was reviewed by Scheuhammer (1987). Toxicity depends on whether mercury is in the organic or inorganic form. Only a small percentage of inorganic mercury is absorbed, but almost all organic mercury is absorbed by the intestine. Biotic and abiotic methylation in nature of inorganic mercury produces methylmercury (MeHg), which fish accumulate from water and their diet; nearly all mercury in fish flesh is MeHg. MeHg can adversely affect developing neural tissue in birds, with fish-eating birds being especially vulnerable.

Historically, mercury was used extensively in gold and silver extraction, in the chlor-alkali industry, in the manufacture of electrical instruments, in pharmaceuticals, in agricultural fungicides, in the pulp and paper industry as a slimicide, and in the production of plastics (Eisler 2000). Other activities that contribute significantly to the global input of environmentally available mercury include the combustion of fossil fuels; mining and reprocessing of copper and lead, runoff from abandoned cinnabar mines; wastes from nuclear reactors, pharmaceutical plants, and military ordinance facilities; incineration of municipal solid wastes and medical wastes; and disposal of batteries and fluorescent lamps (Eisler 2000). Long-range atmospheric transport of mercury has resulted in elevated mercury loadings great distances from source sites, including remote lakes in Canada (Lucotte et al. 1995). Since 1985, mercury has accumulated in flooded soils of the Florida Everglades at a much higher rate than decades earlier. The increase was attributed to increased global and regional deposition, and is similar to increases reported in Sweden and the northern U.S. (Rood et al. 1995). Elevated mercury concentrations have resulted in closing many lakes and rivers to fishing because of human health concerns. In general, the number of mercury-contaminated fish and wildlife habitats has increased progressively. Increased

mercury concentrations in lakes are attributed to increased atmospheric emissions and to acid rain in poorly buffered systems.

Concerns about mercury exposure of raptors were especially high in Europe and North America during the 1960s and 1970s and are again reaching high levels in more recent years. The earlier interest was associated with the agricultural use of alkyl mercury as a fungicide applied as a seed dressing. This killed many seed-eating birds and secondarily poisoned many raptors (Berg et al. 1966, Jenson et al. 1972). Alkyl mercury was introduced around 1940 and was banned as a seed dressing in Sweden in 1966 (Johnels et al. 1979). Most mercury issues have been associated with aquatic systems and species, but the fungicide use resulted in exposure of upland species, including Eurasian Sparrowhawk, Common Buzzard (*B. buteo*), Merlin (*F. columbarius*), and Common Kestrel.

Contemporary interest in mercury includes: (1) atmospheric deposition from coal-fired power plants worldwide, especially the Arctic and the northeastern U.S. and adjacent Canada, which contaminates fish stocks and exposes fish-eating wildlife, (2) the Amazon Basin where mining operations annually discharge 90–120 tons of mercury into local ecosystems (Nriagu et al. 1992) affecting local breeding populations of birds, including raptors, and perhaps neotropical migrants (e.g., fish-eating Ospreys that nest in eastern North America), and (3) in many parts of the world, localized historic mining sites for mercury, or where mercury was used to extract gold or silver.

Criteria and Techniques

Mercury monitoring procedures have included eggs, liver, kidneys, whole blood and feathers (we recommend that personnel at the analytical chemistry laboratory wash feathers with a metal-free alkaline detergent to remove adhering particulate matter). Shunting MeHg into growing feathers is an important sequestering process in birds. And indeed, essentially all mercury in blood, eggs, and feathers is MeHg. Feathers from museum specimens have been used to provide a long-term evaluation of mercury exposure, although care needs to be taken about consistency in the specific feathers analyzed. Livers and kidneys of many raptors found dead were routinely analyzed only for total mercury (THg). THg concentrations reported in birds "dying of mercury poisoning" showed considerable variation, e.g., White-tailed Eagles (all mg/kg ww): Finland, liver 4.6 to 27.1, kidney 48.6 to 123.1; Germany, liver 48.2, 91, kidney 120; Baltic Sea, liver 30, 11, 33 (see Thompson 1996). This variability may be associated with the presence of differing ratios of inorganic mercury and the more toxic MeHg. It has been known for some time that birds (especially seabirds) demethylate MeHg (the form readily absorbed and usually ingested) and sequester it in the liver and kidneys in the less toxic inorganic form. Forms of mercury present in the liver and kidneys have been analyzed in recent years and provide better insight into mercury toxicity and sequestration. Recent studies of waterbirds along the Carson River in Nevada (a highly-contaminated historic mining site) revealed interesting aspects of mercury toxico-dynamics in birds and evidence of some histologic effects (Henny et al. 2002). The theoretical "effect criterion" of mercury in eggs is ~ 0.80 mg/kg (ww) (Heinz 1979, Newton and Haas 1988), but see Oehme (2003). Thompson (1996) rightfully implies that no single mercury criterion in eggs applies to all species, which is similar to the species-specific findings reported earlier for CHs.

Perhaps the best approach to monitoring mercury in raptors is to sample whole blood (highly correlated and 1:1 ratio with MeHg in the liver [Henny et al. 2002]), or to sample newly grown feathers of young (all grown about the same time), which are highly correlated with blood concentrations in young at the time of feather growth. Feathers from adults are more complicated and reflect blood concentrations when the feather was grown (which may represent mercury exposure at different locations for a migratory species), or different degrees of depuration (via feathers) depending upon when in the molt cycle the collected feather was grown. Heinz and Hoffman (2003) reported that once a bird begins ingesting elevated levels of mercury in the diet, it only takes a few days before depositing high levels of mercury in its eggs. High levels of mercury also should appear rapidly in both blood and growing feathers.

ORGANOPHOSPHORUS (OP) AND CARBAMATE (CB) INSECTICIDES

Chemistry and Toxicology

When many of the CH insecticides were banned, they were largely replaced by shorter-lived but more toxic cholinesterase(ChE)-inhibiting OP and CB insecticides. The agents comprising this type of insecticide, which have a common mechanism of action, arise from two

different chemical classes, the esters of phosphoric or phosphorothioic acid (OP) and those of carbonic acid (CB) (Ecobichon 1996). These insecticide classes, primarily developed in the 1950s and 1960s, were generally considered non-persistent and non-bioaccumulative, and, therefore, at low risk for raptor secondary poisoning, occurring through eating intoxicated prey. Many OP and CB compounds have high acute toxicity (low amounts kill vertebrates), especially when compared with the CHs, but they do not bioaccumulate or biomagnify up food chains. Their high acute toxicity results in numerous raptor poisonings and deaths. Secondary poisoning of raptors from these acutely toxic chemicals is most likely from exposure to the unabsorbed compound remaining in the gastro-intestinal tract of the prey (Hill and Mendenhall 1980, Hill 1999), which is in contrast to the importance of residual metabolites accumulated in post-absorptive tissues and fat for CHs. Early reports of OP secondary poisoning involving raptors involved Swamp Harriers (*C. approximans*) in New Zealand killed by parathion and fensulfothion (Mills 1973), and about 400 raptors killed in Israel after eating voles and birds poisoned with monocrotophos (azodrin) (Mendelssohn and Paz 1977).

The principal toxicity of OP and CB pesticides is based on disruption of the nervous system by inhibition of ChE activity in the central nervous system and at neuromuscular junctions with death generally attributed to acute respiratory failure (O'Brien 1967). When an OP or CB binds to ChE, a relatively stable bond is formed and prevents the ChE from deactivating the neurotransmitter, acetylcholine. The clinical signs following an acute exposure include lethargy, labored breathing, excessive bronchial secretion (salivation), vomiting, diarrhea, tremors, and convulsions. These toxic indicators are useful when sick animals are found near an area of recent applications, but the signs are not uniquely different from poisoning by other neurotoxins (Hill 2003).

Criteria and Techniques

OP and CB pesticides have resulted in hundreds of incidents of wildlife mortality from disease vector control and agriculture (including forest and range management). When many dead and moribund animals of mixed species are found in an area of known OP or CB treatment, the casual association may be evident but is not conclusive without biochemical and chemical confirmation (Hill 2003). Proper diagnosis depends upon demonstration of brain ChE inhibition consistent with levels indicative of toxicity or exposure and chemical detection of residues of the causative agent. Hill (2003) pointed out that the last step is sometimes difficult because neither OP nor CB residues tend to accumulate in tissues, but that a strong inferential diagnosis is possible by demonstrating inhibited brain ChE activity and "detection" of the anti-ChE agent in either ingesta or tissues.

Normal brain ChE values are obtained from raptors (same species, because normal values are species-specific) not exposed to OPs or CBs and used as a basis for comparison. Some published normal values for North American raptors (10 species of vultures, hawks, eagles, falcons, and owls) are available (Hill 1988), but before using them for comparative purposes, it is critical that observed values be based upon the same methodology. The concurrent running of "controls" for normal values on the same instrument is preferred. Another alternative is to use a suitable reactivation technique to determine the degree of inhibition. In cases of OP poisoning, ChE activity can be reactivated *in vitro* by the oxime 2-PAM (Fairbrother 1996), and for carbamalated ChE (which is less stable) simple *in vitro* heat will serve as a rapid indicator of CB exposure (Hill and Fleming 1982).

A conservative threshold of 50% inhibition in whole brain ChE activity of a bird found dead is generally considered diagnostic of death from anti-ChE poisoning. Even so, 70–95% is commonly reported for birds killed in nature by OP insecticides (Hill 2003). In contrast, when birds are killed by CB pesticides, whole brain ChE activity often is not nearly as inhibited (ChE levels may vary from near normal to only 70% inhibition). Lesser degrees of ChE inhibition may reflect spontaneous postmortem reactivation of the enzyme (Hill 1989), or that death occurred as a result of initial inhibition of the peripheral nervous system and its control of vital functions prior to the brain being completely inhibited. If immediate analysis is not available, store carcasses frozen (preferably at –80ºC) prior to ChE analyses, especially if CBs are suspected. Freezing, however, will hinder the ability to detect other causes of death (e.g., deaths from infectious diseases).

Toxic consequences to raptors from OP and CB applications usually last only a few days, but exceptions do occur. Treatment of cattle with the OP, famphur (poured directly on back of cattle with ladle), kills warble larvae in the blood stream. Black-billed Magpies (*Pica pica*) died several months following application of famphur, and hawks and owls also died from second-

Figure 4. Bald Eagle (*Haliaeetus leucocephalus*) feeding on duck carcass in the Fraser River Delta of British Columbia, Canada. A single duck carcass can attract many Bald Eagles and other raptors, and is a prime vector of insecticides to birds of prey in that environment *(Photo by S. Lee, CWS).*

Figure 5. Juvenile Bald Eagle (*Haliaeetus leucocephalus*) from Fraser River Delta, British Columbia, Canada, with symptoms of anticholinesterase poisoning. Note dilated pupils, clenched talons and the inability to stand. The bird died 2 days later of phorate poisoning *(Photo by J. Elliott, CWS).*

ary exposure (Henny et al. 1985). Unabsorbed famphur persisted on cattle hair (sampled at weekly intervals) under field conditions for at least 3 months, and magpies that ingested cattle hair died. One Red-tailed Hawk that consumed a magpie died from secondary poisoning 10 days after cattle treatment and another was found incapacitated about 13 days after treatment with blood plasma ChE inhibited 82%.

Plasma ChE, which is more variable than brain ChE, can be used to measure exposure by comparing the observed value to a norm for the species. Caution is indicated for diagnostic use of plasma ChE, because this source of non-specific ChE is more labile and prone to dissociation from inhibitor than is brain ChE (Hill and Fleming 1982). However, acute exposure to potentially lethal levels, at least of OPs, resulted in complete inhibition of plasma ChE activity in many Bald Eagles and other raptors (Elliott et al. 1997b, J. E. Elliott, unpubl. data).

Prior to the famphur study in 1982–83, raptors were not routinely evaluated for OP or CB poisoning. When testing was initiated between March 1984 and March 1985, eight Bald Eagles, two Red-tailed Hawks, and one Great Horned Owl were identified as killed by OP pesticides including fenthion and famphur (Henny et al. 1987). In 1989 and 1990, secondary poisoning of Bald Eagles and Red-tailed Hawks was documented in the Fraser River delta in British Columbia, Canada (Elliott et al. 1996b). Crop contents of the dead raptors, which contained mainly duck parts, included the granular insecticides, carbofuran and fensulfothion (Fig. 4). Elliott et al. (1996b) concluded that enough granular insecticide persists in the low pH conditions of the delta to cause waterfowl kills and secondary poisoning of raptors several months after application, which was supported by subsequent research (Wilson et al. 2002). In 1992–94 additional Bald Eagles and a Red-tailed Hawk in the same area died from phorate, another granular OP insecticide (Elliott et al. 1997b; Fig. 5). Dead eagles usually were found at roost sites rather than in agricultural fields. Persistence of granular formulations causing secondary poisoning is likely not confined to the Fraser River delta, as a similar scenario involving carbofuran poisoning of several hundred waterfowl and some raptors was reported from California (Littrell 1988).

Under laboratory conditions, 14 American Kestrels were presented with House Sparrows (*Passer domesticus*) dermally exposed to Rid-A-Bird (11% fenthion active ingredient). All kestrels died within 3 days (Hunt

et al. 1991). In another scenario, Red-tailed Hawks wintering in orchards in central California were dermally exposed to several dormant-season OP sprays (Hooper et al. 1989, Wilson et al. 1991).

Consumption of freshly sprayed insects by raptors can lead to mortality as well. Large numbers of Swainson's Hawks (*B. swainsoni*) from North America died following grasshopper control in Argentina (Woodbridge et al. 1995). During the 1995–96 austral summer, as many as 3,000 individuals were killed in a single incident and at least 18 different incidents were witnessed totaling about 5,000 Swainson's Hawks (Canavelli and Zacagnini 1996). The OP monocrotophos (first associated with raptor deaths in Israel [Mendelssohn and Paz 1977]) was responsible for the Swainson's Hawk deaths. For additional incidents, see the overall compilation of raptor poisonings by OPs and CBs with emphasis on Canada, U.S., and the U.K. (Mineau et al. 1999), and from the U.S. in 1985–94 (Henny et al. 1999). Raptor poisonings have been frequent under current OP and CB use practices (Henny et al. 1999), although only a few products and formulations have been responsible for most of the incidents.

A high proportion of raptor-poisoning cases in the U.K. resulted from deliberate misuse or abuse of OPs and CBs, whereas the proportion of deliberate poisonings was smaller in North America where problems with labeled uses were as frequent as abuse cases (Mineau et al. 1999).

VERTEBRATE-CONTROL CHEMICALS

Chemistry and Toxicology

A variety of chemicals has been used to control mammal, particularly rodent, and bird populations in urban and agricultural situations. The risk of secondary poisoning of raptors can be high, as many raptor species prey on rodents or other targeted small mammals such as ground squirrels, whereas other species are drawn to scavenge carcasses. Secondary poisoning of raptors has been reported for strychnine (Reidinger and Crabtree 1974) and anti-coagulants (Hegdal and Colvin 1988, Newton et al. 1990, Stone et al. 1999). Chemicals such as sodium monofluoroacetate (Compound 1080) are registered in some jurisdictions for control of livestock predators, whereas CH and anticholinesterase insecticides in particular have seen widespread illegal use for predator control in many countries, and have poisoned many raptors directly or secondarily (Mineau et al. 1999).

Strychnine is a convulsant that works by lowering the stimulation threshold of spinal reflexes. It is toxic to birds at low concentrations, with LD_{50}s ranging from 2.0 to 24.0 mg/kg (ww). The Golden Eagle LD_{50} is 4.8 to 8.1 mg/kg (Hudson et al. 1984). Strychnine was widely used in North America in grain baits to control small mammals, including prairie dogs that were considered pests in range and forestlands. Aboveground use was banned in 1983 by the EPA based on secondary poisoning concerns for listed species.

Anti-coagulants now dominate rodent control worldwide. They function by interfering with the action of Vitamin K-dependent clotting factors in the liver, killing the animal via fatal hemorrhaging. The first generation of 4-hydroxy coumarin-based anticoagulants is typified by warfarin, widely used since the 1940s, but to which rodents became resistant in many areas. Second-generation products, such as difenacoum, bromadialone, and brodifacoum, were subsequently developed. These chemicals are used widely around farm buildings, food storage facilities and in urban settings to control commensal rodents. They have greater potency than the first-generation versions, and also are more persistent and toxic to non-target species. Field and forestry use of second-generation anticoagulants has increased and replaced other poisons such as 1080 and zinc phosphide (Eason et al. 2002).

The hazard to wildlife posed by anticoagulants has been known for some time (Mendenhall and Pank 1980, Townsend et al. 1981). Duckett (1984), for example, reported anticoagulants causing a population collapse of Barn Owls (*Tyto alba*) in Malaysia. A field study in Virginia found that attempts to control orchard voles with brodifacoum resulted in the death of at least five radio-tagged Eastern Screech-Owls (*Megascops asio*) (Hegdal and Colvin 1988). Newton et al. (1990) found that 10% of Barn Owls found dead in Britain contained residues of difenacoum or brodifacoum in their livers, and that exposure to those compounds posed a potential threat to populations. Stone et al. (1999) reported 26 raptors that died from hemorrhage with hepatic residues of anticoagulants, principally brodifacoum, but including warfarin, diphacinone and bromadialone. Secondary ingestion was the presumed source, and Great Horned Owls (13 cases) and Red-tailed Hawks (seven cases) were the most often poisoned species, although a variety of other raptor species were affected.

Brodifacoum, in particular, has been used to

remove rats from island seabird colonies in various locations, and thus posed a risk to raptors and scavengers. Bald Eagles were exposed to brodifacoum, but with no evidence of adverse effects during a successful rat-eradication program on Langara Island, British Columbia (Howald et al. 1999). Swamp Harriers were among the wildlife poisoned by secondary ingestion of brodifacoum during rat-control projects on islands in New Zealand (Eason et al. 2002).

Techniques

Programs to routinely monitor raptor debilitation and mortality following "vertebrate-control operations" can provide valuable information on incidence of exposure and poisoning (Newton et al. 1990, Stone et al. 1999). Considerable variation exists in avian sensitivity to different anticoagulants and among species to each chemical, which makes it difficult to determine diagnostic liver residue concentrations. Brodifacoum appears to pose a particular risk to raptors not only due to its greater toxicity in general, and to some owls in particular (Newton et al. 1990), but also because of its greater persistence and widespread use. Finding of any residues of brodifacoum in livers of raptors is cause for concern and an indication of potentially lethal exposure of local populations. More intensive monitoring methods, including live-capture for blood sampling of residues and clotting times, and telemetry of raptor populations at risk may be indicated in specific circumstances (Colvin and Hegdal 1988, Howald et al. 1999).

ROTENONE AND OTHER PISCICIDES

As many as 30 piscicides have been used extensively in fisheries management in the U.S. and Canada since the 1930s. Today, only four are registered for general or selective fish control or sampling (Finlayson et al. 2000). The general piscicides include antimycin and rotenone (most extensively used in the U.S.). Lampricides include lamprecid and bayluscide. Rotenone is a naturally occurring substance derived from the roots of tropical plants in the bean family (Leguminosae). It has been used for centuries to capture fish in areas where these plants are found naturally. Rotenone inhibits a biochemical process at the cellular level, making it impossible for fish to use the oxygen absorbed in the blood and needed for respiration (Oberg 1967).

Fisheries managers in North America began to use rotenone for fisheries management in the 1930s. By 1949, 34 states and several Canadian provinces used rotenone to manage fish populations. The piscicide was applied first to ponds and lakes, and then to streams in the early 1960s (Schnick 1974). Finlayson et al. (2000) reported that rotenone residues in dead fish are generally very low (< 0.1 mg/kg [ww]) and not readily absorbed through the gut of the animal eating the fish. While secondary toxicity of rotenone by a fish-eating bird or mammal does not appear to be an issue, the loss of food supply following rotenone treatment of a lake has been shown to reduce reproductive success of fish-eating raptors and loons. Bowerman (1991) reported significantly lower Bald Eagle production rates in Michigan at inland breeding areas treated within 3.2 km of nests for rough fish removal during the treatment year and 2 years following compared to the same sites in non-treatment years (0.57 vs. 1.30 young per occupied nest). Production was even more reduced when treatment locations were within one km of nesting sites (0.39 vs. 1.31). At most lakes in Michigan, fish were manually removed and not killed with rotenone. California mitigated an impact to nesting Bald Eagles by transferring eggs from a nest to an approved eagle recovery program (California Department of Fish and Game 1991). Similarly, Oregon provided supplemental salmon for a pair of Bald Eagles nesting at Hyatt Reservoir in 1990 following rotenone treatment in the fall of 1989; the pair produced one young (J. L. Kaiser, pers. comm.). Michigan mitigated impacts on loons by delaying treatments until chicks fledged (Finlayson et al. 2000).

Ospreys were studied in Oregon associated with an operational use of rotenone. Nesting populations at Hyatt Reservoir (the treatment) and Howard Prairie Reservoir (the control) were studied for two years before application (Henny and Kaiser 1995). Production rates (young/occupied nest) in 1988 and 1989 were similar at both Hyatt (1.48 and 1.44) and Howard Prairie (1.50 and 1.50). Rotenone was applied in autumn 1989 (after Osprey departure) and nesting numbers did not change appreciably in 1990 at Hyatt (11 nests) with no fish present (not yet restocked) or at Howard Prairie (29 nests). Productivity in 1990 was higher at the control reservoir (2.07), and lower at the treatment reservoir (1.00) (C. J. Henny and J. L.Kaiser, unpubl. data), and correlated with low prey delivery rates at Hyatt. Several young died shortly before fledging at Hyatt in 1990, and more days were required to fledge at Hyatt in 1990, which implies food shortages and a slower growth rate. As in the Michigan Bald

Eagle study, production rates at Hyatt Reservoir were depressed in the second and third years after fish removal (0.55 and 1.09 young/occupied nest in 1991 and 1992).

Magnitude of the rotenone effect seems to be related to two factors: (1) the distance to alternative sources of fish, and (2) the timing of the restocking program. After treatment and restocking with game fish, foraging must change to a different cohort of fish (e.g., trout or bass) that are likely less abundant, and more difficult to capture. Bullheads, suckers and chubs, the usual target species of rotenone operations, are usually abundant, prefer shallow water and are slow-moving (i.e., fish characteristics preferred by Ospreys).

EMERGING CONTAMINANTS

Polybrominated Diphenyl Ethers (PBDEs)

The group of chemicals termed persistent organic pollutants (POPs), which includes "legacy" contaminants such as CH pesticides and PCBs, have certainly posed the most serious threat to raptors, including global population declines. Many POPs-type chemicals are considered important in a variety of commercial applications with large quantities of some compounds continuing to be produced. Polybrominated diphenyl ethers (PBDEs) are widely used as flame-retardants in plastic and textile products. PBDEs can affect thyroid hormone and neuronal systems in laboratory animals (Danerud et al. 2001, Danerud 2003) and persist, bioaccumulate and biomagnify in predatory fish, mammals and birds in many ecosystems (de Wit 2002). PBDE residues were reported in Swedish raptors (Jansson et al. 1993), and a variety of isomers (including supposedly non-accumulative types) have been reported in eggs of Peregrine Falcons from Sweden (Lindberg et al. 2004). The eggs of Little Owls (*Athene noctua*) in Belgium collected in 1998–2000 contained PBDEs (Jespers et al. 2005). PBDEs also were found in Osprey eggs from Maryland and Virginia in 2000 and 2001 (Rattner et al. 2004), and from Washington and Oregon in 2002–2004 (C. J. Henny, unpubl. data). Osprey eggs collected between 1991 and 1997 along major rivers in British Columbia had PBDE concentrations that increased 10-fold over that time period, raising concerns over possible health effects if increases continued (Elliott et al. 2005b). Hydroxylated PBDE metabolites, including known thyroxine mimics, recently were reported in blood samples of Bald Eagle nestlings from British Columbia and California (McKinney et al. 2006; Fig. 6).

Figure 6. Juvenile Bald Eagles (*Haliaeetus leucocephalus*) at a nest in the Fraser Valley of British Columbia, Canada. Pre-fledging birds weigh about 4 kg and can provide a large quantity of blood for measurement of contaminants and biomarkers without any adverse effects. Sampling should be scheduled when young are about 6 weeks of age *(Photo D. Haycock, CWS).*

Kestrels hatched from eggs injected during incubation with a mixture of PBDEs at a concentration of 1500 ng/g (ww) intended to simulate exposure of Great Lakes Herring Gulls (*Larus argentatus*) exhibited some effects on retinol, thyroid, and oxidative stress parameters (Fernie et al. 2005).

Sulfonated Perfluorochemicals

Perfluoroactane sulfonate (PFOS) was the active ingredient in Scotchguard™ stain and water repellents; perfluoroactanoic acid was used in manufacture of Teflon® and related coatings. In 2000, 3M Corporation committed to eliminate all PFOS use in Scotchguard™ by 2002, while the use of related compounds is undergoing EPA review. These compounds are present as complex mixtures of fluorine atoms substituted on carbon-carbon bonds, which have presented a challenge to the analytical chemist. They have been shown to be persistent and

widely transportable in the environment. There is evidence that structurally similar chemicals affect a variety of biological processes including endocrine function. Blood samples of Bald Eagles from various locations in the U.S. had substantial amounts of PFOS, as did livers of White-tailed Eagles from Poland and Germany (Kannan et al. 2001, 2002). PFOS also were found in Osprey eggs from Chesapeake Bay (Rattner et al. 2004). No data are available to determine whether these chemicals are having a significant effect on wild birds.

Diclofenac

In addition to vultures, which do so regularly, many raptor species scavenge dead prey during periods of inclement weather or when normal prey are scarce. Eagles and buteos, in particular, have been lethally exposed to a wide array of contaminants, particularly lead and various pesticides, from scavenging, as documented elsewhere in this report. As obligate scavengers, vultures are at particular risk of exposure to many chemicals. During the 1990s, catastrophic declines in populations of *Gyps* vulture species took place on the Indian subcontinent (Prakesh et al. 2003). A comprehensive investigation of the causes of mortality in the White-rumped Vulture (*Gyps bengalensis*) in Pakistan identified the main factor as renal failure caused by exposure to diclofenac, a non-steroidal anti-inflammatory drug (Oaks et al. 2004). Diclofenac was readily available in the region and widely used to treat hoofed livestock. Vultures appear to consume the drug while feeding on treated livestock, the carcasses of which are typically left for scavengers. There is further evidence that diclofenac also is the major cause of vulture decline in India and probably across the range of the impacted species (Green et al. 2004). Efforts to restrict or alter the use of diclofenac and similar drugs are presently underway, but may be too late to save the white-rumped and possibly the other vulture species in the wild (Green et al. 2004). In May 2006, a letter from the Drug Controller General (India) indicated that diclofenac formulations for veterinary use in India were to be phased out within three months.

Toxins of Biological Origin

We found no reports of raptors poisoned from toxins in algal blooms although sea eagles and Ospreys in particular, could be at risk. Threats from plant toxins are not confined to marine ecosystems. Beginning in the early 1990s in the southeastern U.S., Bald Eagles were found dying from a nervous system condition referred to as avian vacuolar myelinopathy (AVM), thought to originate from feeding on similarly afflicted American Coots (*Fulica americana*) (Thomas et al. 1998). Recent findings point to a toxin present in cyanobacteria, which grow on the common invasive water plant hydrilla, as the cause of AVM (Birrenkott et al. 2004, Wiley et al. 2004).

Such toxic hazards may occur naturally. Halogenated dimethyl bipyrroles, believed to be of natural origin and structurally similar to products of marine chromobacterium, were found to accumulate in tissues of Bald Eagles and seabirds (Tittlemier et al. 1999). A laboratory dosing study with kestrels found evidence of clinical effects, but concluded that those chemicals did not pose an acute reproductive threat to avian populations (Tittlemier et al. 2003). The increasing perturbation and pollution of ecosystems by exotic species, nutrients and contaminants, along with climatic fluctuations, may increase the future likelihood of similar phenomena.

Newly Registered Chemicals

In addition to the thousands of commercial chemicals presently in use, new products are introduced each year. Many jurisdictions require that all pesticides and pharmaceuticals undergo extensive evaluation for toxicity and environmental fate prior to registration for use (www.epa.gov/opptintr/newchems/pubs/expbased.htm). Concern about the development and use of compounds with endocrine-disrupting properties has prompted extensive new screening requirements and requirements to test other types of commercial chemicals (Huet 2000, Gross et al. 2003). Despite those stringent testing protocols, the increased volume and chemical diversity of new products combined with increasing human populations and economic activity almost guarantees that new chemicals or new usage patterns will pose future environmental threats.

Raptors, and, in particular, scavenging species, face increasing and unexpected threats to their survival from the introduction of new commercial chemicals, despite pre-market testing requirements. From the unpredicted effects of DDE on development of eggshells to the exposure and sensitivity of vultures to diclofenac, most of the ecological consequences of those chemicals would not have been identified even by the current relatively rigorous testing procedures.

CONCLUSIONS

With more chemicals registered each year, raptors are exposed to a seemingly endless number of contaminants. At about the time adverse effects of one contaminant or a group of contaminants diminish (usually following much research and a ban or limitation on its use), other contaminants emerge as problems, and the cycle continues. The diversity of raptors inhabiting the planet, with their many feeding strategies and characteristics, place some species in perilous situations. Some traits help raptors cope with selected contaminants. They include pellet-casting by owls and many other raptors which eliminates much ingested lead shot, and demethylation (by many species, especially adults) of toxic MeHg to a less toxic form. However, other traits make entire species or individual populations exceedingly vulnerable to certain contaminants (e.g., flocking behavior of Swainson's Hawks on wintering grounds in Argentina). Scavenging species, including vultures, and many eagles and buteos, particularly are vulnerable to secondary poisoning by feeding on carcasses contaminated by lead shot, pesticides, and veterinary pharmaceuticals. Populations of some species have recovered from DDT. These include the Osprey, which tolerates humans and is now beginning to nest again in many polluted areas, and is being promoted as an indicator species to monitor the health of large rivers, bays, and estuaries, a role the species initially played many years ago. There is an ongoing need to monitor raptor populations, and to investigate reports of poor productivity or unusual mortality and to report it to appropriate authorities.

Readers of this chapter are on the frontline. Many times initial reports of contaminant issues come from field workers who are studying other aspects of raptor biology. We could cite many examples, but space does not permit. The bottom line is that raptor biologists need to remain vigilant.

ACKNOWLEDGMENTS

We appreciate the review of an earlier draft of this chapter by Elwood F. Hill and D. Michael Fry.

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