

A Review and Assessment of Spent Lead Ammunition and Its Exposure and Effects to Scavenging Birds in the United States

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1 Introduction

Lead is a naturally occurring and highly toxic element that has no known biological function. Lead can affect all body systems due to its ability to compete with calcium for binding sites, disrupting calcium-mediated functions such as cell to cell communication, cell division and communication, and organization of the cytoskeleton (Goyer and Clarkson 2001). Lead poisoning has been documented in humans for at least 2500 years and in waterfowl from spent lead shot for over 100 years (Grinnell 1894; Hough 1894; Eisler 2000). In that time, the ecotoxicological properties of lead have been extensively reviewed (Eisler 2000). Today lead is primarily used in the manufacture of storage batteries, alloys, pigments and chemicals, and in ammunition.

Wildlife can be exposed to lead from numerous sources, including mining and smelter emissions, lead-based paint, lead fishing sinkers, and spent ammunition. Incidental mortality from waterfowl hunting reached population-level effects when over two million ducks and geese (~2 % of all waterfowl) were poisoned annually by ingestion of spent lead shot deposited in sediments (Bellrose 1951). Later, effects to bald eagles (*Haliaeetus leucocephalus*) preying upon lead exposed waterfowl were documented (Griffin et al. 1980; Pattee and Hennes 1983). To alleviate this problem, between 1986 and 1991 the United States Fish and Wildlife Service (USFWS) phased-in a nationwide restriction on the use of lead shot for hunting waterfowl and American coots (*Fulica Americana*; USFWS 1986, 1995). While this ban resulted in a reduction of exposure in waterfowl (Anderson et al. 2000), lead shot and rifle bullets are still widely used for hunting upland birds and large and small game animals. Therefore, lead poisoning from the ingestion of ammunition and fragments persists in some groups of avian species. In addition to waterfowl, lead exposure and poisoning has been reported in a variety of avian species in the United States, including those protected under the Migratory Bird Treaty Act, the

Endangered Species Act, and the Bald and Golden Eagle Protection Act (Bellrose 1959; Pain et al. 2009). This review focuses specifically on scavenging avian species exposed to spent lead via foraging habits.

2 Objectives and Scope

2.1 Objectives

There are three objectives of this review:

1. To ascertain the contribution of ammunition as a source of lead exposure and its contribution to effects, including mortality, to scavenging birds.
2. To examine whether there are viable pathways of exposure for scavenging birds to other environmental sources of lead.
3. To assess the toxicity of other metals that can be used in ammunition to birds.

For this review, the specific focus is a comprehensive evaluation of the scientific evidence regarding lead exposure and poisoning of scavenging birds from ammunition and other environmental sources. Throughout this review, except where otherwise noted, “poisoning” and “toxicosis” are defined as manifestations of adverse effects that can be observed or measured, up to and including death. The regulation of lead ammunition is discussed to provide the evidence that ultimately resulted in the current ban for waterfowl hunting in the United States and to describe the allowable uses of lead ammunition that remain. Several recent reviews have also examined the effect of environmental lead to birds, but incorporate additional topics such as fishing tackle, upland game birds and other classes of vertebrates, human health, research directions, suggested regulatory pathways, and exposure to wildlife in other countries (Fisher et al. 2006; Rattner et al. 2008; Johnson et al. 2013; Haig et al. 2014).

2.2 Geographic Scope

Exposure to lead ammunition is not unique to scavenging bird species found within the United States; the issue has been documented worldwide (e.g., *Germany*: Nadjafzadeh et al. 2013; *Poland*: Komosa and Kitowski 2008; *South Korea*: Nam and Lee 2009; *Spain*: Mateo et al. 2001; Fernandez et al. 2011; *Sweden*: Helander et al. 2009; *United Kingdom*: Knott et al. 2010). However, the geographic scope of this review is limited to the United States. Whereas much of the information reviewed regarding toxicological information can be applied broadly, narrowing the scope to the United States allows for a more detailed review of the availability of ammunition and other sources of lead in the environment. Some of the species

considered herein migrate across borders, such as the bald eagle into Canada. The regulation of lead shot in Canada differs in that it was banned for hunting most migratory bird game species in 1999 and is currently prohibited for all hunting in National Wildlife Areas (reviewed in Scheuhammer and Thomas 2011). Numerous studies have examined lead exposure of scavenging birds in Canada (e.g., Langelier et al. 1991; Elliott et al. 1992; Miller et al. 1998, 2000, 2001a, b; Wayland et al. 1999, 2003). Where examples from outside the United States can be used to enhance a discussion, they have been included, but are not meant to be comprehensive.

2.3 Taxonomic Scope

For this report, scavenging birds are defined as those which feed on carrion exclusively or almost exclusively (obligate scavengers), or in combination with live prey or other food items. The California condor (*Gymnogyps californianus*), black vulture (*Coragyps atratus*), and turkey vulture (*Cathartes aura*) comprise the obligate scavengers found in the United States. Birds that regularly scavenge on carrion in addition to other dietary items include the bald and golden (*Aquila chrysaetos*) eagle, crows and ravens (*Corvus* spp.), and the Audubon's crested caracara (*Polyborus plancus audubonii*) which has a limited range in the United States. Many other avian species, including *Accipiter* and *Buteo* hawks, may scavenge carrion opportunistically. Obligate and regular scavengers are the focus of this review due to the tendency of these birds to be exposed to and affected by lead, as inferred from the greater abundance of documented poisoning cases. California condors and bald eagles are disproportionately represented in some sections as they are particularly well studied relative to other scavengers and may be more apt to be collected due to their large size, conspicuous plumage, and special protection status. Where literature concerning other species is available, including those which feed only opportunistically on carrion, it is described and incorporated into the analysis.

3 Toxicological Effects of Lead in Birds

Lead has no known beneficial role in biological systems and its adverse effects have been detected in birds for over a century. In North America, mortality due to lead poisoning from the ingestion of lead shot was first reported in waterfowl in 1894 in Texas and North Carolina, and by the 1950s an estimated 2–3 % (1.6–2.4 million) of waterfowl across all North American flyways were dying annually of lead shot poisoning (Grinnell 1894; Hough 1894; Bellrose 1959; Anderson et al. 2000). The early accounts by Grinnell (1894) and Hough (1894) include the first descriptions of gross toxicological effects of lead poisoning in wild birds in the United States. Wetmore (1919) reviewed clinical signs and lesions of lead poisoning in waterfowl and reported the results of an experimental study of lead shot poisoning in ducks.

The study showed that mortality varied in mallards (*Anas platyrhynchos*) dosed with one to three #6 lead shot, but six #6 shot were always fatal. Similar findings were noted in northern pintails (*Anas acuta*) and redheads (*Aythya americana*) (Wetmore 1919). Shillinger and Cottam (1937) reported that the frequency of lead shot detection in several thousand gizzards from various species of ducks ranged from 1 to 39 %, depending on the species, and suggested that lead poisoning may be an important factor in the decline of waterfowl populations. In an early study of a variety of waterbird species, lead poisoning was the third largest cause of mortality noted in 3000 carcasses, and the authors frequently observed poikilocytosis (abnormal shape), anisocytosis (unequal size), and reduced hemoglobin content of red blood cells (Quortrup and Shillinger 1941). These early studies laid the groundwork for a more comprehensive investigation into the toxicological effects of lead in birds.

3.1 *Physiological Effects*

When metallic lead is ingested by birds, the stomach's acid and grinding action in species with a muscular gizzard begin to dissolve it, resulting in the formation of toxic lead salts. As these salts are absorbed in the intestinal tract, lead enters the bloodstream and measurable increases in blood lead concentrations occur within hours (Roscoe et al. 1979). The first measurable physiological effect of lead exposure is the inhibition of delta-aminolevulinic acid dehydratase (ALAD), an enzyme necessary for hemoglobin synthesis and a very sensitive indicator of lead exposure in birds (Finley et al. 1976). Birds can tolerate considerable reductions in ALAD activity without adverse hematological effects, but ALAD depression by high levels of lead results in anemia characterized by lowered hemoglobin and hematocrit (Franson et al. 1983; Pain and Rattner 1988). Lead also inhibits ferrochelatase (heme synthetase), an enzyme responsible for combining ferrous iron and protoporphyrin IX (PPIX) to form heme. Lead shot dosing studies with mallards and canvasbacks (*Aythya valisineria*) have shown that blood lead and PPIX concentrations may remain elevated, and ALAD activity may remain depressed, for several weeks to as long as 3 months (Finley and Dieter 1978; Roscoe et al. 1979; Franson et al. 1986). Inhibition of ferrochelatase results in the accumulation of PPIX in the erythrocytes, and its quantification in blood samples has been used as an indicator of lead exposure in birds (Roscoe et al. 1979; Franson et al. 1996).

Lead competes with calcium for binding sites within the body, and can sometimes bind with greater affinity than calcium. This disruption in calcium metabolism can result in neurologic and neuromuscular effects via induction or inhibition of neurotransmitter release, alteration of channels or pumps, and interference with protein kinases (Peraza et al. 1998). Learning and behavioral deficits have been linked to these changes in intra- and extra-cellular signaling (Bressler and Goldstein 1991). Neurotoxic effects, including those on learning and memory, have also been observed in birds. In a series of laboratory experiments with young common terns

(*Sterna hirundo*) and herring gulls (*Larus argentatus*), lead was found to exact behavioral changes on a number of parameters relevant to a chick's survival in the wild (e.g., locomotion, begging behavior, individual recognition, balance, depth perception, behavioral thermoregulation; Burger and Gochfeld 2000). When these tests were repeated in the field, lead-injected chicks showed similar behavioral deficits and had a higher susceptibility to predation (Burger and Gochfeld 2000). High doses of lead can also disrupt the blood–brain barrier in immature animals allowing the entrance of molecules, water, and ions otherwise excluded, leading to cephalic edema, a condition observed in lead poisoned geese (Locke and Thomas 1996).

Other endpoints that may be affected by lead exposure include growth, body and organ mass, feeding activity, reproduction, and immune function. For example, common terns and herring gulls exposed to lead showed a variety of abnormalities, including decreased growth, fledging size, locomotion and balance ability, and decreased feeding activity (Burger et al. 1994). Reduced brain weight has been associated with lead exposure in young mallards, American kestrels (*Falco sparverius*), and European starlings (*Sturnus vulgaris*; Hoffman et al. 1985; Grue et al. 1986; Douglas-Stroebel et al. 2004). Mixed results have been reported in studies of lead effects on immune response. Antibody-mediated immunity was suppressed in Japanese quail (*Coturnix coturnix*) and mallards exposed to lead at levels resulting in other clinical signs (Trust et al. 1990; Rocke and Samuel 1991; Grasman and Scanlon 1995). However, lead ingestion at levels that impaired growth and hematology in Japanese quail did not affect humoral immune response (Morgan et al. 1975). No evidence of immunotoxicity was reported with low-level exposure to lead in Japanese quail and red-tailed hawks (*Buteo jamaicensis*) (Redig et al. 1991; Nain and Smits 2011). Effects of lead exposure on reproduction include lower fertilization rate in ring-necked pheasants (*Phasianus colchicus*), lower egg production in Japanese quail, reduced hatchability in ring-necked pheasants and mourning doves, and smaller clutches and increased nestling mortality in pied flycatcher (*Ficedula hypoleuca*) (Edens et al. 1976; Buerger et al. 1986; Berglund et al. 2010; Gasparik et al. 2012). Testicular changes have been noted in chickens (*Gallus gallus domesticus*), Japanese quail, and ringed turtle-doves (*Streptopelia risoria*) (Morgan et al. 1975; Veit et al. 1983; Mazliah et al. 1989). Decreased mineralization in bones with increased concentrations of lead has been reported in Egyptian vultures (*Neophron percnopterus*) and red-legged partridges (*Alectoris rufa*; Gangoso et al. 2009; Álvarez-Lloret et al. 2014). Bellrose (1959) reported that mallards dosed with lead shot and released were 1.5 times more vulnerable to being shot by hunters than controls.

While some sublethal effects alter health directly, others may render birds more susceptible to causes of mortality such as predation, hunting mortality, collisions with objects, and illness or death from disease. However, it is often difficult to establish a definitive relationship between the detection of elevated concentrations of a contaminant and proximate cause of death. Hunt (2012) argues that population impacts to avian scavengers are likely underestimated, in part due to the difficulty in detecting the health manifestations of sublethal lead exposure. In cases suggestive of lead as a contributing factor, elevated concentrations have been associated

with avian mortality from other causes such as collisions with power lines, cables, or other objects (O'Halloran et al. 1989; Kelly and Kelly 2005; Helander et al. 2009). Elevated lead concentrations were also detected through routine or retroactive screening in a portion of eagles admitted to rehabilitation centers for trauma (Kramer and Redig 1997; Neumann 2009; Nam et al. 2011). However, none of the 1733 bald and 491 golden eagles diagnosed as having succumbed from causes other than lead poisoning (e.g., collision, trauma, electrocution, emaciation, infectious disease) by the National Wildlife Health Center from 1975 to 2013 contained concentrations of lead above background (>1 ppm wet weight in liver; Franson and Russell 2014).

3.2 Clinical Signs of Lead Poisoning

In addition to changes in ALAD and PPIX, signs of lead poisoning vary somewhat among species groups, and include submandibular edema, lethargy, wing droop, ataxia, anorexia, green bile staining of the vent, leg paralysis, and convulsions (Fig. 1; Locke and Thomas 1996; Rattner et al. 2008; Franson and Pain 2011). In a study of lead acetate poisoning in six captive avian species, the most consistent clinical signs across all taxa were weight loss, anemia, and increased concentrations of PPIX (Beyer 1988). Bald eagles dosed with lead shot lost weight and had



Fig. 1 Bald eagle (*Haliaeetus leucocephalus*) at a rehabilitation center displaying clinical signs of lead poisoning, including wing droop and lethargy. Photo courtesy of Kay Neumann, Saving Our Avian Resources, 25494 320th Str, Dedham, IA 51440

reduced hematocrit, hemoglobin, and ALAD activity, as well as changes in serum biochemistries (Hoffman et al. 1981; Pattee et al. 1981).

Birds that die within a few days from an acute exposure to a large concentration or dose of lead may be in good flesh. However, lead poisoning is typically a chronic condition resulting in anorexia, loss of fat reserves, muscle wasting, and debilitation (Locke and Thomas 1996). Time to death in experimental studies varies by species and dosage regimen, with waterfowl generally succumbing within 2–4 weeks, although some raptors survived for more than 15 weeks (Barrett and Karstad 1971; Pattee et al. 1981, 2006; Franson et al. 1986; Beyer et al. 1998). Other gross lesions include impaction of the esophagus, proventriculus, and ventriculus with food (particularly in waterfowl), bile staining of the ventriculus and intestinal contents, distension of the gall bladder with dark green viscous bile, necrosis evidenced by light streaks on the surface of the heart or the cut surface of the gizzard muscle, pale and atrophied internal organs, and flabby heart (Locke and Thomas 1996; Rattner et al. 2008; Franson and Pain 2011; Franson and Russell 2014). In a study of 421 lead-poisoned waterfowl of various species, the most reliable gross indications of lead poisoning were reported to be impactions of the alimentary tract, submandibular edema, necrosis of heart muscle, and bile staining of the liver (Beyer et al. 1998).

Locke et al. (1966) were the first to report inclusion bodies in histologic sections of kidney tissue of lead poisoned birds, in that case mallards. These structures occur within the nuclei of cells in the proximal convoluted tubules of the kidney, and when stained with the Ziehl-Neelson acid-fast technique appear scarlet in color. Other gross and microscopic lesions of lead poisoning are nonspecific and may be observed in association with other conditions, but only lead exposure is known to produce acid-fast intranuclear inclusion bodies in the kidneys of birds. Renal inclusions have been reported in several other species of birds poisoned by lead, including mourning doves (*Zenaida macroura*), rock doves (*Columba livia*), mute swans (*Cygnus olor*), whooper swans (*Cygnus cygnus*), Andean condors (*Vultur gryphus*), turkey vultures, bald eagles, golden eagles, and white-tailed eagle (*Haliaeetus albicilla*; Locke and Bagley 1967; Simpson et al. 1979; DeMent et al. 1987; Ochiai et al. 1992; Kenntner et al. 2001; Carpenter et al. 2003; Pattee et al. 2006; Franson and Russell 2014). However, even though the inclusion bodies are indicative of lead poisoning, they are not present in all cases. Renal inclusions occurred in 64 % of lead poisoned red-winged blackbirds (*Agelaius phoeniceus*), 69 % of brown-headed cowbirds (*Molothrus ater*), 75 % of mallards, 86 % of northern bobwhites (*Colinus virginianus*), and 100 % of common grackles (*Quiscalus quiscula*) and eastern screech-owls (*Otus asio*) that died from lead acetate poisoning (Beyer 1988) and have been infrequently reported in poisoned Canada geese (*Branta Canadensis*; Bagley et al. 1967; Locke et al. 1967; Barrett and Karstad 1971; Sileo et al. 2001). No inclusion bodies were found in bald eagles experimentally dosed with lead, nor observed in 17 lead poisoned eagles (13 bald and 4 golden) submitted to the National Wildlife Health Center (Pattee et al. 1981; Franson and Russell 2014). Additional histopathologic lesions noted in lead poisoned birds include hepatic hemosiderosis, renal tubular cell degeneration, myocardial and gizzard muscle necrosis, fibrinoid necrosis of arterioles, erythroid hyperplasia, encephalopathy, and peripheral neuropathy (Locke and Thomas 1996; Wobeser 1997; Franson and Pain 2011).

3.3 Tissue Distribution and Thresholds for Toxicosis

Lead is distributed throughout the body, including growing feathers, via the circulatory system and a dynamic equilibrium controls deposition and removal in various tissues. Franson and Pain (2011) reviewed the distribution of lead in avian tissues and factors influencing the concentrations of lead in tissues. In general, the highest concentrations are found in bone, liver, and kidney, with intermediate concentrations in brain and blood, and low concentrations in muscle. In birds that survive lead exposure, concentrations in soft tissues will decline over time. Because lead is released from bone far more slowly than from soft tissues, bone functions as a long term repository. Bone lead concentrations may also differ between males and females and among females, depending on season. Finley and Dieter (1978) reported that lead concentrations in femurs of laying mallards were four times higher than in nonlaying females. As calcium is utilized for eggshell formation, intestinal absorption of calcium increases, as well as lead (Krementz and Ankney 1995; Scheuhammer 1996).

Concentrations of lead in blood of live birds and in liver and kidney of dead birds are the tissues commonly used to assess exposure. Concentrations in bone are not a good reflection of recent lead exposure and remain difficult to interpret because of continual accumulation and slow release. Lead concentrations in birds with no history of lead exposure are typically <0.2 ppm wet weight in blood (all blood lead reported herein is wet weight), <2 ppm wet weight in liver and kidney, and <10 ppm dry weight in bone (Table 1; Franson and Pain 2011). Some bird species appear to be more resistant to lead intoxication than others, as indicated by higher lead concentrations in tissues reported in association with lead poisoning. However, suggested guidelines are that lead concentrations >0.2 ppm in blood or >2 ppm wet weight in liver and kidney are evidence of subclinical poisoning, and >0.5 ppm in blood or >6 ppm wet weight in liver and kidney are evidence of clinical poisoning (Table 1; Franson and Pain 2011). Birds with subclinical poisoning are expected to experience physiological effects that are unlikely to severely impair normal biological function and would be likely to recover if lead exposure ceased. Clinical poisoning would likely be accompanied by signs such as anemia, weight loss, and muscular incoordination, and could result in death if lead exposure continued. When monitoring

Table 1 Thresholds for lead toxicosis

Tissue	Background	Subclinical	Clinical poisoning
Blood (ppm, wet weight)	<0.2	0.2–0.5	>0.5
Liver (ppm, wet weight)	<2	2–6	>6
Kidney (ppm, wet weight)	<2	2–6	>6

Note that all tissue concentrations herein have been converted from their original unit to parts per million (ppm) for ease of comparison across studies and against diagnostic thresholds. For measurements taken for blood, the margin of error can be up to 4 % as a result of converting a volumetric measurement to standard SI units. By the nature of the tissue, all blood lead reported is wet weight. Thresholds from Franson and Pain (2011)

reveals elevated blood lead concentrations in live birds, individuals may be given chelation therapy, a treatment used to remove heavy metals. The chelating agent EDTA (ethylenediaminetetraacetic acid) binds lead from soft tissue and bone for excretion (Goyer and Clarkson 2001). Tissues of birds that have undergone recent chelation therapy are likely to have lower concentrations of lead than prior to treatment (e.g. California condor; Rideout et al. 2012), and therefore may not be suitable for diagnostic purposes.

The use of feathers to monitor lead levels can provide a simple, non-invasive method to determine exposure in birds. Although many investigators have measured concentrations of lead in this manner, there have been a limited number of controlled exposure studies to validate this approach and aid in the interpretation of such field data (Kendall and Scanlon 1981; Burger and Gochfeld 1990; Dauwe et al. 2002; Golden et al. 2003). These investigations have shown that under certain conditions feathers can be a reliable indicator of dietary exposure to lead that is associated with lead accumulation in internal organs and biochemical measures of effect. Golden et al. (2003) used ratios of lead concentrations found in these studies as well as those in juvenile birds collected from the field to calculate feather to tissue ratios for nestling or juvenile birds (1:2 for liver, 1:5 for kidney, 1:10 for bone). These ratios, however, may vary based on the specific type of tissue analyzed (e.g., femur versus tibia, primary versus body feather) and in situations of very low or high lead exposure. Golden et al. (2003) also cautioned that adult feathers appear to be a less reliable indicator of endogenous lead exposure, primarily due to complications from external deposition of lead. Feather parts openly exposed to the environment have been found to have higher concentrations of lead than those covered by other feathers and a lack of correlation with concentrations of lead in internal organs (Goede and de Voogt 1985; Goede and de Bruin 1986; Hahn et al. 1993). Rinsing of feathers does not necessarily remove external contamination (Weyers et al. 1988). Dauwe et al. (2002) found evidence that feathers may be also subject to exogenous contamination of lead by excretion from the uropygial gland.

The measurement of lead concentrations in egg content is not known to be particularly useful for monitoring lead exposure in birds. A laboratory experiment with Japanese quail showed that although some lead is transferred to eggs, concentrations were much lower than in the diet (Leonzio and Massi 1989). Field studies with common eiders (*Somateria mollissima*) failed to detect correlations between lead concentrations in eggs and concentrations in feathers or blood of laying females (Grand et al. 2002; Burger et al. 2008). Lead concentrations in eggshells have been measured in several species (Burger 1994; Mora 2003; Dauwe et al. 2005) and may be a suitable indicator of lead contamination (Dauwe et al. 1999).

Wildlife suspected to have died of lead poisoning should be subjected to a complete necropsy to the extent feasible for the condition of the carcass. Although interpretive guidelines are available, lead poisoning as a cause of death should not be distinguished from simple lead exposure based solely on tissue residues. Ideally, a determination of lead poisoning as cause of mortality should be based on an evaluation of field circumstances, observed clinical signs, gross lesions and pathological findings, tissue residues, and when possible, laboratory testing to rule out other

contaminants and infectious or parasitic diseases. However, since birds are generally collected opportunistically and often after death has occurred, observations of clinical signs and comprehensive necropsy findings may not be available due to carcass condition. In these cases, a more conservative diagnostic approach may be warranted. In a study of lead poisoned waterfowl, Beyer et al. (1998) reported that 95 % of fatalities had liver lead concentrations of at least 38 ppm dry weight (10 ppm wet weight), but fewer than 1 % of birds that died of other causes had a concentration that high. The authors concluded that 38 ppm dry weight lead in liver is a defensible criterion for identifying lead poisoning in waterfowl in the absence of pathologic observations.

Key Points: Toxicological Effects of Lead on Birds

- Lead toxicity in birds has been studied in a variety of species and is relatively well understood.
- The progression of lead poisoning can ultimately result in mortality.
- Sublethal effects of lead may render a bird more susceptible to mortality from other causes.
- Lead exposure results in numerous physiological responses that are measurable and specific for diagnostic purposes, but may vary among individuals and species.
- General thresholds have been established in birds to help diagnose lead exposure and poisoning. However, tissue residues can vary and should be interpreted in conjunction with other diagnostic signs, when available.

4 Spent Lead Ammunition in the Environment

4.1 Regulation of Lead Ammunition

Studies conducted in the 1960s further confirmed the toxicity of lead to waterfowl and determined clinical signs such as impairments to normal biological functions, severe weight loss, and mortality (Bellrose 1965; Irby et al. 1967; Locke et al. 1967). Irby et al. (1967) and Locke et al. (1967) examined the effects of lead on mallards dosed with three types of commonly used shot: lead, plastic-coated lead, and lead-magnesium. The study found mortality to 96 % of the mallards dosed with lead, 93 % dosed with plastic-coated shot, and 58 % dosed with lead-magnesium (Irby et al. 1967). The lead-dosed mallards developed anemia, atrophy of adipose and liver tissue, and enlarged gall bladders distended with bile, as well as cellular effects such as hemosiderosis and destruction of the kidney tubule cells, acid-fast intranuclear inclusion bodies, and enlarged nuclei in the cells of the proximal convoluted tubules (Locke et al. 1967). These early studies provided the initial data on the susceptibility of waterfowl to lead poisoning that led to a progression of further studies.

In the early 1980s, an association between bald eagle deaths and lead shot used in waterfowl hunting became apparent. At that time, the bald eagle was listed under the Endangered Species Act. A number of cases determined that bald eagles were exposed to lead by feeding on crippled or non-retrieved hunter-shot waterfowl containing ingested or embedded shot (Griffin et al. 1980; Pattee and Hennes 1983). Between 1967 and 1982, an estimated 7 % of the bald eagle population in the United States was lead poisoned (Pattee and Hennes 1983). In 1986, the USFWS required lead shot in hunting waterfowl and American coots to be phased-out over a 5-year timeframe initiated during the 1987–1988 hunting season (USFWS 1986, 1995). The regulation became a nationwide restriction in 1991 and the USFWS started developing an approval process for nontoxic shot. The USFWS defined that word “non-toxic” as shot that does not cause sickness and death when ingested by migratory birds, and developed testing guidelines to develop and approve new types of shot (USFWS 1997, 2013a, b). More information regarding these testing protocols and the outcome of their use is provided in Sect. 9 of this review.

4.2 Sources of Spent Lead Ammunition Remaining After the Ban

The effectiveness of the 1991 regulations that banned lead for waterfowl hunting in the United States has been evaluated by comparing lead shot ingestion rates before and after the ban. Five years after the ban, a study of 16,651 mallards along the Mississippi Flyway estimated that mortality from lead shot exposure declined 64 %, equating to 1.4 million of 90 million ducks in the 1997 fall flight spared from lead poisoning (Anderson et al. 2000). Although the study indicated a similar overall rate of shot ingestion for these birds (8.9 % post-ban versus 8 % pre-ban; Bellrose 1959; Sanderson and Bellrose 1986), 2/3 of shot ingested were nontoxic. Samuel et al. (1992) and Samuel and Bowers (2000) investigated the effectiveness by studying blood lead concentrations in American black ducks (*Anas rubripes*) in Tennessee before (1986–1988) and after (1997–1999) the ban. Using blood lead concentrations of ≥ 0.2 ppm as an indication of lead exposure, exposure rates were 11.7 % pre-ban and 6.5 % post-ban in adult black ducks. No difference in the exposure rate was detected for juveniles, with differences in food habits or habitat use theorized as a cause. In mottled ducks (*Anas fulvigula*) collected in Texas during the 1987–2002 hunting seasons, lead shot ingestion rates were 7 % along the upper coast and 18 % in those from the central coast (Merendino et al. 2013). This contrasts with an ingestion rate of about 32 % in mottled ducks collected along the gulf coast of Texas from 1973 to 1975, before non-toxic regulations were implemented in the early 1980s (Moulton et al. 1988). A study in Canada reported that bone lead concentrations in ducks declined by about 50 % after the Canadian ban on lead shot for waterfowl hunting (Stevenson et al. 2005).

These studies demonstrate that the lead shot ban was effective at reducing, but not eliminating, lead exposure in waterfowl and birds that prey upon waterfowl.

Lead shot discharged prior to the ban remains on the landscape and is available to be ingested by waterfowl and wading birds unless removed from the environment. Strom et al. (2009) documented lead exposure in trumpeter swans (*Cygnus buccinator*) in the upper Midwest after the lead shot ban. Twenty-five percent of swan mortality from 1991 to 2007 was attributed to lead toxicosis and 39 % of the lead poisoned swans had lead artifacts in their ventriculus at the time of necropsy. The authors suggest that the foraging habitats of swans (digging up sediments to find food) may predispose these species to lead exposure. Bald eagles submitted to the National Wildlife Health Center and diagnosed with lead poisoning significantly increased in all four migratory bird flyways in the United States (Atlantic, Pacific, Central, and Mississippi) after the autumn 1991 ban on the use of lead shot for waterfowl hunting (Russell and Franson 2014). The authors concluded that lead ammunition for use on game other than waterfowl versus the impacts of lead on wildlife populations needs further evaluation.

In addition to the lead remaining from waterfowl hunting, lead ammunition continues to enter the environment from other types of hunting and shooting activities in the United States. Deer hunting is the most popular type of hunting in the United States, with 10.1 million participants nationwide in 2006 (USFWS 2006). At least 3.8 million deer were harvested by resident hunters and 0.7 million deer harvested by non-resident hunters (USFWS 2006). It is common practice to field dress game and discard the internal organs and tissues on the landscape, leaving a lighter carcass to transport out of the field. As a result, each harvested deer may result in an offal pile that may be accessed by scavenging birds and other wildlife. In addition, studies have revealed that a significant number of deer are shot but not retrieved by hunters. Though exact wounding rates are difficult to enumerate and are likely to vary across regions, attempts at estimations have produced similar results: 21–24 % in Illinois, 24 % in Montana, and 17–32 % in Indiana (Stormer et al. 1979; Dusek et al. 1989; Nixon et al. 2001). Other types of hunting also generate substantial participation. Small game such as rabbit, squirrel, pheasant, and quail comprised the next largest group after deer with about 7.5 million hunters followed by turkey (2.6 million) and dove (1.2 million) hunters. Waterfowl hunting accounts for 1.8 million hunters, though no longer contributes to new lead in the environment. Hunting for varmint (coyote, raccoon, fox) and other game (e.g., upland birds and mammals) also takes place and the extent varies by region (USFWS 2006). Poaching of deer and other game species may add to these estimates of wounded prey, carcasses, offal piles, and lead ammunition in the environment, though the magnitude of illegal hunting activity is difficult to evaluate.

4.3 Fragmentation of Ammunition

Modern firearms used for hunting in the United States discharge projectiles of various size and shapes, such as clusters of shot, rifle bullets, and shotgun slugs (Thomas and Guitart 2013). Bullets for hunting are designed to transfer energy from



Fig. 2 The spent remains of the copper jacket and fragmented lead core of a lead-based bullet (*left*) compared with a copper (lead-free) expanding bullet that remained intact. Fragmentation of spent lead ammunition, copper jacket with lead core and pure copper. Photo courtesy of Institute for Wildlife Studies, P.O. Box 1137 Tres Pinos, California 95075

the projectile to the target to quickly maximize power and humanely kill game. This is accomplished by the projectile expanding in diameter when it strikes the animal. For bullets made of frangible metals, like lead, the availability from carcasses and offal left by hunters is greatly enhanced by the tendency to break into small pieces and disperse within a carcass. Several studies have documented that lead-containing bullets fragment and radiate a considerable distance in target animals upon impact. This property makes bullet fragments easily ingested, difficult to avoid when consuming contaminated tissue, and potentially available to multiple scavengers. Fragmentation can also increase the surface area of the ingested material for digestion by stomach acids. Copper bullets, designed to expand into 4–6 frontal petals (or “mushroom”), exhibit much less frangibility and tend to remain intact (as described below; Fig. 2); thus they are less likely to be incidentally consumed than shot or bullet fragments. There exists at least one case of an intact bullet retrieved from an historical California condor nest-site, though evidence suggests that this bullet was fired into the nest rather than having been incidentally ingested by a foraging bird (Snyder et al. 1986).

Fragmentation in Large Game

Several experimental studies of bullet fragmentation in large game, and examination of carcasses and offal piles showed similar patterns of bullet fragment numbers, size, and radiation from the wound site.

Hunt et al. (2006) collected whole or partial remains of white-tailed and mule deer (*Odocoileus virginianus* and *O. hemionus*) killed by hunters with centerfire, breach-loading rifles in California and Wyoming between 2002 and 2004. Thirty-four were killed using copper-jacketed bullets with lead cores, and four with monolithic copper expanding bullets. Local veterinarians radiographed areas of bullet transition, and fragments were counted manually; the presence of metal was verified by dissection in one sample. No attempt was made to distinguish between lead and copper fragments. All whole or eviscerated deer killed with lead-containing bullets (N=24) contained fragments (38–738), with over 100 fragments counted in 74 % of samples. Fragment samples ranged in size about 0.5 to >5 mm, and clusters radiated as far as 15 cm from the wound channel. Ninety percent of offal piles (N=20) contained fragments with counts ranging from 2 to 521. For deer shot with copper bullets, only six fragments in total were found in four whole carcasses, and one fragment in four offal piles. Warner et al. (2014) also observed lead fragmentation in hunter-killed white-tailed deer shot with different firearm types (12 and 20 G shotgun, muzzleloader rifle). Of 25 offal piles examined by radiography, 36 % contained lead fragments, ranging from 1 to 107 per pile (Fig. 3).

Knott et al. (2010) radiographed carcasses and abdominal viscera (stomach, spleen and intestines) of ten red deer (*Cervus elaphus*) and two roe deer (*Capreolus capreolus*) killed by a single shot to the thorax with copper-jacketed lead-core

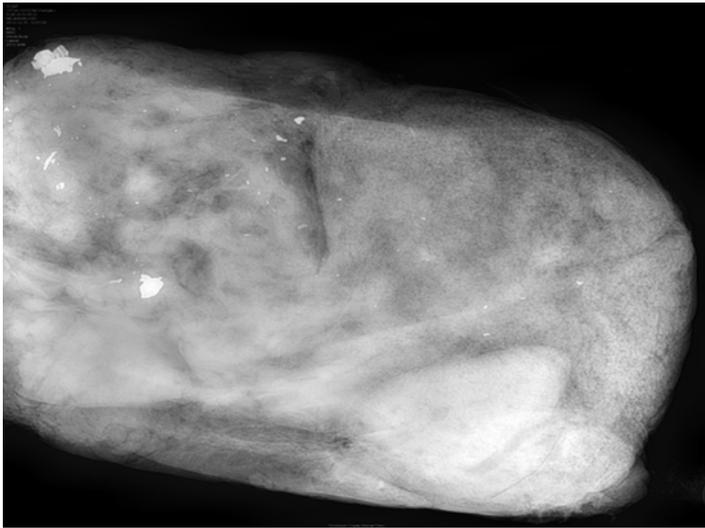


Fig. 3 Radiograph shows lead fragments as white specks in the offal pile from a white-tailed deer (*Odocoileus virginianus*) shot with a .50 caliber muzzleloader in 2012 on the Upper Mississippi River National Wildlife and Fish Refuge. The bullet's intact copper jacket (shown in the upper left corner) mushroomed, exposing the lead core which fragmented into 107 pieces that were spread throughout the offal pile. Photo courtesy of USFWS Upper Mississippi River National Wildlife and Fish Refuge, 7071 Riverview Rd. Thomson IL 61285

bullets. Most of the abdominal viscera were removed from each carcass to mimic local practice and the remaining internal organs (heart, lungs, liver, and kidneys) were retained in the thoracic cavity of the carcass. Fragments on radiographs were compared with images of known lead fragments obtained from copper-jacketed lead bullets fired into a water jug, and bone fragments and grit collected from the study site. The authors found bone and grit to be obviously less opaque than metal fragments and easy to distinguish. An average of 356 metal fragments were visible on radiographs of the carcass and 180 fragments in viscera, accounting for an estimated 17 % of the weight of the bullet. The authors made no attempt to distinguish between copper and lead fragments, but presumed the majority of smaller fragments to be lead due to the larger size of copper fragments produced from firing into water jugs. Fragment counts of carcass and viscera from the same animal were only weakly correlated, and fragments in viscera tended to be smaller: 91 % were <0.01 g as compared to 34 % in carcass. Though mean fragment counts taken from opposite sides of the thoracic area were similar, there was sometimes a substantial difference between counts (a difference of over 300 fragments in one case), suggesting that a single radiograph may underestimate the total number of fragments in a carcass.

Grund et al. (2010) shot euthanized domestic sheep (*Ovis aries*) broadside from a range of 50 m as surrogates for deer. Six types of lead-containing bullets and one copper bullet were studied. Fragments were detected in all carcasses shot with lead-containing bullets, but varied according to bullet type. While all expansion bullets had similar mass and velocity, one of two bullets marketed as “controlled expansion” (i.e., designed to achieve deeper penetration by deforming or “mushrooming” rather than fragmenting) produced fewer fragments and less radiation from the wound site (2–28 fragments per carcass within 25 cm) than other expansion bullets (21–498 fragments per carcass within 45 cm). Similar results were obtained from eight deer shot with rapid expansion bullets as part of a disease management program (Fig. 4). Slugs and muzzleloaders had greater mass and lower velocity than expansion bullets, though still fragmented, albeit to a lesser extent (slugs produced 3–127 fragments, muzzleloaders 1–105 fragments). Lead fragments from these bullet types tended to radiate less. Rinsing of carcasses caused lead to be detected further from the exit wound for all bullet types. Copper bullets produced between 1 and 4 fragments per carcass. The authors concluded that all lead-containing bullets produced fragments, and that hunters could not rely on advertised claims that certain bullets minimized fragmentation and lead deposition into the carcass. The authors also concluded that all meat from deer harvested with lead bullets has the potential to contain at least some lead (e.g., Fig. 4—radiation of lead shot throughout the body cavity).

Dobrowolska and Melosik (2008) collected muscle tissue from wild boar (*Sus scrofa*) and red deer killed by hunters in Poland. Bullet types were selected by hunters and varied, though no attempt was made to establish a relationship between bullet type and lead contamination in their analysis. Elevated lead (>0.3 ppm wet weight, the greatest value detected in control tissues) was detected at least 15 cm from the bullet pathway in all boar carcasses (N=10) and 30 cm from the bullet



Fig. 4 Radiograph of domestic sheep (*Ovis aries*) shot with lead ammunition (rapid expansion bullet fired from a 0.308 Winchester) behind the scapula. Bullet fragments are within the *red circles* and are throughout the thoracic cavity and into the pelvic cavity. Photo courtesy of Minnesota Department of Natural Resources, Farmland Wildlife Populations and Research Group, 35365 800th Ave, Madelia, MN 56062

pathway in three carcasses. Similarly, elevated lead was detected at least 15 cm from the bullet pathway in all deer carcasses (N=10) and 30 cm from the bullet pathway in three carcasses.

Fragmentation in Small Game and Varmints

To study the potential hazard of predators and scavengers consuming small game or varmints, 15 Richardson's ground squirrels (*Spermophilus richardsonii*) were shot using a .22 caliber rifle and hollow-point rimfire ammunition (Knopper et al. 2006). Bullet fragments were visible in 14 of 15 carcasses as minute debris rather than larger pieces of bullet. The area containing fragments (as determined by radiography) was removed from each carcass and the entire section analyzed for lead (bone, hair, and tissue combined). Concentrations in these sections ranged from 0.01 to 17.21 mg (median=3.23 mg). The authors theorized that larger fragments have enough momentum to leave a small carcass, but dust-like lead debris left behind may be an appreciable source of lead for consumers.

In black-tailed prairie dogs (*Cynomys ludovicianus*), fragmentation was studied in individuals shot with non-expanding lead bullets enclosed by a copper jacket and

expanding soft-point lead bullets (Pauli and Buskirk 2007). All prairie dogs were shot at 20–200 m to mimic typical shooting events. Radiographs revealed fragments in 26 of 30 prairie dogs shot with expanding bullets and 2 of 29 shot with non-expanding bullets. Fragments extracted for analysis averaged 317.8 mg (235.7 mg from the lead core, 23.2 mg from the copper jacket) for expanding bullets, and 43.0 mg (19.8 mg lead core, 23.2 mg copper jacket) for the non-expanding bullets. The authors hypothesized that most fragments were too small (<25 mg) to either be avoided during ingestion or egested through regurgitation by a predator or scavenger. Stephens et al. (2008) detected bullet fragments in four of ten black-tailed prairie dog carcasses from shooters at Thunder Basin National Grassland, Wyoming. Lead was the primary metal in fragments of one carcass, and copper the primary metal in the other three carcasses. Lead content averaged 57.3 mg in fragments from three carcasses with measurable amounts of lead.

In three rifle-shot coyotes obtained from a taxidermist, fluoroscopy revealed that lead fragments were distributed in the chest and abdominal cavity (Stauber et al. 2010). Fragments numbered in the hundreds in one carcass to less than ten in another.

Fragments in Meat from Hunter-Killed Game

Lead fragments have also been detected in meat processed from hunter-killed carcasses. Fragments were commonly observed via radiograph in white-tailed deer killed using copper-jacketed lead bullets (all of the same brand, caliber, and bullet weight) during the Wyoming hunting season and then commercially processed, each at a separate facility (Hunt et al. 2009). All 30 carcasses contained bullet fragments (15–409 fragments per carcass), with fragment separation up to 45 cm. In ground meat, fragments were visible in packages from 24 of 30 deer (80 %) and in 74 of 234 packages (32 %). Analysis of fragments excised from ground meat from 13 deer identified lead in 25 of 27 samples (93 %). Nine samples contained copper at greater than background levels. In Wisconsin, lead was detected in 30 of 199 (15 %) commercially processed packages of venison, and 8 of 98 (8 %) samples collected from hunters (Thiboldeaux 2008). Lead concentrations averaged 15.9 ppm wet weight in commercially processed samples that tested positive for lead and 21.8 ppm wet weight in hunter submitted samples that tested positive for lead. Pharmacokinetic modeling using the U.S. EPA *Integrated Exposure Uptake Biokinetic Model* predicted a risk of elevated blood lead concentrations in children (>0.1 ppm) from eating as little as one venison meal per month. In North Dakota, 100 samples of ground venison packages were selected from 15,250 packages donated to food pantries (Cornatzer et al. 2009). High definition computer tomography revealed metal fragments in 59 of 100 packages. Of 15 random samples from all 100 packages, one tested positive for 120 ppm lead dry weight. Lead concentrations in five samples known to contain metal fragments ranged from 4200 to 55,000 ppm dry weight.

In a follow-up investigation to the study by Cornatzer et al. (2009), the Centers for Disease Control and Prevention (CDC) collected blood samples from 736 vol-

unteers in six cities in North Dakota (Iqbal et al. 2009). Participants that consumed wild game had higher blood lead levels than those who did not, with those consuming the greatest serving sizes having the highest concentrations. The average blood lead concentration was 0.0117 ppm, with 1.1 % of samples ≥ 0.05 ppm. No samples exceeded the CDC recommended case management threshold of 0.1 ppm, though the authors note that there is no clinical threshold of lead in the human body that is considered safe, and that increased risk of factors such as myocardial and stroke mortality has been observed at ≥ 0.02 ppm. Since the publication of this investigation, the CDC has lowered its threshold to identify children at risk to 0.05 ppm, and removed the phrase “level of concern” from its publications, noting that no safe blood lead level in children has been identified (CDC 2012).

Key Points: Spent Lead Ammunition in the Environment

Regulation of ammunition

- Studies in the 1960s found waterfowl poisoning from lead shot to affect populations.
- Secondary poisoning to scavenging birds, including bald eagles, resulted from consumption of waterfowl containing embedded or ingested lead shot.
- Lead shot for waterfowl hunting became a nationwide restriction in 1991.

Sources of spent lead ammunition remaining after the ban

- The switch to non-toxic shot resulted in lower rates of lead shot ingestion in waterfowl and was effective at reducing, but not eliminating, lead shot exposure in waterfowl and birds that prey upon waterfowl.
- Lead fragments from bullets and slugs, and lead shot remain on the landscape in game animals that are wounded or killed by hunters but not retrieved, and from discarded offal piles.

Fragmentation

- Regardless of the type of game or lead-based bullet, all studies showed that lead bullets fragment, sometimes substantially, when fired into an animal.
- In samples collected from the field, fragments were often confirmed to contain lead by analysis of the fragments themselves or detection of lead in surrounding tissue.
- Metal fragments on radiographs are more opaque than bone or grit and easily distinguished.
- Fragment size and number varied, with some carcasses containing hundreds of fragments.
- Fragments radiated significantly (up to 45 cm) from the wound channel in large game.

(continued)

(continued)

- The creation of small lead fragments increases availability to scavengers due to the tendency to radiate from wound channels, avoid detection or regurgitation by scavengers, and be abundant enough to expose several scavengers feeding on a single carcass.
- In contrast to lead-containing bullets, monolithic copper bullets produced few, if any, fragments, within carcasses.

5 Vulnerability of Avian Scavengers

The vulnerability of an individual to a toxicant is based on its exposure potential and sensitivity (Golden and Rattner 2003). When coupled with demographic parameters, vulnerability of populations may be assessed.

5.1 *Exposure Potential*

The potential for a bird to be exposed to lead ammunition is a function of multiple factors, including its diet, foraging strategy, and the frequency that lead will be encountered in the environment. Birds that scavenge on carcasses or offal piles are likely to be exposed to spent ammunition in the tissues of hunter-killed game. Scavengers can feed on carcasses and offal, thus removing them from the environment, with efficiency. Removal rates have been documented as high as 98 % after 24 h in controlled studies, but can vary widely, influenced by factors such as season, location, and species (Prosser et al. 2008). Wildlife can also be exposed to spent ammunition via direct ingestion of shot or fragments from the ground; however, this route is unlikely to pose significant risk to scavenging birds.

Influence of Diet on Exposure

Birds can either be obligate scavengers, meaning they feed exclusively (or almost exclusively) on carrion, or combine scavenging with predation on live prey or other food items. Obligate North American avian scavengers include the California condor, black vulture, and turkey vulture, although the diet of the latter may include a high proportion of plant food (Kirk and Mossman 1998). Specific dietary items are dependent on geographic location, but condors and black vultures rely heavily on large mammals, while turkey vultures are more adaptable and forage on smaller and more varied food items (Kirk and Mossman 1998; Buckley 1999; USFWS 2013c). Other avian species such as bald eagles, golden eagles, caracaras, crows, and ravens

consume carrion as part of a broader diet (Boarman and Heinrich 1999; Buehler 2000; Kochert et al. 2002; Verbeek and Caffrey 2002; Morrison and Dwyer 2012). These species tend to be opportunistic, and like obligate scavengers, diets vary with geography, habitat, and availability. For some species, such as eagles, adults may be more apt than immatures to capture live prey due to better developed foraging ability (Buehler 2000). Crows and ravens consume the widest variety of food of birds that scavenge, including plant material, insects, garbage, eggs, small animals, and carcasses (Boarman and Heinrich 1999; Verbeek and Caffrey 2002). Some species, such as the northern harrier (*Circus cyaneus*) and ferruginous hawk (*Buteo regalis*), generally prey on live animals, but may scavenge when resources are available (Peterson et al. 2001; Stephens et al. 2008).

Scavenging birds may vary their diet according to availability. Both species of eagles have been documented to consume a greater percentage of carrion during winter months (Buehler 2000; Kochert et al. 2002). Several studies show a large percentage of deer, in particular, in the diet of bald eagles. In 949 feeding observations of bald eagles in New Brunswick, Canada, white-tailed deer and deer offal accounted for 40 and 30 % of the diet, respectively (Stocek 2000). Of 339 feeding observations of bald eagles wintering in the lower Great Lakes basin, 47 % were on carcasses of white-tailed deer (Ewins and Andress 1995). White-tailed deer remains were the most frequently detected dietary item (found in 67–72 % of the regurgitated castings) of bald eagles wintering along the St. Lawrence River (Lang et al. 2001).

Foraging Strategy

While most scavenging species will do so either individually or in groups, several factors related to foraging strategies can result in group feeding behavior at carcasses (Fig. 5). Turkey vultures have well-developed olfactory organs and are often the first to locate carrion (Kirk and Mossman 1998). Species such as black vultures, condors, and bald eagles are more reliant on visual clues, such as other scavengers feeding on a carcass, and thus tend to fly high, keeping other scavengers in view to follow them to carcasses (Buckley 1999; Buehler 2000; USFWS 2013c). Vultures and smaller birds cannot open thicker skin of some carcasses, so must wait for larger scavengers such as eagles or condors to open intact carcasses, which may displace them in the process (Kirk and Mossman 1998; Buckley 1999). However, specialization by location, type of carcass, and even within the carcass can result in no single species dominating a food source. For example, turkey vultures tend to feed on smaller items, less often near humans, and favor muscle and connective tissue, as opposed to viscera (Kirk and Mossman 1998). Species such as black vultures and bald eagles may visit the same carcasses for several days, thus providing a pathway for others in the roost to discover the carcass and increasing the number of individuals exposed to a single carcass (Buckley 1999; Buehler 2000). A single carcass may attract numerous scavengers. In a study of waterfowl carcass scavenging in agricultural fields, group sizes at a single carcass ranged from 1 to 79 individual birds (Peterson et al. 2001). Mean group size was 16.6 individuals, with



Fig. 5 *Top:* Bald eagles (*Haliaeetus leucocephalus*) feeding on the offal pile from a white-tailed deer (*Odocoileus virginianus*) killed in Monroe County, Iowa in January 2013. *Bottom:* Juvenile bald eagle feeding on a white-tailed deer carcass with an adult bald eagle and an American crow at the Lost Mound Unit of the Upper Mississippi River National Wildlife and Fish Refuge. Top photo courtesy of Peter Eyerhalde, Iowa State University; Bottom photo courtesy of USFWS Upper Mississippi River National Wildlife and Fish Refuge, 7071 Riverview Rd. Thomson IL 61285

maximums of 79 northwestern crows (*Corvus caurinus*) and 16 bald eagles recorded at a carcass at one time. In a study of black vultures, group size increased with carcass size, with as many as 98 individuals recorded at a single feral hog (*Sus scrofa*) carcass (Buckley 1999).

The tendency of scavengers to exhibit group feeding behavior may enhance their vulnerability to lead exposure in a number of ways. The probability of vultures finding food has been found to be related to the density of vultures in the habitat (Jackson

et al. 2008). As the number of foragers increases, so does the probability of finding a carcass. Likewise, if vulture populations experience decline and fall below a critical level, the feeding efficiency of each individual falls dramatically. In addition, the propensity for many individuals to feed on a single food source can expose them to a common source of contamination, as exhibited by group foraging in agricultural fields where carcasses may contain pesticide residues (Peterson et al. 2001). Demographic modeling of vultures in the Indian subcontinent affected by poisoning from use of the non-steroidal anti-inflammatory drug diclofenac revealed that observed population declines (22–50 % annually) could be attributed to a very small proportion of carcasses (between 1:130 and 1:760) containing concentrations of diclofenac that were lethal to old world vultures (Green et al. 2004).

5.2 Sensitivity to Lead

Avian sensitivity to lead is influenced by diet, anatomy, and physiology. Species and individuals may also possess inherent sensitivity driven by genetic makeup: three polymorphic genes have been identified that influence the bioaccumulation and toxicokinetics of lead in mammals though this has not yet been investigated in birds (Onalaja and Claudio 2000).

Physiology

Birds have a unique physiology that can enhance their vulnerability to lead toxicosis by facilitating breakdown and absorption into tissue. Species such as waterfowl that feed on coarse objects like grain or plant material have muscular gizzards for grinding that are larger than birds whose diet is largely meat (Farner 1960). This grinding facilitates the erosion of ingested metallic lead, making it more bioavailable for absorption in the gastrointestinal tract and subsequent transport to other organs (Jordan and Bellrose 1951). Although carnivorous birds may have highly reduced gizzards (and omnivorous birds intermediate to the two groups), other digestive characteristics facilitate the absorption of items from the gastrointestinal tract. While specific values are not available for scavenging birds such as eagles or vultures, some closely related raptors have been found to have especially acidic stomach fluids as compared to other bird species (Duke 1997). The average pH of 1.6 measured during gastric digestion in falcons translates to about six times more hydrogen ion per ml in their basal gastric secretion than was measured in owls (average pH 2.35; Duke 1997). Other species studied included turkeys (*Meleagris gallopavo*; pH 3.0) and domestic ducks (*Anas platyrhynchos* x *Cairina moscata* hybrid; pH 2.1). Raptors have the unique ability to egest indigestible materials, such as bone and fur via casts (Duke et al. 1976; Griffin et al. 1980; Nelson et al. 1989). Casts of raptors exhibit more thorough corrosion of bone as compared to those of owls, likely due to this difference in gastric pH (Duke et al. 1975). In addition, birds

possess a distinctive trait in gastrointestinal mobility that increases residence time of ingested materials in the digestive system, including the highly acidic stomach. Periodic reverse peristalsis moves the contents of the upper ileum and duodenum back into the stomach, an adaptation hypothesized to allow for greater digestion of nutrients without lengthening the gastrointestinal tract, which would be disadvantageous to flying due to added weight (Duke 1997). Raptors feeding on mice had 1–2 refluxes of this nature per hour to aid in digestion (Duke 1997).

Influence of Diet on Sensitivity

Diet can effect lead's toxicity and storage in tissues. Jordan and Bellrose (1951) reported that diet was a more important influence on the toxicity of lead in waterfowl than was the dosage of lead, within a range of one to four #6 shot. Toxicity can be enhanced by nutritional deficiencies in the diet (e.g., calcium, zinc, iron, protein, fat) and decreased by excesses of others (e.g., zinc, protein, fat; Eisler 2000). In particular, because of lead's physical similarity to calcium, these substances compete for absorption in the gut (Quarterman 1986). Studies in waterfowl have found that individuals ingesting foods high in protein and calcium were less susceptible to toxic effects of lead (Sanderson and Bellrose 1986). Dietary influence on lead toxicity has since been studied in other species, and findings indicate that toxic effects are less severe when birds intake nutritionally balanced diets high in protein and calcium. The mitigating effects of such diets may be the result of high calcium and protein levels reducing lead absorption in the gastrointestinal tract and lowering the body burden of lead (Koranda et al. 1979; Sanderson 1992; Scheuhammer 1996). Differences in lead concentrations found in species with similar ingestion rates have been attributed to this variation in diet, with species feeding on increasing amounts of animal matter showing a lower accumulation of lead in tissues (Stendell et al. 1979). Reproductively active female birds have been found to accumulate lead at a greater rate than males, a characteristic presumably linked to increased intestinal calcium absorption during eggshell formation (Scheuhammer 1987). The effects of dietary preferences upon lead toxicity have not been specifically studied in scavengers, but their high intake of animal matter may influence the effects of ingested lead, and could vary among species that specialize on different parts of the carcass.

Results of Toxicity Testing of Scavengers

Laboratory toxicity studies of lead ammunition have been performed on three species of scavenging birds, the Andean condor (Pattee et al. 2006), turkey vulture (Carpenter et al. 2003), and bald eagle (Hoffman et al. 1981; Pattee et al. 1981). For all three species, dosed birds exhibited individual variability in sensitivity, possibly related to factors such as length of shot retention, number of shot retained, amount of lead eroded, and individual susceptibility. Though protocols differed across studies (Table 2), the Andean condor appeared to be more sensitive than other raptor

Table 2 Comparison of dosing protocols in three studies of scavenging species experimentally treated with lead shot

Species	Number shot dosed	Shot number	Shot diameter (mm)	Approx. number shot in 1 oz	Total lead eroded (mg)	Outcome
Andean condor ^a	2 or 6	00	9.14	6.2	126–603	4/4 died or euthanized; clinical signs in 39–49 days
Turkey vulture ^b	3 or 10	BB	4.57	50	111–247	4/6 died or euthanized; clinical signs in 143–183 days; 2/6 survived to study end (211 days)
Bald eagle ^c	10–156	4	3.28	135	19–185	5/5 died in 10–133 days

^aPattee et al. (2006)^bCarpenter et al. (2003)^cPattee et al. (1981)

species tested, with all four birds dosed with either two or six lead shot (size 00) showing signs of lead poisoning within 50 days, and two of those succumbing. This contrasts with a time to mortality of 10–155 days in five bald eagles dosed with 10–156 lead shot (size 4), and a minimum time to death of 143 days in six turkey vultures dosed with up to three or six lead shot (size BB). Two turkey vultures were euthanized at 211 days showing no overt signs of lead poisoning. The authors concluded that the vultures showed considerable tolerance to lead shot when compared to other experimentally treated raptors (Carpenter et al. 2003).

Shot retention varied among individuals of each species. Redosing of turkey vultures was described as “constant” due to defecation or regurgitation of shot (Carpenter et al 2003). Bald eagles retained shot as little as 12 h and as long as 48 days (Pattee et al 1981). Dosed shot was recovered from all four condors at necropsy 39–49 days after dosing (with one bird having been redosed at day 7 following regurgitation) (Pattee et al. 2006). Shot retention has been reported in eagles that have succumbed to lead poisoning in the wild (e.g., 77 shot recovered in one eagle, Jacobson et al. 1977; Fig. 6). The variability in shot retention seen here is consistent with that seen in other species that ingest lead shot, which may erode or pass through the gut before death occurs (Roscoe et al. 1979; Schulz et al. 2006).

Studies on all three scavenging birds confirmed the solubility of dosed metallic lead shot in the digestive tract either by measuring erosion of lead shot post-dosing or by detection of lead in blood and other internal organs shortly after dosing. Total lead eroded after dosing varied among individuals and ranged from 126 to 603 mg (condors), 111–247 mg (turkey vultures), and 19–185 mg (bald eagles) (Table 2). For all species, an increase in blood lead concentrations and a decrease in ALAD activity followed treatment, with blood lead continuing to rise after dosing. These changes were detected in bald eagles within 24 h of treatment (Hoffman et al. 1981), and in condors and vultures at the first blood collection 7 days after dosing.



Fig. 6 *Left:* Radiograph showing two #5 lead shot (*circled*) in the digestive tract of a bald eagle (*Haliaeetus leucocephalus*) admitted to a wildlife rehabilitation center in Iowa. *Right:* Radiograph showing ammunition fragments (*circled*) in bald eagle carcass. Photos courtesy of Kay Neumann, Saving Our Avian Resources, 25494 320th Str, Dedham, IA 51440

Table 3 Comparison of hepatic lead concentrations in scavenging birds showing varying levels of intoxication after being experimentally treated with lead shot

Species	Lead in liver at necropsy (ppm wet weight)			
	Control	No signs of intoxication	Intoxication, euthanized	Mortality
Andean condor ^a	Not analyzed	–	49.88, 109.09	45.46, 58.52
Turkey vulture ^b	0.05, 0.10	1.48, 2.22	6.79, 18.71	20.73, 33.78
Bald eagle ^c	0.4	–	3.4	11.5–27.0

^aPattee et al. (2006)

^bCarpenter et al. (2003)

^cPattee et al. (1981)

Liver concentrations for these three species (Table 3) were in general agreement with previously described diagnostic thresholds (<2 ppm wet weight in liver for birds with no history of lead exposure and >6 ppm wet weight in liver in for birds with evidence of clinical poisoning; see Table 1). However, individual variability among treated birds exhibits the utility in incorporating diagnostic criteria such as field observations, clinical signs, gross lesions and pathological findings, when available, in making a finding of lead poisoning.

Signs of toxicosis in condors, vultures, and eagles showing clinical poisoning included lack of coordination, loss of appetite, lethargy, weakness, reduced activity,

postural change, drooped wing, and frequent opening of the mouth, each occurring in one or more species (Pattee et al. 1981, 2006; Carpenter et al. 2003). All three species exhibited emaciation, with turkey vultures showing a loss of pectoral muscle, subcutaneous fat, and coelomic fat, and condors showing a loss of subcutaneous, abdominal, and coronary fat. Histological findings included spongiosis in brain tissue of vultures and condors, and nephrosis in some individuals of all species. All four eagles examined for myocardial necrosis exhibited this lesion. It is notable that acid-fast inclusions were present in renal tubular epithelium in turkey vultures and condors, but not in bald eagles. As noted above (see Sect. 3), inclusion bodies are indicative of lead exposure, but are not consistently present in all species or all individuals within a species. In general, despite overall evidence of clinical poisoning, individual gross and histological signs varied among individuals and were not consistently displayed.

5.3 Demographic Vulnerability

Species that are long-lived, have delayed maturation, and low reproductive output can be disproportionately sensitive to the loss of breeding adults as opposed to immature individuals. Scavenging species such as eagles, condors, and vultures possess these life history traits (USFWS 1996; Buckley 1999; Buehler 2000; Kochert et al 2002). For example, bald eagles and condors can live well over 20 years, may not begin breeding until 6–7 years old, and typically have only 1–2 offspring (Gerrard et al. 1992; Schempf 1997; Snyder and Schmitt 2002). One study that examined blood lead concentrations in bald eagles admitted to a rehabilitation center found that a greater proportion of admitted adults had elevated lead concentrations compared to juveniles and hatch-years (Cruz-Martinez et al. 2012). The authors hypothesized that the discrepancy was due to the aggressive behavior of adult eagles at scavenging sites. A comprehensive examination of lead poisoning cases of eagles submitted to the National Wildlife Health Center between 1975 and 2013 found that the odds of lead poisoning were greater in adults versus juveniles (Franson and Russell 2014).

Key Points: Vulnerability of Avian Scavengers

Exposure potential

- Birds that feed on carcasses are at risk of ingesting lead ammunition from hunter-killed game.
- The tendency of scavengers to exhibit group feeding behavior can enhance their vulnerability to lead exposure by increasing their ability to locate contaminated carcasses and subsequently exposing multiple individuals simultaneously.

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Sensitivity

- Despite having a reduced gizzard, carnivorous birds contain physiological adaptations (low pH, reverse peristalsis) that facilitate the dissolution of lead after ingestion.
- Dietary components, including calcium and protein, may influence the susceptibility of birds to lead toxicity.
- Scavenging birds can retain ammunition in the gastrointestinal tract. However, when ammunition is not detected this could indicate either a lack of exposure, exposure followed by regurgitation, or complete absorption (e.g., for smaller fragments).
- Metallic lead is soluble in the gastrointestinal tract and is demonstrated in experimental studies by (1) measuring blood lead concentrations or enzyme activity after treatment with lead shot or (2) weighing shot before and after they have passed through the digestive system.
- In controlled studies with scavenging birds, signs considered diagnostic of lead poisoning were not exhibited uniformly by exposed species or individuals, despite known toxicosis.

Demographic vulnerability

- Because lead can kill breeding adults, species that are long-lived, have delayed maturation, and low reproductive output, such as condors and eagles, may be especially vulnerable to its effects.

6 Occurrence of Lead in Avian Scavengers

The sensitivity of scavenging birds coupled with the availability of lead ammunition in the environment has resulted in numerous documented cases of exposure and mortality. One study found that of 130 species documented to be affected by lead ammunition, 24 % were raptors and scavengers (Tranel and Kimmel 2009). Herein, we discuss examples of lead exposure or poisoning in predatory and scavenging birds that are representative of several regions across the United States. For ease of comparison, and to the extent possible, results are presented using tissue thresholds and diagnostic descriptors as defined above (background: <0.2 ppm blood, <2 ppm wet weight liver; subclinical: 0.2–0.5 ppm blood, 2–6 ppm wet weight liver; clinical poisoning: >0.5 ppm blood, >6 ppm wet weight liver; Table 1). Where study authors provided results using other thresholds or criteria, those are described. We only included examples after 1991 when the United States lead shot ban for waterfowl was in full effect in order to focus on exposures that are less likely to be associated with the consumption of waterfowl. Though waterfowl were not devoid of lead shot

following the ban, documented decreases in exposure, as described above, rendered waterfowl a less likely source than prior to the ban. In addition, only cases from the published literature are included, although a number of additional cases have been documented from wildlife rehabilitators, and state agency, university, and federal government labs. Table 4 lists such examples of lead exposure (>0.2 ppm blood or 2 ppm wet weight liver) in raptors. Some cases include evidence suggestive of exposure to lead ammunition, such as seasonality contemporary with hunting activities or radiography of gastrointestinal contents.

Table 4 Examples of lead exposure indicative of subclinical or clinical poisoning (>0.2 ppm in blood or >2 ppm wet weight in liver) in predatory and scavenging birds in the United States

Species	References	Clinical poisoning ^a	Hunting season ^b	Ammunition detected ^c
Bald eagle (<i>Haliaeetus leucocephalus</i>)	Kramer and Redig (1997)	•	•	•
	Strom et al. (2009)	•	•	
	Harris and Sleeman (2007)	•		
	Bedrosian and Criaghead (2009)	•	•	
	Neumann et al. (2009)	•	•	•
	Stauber et al. (2010)	•	•	
	Harmata (2011)	•		
	Nam et al. (2011)	•		•
	Bedrosian et al. (2012)	•	•	
	Cruz-Martinez et al. (2012)	•	•	•
	Pagel et al. (2012)	•	•	
	Mierzykowski et al. (2013)	•	•	
Golden eagle (<i>Aquila chrysaetos</i>)	Franson and Russell (2014)	•	•	•
	Warner et al. (2014)	•		
	Kramer and Redig (1997)	•	•	•
	Bedrosian and Criaghead (2009)	•	•	
	Stephens et al. (2008)			
	Domenech and Langer (2009)	•		
	Stauber et al. (2010)	•	•	
	Kelly et al. (2011)	•		
	Harmata and Restini (2013)	•		
	Franson and Russell (2014)	•	•	•
California condor (<i>Gymnogyps californianus</i>)	Kelly et al. (2014)	•		
	Langner et al. (2015)	•	•	
	Watson and Davies (2015)	•		
	Parish et al. (2009)	•	•	
	Finkelstein et al. (2012)	•		
	Rideout et al. (2012)	•		•
	USFWS (2012)	•	•	•
	USFWS (2013c)	•		•

(continued)

Table 4 (continued)

Species	References	Clinical poisoning ^a	Hunting season ^b	Ammunition detected ^c
Turkey vulture (<i>Cathartes aura</i>)	Kelly et al. (2011)		•	
	Kelly and Johnson (2011)	•	•	
	Kelly et al. (2014)	•		•
Black vulture (<i>Coragyps atratus</i>)	Behmke et al. (2015)	•		
Cooper's hawk (<i>Accipiter cooperii</i>)	McBride et al. (2004)		•	
Red-tailed hawk (<i>Bufo jamaicensis</i>)	Stansley and Murphy (2011)	•	•	
Common raven (<i>Corvus corax</i>)	Craighead and Bedrosian (2008)	•		
	Craighead and Bedrosian (2009)		•	

Columns indicate if any individuals had tissue concentrations consistent with clinical poisoning (>0.5 ppm blood or >6 ppm wet weight liver)^a, if authors indicated that elevated lead levels coincided with the hunting season^b, or if shot or fragments were detected in the gastrointestinal tract via radiography or necropsy. All individuals were collected or sampled after the 1991 United States ban on lead shot use for waterfowl hunting

Though the cases described below are somewhat widespread geographically, they are largely based on opportunistic sampling. For any contaminant, collection of dead or moribund birds is likely to represent only a subset of the actual exposure or mortality attributable to that contaminant. In order to document mortality, a carcass must be observed, reported, collected, and chemically analyzed while still relatively fresh (Vyas 1999). The loss rate of dead birds to scavengers may be up to 98 % in the wild, depending on season, location, and species, with losses generally occurring within 24–96 h after placement of a carcass in experimental studies (Peterson et al. 2001; Prosser et al. 2008). Carcass detection studies have found that even when searches are performed on carcasses known to exist (e.g., placed by a researcher for study), a percentage will never be found due to scavenging, location in remote and inaccessible areas, or size or coloration that renders the carcass inconspicuous (Vyas 1999; Elliott et al. 2008). For these reasons, in addition to the exclusion of unpublished data (as described above), the cases that follow are likely representative of a wider breadth of lead exposure and poisoning, including those jurisdictions where it has not been well documented to date.

Moreover, isolated or infrequent monitoring of lead in the blood of live birds is likely to miss peak values or entire exposure events due to lead's short half-life in blood (e.g., estimated at ~14 days for condors; Fry et al. 2009). A single sample provides information on concentrations at the time of collection only. There is no way to ascertain if blood concentrations are rising, falling, or static, or if previously exposed birds no longer contain detectable concentrations in blood (Barbosa et al. 2005).

Furthermore, lead concentrations from blood can occur from either current exposure or mobilization of previously stored lead from internal tissues (Barbosa et al. 2005). Information from blood samples should be interpreted accordingly.

6.1 *Nationwide*

United States (Bald and Golden eagles): Demographic and pathologic data obtained from case files for lead poisoned bald and golden eagles previously necropsied at the National Wildlife Health Center were analyzed for 484 bald eagles and 68 golden eagles (Franson and Russell 2014). The diagnosis of clinical poisoning was made by pathologists performing necropsies, generally based on gross observations including emaciation and characteristics of bile stasis, microscopic lesions such as damage to the kidney and heart, in combination with liver lead concentrations >6 ppm wet weight. Carcasses were collected from 1982 to 2013 for bald eagles and 1975–2013 for golden eagles from 38 states. The mean liver lead concentration was 28.9 ppm wet weight for bald eagles and 19.4 ppm wet weight for golden eagles. Lead ammunition or fragments were detected in 14.2 % of bald eagles and 11.8 % of golden eagles. Lead poisoned carcasses were found in greater frequency in the late autumn and winter than spring and summer months, and the odds of lead poisoning were greater in eagles from the Mississippi and Central flyway versus the Atlantic and Pacific flyway. In addition, the probability of lead poisoning was greater for bald eagles versus golden eagles, females versus males, and adults versus juveniles. Out of 4064 eagles submitted in total, trauma and poisonings were the leading causes of death for bald eagles, with lead accounting for the greatest percentage of poisonings (Russell and Franson 2014).

6.2 *Northeast*

Maine (Bald eagles): Mierzykowski et al. (2013) found that 19 out of 127 (15 %) bald eagles collected dead in the state of Maine between the years 2001 and 2012 had liver lead concentrations indicative of clinical poisoning (>6 ppm wet weight). The highest lead concentrations were detected in the eagles collected during the winter and early spring months.

New Jersey (Red-tailed hawks, Black vultures, Turkey vultures): Carcasses collected from wildlife rehabilitators in New Jersey from 2008 and 2010 were analyzed for lead exposure in liver (Stansley and Murphy 2011). Of 221 individuals representing 13 raptor species, two red-tailed hawks contained elevated liver concentrations (2.1 ppm wet weight, consistent with subclinical poisoning, and 7.4 ppm wet weight, consistent with clinical poisoning). The red-tailed hawk with the highest lead concentration was submitted during the hunting season for several small game

species in New Jersey. Of 104 red-tailed hawks in total, lead was detected in 52 birds at background concentrations (<2 ppm wet weight). Of scavenging species analyzed, background levels of lead were detected in 1 of 1 black vulture and 5 of 7 turkey vultures. Other species with background levels of lead were barred owl (*Strix varia*), broad-winged hawk (*Buteo platypterus*), Cooper's hawk (*Accipiter cooperii*), great horned owl (*Bubo virginianus*), red-shouldered hawk (*Buteo lineatus*), peregrine falcon (*Falco peregrinus*), eastern screech-owl, and sharp-shinned hawk (*Accipiter striatus*).

6.3 Southeast

Virginia (Bald eagles): From 1993 to 2003, 4 of 95 bald eagles admitted to the Wildlife Center of Virginia were diagnosed with lead poisoning based on blood concentrations of >0.2 ppm and clinical signs, or, when available, elevated liver concentrations (Harris and Sleeman 2007). Trauma was the most common diagnosis (71 %). Eagles were not routinely screened for lead unless showing clinical signs (Jonathan Sleeman, USGS National Wildlife Health Center, personal communication).

Virginia (Black vultures, Turkey vultures): Lead was measured in black vultures and turkey vultures culled by the U.S. Department of Agriculture, Wildlife Services in Chesterfield County, Virginia from July 2011 to May 2012 (Behmke et al. 2015). Mean lead concentrations in liver were 0.78 ppm wet weight (range: 0.012–6.17 ppm, N=96) in black vultures and 0.55 ppm (0.23–1.3 ppm, N=9) in turkey vultures. Of these, concentrations in livers of five black vultures fell within the range of subclinical poisoning (2–6 ppm) and one was over the threshold of clinical poisoning (>6 ppm). Concentrations in livers of turkey vultures did not exceed background levels (>2 ppm). Mean concentrations in femur were 36.99 ppm wet weight (4.5–540 ppm, N=98) in black vultures and 23.02 (6.16–70, N= 10) in turkey vultures, above thresholds that have been associated with background exposure (10 ppm dry weight). The authors indicated that these concentrations were indicative of long-term lead exposure and used isotopic analysis to attribute their origin to multiple potential sources of lead including ammunition, gasoline, coal-fired power plants, and zinc smelting.

6.4 Northwest

Montana (Golden eagles): Of 42 migrant golden eagles sampled in Montana during the fall of 2006 and 2007, 58 % had elevated blood lead levels (Domenech and Langer 2009). For all eagles, blood lead ranged from <0.005 to 4.81 ppm, with 18 exhibiting background levels (0–0.1 ppm), 19 subclinical poisoning (0.1–0.6 ppm), 5 clinical exposure (>0.6 ppm).

Of 74 golden eagles captured in southwestern Montana from 2008 to 2010, 70 (97 %) contained detectable levels of lead in blood (Harmata and Restani 2013). Of these eagles, concentrations were indicative of subclinical poisoning (0.2–0.5 ppm) in 29 %, and clinical poisoning (>0.5 ppm) in 16 %. Blood lead concentrations decreased from winter to spring, which the authors attributed to either a change to a less contaminated food supply locally or population turnover during migration.

Of 178 golden eagles sampled in the Helena National Forest in Montana during fall migration from 2006 to 2012, 118 (66 %) contained blood lead concentrations below background levels (<0.2 ppm), 42 (24 %) within the range of subclinical poisoning (0.2–0.6 ppm), and 18 (10 %) above the threshold for clinical poisoning (>0.6 ppm; Langner et al. 2015). Seven eagles contained blood lead >1.2 ppm and all were captured during the second half of fall migration, when hunter-killed carcasses were presumed to be increasing due to the start of the big-game hunting seasons along the migratory route. Golden eagles trapped using road-killed deer carcasses contained significantly higher concentrations than those trapped using bow-nets with rock pigeons. The authors suggest that these individuals may specialize on carcasses and therefore be more susceptible to increased lead exposure from bullet fragments. Hatch-year birds had lower concentrations than subadults and adults, suggesting that blood lead concentrations may be a function of cumulative lead exposure over time.

Montana (Bald eagles): Bald eagles were sampled from nestling, free-flying, and rehabilitation populations in southwestern Montana from 2006 to 2008 (Harmata 2011). Mean blood lead concentration in nestlings (N=17) was 0.037 ppm and none exceeded the threshold for clinical poisoning (>0.6 ppm). The mean concentration in free-flying individuals (N=88) was 0.272 ppm, 70 % of which exceeded background levels (>0.2 ppm), and 9 % exceeded 1.0 ppm. For birds sampled at rehabilitation centers (N=23), mean concentrations were 0.183 ppm; most were within the range of background concentrations and none exceeded 1 ppm.

Wyoming (Ferruginous hawks, Golden eagles): In 2001, Stephens et al. (2008) investigated blood lead concentrations in nestling ferruginous hawks and golden eagles collected from Thunder Basin National Grasslands, Wyoming, due to local increases of lead poisoned raptors being admitted to wildlife rehabilitation facilities. Lead was detected in blood of all nestlings, but at less than subclinical concentrations (<0.2 ppm). Ferruginous hawks and golden eagles are known to scavenge on black-tailed prairie dogs at this location that have been killed by shooters and not recovered. Concentrations of lead in nestlings were similar to those at a reference site where no hunting took place. As scavenging on prairie dog carcasses is a known exposure pathway (bullet fragments were detected in four of ten carcasses collected), the authors hypothesized that lead levels may have been low due to decreased shooting at the site that season and an increase in alternate food sources for raptors.

Wyoming (Bald eagles, Golden eagles): Bedrosian and Craighead (2009) found that bald eagles and golden eagles accumulated more lead in blood samples during the

hunting season (median 0.56 ppm, just above the threshold for clinical poisoning) in the southern Yellowstone River ecosystem compared to the non-hunting season (median 0.277 ppm, in the range of subclinical poisoning). Nine blood samples out of 63 had concentrations >1.0 ppm.

Wyoming (Bald eagles): Bedrosian et al. (2012) studied free-flying bald eagles near Jackson Hole, Wyoming. The study area included all hunt zones on the National Elk Refuge and Grand Teton National Park where there is an estimated 3000 big game animals harvested annually. There are no other potential sources of lead in the study area as recreational varmint hunting, waterfowl hunting, upland game hunting, and fishing with live bait are not permitted on the Refuge or the National Park Service land. Blood samples were analyzed from 71 free-flying eagles, which included three re-captures and eight nestlings. Samples were taken during and after the big game hunting season for elk and bison in 2005–2010, excluding 2008. The analysis found 93 % (68) of all free-flying eagles tested had concentrations above background levels (>0.1 ppm). Thirty-three percent (14) of these had exposure indicative of clinical poisoning (>0.6 ppm), all of which were sampled during hunting season. For all eagles tested (free-flying and nestlings) 24 % had blood levels indicative of clinical poisoning during the hunting season, and none during the non-hunting season. There was an increase of eagle abundance during the hunting season as compared to after the season. The study tracked ten free-flying eagles with satellite transmitters. These eagles were captured at the site during the hunting season. After the hunting season, the ten eagles migrated south. The following year during the hunting season, 80 % of the eagles with transmitters returned to the site. The authors suggest that offal provides a seasonal attractant for the eagles.

Wyoming (Common ravens): Craighead and Bedrosian (2008) found that of 302 ravens sampled in the southern Yellowstone River ecosystem during 2004 and 2005, 47 % exhibited elevated blood lead levels (≥ 0.1 ppm) during the hunting season as compared to 2 % during the nonhunting season. Expanding upon this data, additional samples were collected through 2008 (Craighead and Bedrosian 2009). For all samples pooled ($N=539$), median blood lead levels were 0.10 ppm for the hunting season and 0.02 ppm during the nonhunting season. A significant relationship was detected between annual median blood lead levels and the combined large-game harvest success from the National Elk Refuge and Grand Teton National Park.

Inland Pacific Northwest (Bald eagles, Golden eagles): Of 67 golden eagles and 63 bald eagles admitted to a raptor rehabilitation program from 1991 to 2008, 46 and 50 were tested for blood lead, respectively (Stauber et al. 2010). Blood lead concentrations above background levels (>0.2 ppm) were detected in 48 % of bald eagles and 62 % of golden eagles, and these eagles were submitted from eastern Washington, northern Idaho, northeast Oregon, and Alaska. Of these, 46 % of bald eagles and 52 % of golden eagles had concentrations associated with clinical poisoning (>0.5 ppm). Admission of eagles to the rehabilitation center with lead concentrations greater than background was strongly seasonal: 91 % of bald eagles were admitted January to March, and 58 and 32 % of golden eagles were admitted January

to March, and October to December, respectively. Elk (*Cervus canadensis*) and deer hunting seasons run from October to December, and the authors indicated that shooting of coyotes intensifies after December and could contribute to lead exposure after that time. All eagles were radiographed and none had evidence of ingested lead particles.

Washington (Golden eagles): From 2005 to 2013, lead was analyzed in blood of resident golden eagles on their nesting territories in the Columbia Basin of eastern Washington between January and June (Watson and Davies 2015). Of 17 eagles sampled, 11 had elevated concentrations (>0.2 ppm), four of which were at levels consistent with clinical poisoning (>0.5 ppm). None of these four exhibited physical impairment upon capture and all survived at least 1 year following capture. Two eagles recovered after colliding with wind turbines had background concentrations of lead (≤ 0.2 ppm).

6.5 Southwest

New Mexico (Cooper's hawk): Blood samples were collected from Cooper's hawks in the Cibola National Forest of north-central New Mexico during fall migration of 2001 and at the spring migration of 2002 (McBride et al. 2004). Three of 98 samples contained lead concentrations indicative of subclinical or clinical poisoning (0.2–1.5 ppm), all collected during spring migration. In total, 50 individuals contained detectable levels of lead. Cooper's hawks may feed on upland game birds such as dove and quail which may contain embedded lead shot. The authors hypothesized that blood lead levels were higher in spring migrants due to the timing of the hunting season in their winter range.

California (Turkey vultures): Lead was elevated (>0.1 ppm) in the blood of 83 of 172 turkey vultures sampled across California in 2008 and 2009 (Kelly and Johnson 2011). Some vultures contained lead concentrations >1.0 ppm, but showed no overt signs of toxicosis. In an intensely hunted area, average blood lead levels were twice as high during the hunting season as during the off-season, with 76 % having elevated concentrations during the hunting season as compared to 36 % prevalence outside of the big game hunting season. Blood lead was also measured in turkey vultures collected within the area of the California condor rangewide ban on lead ammunition for big game hunting (see Sect. 6.7 for more information on this ban; Kelly et al. 2011). Concentrations were elevated (>0.1 ppm) in significantly more vultures collected in 2008 prior to this ban (23 of 38; 61 %) than those collected in 2009 after this ban (3 of 33; 9 %). Blood lead levels indicated subclinical poisoning (0.2–0.49 ppm) in seven vultures (18 %) prior to the ban and one vulture (3 %) after the ban. None of the vultures contained blood lead concentrations associated with clinical poisoning (>0.5 ppm).

California (Golden eagles): Blood lead was measured in golden eagles that were collected within the range of the lead ammunition ban for big game hunting in southern California (Kelly et al. 2011). Concentrations were elevated (>0.1 ppm) in significantly more eagles collected in 2007–2008 prior to the ban (13 of 17) than those collected in 2008–2009 after the ban (12 of 38).

Of non-migratory eagles, the prevalence of elevated lead dropped from 83 % (5 of 6) to 0 % (0 of 9) after the ban. Blood lead levels indicated subclinical poisoning (0.2–0.49 ppm) in eight eagles (48 %) prior to the ban and four eagles (11 % after the ban). Blood levels exceeded the threshold for clinical poisoning (>0.5 ppm) in one eagle (6 %) prior to the ban and three eagles (8 %) after the ban.

California (Bald eagles): Of seven bald eagles released in the northern Channel Islands between 2002 and 2006, and subsequently found dead, three had concentrations of lead in bone considered to be above background levels (>10 ppm wet weight) (Pagel et al 2012). Eagles that spent the most time on Santa Rosa Island, where a deer and elk hunting program occurred, had the highest concentrations of lead. One bird found alive with a broken wing had a blood lead level of 0.522 ppm, above the threshold for clinical poisoning (>0.5 ppm).

California (Golden eagles, Turkey vultures, Common ravens): Cause of death was determined in 21 golden eagles, 23 turkey vultures, and 4 common ravens collected from wildlife biologists and wildlife rehabilitators in 13 counties throughout California between 2007 and 2009 (Kelly et al. 2014a). Forty-five of these birds were found alive and three (two golden eagles and one turkey vulture) were found dead. Lead poisoning (diagnosed from pathological lesions plus lead concentrations >1 ppm in blood or >5 ppm wet weight in liver) was the primary cause of mortality in 17 % (8/48) of cases. Of these, five were turkey vultures, three were golden eagles, and none were ravens. A lead-based fragment was retrieved from the gastrointestinal tract of one vulture. Lead-related mortalities occurred during the winter and early spring months (i.e., outside of the big game hunting season) and were found in various areas throughout the state. Of 39 birds total analyzed for lead, subclinical or clinical concentrations were detected in 21 % of livers (>2 ppm wet weight) and 48 % of bone samples (>6 ppm dry weight).

6.6 *Bald Eagles in the Midwest*

The Great Lakes region is an important habitat for bald eagles in the winter (Millsap 1986; Steenhof et al. 2002) and some of the highest mid-winter bald eagle counts have been recorded for the central United States (Steenhof et al. 2008). During the winter season, bald eagles congregate and forage along lakes and tributaries to feed on fish and waterfowl. As winter progresses in the Midwest and ice freezes over many of the open waterways, bald eagles opportunistically search for other food sources that are available on the landscape. One readily available food source in the winter is discarded offal from hunter-killed white-tailed deer, or deer that were shot

and not retrieved (Harper et al. 1988; Ewins and Andress 1995; Lang et al. 2001; Cruz-Martinez et al. 2012; e.g., Fig. 5).

Iowa: Wildlife rehabilitation facilities in Iowa processed 62 moribund bald eagles from 2004 to 2008 with 39 (62.9 %) of the birds having concentrations >0.2 ppm in the blood or >6 ppm wet weight in liver (Neumann 2009). The radiographs of seven of the 59 (12 %) birds admitted in Iowa showed fragments in the digestive tract presumed to be ammunition. Some of the birds admitted for traumatic injuries had elevated lead exposure. Most of the lead poisoned birds were admitted between the months of September and April, overlapping with the deer hunting season.

Minnesota, Iowa, and Wisconsin: At a raptor rehabilitation center in Minnesota 1227 moribund bald eagles were admitted from 1996 to 2009, with 331 (27 %) of the birds having blood lead concentrations above background levels (>0.2 ppm) (Cruz-Martinez et al. 2012). Over 90 % of these eagles were from Minnesota, Iowa, and Wisconsin. The chance of elevated lead concentrations increased based on hunting season, deer hunting zones, and age of bird. Metal objects were visible by radiograph in the stomachs of 34 eagles with lead levels above background (10 with shot, 24 with metallic shrapnel). An investigation conducted by the USFWS found that 35 of livers from the 58 (60 %) dead bald eagles found between 2009 and 2012 in the states of Iowa, Minnesota, and Wisconsin had detectable concentrations of lead and 22 (37.9 %) had concentrations consistent with clinical poisoning (>6 ppm wet weight in liver; Warner et al. 2014).

Michigan and Minnesota: In a retrospective analysis of 46 eagles that had been diagnosed with various causes of death (mainly trauma) between 2002 and 2010, 30 % exceeded levels suggestive of lead poisoning (>26.4 ppm dry weight in liver) (Nam et al. 2011). Fluoroscopic analysis of 26 birds from Michigan revealed lead shot or fragments (1–19 pieces) in the digestive tract of eight birds.

Wisconsin: The State of Wisconsin diagnostic laboratory processed 583 dead bald eagles between 2000 and 2008 with 87 (16 %) diagnosed with lead poisoning (>6 ppm wet weight in liver; Strom et al. 2009). This study associated bald eagle exposure rates with the hunting season in Wisconsin. Of the remaining admitted eagles, 48 % were diagnosed with trauma; however, lead concentrations were not presented.

Minnesota and Wisconsin: Of 654 bald and golden eagles admitted to a rehabilitation center from 1980 to 1995, 138 contained blood lead levels above background (>0.2 ppm) (Kramer and Redig 1997). Of these eagles, approximately 75 % were collected in Minnesota and Wisconsin, with the remaining from Alaska, Iowa, Illinois, Indiana, Michigan Nebraska, North Dakota, South Dakota, and Ohio. All eagles were routinely screened for lead upon admittance. The prevalence of lead-exposed eagles did not change after lead shot restrictions were implemented (17.5 % before 1991 versus 26.8 % after 1991). Of the 66 cases between 1991 and 1995, 36 % had blood lead concentrations indicative of clinical poisoning (>0.6 ppm). Eagles were admitted in all months of the year, with >40 % in November and

December for eagles collected after 1991, coinciding with deer seasons in Minnesota and Wisconsin. Of the 66 lead exposed birds admitted after 1991, 32 % were admitted for trauma, 23 % for projectile injury, and 17 % for lead toxicity. One eagle had radiographic evidence of lead shot in its gastrointestinal tract.

6.7 California Condor

The California condor, once distributed throughout North America, is now considered one of the rarest bird species in the world. The overall population has steadily increased over the past two decades, chiefly from the propagation and release of captive-reared individuals. Condors can currently be found in two distinct populations in the United States based on release sites: Arizona and Utah (referred to herein as the “Arizona” population), and California. Condors have also been released in Baja, Mexico. As of December 2012 there were 235 free-flying birds in the wild and 169 in captivity. However, the condor population’s high rate of mortality and its low reproductive success have prevented recovery of the species. The leading causes of mortality in birds from all release sites were determined to be anthropogenic, with lead poisoning deemed the most important of those identified (Rideout et al. 2012). Extensive conservation efforts towards recovery continue, including annual captive rearing and release, bi-yearly trapping and drawing of a blood sample, chelation treatment of condors that exhibit elevated lead concentrations, supplemental feeding, radio tracking, and daily monitoring.

While exposure to other sources of lead cannot be definitively ruled out, and some have been documented in select cases (e.g., lead paint; Finkelstein et al. 2012), the feeding habits of the condor make it particularly vulnerable to lead exposure from hunter-killed carcasses left in the field. Condors in interior habitats rely heavily on mule deer, elk, pronghorn antelope (*Antilocapra americana*), feral hogs, and domestic ungulates, but may also scavenge smaller mammals such as rabbits, ground squirrels, and coyotes (USFWS 2012; 2013c). Condors maintain wide-ranging foraging patterns throughout the year, allowing for opportunistic feeding in accordance with food supplies (Meretsky and Snyder 1992).

California: In California, the total harvest of game and varmints from the counties in the condor’s range can be quite high, and in 2010 included approximately 9000 deer, 17,000 cottontail rabbits, 9500 jackrabbits, 8500 wild pigs, 13,000 coyotes, and 7000 tree squirrels (California Department of Fish and Game 2011). In 2007, the State of California passed a law that banned the use of lead ammunition in the range of the condor for big game and varmint hunting. Despite this, declines in blood lead concentrations were not found in the California population in the years following the ban (Finkelstein et al. 2012). Kelly et al. (2014b) suggest that this lack of decline may be attributable to a lesser reliance on intensive management in condors sampled after the ban. Based on their analysis of blood samples collected from

the California population between 1997 and 2011, lead concentrations increased as the time an individual went undetected near provisioning and release sites increased, and as reliance on food provisioning decreased. Time undetected was significantly higher and reliance on food provisioning was significantly lower and post-ban years as compared to pre-ban years (Kelly et al. 2014b). Condors foraging independently of provisioned food may be vulnerable to exposure from lead ammunition remaining on the landscape from poaching, non-compliance with the ban, and shooting activities not addressed under the ban (such as upland game hunting, nuisance animal control, and dispatching domestic livestock).

The USFWS summarized condor mortality up to 2012 for the California population (USFWS 2013c). Condors were first released in California in 1992. By December of 2012, 42 (34 %) out of 123 cases where a cause of death was known were attributed to lead poisoning (USFWS 2013c). Rideout et al. (2012) provided details of condor fatalities occurring between 1993 and 2009. Diagnostic criteria for clinical poisoning in these cases were >0.5 ppm lead in blood (antemortem) or >6 ppm wet weight in liver or kidney (post-mortem). Some cases had additional supporting evidence such as lead fragments in gastrointestinal tracts, or histological findings consistent with lead exposure. In California, three of nine lead poisoning cases were based on liver lead concentrations of 11, 21, and 26.4 ppm wet weight (Rideout et al. 2012). In five cases, condors received chelation therapy but did not recover. Chelation therapy results in lowered lead concentrations in tissues, and as such, these condors had lead concentrations in liver (<3.7 ppm wet weight) below the threshold level for clinical poisoning at the time of death. The remaining case involved a condor with lead toxicosis that did not recover after receiving therapy. Based on feather analysis, this condor was exposed to lead four times since its release 180 days earlier, including one event in which lead concentrations were severely elevated (3 ppm) in blood.

Finkelstein et al. (2012) tested blood samples multiple times throughout the year from up to 150 free-flying condors in the California population between 1997 and 2010. Based on 1154 samples analyzed for lead and lead isotope ratios, an average of 71 % of the birds tested each year were exposed to lead above the selected background concentration of 0.1 ppm (approximately threefold higher than the average background lead level of pre-release condors with no history of lead exposure, 0.0303 ppm). Thirty percent of blood samples collected each year indicated exposure to lead >0.2 ppm, indicative of subclinical health effects (>60 % inhibition of ALAD). About 20 % of the birds tested each year were exposed to lead concentrations >0.45 ppm, the threshold selected for chelation therapy to avert morbidity or mortality. Over the period of 1997–2010, 48 % of the free-flying birds had blood lead concentrations over this threshold. The use of isotopic analysis to identify possible sources of lead exposure revealed that 79 % of the 150 condors sampled contained lead with ratios that overlapped with those measured in ammunition by the researchers (Finkelstein et al. 2012). Other samples contained isotope ratios consistent with leaded paint from a tower where condors were observed roosting. The remaining isotopic ratios were not consistent with known sources of lead.

Arizona: In Arizona, harvest in 2011 within the game management units in the condor's range included over 1000 mule deer, several hundred elk and pronghorn, and potentially thousands of cottontail rabbits, coyotes, and tree squirrels based on statewide totals (Arizona Game and Fish Department 2012). In 2003, the State of Arizona instituted a voluntary program offering incentives for the use of non-toxic ammunition for big game hunting or the removal of offal harvested with lead ammunition. Despite these measures, declines in blood lead concentrations were not found in the Arizona population in the years following the instatement of this program (Parish et al. 2009). Movement from Arizona to the Utah highlands for mule deer and elk hunting season, where no lead reduction program existed until 2012, was associated with a spike of blood lead concentrations during and just after that state's big game hunting season (Parish et al. 2009). Additionally, poaching, non-compliance with lead reduction programs, and shooting activities not addressed by these programs (such as small game and varmint hunting) likely contributes to lead remaining on the landscape throughout the range in Arizona.

The USFWS summarized condor mortality through 2011 for the Arizona population. Condors were first released in Arizona in December of 1996. By the end of 2011, 69 out of 134 released condors had died (USFWS 2012). Of 44 cases where the cause of death was determined, 21 deaths (48 %) have been attributed to lead poisoning (USFWS 2012). Of these birds, eight carcasses contained bullet fragments, one had a whole bullet, and five had shot. Rideout et al. (2012) provided details of condor fatalities occurring between 1993 and 2009. In Arizona, 7 of the 12 lead poisoning cases were based on liver lead concentrations ranging from 17 to 62 ppm wet weight (Rideout et al. 2012). Lead fragments were identified in the gastrointestinal tract of three of these individuals. The remaining five lead poisoning cases were for condors that received chelation therapy but did not recover. As a result of therapy, lead concentrations in liver (<5.2 ppm wet weight) were below the threshold for clinical poisoning at the time of death. Metallic fragments were detected in the gastrointestinal tracts of three of these five treated condors, though were not identified as lead or another metal. Condor mortality in Arizona has predominantly occurred in the fall and winter months and is associated with the big-game hunting seasons (Parish et al. 2009).

Parish et al. (2009) collected blood multiple times throughout the year from up to 64 condors in the Arizona population between 2000 and 2007. Depending on the year, an average of 48–95 % of the birds tested were exposed to lead. Birds were held and monitored when blood lead concentrations were >0.3 ppm. Chelation therapy was administered to condors with concentrations above this threshold whose blood lead continued to increase, or those with concentrations >0.6 ppm, amounting to 4–70 % of the Arizona population treated per year. Since 2002, an increase of condor blood lead concentrations has been found to correspond with deer hunting areas in southern Utah (USFWS 2012).

Key Points: Occurrence of Lead in Avian Scavengers

- Lead exposure and poisoning has been broadly documented in predatory and scavenging birds including eagles, condors, vultures, hawks, and ravens.
- There is broad geographic distribution of lead exposure and poisoning of scavenging birds across the United States.
- Elevated lead concentrations have not only been detected in tissues of dead or moribund birds, but also in random sampling of free-flying birds.
- Elevated lead was detected in tissues of some birds that were diagnosed with mortality from trauma.
- For bald eagles wintering in the Midwest, lead poisoning is a common diagnosis in moribund and dead eagles submitted to rehabilitators and diagnostic labs annually.
- For condors in Arizona and California, nearly half of all fatalities through 2011 and 2012, respectively, have been attributed to lead poisoning.
- Measures to reduce the amount of lead ammunition in the condor's range have likely contributed to a decline in lead availability, but lead ammunition may still be present from upland game hunting, nuisance animal depredation, dispatching domestic livestock, poaching, and non-compliance of lead ban regulations.

7 Association of Lead Exposure with Spent Ammunition

In addition to the diet and foraging strategies discussed above that make scavenging birds susceptible to ingesting ammunition, there are additional factors that support ammunition as the primary source of lead exposure in the cases just described: greater frequency and/or magnitude of lead exposure during or following hunting seasons, observation of ammunition in birds with lead exposure, and isotopic signatures of lead within the range used for the manufacture of ammunition.

7.1 *Temporal Association with Hunting Season*

Seasonality can play an important role in lead exposure as birds opportunistically forage on readily available food sources such as hunter-killed deer carcasses and offal piles. An increase in the frequency of lead exposure events and mortality during or immediately after game hunting seasons was formerly observed in scavenging birds prior to the lead shot ban for waterfowl in the United States (Bloom et al. 1989; Wiemeyer et al. 1988). This temporal trend continues to be observed, as presented in Table 4 and in the previous section (Kramer and Redig 1997; McBride

et al. 2004; Hunt et al. 2007; Craighead and Bedrosian 2008; Bedrosian and Craighead 2009; Parish et al. 2009; Strom et al. 2009; Stauber et al. 2010; Kelly and Johnson 2011; Bedrosian et al. 2012; Cruz-Martinez et al. 2012; USFWS 2012; Mierzykowski et al. 2013; Russell and Franson 2014; Langner et al. 2015). Instances of poaching that occur outside of legal hunting seasons or exposure to small game or varmints shot with lead in other seasons can weaken these temporal trends.

7.2 Detection of Ammunition

Lead fragments or shot may be recovered at necropsy and removed surgically (Fig. 7), or present on radiographs (Fig. 6). In some cases, lead poisoned birds have also been observed feeding on contaminated carcasses. While many cases lack this direct line of evidence, there are numerous citations of such that complete the pathway, as presented in Table 4 and above (Kramer and Redig 1997; Neumann 2009; Nam et al. 2011; Cruz-Martinez et al. 2012; Rideout et al. 2012; USFWS 2012; Franson and Russell 2014; Kelly et al. 2014a).

Birds may also be exposed to lead ammunition despite no detection or recovery of lead fragments from the gastrointestinal tract. As described previously, the fragility of lead can produce fragments small enough to be completely dissolved in the gastrointestinal tract. Absorption of fragments is enhanced by the physiology of

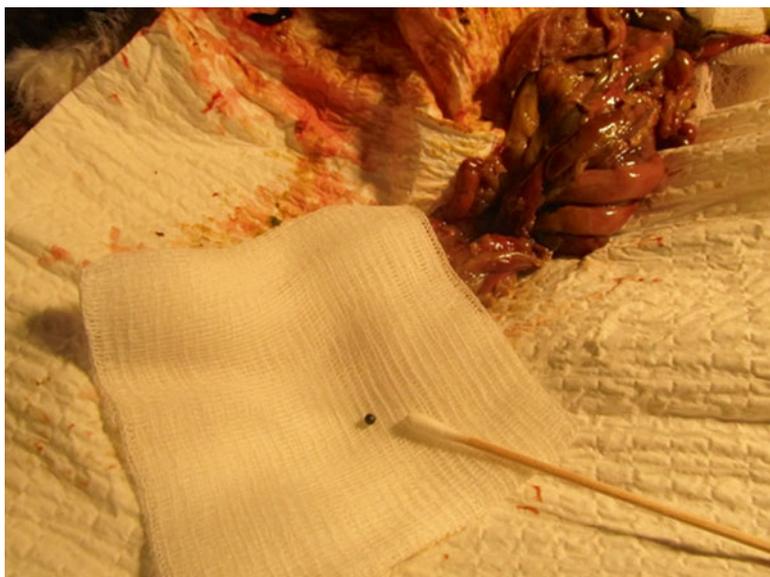


Fig. 7 Retrieval of lead shot from gastrointestinal tract of a bald eagle (*Haliaeetus leucocephalus*). Photo courtesy of Kay Neumann, Saving Our Avian Resources, 25494 320th Str, Dedham, IA 51440

raptors that combines an extremely acidic environment with prolonged digestion. Fragments that are not completely absorbed may also be passed by birds prior to death or capture, leaving no evidence in radiographs or post-mortem examination (Rideout et al. 2012). Fragments that are not completely absorbed may also be egested and lead shot has been found in casts for raptors sampled in the wild (Griffin et al. 1980; Nelson et al. 1989). In one laboratory study, the time between consumption of a meal and cast egestion in captive bald eagles was about 22 h (Duke et al. 1976). Therefore, cast egestion is not a means of completely eliminating ingested lead, as lead can be retained in the stomach for a period of time and absorbed before cast egestion. In laboratory studies of scavenging birds dosed with lead shot, retention of shot varied among individuals of the same species and under similar treatment regimens (Pattee et al. 1981, 2006; Carpenter et al. 2003).

7.3 *Isotopic Ratios in Lead-Exposed Birds*

Lead has four stable isotopes that occur naturally: ^{204}Pb , ^{206}Pb , ^{207}Pb , and ^{208}Pb . Ores from which lead is mined are made up of various percentages of these four isotopes, and those percentages are maintained as lead is extracted and manufactured into products. The ratio of any two of these isotopes is sufficient to provide a unique identifier for the lead, with the ratio of $^{207}\text{Pb}:^{206}\text{Pb}$ most commonly reported (all ratios are reported as such herein). Measuring these ratios in human or wildlife tissue and comparing them to ratios in particular or potential sources of lead (e.g., paint, ammunition, mine tailings) can help to elucidate how biota are exposed to this toxicant.

In an attempt to identify sources of lead exposure for wildlife, several studies have examined isotopic signatures of ammunition for comparison with ratios measured in tissues of exposed individuals. Scheuhammer and Templeton (1998) measured $^{207}\text{Pb}:^{206}\text{Pb}$ ratios in 22 brands of lead shotshells, finding low variability within cartridges and boxes, but higher variability between brands. Ratios from tissues of lead-exposed birds (game birds, waterfowl, and bald and golden eagles) showed ranges and patterns similar to those measured for shot (0.787–0.935). Few samples had ratios within the narrow range associated with environmental lead from gasoline (0.8658–0.8811), with the exception of bones from ten lead-exposed herring gulls, a species that rarely ingests lead ammunition. None of the samples analyzed in this study were consistent with ratios associated with mine and smelter waste.

Church et al. (2006) compared $^{207}\text{Pb}:^{206}\text{Pb}$ isotopic ratios measured in ammunition, condor dietary items, and condor tissue. Ratios from 13 boxes of purchased ammunition and nine individual rifle bullets donated from hunters ranged from 0.8054 to 0.8145, much narrower than that determined by Scheuhammer and Templeton (1998). Lead isotope ratios in seven samples of dietary items (0.8253–0.8394) were similar to background environmental ratios in California obtained from literature (0.8338–0.8453). Blood was collected from both captive (pre-release) and free-ranging condors. Lead levels and isotopic ratios were strongly inversely

associated; as condors were exposed to greater lead concentrations, isotopic ratios declined. Isotopic ratios from pre-release condors (0.8296–0.8351) were significantly different than free-flying condors with blood lead above 0.0375 ppm (0.8101–0.8307), indicating that higher lead concentrations were associated with exposure to a novel source of lead and perhaps shifting downwards towards ratios associated with ammunition.

Whereas Church et al. (2006) contend that the narrow range of isotopic ratios measured in ammunition purchased locally for their analysis is representative for California condor exposure, subsequent studies expanded this range by testing a more expansive set of ammunition. Finkelstein et al. (2012) collected ammunition from an exchange program, hunters, or from shot carcasses and found an isotopic range of approximately 0.78–0.87 $^{207}\text{Pb}:$ ^{206}Pb . Results from condor blood analyses in this study were similar to Church et al. (2006) in that condors exposed to greater lead concentrations showed a decline in isotopic ratios. Isotopic ratios ranged from 0.8296 to 0.8483 in pre-release condors and 0.7602–0.9145 in wild condors. Lead exposure for five birds at the high end of the range (>0.9) was attributed to lead-based paint from a fire lookout tower where condors had been observed roosting (for further discussion, see below: Alternative Sources of Lead: Paint). Lead fragments recovered from six condors that were either lead poisoned or observed feeding on a carcass shot with lead-based ammunition had isotopic ratios that were similar to blood collected from the same bird. The isotopic range of the fragment/blood pairs were approximately 0.81–0.83, overlapping slightly with ratios of pre-release condors. Nine of the 110 condors sampled had isotopic ratios that could not be explained by background, ammunition, or paint.

Ranges of $^{207}\text{Pb}:$ ^{206}Pb ratios were relatively wide in femurs of black vultures (0.8055–0.8813, N=98) and turkey vultures (0.8121–0.8513, N=10) collected from Virginia between July 2011 and May 2012 (Behmke et al. 2015). The authors compared these ratios with those obtained from literature from several sources and found overlap in varying degrees with those measured for ammunition, leaded gasoline, coal emissions, and zinc smelting. Comparisons of liver and bone lead concentrations in these vultures suggested that all had long-term exposure to lead, and few had significant recent exposure.

Isotopic Analysis in Feathers

To examine lead concentrations and isotope ratios in condors, Church et al. (2006) collected one retriex feather that was partially grown at the time of death from a carcass. This condor had been in the wild approximately 2.5 years before its carcass was recovered and was subsequently diagnosed with lead poisoning. Following rinsing in detergent, water, ethanol, and nitric acid, the feather was serially sampled along the length of the rachis and vane. As rinsing may not remove all of the atmospherically deposited lead from a feather, distinguishing the contribution of this exposure route, where possible, helps to better characterize lead exposure from ingestion. Since condor feathers can grow about ~5 mm per day, the 24 cm feather

was likely to have been growing 1–2 months. As condors molt on an approximate 2-year cycle (Snyder et al. 1987), this new feather would have been subject to a relatively low extent of external contamination compared to feathers formed earlier. However, it was unknown how much time elapsed between the condor's death and its discovery that may have added to the feather's exposure to the environment. Results revealed the lowest lead concentrations in the oldest part of the feather, rising sharply in the youngest part of the feather to reach values several times the original concentrations. This pattern of lead concentrations is unlikely to have arisen from external contamination, which would produce a pattern opposite to that seen here, with lead concentrations highest in the outermost, or oldest part of the feather. In addition, the increase in lead levels corresponds with a distinct change in isotopic ratios, indicating an exposure to a novel source of lead during a specific time period. These ratios are similar to those found in liver and kidney of the carcass, where exposure is limited to ingested lead. For these reasons, the source of lead measured in this feather is likely to be from ingested lead deposited in the growing feather, and not external deposition.

Notable in this feather, isotopic ratios of $^{207}\text{Pb}:^{206}\text{Pb}$ increased with lead levels. Ratios associated with lead concentrations below 40 ppm dry weight ranged from approximately 0.720 to 0.790, markedly different than reported ratios from environmental sources or food items reported by the authors (0.8253–0.8453). A spike to lead concentrations of 50 ppm dry weight in the rachis and 90 ppm dry weight in the vane was associated with isotopic ratios of 0.800–0.810. While these values are consistent with ratios found in ammunition, changes in isotopic ratios can also generally be associated with environmental ratios of lead as well as dietary items of condors. However, as lead shot was recovered post-mortem from the gastrointestinal tract, this direct evidence strongly suggests that exposure to ammunition contributed to elevated lead levels.

Finkelstein et al. (2010) expanded upon the work of Church et al. (2006) with more extensive analysis of lead in the feathers, blood, and tissue of six condors chosen to represent three scenarios: a well-documented lead exposure event (condors observed feeding on a carcass from which lead bullets were recovered), fatalities attributed to lead poisoning, and routine exposure monitoring. For two condors with known exposure, feathers showed a spike in lead concentrations and a drop in isotopic ratios that corresponded with the timing of the feeding event. Isotopic ratios of bullet fragments recovered from the scavenged carcass were similar to ratios in blood collected near the time of feeding. For the two condors that died from lead poisoning, feathers concentrations showed a spike at the time of poisoning that corresponds with a change in isotopic ratios. However, for one condor the isotopic ratio increased rather than declined, indicating that exposure may have derived from a source other than ammunition. In each case, isotopic ratios in blood, liver, and kidney corresponded with the acute exposure event, and ratios in bone (the site of long-term storage of lead) corresponded to pre-release ratios. These findings together indicate a change in both the concentration and source of lead. Of three feathers collected during routine monitoring, two had increased lead concentrations that corresponded with shifts in isotopic ratios, while the third gave ambiguous results,

showing decreasing lead concentrations from older to newer parts of the feather, with no corresponding change in isotopic ratios.

If atmospheric deposition had been a source of lead in these feathers, a pattern of increasing concentrations in older parts of feathers would have been expected, as well as a corresponding isotopic signature. For most of the feathers analyzed in these studies, acute rises in concentration tended to correspond with ratios that were lower than known environmental sources. Abiotic sources (dust, snow-fed lake water, urban aerosols, environmental lead, river water) measured in California cited by Church et al (2006) fell within the range of 0.8338–0.8453. This is similar to atmospheric concentrations measured in California cities in the 1990s (0.8418–0.8628; Bollhöfer and Rosman 2001). While this represents only a small sampling of atmospheric lead, it is noteworthy that spikes in lead concentrations measured in feathers tended to drive isotopic ratios below the ranges measured in these studies.

Key Points: Association of Lead Exposure to Spent Ammunition

Temporal association with hunting season

- Seasonality can play an important role in lead exposure as birds opportunistically forage on readily available food sources such as hunter-killed deer and offal piles.
- Many studies found an increased rate of lead exposure and mortality during hunting seasons.

Detection of ammunition

- Several studies report retrieval of lead shot or bullet fragments at necropsy, detection on radiographs, or observation of birds feeding on lead contaminated carcasses.
- The absence of a lead fragment does not negate the possibility of ammunition as the source of exposure due to the possibility that a fragment has been passed, regurgitated or completely dissolved, or because this evidence was not sought by an investigator.

Isotopic ratios in lead-exposed birds

- When used with other lines of evidence such as observations, behavioral ecology, or recovery of ingested items, measurement of isotopic ratios can provide information on the source of lead (e.g., ammunition, dietary items, paint) measured in biota or environmental samples.
- Isotopic ratios can vary for any particular source of lead and measured ranges tend to increase with sample size. Ranges from different sources can overlap.

(continued)

(continued)

- Isotopic ratios in blood of exposed birds will be similar to a known source of exposure (e.g., bullet fragment or paint chip removed from bird).
- Changes in isotopic ratios in avian tissues can reveal when a novel source of lead has been introduced into the diet. A noteworthy example is the decline in ^{207}Pb : ^{206}Pb ratios associated with increased lead exposure as condors transition from captivity to the wild.
- Sampling across the length of a feather can provide a more refined view of lead exposure over time, as changes in isotopic ratios show when an elevation or decline in lead concentrations is associated with a change in the source of lead.

In a study of Andean condor feathers from the Argentine Patagonia region, Lambertucci et al. (2011) compared lead concentrations and isotopic ratios to seven types of ammunition obtained from retail stores and hunters in the study area. Molted feathers (N=152) were collected from the base of cliffs where condors congregate to roost. Isotopic ratios from ammunition fell into two ranges, averaging 0.817 for big game hunting, and 0.857 for hare hunting. The higher isotopic ratios were indistinguishable from background ratios for this region, and most feathers with low concentrations fell within this range. While isotopic ratios in ammunition were not predictive of lead concentrations in feathers, isotopic ratios differed between feathers with high (>4 ppm dry weight) and low (<4 ppm dry weight) concentrations, indicating that elevated lead resulted from a novel, possibly anthropogenic, source of lead. While feathers were rinsed prior to analysis, the possibility of external contamination was not examined by the authors.

8 Alternate Sources of Lead as Potential Exposure Routes

While multiple lines of evidence support the conclusion that scavenging birds are exposed to lead by ingesting spent ammunition, there are numerous sources of lead that contribute to its availability on the landscape. Herein, we examine several alternate sources of lead and evaluate the likelihood of exposure for scavenging birds.

8.1 Fishing Sinkers and Lures

Leaded fishing gear (lures, sinkers, weights) used in recreational fishing activities can be discarded in lakes or other waterways, where it may be incidentally ingested by waterbirds, most notably the common loon (*Gavia immer*). Lead poisoning from

fishing lures and sinkers can account for 10–50 % of adult loon mortality where high populations of loons overlap with recreational fishing activity (Scheuhammer and Norris 1996). Common loons submitted to the Wisconsin Department of Natural Resources for necropsy revealed that 30 % had lead poisoning, all of which had lead fishing lures or sinkers recovered from their gastrointestinal tracts (Strom et al. 2009).

Scavenging birds may be exposed to lead from these sources by feeding on waterbirds or fish that have ingested lead lures or sinkers. However, evidence of this route of exposure is limited in the scientific literature. One study in Canada found a bald eagle that had ingested a lead fishing weight (Langelier 1994, cited in Scheuhammer et al. 2003). Piscivorous species that forage in waterways are likely to be at greater risk of lead exposure from discarded lead fishing gear than scavenging raptors.

8.2 *Microtrash and Other Metal Objects*

The ingestion of metal objects by birds resulting in lead exposure that cannot be positively attributed to ammunition or fishing sinkers has been rarely documented. One notable case was in an endangered Mississippi sandhill crane (*Grus canadensis pulla*) that was diagnosed with lead poisoning based on necropsy findings, liver concentrations of 69–70 ppm wet weight, and an unknown lead object retrieved from the gizzard (Franson and Hereford 1994).

One scavenging species that has been documented to ingest a variety of foreign anthropogenic material, or “microtrash,” is the California condor. These objects can be collected from condor nests, where adults regurgitate ingested material for the consumption by nestlings. While it has been hypothesized that condors may not collect trash until feeding nestlings (Walters et al. 2008), there have been no systematic searches of roost sites to look for evidence of regurgitated shot or microtrash during non-breeding periods (Mee et al. 2007). However, inspection of trash from nest sites shows the spectrum of materials collected by the birds.

Nests of seven condor pairs, representing 11 breeding attempts, were monitored from 2001 to 2005 (Mee et al. 2007). Nest-floor substrates were collected at the end of breeding attempts or opportunistically during nest visits using a fine-mesh window screen that allowed for collection of material greater than 1 mm². Of 650 microtrash items recovered, 148 (22.8 %) were metallic, including items such as bottle-tops, washers, ammunition casings, and electrical wiring (Mee et al. 2007). No objects that could be associated with alternate sources of lead (e.g., wheel weights) were specifically identified. Necropsies were performed on 13 dead nestlings from the California population between 2001 and 2009 (Rideout et al. 2012). Of these, livers were analyzed from six nestlings that were either diagnosed with trash ingestion as the cause of death, or contained microtrash. None had detectable lead concentrations despite the presence of metallic objects such as nuts and washers

(Rideout et al. 2012). Though these studies represent only a subset of condors, the analyses show no evidence of exposure to lead objects in the accumulation of microtrash.

The degree of trash ingestion by condors appears to vary between the California and Arizona populations. Trash ingestion by chicks in Arizona is rare and generally does not contribute to mortality (Walters et al. 2008). The carcass of one chick collected in 2011 contained microtrash, but it was not believed to be the cause of death (USFWS 2012). One juvenile (5 years old) and one adult (11 years old) from the Arizona population were reported to have died from trash ingestion between 1996 and 2010; neither had detectable levels of lead (Rideout et al. 2012). The geographic difference in trash ingestion has been hypothesized to be a function of foraging range and trash density and distribution. Condors in the Arizona population forage in areas that are more remote from anthropogenic activity and may contain less trash (Walters et al. 2008). Although condors of both populations have been observed ingesting metal objects, these cases are few and not a plausible explanation for the widespread lead exposure that occurs in this species.

8.3 *Paint*

Ingestion of lead-based paint from buildings has been documented as a source of lead exposure for birds. Perhaps the most widespread and studied instance of this exposure occurred on Midway Atoll, where lead poisoned Laysan albatross (*Phoebastria immutabilis*) chicks were associated with proximity to historical buildings and discovered with paint chips in the proventriculus (Sileo and Fefer 1987; Work and Smith 1996). Finkelstein et al. (2003) performed lead isotope analysis on blood lead from asymptomatic and symptomatic chicks on Midway, as well as from soil and paint chips found in or near the nests of sampled chicks. While there was wide variation in isotopic composition of samples in both blood and paint, ratios from blood of chicks exhibiting droopwing, a sign of lead toxicosis, were significantly related to the ratios of lead in paint chips collected from nests. No relationship was found between isotopic ratios of blood and soil samples. Few other examples exist of birds exposed to lead through this route. In an isolated incident, a group of 13 captive sandhill cranes (*Grus canadensis*) exhibited signs of lead poisoning after being moved to a facility that was later discovered to have lead-based paint on the walls, though this event occurred indoors with a non-wild population (Kennedy et al. 1977).

Lead-based paint from an inactive fire lookout tower has been identified as a source of lead exposure for a subset of California condors (N=5; Finkelstein et al. 2012). These cases were confirmed both by observations of condors roosting on or near the tower, and by matching isotope ratios between blood of these condors and paint chips sampled from the tower (Finkelstein et al. 2012). Lead-based paint can have a wide range of lead isotope ratios (Rabinowitz and Hall 2002; Finkelstein et al. 2003). However, by matching isotopic ratios from blood of lead-exposed birds

to the specific source suspected of this exposure based upon condor movements and behavior, it was possible to narrow the range of isotopes to the paint used on the roosting tower. Outside of this somewhat unique exposure pathway, there are no other clear pathways for condors to ingest lead-based paint that are likely to elude detection, considering that condors are highly studied and tracked through both radio transmitters and visual observation (Cogan et al. 2012). Condor association with fire lookout towers in central California is a rare occurrence, as indicated by tracking data (Finkelstein et al. 2012), and reports are not available of condors associating with other historical structures. Frequent exposure to lead-based paint appears implausible in consideration of known behavior. In addition, no examples are available from the scientific literature of birds achieving elevated lead concentrations from chronic low-level exposure to various sources of lead-based paint; each example has been tied to a known source providing the exposure (i.e., buildings on Midway, fire lookout tower).

8.4 Mine Tailings

Lead mines exist throughout the nation, with the largest concentration located in southeastern Missouri, known as the “Old Lead Belt”. The process of extracting lead from the earth produces waste tailings that are typically deposited on the landscape if environmental clean-up or storage measures are not followed or in place. Lead tailings have polluted water and sediment in lakes and rivers, and soil in terrestrial habitats. Lead exposure to wildlife can occur when there is direct contact with the tailings dermally or by ingestion, or by consumption of prey items exposed as such.

One of the best known examples of lead poisoning to waterfowl from mine waste is in the Coeur d’ Alene River basin in northern Idaho (Chupp and Dalke 1964). Lead from mining and smelting activities in the Coeur d’ Alene River system in northern Idaho was associated with mortality in tundra swans (*Cygnus columbianus*) that ingested lead-contaminated sediment and plants (Blus et al. 1991). A more recent publication found that lead-contaminated sediment was the cause of mortality in 77 % of 285 waterfowl (mostly tundra swans) found sick or dead from 1992 to 1997, with an additional 7 % of deaths attributed to lead poisoning from ingested lead shot (Sileo et al. 2001).

Waterfowl are thought to be the most common species exposed to lead from mine waste because of their physiology and foraging behavior; however, passerines can be at risk through ingestion of soil and soil-ingesting invertebrates. Hansen et al. (2011) investigated lead exposure from mine wastes and soil ingestion rates of three species of ground-feeding songbirds, American robin (*Turdus migratorius*), Swainson’s thrush (*Catharus ustulatus*), and song sparrow (*Melospiza melodia*), at the Coeur d’Alene River basin. The highest soil ingestion rates and the highest blood lead concentrations were found in the American robin at the most contaminated sites. At these sites, 18 % of 204 American robins had severe poisoning

(>29.4 ppm dry weight), 52 % had clinical poisoning (17.6–29.4 ppm dry weight), and 24 % had subclinical poisoning (5.9–17.6 ppm dry weight). Soil ingestion accounted for almost all of the species' exposure to lead. Beyer et al. (2013) analyzed lead concentrations in soil, earthworms, and songbirds at lead mining and smelting sites in southeast Missouri's Lead Mining District. Lead concentrations in soil sampled from three mining/smelting sites ranged from 1000 to 3000 ppm dry weight, and directly correlated with lead concentrations in earthworms. For six species of songbirds analyzed at the lead mining/smelting sites, lead concentrations were greater in blood by a factor of 8, in liver by a factor of 13, and in kidney by a factor of 23 compared to the reference site. American robins, which are known to feed on earthworms, had lead concentrations up to 1000 ppm dry weight in kidney.

The likelihood of scavenging birds to directly ingest mine tailings is low. However, secondary exposure from dietary sources has been documented, though rarely. For example, blood samples collected from Eurasian eagle owl chicks (*Bubo bubo*) between 2003 and 2007 were significantly higher at a site contaminated from an abandoned mine than at a reference site, suggesting dietary exposure to lead (Gómez-Ramírez et al. 2010). In the Coeur d'Alene River basin, samples collected from adult and nestling osprey (*Pandion haliaetus*), American kestrels (*Falco sparverius*), northern harriers (*Circus cyaneus*), red-tailed hawks (*Buteo jamaicensis*), great horned owls (*Bubo virginianus*), and western screech-owls (*Otus kennicotti*) contained lead concentrations (Henny et al. 1991, 1994) suggesting these individuals had foraged on lead-contaminated fish and other wildlife (Henny et al. 1991, 1994). However, no effects to behavior, reproduction, or survival were detected in the lead-exposed raptors. The authors suggest that raptors have traits that can reduce their potential for accumulating critical levels of lead which is primarily stored in bones of prey species (Henny et al. 1994). For birds exposed via secondary consumption, lead that is biologically incorporated by prey may be less bioavailable due to partitioning into bone (Custer et al. 1984). In a study of American kestrels fed cockerels that were experimentally treated with lead, kestrels accumulated concentrations in tissues, but exhibited no effects on survival, body mass, or hematological endpoints (Custer et al. 1984). The authors concluded that biologically incorporated lead alone was unlikely to cause lead poisoning.

8.5 Shooting Ranges

Shooting ranges, either military or for recreation, are a source of spent lead ammunition and soil contamination. There are approximately 3000 military shooting ranges and 9000 non-military sites in the United States (USEPA 2005). Birds foraging in these areas or in wildlife habitats adjacent to the shooting ranges can be directly exposed by the uptake of spent shot incidental to grit ingestion, or by ingesting contaminated soil or food. A study investigating the toxicity of lead in soil collected from a small arms-range found that rock doves accumulated lead in the blood, tissues, and feathers when dosed with soil, with an accompanying increase in erythrocyte protoporphyrin at blood concentrations >0.5 ppm (Bannon et al. 2011).

Species most likely to be at risk through direct ingestion of spent lead or contaminated soil on or near shooting ranges are waterfowl, mourning doves, and other species that forage on the ground or use grit to help aid in digestion (i.e., quail, grouse, pheasant). Lead poisoning has been documented in northern pintails that foraged in a tidal meadow contaminated with lead from a trap and skeet shooting range (Roscoe et al. 1989). Greater lead exposure was found in passerines that foraged on the ground near a small-arms range as compared to those in a wildlife habitat a distance from the range (Vyas et al. 2000). The likelihood of scavenging birds that normally feed on live or dead animals to directly ingest contaminated soil or spent lead shot from the ground is low. Scavengers could become exposed through consumption of prey items that have directly ingested spent lead shot or contaminated soil from shooting ranges; however, no examples of this resulting in lead poisoning were found in the literature.

Key Points: Alternate Sources of Lead as Potential Exposure Routes

- Numerous sources of lead contribute to its availability on the landscape, including lead fishing tackle, microtrash, lead-based paints, mining operations, and shooting ranges.
- There are few documented cases of lead poisoning to scavenging birds from alternate exposure routes as compared to lead ammunition.
- These exposure pathways cannot be entirely ruled out as potential sources of lead to scavenging birds; however, foraging habits and behavioral traits of scavenging birds make these improbable as sources of widespread exposure.

9 Toxicity of Alternative Metals Used in Ammunition

The use of alternative metals in ammunition has been proposed, and in some cases implemented (e.g. within the range of the California condor), as a means of breaking the exposure pathway of scavenging birds to lead, provided that ammunition derived from these metals is non-toxic (i.e., does not cause toxicity or death when ingested by migratory birds; USFWS 2013a).

9.1 Non-toxic Shot Approval Process

To determine the types of ammunition that are non-toxic and safe to use for waterfowl hunting, a comprehensive testing method was developed for the registration of alternative shot and shot coatings (Rattner and Morehouse 1994). The protocol, formalized and implemented in 1997, requires manufacturers to abide by a

three-tiered test method in order for the USFWS to consider approval of any proposed non-toxic candidate material (USFWS 1997). Tier 1 requires existing data to be compiled on (1) the physical and chemical characterization of the shot or coating, (2) any existing ecological risk assessments and toxicity information of the candidate material, and (3) available tests that determine the effects on the reproduction of waterbirds. Tier 2 requires erosion rate testing and acute toxicity testing on mallards, invertebrates, and early life stage vertebrates to assess potential impacts on waterfowl habitat. Tier 3 requires chronic exposure tests to mallards under adverse environmental conditions to determine effects on reproduction. Based on the Tier 1 information, the USFWS can approve or deny the candidate material or require further testing in the Tier 2 and Tier 3 requirements (USFWS 1997).

Based on toxicity testing and results from the shot approval process, the USFWS established a maximum environmentally acceptable level of lead in shot as <1 % (USFWS 1995), and steel was the first non-toxic shot approved following the three tiered testing requirement (USFWS 1999). To date, the testing has resulted in 12 approved non-toxic shot types, including different combinations of tungsten, bismuth, tin, iron, copper, and nickel (Table 5), illustrating that there are suitable alternatives to lead that present limited threats to waterfowl and species that consume waterfowl (USFWS 2013a). The testing requirements remain the current method used to approve the registration of non-toxic shot for hunting purposes (USFWS 2013b). While these protocols do not include testing of other forms of ammunition (e.g., bullets), results from studies performed under the approval process can provide data on the toxicity of metals to birds treated under similar conditions that may be suitable for extrapolation to other ammunition types.

Table 5 Shot types approved as nontoxic for waterfowl hunting in the United States

Approved shot type ^a	Percent composition by weight
Bismuth-tin	97 bismuth, and 3 tin
Iron (steel)	Iron and carbon
Iron-tungsten	Any proportion of tungsten, and ≥ 1 iron
Iron-tungsten-nickel	≥ 1 iron, any proportion of tungsten, and up to 40 nickel
Tungsten-bronze	51.1 tungsten, 44.4 copper, 3.9 tin, and 0.6 iron, or 60 tungsten, 35.1 copper, 3.9 tin, and 1 iron
Tungsten-iron-copper-nickel	40–76 tungsten, 10–37 iron, 9–16 copper, and 5–7 nickel
Tungsten-matrix	95.9 tungsten, 4.1 polymer
Tungsten-polymer	95.5 tungsten, 4.5 nylon 6 or 11
Tungsten-tin-iron	Any proportions of tungsten and tin, and ≥ 1 iron
Tungsten-tin-bismuth	Any proportions of tungsten, tin, and bismuth.
Tungsten-tin-iron-nickel	65 tungsten, 21.8 tin, 10.4 iron, and 2.8 nickel
Tungsten-iron-polymer	41.5–95.2 tungsten, 1.5–52.0 iron, and 3.5–8.0 fluoropolymer

^aCoatings of copper, nickel, tin, zinc, zinc chloride, and zinc chromate on approved nontoxic shot types also are approved

9.2 Toxicity of Alternative Metals

The tiered testing protocol for the approval of non-toxic ammunition provides comparable data on the toxicity of different metal alloys to mallards. In general, the results of these tests found combinations of tungsten, tin, bismuth, iron, and copper to provide limited threats based on a number of health metrics. Studies found no adverse effects on mallards dosed with bismuth-tin and steel (Sanderson et al. 1997), tungsten-iron, tungsten-polymer, and steel (Kelly et al. 1998), and tungsten, tin, and bismuth (Ringelman et al. 1993). Additionally, Sanderson et al. (1997) found no effects on blood chemistries, body and kidney weight, livers, gonads, or gizzards for mallards dosed with bismuth-tin and steel. However, mild impairment of bile flow was observed in mallards in a study that tested tungsten alloys (tungsten-iron and tungsten-polymer; Kelly et al. 1998). Ringelman et al. (1993) suggested that there is no uptake of tungsten, tin, or bismuth to mallard tissues, and study results found no changes in 23 hematology and blood parameters measured. For toxicity tests with iron, mallard mortality rates were low and weight losses were not significantly different from control groups (Irby et al. 1967). Locke et al. (1967) investigated the histopathological response of mallards dosed with uncoated and coated iron shot and also found limited effects. Mallards fed iron, molybdenum coated iron, and zinc coated iron developed hemosiderosis of the liver (Locke et al. 1967).

The mixture of tungsten-tin-bismuth has been tested in scavenging birds, with similar results as those found in the mallard toxicity tests. Risebrough (2001) tested a composite of tungsten-tin-bismuth on turkey vultures trapped from the wild. Tungsten and bismuth did not accumulate in tissues, although tin concentrations increased in the blood. There was no change in body mass, hematology, plasma biochemistries, or histopathology between dosed and control vultures. Krone et al. (2009) investigated the toxicity of brass and zinc to Pekin ducks (*Anas platyrhynchos domestica*) and found no mortality or organ alterations; however, the ducks dosed with zinc showed a higher weight loss compared to ducks dosed with brass, and zinc showed the highest solubility in duck gizzards.

Birds are generally tolerant to metallic copper, and multiple dosing studies found no mortality or clinical signs of toxicity and limited biochemical effects (Bellrose 1965; Irby et al. 1967; Locke et al. 1967; Krone et al. 2009; Bannon et al. 2011; Franson et al. 2012). Toxicity tests on mallards with metallic copper found no mortality in the dosed group (Irby et al. 1967). Locke et al. (1967) investigated the histopathological response of mallards dosed with copper shot, finding no lesions in tissues, no renal acid-fast inclusion bodies, and no hemosiderin in the liver. Bellrose (1965) tested different weights of single copper shot and dosed mallards lost 15–20 % of their initial weight by day 35 but recovered by day 110. The mallards in the copper tests were given doses over the limit of what they would normally be exposed to in the wild and several times the dose used in previous lead shot tests, suggesting that the quantities of normally ingested copper in the wild would result

in lesser effects. Bannon et al. (2011) investigated the toxicity of copper to rock doves that were exposed to small arms-range soil contaminated by spent ammunition from copper jacketed bullets with a lead core, reporting no copper retention in the tissues or any adverse effects. Similarly, Krone et al. (2009) investigated the toxicity of copper to Pekin ducks and found no mortality or organ alterations in the dosed group. Additionally, the ducks dosed with copper did not show weight loss even after 4 weeks of shot retention in the gizzard.

Tests of metallic copper on raptors and scavenging species found similar results. Franson et al. (2012) tested copper shot on captive raised American kestrels and found no mortality or clinical signs of toxicity in the dosed birds. In addition, biochemistries, hematocrit values, and copper concentrations in the kidney and plasma did not differ between dosed and control birds. Copper concentrations in liver were greater in dosed birds, and were correlated with metallothionein concentrations. The authors suggested that birds sharing similar regurgitation frequency as American kestrels are not likely to be adversely affected by copper ingestion. Risebrough (2001) investigated the toxicity of copper to turkey vultures that were trapped from the wild. The vultures were fed food dosed with copper at a level that was considered equivalent to the ingestion of two copper bullets. The study found no mortality; however, the copper levels were greater in the livers of dosed vultures compared to those in the control group.

Although metallic copper does not seemingly present a health threat to birds, the salts of copper have been found to exhibit toxicity in selected cases. Kobayashi et al. (1992) investigated a die-off of free-ranging mute swans with high copper concentrations in liver tissue, and attributed the deaths to copper poisoning of unknown source. Henderson and Winterfield (1975) recorded two cases of copper poisoning in Canada geese; copper sulfate used as an algicide was the suggested cause.

Tissue concentrations of copper can vary widely among individuals and species, and can reach seemingly elevated levels without detectable effects to individuals. For example, copper concentrations in the livers of California condors ranged from 2.2 to 531 ppm wet weight at necropsy (Rideout et al. 2012). None of these birds were diagnosed with copper toxicosis. In American kestrels dosed with copper shot the mean liver concentration was 20.7 ppm dry weight (Franson et al. 2012). Multiple studies report much higher concentrations in liver, notably mute swans and common eiders. The copper concentration in the livers of three mute swans found dead in Mamaroneck Harbor, New York, ranged from 1562 to 5857 ppm dry weight, while concentrations in two captive mute swans were much lower, 64 and 121 ppm dry weight (Molnar 1983). The copper contamination was suspected to be from antifouling paint released in water through the scraping of vessel hulls. Clausen and Wolstrup (1978) and Kobayashi et al. (1992) also detected high concentrations in mute swans, and other studies found copper concentrations of over 1000 ppm wet weight in the livers of common eiders (Norheim and Borch-Iohnsen 1990; Hollmén et al. 1998; Franson et al. 2000; Stout et al. 2002). Because of this variability, interpretation of copper concentrations in tissue should be done with caution, as seemingly elevated levels do not necessarily signify adverse effects.

Key Points: Toxicity of Alternative Metals used in Ammunition

- A comprehensive testing method exists in the United States to ensure alternative metals used to hunt waterfowl do not cause sickness and death when ingested by migratory birds. These data can be extrapolated to ammunition used for other forms of hunting.
- The non-toxic shot approval process has resulted in the approval of suitable alternatives to lead that present limited environmental threats.
- Copper, the primary alternative currently used in bullets, exhibits low toxicity to birds in its metallic form.

10 Conclusion

Although there are multiple sources of lead in the environment, scientific evidence points to spent lead ammunition as the most frequent cause of lead exposure and poisoning in scavenging birds. Numerous lines of evidence support this conclusion, including the extent of lead ammunition currently used for hunting and its tendency to fragment, the behavioral ecology and physiology of scavenging birds, their susceptibility to lead as exhibited in controlled dosing studies, the diagnosis of lead poisoning by well-established tissue thresholds and clinical signs, the recovery of ingested lead fragments or shot from exposed birds, observations of birds feeding on contaminated carcasses, isotopic analyses relating tissue concentrations to ammunition, patterns of mortality coincident with hunting seasons, and the lack of abundant evidence for other sources of lead. While few cases may exhibit all of these lines of evidence, there are numerous documented cases of lead poisoned scavengers in the literature and many are supported by one or more lines of evidence. Lead can be replaced in ammunition by alternative metals that are currently available and present limited environmental threats. Scientific literature shows spent lead ammunition to be the primary pathway for widespread lead exposure to scavenging birds in the United States. Reducing this route of exposure will result in the greatest alleviation of mortality and other adverse effects to these species from lead in the environment.

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