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16 Lead in Birds

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CONTENTS

16.1 INTRODUCTION

Lead is a highly toxic heavy metal that acts as a nonspecific poison affecting all body systems and has no known biological requirement. Absorption of low concentrations may result in a wide range of sublethal effects in animals, and higher concentrations may result in mortality (Demayo et al. 1982).

Lead has been mined and smelted by humans for centuries, but the use of lead-based products increased greatly following the Industrial Revolution. Consequently, lead today is ubiquitous in

air, water, and soil, in both urban and rural environments (Eisler 2000). Vertebrates are exposed to lead mainly via inhalation and ingestion. A proportion of lead entering the body is absorbed into the bloodstream and subsequently becomes distributed among body tissues, primarily the blood, liver, kidney, and bone. As a result of anthropogenic activities, most animals have higher tissue lead concentrations than in preindustrialized times. Although even very low tissue lead concentrations have some measurable physiological effects, the concentrations usually encountered in the wider environment (i.e., distant from lead emission sources) have not generally been considered to directly affect survival of most wildlife.

However, significant numbers of wild birds may be exposed to large amounts of lead through the ingestion of spent lead from ammunition (i.e., shotgun pellets, bullets, and fragments thereof). This source of exposure primarily affects birds in two groups. The first includes birds that ingest lead shotgun pellets, presumably as grit or food particles, in areas that are hunted over, including wetlands, farmland, terrestrial game shooting areas, and shooting ranges. The second includes birds that prey upon or scavenge the flesh of game species, or other hunted species, such as pests or predators. This latter group consists primarily of hawks, eagles, falcons, owls, vultures, and other scavenging birds that ingest lead from ammunition in carcasses of wildlife that have been killed but unretrieved or shot but survived, or that ingest lead bullet fragments embedded in the offal and other tissues of field-dressed game animals. Ingestion of lead from ammunition has resulted in widespread avian mortality in the United States, Europe, and elsewhere for over a century (Bellrose 1959, Sanderson and Bellrose 1986, Locke and Friend 1992, Pain 1992, Kendall et al. 1996, Kurosawa 2000, Fisher et al. 2006, Kreager et al. 2008, Mateo 2009, Pain et al. 2009). Consequently, the use of lead ammunition has been restricted in at least 29 countries around the world (Avery and Watson 2009). Lead restrictions have been introduced primarily due to concerns over lead poisoning in waterfowl or avian scavengers. The most common restriction is a ban on the use of lead gunshot for shooting waterfowl and/or over wetlands, although a few countries have banned the use of lead shot for all hunting (Avery and Watson 2009). The ingestion of lead fishing weights also has resulted in lead exposure and mortality in waterfowl, particularly swans, common loons (*Gavia immer*), and a variety of other waterbirds (Sears 1988, Blus et al. 1989, Franson et al. 2003, Scheuhammer et al. 2003b, Sidor et al. 2003, Pokras et al. 2009).

Nonparticulate lead exposure is frequently the result of the ingestion of sediments containing lead, particularly at locations surrounding mines and smelters, but also occurs from the consumption of lead-contaminated prey (Beyer et al. 1985, Henny et al. 1994, Johnson et al. 1999, Sileo et al. 2001, Beyer et al. 2004, Bostan et al. 2007). In addition, lead exposure of birds has occurred in urban environments from airborne sources (Ohi et al. 1974, Hutton 1980, Hutton and Goodman 1980, Ohi et al. 1981, Grue et al. 1986, Nam and Lee 2005, Scheifler et al. 2006, Roux and Marra 2007) and from the ingestion of paint chips near buildings painted with lead-based paint (Sileo and Fefer 1987, Work and Smith 1996, Finkelstein et al. 2003).

The primary focus of this chapter is the interpretation of tissue lead concentrations resulting from exposure to metallic lead, including lead shotgun pellets, bullets, bullet fragments, and fishing weights. However, the guidelines provided can also be applied to evaluating the effects of other sources of lead exposure in birds, such as contaminated sediments, airborne emissions, and lead-based paints.

Research on lead poisoning of birds, under both experimental and field conditions, has been conducted since the 1950s, when Bellrose (1959) completed the first comprehensive study of lead poisoning in waterfowl. More recently, lead poisoning has been investigated in many additional species of aquatic and terrestrial birds. This chapter draws upon this research, covering the distribution of lead within the body, factors that influence tissue lead concentrations, and the effects of lead poisoning. The concentrations of lead found in tissues of unexposed and lead-poisoned birds are discussed, along with the threshold tissue concentrations that are considered to indicate excessive lead absorption or poisoning. The chapter concludes with suggested thresholds for tissue lead concentrations indicative of different levels of exposure and poisoning and discusses factors associated with their interpretation at both individual and population levels.

16.2 NOTES ON TERMINOLOGY

1. "Natural" environmental concentrations no longer exist, because lead resulting from anthropogenic emissions is ubiquitous. Concentrations in the wider environment far from lead emission sources have consequently been described as "background." The magnitude of such "background exposure" in birds varies according to the specific geographic areas used throughout the life cycle. "Elevated exposure" is that resulting from exposure to above-background concentrations.
2. Lead exposure can be characterized according to duration as acute (exposure over a short period of time) or chronic (sustained exposure), and magnitude, depending on the amount of lead the animal is exposed to. However, "acute" is generally used to describe a high magnitude of exposure over a short time period, and "chronic" to describe exposure over a more protracted time period, often at a lower level.
3. Residues from the literature that were reported as parts per million (ppm) were converted to milligram per kilogram and expressed on a wet weight or dry weight basis when specified as such. Blood lead data reported in the literature as ppm, μg/g, or mg/kg were assumed to be on a wet weight basis. For reference, 1 μg/g = 1 mg/kg and is equal to 1 ppm. In blood, 1 ppm lead is approximately equal to 1 μg/mL, 1 μg/g, or 100 μg/dL. Interpretive threshold concentrations for soft tissues are given on wet weight basis, as is commonly done, although reporting on a dry weight basis is often recommended to control for variation in moisture content (Adrian and Stevens 1979). Unless otherwise noted, conversions between wet and dry weight concentrations were calculated using moisture levels reported for mallard (*Anas platyrhynchos*) tissues by Scanlon (1982). Thus, 1 μg/g wet weight equals approximately 4.6 μg/g dry weight for blood, 3.1 μg/g dry weight for liver, 4.3 μg/g dry weight for kidney, and 1.2 μg/g dry weight for bone.

16.3 LEAD DISTRIBUTION AMONG AVIAN TISSUES

Ingested lead shot, bullets, fishing weights, or fragments from these are subject to dissolution by acids in the avian stomach, as well as mechanical grinding in those species that retain grit in their muscular gizzards. The resultant toxic lead salts are absorbed into the bloodstream. However, retention of lead particles, such as shot, is variable. They may be evacuated immediately or soon after ingestion with little or no absorption of lead, retained and partially eroded/dissolved with significant lead absorption, or retained until they are completely eroded/dissolved and absorbed. For example, all black ducks (*Anas rubripes*) that died 4–6 days after being dosed retained the lead shot that was administered (Pain and Rattner 1988). However, if waterfowl that have ingested shot survive long enough, most lead shot disappear from the gizzard within about 20 days, either because it has passed through the gastrointestinal tract or has been eroded (Franson et al. 1986, Sanderson and Bellrose 1986). In mourning doves (*Zenaida macroura*), 33–100% of administered lead shot were voided in 2 days (Schulz et al. 2007), while another study reported that up to 95% of shot were voided within 3 weeks (Marn et al. 1988). In a 211-day study of lead poisoning in turkey vultures (*Cathartes aura*), some of the birds were redosed because lead shot was regurgitated or defecated, whereas regurgitation occurred only once in Andean condors (*Vultur gryphus*) and they retained the administered lead shot for 39–49 days (Carpenter et al. 2003, Pattee et al. 2006).

Once absorbed, some of the lead in the bloodstream is deposited rapidly into soft tissues, primarily the liver and kidney, bone, and in growing feathers. Relative concentrations in different tissues depend on time postexposure and absorption. However, in general, the highest lead concentrations are found in bone, followed by kidney and liver, with intermediate concentrations in brain and blood, and the lowest concentrations in muscle (Longcore et al. 1974b, Johnson et al. 1982, Custer et al. 1984, Garcia-Fernandez et al. 1995). In lead-exposed birds, lead concentrations are often greater in kidney than liver of many species (Longcore et al. 1974b, Custer et al. 1984, Beyer et al. 1988,

Marn et al. 1988, Carpenter et al. 2003, Beyer et al. 2004, Pattee et al. 2006, Schulz et al. 2006). However, the reverse has been reported for eagles and some other raptors (Pattee et al. 1981, Wayland et al. 1999, Iwata et al. 2000, Kenntner et al. 2001, Krone et al. 2004, Battaglia et al. 2005, Martin et al. 2008).

In cases of acute exposure, lead in blood and soft tissues retains a fairly mobile equilibrium and usually remains elevated from several weeks to several months following exposure, in relation to the initial amount absorbed. When exposure is chronic blood lead will remain elevated for proportionately longer. Lead in bone is relatively immobile, loss is very slow, and lead accumulates in bone throughout the lifetime. However, the dynamics of lead absorption and release appear to be accelerated in the medullary bone of egg-laying females as described later. Bone lead concentrations in bird populations generally increase with age, although they may be very high in young individuals subject to high levels of exposure (Stendell et al. 1979, Clausen et al. 1982, García-Fernández et al. 1997, Scheuhammer et al. 1999, Pain et al. 2005). The use of feathers for monitoring of metals in birds is predicated on the fact that metals are deposited during the period of feather growth, but a variety of factors can affect concentrations (Burger 1993). Lead concentrations in feathers of juvenile birds have been shown to be a reliable indicator of dietary exposure at the time of feather formation and to be correlated with lead in some tissues, particularly kidney and bone (Golden et al. 2003). However, the use of adult feathers for assessing lead exposure can be problematic because they are subject to the deposition of atmospheric lead for a longer time, and must be cleaned thoroughly before analysis. Pain et al. (2005) found that, even after cleaning, lead concentrations in the feathers of Spanish imperial eagles (*Aquila adalberti*) from museums were positively correlated with specimen age, suggesting that externally deposited lead can be difficult to remove. Fry and Maurer (2003) and Fry (2004) have proposed using lead analysis of portions of feathers along their length as indicators of lead circulating in the feather pulp at the time of growth in California condors (*Gymnogyps californianus*), relating the findings to feather growth rates to evaluate lead exposure over time.

The chronicity of exposure to lead has an important influence upon the concentrations of lead in various tissues of birds. In cases of chronic exposure, the highest lead concentrations are generally found in bone, with lower concentrations in soft tissues such as liver, kidney, and blood (Custer et al. 1984, Pattee 1984, Mautino and Bell 1986, Mautino and Bell 1987). However, when birds die following acute exposure after the ingestion and absorption of large amounts of lead, concentrations in kidney and/or liver may exceed those in bone. For example, in a die-off of lesser scaup (*Aythya affinis*), recent lead exposure and rapid poisoning were suspected as body weights were positively correlated with the number of shot in the gizzard, indicating that birds exposed to the most lead died before the effects of lead poisoning resulted in weight loss (Anderson 1975). Tissue lead concentrations were consistent with the rapid absorption of large amounts of lead, as mean lead concentrations were 46 mg/kg wet weight (c. 141 mg/kg dry weight) in liver, 66 mg/kg wet weight (c. 282 mg/kg dry weight) in kidney, and 40 mg/kg dry weight in bone (Anderson 1975). Concentrations of lead in liver and kidney of Canada geese (*Branta canadensis*) found during a lead poisoning die-off were more than twice as high as those in bone (Szymczak and Adrian 1978). In a study with bald eagles (*Haliaeetus leucocephalus*), three of four birds that died did so within 20 days after dosage with 10 or more No. 4 lead shot and lead concentrations in tissues, on a dry weight basis were approximately 2.5–10 times higher in liver and kidney than in bone (Pattee et al. 1981). Zebra finches (*Taeniopygia guttata*) that received lead acetate in drinking water for 38 days and mourning doves dosed with lead shot and held for 3 weeks also had higher mean concentrations of lead in kidney than in bone (Marn et al. 1988, Snoeijs et al. 2005). In another study with mourning doves, birds that died within 5 weeks after receiving 1–4 lead shot had kidney lead concentrations that were 1.3–2 times greater than concentrations in bone (Buerger et al. 1986).

Because lead is lost far more slowly from bone than from soft tissues, few birds sampled from wild populations have higher lead concentrations in soft tissues than bone. Clausen et al. (1982) found only 10% of mute swans (*Cygnus olor*) had high liver and low bone lead concentrations, whereas 31% had low liver and high bone lead concentrations. Franson et al. (2009) found that in

mourning doves shot by hunters, birds both with and without ingested shot had higher lead concentrations in bone than in liver. In 12 species of raptors, mean lead concentrations were consistently higher in bone than in liver (Martin et al. 2008).

Thus, bone lead concentration is generally considered the best indicator of lead exposure over the total lifetime of the bird, but the least useful indicator of recent lead exposure and absorption. The tissues usually chosen to evaluate recent exposure are blood, liver, and occasionally kidney.

16.4 FACTORS INFLUENCING CONCENTRATIONS OF LEAD IN TISSUES

It is often difficult to relate lead exposure directly to tissue lead concentrations in birds, as many factors influence lead retention and its absorption and deposition into body tissues. The influence of the magnitude and duration (chronic or acute) of lead exposure on tissue lead concentrations was discussed earlier. Both the absorption of lead and its deposition in tissues are affected by a variety of additional factors, including gender, breeding condition, age, stomach type, and diet, as discussed later.

Finley et al. (1976a) found that laying mallards dosed with one No. 4 lead shot (193 mg) accumulated significantly higher liver, kidney, and bone lead concentrations than did males. Although lead concentrations remained relatively low in soft tissues of both genders (maximum means of 1.2 mg/kg wet weight in liver and 3.5 mg/kg wet weight in kidney), mean bone lead concentrations were 112 mg/kg dry weight in females compared with 10 mg/kg dry weight in males. Subsequent studies revealed a similar result and additionally found lead concentrations in femurs to be 4 times higher in laying than nonlaying females (Finley and Dieter 1978). As females mobilize calcium from medullary bone for eggshell formation, the intestinal absorption of calcium (and concurrently lead) increases, resulting in higher bone lead concentrations in females (Krementz and Ankney 1995, Scheuhammer 1996). Lead also is deposited to a greater extent in bones of high medullary content (e.g., femur, sternum, and tibia) than in those with low medullary content (e.g., humerus, ulna, and radius) (Finley and Dieter 1978, Pattee 1984, Rocke and Samuel 1991). Medullary bone is of particular significance in females because of its accumulation, before egg laying, as a source of calcium storage for eggshells, followed by its destruction during eggshell formation (Simkiss 1961). Although lead concentrations of wing bones (low medullary bone content) of waterfowl collected in the autumn did not differ by gender (White and Stendell 1977), a study of American woodcock (*Scolopax minor*) collected in the autumn showed that females had higher lead concentrations in wing bones than males (Scheuhammer et al. 1999).

Although, as mentioned previously, lead concentrations in bones generally increase with age, evidence also exists that large amounts of lead are deposited in bones of immature birds during the time when calcium absorption and deposition are rapid. For example, 70% of American woodcock chicks collected from nests or broods had bone lead concentrations of >20 mg/kg dry weight, compared with 43% of hatch year woodcock with >20 mg/kg collected in the autumn (Strom et al. 2005) and Anderson (1975) reported mean bone lead concentrations of 56 mg/kg dry weight in immature lesser scaup versus 26 mg/kg dry weight in adults during a lead poisoning mortality event. Mateo et al. (2001) also reported higher bone lead concentrations in duckling (<9-day old) than fully grown marbled teal (*Marmaronetta angustirostris*).

The anatomical characteristics of the avian ventriculus (stomach) vary by species and can influence the retention, and thus to some extent the absorption, of metallic lead objects. Stomachs of carnivores, scavengers, and fish-eating birds are adapted for a relatively soft diet, whereas birds that consume grain, vegetation, and insects have a more muscular stomach (or gizzard) adapted for hard diets (Denbow 2000). The acidic conditions in both types of stomachs facilitate the dissolution of lead, but ingested lead may be retained with grit and mechanically ground down in the muscular gizzard of some birds, particularly waterfowl. Although passerines and columbiforms also have muscular gizzards, lead shot dosing experiments suggest that these groups of birds may void shot relatively quickly (Vyas et al. 2001, Schulz et al. 2007). On the other hand, raptors and many other carnivorous birds have a thin-walled stomach and share the particular characteristic of forming

and regurgitating pellets, or casts, consisting of undigested bones, hair, and feathers of prey (Duke 1986). Field studies confirm that particulate lead may sometimes be egested with the casts (Platt 1976, Nelson et al. 1989, Mateo et al. 1999, 2007). However, the significance of such regurgitation on total lead absorption is variable, as one experimental study found that bald eagles regurgitated lead from 12 h to 48 days after dosage (Pattee et al. 1981).

Diet, one of the most important factors influencing lead absorption and deposition in tissues, has been extensively studied in waterfowl and to a lesser extent in other species (Jordan and Bellrose 1951, Longcore et al. 1974a, Sanderson and Irwin 1976, Koranda et al. 1979, Sanderson and Bellrose 1986, Marn et al. 1988, Scheuhammer 1996, Vyas et al. 2001). Findings indicate that nutritional, chemical, and physical characteristics of diet are important. In general, nutritionally balanced species-appropriate diets high in protein and calcium tend to mitigate the effects of lead exposure. Much of the mitigating effect of nutrient-rich diets appears to occur in the digestive system, with high calcium and protein levels reducing the gastrointestinal absorption of lead and lowering the total body burden (Koranda et al. 1979, Sanderson 1992, Scheuhammer 1996). Furthermore, when lead is ingested along with food, certain chemical groups in food components have a ligand effect, binding lead in an insoluble form in the intestine (Morton et al. 1985). In waterfowl, there is evidence that the selection of larger grit size is positively related to the prevalence of lead shot ingestion (Pain 1990, Mateo et al. 2000, Figuerola et al. 2005).

Finally, when evaluating reports of lead concentrations in tissues, one should consider the quality assurance/quality control data for the analytical technique used, such as lead concentrations in procedural blanks, spiked samples, duplicates, and standard reference materials. Although standard practice today, these parameters were not reported in many of the older studies, when inadequate sensitivity and contamination issues may have resulted in less precise analyses.

16.5 TISSUE LEAD CONCENTRATIONS AND SUBLETHAL EFFECTS ON ENZYME SYSTEMS

Tissue lead "threshold" concentrations for lead poisoning can be defined according to the tissue lead concentrations at which measurable effects occur. Because of the dynamics of lead uptake and retention, lead concentrations in blood and soft tissues are more easily related to effects than are bone lead concentrations. The effects of blood lead upon hematological parameters and enzyme systems have been extensively studied in birds. Blood lead studies provide useful information, as blood lead concentrations can be related to effect over time, with the extent and duration of lead exposure experimentally controlled.

Lead inhibits the activities of several enzymes necessary for the synthesis of heme, for example, delta-aminolevulinic acid dehydratase (ALAD) and heme synthetase. Heme is incorporated into hemoglobin and mitochondrial cytochromes and is part of cytochrome P-450, which is required in the liver for certain detoxification processes (Sassa et al. 1975, Dieter and Finley 1979). Heme synthetase is responsible for the incorporation of ferrous iron into protoporphyrin IX (PPIX) at the last stage of heme formation. Inhibition of heme synthetase activity results in an accumulation of PPIX in the blood. PPIX fluoresces when exposed to specific wavelengths, and this fluorescence has been used for the quantitative estimation of the PPIX concentration in birds and, consequently, as an indicator of lead exposure (Roscoe et al. 1979, Beyer et al. 1988, O'Halloran et al. 1988a, 1988b, Franson et al. 1996a, Vyas et al. 2000, Carpenter et al. 2003, Pattee et al. 2006). However, the first measurable biochemical change resulting from lead absorption appears to be the inhibition of erythrocyte ALAD activity (Hernberg et al. 1970, Tola et al. 1973). The inhibition of erythrocyte ALAD activity by blood lead is described in some detail later and illustrates the difficulty of interpreting the biological significance of sublethal effects, even when they can be directly related to tissue lead concentrations.

Inhibition of erythrocyte ALAD activity persists for several weeks to several months following lead absorption, in relation to elevated blood lead concentrations (Finley et al. 1976b, Dieter and Finley 1978, Pain 1987, Redig et al. 1991, Carpenter et al. 2003, Pattee et al. 2006). Inhibition of

ALAD activity in avian blood has been reported at blood lead concentrations of <5 μg/dL (c. 0.05 mg/kg) (Pain 1989, Martínez-López et al. 2004). Blood lead concentrations of 15–20 μg/dL (c. 0.15–0.2 mg/kg), 30–80 μg/dL (c. 0.3–0.8 mg/kg), and ≥100 μg/dL (c. 1 mg/kg) have been reported to result in inhibition in ALAD of 50%, 60–80%, and 75% to nearly 100%, respectively (Finley et al. 1976b, 1976c, Dieter and Finley 1978, Hoffman et al. 1981, Pain 1989, Redig et al. 1991, Work and Smith 1996, Franson et al. 2002, Beyer et al. 2004, Pattee et al. 2006).

Birds appear to be able to tolerate some reduction of erythrocyte ALAD activity without showing signs of reduced hematocrit or hemoglobin concentration, although anemia may occur following sustained low level ALAD inhibition. Rapid decreases in hematocrit after exposure to a large amount of lead may be associated with hemolytic anemia, as well as severe (e.g., >75%) ALAD inhibition (Pain and Rattner 1988, Mateo et al. 2003). However, although the significance of reduced erythrocyte ALAD activity is not always easily determined, ALAD activity is also inhibited in other body tissues. One month after administering doses of one lead shot to mallards, Dieter and Finley (1979) recorded a 75% reduction in erythrocyte ALAD activity (blood lead, 98 μg/dL, c. 0.98 mg/kg), a 42% inhibition in liver ALAD activity (liver lead, 2.24 mg/kg of wet weight), a 50% reduction in ALAD activity in the cerebellum, and a 35% reduction in the cerebral hemisphere (brain lead, 0.43 mg/kg of wet weight). ALAD activity was correlated with the lead concentration in all tissues but was more sensitive to lead in the brain than in the liver, where some lead may possibly be bound in a biologically inactive form. The authors also recorded a significant increase in butylcholinesterase (a marker enzyme for glial or supportive cells) activity in the brain, suggesting possible brain damage. The results of this study suggest that blood lead concentrations of 100 μg/dL (c. 1 mg/kg) may be associated with pathological changes in waterfowl brains, but the histologic lesions found in the central nervous system of lead-exposed birds have been minor (Wobeser 1997).

16.6 LEAD AND IMMUNOSUPPRESSION

Several studies have addressed the immunosuppressive effects of lead exposure in birds. Mallards dosed with one No. 4 lead shot exhibited depressed hemagglutination titers to sheep red blood cells, indicating an effect on antibody-mediated immunity by day 7 after treatment, and titers remained low until the end of the 3-week experiment (Trust et al. 1990). Three ducks died during the experiment, exhibiting clinical signs and lesions consistent with lead poisoning. In Japanese quail (*Coturnix coturnix*), lead acetate in drinking water suppressed antibody-mediated response, but only at dosages that also caused clinical lead poisoning (Grasman and Scanlon 1995). In two other studies with Japanese quail, lead exposure did not affect any of the immune system parameters measured (Morgan et al. 1975, Fair and Ricklefs 2002). Western bluebird (*Sialia mexicana*) nestlings dosed with three No. 9 lead shot showed a reduction in cell-mediated immune response, although those receiving one or two lead shots were not affected (Fair and Myers 2002). Redig et al. (1991) reported depressed cell-mediated immunity in red-tailed hawks (*Buteo jamaicensis*) that received increasing amounts of lead acetate over a 10-week period. As discussed previously, the distribution of lead among tissues may be different between females and males during the prebreeding and breeding seasons. The storage of higher proportions of lead in bone prior to and during breeding by females could result in gender differences in the immune response of birds exposed to a similar amount of lead during this period. For example, Rocke and Samuel (1991) noted an increased immunosuppressive effect of lead in male compared with female mallards during the prebreeding season. In nonbreeding zebra finches, however, antibody-mediated immunity was suppressed by lead in females compared with males, but only in birds on a low calcium diet (Snoeijs et al. 2005).

16.7 BACKGROUND LEAD CONCENTRATIONS IN AVIAN TISSUES

As discussed earlier, there are difficulties associated with relating exposure to tissue lead concentrations and with relating tissue lead concentrations to effect. However, for managing wildlife on

contaminated areas, it is important to provide recommendations for interpreting tissue concentrations. These recommendations are based on the tissue lead concentrations in unexposed wild birds and the concentrations at which clinical effects and mortality may occur.

Background blood lead concentrations in birds are generally low, usually <20 μg/dL (c. 0.2 mg/kg) and frequently well below 10 μg/dL (c. 0.1 mg/kg) (Dieter et al. 1976, Szymczak and Adrian 1978, Birkhead 1983, Franson et al. 1986, 2004, Pain 1989, Trust et al. 1990, Redig et al. 1991, Carpenter et al. 2003, Pattee et al. 2006). Birds with no history of lead poisoning usually have liver and kidney lead concentrations of <2 mg/kg wet weight and frequently of <1 mg/kg wet weight (Bagley and Locke 1967, Irwin 1975, Clausen and Wolstrup 1979, Custer et al. 1984, Spray and Milne 1988, Kingsford et al. 1989, Franson et al. 1995a, Beyer et al. 1998, Carpenter et al. 2003, Kenntner et al. 2003, Martin et al. 2008). Bone lead concentrations are more difficult to interpret, and concentrations tend to be higher because of accumulation. Different values for background concentrations have been proposed, but concentrations of <10–20 mg/kg dry weight are generally considered as background or are reported in unexposed birds (Moore 1978, Szymczak and Adrian 1978, Pain et al. 1992, Martin et al. 2008, Franson et al. 2009).

16.8 RECOMMENDATIONS FOR THE INTERPRETATION OF TISSUE LEAD CONCENTRATIONS IN BIRDS

The interpretation of tissue lead concentrations is facilitated by the availability of information on exposure and clinical signs of poisoning. In live birds, useful information may include the presence of lead in the casts regurgitated by raptors, the presence of lead particles in the gastrointestinal tracts of birds identified by x-ray, and clinical signs of lead poisoning. At necropsy, pathological observations and the presence of ingested lead particles are useful indicators, along with history about the bird before death. However, detailed history information is not always available. In these situations, a prediction can be made regarding the degree to which both live and dead birds were affected by lead poisoning by evaluating tissue residues. We categorize lead residues in the blood, liver, and kidney of Anseriformes, Falconiformes, and Columbiformes according to increasing severity of effects (Table 16.1): (1) subclinical poisoning, a range of residues reported to cause physiological effects that are insufficient to severely impair normal biological functioning, resulting in no external signs of poisoning, and from which the bird would probably recover if lead exposure were terminated; (2) clinical poisoning, an approximate threshold level marking the initiation of clinical signs (pathological manifestations of physiological effects) such as anemia, microscopic lesions in tissues, weight loss, muscular incoordination, green diarrhea, and anorexia leading to probable death if lead exposure were to continue; and (3) severe clinical poisoning, an approximate threshold value at which the effects may be directly life threatening in field, captive, and/or experimental cases of lead poisoning. We consider residues below the subclinical poisoning range as "background," that is, evidence of environmental exposure distant from any specific source of lead contamination. Table 16.1 is meant to provide general guidance in the assessment of residues, with an awareness that numerous factors, some of which are discussed later, may contribute to an overlap of residue values in the three categories. It is also important to note that birds in experimental studies may accumulate higher levels of lead in tissues than wild birds subject to stressors in the environment.

16.8.1 Individual Birds

It is important to note that for lead, as for many other contaminants, toxic effects may depend upon factors other than simply the concentrations in tissues. These factors include the level and duration of lead exposure, previous history of exposure, species variability in response to exposure, the overall health of the bird, the extent of damage already done, and the potential interactions between lead and other disease agents. The biological significance of a tissue lead concentration may, therefore, be difficult to determine if the history of the bird is unknown. The chronicity of exposure is

TABLE 16.1
Suggested Interpretations of Tissue[a] Lead Concentrations in Three Orders of Birds

Order	Blood (μg/dL)[b]	Liver[c] (mg/kg ww)	Kidney (mg/kg ww)	Reference
Anseriformes				
Subclinical poisoning	20 < 50	2 < 6	2 < 6	Dieter and Finley (1979), Degernes (1991)
Clinical poisoning	50–100	6–10	6–15	Longcore et al. (1974b), Degernes (1991), Beyer et al. (2000)
Severe clinical poisoning	>100	>10	>15	Cook and Trainer (1966), Longcore et al. (1974b), Mautino and Bell (1986), Beyer et al. (1988, 2000), Pain and Rattner (1988), Pain (1989), Blus et al. (1991, 1999), Degernes (1991), Kelly et al. (1998), Nakade et al. (2005), Degernes et al. (2006)
Falconiformes				
Subclinical poisoning	20 < 50	2 < 6	2 < 4[d]	Custer et al. (1984), Henny et al. (1991), Kramer and Redig (1997)
Clinical poisoning	50–100	6–10	4–6[d]	Lumeij et al. (1985), Kramer and Redig (1997)
Severe clinical poisoning	>100	>10	>6[d]	Redig et al. (1980), Hoffman et al. (1981), Pattee et al. (1981, 2006), Langelier et al. (1991), Kramer and Redig (1997)
Columbiformes				
Subclinical poisoning	20 < 200	2 < 6	2 < 15	Ohi et al. (1974), Cory-Slechta et al. (1980), Kendall et al. (1982), Kendall and Scanlon (1982), DeMent et al. (1987), Scheuhammer and Wilson (1990)
Clinical poisoning	200–300	6–15	15–30	Cory-Slechta et al. (1980), Anders et al. (1982), Boyer et al. (1985)
Severe clinical poisoning	>300	>15	>30	Locke and Bagley (1967), Barthalmus et al. (1977), Cory-Slechta et al. (1980), Anders et al. (1982), Boyer et al. (1985), Schulz et al. (2006)

[a] Lead concentrations in bone reflect lifetime accumulation and chronic low exposure to lead may result in similar concentrations in bone as acute exposure to higher levels. If evidence of acute exposure exists, we recommend that bone lead concentrations (dry weight basis) of <10 μg/g be considered background, 10–20 μg/g be considered evidence of subclinical to clinical poisoning, and >20 μg/g be considered evidence of severe clinical poisoning.

[b] Divide μg/dL by 100 for an approximate conversion to mg/kg.

[c] In general, a diagnosis of lead poisoning in an individual bird can be reached if necropsy observations are consistent with lead poisoning and the lead concentration in the liver is ≥6 mg/kg wet weight.

[d] Although in many species lead concentrations are often higher in kidney than in liver, the reverse has been found in lead-exposed and poisoned eagles and some species of hawks; thus, for Falconiformes, we have suggested a conservative lead threshold concentration in kidney.

particularly important. Birds exposed to relatively low lead levels on a sustained basis may suffer similar effects, but with lower soft tissue lead concentrations, than birds acutely exposed to higher levels of lead for a short period of time. Also, exposure and tissue lead concentrations are not always associated in individual birds because of the varying retention time of shot in the gizzard and the uptake/retention dynamics of lead in tissues. However, in a live bird, sequential blood lead analyses from an individual give a much clearer picture of the significance of contamination as chronicity can be established. In addition, hematological measurements such as ALAD activity, PPIX concentration, hematocrit, and hemoglobin concentrations will indicate biochemical damage.

A wide range of clinical signs may be observed in lead-poisoned birds following chronic exposure, including anorexia, emaciation, anemia, lethargy, wing droop, ataxia, green diarrhea staining the vent, and neurological signs such as leg paralysis or convulsions (Locke and Thomas 1996, Wobeser 1997, Friend 1999, Eisler 2000, Pattee and Pain 2003). Some of the gross and microscopic lesions in waterfowl with lead poisoning (Coburn et al. 1951, Cook and Trainer 1966, Karstad 1971, Clemens et al. 1975, Forbes and Sanderson 1978, Hunter and Wobeser 1980, Wobeser 1997) have been reported for other avian species (Locke and Bagley 1967, Hunter and Haigh 1978, Pattee et al. 1981, 2006, Beyer et al. 1988, Langelier et al. 1991, Vyas et al. 2001, Carpenter et al. 2003). Muscle wasting and loss of fat reserves are some of the most consistent lesions associated with lead poisoning across avian taxa. Other gross lesions include impactions of the esophagus or proventriculus, distended gallbladder, dark discolored gizzard lining, light areas (gross evidence of necrosis) in heart or gizzard muscle, wasting of internal organs, pale flabby heart, pale internal organs and muscle tissue, and atrophied internal organs. Particulate lead may or may not be present in the stomach. The presence of acid-fast intranuclear inclusion bodies in kidney tubular cells are indicative of lead poisoning, but are not present in all cases. Additional microscopic lesions include hemosiderosis in the liver, necrosis of myocardial and gizzard muscle, and degenerative changes in the brain and peripheral nerves. In cases where birds die rapidly following acute exposure to high levels of lead, many of these lesions may be absent. It should be noted that while acid-fast intranuclear inclusion bodies are a relatively specific indicator of lead poisoning, other signs are nonspecific and may be observed in association with a variety of other conditions. In waterfowl, Beyer et al. (1998) found impactions of the upper alimentary tract, submandibular edema, myocardial necrosis, and biliary discoloration of the liver to be the gross lesions most reliably associated with lead poisoning.

In individual birds, lead poisoning as a cause of death should be distinguished from the observation that the bird simply has been exposed to lead, based solely on tissue residues. The confidence with which a diagnosis of lead poisoning as the cause of death can be made will increase with the amount of information available. Ideally, a diagnostic evaluation will include exposure history data, necropsy observations, and pathological findings. Necropsy and pathological findings consistent with lead poisoning, in conjunction with lead residues of ≥6 mg/kg wet weight in liver, for example, support a diagnosis of lead poisoning as the cause of death. On the other hand, if necropsy observations and pathological findings are not available, a more conservative approach is recommended. Beyer et al. (1998) studied liver lead concentrations in waterfowl diagnosed as lead-poisoned, finding that 95% of 421 ducks and geese diagnosed with lead poisoning had lead concentrations of at least 38 mg/kg dry weight (10 mg/kg wet weight), but less than 1% of birds that died of other causes had a level that high. The authors concluded that 38 mg/kg dry weight in liver is a defensible criterion for identifying waterfowl suffering from lead poisoning, although a definitive diagnosis requires the finding of concurrent lesions compatible with lead toxicosis. Single bone lead concentrations are the least useful indices of poisoning due to accumulation over time. However, high liver and low bone lead concentrations may suggest severe clinical poisoning following recent exposure to a large amount of lead.

16.8.2 Populations

The determination of lead concentrations in blood is a useful, and nonlethal, way to study lead exposure in populations of birds. Flint et al. (1997) collected blood samples from a population of

threatened spectacled eiders (*Somateria fischeri*) in Alaska, finding that the probability of lead exposure (≥20 µg/dL blood lead, or c. 0.2 mg/kg) increased through the breeding season. In a follow-up study with spectacled eiders, adult females with ≥20 µg/dL blood lead before hatching their eggs were found to survive at a much lower rate than females with blood lead concentrations of <20 µg/dL before hatch (Grand et al. 1998). Sympatrically nesting common eiders (*Somateria mollissima*) were found to have much lower frequencies of elevated lead than spectacled eiders, probably as a result of differing foraging behavior and brood rearing strategies (Flint et al. 1997, Wilson et al. 2007). In canvasbacks (*Aythya valisineria*), Hohman et al. (1995) found that winter survival rates for immatures with ≥0.2 mg/kg blood lead were lower than in those with <0.2 mg/kg during two of three winters. DeStefano et al. (1991) measured blood lead concentrations and studied lead exposure in the Eastern Prairie Population of Canada geese, reporting increasing levels of exposure as geese moved from breeding to wintering grounds. Pain et al. (1997) found a temporal variation in the proportion of marsh harriers (*Circus aeruginosus*) with elevated blood lead concentrations, with a far higher incidence during than outside the hunting season. Blood lead concentrations have been used in recent years to monitor California condor populations for lead exposure, and to form the basis for decisions regarding chelation therapy in individuals of this highly endangered species (Hall et al. 2007, Parish et al. 2007).

The activity of ALAD is well correlated with blood lead concentrations, particularly up to about 100–150 µg/dL (c. 1–1.5 mg/kg). As absolute levels of ALAD vary among individuals, the ratio of lead inhibited ALAD activity to reactivated ALAD activity, with the lead displaced, has been recommended for monitoring lead exposure in populations (Dieter 1979, Pain 1989, Scheuhammer 1989). Although less useful than other tissues in individual cases, bone lead concentrations have been used to determine geographical patterns of lead poisoning in populations (Stendell et al. 1979, Scheuhammer and Dickson 1996). In addition, at a population level, bone and liver lead concentrations may be correlated (Anderson 1975), and tissue lead concentrations may sometimes be correlated with exposure, as measured by the presence of shot in gizzards (White and Stendell 1977). Pain et al. (1992) found a positive correlation between exposure to shot (measured as the percentage of shot ingestion in eight species of waterfowl and shore birds) and liver and bone lead concentrations.

Concentrations of lead in tissues will sometimes be elevated via pathways other than metallic lead from ammunition or angler's weights, such as exposure to mining or smelting wastes, lead-based paint, or industrial effluents. An evaluation of the distribution of blood or liver lead concentrations may help identify possible sources of exposure. Distributions of blood and soft tissue lead concentrations from populations in which some birds have ingested metallic lead tend to be very skewed or have distinct outliers (Dieter 1979, Beyer et al. 1998), whereas concentrations resulting from a more general source of lead, for example, in water or the atmosphere, may be more normally distributed. The determination of stable lead isotope ratios, $Pb^{204,206,207,208}$, in bird tissues may also assist in determining potential sources of lead exposure (Scheuhammer and Templeton 1998, Meharg et al. 2002, Scheuhammer et al. 2003a, Pain et al. 2007). For example, Church et al. (2006) found lead isotope values in free-flying California condors with relatively high blood lead concentrations to be consistent with lead from ammunition and distinct from those of pre-release condors with low blood lead concentrations.

16.8.3 Species

Lead concentrations in tissues associated with poisoning in birds vary among orders, with Galliformes and Columbiformes often having higher residues than others (Pattee et al. 1981, Kendall et al. 1983, Gjerstad and Hanssen 1984, Reichel et al. 1984, Beyer et al. 1988, 1998, Vyas et al. 2001, Lewis et al. 2001, Schulz et al. 2006). Within-order differences among species also have been noted, as lead-poisoned Anseriformes of lighter weight tended to have higher lead concentrations in the liver than species of greater weight (Beyer et al. 1998). Within Falconiformes, one study of birds

similarly dosed with lead acetate found that the blood lead concentration of a turkey vulture was nearly 8 times greater than in two red-tailed hawks after 6 weeks of dosage and that the vulture developed clinical signs of lead poisoning, but the hawks did not (Reiser and Temple 1981). In a more recent study with turkey vultures, Carpenter et al. (2003) reported that, although four of six birds died or were euthanized because of severe clinical signs, survival time, even with frequent redosing because of regurgitated or defecated lead shot, was quite long and lead pellet dosage was high compared with other avian studies. The authors concluded that turkey vultures are relatively tolerant to lead exposure and would not be a good model for evaluating the risk of lead exposure in California condors (Carpenter et al. 2003).

Although nontoxic shot regulations have been introduced in many countries, with a few exceptions these primarily restrict the use of lead gunshot over wetlands or for waterfowl hunting. While, where compliance is good, this should reduce lead ingestion in waterfowl and their main predators, other avian predators and scavengers, a range of other terrestrial birds continue to be subject to lead exposure and poisoning because of their feeding habits. Lead poisoning in the California condor, which feeds almost exclusively on carrion, is now considered a major obstacle to the recovery of the species (Meretsky et al. 2000, Cade 2007, Snyder 2007). California condors ingest lead fragments from ammunition in carcasses of animals or offal left in the field (Church et al. 2006, Hunt et al. 2006, Cade 2007), and considerable effort is devoted to monitoring and attempts to reduce lead exposure (Hall et al. 2007, Parish et al. 2007, Sullivan et al. 2007). Lead poisoning of wintering bald eagles and golden eagles (*Aquila chrysaetos*) was long thought to be primarily due to the ingestion of shotgun pellets in dead or crippled waterfowl, but Kramer and Redig (1997) found that the prevalence of lead poisoning in eagles did not decrease after the implementation of the 1991 nontoxic shot regulations for hunting waterfowl and American coots (*Fulica americana*) in the United States. The authors suggested that carcasses of animals still hunted with lead ammunition, such as small mammals and birds, and deer, could be additional sources of exposure (Kramer and Redig 1997). Lead exposure and poisoning has been reported in a variety of eagle species and other raptors throughout the world (Pain et al. 2009).

16.9 LEAD RESIDUES REPORTED IN SELECTED FIELD AND LABORATORY STUDIES IN BIRDS

16.9.1 Anseriformes (Ducks, Geese, and Swans)

Lead concentrations in the liver and kidney of sick and dead Canada geese in an early field report ranged from 9 to 27 mg/kg and 12 to 57 mg/kg (wet weight presumed but not stated) (Adler 1944). One of the early experimental studies of lead exposure in waterfowl that reported concentrations in tissues was that of Coburn et al. (1951) who dosed mallards with lead nitrate. Birds that died in that study had average bone and liver lead concentrations of 469 and 208 mg/kg dry weight, respectively (Coburn et al. 1951). Canada geese that died of experimental lead shot exposure had lead concentrations in the liver of 5–32 mg/kg (wet weight presumed but not stated) (Cook and Trainer 1966). Longcore et al. (1974b) summarized results of seven experimental lead exposure studies in Canada geese and mallards, in which birds both died and were euthanized, finding that mean liver lead concentrations ranged from 12 to 51 mg/kg wet weight. Mallards that died after being dosed with one No. 4 lead buckshot had mean liver and kidney lead concentrations (mg/kg wet weight) of 51–64 and 158–259, respectively (Longcore et al. 1974b). In wild Canada geese, tundra swans (*Cygnus columbianus*), and mallards picked up during lead poisoning die-offs, mean liver lead concentrations were 12–28 mg/kg wet weight (Longcore et al. 1974b). In another lead poisoning event, the mean lead concentration in livers of Canada geese was 16 mg/kg wet weight (Bagley et al. 1967). Lesser scaup found sick or dead during a lead poisoning mortality event had average lead concentrations in tissues of 46 mg/kg wet weight in liver, 66 mg/kg wet weight in kidney, and 40 mg/kg dry weight in wing bones (Anderson 1975). Average lead concentrations in liver and kidney (mg/kg wet

weight assumed but not stated) of lead poisoned mallards and mute swans were 40 and 58, and 33 and 105, respectively (Clausen and Wolstrup 1979). Mean liver lead concentrations in four species of waterfowl picked up during a lead poisoning die-off ranged from 15 to 31 mg/kg wet weight, or 86–131 mg/kg dry weight (Zwank et al. 1985). Liver lead concentrations in four spectacled eiders and a common eider found dead or moribund and diagnosed with lead poisoning ranged from 26 to 52 mg/kg wet weight (Franson et al. 1995b). The lead concentration in a blood sample from one of the spectacled eiders was 8.5 mg/kg (Franson et al. 1995b). In a field study of lead poisoning in tundra swans and trumpeter swans (*Cygnus buccinator*), Degernes et al. (2006) reported mean liver lead concentrations of 42–203 mg/kg dry weight in lead-poisoned swans, and 0.5–2.1 mg/kg dry weight in swans not poisoned by lead. Whooper swans (*Cygnus cygnus*) that were diagnosed as lead poisoned had liver lead concentrations of 5.5–44.3 mg/kg wet weight (Ochiai et al. 1992). The mean and fifth percentiles of liver lead concentrations in waterfowl diagnosed with lead poisoning were 115 and 38 mg/kg dry weight, respectively (Beyer et al. 1998). Mallards dosed with lead shot, half of which died, had liver and kidney lead concentrations of 78 and 256 mg/kg dry weight (Kelly et al. 1998). When given a chronic dose of lead acetate, mallards died with lower tissue lead concentrations than when given a large dose of lead from lead pellets (Beyer et al. 1988).

In experimental studies of lead exposure in waterfowl, blood lead concentrations can reach 7 mg/kg or greater. After one week, concentrations of lead in blood of ring-necked ducks (*Aythya collaris*) dosed with one No. 4 lead shot and in mallards dosed with two No. 4 lead shot were 7.6 and 7.8 mg/kg (Mautino and Bell 1986, 1987). Three and six days after canvasbacks were dosed with one No. 4 lead shot, blood lead concentrations were 7.4 mg/kg and about 4.5 mg/kg, respectively (Franson et al. 1986). In Canada geese dosed with multiple No. 4 lead shot, peak blood lead concentrations at days 3 and 10 were 16.8 mg/kg and 6.7 mg/kg, respectively (Cook and Trainer 1966). Concentrations of lead in blood of ducks that survive experimental lead shot exposure generally decline to about 1 mg/kg or less after 1 month (Dieter and Finley 1978, 1979, Franson et al. 1986, Mautino and Bell 1986, 1987). Concentrations of lead in blood associated with clinical signs in field cases of lead-poisoned waterfowl tend to be lower than in experimental studies. Trumpeter swans with 1.0–1.99 mg/kg lead in their blood had pronounced clinical signs, including weakness and neurological abnormalities (Degernes 1991). With treatment, 6 of 10 birds in this group survived (Degernes 1991). Concentrations of lead in blood of six whooper swans that were diagnosed with lead poisoning were 300–630 μg/dL (c. 3–6.3 mg/kg) (Ochiai et al. 1992). Six whooper swans and two tundra swans that were captured exhibiting signs of weakness, green feces, and pale conjunctiva had blood lead concentrations ranging from 2.5 to 6.7 mg/kg (Nakade et al. 2005). Seven of the eight swans died on the second day after capture, lead shot were recovered from gizzards, and liver and kidney lead concentrations (wet weight presumed but not stated) ranged from 14.0 to 30.4 mg/kg and 30.2 to 122 mg/kg, respectively. The single whooper swan that survived had an original blood lead concentration of 2.9 mg/kg, which declined to about 0.6 mg/kg at 30 days (Nakade et al. 2005). Sick Canada geese captured during a lead poisoning die-off had a mean blood lead concentration of 5.2 mg/kg (Szymczak and Adrian 1978).

Tissue residues described above are primarily the result of lead shot ingestion. Poisoning of waterfowl from the ingestion of lead in sediments can result in similar tissue concentrations. Mortality of tundra swans near an area of lead mining and smelting was reported as early as 1924 and early analysis of liver samples revealed lead concentrations of 18–37 mg/kg wet weight (Chupp and Dalke 1964). Later studies in the same area reported mean liver lead concentrations of 10–24 mg/kg wet weight and mean blood lead concentration of 3.3 mg/kg in swans and up to 8 mg/kg in blood and 14 mg/kg wet weight in liver of wood ducks (*Aix sponsa*) (Blus et al. 1991, 1993, 1999). The mean and fifth percentile blood lead concentrations in tundra swans moribund from lead poisoning were 3.6 and 1.9 mg/kg, respectively (Beyer et al. 2000). Sileo et al. (2001) diagnosed lead poisoning in 219 waterfowl found sick or dead, none of which had metallic lead in their gizzards. Liver lead concentrations in 216 birds in that study ranged from 6.3 to 90 mg/kg wet weight. The three others were diagnosed with lead poisoning as follows: a tundra swan and a Canada goose had <6 mg/kg

wet weight lead in liver, but had inclusion bodies in the kidney, and a mallard had 38.7 mg/kg wet weight lead in the kidney (Sileo et al. 2001). Heinz et al. (1999) conducted a series of experiments feeding mallards 3–48% sediment (containing lead concentrations of 3400 and 4000 mg/kg dry weight) from the mining and smelting area. Lead concentrations in blood, liver, and kidney were as high as 6.9, 38, and 31 mg/kg wet weight, respectively (Heinz et al. 1999).

16.9.2 Falconiformes (Hawks, Falcons, and Vultures)

A wide range of liver and kidney residues have been reported from raptors dying of lead poisoning, but levels are often in the range of 5–40 mg/kg wet weight. In lead-exposed or poisoned eagles, lead concentrations are often higher in liver than in kidney. Wayland et al. (1999) studied relationships of lead concentrations among tissues of bald and golden eagles, finding that when the lead concentration in liver was 6 mg/kg dry weight, the predicted renal concentration was 4.6 mg/kg, but when the concentration in the liver was 30 mg/kg dry weight, the predicted renal concentration was 18 mg/kg. The published records of 37 wild bald eagles that died of lead poisoning report liver lead residues of 5–61 mg/kg wet weight and of 5 and 12 mg/kg wet weight in the two kidneys tested (Mulhern et al. 1970, Kaiser et al. 1980, Reichel et al. 1984, Frenzel and Anthony 1989, Craig et al. 1990, Langelier et al. 1991, Gill and Langelier 1994). The mean lead concentration in kidneys of five bald eagles that showed evidence of lead poisoning was 34 mg/kg dry weight (Elliott et al. 1992). Of three captive bald eagles that died of lead poisoning, two had liver lead residues of 23 and 15 mg/kg and kidney residues of 11 mg/kg each (wet weight presumed but not stated) (Jacobson et al. 1977, Redig et al. 1980). The third had 26 mg/kg wet weight of lead in the liver and 9 mg/kg wet weight in the kidney (Janssen et al. 1979). In an experimental study of lead shot poisoning, four eagles died with mean lead residues of 17 mg/kg wet weight in the liver, 6 mg/kg wet weight in the kidney, 1.4 mg/kg wet weight in the brain, and 10 mg/kg dry weight in the bone (Pattee et al. 1981). A fifth eagle in that study that became blind and was euthanized had lead concentrations of 3 mg/kg wet weight in the liver and kidney, 2 mg/kg wet weight in the brain, and 13 mg/kg dry weight in the bone.

A Steller's sea eagle (*Haliaeetus pelagicus*) and a white-tailed sea eagle (*Haliaeetus albicilla*) with lead fragments in the intestine and gizzard, respectively, had liver lead concentrations of 139 and 79 mg/kg dry weight and kidney lead concentrations of 67 and 58 mg/kg dry weight (Kim et al. 1999). Mean lead concentrations in liver and kidney of two Steller's sea eagles and three white-tailed sea eagles with lead bullet fragments in their stomachs were 92 and 156 mg/kg dry weight, respectively (Iwata et al. 2000). Five white-tailed sea eagles diagnosed with lead poisoning had mean liver and kidney lead concentrations of 22.4 and 9.6 mg/kg wet weight, respectively (Krone et al. 2004, 2006, 2009).

Liver lead concentrations in three California condors that died of lead poisoning were 6, 23, and 35 mg/kg wet weight, respectively (Wiemeyer et al. 1988). The relatively low liver lead concentration in the first bird was probably the result of chelation therapy administered before it died. The liver and kidney lead concentrations (mg/kg wet weight) in Andean condors dosed with lead shot, and that died or were euthanized, ranged from 45 to 110 and 124 to 179, respectively (Pattee et al. 2006). A wild griffon vulture (*Gyps fulvus*) that died after admittance to a rehabilitation center had a piece of lead in its gizzard and a liver lead concentration of 52 mg/kg dry weight (Mateo et al. 1997b). In a lead shot dosing study with turkey vultures, lead concentrations (mg/kg wet weight) in liver and kidney of birds that died or were euthanized with severe clinical signs ranged from 6.8 to 34 and 180 to 246, respectively (Carpenter et al. 2003). Reports of lead poisoning mortality in captive condors and vultures include an Andean condor with a liver lead level of 38 mg/kg wet weight (Locke et al. 1969) and two captive king vultures (*Sarcoramphus papa*) with liver lead concentrations of 63 and 7 mg/kg and kidney residues of 71 and 25 mg/kg wet weight (Decker et al. 1979).

A wild red-tailed hawk that died of lead poisoning had a liver lead concentration of 71 mg/kg (wet weight presumed but not stated) and eight ingested lead shot (Sikarskie 1977). Two red-tailed hawks found emaciated, but with no lead in their stomachs, had lead concentrations in liver of 4.3 and 10 mg/kg wet weight (Franson et al. 1996b) and a third emaciated red-tailed hawk with green staining around the vent had lead concentrations of 22.3 mg/kg dry weight in liver and 8.2 mg/kg dry weight in kidney (Martin et al. 2008). Two additional red-tailed hawks had necropsy lesions consistent with lead poisoning and lead concentrations in liver or kidney of >30 or >20 mg/kg dry weight, respectively (Clark and Scheuhammer 2003). A common buzzard (*Buteo buteo*) with paresis of the legs and contracted talons had lead concentrations (all on dry weight basis) of 47.7 mg/kg in liver, 6.6 mg/kg in kidney, and 42.0 mg/kg in bone (Battaglia et al. 2005). MacDonald et al. (1983) reported lead poisoning mortality, probably caused by the presence of metallic lead in food items, in captive Falconiformes including a common buzzard, Eurasian sparrowhawk (*Accipiter nisus*), two peregrine falcons (*Falco peregrinus*), and a lagger falcon (*Falco jugger*). Liver lead concentrations were 36–175 mg/kg dry weight, and kidney residues were 31–221 mg/kg dry weight. Two prairie falcons (*Falco mexicanus*) that died of lead poisoning after being fed parts of animals shot by hunters had lead residues of 17 and 57 mg/kg in the liver and 6 and 78 mg/kg (wet weight presumed but not stated) in the kidney (Benson et al. 1974, Redig et al. 1980). Three raptors that died after receiving daily doses of lead acetate for several weeks had lead residues (dry weight basis) of 2.8–19.7 mg/kg in the liver, 4.2–17.4 mg/kg in the kidney, and 33.4–41 mg/kg in bone (Reiser and Temple 1981). Nestling American kestrels (*Falco sparverius*) dosed with metallic lead powder exhibited reduced ALAD activity and anemia with liver and kidney lead concentrations of 4 and 7 mg/kg wet weight, while survivors of a dosage that killed 40% of the treatment group had lead residues of 6 and 16 mg/kg wet weight in the liver and kidney (Hoffman et al. 1985a, 1985b). American kestrels fed a diet containing about 450 mg/kg dry weight of biologically incorporated lead had mean tissue residues of 10 mg/kg dry weight in the liver, 15 in the kidney, 2 in the brain, and 18 in bone with no effects on body weight, hematocrit, hemoglobin concentration, or red blood cell count (Custer et al. 1984). The authors concluded that their study provided further evidence that lead poisoning of raptors is probably due to the ingestion of metallic lead, not biologically incorporated lead.

Raptors exhibited anemia, anorexia, and bile-stained feces when blood lead residues reached 5–8 mg/kg (Reiser and Temple 1981). After 14 days of exposure to lead shot, five bald eagles had a mean blood lead concentration of 5.4 mg/kg and exhibited 80% ALAD depression and a 20–25% reduction in hematocrit and hemoglobin concentration (Hoffman et al. 1981). A California condor that was captured in a weakened condition and later died had a blood lead concentration of 420 μg/dL (c. 4.2 mg/kg) (Janssen et al. 1986). Of 437 blood samples collected from wild California condors in Arizona, 137 had lead concentrations of 15–59 μg/dL (c. 0.15–0.59 mg/kg), 25 were >100 μg/dL (c. 1 mg/kg), 10 were >200 μg/dL (c. 2 mg/kg), and 5 were >400 μg/dL (c. 4 mg/kg) (Parish et al. 2007). Among 214 blood samples from 44 individual California condors in southern California, 95 had lead concentrations >20 μg/dL (c. 0.2 mg/kg), 18 had 60–99 μg/dL (c. 0.6–0.99 mg/kg), and 7 had >100 μg/dL (c. 1 mg/kg) (Hall et al. 2007). Lead concentrations in 126 blood samples from 33 wild Big Sur California condors were >20 μg/dL (c. 0.2 mg/kg) in 27, 60–99 μg/dL (c. 0.6–0.99 mg/kg) in 4, and >100 μg/dL (c. >1 mg/kg) in 2 (Sorenson and Burnett 2007). In captive Andean condors dosed with lead shot, blood lead concentrations reached 16 and 17 mg/kg wet weight (Pattee et al. 2006). Two turkey vultures with clinical signs consistent with lead toxicosis had blood lead residues of 320 μg/dL (c. 3.2 mg/kg) and 11 mg/kg, but both recovered (Janssen et al. 1979, Reiser and Temple 1981). Turkey vultures that died after exposure to lead shot had 6–30 mg/kg wet weight lead in the heart blood clot (Carpenter et al. 2003). A prairie falcon with weakness, weight loss, and anemia had 11 mg/kg of lead in its blood but recovered after chelation therapy (Redig et al. 1980). A honey buzzard (*Pernis apivorus*) with a blood lead concentration of 80 μg/dL (c. 0.8 mg/kg) exhibited clinical signs of green diarrhea, muscle wasting, and weakness and had one lead shot in its stomach (Lumeij et al. 1985). Two weeks after removal of the lead shot when the blood lead concentration was 16 μg/dL (c. 0.16 mg/kg), the bird was released to the wild.

American kestrels fed lead-contaminated diets had blood lead levels of 1.69 mg/kg with no resultant anemia (Custer et al. 1984). Redig et al. (1991) reported no mortality or clinical signs of toxicity, based on gross observations and body weight, in red-tailed hawks with blood lead concentrations of up to 1.58 μg/mL (c. 1.58 mg/kg). However, Kramer and Redig (1997) stated that in bald and golden eagles, blood lead concentrations >1.2 mg/kg were invariably associated with mortality. In a survey of blood lead concentrations in 162 wild golden eagles Pattee et al. (1990) considered 36% of the birds to have been exposed to lead, but the overall mean blood lead concentration was 0.25 mg/kg. Harmata and Restani (1995) found ≥1.0 mg/kg lead in blood of 5.4% of bald eagles and 2.3% of golden eagles. Ospreys (*Pandion haliaetus*) from a mining and smelting area had a mean blood lead concentration of 0.20 mg/kg and inhibited ALAD activity, but there was no effect on hemoglobin or hematocrit (Henny et al. 1991).

16.9.3 Columbiformes (Doves and Pigeons)

Soft tissue lead residues associated with mortality in doves and pigeons tend to be in the range of 20–60 mg/kg wet weight or even higher, as in the case of a wild mourning dove that died of lead poisoning with 72 mg/kg wet weight of lead in the liver (Locke and Bagley 1967). Clausen and Wolstrup (1979) reported liver and kidney lead residues of 48 and 200 mg/kg (wet weight presumed but not stated), respectively, in a wood pigeon (*Columba palumbus*) that died of lead poisoning. Mourning doves that died after dosage with lead shot had mean lead residues of 80–93 mg/kg dry weight in the liver, 230–300 mg/kg in the kidney, and 116–192 mg/kg in bone (Buerger et al. 1986). In another lead shot dosing study, a mourning dove that died had lead residues of 267 mg/kg dry weight in the liver, 1901 mg/kg in the kidney, 11 mg/kg in the brain, and 403 mg/kg in bone (Kendall et al. 1983). Mourning doves in that study that were euthanized after 9 days of lead shot exposure had microscopic lesions of lead poisoning and lead concentrations of 150–179 mg/kg dry weight in the liver, 1182–1298 mg/kg in the kidney, 11–12 mg/kg in the brain, and 473–528 mg/kg in bone. Castrale and Oster (1993) reported that blood lead concentrations in mourning doves dosed with 4 #8 lead shot reached 10 mg/kg (wet weight presumed but not stated) after about 5 days, and eight of ten birds receiving this dose survived to 28 days, when blood lead levels had approached those in control birds. Ringed turtle-doves (*Streptopelia risoria*) given doses of lead shot and euthanized 9 days later had liver lead residues of 24–128 mg/kg and kidney residues of 633–2384 mg/kg dry weight (Kendall et al. 1981). Several of these birds had seizures, and all had microscopic lesions in the kidney and liver, but none died during the course of the experiment. Ringed turtle-doves that received lead acetate in drinking water for 90 days had mean kidney and liver residues of 900 and 8 mg/kg dry weight, respectively (Kendall and Scanlon 1981). Although no birds died, cellular necrosis and lead inclusions were noted in the kidneys. Two clinically normal feral pigeons (*Columba livia*) with lead shot in their gizzards had liver lead concentrations of 5 and 13 mg/kg and kidney residues of 14 and 81 mg/kg wet weight (DeMent et al. 1987).

Reduced ALAD activity occurred in ringed turtle-doves that had liver and kidney lead residues of 1 and 6 mg/kg wet weight, respectively (Scheuhammer and Wilson 1990). In other studies with ringed turtle-doves, ALAD activity was inhibited with tissue lead levels of 4–9 mg/kg dry weight in the brain, 9–19 mg/kg in the liver, and 84–839 mg/kg in the kidney (Kendall and Scanlon 1982, Kendall et al. 1982). The hemoglobin concentration was reduced when tissue residues reached 28 mg/kg dry weight in the liver, 12 mg/kg in the brain, and 457 mg/kg in the kidney (Kendall and Scanlon 1982). ALAD activity was reduced in feral pigeons with lead residues as low as 1.7 mg/kg wet weight in kidney and 16.5 mg/kg wet weight in bone (Ohi et al. 1974). Domestic pigeons that became anemic after treatment with lead acetate had lead residues of 8–20 mg/kg wet weight in the liver, 33–603 mg/kg in the kidney, 0.9–2.3 mg/kg in the brain, and 57–501 mg/kg in bone (Anders et al. 1982).

Blood lead concentrations associated with death in Columbiformes can be extremely high. Two domestic pigeons treated with lead acetate had blood lead residues of 2320 and 4000 μg/dL

(c. 23.2 and 40 mg/kg) shortly before they died (Dietz et al. 1979, Anders et al. 1982). Other pigeons that were treated with lead acetate developed blood lead concentrations of 250–440 µg/dL (c. 2.5–4.4 mg/kg), resulting in anemia and lead inclusions in the kidneys, but survived the dosage regimen for up to 64 weeks (Anders et al. 1982). Domestic pigeons that died after experimental lead acetate exposure had blood lead levels of 569–1235 and 1245 µg/dL (c. 5.69–12.35, and 12.45 mg/kg), crop stasis occurred at 450–1100 µg/dL (c. 4.5 and 11 mg/kg), but there were no observable clinical signs when blood lead was less than 200 µg/dL (c. 2 mg/kg) (Barthalmus et al. 1977, Cory-Slechta et al. 1980, Boyer et al. 1985). ALAD activity was inhibited in ringed turtle-doves with blood lead concentrations of 21, 81–122, and 142–245 µg/dL (c. 0.21, 0.81–1.22, and 1.42–2.45 mg/kg), and hemoglobin concentration was reduced with mean blood lead concentrations of 395 µg/dL (c. 3.95 mg/kg) (Kendall and Scanlon 1982, Kendall et al. 1982, Scheuhammer and Wilson 1990). Depression of ALAD in feral pigeons has been associated with lead residues as low as 15 µg/dL (c. 0.15 mg/kg) in the blood (Ohi et al. 1974). Two urban pigeons with lead shot in their gizzards had blood lead concentrations of 95 and 1870 µg/dL (c. 0.95 and 18.7 mg/kg), but they were not anemic or emaciated (DeMent et al. 1987).

16.9.4 Galliformes (Pheasants, Grouse, Quail, and Partridge)

Data suggest that Galliformes are relatively less sensitive to lead than some other groups as evidenced by high tissue lead residues in individuals poisoned by lead. A wild turkey (*Meleagris gallopavo*) that died of lead poisoning had a liver lead concentration of 17 mg/kg wet weight (Stone and Butkas 1978). A wild ring-necked pheasant (*Phasianus colchicus*) found dead with 29 lead shot in its gizzard had 168 mg/kg (wet weight presumed but not stated) of lead in the liver (Hunter and Rosen 1965). Two female ring-necked pheasants from shooting estates with ingested lead shot had lead concentrations of 378 and 220 mg/kg dry weight in wing bones (Butler et al. 2005). A northern bobwhite (*Colinus virginianus*) observed in the field with partial paralysis one day and found dead the next had two lead shot pellets in its gizzard and a liver lead concentration of 399 mg/kg wet weight (Lewis and Schweitzer 2000). Keymer and Stebbings (1987) reported lead poisoning as the cause of death in a gray partridge (*Perdix perdix*) with 40 mg/kg wet weight of lead in the liver and 100 mg/kg wet weight in the kidney. An emaciated gray partridge had lead residues of 130 mg/kg in the liver and 440 in the kidney (wet weight presumed but not stated) with 34 lead pellets in the gizzard (Clausen and Wolstrup 1979). Gjerstad and Hanssen (1984) administered doses to willow ptarmigan (*Lagopus lagopus*) of one, three, or six lead shot. Three ptarmigan that died had liver lead residues of 64, 134, and 274 mg/kg wet weight. Birds given doses of one lead shot survived with no clinical signs and had mean liver lead residues of about 3 mg/kg wet weight 15 days after dosing. Thirty-three greater sage-grouse (*Centrocercus urophasianus*) diagnosed with West Nile virus or trauma as cause of death had liver lead concentrations of ≤0.66 mg/kg wet weight (Dailey et al. 2008). When northern bobwhite were fed increasing amounts of lead acetate until half of the birds died, lead residues in the liver and kidney were 21–277 and 85–500 mg/kg wet weight, respectively (Beyer et al. 1988). Chickens (*Gallus gallus*) that died after being fed lead-containing grit had liver lead residues of up to 54 mg/kg wet weight, while liver lead in chickens that exhibited no clinical signs and were euthanized was 1–6 mg/kg wet weight (Salisbury et al. 1958). ALAD was inhibited but there was no effect on hemoglobin or packed cell volume in Japanese quail that had liver lead concentrations of about 0.5, 4, and 6 mg/kg and kidney concentrations of 6, 20, and 30 mg/kg wet weight (Stone and Soares 1976, Stone et al. 1977, 1979). Northern bobwhite given doses of lead shot had a pooled blood lead concentration of 43 mg/kg 9 days post exposure (McConnell 1967). Weakness, lethargy, and loss of weight were seen in 19% of the quail and 10% died. Chickens fed lead acetate exhibited severely inhibited ALAD activity, weight loss, and mild anemia with blood lead concentrations of 322–832 µg/dL (c. 3.22–8.32 mg/kg) (Franson and Custer 1982). Mean liver and kidney lead residues were 17 and 56 mg/kg dry weight after 28 days. Japanese quail given doses of lead shot had lead residues of 7 mg/kg wet weight in the blood, 3 mg/kg in the liver, and

5 mg/kg in the kidney (Yamamoto et al. 1993). ALAD was inhibited, but no other clinical signs were reported.

16.9.5 Passeriformes (Perching Birds)

Two yellow-billed cuckoos (*Coccyzus americanus*) collected near a zinc smelter had liver lead concentrations of 18 and 25 mg/kg wet weight and kidney residues of 21 and 14 mg/kg wet weight (Beyer et al. 1985). ALAD was inhibited, but there was no anemia or gross or microscopic lesions of lead poisoning. Two American robins (*Turdus migratorius*), a northern cardinal (*Cardinalis cardinalis*), and a brown thrasher (*Toxostoma rufum*) sampled in a area with a history of mining and smelting lead and zinc had liver lead concentrations of 12–94 mg/kg dry weight, kidney lead concentrations of 25–150 mg/kg dry weight, and ALAD activities of <10% of reference values (Beyer et al. 2004). Getz et al. (1977) measured tissue lead residues in songbirds collected in urban and rural areas. House sparrows (*Passer domesticus*), starlings (*Sturnus vulgaris*), common grackles (*Quiscalus quiscula*), and American robins had maximum liver lead residues of 10–16 mg/kg dry weight and kidney residues of 14–98 mg/kg dry weight. The concentration of lead in the liver of a dark-eyed junco (*Junco hyemalis*) collected at a trap and skeet range was 9.3 mg/kg dry weight (Vyas et al. 2000). Three brown-headed cowbirds (*Molothrus ater*) that died after being dosed with lead shot had liver lead concentrations of 71–137 mg/kg dry weight (Vyas et al. 2001). Starlings given doses of trialkyl lead died within 6 days of treatment, with mean liver and kidney residues of 40 and 20 mg/kg wet weight, respectively (Osborn et al. 1983). Beyer et al. (1988) fed lead acetate to red-winged blackbirds (*Agelaius phoeniceus*), brown-headed cowbirds, and common grackles until half of the birds in each group died. Median liver lead concentrations were 20–50 mg/kg wet weight (range, 4–97 mg/kg), and median kidney residues were 22–160 mg/kg wet weight (range, 2–740 mg/kg).

16.9.6 Charadriiformes (Shorebirds, Gulls, and Alcids)

Locke et al. (1991) reported a liver lead concentration of 52 mg/kg wet weight in a marbled godwit (*Limosa fedoa*) that died of lead poisoning after ingesting lead shot. Seven herring gull (*Larus argentatus*) chicks that received a one-time injection of lead nitrate survived 45 days but exhibited slower growth than did controls, with mean lead concentrations of 197 μg/dL (c. 1.97 mg/kg) in the blood, 21 mg/kg dry weight in the liver, and 41 mg/kg dry weight in the kidney (Burger and Gochfeld 1990). Apparently healthy adult laughing gulls (*Larus atricilla*) collected in a lead-contaminated area had mean liver lead residues of 4–5 mg/kg and kidney residues of 2 mg/kg wet weight (Munoz et al. 1976, Hulse et al. 1980). Adult royal terns (*Thalasseus maximus*) collected in the same region had liver and kidney residues of 0.4 and 1.1 mg/kg wet weight, while sandwich terns (*Thalasseus sandvicensis*) had liver and kidney residues of 0.5 and 0.8 mg/kg wet weight (Maedgen et al. 1982). Herring gulls feeding at a dump site had liver and kidney lead residues of 3 and 13 mg/kg dry weight, respectively (Leonzio et al. 1986). Black-headed gulls (*Larus ridibundus*) collected at the same location had liver and kidney residues of 8 and 31 mg/kg dry weight, respectively.

16.9.7 Gruiformes (Cranes and Their Allies)

Windingstad et al. (1984) reported on two wild sandhill cranes (*Grus canadensis*) that died of lead poisoning with parts of fishing sinkers in their gizzards. One bird had liver and kidney lead concentrations of 23 and 30 mg/kg wet weight, while the other had liver and kidney lead residues of 259 and 113 mg/kg dry weight. A captive sandhill crane that died after ingesting two 0.22-caliber rifle cartridges had 30 mg/kg wet weight of lead in its liver, and another dead captive sandhill crane with an ingested copper-coated penny had a liver lead concentration of 24 mg/kg wet weight

(Windingstad et al. 1984). A sandhill crane that died after exposure to lead-based paint in a zoo facility had liver and kidney lead concentrations of 29 and 19 mg/kg (wet weight presumed but not stated), respectively (Kennedy et al. 1977). Four additional cranes in this facility developed clinical signs of lead poisoning with blood lead concentrations of 146–378 μg/100 mL (c. 1.46–3.78 mg/kg), but they recovered following chelation treatment. Lead concentrations in kidneys of two houbara bustards (*Chlamydotis undulata maqueenii*) that died on a farm and were diagnosed with lead poisoning resulting from the ingestion of paint flakes were 47 and 5.5 mg/kg, wet weight (Bailey et al. 1995). A whooping crane (*Grus americana*) had a blood lead concentration of 5.6 mg/kg shortly before it died and was found to have liver and kidney concentrations of 24 and 10 mg/kg wet weight (Snyder et al. 1991). An emaciated Mississippi sandhill crane (*Grus canadensis pulla*) found with an unidentified lead object in its stomach had 70 mg/kg wet weight lead in liver tissue (Franson and Hereford 1994). Two Japanese cranes (*Grus japonensis*) found dead had liver lead concentrations of 32 and 62 mg/kg dry weight and kidney lead concentrations of 31 and 34 mg/kg dry weight (Teraoka et al. 2007). Apparently healthy soras (*Porzana carolina*) collected with lead shot in their gizzards had mean liver lead residues of up to 3 mg/kg wet weight and up to 11 mg/kg dry weight in bone (Stendell et al. 1980).

16.9.8 Ciconiiformes (Herons and Their Allies)

A black-crowned night heron (*Nycticorax nycticorax*) with a lead jig head in its stomach had a liver lead concentration of 26 mg/kg wet weight (Franson et al. 2003), but a number of surveys of lead in herons have reported liver concentrations of <6.7 mg/kg dry weight (see Custer 2000). Apparently healthy cattle egrets (*Bubulcus ibis*) collected in an industrialized area had liver lead concentrations of 0.07–1.3 mg/kg and kidney concentrations of 0.08–3.5 mg/kg wet weight (Hulse et al. 1980).

16.9.9 Gaviiformes (Loons)

Locke et al. (1982) reported liver lead residues of 21–38 mg/kg wet weight in three common loons that died of lead poisoning. Two of the three had lead fishing sinkers in gizzard contents. Liver lead concentrations were 5–41 mg/kg wet weight in 36 additional cases of lead-poisoned loons, 33 of which had ingested lead fishing weights (Franson and Cliplef 1992, Pokras and Chafel 1992, Stone and Okoniewski 2001). Of 522 common loons examined from New England, Sidor et al. (2003) confirmed lead poisoning in 68 and assigned a diagnosis of suspected lead poisoning in 50 more. The average lead concentration in the livers of confirmed lead poisoning cases in that study was 17.6 mg/kg wet weight. Franson et al. (2003) found liver lead concentrations of 8.0–16.9 mg/kg wet weight in four common loons with ingested lead fishing weights. In two other common loons with ingested lead, blood and liver lead levels (mg/kg wet weight) were 4.2 and 16.6, and 6.0 and 12.8, respectively, whereas one loon with an ingested jig head had 0.30 mg/kg lead in blood (liver was not tested) (Franson et al. 2003). In a summary of Canadian data, the average lead concentrations in liver (n = 12) and kidney (n = 25) of common loons with ingested lead artifacts were 59 and 218 mg/kg dry weight, respectively (Scheuhammer et al. 2003b). Kidney lead concentrations were 15–167 mg/kg wet weight in six common loons that died of lead poisoning, four of which were determined to have remnants of lead fishing weights in their gizzards (Daoust et al. 1998). Although lead fishing weights account for most of the reported lead poisonings in loons, a Pacific loon (*Gavia pacifica*) found in Alaska had three ingested lead shot, and a liver lead concentration of 31 mg/kg wet weight (Wilson et al. 2004).

16.9.10 Strigiformes (Owls)

Two captive snowy owls (*Nyctea scandiaca*) that died of lead poisoning had 45 and 204 mg/kg dry weight of lead in the liver and 68 and 146 mg/kg dry weight in the kidney (MacDonald et al. 1983). Exposure was thought to have resulted from consumption of bullet fragments in food items. Eastern

screech owls (*Otus asio*) fed lead acetate until half the birds died had median liver and kidney lead concentrations of 22 and 33 mg/kg wet weight (Beyer et al. 1988). In a great-horned owl (*Bubo virginianus*) with necropsy lesions consistent with lead poisoning, the lead concentration in the liver or kidney was >30 or >20 mg/kg dry weight, respectively (Clark and Scheuhammer 2003).

16.9.11 Procellariiformes (Tubenoses)

Lead poisoning from consumption of paint chips in association with buildings contributed to epizootic mortality in Laysan albatross (*Diomedea immutabilis*) chicks on Midway Atoll (Sileo and Fefer 1987). Some of the sick birds were unable to retract their wings, resulting in a "droop-wing" appearance, and in 10 of these birds diagnosed with lead poisoning, lead was detected in the blood of each (maximum of 4.8 mg/kg wet weight), in the liver of 8 (maximum 110 mg/kg dry weight), and in the kidney of 1 (44 mg/kg dry weight) (Sileo and Fefer 1987). In later studies on Midway, droop-wing albatross chicks had mean lead concentrations of 14.4 and 14.0 mg/kg wet weight in liver and kidney, respectively, and 410 µg/dL (c. 4.10 mg/kg) in blood (Burger and Gochfeld 2000, Finkelstein et al. 2003). Work and Smith (1996) found that ALAD in albatross chicks decreased at blood lead concentrations >0.05 µg/mL (c. >0.05 mg/kg).

16.9.12 Pelecaniformes (Pelicans and Their Allies)

Seven brown pelicans (*Pelecanus occidentalis*) with ingested lead fishing weights, sampled when still alive, had blood lead concentrations of 0.04–13.9 mg/kg wet weight (Franson et al. 2003).

16.9.13 Phoenicopteriformes (Flamingos)

Liver lead concentrations of 17 Caribbean flamingos (*Phoenicopterus ruber ruber)* that died of lead poisoning after consumption of lead shot ranged from 128 to 771 mg/kg dry weight (Schmitz et al. 1990). In two lead poisoning events in greater flamingos (*Phoenicopterus ruber*), dead birds had eight or more ingested lead shot and minimum liver lead concentrations were 12.6 mg/kg wet weight and 77.2 mg/kg dry weight (Ramo et al. 1992, Mateo et al. 1997a).

16.9.14 Piciformes (Woodpeckers)

Liver and kidney lead residues were 20 and 76 mg/kg wet weight, respectively, in an emaciated gray-headed woodpecker (*Picus canus*), 9.4 and 1.4 mg/kg wet weight, respectively in a second gray-headed woodpecker that died of traumatic injuries, and 26.2 and <0.02 mg/kg, respectively, in a white-backed woodpecker (*Dendrocopus leucotos*) that died during a translocation program (Mörner and Petersson 1999).

Summary

Lead is a nonessential, highly toxic heavy metal that affects all body systems. Birds are exposed to lead from direct consumption of spent lead shot, bullets, or fragments thereof in prey items, ingestion of lead fishing weights and chips of lead-based paints, and environmental contamination of urban and industrial areas. However, the majority of cases of poisoning result from the ingestion of lead from spent ammunition or ammunition fragments, or angler's lead weights. First recognized more than a century ago, lead poisoning of birds has been reported in many areas of the world.

When lead is absorbed into the bloodstream, the first measurable effects are the inhibition of heme-biosynthetic enzymes. There does not appear to be a no-effect level for lead, because the activities of certain enzymes have been reported to be inhibited at blood lead concentrations of <5 μg/dL (c. <0.05 mg/kg). From the blood, lead is deposited into soft tissues, particularly liver and kidney, growing feathers, and bone. Lead has different retention times in these tissues, with blood and other soft tissue concentrations reflecting recent exposure, lead in feathers reflecting exposure at the time of feather growth, and bone lead concentrations reflecting long-term absorption and accumulation over the lifetime of the bird. Many factors influence lead absorption and distribution within the body, including age, gender, physiological condition, diet, and exposure level and duration. Tissue lead residues associated with physiological injury, clinical signs, and death due to lead poisoning also vary among species, and assessments of toxicosis should be done by comparison with data from phylogenetically related groups whenever possible.

We consider background concentrations of lead to be <20 μg/dL (c. <0.2 mg/kg) in blood, <2 mg/kg wet weight in liver and kidney, and <10 mg/kg dry weight in bone of birds. The suggested thresholds of increasing severity of effects for Anseriformes are: 20–<50 μg/dL (c. 0.2–<0.5 mg/kg) in blood, 2 < 6 mg/kg wet weight in liver and kidney (subclinical poisoning); 50–100 μg/dL (c. 0.5–1 mg/kg) in blood, 6–10 mg/kg wet weight in liver, 6–15 mg/kg wet weight in kidney (clinical poisoning); >100 μg/dL (c. >1 mg/kg) in blood, >10 mg/kg wet weight in liver, >15 mg/kg wet weight in kidney (severe clinical poisoning). Suggested thresholds for Falconiformes: 20–<50 μg/dL (c. 0.2–<0.5 mg/kg) in blood, 2 < 6 mg/kg wet weight in liver, 2 < 4 mg/kg wet weight in kidney (subclinical poisoning); 50–100 μg/dL (c. 0.5–1 mg/kg) in blood, 6–10 mg/kg wet weight in liver, 4–6 mg/kg wet weight in kidney (clinical poisoning); >100 μg/dL (c. >1 mg/kg) in blood, >10 mg/kg wet weight in liver, >6 mg/kg wet weight in kidney (severe clinical poisoning). Suggested thresholds for Columbiformes: 20 < 200 μg/dL (c. 0.2 < 2 mg/kg) in blood, 2 < 6 mg/kg wet weight in liver, 2 < 15 mg/kg wet weight in kidney (subclinical poisoning); 200–300 μg/dL (c. 2–3 mg/kg) in blood, 6–15 mg/kg wet weight in liver, 15–30 mg/kg wet weight in kidney (clinical poisoning); >300 μg/dL (c. >3 mg/kg) in blood, >15 mg/kg wet weight in liver, >30 mg/kg wet weight in kidney (severe clinical poisoning). For birds in general, liver lead concentrations within the clinical poisoning range (≥6 mg/kg wet weight) support a lead poisoning diagnosis where necropsy observations are consistent with lead poisoning.

Bone lead concentrations of >20 mg/kg dry weight are considered to suggest excessive exposure. Because of the rapid uptake and slow release of lead from bone, bone lead concentrations are the least useful index of recent lead poisoning, although bone lead concentrations can be used to determine geographical patterns of poisoning in populations.

Care should be exercised when comparing tissue lead residues from experimental studies with field data. Wild birds with inadequate diets and exposed to ambient environmental conditions may be more susceptible to lead toxicosis than are birds in controlled situations. A clinical evaluation of health and the determination of lead residues in sequential blood samples will enhance the assessment of lead exposure and toxicity in live birds. Although conclusions can be drawn regarding the severity of lead poisoning from tissue concentrations alone, a definitive diagnosis of lead poisoning as the cause of death in an individual bird requires interpretation of tissue lead residues in dead birds accompanied by an examination of carcasses for gross and microscopic lesions of lead poisoning.

REFERENCES

Adler, F. E. W. 1944. Chemical analyses of organs from lead-poisoned Canada geese. *J. Wildl. Manag.* 8:83–85.

Adrian, W. J., and M. L. Stevens. 1979. Wet versus dry weights for heavy metal toxicity determinations in duck liver. *J. Wildl. Dis.* 15:125–126.

Anders, E., et al. 1982. Morphological, pharmacokinetic, and hematological studies of lead-exposed pigeons. *Environ. Res.* 28:344–363.

Anderson, W. L. 1975. Lead poisoning in waterfowl at Rice Lake, Illinois. *J. Wildl. Manag.* 39:264–70.

Avery, D., and R. T. Watson. 2009. Regulation of lead-based ammunition around the world. In *Ingestion of lead from spent ammunition: Implications for wildlife and humans*, eds. R. T. Watson, M. Fuller, M. Pokras, and W. G. Hunt. Boise: The Peregrine Fund. DOI 10.4080/ilsa.2009.0115.

Bagley, G. E., and L. N. Locke. 1967. The occurrence of lead in tissues of wild birds. *Bull. Environ. Contam. Toxicol.* 2:297–305.

Bagley, G. E., L. N. Locke, and G. T. Nightingale. 1967. Lead poisoning in Canada geese in Delaware. *Avian Dis.* 11:601–608.

Bailey, T. A., J. H. Samour, J. Naldo, and J. C. Howlett. 1995. Lead toxicosis in captive houbara bustards (*Chlamydotis undulata maqueenii*). *Vet. Rec.* 137:193–194.

Barthalmus, G. T., J. D. Leander, D. E. McMillan, P. Mushak, and M. R. Krigman. 1977. Chronic effects of lead on schedule-controlled pigeon behavior. *Toxicol. Appl. Pharmacol.* 42:271–284.

Battaglia, A., S. Ghidini, G. Campanini, and R. Spaggiari. 2005. Heavy metal contamination in little owl (*Athene noctua*) and common buzzard (*Buteo buteo*) from northern Italy. *Ecotoxicol. Environ. Saf.* 60:61–66.

Bellrose, F. C. 1959. Lead poisoning as a mortality factor in waterfowl populations. *Ill. Nat. Hist. Surv. Bull.* 27:235–288.

Benson, W. W., B. Pharaoh, and P. Miller. 1974. Lead poisoning in a bird of prey. *Bull. Environ. Contam. Toxicol.* 11:105–108.

Beyer, W. N., O. H. Pattee, L. Sileo, D. J. Hoffman, and B. M. Mulhern. 1985. Metal contamination in wildlife living near two zinc smelters. *Environ. Pollut. Ser. A* 38:63–86.

Beyer, W. N., J. W. Spann, L. Sileo, and J. C. Franson. 1988. Lead poisoning in six captive avian species. *Arch. Environ. Contam. Toxicol.* 17:121–130.

Beyer, W. N., J. C. Franson, L. N. Locke, R. K. Stroud, and L. Sileo. 1998. Retrospective study of the diagnostic criteria in a lead-poisoning survey of waterfowl. *Arch. Environ. Contam. Toxicol.* 35:506–512.

Beyer, W. N., D. J. Audet, G. H. Heinz, D. J. Hoffman, and D. Day. 2000. Relation of waterfowl poisoning to sediment lead concentrations in the Coeur d'Alene River Basin. *Ecotoxicology* 9:207–218.

Beyer, W. N., et al. 2004. Zinc and lead poisoning in wild birds in the tri-state mining district (Oklahoma, Kansas, and Missouri). *Arch. Environ. Contam. Toxicol.* 48:108–117.

Birkhead, M. 1983. Lead levels in the blood of mute swans *Cygnus olor* on the River Thames. *J. Zool. (Lond.)* 199:59–73.

Blus, L. J., R. K. Stroud, B. Reiswig, and T. McEneaney. 1989. Lead poisoning and other mortality factors in trumpeter swans. *Environ. Toxicol. Chem.* 8:263–271.

Blus, L. J., C. J. Henny, D. J. Hoffmann, and R. A. Grove. 1991. Lead toxicosis in tundra swans near a mining and smelting complex in northern Idaho. *Arch. Environ. Contam. Toxicol.* 21:549–555.

Blus, L. J., C. J. Henny, D. J. Hoffman, and R. A. Grove. 1993. Accumulation and effects of lead and cadmium on wood ducks near a mining and smelting complex in Idaho. *Ecotoxicology* 2:139–154.

Blus, L. J., C. J. Henny, D. J. Hoffman, L. Sileo, and D. J Audet. 1999. Persistence of high lead concentrations and associated effects in tundra swans captured near a mining and smelting complex in Northern Idaho. *Ecotoxicology* 8:125–132.

Bostan, N., M. Ashraf, A. S. Mumtaz, and I. Ahmad. 2007. Diagnosis of heavy metal contamination in agro-ecology of Gujranwala, Pakistan using cattle egret (*Bubulcus ibis*) as bioindicator. *Ecotoxicology* 16:247–251.

Boyer, I. J., D. A. Cory-Slechta, and V. DiStefano. 1985. Lead induction of crop dysfunction in pigeons through a direct action on neural or smooth muscle components of crop tissue. *J. Pharmacol. Exp. Ther.* 234:607–615.

Buerger, T. T., R. E. Mirarchi, and M. E. Lisano. 1986. Effects of lead shot ingestion on captive mourning dove survivability and reproduction. *J. Wildl. Manag.* 50:1-8.

Burger, J. 1993. Metals in avian feathers: Bioindicators of environmental pollution. *Rev. Environ. Toxicol.* 5:203–311.

Burger, J., and M. Gochfeld. 1990. Tissue levels of lead in experimentally exposed herring gull (*Larus argentatus*) chicks. *J. Toxicol. Environ. Health* 29:219–233.

Burger, J., and M. Gochfeld. 2000. Metals in Laysan albatrosses from Midway Atoll. *Arch. Environ. Contam. Toxicol.* 38:254–259.

Butler, D. A., R. B. Sage, R. A. H. Draycott, J. P. Carroll, and D. Potts. 2005. Lead exposure in ring-necked pheasants on shooting estates in Great Britain. *Wildl. Soc. Bull.* 33:583–589.

Cade, T. J. 2007. Exposure of California condors to lead from spent ammunition. *J. Wildl. Manag.* 71:2125–2133.

Carpenter, J. W., et al. 2003. Experimental lead poisoning in turkey vultures (*Cathartes aura*). *J. Wildl. Dis.* 39:96–104.

Castrale, J. S., and M. Oster. 1993. Lead and delta-aminolevulinic acid dehydratase in the blood of mourning doves dosed with lead shot. *Proc. Indiana Acad. Sci.* 102:265–272.

Chupp, N. R., and P. D. Dalke. 1964. Waterfowl mortality in the Coeur d'Alene River Valley, Idaho. *J. Wildl. Manag.* 28:692–702.

Church, M. E., et al. 2006. Ammunition is the principal source of lead accumulated by California condors re-introduced to the wild. *Environ. Sci. Technol.* 40:6143–6150.

Clark, A. J., and A. M. Scheuhammer. 2003. Lead poisoning in upland-foraging birds of prey in Canada. *Ecotoxicology* 12:23–30.

Clausen, B., and C. Wolstrup. 1979. Lead poisoning in game from Denmark. *Dan. Rev. Game Biol.* 11:1–22.

Clausen, B., K. Elvestad, and O. Karlog. 1982. Lead burden in mute swans from Denmark. *Nord. Veterinaermed.* 34:83–91.

Clemens, E. T., L. Krook, A. L. Aronson, and C. E. Stevens. 1975. Pathogenesis of lead shot poisoning in the mallard duck. *Cornell Vet.* 65:248–285.

Coburn, D. R., D. W. Metzler, and R. Treichler. 1951. A study of absorption and retention of lead in wild waterfowl in relation to clinical evidence of lead poisoning. *J. Wildl. Manag.* 15:186–192.

Cook, R. S., and D. O. Trainer. 1966. Experimental lead poisoning of Canada geese. *J. Wildl. Manag.* 30:1-8.

Cory-Slechta, D. A., R. H. Garman, and D. Seidman. 1980. Lead-induced crop dysfunction in the pigeon. *Toxicol. Appl. Pharmacol.* 52:462–467.

Craig, T. H., J. W. Connelly, E. H. Craig, and T. L. Parker. 1990. Lead concentrations in golden and bald eagles. *Wilson Bull.* 102:130–133.

Custer, T. W. 2000. Environmental contaminants. In *Heron conservation,* eds. J.A. Kushlan and H. Hafner, pp. 251–267. San Diego: Academic Press.

Custer, T. W., J. C. Franson, and O. H. Pattee. 1984. Tissue lead distribution and hematologic effects in American kestrels (*Falco sparverius* L.) fed biologically incorporated lead. *J. Wildl. Dis.* 20:39–43.

Dailey, R. N., M. F. Raisbeck, R. S. Siemion, and T. E. Cornish. 2008. Liver metal concentrations in greater sage-grouse (*Centrocercus urophasianus*). *J. Wildl. Dis.* 44:494–498.

Daoust, P. Y., G. Conboy, S. McBurney, and N. Burgess. 1998. Interactive mortality factors in common loons from Maritime Canada. *J. Wildl. Dis.* 34:524–531.

Decker, R. A., A. M. McDermid, and J. W. Prideaux. 1979. Lead poisoning in two captive king vultures. *J. Am. Vet. Med. Assoc.* 175:1009.

Degernes, L. A. 1991. The Minnesota trumpeter swan lead poisoning crisis of 1988–89. In *Proceedings and papers of the 12th trumpeter swan society conference*, pp. 114–118. Minneapolis: Trumpeter Swan Society.

Degernes, L., et al. 2006. Epidemiologic investigation of lead poisoning in trumpeter and tundra swans in Washington state, USA, 2000–2002. *J. Wildl. Dis.* 42:345–358.

Demayo, A., M. C. Taylor, K. W. Taylor, and P. V. Hodson. 1982. Toxic effects of lead and lead compounds on human health, aquatic life, wildlife plants, and livestock. *CRC Crit. Rev. Environ. Control* 12:257–305.

DeMent, S. H., J. J. Chisolm, Jr., M. A. Eckhaus, and J. D. Strandberg. 1987. Toxic lead exposure in the urban rock dove. *J. Wildl. Dis.* 23:273–278.

Denbow, D. M. 2000. Gastrointestinal anatomy and physiology. In *Sturkie's avian physiology*, 5th ed., ed. G. C. Whittow, pp. 299–325. San Diego: Academic Press.

DeStefano, S., C. J. Brand, D. H. Rusch, D. L. Finley, and M. M. Gillespie. 1991. Lead exposure in Canada geese of the eastern prairie population. *Wildl. Soc. Bull.* 19:23–32.

Dieter, M. P. 1979. Blood delta-aminolevulinic acid dehydratase (ALAD) to monitor lead contamination in canvasback ducks (*Aythya valisineria*). In *Animals as monitors of environmental pollutants*, eds. S. W. G. Nielsen, G. Migaki, and D. G. Scarpelli, pp. 177–191. Washington, D.C.: National Academy of Sciences.

Dieter, M. P., and M. T. Finley. 1978. Erythrocyte delta-aminolevulinic acid dehydratase activity in mallard ducks: duration of inhibition after lead shot dosage. *J. Wildl. Manage.* 42:621–625.

Dieter, M. P., and M. T. Finley. 1979. Delta-aminolevulinic acid dehydratase enzyme activity in blood, brain, and liver of lead-dosed ducks. *Environ. Res.* 19:127–135.

Dieter, M. P., M. C. Perry, and B. M. Mulhern. 1976. Lead and PCB's in canvasback ducks: relationship between enzyme levels and residues in blood. *Arch. Environ. Contam. Toxicol.* 5:1–13.

Dietz, D. D., D. E. McMillan, and P. Mushak. 1979. Effects of chronic lead administration on acquisition and performance of serial position sequences by pigeons. *Toxicol. Appl. Pharmacol.* 47:377–384.

Duke, G. E. 1986. Raptor physiology. In *Zoo and wild animal medicine*, 2nd ed., ed. D. Pedersen, pp. 370–376. Philadelphia: W.B. Saunders Company.

Eisler, R. 2000. *Handbook of chemical risk assessment*, Vol. 1. Metals. Boca Raton, FL: Lewis Publishers.

Elliott, J. E., K. M. Langelier, A. M. Sheuhammer, P. H. Sinclair, and P. E. Whitehead. 1992. Incidence of lead poisoning in bald eagles and lead shot in waterfowl gizzards from British Columbia, 1988–91. *Canadian Wildlife Service Progress Notes No.* 200:1–7.

Fair, J. M., and O. B. Myers. 2002. The ecological and physiological costs of lead shot and immunological challenge to developing western bluebirds. *Ecotoxicology* 11:199–208.

Fair, J. M., and R. E. Ricklefs. 2002. Physiological, growth, and immune responses of Japanese quail chicks to the multiple stressors of immunological challenge and lead shot. *Arch. Environ. Contam. Toxicol.* 42:77–87.

Figuerola, J., R. Mateo, A. J. Green, J. Y. Mondain-Monval, H. LeFranc, and G. Mentaberre. 2005. Grit selection in waterfowl and how it determines exposure to ingested lead shot in Mediterranean wetlands. *Environ. Conserv.* 32:226–234.

Finkelstein, M. E., R. H. Gwiazda, and D. R. Smith. 2003. Lead poisoning of seabirds: environmental risks from leaded paint at a decommissioned military base. *Environ. Sci. Technol.* 37:3256–3260.

Finley, M. T., M. P. Dieter, and L. N. Locke. 1976a. Lead in tissues of mallard ducks dosed with two types of lead shot. *Bull. Environ. Contam. Toxicol.* 16:261–269.

Finley, M. T., M. P. Dieter, and L. N. Locke. 1976b. Delta-aminolevulinic acid dehydratase: inhibition in duck dosed with lead shot. *Environ. Res.* 12:243–249.

Finley, M. T., M. P. Dieter, and L. N. Locke. 1976c. Sublethal effects of chronic lead ingestion in mallard ducks. *J. Toxicol. Environ. Health* 1:929–937.

Finley, M. T., and M. P. Dieter. 1978. Influence of laying on lead accumulation in bone of mallard ducks. *J. Toxicol. Environ. Health* 4:123–129.

Fisher, I. J., D. J. Pain, and V. G. Thomas. 2006. A review of lead poisoning from ammunition sources in terrestrial birds. *Biol. Conserv.* 131:421–432.

Flint, P. L., M. R. Petersen, and J. B. Grand. 1997. Exposure of spectacled eiders and other diving ducks to lead in western Alaska. *Can. J. Zool.* 75:439–443.

Forbes, R. M., and G. C. Sanderson. 1978. Lead toxicity in domestic animals and wildlife. In *The biogeochemistry of lead in the environment. Part B. Biological effects*, ed. J. O. Nriagu, pp. 225–277. Amsterdam: Elsevier/North-Holland Biomedical Press.

Franson, J. C., and T. W. Custer. 1982. Toxicity of dietary lead in young cockerels. *Vet. Hum. Toxicol.* 24:421–423.

Franson, J. C., G. M. Haramis, M. C. Perry, and J. F. Moore. 1986. Blood protoporphyrin for detecting lead exposure in canvasbacks. In *Lead poisoning in wild waterfowl—a workshop*, eds. J. S. Feierabend, and A. B. Russell, pp. 32–37. Washington, D.C.: National Wildlife Federation.

Franson, J., and D. J. Cliplef. 1992. Causes of mortality in common loons. In *Proceedings from the 1992 conference on the loon and its ecosystem: status, management, and environmental concerns*, eds. L. Morse, S. Stockwell, and M. Pokras, pp. 2–12. Concord: U.S. Fish and Wildlife Service.

Franson, J. C., and S. G. Hereford. 1994. Lead poisoning in a Mississippi sandhill crane. *Wilson Bull.* 106:766–768.

Franson, J. C., P. S. Koehl, D. V. Derksen, T. C. Rothe, C. M. Bunck, and J. F. Moore. 1995a. Heavy metals in seaducks and mussels from Misty Fjords National Monument in southeast Alaska. *Environ. Monit. Assess.* 36:149–167.

Franson, J. C., M. R. Petersen, C. U. Meteyer, and M. R. Smith. 1995b. Lead poisoning of spectacled eiders (*Somateria fischeri*) and of a common eider (*Somateria mollissima*) in Alaska. *J. Wildl. Dis.* 31:268–271.

Franson, J. C., W. L. Hohman, J. L. Moore, and M. R. Smith. 1996a. The efficacy of protoporphyrin as a predictive biomarker for lead exposure in canvasback ducks: effect of sample storage time. *Environ. Monit. Assess.* 43:181–188.

Franson, J. C., N. J. Thomas, M. R. Smith, A. H. Robbins, S. Newman, and P. C. McCartin. 1996b. A retrospective study of postmortem findings in red–tailed hawks. *J. Raptor. Res.* 30:7–14.

Franson, J. C., T. Hollmén, M. Hario, M. Kilpi, and D. L. Finley. 2002. Lead and delta-aminolevulinic acid dehydratase in blood of common eiders (*Somateria mollissima*) from the Finnish archipelago. *Ornis Fenn.* 79:87–91.

Franson, J. C., et al. 2003. Lead fishing weights and other fishing tackle in selected waterbirds. *Waterbirds* 26:345–352.

Franson, J. C., T. E. Hollmén, P. L. Flint, J. B. Grand, and R. B. Lanctot. 2004. Contaminants in molting long-tailed ducks and nesting common eiders in the Beaufort Sea. *Mar. Pollut. Bull.* 48:504–513.

Franson, J. C., S. P. Hansen, and J. H. Schulz. 2009. Ingested shot and tissue lead concentrations in mourning doves. In *Ingestion of lead from spent lead ammunition: Implications for wildlife and humans*, eds. R.T. Watson, M. Fuller, M. Pokras, and W.G. Hunt. Boise: The Peregrine Fund. DOI 10.4080/ilsa.2009.0202.

Frenzel, R. W., and R. G. Anthony. 1989. Relationship of diets and environmental contaminants in wintering bald eagles. *J. Wildl. Manag.* 53:792–802.

Friend, M. 1999. Lead. In *Field manual of wildlife diseases, general field procedures and diseases of birds*, eds. M. Friend and J. C. Franson, pp. 317–334. U.S. Geological Survey Information and Technology Report 1999-001. Washington, D. C.: U.S. Department of the Interior.

Fry, D. M. 2004. Analysis of lead in California condor feathers: determination of exposure and depuration during feather growth. California Department of Fish and Game, Species Conservation and Recovery Program Report, 2004-02, Sacramento.

Fry, D. M., and J. R. Maurer. 2003. Assessment of lead contamination sources exposing California condors. Final report to the California Department of Fish and Game, Sacramento.

Garcia-Fernandez, A. J., J. A. Sanchez-Garcia, P. Jimenez-Montalban, and A. Luna. 1995. Lead and cadmium in wild birds in southeastern Spain. *Environ. Toxicol. Chem.* 14:2049–2058.

García-Fernández, A. J., M. Motas-Guzmán, I. Navas, P. María-Mojica, A. Luna, and J. A. Sánchez-García. 1997. Environmental exposure and distribution of lead in four species of raptors in southeastern Spain. *Arch. Environ. Contam. Toxicol.* 33:76–82.

Getz, L. L., L. B. Best, and M. Prather. 1977. Lead in urban and rural song birds. *Environ. Pollut.* 12:235–238.

Gill, C. E., and K. M. Langelier. 1994. Acute lead poisoning in a bald eagle secondary to bullet ingestion. *Can. Vet. J.* 35:303–304.

Gjerstad, K. O., and I. Hanssen. 1984. Experimental lead poisoning in willow ptarmigan. *J. Wildl. Manag.* 48:1018–1022.

Golden, N. H., B. A. Rattner, J. B. Cohen, D. J. Hoffman, E. Russek-Cohen, and M. A. Ottinger. 2003. Lead accumulation in feathers of nestling black-crowned night herons (*Nycticorax nycticorax*) experimentally treated in the field. *Environ. Toxicol. Chem.* 22:1517–1524.

Grand, J. B., P. L. Flint, M. R. Petersen, and C. L. Moran. 1998. Effect of lead poisoning on spectacled eider survival rates. *J. Wildl. Manag.* 62:1103–1109.

Grasman, K. A., and P. F. Scanlon. 1995. Effects of acute lead ingestion and diet on antibody and T-cell-mediated immunity in Japanese quail. *Arch. Environ. Contam. Toxicol.* 28:161–167.

Grue, C. E., D. J. Hoffman, W. N. Beyer, and L. P. Franson. 1986. Lead concentrations and reproductive success in European starlings *Sternus vulgaris* nesting within highway roadside verges. *Environ. Pollut. Ser. A* 42:157–182.

Hall, M., J. Grantham, R. Posey, and A. Mee. 2007. Lead exposure among reintroduced California condors in southern California. In *California condors in the 21st century*. Series in ornithology, no. 2, eds. A. Mee and L. S. Hall, pp. 139–162. Cambridge, MA: Nuttall Ornithology Club and Washington, D.C.: The American Ornithologists' Union.

Harmata, A. R., and M. Restani. 1995. Environmental contaminants and cholinesterase in blood of vernal migrant bald and golden eagles in Montana. *Intermountain J. Sci.* 1:1–15.

Heinz, G. H., D. J. Hoffman, L. Sileo, D. J. Audet, and L. J. LeCaptain. 1999. Toxicity of lead-contaminated sediment to mallards. *Arch. Environ. Contam. Toxicol.* 36:323–333.

Henny, C. J., L. J. Blus, D. J. Hoffman, R. A. Grove, and J. S. Hatfield. 1991. Lead accumulation and osprey production near a mining site on the Coeur d'Alene River, Idaho. *Arch. Environ. Contam. Toxicol.* 21:415–424.

Henny, C. J., L. J. Blus, D. J. Hoffman, and R. A. Grove. 1994. Lead in hawks, falcons and owls downstream from a mining site on the Coeur D'Alene River, Idaho. *Environ. Monit. Assess.* 29:267–288.

Hernberg, S., J. Nikkanen, G. Mellin, and H. Lilius. 1970. Delta-aminolevulinic acid dehydrase as a measure of lead exposure. *Arch. Environ. Health* 21:140–145.

Hoffman, D. J., O. H. Pattee, S. N. Wiemeyer, and B. Mulhern. 1981. Effects of lead shot ingestion on delta-aminolevulinic acid dehydratase activity, hemoglobin concentration, and serum chemistry in bald eagles. *J. Wildl. Dis.* 17:423–431.

Hoffman, D. J., J. C. Franson, O. H. Pattee, C. M. Bunck, and A. Anderson. 1985a. Survival, growth, and accumulation of ingested lead in nestling American kestrels (*Falco sparverius*). *Arch. Environ. Contam. Toxicol.* 14:89–94.

Hoffman, D. J., J. C. Franson, O. H. Pattee, C. M. Bunck, and H. C. Murray. 1985b. Biochemical and hematological effects of lead ingestion in nestling American kestrels (*Falco sparverius*). *Comp. Biochem. Physiol.* 80C:431–439.

Hohman, W. L., J. L. Moore, and J. C. Franson. 1995. Winter survival of immature canvasbacks in inland Louisiana. *J. Wildl. Manag.* 59:384–392.

Hulse, M., J. S. Mahoney, G. D. Schroder, C. S. Hacker, and S. M. Pier. 1980. Environmentally acquired lead, cadmium, and manganese in the cattle egret, *Bubulcus ibis*, and the laughing gull, *Larus atricilla*. *Arch. Environ. Contam. Toxicol.* 9:65–78.

Hunt, W. G., W. Burnham, C. N. Parish, K. K. Burnham, B. Mutch, and J. L. Oaks. 2006. Bullet fragments in deer remains: Implications for lead exposure in avian scavengers. *Wildl. Soc. Bull.* 34:167–170.

Hunter, B., and J. C. Haigh. 1978. Demyelinating peripheral neuropathy in a guinea hen associated with subacute lead intoxication. *Avian Dis.* 22:344–349.

Hunter, B. F., and M. N. Rosen. 1965. Occurrence of lead poisoning in a wild pheasant (*Phasianus colchicus*). *Calif. Fish Game* 51:207.

Hunter, B., and G. Wobeser. 1980. Encephalopathy and peripheral neuropathy in lead-poisoned mallard ducks. *Avian Dis.* 24:169–178.

Hutton, M. 1980. Metal contamination of feral pigeons *Columba livia* from the London area: Part 2—Biological effects of lead exposure. *Environ. Pollut. Ser. A* 22:281–293.

Hutton, M., and G. T. Goodman. 1980. Metal contamination of feral pigeons *Columba livia* from the London area: Part 1—Tissue accumulation of lead, cadmium, and zinc. *Environ. Pollut. Ser. A* 22:207–217.

Irwin, J. C. 1975. Mortality factors in whistling swans at Lake St. Clair, Ontario. *J. Wildl. Dis.* 11:8–12.

Iwata, H., et al. 2000. Contamination by chlorinated hydrocarbons and lead in Steller's sea eagle and white-tailed sea eagle from Hokkaido, Japan. In *First symposium on Steller's and white-tailed sea eagles in east Asia*, eds. M. Ueta, and M. J. McGrady, pp. 91–106. Tokyo: Wild Bird Society of Japan.

Jacobson, E., J. W. Carpenter, and M. Novilla. 1977. Suspected lead toxicosis in a bald eagle. *J. Am. Vet. Med. Assoc.* 171:952–954.

Janssen, D. L., P. T. Robinson, and P. K. Ensley. 1979. Lead toxicosis in three captive avian species. In *Proc. 1979 Annu. Meet. Am. Assoc. Zoo Vet.*, 40–42.

Janssen, D. L., J. E. Oosterhuis, J. L. Allen, M. P. Anderson, D. G. Kelts, and S. N. Wiemeyer. 1986. Lead poisoning in free-ranging California condors. *J. Am. Vet. Med. Assoc.* 189:1115–1117.

Johnson, G. D., et al. 1999. Lead exposure in passerines inhabiting lead-contaminated floodplains in the Coeur D'Alene River Basin, Idaho, USA. *Environ. Toxicol. Chem.* 18:1190–1194.

Johnson, M. S., H. Pluck, M. Hutton, and G. Moore. 1982. Accumulation and renal effects of lead in urban populations of feral pigeons, *Columba livia*. *Arch. Environ. Contam. Toxicol.* 11:761–767.

Jordan, J. S., and F. C. Bellrose. 1951. Lead poisoning in wild waterfowl. *Ill. Nat. Hist. Surv. Biol. Notes* No. 26.

Kaiser, T. E., et al. 1980. Organochlorine pesticide, PCB, and PBB residues and necropsy data for bald eagles from 29 states—1975–77. *Pestic. Monit. J.* 13:145–149.

Karstad, L. 1971. Angiopathy and cardiopathy in wild waterfowl from ingestion of lead shot. *Conn. Med.* 35:355–360.

Kelly, M. E., et al. 1998. Acute effects of lead, steel, tungsten-iron, and tungsten-polymer shot administered to game-farm mallards. *J. Wildl. Dis.* 34:673–687.

Kendall, R. J., and P. F. Scanlon. 1981. Chronic lead ingestion and nephropathy in ringed turtle doves. *Poult. Sci.* 60:2028–2032.

Kendall, R. J., and P. F. Scanlon. 1982. The toxicology of ingested lead acetate in ringed turtle doves *Streptopelia risoria*. *Environ. Pollut. Ser. A* 27:255–262.

Kendall, R. J., P. F. Scanlon, and R. T. Di Giulio. 1982. Toxicology of ingested lead shot in ringed turtle doves. *Arch. Environ. Contam. Toxicol.* 11:259–263.

Kendall, R. J., P. F. Scanlon, and H. P. Veit. 1983. Histologic and ultrastructural lesions of mourning doves (*Zenaida macroura*) poisoned by lead shot. *Poult. Sci.* 62:952–956.

Kendall, R. J., H. P. Veit, and P. F. Scanlon. 1981. Histological effects and lead concentrations in tissues of adult male ringed turtle doves that ingested lead shot. *J. Toxicol. Environ. Health* 8:649–658.

Kendall, R. J., et al. 1996. An ecological risk assessment of lead shot exposure in non-waterfowl avian species: upland game birds and raptors. *Environ. Toxicol. Chem.* 15:4–20.

Kennedy, S., J. P. Crisler, E. Smith, and M. Bush. 1977. Lead poisoning in sandhill cranes. *J. Am. Vet. Med. Assoc.* 171:955–958.

Kenntner, N., O. Krone, R. Altenkamp, and F. Tataruch. 2003. Environmental contaminants in liver and kidney of free-ranging northern goshawks (*Accipiter gentilis*) from three regions of Germany. *Arch. Environ. Contam. Toxicol.* 45:128–135.

Kenntner, N., F. Tataruch, and O. Krone. 2001. Heavy metals in soft tissue of white-tailed eagles found dead or moribund in Germany and Austria from 1993 to 2000. *Environ. Toxicol. Chem.* 20:1831–1837.

Keymer, I. F., and R. St. J. Stebbings. 1987. Lead poisoning in a partridge (*Perdix perdix*) after ingestion of gunshot. *Vet. Rec.* 120:276–277.

Kim, E. Y., R. Goto, H. Iwata, Y. Masuda, S. Tanabe, and S. Fujita. 1999. Preliminary survey of lead poisoning of Steller's sea eagle (*Haliaeetus pelagicus*) and white-tailed sea eagle (*Haliaeetus albicilla*) in Hokkaido, Japan. *Environ. Toxicol. Chem.* 18:448–451.

Kingsford, R. T., J. Flanjak, and S. Black. 1989. Lead shot and ducks on Lake Cowal. *Aust. Wildl. Res.* 16:167–172.

Koranda, J., K. Moore, M. Stuart, and C. Conrado. 1979. Dietary effects on lead uptake and trace element distribution in mallard ducks dosed with lead shot. UCID-18044. Livermore: Lawrence Livermore Laboratory, Environmental Sciences Division.

Kramer, J. L., and P. T. Redig. 1997. Sixteen years of lead poisoning in eagles, 1980-95: an epizootilogic view. *J. Raptor Res.* 31:327–332.

Kreager, N., B. C. Wainman, R. K. Jayasinghe, and L. J. S. Tsuji. 2008. Lead pellet ingestion and liver-lead concentrations in upland game birds from southern Ontario, Canada. *Arch. Environ. Contam. Toxicol.* 54:331–336.

Krementz, D. G., and C. D. Ankney. 1995. Changes in total body calcium and diet of breeding house sparrows. *J. Avian Biol.* 26:162–167.

Krone, O., A. Berger, and R. Schulte. 2009. Recording movement and activity pattern of a white-tailed sea eagle (*Haliaeetus albicilla*) by a GPS datalogger. *J. Ornithol.* 150:273–280.

Krone, O., T. Stjernberg, N. Kenntner, F. Tataruch, J. Koivusaari, and I. Nuuja. 2006. Mortality factors, helminth burden, and contaminant residues in white-tailed sea eagles (*Haliaeetus albicilla*) from Finland. *Ambio* 35:98–104.

Krone, O., F. Wille, N. Kenntner, D. Boertmann, and F. Tataruch. 2004. Mortality factors, environmental contaminants, and parasites of white-tailed sea eagles from Greenland. *Avian Dis.* 48:417–424.

Kurosawa, N. 2000. Lead poisoning in Steller's sea eagles and white-tailed sea eagles. In *First symposium on Steller's and white-tailed sea eagles in east Asia*, eds. M. Ueta and M. J. McGrady, pp. 107–109. Tokyo: Wild Bird Society of Japan.

Langelier, K. M., C. E. Andress, T. K. Grey, C. Wooldridge, R. J. Lewis, and R. Marchetti. 1991. Lead poisoning in bald eagles in British Columbia. *Can. Vet. J.* 32:108–109.

Leonzio, C., C. Fossi, and S. Focardi. 1986. Lead, mercury, cadmium, and selenium in two species of gull feeding on inland dumps, and in marine areas. *Sci. Total Environ.* 57:121–127.

Lewis, L. A., and S. H. Schweitzer. 2000. Lead poisoning in a northern bobwhite in Georgia. *J. Wildl. Dis.* 36:180–183.

Lewis, L. A., R. J. Poppenga, W. R. Davidson, J. R. Fischer, and K. A. Morgan. 2001. Lead toxicosis and trace element levels in wild birds and mammals at a firearms training facility. *Arch. Environ. Contam. Toxicol.* 41:208–214.

Locke, L. N., and G. E. Bagley. 1967. Lead poisoning in a sample of Maryland mourning doves. *J. Wildl. Manage.* 31:515–518.

Locke, L. N., G. E. Bagley, D. N. Frickie, and L. T. Young. 1969. Lead poisoning and aspergillosis in an Andean condor. *J. Am. Vet. Med. Assoc.* 155:1052–1056.

Locke, L. N., and M. Friend. 1992. Lead poisoning of avian species other than waterfowl. In *Lead poisoning in waterfowl*, ed. D. J. Pain, pp. 19–22. IWRB Spec. Publ. 16. Slimbridge: International Waterfowl and Wetlands Research Bureau.

Locke, L. N., S. M. Kerr, and D. Zoromski. 1982. Lead poisoning in common loons (*Gavia immer*). *Avian Dis.* 26:392–396.

Locke, L. N., M. R. Smith, R. M. Windingstad, and S. J. Martin. 1991. Lead poisoning of a marbled godwit. *Prairie Nat.* 23:21–24.

Locke, L. N., and N. J. Thomas. 1996. Lead poisoning of waterfowl and raptors. In *Noninfectious diseases of wildlife*, 2nd ed., eds. A. Fairbrother, L. N. Locke, and G. L. Hoff, pp. 108–117. Ames, IA: Iowa State University Press.

Longcore, J. R., R. Andrews, L. N. Locke, G. E. Bagley, and L. T. Young. 1974a. Toxicity of lead and proposed substitute shot to mallards. *U.S. Fish Wildl. Serv. Spec. Sci. Rep. Wildl.* No. 183. Washington, D.C.: U.S. Department of the Interior.

Longcore, J. R., L. N. Locke, G. E. Bagley, and R. Andrews. 1974b. Significance of lead residues in mallard tissues. *U.S. Fish Wildl. Serv. Spec. Sci. Rep. Wildl.* No. 182. Washington, D.C.: U.S. Department of the Interior.

Lumeij, J. T., W. T. C. Wolvekamp, G. M. Bron-Dietz, and A. J. H. Schotman. 1985. An unusual case of lead poisoning in a honey buzzard (*Pernis apivorus*). *Vet. Q.* 7:165–168.

MacDonald, J. W., C. J. Randall, H. M. Ross, G. M. Moon, and A. D. Ruthven. 1983. Lead poisoning in captive birds of prey. *Vet. Rec.* 113:65–66.

Maedgen, J. L., C. S. Hacker, G. D. Schroder, and F. W. Weir. 1982. Bioaccumulation of lead and cadmium in the royal tern and sandwich tern. *Arch. Environ. Contam. Toxicol.* 11:99–102.

Marn, C. M., R. E. Mirarchi, and M. E. Lisano. 1988. Effects of diet and cold-exposure on captive female mourning doves dosed with lead shot. *Arch. Environ. Contam. Toxicol.* 17:589–594.

Martin, P. A., D. Campbell, K. Hughes, and T. McDaniel. 2008. Lead in the tissues of terrestrial raptors in southern Ontario, Canada, 1995–2001. *Sci. Total Environ.* 391:96–103.

Martínez-López, E., et al. 2004. Lead in feathers and delta-aminolevulinic acid dehydratase activity in three raptor species from an unpolluted Mediterranean forest (southeastern Spain). *Arch. Environ. Contam. Toxicol.* 47:270–275.

Mateo, R. 2009. Lead poisoning in wild birds in Europe and the regulations adopted by different countries. In *Ingestion of lead from spent ammunition: implications for wildlife and humans*, eds. R. T. Watson, M. Fuller, M. Pokras, and W. G. Hunt. Boise: The Peregrine Fund. DOI 10.4080/ilsa.2009.0107.

Mateo, R., W. N. Beyer, J. W. Spann, D. J. Hoffman, and A. Ramis. 2003. Relationship between oxidative stress, pathology, and behavioral signs of lead poisoning in mallards. *J. Toxicol. Environ. Health Part A* 66:1371–1389.

Mateo, R., J. C. Dolz, J. M. Aguilar Serrano, J. Belliure, and R. Guitart. 1997a. An epizootic of lead poisoning in greater flamingos (*Phoenicopterus ruber roseus*) in Spain. *J. Wildl. Dis.* 33:131–134.

Mateo, R, A. J. Green, C. W. Jeske, V. Urios, and C. Gerique. 2001. Lead poisoning in the globally threatened marbled teal and white-headed duck in Spain. *Environ. Toxicol. Chem.* 20:2860–2868.

Mateo, R., A. J. Green, H. Lefranc, R. Baos, and J. Figuerola. 2007. Lead poisoning in wild birds from southern Spain: a comparative study of wetland areas and species affected, and trends over time. *Ecotoxicol. Environ. Saf.* 66:119–126.

Mateo, R., R. Guitart, and A. J. Green. 2000. Determinants of lead shot, rice and grit ingestion in ducks and coots. *J. Wildl. Manage.* 64:939–947.

Mateo, R., R. Molina, J. Grífols, and R. Guitart. 1997b. Lead poisoning in a free ranging griffon vulture (*Gyps fulvus*). *Vet. Rec.* 140:47–48.

Mateo, R., et al. 1999. Lead shot ingestion by marsh harriers Circus aeruginosus from the Ebro delta, Spain. *Environ. Pollut.* 104:435–440.

Mautino, M., and J. U. Bell. 1986. Experimental lead toxicity in ring-necked duck. *Environ. Res.* 41:538–545.

Mautino, M., and J. U. Bell. 1987. Hematological evaluation of lead intoxication in mallards. *Bull. Environ. Contam. Toxicol.* 38:78–85.

McConnell, C. A. 1967. Experimental lead poisoning of bobwhite quail and mourning doves, pp. 208–219. New Orleans: Proc. 21st Annu. Conf. Southeast. Assoc. Game Fish Comm.

Meharg, A. A., et al. 2002. Isotopic identification of the sources of lead contamination for white storks (*Ciconia ciconia*) in a marshland ecosystem (Doñana, S. W. Spain). *Sci. Total Environ.* 300: 81–86.

Meretsky, V. J., N. F. R. Snyder, S. R. Beissinger, D. A. Clendenen, and J. W. Wiley. 2000. Demography of the California condor: implications for reestablishment. *Conserv. Biol.* 14:957–967.

Moore, K. C. 1978. Investigations of lead poisoning in waterfowl in California. In *Trans. West. Sec. Wildl. Soc. Annu. Mtg.* 209–220.

Morgan, G. W., F. W. Edens, P. Thaxton, and C. R. Parkhurst. 1975. Toxicity of dietary lead in Japanese quail. *Poult. Sci.* 54:1636–1642.

Mörner, T., and L. Petersson. 1999. Lead poisoning in woodpeckers in Sweden. *J. Wildl. Dis.* 35:763–765.

Morton, A. P., S. Partridge, and J. A. Blair. 1985. The intestinal uptake of lead. *Chem. Br.* Oct:923–927.

Mulhern, B. M., et al. 1970. Organochlorine residues and autopsy data from bald eagles 1966-68. *Pestic. Monit. J.* 4:141–144.

Munoz, R. V., Jr., C. S. Hacker, and T. F. Gesell. 1976. Environmentally acquired lead in the laughing gull, *Larus atricilla*. *J. Wildl. Dis.* 12:139–142.

Nakade, T., et al. 2005. Lead poisoning in whooper and tundra swans. *J. Wildl. Dis.* 41:253–256.

Nam, D. H., and D. P. Lee. 2005. Possible routes for lead accumulation in feral pigeons (*Columba livia*). *Environ. Monit. Assess.* 121:355–361.

Nelson, T. A., C. Mitchell, and C. Abbott. 1989. Lead-shot ingestion by bald eagles in western Arkansas. *Southwest. Nat.* 34:245–249.

Ochiai, K., et al. 1992. Pathological study of lead-poisoning in whooper swans (*Cygnus cygnus*) in Japan. *Avian Dis.* 36:313–323.

O'Halloran, J., P. F. Duggan, and A. A. Myers. 1988a. Biochemical and haematological values for mute swans (*Cygnus olor*): effects of acute lead poisoning. *Avian Pathol.* 17:667–678.

O'Halloran, J., A. A. Myers, and P. F. Duggan. 1988b. Blood lead levels and free red blood cell protoporphyrin as a measure of lead exposure in mute swans. *Environ. Pollut.* 52:19–38.

Ohi, G., H. Seki, K. Akiyama, and H. Yagyu. 1974. The pigeon, a sensor of lead pollution. *Bull. Environ. Contam. Toxicol.* 12:92–98.

Ohi, G., H. Seki, K. Minowa, M. Ohsawa, I. Mizoguchi, and F. Sugimori. 1981. Lead pollution in Tokyo—the pigeon reflects its amelioration. *Environ. Res.* 26:125–129.

Osborn, D., W. J. Every, and K. R. Bull. 1983. The toxicity of trialkyl lead compounds to birds. *Environ. Pollut. Ser. A* 31:261–275.

Pain, D. J. 1987. Lead poisoning in waterfowl: an investigation of sources and screening techniques. Ph.D. Thesis. Oxford Univ. Oxford, UK.

Pain, D. J. 1989. Haematological parameters as predictors of blood lead and indicators of lead poisoning in the black duck (*Anas rubripes*). *Environ. Pollut.* 60:67–81.

Pain, D. J. 1990. Lead shot ingestion by waterbirds in the Camargue, France: an investigation of levels and interspecific differences. *Environ. Pollut.* 66:273–285.

Pain, D. J. 1992. Lead poisoning of water fowl: a review. In *Lead poisoning in waterfowl*, ed. D. J. Pain, pp. 7–13. IWRB Spec. Publ. 16. Slimbridge: International Waterfowl and Wetlands Research Bureau.

Pain, D. J., and B. A. Rattner. 1988. Mortality and hematology associated with the ingestion of one number four lead shot in black ducks, *Anas rubripes*. *Bull. Environ. Contam. Toxicol.* 40:159–164.

Pain, D. J., C. Amiard-Triquet, and C. Sylvestre. 1992. Tissue lead concentrations and shot ingestion in nine species of waterbird from the Camargue (France). *Ecotoxicol. Environ. Saf.* 24:217–233.

Pain, D. J., C. Bavoux, and G. Burneleau. 1997. Seasonal blood lead concentrations in marsh harriers *Circus aeruginosus* from Charente-Maritime, France: relationship with the hunting season. *Biol. Conserv.* 81:1–7.

Pain, D. J., A. A. Meharg, M. Ferrer, M. Taggart, and V. Penteriani. 2005. Lead concentrations in bones and feathers of the globally threatened Spanish imperial eagle. *Biol. Conserv.* 121:603–610.

Pain, D. J., et al. 2007. Lead contamination and associated disease in captive and reintroduced red kites *Milvus milvus* in England. *Sci. Total Environ.* 376:116–127.

Pain, D. J., I. J. Fisher, and V. G. Thomas. 2009. A global update of lead poisoning in terrestrial birds from ammunition sources. In *Ingestion of lead from spent ammunition: implications for wildlife and humans*, eds. R. T. Watson, M. Fuller, M. Pokras, and W. G. Hunt. Boise: The Peregrine Fund. DOI 10.4080/ilsa.2009.0108.

Parish, C. N., W. R. Heinrich, and W. G. Hunt. 2007. Lead exposure, diagnosis, and treatment in California condors released in Arizona. In *California condors in the 21st century*. Series in ornithology, no. 2, eds. A. Mee and L. S. Hall, pp. 97–108. Cambridge, MA: Nuttall Ornithology Club and Washington, D.C.: The American Ornithologists' Union.

Pattee, O. H. 1984. Eggshell thickness and reproduction in American kestrels exposed to chronic dietary lead. *Arch. Environ. Contam. Toxicol.* 13:29–34.

Pattee, O. H., S. N. Wiemeyer, B. M. Mulhern, L. Sileo, and J. W. Carpenter. 1981. Experimental lead-shot poisoning in bald eagles. *J. Wildl. Manage.* 45:806–810.

Pattee, O. H., P. H. Bloom, J. M. Scott, and M. R. Smith. 1990. Lead hazards within the range of the California condor. *Condor* 92:931–937.

Pattee, O. H., and D. J. Pain. 2003. Lead in the Environment. In *Handbook of ecotoxicology*, 2nd ed., eds. D. J. Hoffman, B. A. Rattner, G. A. Burton, Jr., and J. Cairns, Jr., pp. 373–408. Boca Raton: Lewis Publishers.

Pattee, O. H., et al. 2006. Lead poisoning in captive Andean condors (*Vultur gryphus*). *J. Wildl. Dis.* 42:772–779.

Platt, J. B. 1976. Bald eagles wintering in a Utah desert. *Am. Birds* 30:783–788.

Pokras, M. A., and R. Chafel. 1992. Lead toxicosis from ingested fishing sinkers in adult common loons (*Gavia immer*) in New England. *J. Zoo Wildl. Med.* 23:92–97.

Pokras, M. A., M. R. Kneeland, A. Major, R. Miconi, and R. H. Poppenga. 2009. Lead objects ingested by common loons in New England. Extended abstract. In *Ingestion of lead from spent ammunition: implications for wildlife and humans*, eds. R. T. Watson, M. Fuller, M. Pokras, and W. G. Hunt. Boise: The Peregrine Fund. DOI 10.4080/ilsa.2009.0116.

Ramo, C., C. Sánchez, and L. H. Saint-Aubin. 1992. Lead poisoning of greater flamingos *Phoenicopterus ruber*. *Wildfowl* 43:220–222.

Redig, P. T., C. M. Stowe, D. M. Barnes, and T. D. Arent. 1980. Lead toxicosis in raptors. *J. Am. Vet. Med. Assoc.* 177:941–943.

Redig, P. T., E. M. Lawler, S. Schwartz, J. L. Dunnette, B. Stephenson, and G. E. Duke. 1991. Effects of chronic exposure to sublethal concentrations of lead acetate on heme synthesis and immune function in red-tailed hawks. *Arch. Environ. Contam. Toxicol.* 21:72–77.

Reichel, W. L., et al. 1984. Pesticide, PCB, and lead residues and necropsy data for bald eagles from 32 states—1978-81. *Environ. Monit. Assess.* 4:395–403.

Reiser, M. H., and S. A. Temple. 1981. Effects of chronic lead ingestion on birds of prey. In *Recent advances in the study of raptor diseases*, eds. J. E. Cooper and A. G. Greenwood, pp. 21–25. West Yorkshire: Chiron Publications, Ltd.

Rocke, T. E., and M. D. Samuel. 1991. Effects of lead shot ingestion on selected cells of the mallard immune system. *J. Wildl. Dis.* 27:1–9.

Roscoe, D. E., S. W. Nielsen, A. A. Lamola, and D. Zuckerman. 1979. A simple quantitative test for erythrocytic protoporphyrin in lead-poisoned ducks. *J. Wildl. Dis.* 15:127–136.

Roux, K. E., and P. P. Marra. 2007. The presence and impact of environmental lead in passerine birds along an urban to rural land use gradient. *Arch. Environ. Contam. Toxicol.* 53:261–268.

Salisbury, R. M., E. L. J. Staples, and M. Sutton. 1958. Lead poisoning of chickens. *N. Z. Vet. J.* 6:2–7.

Sanderson, G. C. 1992. Lead poisoning mortality. In *Lead poisoning in waterfowl*, ed. D. J. Pain, pp. 14–18. IWRB Spec. Publ. 16. Slimbridge: International Waterfowl and Wetlands Research Bureau.

Sanderson, G. C., and F. C. Bellrose. 1986. A review of the problem of lead poisoning in waterfowl. *Ill. Nat. Hist. Surv. Spec. Publ.* No. 4. Champaign, IL: Illinois Natural History Survey.

Sanderson, G. C., and J. C. Irwin. 1976. Effects of various combinations and numbers of lead:iron pellets dosed in wild-type captive mallards. Final Report Contract No. 14-16-0008-914. U. S. Fish and Wildlife Service and Illinois Natural History Survey.

Sassa, S., S. Granick, and A. Kappas. 1975. Effect of lead and genetic factors on heme biosynthesis in the human red cell. *Ann. N. Y. Acad. Sci.* 244:419–440.

Scanlon, P. F. 1982. Wet and dry weight relationships of mallard (*Anas platyrhynchos*) tissues. *Bull. Environ. Contam. Toxicol.* 29:615–617.

Scheifler, R., et al. 2006. Lead concentrations in feathers and blood of common blackbirds (*Turdus merula*) and in earthworms inhabiting unpolluted and moderately polluted urban areas. *Sci. Total Environ.* 371:197–205.

Scheuhammer, A. M. 1989. Monitoring wild bird populations for lead exposure. *J. Wildl. Manage.* 53:759–765.

Scheuhammer, A. M. 1996. Influence of reduced dietary calcium on the accumulation and effects of lead, cadmium, and aluminum in birds. *Environ. Pollut.* 94:337–343.

Scheuhammer, A. M., and K. M. Dickson. 1996. Patterns of environmental lead exposure in waterfowl in eastern Canada. *Ambio* 25:14–20.

Scheuhammer, A. M., and D. M. Templeton. 1998. Use of stable isotope ratios to distinguish sources of lead exposure in wild birds. *Ecotoxicology* 7:37–42.

Scheuhammer, A. M., and L. K. Wilson. 1990. Effects of lead and pesticides on delta-aminolevulinic acid dehydratase of ring doves (*Streptopelia risoria*). *Environ. Toxicol. Chem.* 9:1379–1386.

Scheuhammer, A. M., C. A. Rogers, and D. Bond. 1999. Elevated lead exposure in American woodcock (*Scolopax minor*) in eastern Canada. *Arch. Environ. Contam. Toxicol.* 36:334–340.

Scheuhammer, A. M., D. E. Bond, N. M. Burgess, and J. Rodrigue. 2003a. Lead and stable lead isotype ratios in soil, earthworms, and bones of American woodcock (*Scolopax minor*) from eastern Canada. *Environ. Toxicol. Chem.* 22:2585–2591.

Scheuhammer, A. M., S. L. Money, D. A. Kirk, and G. Donaldson. 2003b. Lead fishing sinkers and jigs in Canada: review of their use patterns and toxic impacts on wildlife. Occasional Paper #108. Ottawa: The Canadian Wildlife Service.

Schmitz, R. A., A. A. Aguirre, R. S. Cook, and G. A. Baldassarre. 1990. Lead poisoning of Caribbean flamingos in Yucatan, Mexico. *Wildl. Soc. Bull.* 18:399–404.

Schulz, J. H., et al. 2006. Acute lead toxicosis in mourning doves. *J. Wildl. Manage.* 70:413–421.

Schulz, J. H., X. Gao, J. J. Millspaugh, and A. J. Bermudez. 2007. Experimental lead pellet ingestion in mourning doves (*Zenaida macroura*) *Am. Midl. Nat.* 158:177–190.

Sears, J. 1988. Regional and seasonal variations in lead poisoning in the mute swan *Cygnus olor* in relation to the distribution of lead and lead weights, in the Thames area, England. *Biol. Conserv.* 46:115–134.

Sidor, I. F., M. A. Pokras, A. R. Major, R. H. Poppenga, K. M. Taylor, and R. M. Miconi. 2003. Mortality of common loons in New England, 1987 to 2000. *J. Wildl. Dis.* 39:306–315.

Sikarskie, J. 1977. The case of the red-tailed hawk. *Intervet* 8:4.

Sileo, L., and S. I. Fefer. 1987. Paint chip poisoning of Laysan albatross at Midway Atoll. *J. Wildl. Dis.* 23:432–437.

Sileo, L., et al. 2001. Lead poisoning of waterfowl by contaminated sediment in the Coeur d'Alene River. *Arch. Environ. Contam. Toxicol.* 41:364–368.

Simkiss, K. 1961. Calcium metabolism and avian reproduction. *Biol. Rev.* 36:321–367.

Snoeijs, T., T. Dauwe, R. Pinxten, V. M. Darras, L. Arckens, and M. Eens. 2005. The combined effect of lead exposure and high or low dietary calcium on health and immunocompetence in the zebra finch (*Taeniopygia guttata*). *Environ. Pollut.* 134:123–132.

Snyder, N. F. R. 2007. Limiting factors for wild California condors. In *California condors in the 21st century.* Series in ornithology, no. 2, eds. A. Mee, and L. S. Hall, pp. 9–33. Cambridge, MA: Nuttall Ornithological Club and Washington, D.C.: The American Ornithologists' Union.

Snyder, S. B., M. J. Richard, R. C. Drewien, N. Thomas, and J. P. Thilsted. 1991. Diseases of whooping cranes seen during annual migration of the Rocky Mountain flock. In *1991 Proc. Am. Assoc. Zoo Vet.*, ed. R. E. Junge, pp. 74–80. Cambridge, MA: The Nutthall Ornithological Club and Washington, D.C.: The American Orithologists' Union.

Sorenson, K. J., and L. J. Burnett. 2007. Lead concentrations in the blood of Big Sur California condors. In *California condors in the 21st century*. Series in ornithology, no. 2, eds. A. Mee and L. S. Hall, pp. 185–195. Cambridge, MA: Nuttall Ornithological Club and Washington, D.C.: The American Ornithologists' Union.

Spray, C. J., and H. Milne. 1988. The incidence of lead poisoning among whooper and mute swans *Cygnus cygnus* and *C. olor* in Scotland. *Biol. Conserv.* 44:265–281.

Stendell, R. C., R. I. Smith, K. P. Burnham, and R. E. Christensen. 1979. Exposure of waterfowl to lead: a nationwide survey of residues in wing bones of seven species, 1972–73. *U.S. Fish Wildl. Serv. Spec. Sci. Rep. Wildl.* No. 223. Washington, D.C.: U.S. Department of the Interior.

Stendell, R. C., J. W. Artmann, and E. Martin. 1980. Lead residues in sora rails from Maryland. *J. Wildl. Manage.* 44:525–527.

Stone, W. B., and S. A. Butkas. 1978. Lead poisoning in a wild turkey. *N. Y. Fish Game J.* 25:169.

Stone, C. L., and J. H. Soares, Jr. 1976. The effect of dietary selenium level on lead toxicity in the Japanese quail. *Poult. Sci.* 55:341–349.

Stone, C. L., M. R. S. Fox, A. L. Jones, and K. R. Mahaffey. 1977. Delta-aminolevulinic acid dehydratase—a sensitive indicator of lead exposure in Japanese quail. *Poult. Sci.* 56:174–181.

Stone, C. L., K. R. Mahaffey, and M. R. S. Fox. 1979. A rapid bioassay system for lead using young Japanese quail. *J. Environ. Pathol. Toxicol.* 2:767–779.

Stone, W. B., and J. C. Okoniewski. 2001. Necropsy findings and environmental contaminants in common loons from New York. *J. Wildl. Dis.* 37:178–184.

Strom, S. M., K. A. Patnode, J. A. Langenberg, B. L. Bodenstein, and A. M. Scheuhammer. 2005. Lead contamination in American woodcock (*Scolopax minor*) from Wisconsin. *Arch. Environ. Contam. Toxicol.* 49:396–402.

Sullivan, K., R. Sieg, and C. Parish. 2007. Arizona's efforts to reduce lead exposure in California condors. In *California condors in the 21st century*. Series in ornithology, no. 2, eds. A. Mee and L. S. Hall, pp. 109–121. Cambridge, MA: Nuttall Ornithology Club and Washington, D.C.: The American Ornithologists' Union.

Szymczak, M. R., and W. J. Adrian. 1978. Lead poisoning in Canada geese in southwest Colorado. *J. Wildl. Manage.* 42:299–306.

Teraoka, H., et al. 2007. Heavy metal contamination status of Japanese cranes (*Grus japonensis*) in east Hokkaido, Japan—extensive mercury pollution. *Environ. Toxicol. Chem.* 26:307–312.

Tola, S., S. Hernberg, S. Asp, and J. Nikkanen. 1973. Parameters indicative of absorption and biological effect in new lead exposure: a prospective study. *Br. J. Ind. Med.* 30:134–141.

Trust, K. A., M. W. Miller, J. K. Ringelman, and I. M. Orme. 1990. Effects of ingested lead on antibody production in mallards (*Anas platyrhnchos*). *J. Wildl. Dis.* 26:316–322.

Vyas, N. B., J. W. Spann, G. H. Heinz, W. N. Beyer, J. A. Jaquette, and J. M. Mengelkoch. 2000. Lead poisoning of passerines at a trap and skeet range. *Environ. Pollut.* 107:159–166.

Vyas, N. B., J. W. Spann, and G. H. Heinz. 2001. Lead shot toxicity to passerines. *Environ. Pollut.* 111:135–138.

Wayland, M., and T. Bollinger. 1999. Lead exposure and poisoning in bald eagles and golden eagles in the Canadian prairie provinces. *Environ. Pollut.* 104:341–350.

White, D. H., and R. C. Stendell. 1977. Waterfowl exposure to lead and steel shot on selected hunting areas. *J. Wildl. Manage.* 41:469–475.

Wiemeyer, S. N., J. M. Scott, M. P. Anderson, P. H. Bloom, and C. J. Stafford. 1988. Environmental contaminants in California condors. *J. Wildl. Manage.* 52:238–247.

Wilson, H. M., J. L. Oyen, and L. Sileo. 2004. Lead shot poisoning of a pacific loon in Alaska. *J. Wildl. Dis.* 40:600–602.

Wilson, H. M., P. L. Flint, and A. N. Powell. 2007. Coupling contaminants with demography: effects of lead and selenium in pacific common eiders. *Environ. Toxicol. Chem.* 26:1410–1417.

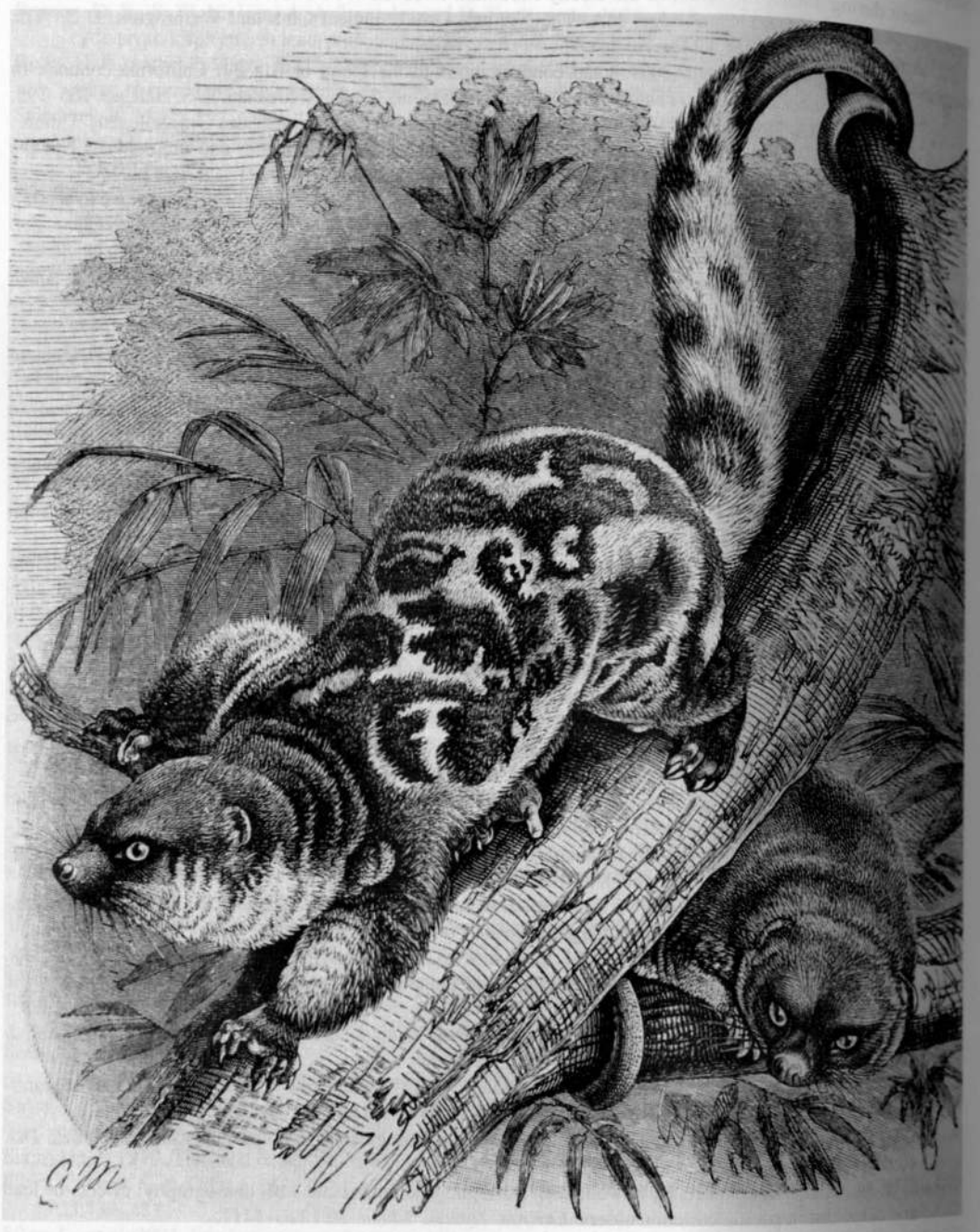
Windingstad, R. M., S. M. Kerr, L. N. Locke, and J. J. Hurt. 1984. Lead poisoning of sandhill cranes (*Grus canadensis*). *Prairie Nat.* 16:21–24.

Wobeser, G. A. 1997. *Diseases of wild waterfowl*, 2nd ed. New York: Plenum Press.

Work, T. M., and M. R. Smith. 1996. Lead exposure in Laysan albatross adults and chicks in Hawaii: prevalence, risk factors, and biochemical effects. 1996. *Arch. Environ. Contam. Toxicol.* 31:115–119.

Yamamoto, K., M. Hayashi, M. Yoshimura, H. Hayashi, A. Hiratsuka, and Y. Isii. 1993. The prevalence and retention of lead pellets in Japanese quail. *Arch. Environ. Contam. Toxicol.* 24:478–482.

Zwank, P. J., V. L. Wright, P. M. Shealy, and J. D. Newsom. 1985. Lead toxicosis in waterfowl on two major wintering areas in Louisiana. *Wildl. Soc. Bull.* 13:17–26.



Spotted Cuscus

By G. Mutzel, from *The Royal Natural History*, edited by Richard Lydekker, Frederick Warne & Co., London, 1893–94.