

## A GLOBAL UPDATE OF LEAD POISONING IN TERRESTRIAL BIRDS FROM AMMUNITION SOURCES

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**ABSTRACT.**—Lead poisoning mortality, through the ingestion of spent shot, is long established in waterfowl, and more recently in raptors and other avian taxa. Raptors (vultures, hawks, falcons, eagles and owls) are exposed to lead from spent ammunition (shot, bullets, or fragments from either) while feeding on game species, and other avian taxa are exposed when feeding in shot-over areas, including shooting ranges. Here we review the published literature on ingestion of and poisoning by lead from ammunition in terrestrial birds. We briefly discuss methods of evaluating exposure to and poisoning from ammunition sources of lead, and the use of lead isotopes for confirming the source of lead. Documented cases include 33 raptor species and 30 species from *Gruiformes*, *Galliformes* and various other avian taxa, including ten Globally Threatened or Near Threatened species. Lead poisoning is of particular conservation concern in long-lived slow breeding species, especially those with initially small populations such as the five Globally Threatened and one Near Threatened raptor species reported as poisoned by lead ammunition in the wild. Lead poisoning in raptors and other terrestrial species will not be eliminated until all lead gunshot and rifle bullets are replaced by non-toxic alternatives. *Received 29 May 2008, accepted 24 July 2008.*

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### TERRESTRIAL BIRDS AT RISK

LEAD POISONING IN WILDFOWL AND WADERS from the ingestion of spent lead gunshot has been extensively studied, documented and reviewed over the last half century (Bellrose 1959, Kaiser and Fry 1980, Hall and Fisher 1985, Veiga 1985, Pain 1990a, 1990b, 1991a, 1992, 1996, Locke and Friend 1992, Sharley et al. 1992, Scheuhammer and

Norris 1995, Beck and Granval 1997). Poisoning from the ingestion of lead from both gunshot and bullets by terrestrial birds initially received less attention, but today a considerable and increasing literature exists. While ingestion of discharged lead from firearms has been recorded in a diverse range of terrestrial birds, two groups of birds are particularly susceptible.

One group includes birds that are exposed in a similar way to wildfowl and waders, while feeding in any shot over areas where spent lead is deposited. Such areas include upland and lowland game shooting areas, shooting ranges including clay-pigeon shoots, and areas where species such as squirrels and other rodents are shot as pests. Exposure is likely to be higher in species that, like wildfowl, deliberately ingest grit to help break down food and/or ingest food items similar in appearance to lead shot (such as seeds) and may therefore mistakenly ingest lead. Many species fall into this category, but species are predominantly *Gruiformes* and *Galliformes* (Table 1). The second group at risk comprise species that ingest lead from bullets or shot while preying upon or scavenging game or other species of mammals or birds (such as 'pest' species, e.g., Knopper et al. 2006) that have been shot but survived, or killed but unretrieved. Species at risk are predominantly raptors, herein defined as vultures (New and Old World), hawks, falcons, eagles and owls (Table 1). Here, risk of exposure is likely to be particularly high in species that feed more frequently on hunter-shot quarry and have a propensity to scavenge. Species are also more likely to be exposed if there is intensive hunting within their foraging range (e.g. Mateo et al. 1998a, Wayland and Bollinger 1999) and during the hunting season (Elliott et al. 1992, Pain et al. 1997).

One of the earliest reports of lead poisoning from lead shot ingestion in a non-wildfowl species involved a Ring-necked Pheasant (*Phasianus colchicus*) in the UK (Calvert 1876), and a recent study from British shooting estates found an overall shot ingestion rate of 3%, with correspondingly elevated bone lead levels (Butler et al. 2005). Evidence for lead poisoning in this species therefore spans 125 years. Species of *Galliformes* and *Gruiformes* have been reported to have ingested shot and/or suffered lead poisoning through exposure to lead shot in a wide range of countries across Europe, and North America (Table 1).

In areas where lead ammunition is used, all raptors that feed on game or other hunted species are potentially at risk from lead poisoning. Species that take live prey, such as the Goshawk (*Accipiter gentilis*), are exposed through ingesting animals that have been shot but survived carrying shot in their

flesh. In some cases exposure rates can be high, e.g., 25% and 36% of first year and adult live-trapped Pink-footed Geese (*Anser brachyrhynchus*) carried shot (Noer and Madsen 1996). However, species that scavenge, or both hunt and scavenge, are likely to be at particular risk from lead ingestion through feeding on injured or hunter-killed but unretrieved prey. Season and local hunting intensity play an important part in determining the level of dietary lead exposure (Pain et al. 1993, Mateo 1998a). For example, of Bald Eagles (*Haliaeetus leucocephalus*) found sick, injured or dead with significantly elevated lead exposure in British Columbia, the greatest number were found between January and March when they were feeding heavily on wintering waterfowl (Elliott et al. 1992). The prevalence of Bald Eagles with elevated lead exposure was found to be higher in areas of high waterfowl hunting intensity than areas with low hunting intensity (Wayland and Bollinger 1999). Similarly, Pain et al. (1997) found a significantly higher prevalence of elevated blood lead concentrations in Marsh Harriers (*Circus aeruginosus*) in France during than outside the hunting season.

Raptors are at risk both from prey killed with shotguns, such as wildfowl, rabbits and rodents, and with rifles, such as deer and other large game. The prevalence of exposure to lead from bullets is likely to be higher in larger raptors, and poisoning from bullet fragments has been reported in wild Bald Eagles, Golden Eagles (*Aquila chrysaetos*), Steller's Sea Eagles (*Haliaeetus pelagicus*), White-tailed Eagles (*H. albicilla*), and California Condors (*Gymnogyps californianus*) (Table 1).

Both captive and supplementary-fed wild birds may also be at risk if food includes animals that have been killed with lead ammunition, even when shot and bullets have been removed (e.g., Pain et al. 2007). Until recently, the extent to which lead shot and bullets fragment while passing through the tissues of prey was not realised. However, radiographs have shown that both shot and bullets undergo considerable fragmentation and fragments may be both distant from the wound canal, and too small to be visually detected (e.g., Pain et al. 2007). Many authors have now shown that feeding on species killed by lead gunshot or bullets can present a significant risk to scavengers, whether or not they

are exposed to whole shot or bullets (Scheuhammer et al. 1998, Sergeyeve and Shulyatieva 2005, Hunt et al. 2006, Knopper et al. 2006, Pain et al. 2007). For example, Scheuhammer et al. (1998) found that 11% (92 of 827) of pectoral muscle pools from hunter-killed game (predominantly waterfowl) had elevated lead concentrations, in excess of  $0.5 \mu\text{g g}^{-1}$  ww (mean of  $12 \pm 38 \mu\text{g g}^{-1}$  ww). While no lead could be detected visibly in the samples, radiography showed tiny embedded metal fragments.

#### EXPOSURE TO LEAD GUNSHOT AND BULLETS

Lead exposure is usually measured through the presence of lead shot or lead fragments in pellets regurgitated by raptors (e.g., Platt 1976, Pain et al. 1997, 2007), or the presence of shot, bullets or fragments in the alimentary tracts of birds post-mortem (e.g. Church et al. 2006 and numerous authors in Table 1).

The regurgitation of ingested shot in pellets illustrates that not all exposure to lead need be fatal, and regurgitation will undoubtedly reduce the amount of lead absorbed and the level of poisoning in raptors. However, repeated exposure and regurgitation of lead shot can result in poisoning (Pattee et al. 1981). In areas of high exposure, the likelihood of picking up shot (and of repeated exposure) increases. For example, as Bald Eagles and their waterfowl prey become concentrated in smaller areas, the likelihood of eagles picking up shot increases (Pattee and Hennes 1983). Consequently, exposure measured through the presence of shot in regurgitated pellets is likely to give a good indication of the likelihood of a species to become poisoned. Pain et al. (1997) found that the incidence of shot in regurgitated Marsh Harrier pellets dropped from up to 25% during the hunting season to just 1.4% after, in parallel with a significant reduction in blood lead concentrations in the birds outside the hunting season. Other studies have similarly found both the presence of lead shot in regurgitated pellets and evidence of poisoning and mortality from lead in wild birds (e.g., Red Kites [*Milvus milvus*] in the UK, Pain et al. 2007).

The presence of lead particles in the alimentary tracts of birds at post mortem is a widely used indicator of exposure, and both the number of lead shot

or fragments found and the degree of abrasion or dissolution may provide supplementary evidence to help in the diagnosis of lead poisoning. Occasionally, large numbers of shot are ingested, and in Spain, where Golden Eagles have been found with lead toxicosis from shot ingestion, one bird had 40 pellets in its proventriculus (Cerradello et al. 1992).

It is possible that birds that have not ingested lead from ammunition but carry shot-in lead in their tissues may also have elevated blood lead concentrations, as has been found in humans that have been shot with lead bullets (McQuirter et al. 2004), although these are likely to be far lower than in birds that have ingested lead fragments.

#### EVALUATING LEAD POISONING

Lead is not an essential element, and is a non-specific poison affecting all body systems. Lead poisoned birds often exhibit a distended proventriculus, green watery faeces, weight loss, anaemia and drooping posture, among other signs (Redig et al. 1980, Reiser and Temple 1981, Franson et al. 1983, Custer et al. 1984, Sanderson and Bellrose 1986, Friend 1987, Mateo 1998b). Lead affects the nervous system, kidneys and the circulatory system, resulting in a range of sub-lethal physiological, biochemical and behavioural changes (Scheuhammer 1987). There is no true 'no-effect' threshold level for lead; for example even at extremely low concentrations lead depresses the activity of the blood enzyme delta aminolevulinic acid, essential for haemoglobin production (Grasman and Scanlon 1995, Redig et al. 1991).

Once ingested, lead ammunition or fragments are readily dissolved in the acidic conditions in the intestine, and in birds that ingest grit to aid digestion (e.g., some *Gruiformes* and *Galliformes*) abrasion of the lead may accelerate this process. Directly following absorption the highest lead concentrations tend to be found in the blood, from which lead is transported around the body and deposited in soft tissue and bone. Blood lead generally has a half-life of around two weeks in birds (e.g., 14 days in the California Condor, Fry et al. 2009 this volume), whereas liver and kidney tissues generally retain elevated lead concentrations for weeks to several

**Table 1.** Lead shot ingestion and poisoning.

<b>Species</b>	<b>Status</b>	<b>Evidence</b>	<b>Countries</b>	<b>References</b>
Common Raven ( <i>Corvus corax</i> )	LC	Poisoning	USA	Craighead and Bedrosian 2008
Chukar ( <i>Alectoris chukar</i> )	LC	Ingestion	USA	Hanspeter and Kerry 2003
Grey Partridge ( <i>Perdix perdix</i> )	LC	Ingestion and Poisoning	Denmark, UK	Clausen and Wolstrup 1979, Keymer and Stebbings 1987, Potts, 2005
Common Pheasant ( <i>Phasianus colchicus</i> )	LC	Ingestion and Poisoning	Denmark, UK, USA	Calvert 1876, Elder 1955, Clausen and Wolstrup 1979, NWHL 1985, Dutton and Bolen 2000, Butler et al. 2005.
Wild Turkey ( <i>Meleagris gallopavo</i> )	LC	Ingestion	USA	Stone and Butkas 1978
Scaled Quail ( <i>Callipepla squamata</i> )	LC	Ingestion	USA	Campbell 1950
Northern Bobwhite Quail ( <i>Colinus virginianus</i> )	NT	Ingestion	USA	Stoddard 1931, Keel et al. 2002
Great Horned Owl ( <i>Bubo virginianus</i> )	LC	Poisoning	Canada	Clark and Scheuhammer 2003
Eurasian Eagle Owl ( <i>B. bubo</i> )	LC	Poisoning	Spain	Mateo et al. 2003
Snowy Owl ( <i>Nyctea scandiaca</i> )	LC	Poisoning	Captive	MacDonald et al. 1983
Long-eared Owl ( <i>Asio otus</i> )	LC	Poisoning	Spain	Brinzal 1996
Rock Pigeon ( <i>Columba livia</i> )	LC	Ingestion	USA , Belgium	Dement et al. 1987, Tavernier et al. 2004
Common Wood-pigeon ( <i>C. palumbus</i> )	LC	Poisoning	Denmark	Clausen and Wolstrup 1979
Mourning Dove ( <i>Zenaida macroura</i> )	LC	Ingestion	USA	Locke and Bagley 1967, Lewis and Legler 1968, Best et al. 1992, Schulz et al. 2002
Sandhill Crane ( <i>Grus canadensis</i> )	LC	Ingestion	USA	Windingstad et al. 1984, NWHL 1985
Whooping Crane ( <i>G. americana</i> )	EN	Poisoning	USA	Hall and Fisher 1985
Clapper Rail ( <i>Rallus longirostris</i> )	LC	Ingestion	USA	Jones 1939
King Rail ( <i>R. elegans</i> )	LC	Ingestion	USA	Jones 1939
Virginia Rail ( <i>R. limicola</i> )	LC	Ingestion	USA	Jones 1939
Sora ( <i>Porzana carolina</i> )	LC	Poisoning	USA	Jones 1939, Artman and Martin 1975, Stendell et al. 1980
Common Moorhen ( <i>Gallinula chloropus</i> )	LC	Ingestion	Europe, USA	Jones 1939, Locke and Friend 1992
Common Coot ( <i>Fulica atra</i> )	LC	Ingestion	France	Pain 1990a
American Coot ( <i>F. americana</i> )	LC	Ingestion	USA	Jones 1939

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<b>Species</b>	<b>Status</b>	<b>Evidence</b>	<b>Countries</b>	<b>References</b>
American Woodcock ( <i>Scolopax minor</i> )	LC	Ingestion	Canada	Scheuhammer et al. 1999, 2003
Ruffed Grouse ( <i>Bonasa umbellus</i> )		Ingestion	Canada	Rodrigue et al. 2005
California Gull ( <i>Larus californicus</i> )	LC	Ingestion	USA	Quortrup and Shillinger 1941
Glaucous-winged Gull ( <i>L. glaucescens</i> )	LC	Ingestion	USA	NWHL 1985
Herring Gull ( <i>L. argentatus</i> )	LC	Ingestion	USA	NWHL 1985
European Honey-buzzard ( <i>Pernis apivorus</i> )	LC	Unknown (ingestion or shot)	Netherlands	Lumeiji et al. 1985
Red Kite ( <i>Milvus milvus</i> )	LC	Ingestion or poisoning	Germany, Spain, UK, Captive	Mateo 1998a, Mateo et al. 2001, 2003, Pain et al. 1997, Kenntner et al. 2005
Bald Eagle ( <i>Haliaeetus leucocephalus</i> )	LC	Poisoning, shot and bullets	Canada, USA	Platt 1976, Jacobson et al. 1977, Kaiser et al. 1980, Patee and Hennes 1983, Reichel et al. 1984, Frenzel and Anthony 1989, Craig et al. 1990, Langelier et al. 1991, Elliott et al. 1992, Gill and Langelier 1994, Scheuhammer and Norris 1996, Wayland and Bollinger 1999, Miller et al. 2000, 2001, Clark and Scheuhammer 2003, Wayland et al. 2003
Steller's Sea-eagle ( <i>H. pelagicus</i> )	VU	Poisoning, bullets	Japan	Kim et al. 1999, Iwata et al. 2000, Kurosawa 2000
White-rumped Vulture ( <i>Gyps bengalensis</i> )	CR	Poisoning (origin unknown)	Pakistan	Oaks et al. 2004
Eurasian Griffon ( <i>G. fulvus</i> )	LC	Poisoning	Spain	Mateo et al. 1997, Guitart 1998, Mateo et al. 2003, Garcia-Fernandez et al. 2005
Egyptian Vulture ( <i>Neophron percnopterus</i> )	EN	Poisoning	Canary Islands; Iberian peninsula	Donazar et al. 2002, Rodriguez-Ramos et al. 2009, this volume
Cinereous Vulture ( <i>Aegypius monachus</i> )	NT	Poisoning	Spain	Hernandez and Margalida 2008
Eastern Marsh-harrier ( <i>Circus spilonotus</i> )		Ingestion	Japan	Hirano et al. 2004
Western Marsh-harrier ( <i>Circus aeruginosus</i> )	LC	Poisoning	France, Germany, Spain	Pain et al. 1993, 1997, Mateo et al., 1999, Kenntner et al. 2005
Northern Harrier ( <i>C. cyaneus</i> )	LC	Ingestion	Canada, USA	Martin and Barrett 2001, Martin et al. 2003
Eurasian Sparrowhawk ( <i>Accipiter nisus</i> )	LC	Ingestion	France, Captive	MacDonald et al. 1983, Pain and Amiard-Triquet 1993
Sharp-shinned Hawk ( <i>A. striatus</i> )	LC	Ingestion	Canada, USA	Martin and Barrett 2001
Cooper's Hawk ( <i>A. cooperii</i> )	LC	Ingestion	Canada, USA	Snyder et al. 1973, Martin and Barrett 2001
Northern Goshawk ( <i>A. gentilis</i> )	LC	Poisoning	Canada, France, Germany, USA, Captive	Stehle 1980, Pain and Amiard-Triquet 1993, Martin and Barrett 2001, Kenntner et al. 2003, 2005

Species	Status	Evidence	Countries	References
Red-tailed Hawk ( <i>Buteo jamaicensis</i> )	LC	Poisoning	Canada, USA	Franson et al. 1996, Martin and Barrett 2001, Clark and Scheuhammer 2003, Martina et al. 2008
Common Buzzard ( <i>B. buteo</i> )	LC	Poisoning	France, Germany; UK, Italy, Captive	Stehle 1980, MacDonald et al. 1983, Pain and Amiard-Triquet 1993, Pain et al. 1995, Kenntner et al. 2005, Battaglia et al. 2005
Rough-legged Buzzard ( <i>B. lagopus</i> )	LC	Poisoning	USA	Locke and Friend 1992
Spanish Imperial Eagle ( <i>Aquila adalberti</i> )	VU	Poisoning	Spain	González and Hiraldo 1988, Hernández 1995, Mateo 1998a, Mateo et al. 2001, Pain et al. 2004
Golden Eagle ( <i>A. chrysaetos</i> )	LC	Poisoning	Canada, Germany, Spain, Switzerland, USA	Bloom et al. 1989, Craig et al. 1990, Pattee et al. 1990, Cerradello et al. 1992, Bezzel and Fünfstück 1995, Scheuhammer and Norris 1996, Wayland and Bollinger 1999, Clark and Scheuhammer 2003, Wayland et al. 2003, Kenntner et al. 2007
White-tailed Eagle ( <i>Haliaeetus albicilla</i> )	LC	Ingestion and Poisoning – shot and bullets	Greenland, Poland; Austria, Germany, Japan, Finland	Falandysz et al. 1988, Kim et al. 1999, Iwata et al. 2000, Kurosawa 2000, Kenntner et al. 2001, 2004, 2005, Krone et al. 2004, 2006, Kalisinska et al. 2006
American Kestrel ( <i>Falco sparverius</i> )	LC	Ingestion	Canada, USA	Martin and Barrett 2001
Laggar Falcon ( <i>F. jugger</i> )	NT	Poisoning	Captive	MacDonald et al. 1983
Prairie Falcon ( <i>F. mexicanus</i> )	LC	Poisoning	Captive	Benson et al. 1974, Stehle 1980
Peregrine Falcon ( <i>F. peregrinus</i> )	LC	Poisoning	UK, Captive	MacDonald et al. 1983, Pain et al. 1995
Turkey Vulture ( <i>Cathartes aura</i> )	LC	Ingestion	Canada, USA	Wiemeyer et al. 1986, Martin et al. 2003, Martina et al. 2008
California Condor ( <i>Gymnogyps californianus</i> )	CR	Poisoning, bullets	USA	Wiemeyer et al. 1983, 1986, 1988, Janssen et al. 1986, Bloom et al. 1989, Pattee et al. 1990, Meretsky et al. 2000, Snyder and Snyder 2000
Andean Condor ( <i>Vultur gryphus</i> )	NT	Poisoning	Captive	Locke et al. 1969
King Vulture ( <i>Sarcorhampus papa</i> )	LC	Poisoning	Captive	Decker et al. 1979
White-throated Sparrow ( <i>Zonotrichia albicollis</i> )	LC	Ingestion	USA	Vyas et al. 2000
Dark-eyed Junco ( <i>Junco hyemalis</i> )	LC	Ingestion	USA	Vyas et al. 2000
Brown-headed Cowbird ( <i>Molothrus atar</i> )	LC	Ingestion	USA	Vyas et al. 2000
Yellow-rumped Warbler ( <i>Dendroica coronata</i> )		Poisoning	USA	Lewis et al. 2001
Brown Thrasher ( <i>Toxostoma rufum</i> )		Poisoning	USA	Lewis et al. 2001
Solitary Vireo ( <i>Vireo solitarius</i> )		Poisoning	USA	Lewis et al. 2001

Countries are given for wild birds; captive birds are simply listed as captive, and were poisoned through shot present in their feed. Conservation status is from BirdLife International (2004) and is coded as: CR—Critically Endangered, EN—Endangered, NT—Near Threatened, LC—Least Concern. Evidence of poisoning indicates tissue lead concentrations indicative of poisoning (e.g., Franson 1996) with the source of poisoning most likely to be lead gunshot and/or diagnosis of lead poisoning in one or more individuals; ‘Ingestion’ indicates evidence of ingestion of shot usually in the absence of tissue analysis. Rare cases of lead poisoning believed to be from non-ammunition sources are excluded.

**Table 2.** Guidelines for interpretation of tissue lead concentrations in *Falconiformes* and *Galliformes* (ppm ww).

	Falconiformes (ppm ww)			Galliformes (ppm ww)		
	Blood	Liver	Kidney	Blood	Liver	Kidney
Sub clinical	0.2-1.5	2-4	2-5	0.2-3	2-6	2-20
Toxic	>1	>3	>3	>5	>6	>15
Compatible with death	>5	>5	>5	>10	>15	>50

Adapted from Franson (1996)

months following absorption. Once deposited in bone, lead is far less mobile, and bone lead concentrations tend to remain elevated for months to years, reflecting lifetime exposure (Pain 1996). Several authors have suggested guidelines to help interpret tissue lead concentrations in different avian taxa (Franson 1996, Pain 1996, Table 2). Differences in sensitivity exist both among and within taxa (e.g., Table 2, Carpenter et al. 2003), and other factors, such as the duration and level of exposure, may influence the tissue lead concentrations at which effects are observed. There is no simple way of relating tissue lead concentrations to effect. However, the general conclusions on tissue lead levels associated with sub-lethal poisoning, toxicity, and death from lead poisoning that have been drawn from the vast field and experimental data reported in the published literature are useful guidelines (Table 2, Franson 1996).

The definition of 'background' lead concentrations is rather more complex and will differ with the context of the question being asked and the population under study. Natural levels of exposure to lead in the environment no longer exist as lead resulting from anthropogenic emissions is ubiquitous. Concentrations in the environment distant from emission sources are generally described as 'background.' In remote areas most birds would be expected to have <2 and certainly <5 µg/dL blood lead from exposure to background levels. However, populations of scavengers that feed in urban areas or at rubbish dumps may have median population blood lead concentrations far higher than this as they will be exposed to a mix of anthropogenic sources of lead. These higher concentrations could still usefully be defined as 'background for this population' when attempting to distinguish between low level chronic exposure from a mix of general

environmental sources, and acute exposure, for example through the ingestion of lead from ammunition.

The diagnosis of lead poisoning is usually based upon clinical signs of poisoning in combination with blood lead concentrations in live birds. In dead birds, diagnosis is based upon tissue (liver or kidney) lead concentrations and clinical signs of poisoning, sometimes, but not necessarily, in combination with evidence of exposure to lead. Clinical signs of poisoning and/or evidence of exposure alone are insufficient to positively diagnose lead poisoning and tissue lead analysis is required. The presence of ingested lead objects in the intestine, while a good indicator, is not diagnostic of poisoning as little absorption may have occurred if objects have been ingested very recently. Similarly, the absence of lead objects in the intestine does not mean that birds have not been lead poisoned, as lead objects may be regurgitated or passed through after considerable absorption has occurred, or may be ground down and/or totally dissolved and absorbed.

Clinical signs are more frequently recorded in cases of chronic poisoning, whereas acutely poisoned birds may die rapidly in apparently good body condition (e.g., Gill and Langelier 1994). Liver and kidney lead concentrations are most useful in lead poisoning diagnosis post-mortem. Bone lead concentrations are less useful as, in cases of acute poisoning, lead poisoned birds may die rapidly with comparatively low bone lead concentrations, and conversely, high bone lead concentrations may result from chronic lifetime exposure in the absence of lead toxicity. Except in cases of acute exposure, bone lead concentrations have been shown to be positively correlated with age in waterfowl populations (Stendell et al. 1979, Clausen et al. 1982).

## IDENTIFYING THE SOURCE OF LEAD

There are relatively few sources of lead to which exposure is sufficient to result in tissue concentrations indicative of toxicity (Table 2) or to result in mortality. By far the majority of cases in birds result from the ingestion of shot or bullet fragments (Table 1), or anglers' lead weights in certain water birds (Scheuhammer and Norris 1995). Rare cases result from point source exposure, e.g., to mine waste (Henny et al. 1991, 1994, Sileo et al. 2001) or industrial pollution (Bull et al. 1983), or the ingestion of lead in paint (Sileo and Fefer 1987). In cases where lead poisoning has been diagnosed and lead shot and/or bullet fragments are found in the bird's intestine, the likely source is self-evident. In most cases where no lead from ammunition is present, knowledge of feeding habits or areas of the species is often sufficient to conclude that lead from ammunition is the likely source.

One additional tool to help identify likely sources of lead contamination is the examination of the distribution of blood or soft tissue lead concentrations in a population of birds. Blood and soft tissue lead concentrations in birds that have simply been exposed to generally distributed anthropogenic lead (e.g., from atmospheric deposition) should follow a relatively normal distribution. Dramatic outliers from this, or distributions particularly skewed to higher lead concentrations, suggest elevated lead ingestion from an additional source in a proportion of birds. Distributions are likely to be normal with a few elevated outliers in birds that infrequently ingest lead ammunition, and heavily skewed to elevated concentrations, with more very elevated outliers in populations that more frequently ingest lead ammunition. Although the examination of population lead distributions is a useful indicator and can provide supporting evidence, and while lead from ammunition is by far the most frequently reported source of exposure, most distributions could potentially result from other forms of exposure, and the examination of tissue lead distributions should simply be used as a tool.

Recently, lead isotopes have been used to help source lead exposure (e.g., Meharg et al. 2002). Ratios of lead isotopes,  $Pb^{206, 207, 208}$ , can vary with the origin of the lead. For example, lead in background

sources such as soil and air may have markedly different isotopic ratios from certain industrial sources, or from lead mined in different geographical regions. The utility of lead isotopes for distinguishing sources of lead contamination in wild birds was illustrated by Scheuhammer and Templeton (1998). They compared isotopic ratios in lead-exposed waterfowl and eagles and found them to be similar to those from lead shot pellets, whereas juvenile Herring Gulls (*Larus argentatus*) had isotopic ratios within the range characterising lead from gasoline combustion. Stable lead isotope ratios were used by Martin and Barrett (2001) to determine the source of lead exposure to wildlife on the north shore of Lake Erie. While none of the migrating birds sampled had lead levels indicating lead poisoning, one or more individuals of the following species were found to have levels indicative of sub-lethal lead exposure, with the lead isotope ratios for most of the samples falling within the range of gunshot pellets: American Kestrel (*Falco sparverius*), Sharp-shinned Hawk (*Accipiter striatus*), Cooper's Hawk, Northern Goshawk, Northern Harrier (*Circus cyaneus*), and Red-tailed Hawk (*Buteo jamaicensis*). More recently, Pain et al. (2007) found lead isotope ratios in the livers of Red Kites in the UK to be consistent with ratios in lead pellets, and Church et al. (2006) found that free-flying California Condors with relatively high blood lead concentrations had isotope values consistent with lead from ammunition and distinct from those of pre-release condors with low blood lead concentrations.

In most cases of poisoning from lead ammunition sources, there is sufficient knowledge of the feeding habits of birds, and their proclivity to ingest lead shot and bullet fragments to negate the need for isotopic studies. Isotopic studies may be particularly useful in those rare cases when the source of lead is unclear or where unusual patterns of tissue lead concentrations are found.

## CASE STUDIES

Many lead poisoning studies are based upon knowledge of a species' feeding habits and ranges, the likelihood of shot and bullet fragment ingestion, and tissue lead concentrations, or upon the presence of ingested shot or bullet fragments. While these

factors are generally sufficient to evaluate lead poisoning from ammunition sources, many other studies have included a much wider range of data. A few illustrative examples are outlined here. Evidence for lead poisoning of Marsh Harriers in southern Europe came from (1) knowledge of the propensity of Marsh Harriers to scavenge waterfowl, (2) temporal variation in the proportion of regurgitated pellets containing lead shot, with far higher incidence during the hunting season and, (3) temporal variation in the proportion of birds with elevated blood lead concentrations, with a far higher incidence during than outside the hunting season (Pain et al. 1993, 1997, Mateo et al. 1999). In the UK, evidence for lead shot poisoning of Red Kites included (1) knowledge of propensity to scavenge game and other shot species, (2) the presence of lead gunshot in regurgitated pellets, (3) tissue lead concentrations consistent with lead poisoning mortality at post mortem, and (4) lead isotope values in the tissues of dead birds with elevated lead concentrations consistent with those of lead shot (Pain et al. 1997). By far the most comprehensively studied species is the California Condor, for which evidence for lead poisoning from spent shot and bullets includes all of the above. It includes: post-mortem evidence and/or evidence of ammunition ingestion, moribund birds with clinical signs of lead poisoning and elevated blood lead levels, elevated blood lead levels in free-flying birds, temporal and spatial correlations between big game hunting seasons and areas and elevated lead levels in condors, and lead isotope ratios in the tissues of exposed condors consistent with those of lead shot and bullets (Church et al. 2006 and authors in Table 1). This provides only a small sample of the numerous studies that combine a wide range of evidence of lead poisoning from ammunition sources in individual species.

#### **SPECIES AFFECTED BY LEAD POISONING**

Cases of lead shot ingestion and/or poisoning in terrestrial birds are documented in Table 1. This currently includes 33 raptor species and 30 species from *Gruiformes*, *Galliformes* and various other avian taxa. Twenty-eight raptor species and all species from other taxa were free-flying, and an additional ten raptor species were poisoned accidentally

in captivity from lead shot in food carcasses. This was usually hunter-killed rabbits, squirrels and other small mammals, including an example where shot were manually removed from prey, but where small lead fragments likely remained (Locke et al. 1969, Benson et al. 1974, Decker et al. 1979, Jacobsen et al. 1977, Stehle 1980, MacDonald et al. 1983, Pain et al. 1997). While most cases of poisoning are in raptors, *Gruiformes* or *Galliformes*, several species from other taxa have been shown to have ingested shot, including pigeons, doves, gulls, and a range of passerines.

In addition to published cases reviewed for this study, records of many other species poisoned by lead ammunition exist at veterinary schools and similar institutions (M. Pokras pers. comm.).

#### **PHYSIOLOGICAL AND POPULATION IMPACTS OF LEAD POISONING**

Lead poisoning through the ingestion of shot and bullet fragments by terrestrial birds causes a wide range of sub-lethal impacts affecting physiology and behaviour, and when exposure is sufficiently high or of sufficient duration results in mortality. Experimental studies in birds have shown that, among other effects, lead can impair blood synthesis, immune function and reproduction (Grandjean 1976, Kendall et al. 1981, Kendall and Scanlon 1981, Veit et al. 1982, Edens and Garlich 1983, Scheuhammer 1987, Grasman and Scanlon 1995, Redig et al. 1991). Consequently, free-living birds exhibiting sub-lethal poisoning will likely be more susceptible to disease, starvation and predation, and an increased probability of death from other causes (Scheuhammer and Norris 1996). Although the extent to which survival is reduced by sub-lethal lead poisoning is difficult to quantify in field studies, it is clearly undesirable in any species.

Mortality from lead poisoning is of greatest concern in long-lived, slow-breeding species, characteristics typical of many raptors, and particularly species with populations that are either naturally small or have been reduced by other factors. Such species are particularly vulnerable to increases in adult mortality, and all age classes can be affected by lead poisoning.

Lead poisoning in the Bald Eagle, a species whose population was depressed through losses due to pesticide abuses in the 1950s and 1960s, is well documented (Elliott et al. 1992, Pattee and Hennes 1983, Wayland and Bollinger 1999, Table 1). In Canada and the USA, approximately 10–15% of recorded post-fledging mortality in Bald and Golden Eagles was attributed to the ingestion of lead shot from prey animals (Scheuhammer and Norris 1996). Elliott et al. (1992) found that 14% of 294 sick, injured, or dead Bald Eagles in British Columbia (1988 to 1991) were lead-poisoned and an additional 23% sub-clinically exposed, with the majority of exposure occurring during January to March, when the birds were feeding heavily on wintering wildfowl.

Of particular concern is lead poisoning in globally threatened species. To date, eight globally threatened (Critically Endangered, Endangered or Vulnerable) or Near Threatened (BirdLife International 2008) free-living species have been reported as lead-poisoned (Table 1), of which six are raptors. While there were isolated reports in two cases, the Endangered Whooping Crane (*Grus americana*) and Critically Endangered Oriental White-backed Vulture (*Gyps bengalensis*), multiple reports exist for the other species. In addition, two captive Near Threatened species have been reported with lead poisoning from ammunition sources: Laggar Falcon (*Falco jugger*) and Andean Condor (*Vultur gryphus*). High levels of adult mortality occurred in globally Vulnerable Steller's Sea-eagles and White-tailed Eagles (Least Concern) in Hokkaido, Japan, until a ban on the use of lead bullets in this area (Kurosawa 2000). Population modelling indicated that in the absence of preventative measures Steller's Sea Eagle populations would decline as a result of lead poisoning, and that populations were most sensitive to changes in adult mortality (Ueta and Masterov 2000). The Vulnerable Spanish Imperial Eagle (*Aquila adalberti*) is an example of a species with a small population, estimated at 200 pairs (BirdLife International 2008) that cannot sustain high, especially adult, mortality (Ferrer 2001, Ferrer et al. 2003). The most significant impacts of lead poisoning on any terrestrial species have been on the Critically Endangered California Condor. Lead poisoning from lead shot and bullet fragments was a major factor in the decline of this species,

which became extinct in the wild, and has seriously hampered reintroduction efforts (Meretsky et al. 2000, Cade 2007).

In addition to lead poisoning in globally threatened species, many of the species in Table 1 are of national or regional conservation concern (e.g., Gregory et al. 2002 for the UK).

Not only does lead poisoning cause unnecessary mortality in many terrestrial avian species, and present a significant problem at a population level to a few, it also severely compromises the welfare of large numbers of wild birds, both terrestrial and aquatic. Sainsbury et al. (1995) investigated the scale and severity of welfare issues in wild European birds and mammals. They described the nature and level of harm caused (pain, stress and fear), the duration of effects and numbers of individuals affected, and found lead poisoning through shot ingestion to be among the most significant human activities that severely compromise the welfare of large numbers of animals.

#### REMOVING THE THREAT OF LEAD POISONING IN TERRESTRIAL BIRDS

Lead poisoning from ammunition affects the populations of several globally threatened species, and hampers the reintroduction of the critically endangered California Condor. In many species it causes mortality or sub-lethal effects which may influence survival. Limited legislative steps have been taken to reduce the threat of lead poisoning to terrestrial birds, including nationwide legislation banning the use of lead shot for all waterfowl hunting in the USA from 1991, aimed at preventing secondary lead toxicosis of the Bald Eagle (Anderson 1992), and a ban on the use of lead bullets for hunting on Hokkaido, Japan, where secondary poisoning affected many Steller's Sea Eagles (Matsuda 2003). More recently, and in the face of considerable opposition from the gun lobby, the State of California passed Assembly Bill 821 in 2007 to restrict the use of lead shot and bullets in the range of the California Condor. Such legislation is an important step towards reducing the risk of lead poisoning in terrestrial birds; policing and compliance are also essential, and much remains to be done in these areas.

The risk to terrestrial and water birds, of lead poisoning from ammunition is just one of many reasons for replacing lead in all ammunition with non-toxic alternatives. Other reasons have been covered elsewhere in this volume, and include the following:

- Lead is a highly toxic heavy metal with no known biological function that affects humans and other animals at the lowest measurable concentrations. Lead has already been banned from most uses that could result in human and wildlife exposure (e.g., from paint, and as an anti-knocking agent and octane booster in gasoline/petrol).
- The use of lead ammunition results in huge quantities of lead deposited in the environment annually (Thomas 1997), where it is extremely persistent in contaminating soils and waterways (e.g., Sorvari et al. 2006) and augmenting potential future risks to wildlife inhabiting shot-over areas.
- Environmental clean-up following the deposition of lead ammunition is both difficult and costly.
- Some lead from ammunition sources is or becomes bioavailable and may be bioaccumulated by edaphic organisms (Migliorini et al. 2004), and puts at risk vegetation, invertebrates and other organisms (Bennett et al. 2007).
- Lead poisoning may severely compromise the welfare of large numbers of wild animals.
- Lead from ammunition can present a human health risk, e.g., at firing ranges and to people who consume game shot with lead (Scheuhammer et al. 1998, Tsuji et al. 1999, Guitart et al. 2002, Johansen et al. 2004, Gustavsson and Gerhardsson 2005). While not all people that consume game will necessarily develop elevated blood lead concentrations (e.g., Haldimann et al. 2002), those that frequently consume game such as subsistence hunters appear particularly at risk (e.g., Bjerregaard et al. 2004) as may be people who cook game using recipes of low pH that facilitate lead dissolution (e.g., Mateo et al. 2007).

There are few reasons to continue with the use of lead ammunition. Non-toxic shot regulations have been shown to be successful at reducing the incidence of lead poisoning (e.g., Anderson et al. 2000, Stevenson et al. 2005) and are unlikely to result in

increased crippling rates (Schultz et al. 2006). Cade (2007) reviewed the compelling evidence for the impacts of lead poisoning from ammunition on the California Condor population, and concluded that the use of non-toxic shot and bullets would be highly efficacious for hunting, economically feasible, and ethically the right thing to do.

While previous regulatory emphasis has been placed on wetland habitats or waterfowl hunting, there is now an urgent and compelling need to replace all lead gunshot and rifle bullets for shooting game, pest species, and targets with non-toxic alternatives. The wide extent of primary and secondary lead poisoning has been demonstrated across a large range of bird species and continents where hunting has been practiced. The burden of proof that lead from spent gunshot and bullets causes lead toxicosis has already been achieved for wetland species, upland species, and avian predators and scavengers. More recently, the risks to humans have been documented, especially those who regularly eat hunted game meat (see the collection of papers in this volume). Thomas and Guitart (2005) indicated that several global treaties and agreements exist that could authorize the transition to non-toxic shot and bullets in many countries, and presage the passing of binding legislation to support bans on the use of lead products. The removal of lead shot, bullets and fishing weights from sporting use is also completely consistent with the Precautionary Principle (Matsuda 2003), and does not appear compromised by considerations of economic costs, commercial availability, or ballistic effectiveness. The transition to use of non-toxic lead substitutes is vital to ensure the sustainable use of wildlife, and to reduce the risks of lead poisoning from lead bullets and shot to human health.

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