PETITION TO THE ENVIRONMENTAL PROTECTION AGENCY TO BAN LEAD SHOT, BULLETS, AND FISHING SINKERS UNDER THE TOXIC SUBSTANCES CONTROL ACT

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Lead-poisoned bald eagle, photo credit U.S. Fish and Wildlife Service, Dr. Rhoda M. Ralston

PETITIONERS

CENTER FOR BIOLOGICAL DIVERSITY
AMERICAN BIRD CONSERVANCY
ASSOCIATION OF AVIAN VETERINARIANS
PROJECT GUTPILE
PUBLIC EMPLOYEES FOR ENVIRONMENTAL RESPONSIBILITY

PETITION FOR RULEMAKING
UNDER THE TOXIC SUBSTANCES CONTROL ACT

August 3, 2010

EXECUTIVE SUMMARY

Pursuant to the Toxic Substances Control Act ("TSCA", 15 U.S.C. § 2601 *et seq.*), Petitioners Center for Biological Diversity, American Bird Conservancy, Association of Avian Veterinarians, Project Gutpile and Public Employees for Environmental Responsibility hereby petition the Environmental Protection Agency ("EPA") to revise rules governing toxic substances to ban the manufacture, processing and distribution in commerce of lead shot, bullets, and fishing sinkers. Petitioners request that the EPA consider this rulemaking pursuant to section 6(a) of TSCA.

Based on information extending back to Roman times more than 2,000 years ago, lead has long been identified as a highly toxic substance with lethal properties and numerous pathological effects on living organisms. Health effects from lead exposure can run the gamut from acute, paralytic poisoning and seizures to subtle, long-term mental impairment, miscarriage and impotence. Lead is a cumulative metabolic poison affecting a large number of biological functions including reproduction, growth, development, behavior and survival. Even low levels of exposure to lead can cause neurological damage, and there may be no safe level of lead in the body tissues of fetuses and young. Despite this knowledge, lead continues to be used in manufactured products, many of which are sources of toxic lead exposure to wildlife and to human beings.

In recent decades the federal government has begun to implement regulations to reduce the exposure of human beings to lead in drinking water, paint, gasoline, toys, toxic dumps, lead wheel balancing weights and both indoor and outdoor shooting ranges. Strict recycling regulations have been imposed on disposal of lead-acid batteries. However, spent lead ammunition and lost lead fishing tackle are uncontrolled and lead remains widely encountered and distributed in the environment from these sources. The continued availability of traditional lead bullets and shot exposes any animal that preys or scavenges on targeted wildlife to lead's toxic effects. Particularly susceptible are avian scavengers that encounter lead in carcasses left in the wild, in gut piles (viscera) from animals cleaned in the wild, and in wounded prey species that survive hunting and carry lead ammunition in their bodies. Sensitive species such as bald and golden eagles and endangered California condors are frequently killed by lead poisoning or suffer chronic sublethal effects of lead poisoning from scavenging meat containing lead fragments from ammunition. Lead shotgun pellets and lead fishing tackle accumulate in both aquatic and terrestrial habitats, where animals encounter and ingest these lead items, often mistaking them for food, grit or bone fragments. More than 130 species of animals (including mammals, upland birds, raptors, waterfowl, amphibians and reptiles) have been reported in scientific literature as being exposed or killed by ingesting lead shot, bullets, bullet fragments, fishing tackle or prey contaminated with lead ammunition.

Ducks, geese and swans have received protection from hunting sources of lead poisoning since 1991 by a federal requirement to use only nontoxic shot for hunting waterfowl, but similar restrictions in terrestrial habitats are scattered and localized. Data now show that over 75 terrestrial species of birds are known to be poisoned by spent lead from ammunition. Mourning doves are particularly susceptible to ingesting lead shot pellets,

and lead poisoning may kill as many as 20 million doves per year in the United States. Lead fishing sinkers and jigs continue to cause the needless deaths of waterfowl species such as trumpeter swans, ducks, geese and loons.

Ammunition and tackle manufacturers now market a wide variety of non-lead, nontoxic bullets, shotgun pellets and fishing tackle that can replace lead projectiles and weights. There is no technological or commercial reason why nontoxic ammunition and fishing tackle with comparable effectiveness should not be substituted for their lead counterparts. Several states have mandated nontoxic shotgun ammunition for upland game bird hunting, and states in the Northeast have begun to require non-lead fishing weights and lures in an effort to protect loons and other wildlife. Those states with only a partial ban, such as California's requirement for big game hunting with nontoxic ammunition within the eight-county range of California condors, continue to have high rates of lead poisoning in wildlife.

The EPA has long held that whenever a toxic substance customarily used in the manufacture of commercial products can be replaced by a nontoxic substitute, articles made of the toxic substance should be removed from the market. All hunting and fishing gear containing lead could economically be replaced with effective, nontoxic alternatives, thus making a strong argument for EPA regulatory action.

TSCA grants the EPA the broad authority to regulate chemical substances that "present an unreasonable risk of injury to health or the environment" (15 U.S.C. § 2061). The EPA may regulate the manufacture, processing, distribution, use or disposal of such chemical substances. Specific control mechanisms include: prohibitions on an entire or certain use of a chemical substance; limitations on allowable concentration levels; labeling or recordkeeping requirements; and obligations to issue notice of risks of injury (15 U.S.C. § 2605(a)). The EPA has already declared that lead is a toxic substance, and has removed nearly all products containing lead from the market. The requirement for nontoxic shotgun shot, bullets, and fishing gear may be achieved with the EPA prohibiting the manufacture, processing, or distribution in commerce of a chemical substance for a particular use (15 U.S.C. § 2605(a)(2)(A)(i)). The EPA is specifically prohibited from regulating ammunition or firearms under TSCA, but toxic components of ammunition can be regulated if nontoxic alternatives are commercially available. The petitioners have waited until nontoxic alternatives have become available to submit this petition in an effort to clearly indicate that this petition is not an attempt to regulate ammunition or firearms.

States that have mandated nontoxic shotgun ammunition for upland game bird hunting and nontoxic fishing gear continue to have active hunting and fishing communities that have successfully transitioned away from lead products. Market forces in these states have caused a full line of nontoxic replacement products to be made available to the public, demonstrating that commercially available alternatives exist and the economic consequences of removing lead from the environment will be minimal. The EPA is compelled under TSCA to grant this petition and develop regulations to require nontoxic alternatives to lead sporting products.

TABLE OF CONTENTS

1. PETITIONERS AND STANDING TO FILE

Section 21 of the Toxic Substances Control Act ("TSCA" or the "Act", 15 U.S.C. § 2601 *et seq.*) provides that "any person" may petition the Environmental Protection Agency Administrator ("EPA") to initiate a proceeding for the "issuance, amendment, or repeal of a rule" (15 U.S.C. § 2620(b)(3)). Petitioners therefore have standing to petition the EPA to initiate proceedings to ban lead shot, bullets, and fishing sinkers under section 2605 of TSCA.

Petitioner Center for Biological Diversity ("Center") is a non-profit organization that works to protect endangered species and wild places through science, policy, education, citizen activism, and environmental law. The Center and its 255,000 online activists and members have an ongoing interest in protecting wildlife from lead poisoning. Since 2004, the Center has taken action through a "Get the Lead Out" campaign to change policies to regulating lead to prevent toxic lead from entering the food chain. The Center has been a leading proponent of regulations on lead ammunition to protect endangered California condors, bald and golden eagles, and other wildlife species at risk from lead poisoning.

Center for Biological Diversity
351 California Street, Suite 600
San Francisco, CA 94104
Contact: Jeff Miller
E-mail: jmiller@biologicaldiversity.org

Petitioner American Bird Conservancy conserves native wild birds and their habitats throughout the Americas, and acts to safeguard the rarest bird species, restore habitats, and reduce threats to bird species. The American Bird Conservancy has been a leading organization working to reduce all mortality threats to birds from habitat destruction, collisions with buildings and wind turbines, and from toxins such as hazardous pesticides and lead.

American Bird Conservancy
1731 Connecticut Ave., NW
Washington, DC 20009
Contact: Michael Fry, Director of Conservation Advocacy
E-mail: mfry@abcbirds.org

Petitioner Association of Avian Veterinarians is an international professional organization of practitioners advancing and promoting avian medicine, stewardship, and conservation through education of its members, the veterinary community and those they serve.

Association of Avian Veterinarians
P.O. Box 811720
Boca Raton, FL 33841

Contact: Adina Rae Freedman, Executive Director
E-mail: aavcentraloffice@gmail.com

Petitioner Project Gutpile is an educational organization comprised of hunters that provides educational resources for lead-free hunters and anglers. Project Gutpile has been promoting non-lead ammunition and raising lead awareness in the hunting community since 2002.

Project Gutpile
Contact: Anthony Prieto
E-mail: projectgutpile@gmail.com

Petitioner Public Employees for Environmental Responsibility ("PEER") is a 10,000 member national alliance of local, state and federal resource professionals working to protect the environment. PEER members include government scientists, land managers, environmental law enforcement agents, field specialists, and other resource professionals committed to responsible management of America's public resources.

Public Employees for Environmental Responsibility
P.O. Box 4057
Georgetown, CA 95634
Contact: Karen Schambach, California Field Director
E-mail: capeer@peer.org

TSCA requires that within 90 days after filing of a petition, the EPA shall either grant or deny the petition (15 U.S.C. § 2620(b)(3)). If the Administrator grants the petition, the Administrator shall promptly commence an appropriate proceeding. If the Administrator denies the petition, the Administrator shall publish in the Federal Register the Administrator's reasons for such denial (15 U.S.C. § 2620(b)(3)).

II. NATURE OF THE REQUESTED ACTION

Petitioners Center for Biological Diversity, American Bird Conservancy, Association of Avian Veterinarians, Project Gutpile and Public Employees for Environmental Responsibility request that the EPA adopt regulations prohibiting the manufacture, processing, and distribution in commerce of lead shot, lead bullets, lead fishing sinkers, and other lead-containing fishing gear, pursuant to TSCA (15 U.S.C. § 2605(a)(2)(A)(i)). Such regulations are needed to protect vulnerable wildlife species from the ongoing threat of lead poisoning, as well as to safeguard human health.

TSCA mandates that the EPA must regulate chemical substances where there is a "reasonable basis to conclude" that such substances "present an unreasonable risk of injury to health and or the environment" (15 U.S.C. § 2605(a)). TSCA authorizes the EPA to prohibit "the manufacturing, processing, or distribution in commerce" of a chemical substance for a particular use or uses (15 U.S.C. § 2605(a)(2)(A)(i)).

Lead used in shot, bullets, and fishing sinkers is a "chemical substance" falling within the scope of TSCA. As defined by the Act, "Except as provided in subparagraph (B), the term "chemical substance" means any organic or inorganic substance of a particular molecular identity, including (i) any combination of such substances occurring in whole or in part as a result of a chemical reaction or occurring in nature and (ii) any element or uncombined radical" (15 U.S.C. § 2602(2)(A)).

Most other uses of lead, such as lead-based paints, plumbing pipe and fixtures, and leaded gasoline, are already subject to strict regulation (15 U.S.C. §§ 2681-2692). In January 2008, lead and lead compounds were added to the Priority Testing List (40 C.F.R. 716.120; *see also* 15 U.S.C. § 2603(e)), requiring certain lead manufacturers to submit unpublished health and safety reports to the EPA (73 Fed. Reg. 5109-5115; Jan. 29, 2008). Automobile wheel balancing weights will be phased out with an EPA proposed rule scheduled for 2011. Manufacturers of consumer products intended for use by children who also manufacture lead or lead compounds are required to report certain health and safety data to the EPA. However, there is currently no specific regulation of lead shot, bullets or fishing sinkers under TSCA.

III. REASON FOR THE REQUEST

A. INTRODUCTION

Lead has been used by humankind for millennia. Lead had numerous uses in ancient Egypt and it is believed that toxicity arising from the use of lead in water pipes, pottery, cosmetics, food and wine may have contributed to the fall of the Roman Empire (Hernberg 2000). The properties of lead as a biocide have been well known for hundreds of years. It is now unquestioned scientific knowledge that lead is a toxic substance with potentially lethal as well as numerous pathological effects on living organisms of all sorts. Despite this knowledge, lead has continued to be used in a wide variety of manufactured products, many of which are continued sources of toxic lead exposure to human beings and to wildlife.

The use of lead for hunting dates back hundreds of years, and lead has been used in fishing activities for thousands of years. The effects of lead in modern shot, bullets, and fishing sinkers on fish and wildlife have been well documented and reviewed (e.g. Pain 1992; Fisher et al. 2006; Rattner et al. 2007). Recognizing these threats, some jurisdictions began placing restrictions on the use of lead ammunition and tackle in the 1970s and 1980s. Despite restrictions, significant amounts of lead continue to be deposited in aquatic and upland habitats and enter the food chain from hunting and fishing activities. Legal and illegal hunting using lead ammunition may directly or secondarily expose wildlife to lead and deposit bioavailable lead into the environment. Skeet/trap shooting and target practice activities also deposit significant amounts of lead in geographically localized areas. Significant amounts of lead end up in aquatic environments from lost or discarded fishing tackle, including lures, sinkers, lead core fishing line, downrigger cannonballs, weights, and a variety of fishing traps and nets that employ the use of lead.

Lead can remain in the environment relatively intact and stable for decades, and, under some environmental conditions it can be readily released and taken up by plants or animals (ATSDR 2007). Absorbed or ingested lead can cause a range of biochemical, physiological, and behavioral effects in species of invertebrates, fish, amphibians, reptiles, birds, and mammals. Wildlife can be exposed to lead through feeding in aquatic environments and ingesting contaminated vegetation and sediments, feeding on invertebrates or vertebrates containing lead, or ingesting lead pellets or fragments directly, mistaking them for food, grit, or bone. Although lead is a naturally occurring metal in the environment, for biological systems it is a nonessential metal with no functional or beneficial role at the molecular or cellular level. Ingested lead substitutes in dysfunctional ways for calcium in biochemical interactions, with harmful effects on neurological functions, bone structure, renal function, reproductive functions, pancreatic functions, and muscular functions, among others.

Lead is toxic to organisms at very low levels, and has lethal and severe sublethal effects at higher levels (IPCS 1989; Nordic Council of Ministers 2003). Lead can act as a neurotoxin, and numerous studies indicate that blood lead concentrations even below 10

micrograms per deciliter can have adverse developmental effects on intellectual functioning and social-behavioral conduct in humans (Needleman et al. 1990; Canfield et al. 2003; Ris et al. 2004). Human fetuses and young children are particularly sensitive to even low levels of lead exposure and can easily suffer permanent neurological damage. Clinicians now assert that there is no safe level of lead in the body tissues for fetuses and young children (e.g. Canfield et al. 2003; Lanphear et al. 2005, 2006; Carlisle et al. 2009).

In recent decades the federal government has taken various regulatory actions to reduce the exposure of humans to lead in drinking water, paint, gasoline, toys, toxic dumps, automobile wheel balancing weights, and indoor and outdoor shooting ranges. However, other lead sources causing significant contamination are still uncontrolled, and lead exposure to wildlife has recently been widely documented and is not adequately regulated.

Toxic lead shotgun pellets and bullet fragments from hunting accumulate in both aquatic and terrestrial habitats, where animals often eat them because they are mistaken for grit or food (seeds). Birds frequently ingest spent lead shotgun pellets as grit normally consumed to aid grinding of foods in the gizzard during digestion. Ducks, geese, and swans have received much protection from this source of lead poisoning since 1991 through the federal requirement to use only nontoxic shot for hunting waterfowl, but similar restrictions in terrestrial habitats are few, scattered, and localized. Research has shown that over 75 terrestrial species of birds are known to be poisoned by spent lead ammunition (Eisler 1988; Fisher et al. 2006).

Evidence has accumulated over the past century and it is now incontrovertible fact that lead fragments in the bodies of animals shot with lead bullets or lead shotgun pellets are a serious source of lead exposure to scavenging animals that eat meat containing lead fragments and residue (Calvert 1876; Holland 1882; Grinnell 1894; Bowles 1908). Lead fragments in meat also pose potential health risks to humans who eat lead-tainted game, especially subsistence hunters dependent upon hunted game for food. Critically endangered California condors, bald and golden eagles, ravens, and other scavenging birds are frequently killed or harmed by this source of lead poisoning. Spent lead shotgun pellets on the ground in fields where upland game birds are hunted are also ingested by birds as grit making herbivorous birds as well as carnivorous birds victims of lead poisoning. Mourning doves are particularly sensitive to this, as they are hunted in fields where they congregate to feed, and spent lead pellets are the appropriate sized grit for doves.

More than 130 species of animals (including upland birds, raptors, waterfowl, mammals and reptiles) have been documented being exposed or killed by ingesting lead shot, bullets, bullet fragments, or prey contaminated with lead ammunition (Environment Canada 1995; Tranel and Kimmel 2009). As long as ammunition manufactured with lead projectiles and lead fishing tackle remains available for purchase and use, numerous species of wildlife will continue to be poisoned by lead and human health will be threatened, posing an unreasonable risk of injury to human health and the environment.

B. Sources and Quantities of Lead in the Environment from Hunting, Shooting Sports, and Fishing Activities

Hunting

The density of spent lead shot in wetlands or fields is related to hunting intensity. In waterfowl hunting areas, prior to the national requirement for nontoxic shot, densities of spent shot have been reported from about 50,000 pellets to over 2 million pellets per acre (Bellrose 1959; Pain 1992; Rocke et al. 1997). Areas with regular hunting from fixed position blinds or pits resulted in significant accumulation of spent lead. Prior to the banning of lead shot for waterfowl hunting, an estimated 2,721 metric tons of spent lead shot were deposited in U.S. wetlands each year (Pain 1992), and spent shot accumulated near the surface of sediments in aquatic settings, increasing the amount of lead shot available to waterfowl over time (Pain 1992). The depth of lead fragments in soil and their availability can be influenced by land management practices such as cultivation, and lead shot and bullets can persist for decades to hundreds of years (Fredrickson et al. 1977; Jorgensen and Willems 1987; Kendall et al. 1996).

Significant lead shot deposition continues today in upland fields used for hunting and densities of spent lead shot can reach over 400,000 pellets per acre (Schulz et al. 2002). Castrale (1989) estimated densities of 11,000 pellets/acre in a field managed for dove hunting in Indiana. Lewis and Legler (1968) estimated 43,600 pellets/acre in a field managed for dove hunting in Tennessee. Esslinger and Klimstra (1983) estimated 44,000 pellets/acre in a field managed for goose hunting in Illinois. Fredrickson et al. (1977) estimated 122,800 pellets/acre in uncultivated fields near duck blinds in Missouri. Best et al. (1992a) estimated 344,000 pellets/acre in an area frequented by dove and quail hunters in New Mexico. The Washington Fish and Wildlife Nontoxic Shot Working Group in 2001 estimated densities of 188,000 and 344,000 pellets/acre at two pheasant release sites in Washington.

Despite the ban on lead shot for waterfowl hunting, large amounts of spent lead ammunition continue to be deposited in the environment through hunting of big game, upland species, furbearers, and from predator control activities (Scheuhammer and Norris 1995; Schulz et al. 2002). Lead from shot, bullets and bullet fragments in tissue or entrails of wounded or dead animals has been increasingly recognized as a threat to many avian species (Jannsen et al. 1986; Hunt et al. 2006; Knopper et al. 2006).

To give an idea of the quantity of potentially lead-tainted carcasses available to scavengers, Fry and Maurer (2003) quantified hunter-shot carcasses available to condors in their California range before the California lead ammunition ban went into effect, and concluded that gut piles and whole carcasses left in the field by hunters were a highly significant source of lead within the condor range. From hunting survey data for the eight counties encompassing the condor range in California, Fry and Maurer (2003) estimated an annual average of 36,000 big game animals (17,000 wild pigs, 11,000 coyotes and 8,000 deer) were taken each year by sport hunters in this area. Fry and Maurer (2003)

assumed that only a very few gut piles are actually buried, hidden successfully, or removed from the field. Deer and pigs are generally field dressed and gut piles discarded in the field; coyotes are generally left in the field intact. The Fry and Maurer (2003) figures do not account for poaching, which likely significantly increases the number of deer carcasses available. The data also do not account for the take of thousands of permitted depredated pigs and deer shot by ranchers, and do not account for small game such as ground squirrels shot by varmint hunters. The carcasses of these large animals left in the field would be the primary source of hunter-shot food for condors. Fry and Maurer (2003) estimated that almost 28,000 tree squirrel, rabbit, and ground squirrel carcasses are left in the field within the condor range annually. Even animals as small as ground squirrels shot with .22 caliber bullets can contain lead fragments at biologically relevant levels that may constitute a lead-hazard for other scavenging birds of prey (Harmata and Restani 1995: Knopper et al. 2006).

Shooting Sports

Target, trap and skeet shooting can result in substantial accumulation of spent lead in localized areas, and lead from spent shot at shooting ranges can become bioavailable to terrestrial and aquatic plants, invertebrates, and vertebrates (Scheuhammer and Norris 1995). Individual shooting ranges can deposit as much as from 1.4 to 15 tons of lead shot and bullets each year; estimates of the annual amount of lead shot and bullets deposited at the roughly 9,000 outdoor shooting ranges in the U.S. range from 72,600 to 80,000 metric tons (Tanskanen et al. 1991; USEPA 2001; Craig et al. 2002; USGS 2008). Kendall et al. (1996) estimated mean densities of spent lead shot at trap, skeet, and sporting clay ranges in the United States at 3.7 billion pellets/acre. Large concentrations of lead at shooting ranges (up to 17,000 grams per square meter) cluster in small areas because of stationary targets, trajectories of launched targets, and concentration of shooters (Darling and Thomas 2003). Significant amounts of fine particulate lead can concentrate near shooting stations.

Studies of the dissolution of lead from shot at terrestrial shooting ranges suggest that one-half of a lead shot pellet would release into the soil within 40 to 70 years and that the entire lead shot would transform in 100 to 300 years, with mechanical disturbance of soil enhancing transformation rates (Jorgensen and Willems 1987; Scheuhammer and Norris 1995; Hardison et al. 2004). Lead concentrations in soil at shooting ranges have been shown to be up to 55,000 mg/kg, over 10,000 times background levels (Scheuhammer and Norris 1995). Lead leaching into soil or sediment exceeding criteria for hazardous waste and dissolved lead entering surface water and ground water exceeding water quality criteria has been documented at many U.S. shooting ranges (Murray et al. 1997; Bruell et al. 1999; Craig et al. 1999; Rooney et al. 1999; Chen et al. 2002; Cao et al. 2003a, 2003b; Soeder and Miller 2003; Sorvai et al. 2006). A study of shooting ranges over water showed high shot density in the soil/sediment fall zone and lead concentrations in water samples two orders of magnitude above EPA water quality criteria (Stansley et al. 1992).

Fishing

Accurate quantitative information on how much lead is entering the environment from lead fishing weights and tackle sinkers is not available, but approximations can be made from the quantities of lead fishing tackle sold in the U.S., assuming most or many sinkers are purchased to replace those lost while fishing (Scheuhammer et al 2003), and from studies of sinker and tackle loss by anglers. Roughly 4,000 metric tons of lead fishing sinkers are sold annually in the U.S. (Scheuhammer et al 2003; USGS 2008). Studies of sinker and tackle loss rates among recreational anglers vary - a variety of factors can influence whether lead will be lost, including the type of fishing activity, the location of the activity, the time of year, and the skill of the angler. Fishers lost 2-3 sinkers per angling day in the United Kingdom (Bell et al. 1985). Anglers in the U.S. reported losing 0.18 sinkers/hour, and 0.23 hooks and lures/hour, with 2% of anglers reporting losing a fish with tackle still attached (Duerr 1999). Radomski et al. (2006) reported average loss rates on Minnesota lakes of 0.0127 lures per hour, 0.0081 large sinkers per hour, 0.0057 split shot sinkers per hour, 0.0247 jigs per hour, and 0.0257 hooks per hour; for a estimated total of one metric ton of lead lost for 6,000 anglers in 2004. Assessments of lead fishing tackle along U.S shorelines have found 0.01 sinkers/square meter of shoreline in areas of low angling pressure up to 0.47 sinkers/m^2 in areas of high angling pressure (Duerr and DeStefano 1999), with much higher densities (up to 190 sinkers/m^2) in studies in Europe (Cryer et al. 1987; Sears 1988). Amounts of lead fishing weights produced and approximations of lost tackle indicate that fishing can introduce significant amounts of lead into aquatic environments. The USEPA (1994) estimated in 1994 that 450 million toxic fishing sinkers containing lead or zinc are produced each year and potentially entering the environment.

The fate of elemental lead in aquatic environments is influenced by water chemistry, wave action, water flow, and pH (see Scheuhammer and Norris 1995). In lakes, lead particles may be adsorbed onto sediment and soil particles. The bioavailability of lead is related to the presence of organic matter and sediments and acidity. In coastal ocean waters, lead sinkers may easily be abraded by wave action against rocks, releasing small fragments into the water column.

C. Pathways of Lead Exposure

Lead has been widely dispersed throughout the environment from activities such as mining, smelting, manufacturing, and engine combustion. Many historical documented instances of lead exposure among terrestrial wildlife species have been associated with small contaminated areas, such as around metal smelters, shooting ranges, lead paint contaminated buildings, or locations with intense hunting pressure (Blus et al. 1991, Henny et al. 1991; Blus et al. 1995; Sileo et al. 2001; Lewis et al. 2001). Manufacture of leaded gasoline, lead-based paints and pesticides, and use of lead solder in cans has now been nearly eliminated in the U.S. The EPA recently granted a petition to ban lead automobile wheel balancing weights, and will produce regulations requiring non-lead wheel weight alternatives in 2011 (USEPA 2009). A petition to phase out the largest

remaining permitted use of leaded gasoline, that for piston-driven aircraft, was filed with the EPA in 2010, and EPA recently solicited comments on that petition (USEPA 2010).

Environmental distribution of lead from hunting and fishing is widespread, although it is difficult to estimate the magnitude of lead exposure compared to other sources, such as legacy residues of leaded gasoline exhaust deposition, emissions from smelters, improper disposal of paint chips and dust, and lead ground to dust from lead wheel weights falling off vehicles. Lead from shot, bullets and fragments in heavily hunted fields, wetlands, and shooting areas can be directly ingested or solubilized and biologically incorporated into food items (Ma 1989; Stansley and Roscoe 1996; Hui 2002). Studies at shooting ranges have shown increased lead levels in terrestrial and aquatic plants (Manninen and Tanskaanen 1993; Peterson et al. 2993; Mellor and McCartney 1994; Rooney et al. 1999; Hui 2002), and lead concentrations in invertebrates have been shown to be elevated due to uptake of lead near shooting ranges (Hui 2002; Labare et al. 2004). There has been increasing evidence documenting significant deposition of spent lead shot and bullets and lost fishing sinkers and tackle, and subsequent ingestion by numerous bird and mammals species, as discussed below.

Aquatic Environments

There is extensive documentation of direct ingestion of lead shot, bullet fragments, sinkers, and jigs by dabbling and diving ducks, swans, loons and other water birds.

There is very little information on lead released from spent shot or fishing weights in aquatic habitats which could be solubilized and taken up by invertebrates or fish (Stansley et al. 1992; Hui 2002). For reptiles and amphibians near heavily hunted wetlands and shooting ranges, consumption of waterborne lead and ingestion of lead-contaminated sediments and food items are likely exposure pathways (Stansley and Roscoe 1996; Borkowski 1997; Camus et al. 1998; Hammerton et al. 2003; Pattee and Pain 2003; Lance et al. 2006). Although direct ingestion of lead shot or ammunition fragments by aquatic invertebrates, reptiles, and amphibians seems unlikely, numerous studies describe injuries to fish incurred by tackle (often containing lead) and small fragments passing through fish digestive track could release lead. Although there is no documentation of such lead uptake or poisoning in fish, there are reported cases of lead poisoning of turtles that have ingested lead fishing tackle (Borkowski 1997; Scheuhammer et al. 2003b). Hooked fish can ingest or retain attached lead fishing tackle such as hooks and jigs, and become a potential pathway of lead exposure for predatory birds consuming fish containing such lead fishing gear. Marsh birds feeding in wetland areas that are hunted with lead ammunition ingest lead, which has been documented in flamingoes, rails, shorebirds, terns, and herons (Artman and Martin 1975; Kaiser et al. 1980; Maedgen et al. 1982; Custer and Mulhern 1983; Hall and Fisher 1985; Locke et al. 1991; Beck 1997; Mateo et al. 1997; Acora 2005).

Terrestrial Environments

The most significant lead exposures and effects are due to direct ingestion of spent lead shot and bullet fragments by waterfowl (Sanderson and Bellrose 1986) and certain upland game species (Kendall et al. 1996, Schulz et al. 2006). Secondary poisoning of birds consuming wounded or dead prey contaminated with lead ammunition and scavenging of gut piles with spent lead ammunition or fragments is a significant source of toxic exposure to predatory and scavenging birds, with particularly deadly effects on bald eagles and California condors (Pattee and Hennes 1983; Kramer and Redig 1997; Meretsky et al. 2000; Church et al. 2006; Hunt et al. 2006; Pauli and Buskirk 2007). The recent use of stable lead isotope ratios has provided evidence that ammunition sources are responsible for lead exposure in wild birds (Scheuhammer and Templeton 1998; Scheuhammer et al. 2003a; Church et al. 2006, Finkelstein et al. 2010).

Granivorous (seed-eating) bird species may ingest lead shot as grit, or perhaps mistaking it for berries, which may be similar in appearance after drying and falling (Calvert 1876; Campbell 1950; Hunter and Rosen 1965; Fimreite 1984; Best 1992; Scheuhammer et al. 1999; Lewis et al. 2001; Potts 2004; Butler 2005a, 2005b; Rodrigue et al. 2005). Lead exposure has been documented in doves foraging at intensive hunting or target-shooting areas (Fisher et al. 2006 Schulz et al. 2002). Species that forage primarily on seeds on the ground may have higher risk, but even bird species with very different foraging strategies, such as woodpeckers, can acquire lead - presumably by ingesting lead fragments embedded in trees or on the ground (Mörner and Peterson 1999). Of birds and mammals examined in a firearm shooting field, 33% were found to have elevated lead tissue levels and 17% to have potential subclinical or clinical lead exposure (Lewis et al. 2001). Deer are thought to ingest lead fragments on the ground at shooting ranges because of the taste of lead salts on oxidized fragment surfaces (Lewis et al. 2001).

Animals that scavenge hunter-killed carcasses are at the highest risk of encountering severely toxic concentrations of lead. A recent study by Hunt et al. (2006, 2009) evaluated radiographic evidence of lead fragments in 38 deer killed by licensed hunters using center fire rifles with lead-based copper jacketed, soft point bullets in Arizona from 2002 to 2004. Metal fragments were found to be broadly distributed along wound channels, 94% of samples of deer killed with lead-based bullets contained fragments and 18 out of 20 (90%) offal piles contained lead fragments. The authors concluded that the data demonstrated a high potential for scavenger exposure to lead. Meanwhile, the carcasses and gut piles from deer killed by non-lead copper expanding bullets (or "Xbullets") showed little evidence of fragmentation (Hunt et al. 2006, 2009).

Reports from experimental and field observations conclude that all bird species would be susceptible to lead poisoning after ingesting and retaining shot in the gastrointestinal system (Fisher et al. 2006). Raptor and scavenger species that feed on animals killed with lead ammunition would be at high risk for exposure to lead in this way. Animals that consume lead particles that have fragmented in hunter-killed carcasses may be at particular risk because the small size and irregular shape of fragments make them more absorbable in the digestive process.

Fisher et al. (2006) listed fifty-nine terrestrial bird species worldwide that have been exposed to lead from ammunition sources, including raptors, galliforms, gruiforms, columbiforms, and gulls. Vyas et al. (2000, 2001) identified lead in song birds resident on a shotgun trap and skeet range. Fisher et al. (2006) reviewed published literature on lead poisoning of 32 species of wild birds in the United States from spent lead ammunition. Documented cases of ingestion and poisoning by lead from ammunition in terrestrial birds globally include 33 raptor species and 30 species from *Gruiformes*, *Galliformes* and various other avian taxa, including ten globally threatened or near threatened species (Pain et al. 2009). Lead poisoning is of particular conservation concern in long-lived slow breeding species, especially those with initially small populations. A recent review by the Minnesota Department of Natural Resources found over 130 species of animals (including upland birds, raptors, waterfowl, and reptiles) have been reported in scientific literature as being exposed or killed by ingesting lead shot, bullets, bullet fragments, or prey contaminated with lead ammunition (Tranel and Kimmel 2009). In the United States, Kendall et al. (1996) found that upland game birds ingest substantial amounts of lead shotgun pellets and deduced that raptors must incur secondary ingestion of pellets because their prey ingested it. Rifle-shot prairie dogs and ground squirrels may contain fragmented lead particles that could be ingested by scavengers or raptors (Knopper et al. 2006; Pauli and Buskirk 2007). Kramer and Redig (1997) compiled data on more than 2,000 bald eagles, demonstrating that lead shot pellets, likely from crippled waterfowl and lead fragments in offal and unrecovered deer carcasses, were responsible for elevated lead levels in more than 98% of birds admitted to a veterinary hospital and raptor center. Recently, Church et al. (2006) and Chesley et al. (2009) linked isotopically labeled lead in California condors with rifle bullets sold in the same region, substantiating that condors were ingesting lead and were dying and suffering sublethal effects from bullet fragments.

Terrestrial birds are exposed to lead mainly through ingestion. Galliforms and doves probably ingest spent shot as grit which is retained in their gizzards, although there is considerable uncertainty as to why doves ingest lead and steel shot pellets (Schulz et al. 2002). Approximately 2.5% of hunter-shot doves contained lead shot in their digestive system, giving a rough estimate of the proportion of doves that ingest shot. A similar percentage of doves collected on fields where hunters used steel shot ingested steel shot. Estimates of the 2005 U.S. dove population are 350-600 million birds (Dunks et al. 1982; Schulz et al. 2006), and experimental studies indicate that nearly all doves that ingest shot will die as a result of this ingestion. Schulz et al. (2006) estimated that from 8.8 to 15 million doves may be killed each year from ingesting lead shot pellets. If scavengers consume these poisoned doves and secondarily consume the lead pellets, it is estimated that up to one million scavenging birds and mammals could die annually from ingesting poisoned doves alone.

Raptors and other scavenging birds are usually poisoned through ingesting lead shot or bullet fragments in dead or injured prey or gut piles (Friend 1987; Kendall et al. 1996). Common ravens have been shown to have elevated blood lead levels during hunting season due to ingestion of lead in rifle-shot big game offal piles (Hatch 2006; Craighead

and Bedrosian 2007, 2008). In Canada, upland game birds and mammals, the primary food source of many raptors, are now more likely to contain lead shot than waterfowl, as lead shot is prohibited for waterfowl hunting (Clark and Scheuhammer 2003).

D. Toxic Effects of Lead Ammunition on Wildlife

Lead has long been recognized as a poison to living organisms (Grinnell 1894; Engsted 1932; Horton 1933), with negative effects on general health, reproduction, and behavior (Ris et al. 2004). Lead was highlighted as an important cause of mortality in wildlife populations in the late 1950s, when ingestion of spent hunting lead pellets or fishing sinkers was recognized to cause death in a wide range of wild waterfowl (Bellrose 1959). Reports of poisoned wildlife have continued frequently since that time (e.g. Bates et al. 1968; Irwin and Karstad 1972; Sanderson and Bellrose 1986; Kramer and Redig 1997; Schulz et al. 2006).

It is well recognized that lead fragments can be absorbed from the gastrointestinal tract of birds and mammals, cause damage in various organs, and result in behavioral changes, significant illness, and even death depending on the amount ingested (Reiser and Temple 1981; Kramer and Redig 1997; Fisher et al. 2006).

Lead fragments or pellets ingested by birds may be rapidly regurgitated (in the pellets of raptors, for example), retained for varying periods, or completely dissolved with the resulting lead salts absorbed into the bloodstream. The likelihood of a bird becoming poisoned is related to the retention time of lead items, frequency and history of exposure to lead, and factors such as nutritional status and environmental stress (Pattee and Pain 2003). A proportion of exposed birds will die, and mortality can occur following the ingestion of just one pellet of lead shot (Pain and Rattner 1988). Ingestion of lead particles usually results in some absorption, and in cases where sufficient lead is absorbed, poisoning ensues. Lead concentrations are generally highest in the blood directly after absorption, and in liver and kidneys for days to months after absorption. Lead deposited in bone can remain for years, and reflects lifetime exposure (Pain 1996). Lead is a non-essential element and the activity of blood enzymes appears to be affected by extremely low concentrations. Other than in cases of point source contamination, high concentrations of lead in the tissues of birds result primarily from the ingestion of lead ammunition or fishing weights.

Various authors have attempted to define tissue concentrations in birds indicative of excessive lead exposure, sub-lethal poisoning and acute poisoning (Franson et al. 1996; Pain 1996), but there is no definitive consensus on “background” lead levels for wild birds. Environmental sources of lead are almost exclusively anthropogenic, with a small contribution from natural sources such as volcanoes. Lead is rarely found in nature in its elemental metal form, and the most common source is galena or PbS, which has a very low solubility in water. Wildlife can get low level exposure to lead from unknown sources, including natural accumulation in plants and ingestion by herbivores, and deposition by leaded gasoline exhaust, now attenuated with regulation. “Baseline” lead concentrations in wildlife can vary between taxa, and the diagnosis of poisoning is

usually based on signs of poisoning in combination with blood lead levels in live birds, and on tissue concentrations, sometimes in combination with evidence of exposure to lead in dead birds. For example, the Diagnostic Center for Population and Animal Health (Michigan State University, Lansing MI) defined background blood lead levels as <35 µg/dL for eagles (W. Rumbeiha, pers. comm.), while Pattee et al. (1990) defined background levels as <20 µg/dL, and Feierabend and Myers (1984) defined them as <10 µg/dL. The generally accepted blood lead levels for wild birds have been <20 µg/dL as background; 20 to <50 µg/dL indicating subclinical poisoning; 50 to 100 µg/dL indicating clinical poisoning; and >100 µg/dL representing severe clinical poisoning (Friend 1985, 1999; Franson 1996; Pain 1996; Pattee and Pain 2003). For condors, blood lead levels above 10 µg/dl, rather than 20 µg/dl, could have detrimental effects on condors and ought to be considered the beginning of toxic exposure (Fry et al. 2009). The background levels of 20ug/dl are now understood to indicate significant exposure, because animals held in captivity usually have background levels of 4 µg/dl or less (Walters et al. 2010).

A threshold toxic level for wildlife is difficult to measure because the effects on the nervous system at low doses can be subtle and difficult to detect without specific quantifiable behaviors. In addition, predisposition and susceptibility to lead can vary between individuals within a species (Pattee et al. 1981, Carpenter et al. 2003). There is probably no toxic lead threshold for any animal, as lead is a neurotoxin with no biological function. Lead salts are rarely encountered in the environment, and animals do not have well established metabolic or detoxification mechanisms to biochemically protect themselves from adverse effects of exposure. Even a minor decrease in fitness to a bird surviving in a hostile and competitive environment caused by small amounts of lead ingestion may result in a proximate death from many causes. In long-lived bird species, such as condors, eagles, and ravens, this has the potential to skew the normal age structure toward younger and non-breeding birds and negatively influence long-term population viability. As the duration of periodic and chronic exposure increases in the condor population so does the likelihood of death by lead-poisoning. It is unknown whether wildlife species sustain sublethal effects on coordination and cognitive behaviors similar to those demonstrated in humans, but it is likely that repetitive sub-lethal exposures to lead will cause permanent neurological and behavioral decrements in all species of wildlife (Canfield et al. 2003; Lanphear et al. 2003; Ris et al. 2004).

Lead is a non-specific poison affecting all body systems. Birds can suffer from both acute and chronic lead poisoning (Bellrose 1959; Redig 1985; Sanderson and Bellrose 1986; Eisler 1988; Scheuhammer and Norris 1996). Birds with acute lead poisoning can appear normal, but experience massive tissue destruction to internal organs and death within a few days (Sanderson and Bellrose 1986). Birds with chronic lead poisoning may develop appetite loss, anemia, anorexia, reproductive or neurological impairment, immune suppression, weakness, and susceptibility to predation and starvation (Grandy et al. 1968; Kimball and Munir 1971; Finley and Deiter 1978; Hohman et al. 1995).

The effects of toxicosis in birds commonly include distension of the proventriculus, green watery feces, weight loss, anemia and drooping posture (Hanzlik 1923; Quortrup and

Shillinger 1941; Redig et al. 1980; Reiser and Temple 1981; Franson et al. 1983; Custer et al. 1984; Sanderson and Bellrose 1986; Mateo 1998). Sub-lethal toxic effects are exerted on the nervous system, kidneys and circulatory system, resulting in physiological, biochemical and behavioral changes (Scheuhammer 1987). Vitamin metabolism can be affected (Baski and Kenny 1978) and birds can go blind (Pattee et al. 1981). Lead toxicosis depresses the activity of certain blood enzymes, such as delta aminolevulinic acid dehydratase, essential for cellular energy and hemoglobin production, and may impair immune function (Redig et al. 1991; Grasman and Scanlon 1995). Over longer periods, haematocrit and hemoglobin levels are often reduced. Finkelstein et al. (2010) found that sub-lethal concentrations of lead in blood (20 µg/dL), resulted in a 60% decrease in the levels of aminolevulinic acid dehydratase in condors.

As a result of physiological and behavioral changes, birds may become increasingly susceptible to predation, starvation and infection by disease, increasing the probability of death from other causes (Scheuhammer and Norris 1996). Lead can also affect reproductive success (Cheatum and Benson 1945; Elden 1954; Buerger 1984; Buerger et al. 1986). Grandjean (1976) showed a correlation between thin eggshells and high concentrations of lead in European kestrels (*Falco tinnunculus*). Lead poisoning significantly decreased egg production in captive Japanese quail, *Coturnix japonica* (Edens and Garlich 1983). In ringed turtle doves (*Streptopelia risoria*), significant testicular degeneration has been reported in adults following shot ingestion and seminiferous tubules may be devoid of sperm (Kendall and Scanlon 1981; Veit et al. 1982). Experimental studies on Cooper's hawks (*Accipiter cooperii*) showed detectable amounts of lead in eggs when adults had high levels in their blood (Snyder et al. 1973). In nestlings of altricial species, such as the American kestrel (*Falco sparverius*), body length, brain, liver and kidney weights can be depressed (Hoffman et al. 1985a), along with reduced survival and disrupted brain, liver and kidney function (Hoffman et al. 1985b).

Under some circumstances, there may be sex differences in the probability of exposure to or poisoning by lead, at least in western marsh-harriers (*Circus aeruginosus*), as significantly more females than males trapped had elevated lead concentrations, for unexplained reasons (Pain et al. 1993). Lead exposure may also reduce the likelihood of birds returning to an area to breed (Mateo et al. 1999). Locke and Friend (1992) concluded from their wide-ranging study that all bird species would be susceptible to lead poisoning after ingesting and retaining shot. All raptor species that feed on game could potentially be exposed at some time to lead ingestion, the likelihood varying according to the proportion of game in the diet, the size of game taken, the season, and the local hunting intensity (Pain et al. 1993).

Burger and Gochfeld (2000) found that chronic lead exposure resulted in delayed behavioral response time in both laboratory and wild herring gulls (*Larus argentatus*). Kelly and Kelly (2005) documented moderately elevated blood lead levels increased the risk of collision with overhead power lines for mute swans (*Cygnus olor*). Mallards (*Anas platyrhynchos*) experimentally fed lead exhibited hemolytic anemia during the first week of exposure and neurological impairment during the second week (Mateo et al. 2003). In

experimentally fed turkey vultures (*Cathares aura*) and bald eagles (*Haliaeetus luecocephalus*), lead ingestion decreased weight and muscle mass and caused blindness (Pattee et al. 1981, 2003). Blood pressure increases and renal damage have also been observed in rodents after experimental lead exposure (Victery 1988; Staessen et al. 1994). Bagchi and Preuss (2005) found that acute lead exposure had lasting effects including lowered bone density and increased blood pressure one year after exposure in laboratory rats.

In spite of the abundance of evidence that lead is toxic to wildlife, poisoning rates are not well understood. While massive die-offs are readily visible, daily losses of individual animals are more difficult to detect. This is because sick animals will often isolate themselves, and then are quickly predated upon after death. In one study, observers were given 30 minutes to discover 100 placed carcasses and only found 6 (Stutzenbaker et al. 1983). In another study in which researchers planted carcasses, over 60% of the carcasses were gone within 3 days and over 90% were gone within 8 days (Humburg et al. 1983; Stutzenbaker et al. 1983).

Sub-lethal lead poisoning may weaken raptors and leave them unable to hunt, or make them more susceptible to mortality from vehicles, power lines, and steel traps (Redig et al. 1980; Fry and Maurer 2003). It has also been suggested that raptors intoxicated with lead may suffer impaired hunting ability and may scavenge to a greater extent or be less selective in their choice of prey (Pain et al. 1993). Sampling methods to determine the exposure to lead intoxication in wildlife have inherent biases as with any wildlife health assessment in the field.

Long-lived species are particularly susceptible to bioaccumulation of lead in bone tissues, and repeated lead ingestion and accumulation in long-lived species can reduce bone mineralization, which could mean an increase in bone fragility (Gangoso et al. 2009). . Gangoso et al. (2009) found unusually high level of frequency of fractures and even leg amputations in an Egyptian vulture (*Neophron percnopterus*) population with high exposure to ingestion of lead ammunition.

The non-lethal effects of lead toxicosis may be difficult to recognize at a distance in free-ranging wild animals. Subtle neurological signs are easy to miss even in domesticated animals that can be physically examined. Wild animals that have died from or have been debilitated by lead poisoning may elude capture due to behavioral or physiological changes, or be removed from the population if lead exposure is associated with high levels of mortality (Miller et al. 1998).

Lead poisoning due to ingestion of spent shot or bullet fragments has had population-level effects for some bird species with low recruitment rates, depressed populations, or in recovery, such as the California condor, bald eagle, trumpeter swan, sandhill crane, and spectacled eider (Hennes 1983; Grand et al. 1998; Church et al. 2006).

E. Lead Ammunition Poisonings by Species

Information on lead poisoning of wildlife species in the United States from lead ammunition is detailed below. Information on lead poisoning of wildlife species in Canada and other countries is included in some instances where there is additional research on the effects and prevalence of lead toxicosis for certain species.

1. California Condor (*Gymnogyps californianus*)

The potential effects of lead ammunition in non-waterfowl hunting practices has now received national attention in part because of recent documentation of harmful levels of lead exposure in the endangered California condor population. Elevated blood lead levels in free-flying California condors have been well described (Locke et al.1969; Wiemeyer et al. 1986; Janssen et al. 1986; Pattee et al. 1990; Meretsky et al. 2000; Fry and Maurer 2003; Redig et al. 2003; Woods et al. 2006; Hunt et al. 2006; Sullivan et al. 2006; Parish et al. 2006; Church et al. 2006). Wild condors in California are captured once or twice per year, and blood samples are taken as part of an extensive lead monitoring program for the reintroduced population. Condors with concentrations below 20 µg/dl in blood are considered to have only background exposure, concentrations between 20–59 µg/dl indicate elevated exposure to lead, concentrations between 60–99 µg/dl suggest birds may be clinically affected, and levels above 100 µg/dl indicate acute toxicity (Redig et al. 1983). Fry and Maurer (2003) and Fry et al. (2009) report that since blood monitoring was implemented in California in 1997, 83% of all free-flying condors tested have had detectable exposure to lead. A recent review of medical records from captive condors at the San Diego Wild Animal Park was conducted to identify blood lead reference ranges for condors not exposed to lead sources in the wild (Dujowich et al. 2005). Among 95 captive born condors tested, all had blood lead levels below detection limits of 6 µg/dl with one exception testing at 11.0 µg/dl (Dujowich et al. 2005).

For the condors released in southern California (Ventura, Santa Barbara and San Luis Obispo counties) since 1992, blood lead concentrations were evaluated in 214 samples from 44 individuals (Hall et al. 2007). Forty-four percent (95/214) of these blood samples obtained during captures from 1997 to 2004 had lead concentrations consistent with elevated levels of exposure (>20 µg/dl), with 8% (18/214) at clinically significant concentrations and 3% (7/214) at acutely toxic concentrations (Hall et al. 2007). Seventy seven percent of the individual condors tested (34/44) showed elevated exposure; 32% of condors (14/44) had concentrations considered to be clinically significant, and 14% (6/44) had concentrations consistent with acute toxicity in at least one of their samples. Half of the individuals had elevated levels in multiple samples suggesting repeated exposure events (Hall et al. 2007). Subadults (age 4-5 years) in this cohort had higher exposure than adults classified as 6 years and older (Hall et al. 2007). Condors were found to have increased blood lead concentrations the second year after release reaching a peak 4 years post-release and then generally declining (Hall et al. 2007). Highest lead concentrations coincide with the age class most likely to forage widely, but detailed comparisons of condor movements and lead concentrations have not been done on a large scale for the California population.

The frequency of elevated blood lead concentrations reported for the condors released in southern California is higher than that observed in the condors released by the Ventana Wildlife Society in Big Sur, California. Since 1997, 33 condors released in Big Sur were repeatedly sampled to produce a total of 126 independent measurements of blood lead concentration. This group reports 21% (27/126) of samples to be above background levels, with only 3% (4/126) of samples at clinically significant levels and only 2% (2/126) indicative of acute toxicity (Sorenson and Burnett 2007). Condor lead concentrations were significantly higher in year 6 and year 8 post-release (Sorenson and Burnett 2007). Most of the birds released at Big Sur (21/33) visited southern California at some point, and for those that did so, they did this on average 2 years post release (Sorenson and Burnett 2007). The authors attributed the lower prevalence of lead exposure in the Big Sur population to their finding that out of 26 observed feeding events on wild prey, 20 were California sea lions in contrast to only 3 observations of condors feeding on deer, which were far more likely to be hunter-shot (Sorenson and Burnett 2007).

Petterson et al. (2009) collected 63 blood samples from 20 condors at Pinnacles National Monument from 2003 to 2007 and compared blood lead values before and after release. Of 63 post-release samples, 24 (38%) were above background (20–59 µg/dL), two (3.2%) were clinically affected (60–99 µg/dL), and two more (3.2%) were indicative of acute toxicity (≥100 µg/dL). Fifteen (75%) of individuals sampled were exposed at least once and eight (40%) were exposed on two or more occasions. Petterson et al. (2009) found a significant difference comparing samples collected before release and within one year after release from the same individuals, revealing that even young, inexperienced condors in this area are vulnerable to lead exposure.

A complete recount on lead intoxication events of wild and captive-reared condors in California and Arizona (1992 to 2002) was compiled by Fry in 2003 with information from the Condor Recovery Program. Currently, condors with greater than 40 µg/dl lead in blood measured with a portable lead analyzer in the field are brought into captivity for chelation treatment to reduce blood lead concentrations. Among condors released in southern California, at least 8 had been brought back into captivity and received emergency chelation therapy as of 2007 (Hall et al. 2007). Four of these birds had lead levels exceeding 180 µg/dl and were likely to have died or been severely debilitated without emergency intervention (Hall et al. 2007).

Free-flying condors are also captured once or twice per year in Arizona to measure lead exposure. Out of a total of 437 samples, 31% (137/437) had elevated lead concentrations (15-59 µg/dl), and 9% (39/437) exceeded 60µg/dl (Parish et al. 2006). Chelation therapy was administered 66 times to a total of 28 individuals from 1996 to 2005 in Arizona (Parish et al. 2006).

In addition to capture and treatment of condors found with high lead levels, management practices in both California and Arizona include supplemental feeding to reduce lead exposure. Several reports suggest that the condor mortality due to lead would be much

higher if the free-flying population were not intensively managed by supplemental feeding and chelation treatment to minimize the impact of lead exposure events (Fry and Maurer 2003; Woods et al. 2006; Pattee et al. 2006; Hall et al. 2007; Mee and Snyder 2007).

Causes of condor mortality were reviewed in detail for 41 free flying condors that died in California and Arizona between 1992 and 2002 (Fry and Maurer 2003). Of the 41 condor carcasses found and examined, lead toxicity was documented as the cause of death in 12% (5/41) (Meretsky et al. 2000; Fry and Maurer 2003). Predation (22%; 9/41) and power line electrocutions and collisions (20%; 8/41) were identified as the leading causes of reintroduced condor mortality in California and Arizona during this time frame (Fry and Maurer 2003). While power line collisions and electrocutions were particularly problematic for the early condor releases in California, mortality due to this cause has been reduced substantially in recent years since power line aversion training was instituted as part of the reintroduction program (Mee and Snyder 2007). The coarse terrain and wide-ranging movements of the condor make it difficult to find deceased animals within a timeframe that allows for the accurate determination of causes of death. Unless carcasses are recovered in fresh post-mortem condition, it can be exceedingly difficult to identify causes of death that are only recognizable by microscopic examination of tissues or laboratory analyses of samples. It can also be difficult to distinguish whether or not disease or intoxications have altered an individual's behavior and initiated a chain of events leading to trauma or some other cause of death. The exceedingly high blood lead levels reported in some free-flying condors suggest that these birds may have compromised abilities to avoid hazards. Therefore, cause of death could not be accurately determined for 20% (8/41) of recovered condor carcasses. An additional 11 condors were lost to follow up during this time period. While likely dead, these condors were never recovered.

A more recent review of the 66 condors released in southern California found that among the 34 condors that died in this area from 1992-2005, exact cause of death could be determined for 18 birds (Hall et al. 2007). Lead toxicity is believed to be the primary cause of death in 3 of these birds (17%). Ages at death for the three condors with lead poisoning were 1.7, 2.5 and 4.8 years. Condor deaths due to lead toxicity have not been reported for the 33 condors released in Big Sur, California (Sorenson and Burnett 2007). Out of a total of 26 condor deaths observed in Arizona between 1996 and 2005, at least 6 and perhaps as many as 8 (23-31%) died from lead poisoning (Woods et al. 2006; Parish et al. 2006). Mainly because of a marked recent increase in the number of lead-related mortalities in condors released in Arizona, lead poisoning is now the leading known cause of death in free-flying condors. Data presented in these reports do not include the additional 5 released condors in California and 3 condors in Arizona that have died due to lead poisoning from lead ammunition since 2007 (Peregrine Fund 2010; VWS 2010). At least 30 condors in California and Arizona have now died from lead exposure proven or thought to be from ingestion of lead ammunition fragments or lead shot, with many more deaths suspected to involve lead poisoning.

Chronic and frequent sub-lethal lead exposure for condors has been well-documented. A disturbing number of the released condors must undergo frequent chelation therapy to save their lives from lead poisoning due to continual exposure to lead. In 2006 alone, 95% of all Arizona condors had lead exposure and 70% of the Arizona population had to receive life-saving chelation treatment. In 2007 there were 50 cases of lead exposure in Arizona condors, and although data have not been released for 2008 and 2009, the Peregrine Fund reported having to treat "a large portion" of the wild Arizona condor population with chelation therapy for increased lead levels during the winter of 2008 and a "significant amount" of lead exposure to condors in the winter of 2009 (Peregrine Fund 2010). In June of 2008, 7 condors fell ill from lead poisoning in one feeding event near or on Tejon Ranch, the highest lead exposure event in Southern California in 10 years. In 2008 the blood lead levels of 72 free-flying condors in California were tested; from January to June, 59 percent of the condors sampled had blood lead levels that were considered above background (>10 micrograms/deciliter) levels; 45 percent of condors exhibited blood lead levels above background levels during July-December 2008 (CFGC 2009).

Lead poisoning has begun to interfere with the first breeding of released condors in the wild; one of the lead poisoning deaths in California in 2008 was a parent with a chick in the nest; in 2009 a breeding condor died of lead poisoning; and in December 2008 the first condor female to successfully hatch, and fledge, a wild-produced condor in Arizona in close to a century died of lead poisoning, as did her wild-produced offspring. In early 2010, the first California condor chick to hatch inside a national park in more than a century was severely lead poisoned, likely from eating carrion contaminated with fragments of lead bullets. The condor chick and its male parent had to be taken from the nest at Pinnacles National Monument for treatment and the chick could suffer lasting neurological damage as a result.

If lead exposure is not high enough to cause acute intoxication and death or to impair survival skills, lead should be eliminated from the body gradually through natural processes. Fry and Maurer (2003) calculated an average depuration rate (or half-time for lead elimination from blood) of 13.34±2.87 days. This finding, along with the high proportion of samples with elevated blood levels (see Fry et al. 2009), suggest that condors are frequently and repeatedly exposed to lead in the wild. The clinical consequences of recurrent lead poisoning are uncertain, but will likely result in long-term neurological injury (Fry et al. 2009). Fry et al. (2009) analyzed 469 blood samples taken from 95 different condors in California since 2000, and found that 79 of these condors (83%) have had at least one significant lead exposure incident, and some condors have been intoxicated multiple times (up to 13 times for one condor). There were 276 separate documented incidents of blood lead levels in excess of 10 µg/dL; and 27 poisonings in excess of 50 µg/dL that required emergency clinical care to prevent permanent injury or death. Similar observations of high lead levels in blood and tissues of sympatric species, such as vultures, eagles, hawks and ravens in the condor range support the conclusion that environmental lead is widely available to scavenging birds (Wiemeyer et al. 1988; Pattee et al. 1990).

Many studies have attributed lead exposure in condors to lead bullet fragment ingestion when eating hunted animal carcasses (Locke et al. 1969; Janssen et al. 1986; Meretsky et al. 2000; Fry and Maurer 2003; Hunt et al. 2006; Woods et al. 2006; Church et al. 2006). Lead from environmental sources, such as air and water pollution, may accumulate in animals, but environmental exposure is not likely to result in levels high enough to cause mortality (Pattee et al. 1990). The very high level of lead detected in most individuals of the free-flying condor population is consistent with a highly concentrated source of exposure not typically found in air, water or soil unless in an area contaminated from lead mining and smelting activities.

Condors are exclusively carrion feeders, and the condor diet includes deer, sea lions, whales, squirrels, rabbits, skunks, coyotes, pigs and cattle. The relative proportion of these various components in condor diet is very hard to assess given the difficulties involved in directly observing condor feeding behavior in the wild. Observed condor feeding behavior in southern California, although sporadic, has most commonly involved deer and cow or calf carcasses (Hopper Mountain NWR unpublished data). Intensive monitoring of the condors released in Arizona has resulted in the documentation of condors feeding on 78 deer, 42 elk, 10 coyotes, 51 domestic livestock and 16 miscellaneous animals (Hunt et al. 2007). Condors released in Big Sur are the only population with a diet that includes marine mammals. Out of 26 feeding observations made on condors released in Big Sur, 77% involved sea lion carcasses and only 15% involved deer and elk carcasses (Sorenson and Burnett 2007). In 1984, Wiemeyer et al. evaluated environmental contaminants including biologically incorporated lead in condor prey species, testing muscle, fat and placenta from cattle, sheep and mule deer (*Odocoileus hemionus*). Lead levels in these potential food items were low in all but one muscle sample from the head of a hunter killed deer (17.5 parts per million, ppm) and one cattle placenta sample (1.82 ppm; Wiemeyer et al. 1984).

Deer killed by hunters, predation, vehicular collisions, fire and disease are potential food sources for condors. Condors have been directly observed feeding on deer killed by hunters, and there are several observations of multiple condors feeding on deer offal piles in California (Hopper Mountain NWR unpublished data) and Arizona (Hunt et al. 2007). In the Kaibab Plateau in Arizona 15 of the 55 deer carcasses involved in condor foraging events were hunter-killed (Hunt et al. 2007). Animal carcasses that have been shot with lead ammunition are likely to contain fragments of lead even if the bullet passed through the carcass or if the primary shot fragment has been removed (Hunt et al. 2006). Offal piles left in the field are also very likely to contain lead fragments, since these piles usually contain thoracic organs and hunters often aim for the thorax when targeting large mammals (Hunt et al 2006).

Inter-annual variation and seasonal trends in lead exposure have been observed in all condor populations. The 44 condors released in southern California showed substantial inter-annual variation in blood lead concentrations with samples from 2001-2004 having a significantly lower mean than samples from 1997-2000 (Hall et al. 2007). This temporal trend has been explained by a move in release, food provisioning and trapping (sampling) location from the Sierra Madre Mountains to Hopper Mountain NWR in

2001. Condors trapped at the Sierra Madre site had a significantly higher mean lead concentration than condors trapped at Hopper Mountain. The Sierra Madre site is characterized by greater public access and hunting activity than the Hopper Mountain site (Hall et al. 2007). Increases in blood lead levels in condors tested during the deer hunting season have also been reported by Hall et al (2007), but sampling effort was not distributed evenly in all seasons. In fact, sampling between January and May was very limited (with only 20/214 samples in these months). This study noted that while mean lead concentration was significantly higher in condors sampled during deer hunting season, elevated lead exposure was detected at other times of the year with 38% (20/53) of blood samples collected in June having lead concentrations exceeding 20 μg/dl. Blood samples in the 33 condors at Big Sur also showed inter-annual and seasonal variation in lead concentration with samples obtained in 2005 and samples obtained in September and October showing the highest mean concentration of lead (Sorenson and Burnett 2007). This peak in lead concentrations in the Big Sur condor population does correspond with the time period in which hunter-shot deer are most prevalent in the coast range (Fry and Maurer 2003).

The studies conducted on the introduced condor population in Arizona by Hunt et al. (2006) show correlations between increased lead exposure and foraging in deer hunting areas during and just following the hunting season. Spikes in blood lead levels of condors during November and December correspond with the deer hunting seasons and condor movement to deer hunting areas (Parish et al. 2009). Since condors began foraging in the Kaibab Plateau in 2002, detected lead exposures have been temporally and spatially clustered and highly predictable (Hunt et al. 2007). Blood lead levels in condors visiting the Kaibab Plateau were significantly higher than condors not visiting this intensively hunted area, and evidence of lead intoxication in live and dead condors have peaked annually in November and December from 2002 to 2004, coincident with the deer hunting season (Hunt et al. 2007). Increased proficiency of condors at finding carrion in the wild corresponds with a greater incidence of lead exposure, and information collected on food types supports the hypothesis that lead ammunition residues in rifle- and shotgun-killed animals are the principle source of lead contamination among condors in northern Arizona and southern Utah (Parish et al. 2009). Chesley et al. (2009) evaluated lead isotopic ratios from blood samples of 47 condors in Arizona over 3 years and collected 12 metal fragments from 6 birds with elevated blood lead levels, directly linking ingested lead ammunition fragments to lead in the blood of Arizona condors. Lead poisoning has been the leading cause of death among reintroduced condors in Arizona from 1996 to 2007 (Parish et al. 2009).

Condor lead intoxications reported in both California and Arizona during non-deer hunting seasons suggest that deer hunting practices are not the only potential source of lead for condors. Firearms are used in the California condor range year-round for taking non-game animals, such as ground squirrels and coyotes, which are typically left in the field and available for scavenging species (Pattee et al.1990). Rabbits, squirrels, coyotes, and pigs shot with lead ammunition likely pose a similar risk for exposure as do hunter-killed deer carcasses, and these animals are more likely to be left in the field if they are not a food source or trophy for hunters. Condors have been observed feeding on hunted

pigs at private dumps and piles of dead ground squirrels shot for pest control (Johnson et al. 2007). Two rifle-killed coyotes were observed as a food source for condors in Arizona, and hunted coyotes have been suggested as a potential source for summer lead exposure in condors (Parish et al. 2006, Hunt et al. 2007).

Direct evidence of consumption of ammunition by condors is extremely difficult to obtain given the lag time between likely ingestion/exposure and debilitation or death. In addition, ingested lead fragments can pass through the digestive tract or be completely digested and absorbed if very small. Radiographs are unlikely to detect radio-opaque particles less than 1 mm in diameter, and similarly sized particles may be easily missed at surgery or necropsy. Nonetheless, physical evidence of ammunition inside the stomachs of individual condors that have died or have been diagnosed with high blood lead levels, have occurred in 14 cases in Arizona (Parrish et al. 2006). Of these 14 condors, 7 (3 alive, 4 dead) had shotgun pellets and 7 (6 alive, 1 dead) had spent rifle bullets in their digestive tracts either on radiographs or during necropsy (Parish et al. 2006). In California, from 1984 to 2002, 7 condors had metal detected in their gastro-intestinal tracts, but identification of fragments and analysis of samples for lead content were not performed (Fry and Maurer 2003).

Ammunition was implicated as the main source of lead exposure in released condors in recent studies by Church et al. (2006) and Chesley et al. (2009). The Church et al. (2006) study compared lead isotopic ($207^{Pb}/206^{Pb}$) ratios in blood samples from released free-flying condors in Central California and prerelease condors in Southern California; tissue samples from possible condor diet items (calves, road-killed deer and a sea lion); and samples from ammunition sold for bird and mammal hunting at stores within the condor range. The lead isotope ratios in free-flying condors differed significantly from those in captive pre-release condors providing further evidence that the sources of exposure for free-flying condors were different from the sources causing background levels of exposure in captive condors. Furthermore, the low background concentrations of lead detected in non-hunter killed condor diet samples had isotopic ratios similar to those described for environmental lead in rivers, lakes, atmospheric dust and urban aerosols. These findings provided evidence that “environmental” sources of lead, such as background levels in water, soil and non-hunter killed carcasses, were not responsible for the elevated lead exposures observed in free-flying condors in California. The lead isotopic ratios in the elevated free-flying condor samples were similar to those found in the ammunition samples tested (Church et al. 2007).

The lead isotope ratio technique has been also recently applied as a forensics tool to trace lead sources involved in condor deaths. A feather from one condor (#165) released in Arizona and found dead nearly 3 years later (June 2000) from acute lead poisoning with 16-17 shotgun pellets in its stomach was analyzed for lead concentration and lead isotope composition (Church et al. 2006). The lead isotopic ratio in the feather from this condor (in the area of the rachis and vane with most recent growth) closely matched that detected in ammunition samples from stores in California (Church et al. 2006). Further analysis of concentrations of total lead in the bones of three other dead condors (#132, 175 and 181) strongly suggest that lead poisoning or debilitation induced by lead contributed to their

deaths, but the isotopic ratio in tissues from condors #175 and # 181 were unlike those of the ammunition samples tested to date. However, the isotope ratio in tissues from condor #132 closely matched that of the ammunition samples tested in the study by Church et al (2006).

Finkelstein et al. (2010) compared blood and feather lead isotopic composition in lead-poisoned condors to spent ammunition from a recovered pig carcass fed upon by the birds to provide irrefutable evidence for exposure to lead-based ammunition. Finkelstein et al. (2010) also analyzed lead isotope ratios in 65 released California condors and demonstrated that lead in the condors (isotope ratio values of between 0.81 and 0.83) is within the range of that found in bullet samples, most of which were turned in by hunters in California. Pre-release birds had lower concentrations of lead in their blood, and their isotopic ratios were higher (0.83 to 0.85). Parmentier et al. (2009) showed that the lead concentration and isotopic composition of condor feathers examined changed following a documented ammunition-exposure event, arriving at values that matched exactly the isotopic composition of recovered ammunition.

Further lead isotopic analyses of ammunition and other potential point sources in the condor's habitat, along with bones, feathers and blood of other condors have developed a more conclusive association between ammunition-derived lead intoxications and condor mortalities. Feathers are a particularly instructive forensic tool to reconstruct lead poisoning in California condors. Finkelstein et al. (2010) used sequential feather sampling and analysis to show that feather lead concentrations can help estimate annual lead exposure risk and give better long-term monitoring of lead exposure. Sampling two or more growing primary feathers over a year will provide a time series of lead exposure and capture 60% of a bird's annual lead exposure history, compared to only 10% of an annual exposure history reflected in one or two blood samples collected over the same time period.

Parmentier et al. (2009) demonstrated from evaluating lead concentration and stable isotopic measurements in growing condor feathers that lead-exposed condors are suffering sub-clinical toxicity, based on δ-aminolevulinic acid dehydratase (ALAD) inhibition by lead. Preliminary results of nine condors with blood lead concentrations ranging between 1.9-64.0 μg/dL show a significant inverse relationship between blood lead concentration and ALAD activity, indicating significant inhibition of ALAD at blood lead levels below those where clinical chelation treatment is indicated. Church et al. (2009) showed increases in phosphorus and enzymes in condors with high blood lead levels that may be indicative of lead-induced nerve tissue damage or renal disease and kidney dysfunction.

The population-level impact of condor exposure to lead may be difficult to quantify but is clearly significant. For small populations in particular, increased adult bird mortality at any measurable rate is likely to affect population dynamics (Westemeier et al. 1998; Fisher et al. 2006). Re-introduced condor populations are currently being intensively managed to reduce lead exposure, and the proportionate mortality due to lead exposure would almost certainly be higher if individual animal interventions ceased. While

intensive management has been an important component of condor recovery efforts to date, its practicality in the long term is questionable as the condor population grows in size. More importantly, such close monitoring and frequent recapturing are counterproductive to the establishment of a behaviorally normal self-sustaining wild condor population (Condor Recovery Team, Lead Exposure Reduction Steering Committee, 2003). In order to ensure the recovery of the California condor, lead ammunition must cease being one of the species' greatest threats.

2. Bald Eagle (*Haliaeetus leucocephala*), Golden Eagle (*Aquila chrysaetos*), and Other Eagles

Bald eagles share some demographic and ecological factors with free-ranging condors that make this species vulnerable to lead intoxication; they scavenge on carcasses, they are long-lived, they have low recruitment rates, and their numbers have been reduced in recent decades (Pattee et al. 1990). Bald eagles that ingest lead shot embedded in the tissues or the intestinal tract of waterfowl demonstrate acute and chronic symptoms of lead poisoning (Hoffman et al. 1981; Miller et al. 2001). The experimental intoxication of bald eagles with lead shot conducted by Pattee et al. (1981) found that it took between 10 and 133 days (median 20 days) for mortality to occur. The range of time for lead shot retention in the stomach varied between 0.5 and 48 days. Mean lead levels in dead animals were 16.6 ppm (wet weight) in liver and 6.0 ppm (wet weight) in kidney (Pattee et al. 1981). In a complementary study, Hoffmann et al. (1981) report mean blood lead levels in eagles dosed with 10 #4 lead shot (0.21g each) to be 80 µg/dl after 24 hours and 280 µg/dl after 72 hours. Mean blood lead levels as high as 270 µg/dl have been detected in apparently healthy free-ranging bald eagles but subclinical effects may be difficult to document (Reiser and Temple 1981). Foreign bodies, including lead fragments, may be regurgitated by eagles so that fragments may not be detected in the gastrointestinal tract at the time of capture or blood tests, even if the fragments contributed substantially to elevated lead exposure levels prior to being ejected. Mateo et al. (2003) recognized the importance of accounting for this unique physiology in raptors and recommend collecting regurgitated pellets at raptor roosting sites to study the presence, frequency, seasonality and prey associated with the ingestion of lead shot.

The secondary poisoning of bald eagles by lead shot in crippled waterfowl was part of the impetus for the final decision to ban the use of lead for hunting waterfowl (Kendall et al. 1996; Kramer et al. 1997). Coon et al. (1969) reported that 7% of 45 bald eagle carcasses had high enough lead levels to be lethal. Kaiser et al. (1990) reported 9% of 158 bald eagle carcasses had elevated lead levels in the liver. In one study, 97% of bald eagles and 86% of golden eagles had elevated blood levels of lead (Harmata and Restani 1995).

Pattee and Hennes (1983) found that elevated lead levels in bald eagles corresponded well (89%) with late fall and winter waterfowl hunting seasons. However, a study attempting to trace lead poisoning in bald eagles to diet preference did not find significant differences in blood lead levels among eagles feeding on fish and eagles feeding on waterfowl in an area where waterfowl hunting was intensive (Miller et al. 1998). According to the Wisconsin Department of Natural Resources, about 15 to 20 percent of

all bald eagle deaths in that state are due to lead poisoning (Eisele 2008; Strom et al. 2009), usually from eating animals that were wounded with lead ammunition or from scavenging gut piles during and after the deer hunting season. Lead poisoning cases in bald eagles begin to increase in October, peak in December and tail off in late winter, which coincides exactly with Wisconsin's deer hunting seasons, suggesting hunter-crippled game and lead contaminated offal are the cause.

A 16 year review of lead levels in bald and golden eagles in Minnesota and Wisconsin by Kramer and Redig (1997) found that observed blood lead concentrations in both species declined following the ban on lead shot in waterfowl hunting, but there was no change in the prevalence of lead poisoning, attributable in part to continued availability of gut piles from hunter-killed deer. In that study, 21% (138/654) of eagles admitted to the centers had evidence of lead poisoning, and only one had radiographic evidence of lead fragments in the gastro-intestinal tract (Kramer and Redig 1997). Other potential sources of lead, such as fish contaminated with lead fishing sinkers, and hunting activities not included in the lead shot ban were suggested as causes for the substantial number of cases reported during this time period. Clark and Scheuhammer (2003b) found, not surprisingly, that upland game birds and mammals, the primary foods for many raptors, were more likely to contain lead shot than waterfowl 12 years after the ban on lead shot for waterfowl hunting. Lead shot from upland game hunting and lead bullet fragments from big game hunting and “varmint” shooting are a significant cause of continued lead toxicity for bald and golden eagles (Harmata and Restani 1995; Fisher et al. 2006; Hunt et al. 2006; Pauli and Buskirk 2007).

Golden eagles share some feeding ecology and behaviors with California condors and bald eagles and therefore may be exposed to some of the same factors that predispose condors to lead intoxication. In the study by Pattee et al. (1990) on the lead hazards within the California condor range, golden eagles were suggested as a model species to assess lead exposure in California condors because they are abundant in the condor range and they have been observed feeding on the same carcasses as condors. Between 1985 and 1986, 36% of the 162 golden eagles evaluated within the California condor range had elevated blood lead levels, and 2.5% had levels greater than 100ug/dl, indicative of clinical lead poisoning. This study also reported seasonal trends in lead levels in tissues of golden eagles within the California condor range which coincided with the deer hunting season (Pattee et al. 1990).

Wildlife rehabilitators in Iowa began gathering lead poisoning information on bald eagles in 2004, analyzing blood, liver, or bone samples for 62 eagles (Neumann 2009). Thirty-nine eagles showed lead levels in their blood above 0.2 ppm or lead levels in their liver above 6 ppm, which could be lethal poisoning without chelation treatment. Seven eagles showed exposure levels of lead (between 0.1 ppm and 0.2 ppm in blood samples, between 1 ppm and 6 ppm in liver samples, and between 10 ppm and 20 ppm in bone). Several of the eagles admitted with traumatic injuries showed underlying lead exposure or poisoning. Over fifty percent of the eagles being admitted to Iowa wildlife rehabilitators have ingested lead. Behavioral observations, time-of-year data analysis, and x-ray information point to lead shrapnel left in slug-shot white-tailed deer (*Odocoileus*

virginianus) carcasses to be a source of this ingested lead (Neumann 2009). Thousands of bald eagles winter in Iowa, up to one fifth of the lower 48 states population.

The Washington Department of Fish and Wildlife conducted a four-year study of golden eagles in Washington that showed increased lead levels in golden eagles.

Spring migrating eagles sampled in west-central Montana between 1983 and 1985 showed elevated blood-lead levels in 85% of 86 golden eagles and 97% of 37 bald eagles, with the source thought to be shot from waterfowl hunting and fragmented lead-core rifle bullets in ground squirrels (Harmata and Restani 1995). Domenech and Langner (2009) sampled blood from 42 golden eagles in Montana captured on migration during the fall of 2006 and 2007 and found that 58% had elevated blood-lead levels, attributed to ingestion of lead-tainted carcasses or offal piles. Of the eagles evaluated by Domenech and Langner (2009), 18 contained background lead levels of 0–10 µg/dL, 19 eagles were considered sub-clinically exposed at 10–60 µg/dL, two birds were clinically exposed (60–100 µg/dL), and three exhibited acute exposure of >100 µg/dL. Eagles with lower, but detectable blood lead levels may have had earlier exposure with the majority of the lead already deposited in other organs and bone.

Bedrosian and Craighead (2009) measured blood lead levels of 47 bald eagles and 16 golden eagles in the southern Yellowstone Ecosystem around Grand Teton National Park, Wyoming during and after large-game hunts for two years. They found a median blood lead level of 41.0 µg/dL (range = 3.2–523 µg/dL), 74.9% of all birds tested exhibited elevated lead levels (>20 µg/dL), and 14.3% exhibited levels associated with clinical poisoning (>100 µg/dL). The median blood lead levels for eagles during the hunting season was significantly higher than the non-hunting season (56.0 vs. 27.7 µg/dL, respectively; $P = 0.01$). The magnitude of lead in the blood of Wyoming eagles is extremely high and likely results in the death of some individuals (Bedrosian and Craighead 2009).

Lead poisoning is a significant cause of death in some upland-foraging raptors in Canada (such as golden eagles) that may feed on dead or wounded upland prey with embedded lead shot or bullet fragments (Scheuhammer 2009).

Kurosawa (2000) first reported lead poisoning in sea eagles in Japan, and Saito (2009) reported on 129 mortalities of Steller's sea-eagles (*Haliaeetus pelagicus*) and white-tailed eagles (*Haliaeetus albicilla*) on the island of Hokkaido, Japan from 1996 to 2007 diagnosed as lead poisoning fatalities. Necropsies and radiographs revealed lead fragments from rifle bullets and from shotgun slugs in the digestive tracts of poisoned eagles, providing evidence that a source of lead was spent ammunition from lead-contaminated Sika deer carcasses, which are a major food source for wintering eagles. Post mortem examinations of more than 390 white-tailed sea eagles (*Haliaeetus albicilla*) in Germany, an umbrella species for other scavenging birds, have shown that lead poisoning is the most significant cause (23% of mortality) of death (Krone et al. 2009). Krone (2004) also reported lead poisoning of seal eagles in Greenland. Potential sources of lead were waterfowl such as geese and carcasses of game animals or their remains (gut

piles) shot with lead-containing bullets. Captured geese and shot game animals examined by radiograph revealed embedded shot pellets and large numbers of lead particles.

3. Turkey Vulture (*Cathartes aura*) and other Vultures

While mortality due to lead exposure in turkey vultures is not well documented, dead turkey vultures sampled within the condor range have been documented as having elevated lead exposure (Weimeyer et al. 1988). Experimental lead intoxication studies in turkey vultures suggest that vultures can succumb to lead poisoning, although studies have demonstrated that turkey vultures are able to tolerate very highly elevated lead concentrations in blood. One experimental intoxication study, conducted by Carpenter et al. (2003), administered turkey vultures daily oral doses of one, three or ten BB-sized lead shot (0.35 to 0.45 grams) over a six month observation period. While most measured parameters were similar to those reported for other avian species, survival time (143 to 211 days), even at the higher level of exposure, was much longer than reported for other species, suggesting turkey vultures may be less sensitive to the deleterious effects of lead ingestion (Carpenter et al. 2003). In a separate experimental trial by Reiser and Temple (1981), one turkey vulture was more susceptible to lead intoxication than two red-tailed hawks (it is difficult to generalize to the species level and rule out individual responses with this sample size).

Additional reports on individual cases of lead toxicosis in turkey vultures have been published. Clark and Scheuhammer (2002) evaluated 184 raptors (16 different species) in Canada and the highest bone lead concentration was found in a turkey vulture, suggesting this bird was likely exposed to a series of sublethal doses of lead in carrion. Platt et al. (1999) observed histopathological peripheral neuropathy in a turkey vulture with toxic blood lead concentrations. Another case report describes a griffon vulture (*Gyps fulvus*) evaluated at a wildlife rescue hospital in Spain that was clinically ill and died after eight days of supportive care with one 0.4 gram lead shot fragment found in its gizzard (Mateo et al. 1997).

Gangoso et al. (2009) examined the sub-lethal effects of lead contamination on Egyptian vultures (*Neophron percnopterus*), comparing two populations in Spain with differing exposures to the ingestion of lead ammunition. Blood lead levels were higher in the Canary Island population than the Iberian Peninsula population, showing clear seasonal trends peaking during the Canary Island hunting season. Bone lead concentration increased with age, reflecting a bioaccumulation effect. The bone composition was significantly altered by this contaminant: the mineralization degree decreased as lead concentration levels increased (Gangoso et al. 2009).

4. Other Raptors

Lethal effects from ingestion of lead shot by predatory and scavenging raptors feeding on hunter-killed carcasses have been documented in red-tailed hawks (*Buteo jamaicensis*), northern goshawks (*Accipiter gentilis*), and great horned owls (*Bubo virginianus*). Pain

and Amiard-Triquer (1993) and Pain et al. (1993) documented exposure of marsh harriers in France.

5. Waterfowl

In North America, ducks and geese for years suffered significant losses from lead poisoning by ingesting spent lead shot on the bottom of ponds and marshes. Grinnell (1894) first reviewed lead poisoning in waterfowl and others documented poisonings shortly thereafter (Bowles 1908; McAtee 1908; Wetmore 1919; Munro 1925). It was estimated that about 1.6 to 3.9 million waterfowl died each year in North America from lead poisoning before the national ban on lead shot for waterfowl hunting in 1991 (Bellrose 1959; Feierabend 1983). Lead poisoning from spent lead shot caused an estimated 2 to 3 percent of the annual losses of North American waterfowl between 1938 and 1954 (Bellrose 1959). In Washington, prior to the ban on waterfowl hunting with lead shot, biologists reported that 4.1-4.5% of harvested waterfowl contained ingested lead shot in their digestive tracts (Jeffrey 1977; Driver and Kendall 1984), however the extent of lead poisoning in the entire Washington waterfowl population was unknown. Within six years of the ban, there was an estimated 64% decline in ingestion of lead shot by waterfowl on the Mississippi flyway (Anderson et al. 2000). Of examined ducks whose gizzards contained ingested pellets, 68% of mallards, 45% of ring-necked ducks, 44% of scaup, and 71% of canvasbacks contained only nontoxic shot (Anderson et al. 2000). Samuel and Bowers (2000) demonstrated a 44% reduction in lead exposure (defined as >0.2 ppm in blood) and of black ducks in Tennessee comparing exposure from 1986-1988 with the post-lead shot ban from 1997-1999. Similar decreases in mean bone lead concentrations in hatch-year ducklings were shown in Canada after implementation of lead shot bans (Stevenson et al. 2005). After nontoxic shot regulations for most migratory game birds were established and implemented nationwide from 1990 to 1999 in Canada, the incidence of elevated lead exposure in hatch year ducks declined dramatically, testifying to the effectiveness of the regulations and a generally high compliance by hunters (Scheuhammer 2009).

The effects of ingested lead shot on waterfowl have been well documented (Bellrose 1959; Dieter and Finley 1978; Roscoe et al. 1979; Sanderson and Bellrose 1986; Pain and Rattner 1988; Rattner et al. 1989; Pain 1996; Franson et al. 1996; Friend 1999; Pattee and Pain 2003). As few as one or two ingested lead shot pellets can cause waterfowl to waste away and die over a period of several weeks. Ducks debilitated by lead may be more vulnerable to hunting and may have impaired migratory behavior (Bellrose 1951). Lead shot ingestion rates for waterfowl are related to density of spent shot (Rocke et al. 1997).

Swans

Swan lead fatalities from ingestion of spent lead shot have been occurring since at least 1925 (Munro 1925). Some of the most dramatic examples of mass mortality of waterfowl due to ingestion of lead shot involve the deaths of thousands of wintering trumpeter swans and tundra swans in northwestern Washington state and southern British Columbia (Lagerquist et al. 1994; Degernes et al. 2006). Swan mortalities continue to regularly

occur although use of lead shot was prohibited in wetland areas over 10 years previously. The use of lead shot for waterfowl hunting was banned in Whatcom County, Washington in 1989 and Sumas Prairie, British Columbia in 1992 but lead shot continues to be permitted for upland hunting and target shooting and for hunting doves, pigeons, and American woodcock. Lagerquist et al. (1994) found that 35 percent of the 110 trumpeter and tundra swan carcasses collected and diagnosed from 1986 to 1992 had lead liver concentrations diagnostic of lead poisoning. Degernes et al. (2006) found that 81 percent of 400 trumpeter and tundra swan carcasses collected from 2000 to 2002 died from lead poisoning. Swan mortality could be high because swans can forage deeper into bottom sediments than other waterfowl, and be exposed to shot deposited years earlier. Large die-offs or consistent mortality prompt concern that lead poisoning could negatively impact populations. From 2000 to 2001, over 300 trumpeter swans died in Whatcom County from ingestion of lead shot (WDFW 2000). The 2001 population of trumpeter swans in this area was 916, and it is likely that lead poisoning is affecting the population in this area.

From 1999 to 2008, more than 2,500 trumpeter (*Cygnus buccinator*) and tundra swans (*Cygnus columbianus*) have died from lead poisoning from ingesting lead shot in northwestern Washington and southwestern British Columbia around just a single lake in (Shore 2009; Wilson et al. 2009). Swans at Judson Lake, which straddles the U.S./Canadian border in the Fraser Valley, begin to die from lead poisoning about three weeks after their arrival in November from summer nesting grounds in the Yukon. Swans arrive on the wintering grounds with low blood lead levels, but some birds subsequently become exposed to lead after ingesting lead shot (Smith et al. 2009). Some of the poisoned swans were found to have ingested more than 100 lead shot. Ingestion of only two to three pellets may cause mortality in approximately three weeks. It is unknown whether the swans are ingesting lead shot from feeding on the bottom of the lake, left over from the days before the lead shot ban, or from nearby fields where they also forage, and where lead shot is still legal to use in the hunting of doves and other upland game birds. Relatively high densities of spent lead shot occur in fields and water bodies where hunting and target shooting occur on the U.S. side of Judson Lake (Smith et al. 2009).

Wilson et al. (2009) confirmed lead shot in 80% of intact remains of dead swans from Judson Lake given toxicological testing. A multi-agency working group identified suspected sources of the lead shot, determining the type and size of shot in lead poisoned swans and measuring lead isotope ratios in lead poisoned and non-lead poisoned swans. Patterns of lead ratios in shot from suspected source areas, in shot from gizzards of lead-poisoned swans, and in liver of lead poisoned swans were compared. Swan gizzards had a mix of lead shot sizes typically used for upland game bird hunting (#6) and target shooting (#7.5–8) and predominately nontoxic shot sizes typically used for upland game bird hunting (#4–6). 56% of the lead ratios measured in the shot collected from sediment/soil fell within the range found for shot collected from the swan gizzards, indicating that swans are not consuming the whole range of shot sizes recovered from agricultural fields and water-bodies. Preliminary results are inconclusive, but Wilson et al. (2009) suspect lead shot for trap or skeet practice in areas frequented by waterfowl may be partially responsible. Two recent tests of hazing or preventing swans from using a

major roost site at Judson Lake (2006-2007 and 2007-2008) resulted in a 50% reduction in lead-related swan fatalities, compared to the average of the five previous years (Smith et al. 2009). Judson Lake is a source of lead shot poisoning swans but it clearly is not the only source.

Efforts to restore trumpeter swans in Wisconsin are also being hampered by persistent die-offs due to lead poisoning. According to the Wisconsin Department of Natural Resources, about 30 percent of all trumpeter swan deaths in Wisconsin are related to lead poisoning from ingesting lead shot or lead fishing sinkers on the bottom of water bodies (Eisele 2008). Strom et al. (2009) reported that approximately 25% of trumpeter swan fatalities in Wisconsin have been attributed to lead toxicity, and about 15% of live-sampled trumpeter swans in Wisconsin had blood lead levels above background concentrations (20 µg/dL).

Loons

According to the Wisconsin Department of Natural Resources, about 35 percent of all loon deaths in Wisconsin are related to lead poisoning, from picking up lead shot or sinkers on the bottom of water bodies (Eisele 2008). Pokras et al. (2009) demonstrated that common loons in New England ingest spent lead shot through examining lead objects found in 118 of 522 loon carcasses recovered from the six New England states from 1987 to 2000. A total of 222 lead objects were recorded from the loons' gizzards, with 11% of the items recovered being lead ammunition, primarily shotgun pellets, but also one .22 caliber bullet and one .44 –.45 caliber bullet. Evers (2004) reported that in New England, a 14-year study diagnosing causes of mortality in 522 common loons documented 44% of the breeding adults died from lead toxicosis, from either lead shot or sinkers. Substantial rates of lead-related mortality are also known for loons in Michigan and Minnesota. In New Hampshire, the ingestion of lead sinkers and jigs accounted for 40 to 71% of identified annual adult mortality during the breeding season.

Eiders

Lead shot is still used in many parts of rural Alaska for subsistence waterfowl hunting and legal use of lead shot for upland game hunting can occur in waterfowl breeding habitats. Availability of spent shot may be prolonged by permafrost, which frequently underlies wetlands used for breeding and retards the sinking of shot beyond the reach of feeding waterfowl. Lead poisoning of spectacled eiders (*Somateria fischeri*), a federally threatened species, in the Yukon-Kuskokwim Delta of Alaska, has been shown to be due to ingestion of lead shot (Franson et al. 1995; Brown et al. 2006). Radiograph studies showed that nearly 12 percent of eider adults and 2.5 percent of eider ducklings had ingested lead shot (Flint et al. 1997; Franson et al. 1998). Adult female eiders died of lead poisoning and predation, and eggs from females exposed to lead survived at much lower rates (Flint and Grand 1997; Grand et al. 1998). Mortality from lead exposure was suggested as a significant impact impeding the recovery of the local spectacled eider populations in the 1990s, which were already depressed (Grand et al. 1998). Matz and Flint (2009) analyzed blood of spectacled eiders (*Somateria fischeri*), king eiders (*S.*

spectabilis), common eiders (*S. mollissima*) and long-tailed ducks (*Clangula hyemalis*) on the Yukon-Kuskokwim Delta and the North Slope of Alaska for total lead and lead isotope ratios. Lead shot can have distinct, ore-specific signatures. Isotopic signatures from birds with relatively high blood lead concentrations were most similar to the isotopic signatures of lead shot, while signatures from birds with low blood lead concentrations closely matched those of local sediments. Lead concentrations in sediment samples were very low making sediments an unlikely source for high blood concentrations.

6. Game Birds

Lead exposure and poisoning from ingesting spent lead shot has been documented in many species of upland game birds such as chukar (*Alectoris chukar*), grey partridge (*Perdix perdix*), ring-necked pheasant (*Phasianus colchicus*), wild turkey (*Meleagris gallopavo*), scaled quail (*Callipepla squamata*), northern bobwhite (*Colinus virginianus*), American woodcock (*Scolopax minor*), ruffed grouse (*Bonasa umbellus*), and mourning dove (*Zenaida macroura*) (Campbell 1950; Damron and Wilson 1975; Best et al. 1992; Yamamoto et al. 1993; Kendall et al. 1996; Akoshegyi 1997; Keel et al. 2002; Battaglia et al. 2005; Butler 2005; Fisher et al. 2006, Schulz et al. 2006).

Mourning doves are particularly at risk for lead poisoning because they frequent and feed at high-risk habitats in terms of high concentrations of spent lead shot (Lewis and Legler 1968; Hass 1977; Kendal and Scanlon 1979a, 1979b; Kendall 1980; Burger et al. 1983; Carrington and Mirarchi 1989; Castrale 1989; Best et al. 1992; Kendall et al. 1996; Burger et al. 1997; Schulz et al. 2002). Portions of the dove populations feeding on these sites ingest lead pellets, and shot ingestion by doves increases during the hunting season ((Kendall et al. 1996; Otis et al. 2008; Franson et al. 2009). Virtually all doves that ingest pellets succumb to the direct or indirect effects of lead poisoning (Schulz et al. 2006; Schulz et al. 2007). Kendell et al. (2006) identified increased susceptibility to cold as a mortality mechanism caused by lead toxicosis in doves. Spent shot concentrations on managed dove fields have been documented as high as 348,000 pellets per acre (Best et al. 1992).

Sampling and evaluation of lead exposure of hunter-harvested doves is the usual source for estimating lead ingestion (Schulz et al. 2002, 2006) with 2.5-45.3% of doves having lead shotgun pellets in their digestive tracts. Schulz et al. (2009) suggested that doves feeding in fields hunted with lead shot that ingest multiple lead pellets may die quickly of acute lead toxicosis and become unavailable to harvest, resulting in an underestimates of lead shot ingestion rates, such as for previous studies finding relatively few doves with ingested lead shot despite feeding in areas with high lead shot availability. Schulz et al. (2007, 2009) administered lead shot to captive doves and confirmed rapid and acute lead toxicosis.

Franson et al. (2009) evaluated lead exposure in 4,884 hunter-harvested mourning doves from Arizona, Georgia, Missouri, Oklahoma, Pennsylvania, South Carolina, and Tennessee. The frequency of ingested lead pellets in gizzards of doves on hunting areas

where the use of lead shot was permitted was 2.5%. On areas where nontoxic shot was required, 2.4% of mourning doves had ingested steel shot. Doves without ingested lead pellets had lower bone lead concentrations in areas requiring the use of nontoxic shot than in areas allowing the use of lead shot. Schulz et al. (2006) calculated from comparing hunting statistics and population estimates that approximately as many doves are poisoned lethally by lead shotgun pellets (8.8 million to15 million per year) as are shot by sport hunters on an annual basis. The number of mourning doves harvested in the U.S. is approximately 20 million birds annually.

Bingham et al. (2009) documented ingestion of lead pellets by hunter-harvested chukars in four counties in western Utah, finding ingested lead-pellets in 8.74% of gizzards from 286 birds. Toxicology results show elevated concentrations of lead (>0.5 ppm, ranging from 0.7 to 42.6 ppm) in 50 bird livers (14%). The arid, rocky, and alkaline nature of chukar habitat which reduces pellet settlement and dissolution, and the similar appearance of lead pellets to chukar food sources leads to ingestion of lead pellets by chukars.

American woodcock are exposed to lead on their breeding grounds in Wisconsin, resulting in high accumulations of lead in bone tissue (Strom et al. 2009). Bone lead concentrations considered to be toxic in waterfowl were observed in all age classes of woodcock. Although stable isotope analysis of bone samples was not able to conclusively identify the source of the lead, the data suggest a local and dietary source (Strom et al. 2009).

The frequency of elevated lead accumulation in some hatch year upland game birds (such as Hungarian partridge) in Canada can be comparable to that experienced by hatch year ducks prior to nontoxic shot regulations.

7. Cranes and Rails

A number of gruiformes have been shown to ingest lead shot, including greater sandhill cranes (*Grus canadensis tabida*), American coots (*Fulica americana*), clapper rails (*Rallus longirostris*), king rails (*Rallus elegans*), Virginia rails (*Rallus limicola*), and sora (*Porzana carolina*) (Jones 1939; Kennedy et al. 1979; Windingstad et al. 1984; Franson and Hereford 1994; Wingstad 1998; Fisher et al. 2006). The consequences of poisoning incidents for the critically endangered Mississippi sandhill crane (*Grus canadensis pulla*) could be considerable, given a population that has only recently grown to about 100 individuals (Johnsgard 1983; LaRoe et al. 1995). Whether endangered whooping cranes (*Grus americana*) ingest lead pellets during their migration across Canada and the U.S. is unknown.

8. Corvids

Scientists tested blood lead levels in 302 ravens that scavenged on hunter-killed large ungulates and their offal in and around Grand Teton National Park, Wyoming in 2004 and 2005 (Craighead and Bedrosian 2007, 2008). Blood-lead levels of ravens increased

dramatically during hunting season, roughly five times higher than the rest of the year, likely due to ravens consuming lead bullet fragments left behind in gut piles of hunted elk, deer, and moose. Blood samples were taken during a 15-month period spanning two hunting seasons, from mid-September 2004 to mid-December 2005. Forty-seven percent of the ravens tested during the hunting season exhibited elevated blood lead levels (≥10 µg/dL) while only 2% tested during the non-hunting season exhibited elevated lead levels. Offal is the primary food source of ravens during the time of exposure and Craighead and Bedrosian (2007) also identified un-retrieved offal piles of hunter-killed game as a point source for lead contamination in the area. These substantial increases in blood-lead levels correspond almost exactly with the open and close of hunting season. Just after the start of hunting season, blood-lead levels begin to rise. Shortly after the end of hunting season, they return to normal. Blood-lead levels show a spike again in the late spring, when melting snow uncovers gut piles left from the previous hunting season. 100 percent of the ravens at the study site feed on gut piles at some point throughout the hunting season and get exposed to lead.

Craighead and Bedrosian (2009) collected an additional 237 blood samples from ravens in the same study area spanning an additional two hunting seasons. The samples had a median blood lead level of 10.0 µg/dL with a range of 2.7–51.7 µg/dL. The median blood lead level of 84 additional samples collected during the non-hunting season was only 2.2 µg/dL with a range of 0.0–19.3 µg/dL. Fifty percent of the hunting season samples had blood lead levels >10µg/dL, while only 3% were greater than 10µg/dL during the non-hunting season.

Craighead and Bedrosian also documented that the blood lead levels of ravens around Grand Teton dropped corresponding with increased use of non-lead ammunition by hunters on the National Elk Refuge and in Grand Teton National Park. In fall of 2009 researchers distributed 194 boxes of copper bullets to hunters with permits for the park and the refuge, captured 46 ravens (which typically scavenge the discarded gut piles) during hunting season and tested their blood for lead. An estimated 24 percent of hunters in the area used copper bullets in 2009, and there was a corresponding 28 percent drop in blood lead levels in ravens compared with what would have been expected (Hatch 2010).

Research has yet to be done on lead exposure to magpies, which occasionally feed on carrion and could also ingest lead by eating hunter-killed carcasses.

9. Song Birds

Lead poisoning from ingested spent lead ammunition has been documented in several songbird species in the United States, including white-throated sparrow (*Zonotrichia albicollis*), dark-eyed junco (*Junco hyemalis*), brown-headed cowbird (*Molothrus atar*), yellow-rumped warbler (*Dendroica coronata*), brown thrasher (*Toxostoma rufum*), and blue-headed vireo (*Vireo solitarius*) (Vyas et al. 2000, 2001; Lewis et al. 2001).

10. Mammals

Elevated levels of lead have been found in several species of small mammals near shooting ranges, such as shrews, mice, voles, and squirrels, hares, opossums, and raccoons (Tataruch and Onderscheka 1981; Erickson and Lindsey 1983; Ma 1989; Stansley and Roscoe 1996; Lewis et al. 2001). It was not determined whether elevated blood lead levels were due to direct ingestion of lead particles, or whether plants growing on lead-contaminated soil bioaccumulated lead, which was ingested by the herbivorous mammals. Woolf et al. (1982) identified lead in liver of wild white-tailed deer (*Odocoileus virginianus*).

Ingestion of lead by carrion scavenging mammals, such as coyotes, grizzly bears, black bears, wolves, wolverines, and mountain lions feeding on varmint carcasses, and gut piles and carcasses of big game during the hunting season has rarely been studied. Large carnivores such as black bears (*Ursus americanus*), grizzly bears (*U. arctos)*, wolves (*Canis lupis),* and coyotes (*C. latrans)* scavenge to varying degrees on ungulate offal piles abandoned by hunters. Cougars (*Puma concolor*) may periodically be exposed to lead at biologically significant levels because of the tendency to occasionally scavenge. Rogers et al. (2009) have begun collecting samples of liver, hair, blood, and feces from black and grizzly bears, wolves, coyotes, and cougars in Grand Teton, Wyoming, and tested samples for the presence of lead. Rogers et al. (2009) documented elevated lead blood levels in grizzly bears during hunting season, when they scavenge the remains of big game. Preliminary data by Rogers et al. (2009) showed that of 13 Grand Teton grizzly bears sampled during hunting season, 46 percent showed elevated blood lead levels above 10 μg/dl, while 11 bears sampled outside of hunting season had undetectable lead in their blood. The potential consequences for large mammalian scavengers are as yet unstudied.

11. Amphibians and Reptiles

A few studies have found elevated concentrations of lead in tissues of amphibians and reptiles near shooting ranges and heavily hunted areas (Stansley and Roscoe 1997; Stansley et al. 1997; Hammerton et al. 2003; Pattee and Pain 2003), with exposure presumed to be due to ingestion of lead in food items or dissolved in water, although ingestion of small lead fragments may be possible. Lead poisoning has been shown to cause mortality and to impact egg and tadpole development and growth rates in amphibians (Dilling and Healey 1926; Kaplan et al. 1967; Khangarot et al. 1985; Perez-Coll et al. 1988; IPCS 1989; Stansley et al. 1997; Rice et al. 1999; Sparling et al. 2006).

American alligators have been documented ingesting lead bullets after feeding on nutria that had been shot (Camus et al. 1998).

F. Toxic Effects of Lead Sinkers and Fishing Tackle on Wildlife

Waterbirds are lead poisoned from ingesting lead fishing sinkers or jigs lost by anglers on the bottom of water bodies. Sport anglers attach lead weights to fishing lines to sink the hook, bait, or lure into the water. Some anglers use lead-weighted hooks, called jigs. A sinker or jig can accidentally detach from a line and fall into the water or the hook or line may become tangled and the line may break or be cut. Aquatic birds may ingest lead objects while collecting gizzard stones or by preying on live bait or escaped fish with attached fishing gear. Many ducks and other water birds forage for food in the mud at the bottom of lakes. Most of these birds also swallow small stones and grit that aid in grinding up their food. Some of the grit may contain lead from fishing tackle.

Since birds do not generally ingest lead fishing weights greater than 2 ounces, the greatest hazard to water birds from lead fishing tackle seem to be the smaller weights used by sport anglers (Scheuhammer and Norris 1995). However, Franson et al. (2003) found a pyramid sinker weighing 2.75 ounces in a common loon and found 5 sinkers in other water birds greater than 25 mm in diameter. Observed sizes of lead objects in the gizzards of waterfowl may be somewhat smaller at necropsy than at the time they were first ingested, due to the grinding action of the gizzard and the presence of small stones against which lead objects are abraded. Birds such as loons may ingest fishing weights while ingesting bait attached to tackle (Franson and Cliplef 1992; Stone and Okoniewski 2001; Evers 2004). Once ingested, lead objects retained within the ventriculus of birds will be abraded and will be partially dissolved by acid in the digestive tract, and absorbed into the blood with potentially toxic effects (IPCS 1989; Scheuhammer and Norris 1995, 1996; NCM 2003; Scheuhammer et al. 2003b; Pokras et al. 2009).

Lead fishing sinkers and jigs are documented to cause lead poisoning mortality in numerous species of water birds and wading birds, and the problem is particularly acute for mute swans (*Cygnus olor*), whooper swans (*Cygnus cygnus*), trumpeter swans (*Cygnus buccinator*), sandhill cranes (*Grus canadensis,*), Canada geese (*Branta canadensis*), mallards (*Anas platyrhynchos*), brown pelicans (*Pelecanus occidentalis*), and common loons (*Gavia immer*) (Locke et al. 1982; Windingstad et al. 1984; Blus et al. 1989; Pain 1992; Pokras and Chafel 1992; USEPA 1994; Scheuhammer and Norris 1995, 1996; Daoust et al. 1998; Friend 1999; Stone and Okoniewski 2001; Franson et al. 2003; Sidor et al. 2003).

Loons

Common loons and pelicans are known to ingest lead objects more frequently compared to 26 other species of water birds sampled across the United States (Franson et al. 2003). Franson et al. (2003) found that 11 of 313 common loons brought sick to rehabilitation centers or live-trapped had ingested lead fishing weights, including split shot, jig heads, and a pyramid sinker. Franson and Cliplef (1992) reviewed records of 222 dead loons examined between 1976 and 1991 and found that 14 died of lead poisoning, with lead fishing weights found in the stomachs of 11 of these loons. Waterbirds trapped when

apparently healthy rarely show evidence of lead sinker ingestion (Franson and Cliplef 1992).

Lead poisoning from ingesting lead fishing sinkers or jigs is a frequent cause of death for adult common loons in the northeastern United States, accounting for about 50 percent of mortality of examined dead loons, and is the single most important mortality source in for loons new England (Pokras et al. 1992). Locke et al. (1982) reported a common loon poisoned that died from ingesting lead fishing sinkers in New Hampshire; Pokras et al (1992) examined 60 dead loons collected from 1989 to 1992 and 27 (45%) had ingested lead sinkers; Pokras and Chafel (1992) found that 16 of 31 (52%) dead adult loons collected from 1989 to 1990 ingested lead sinkers; Stone and Okoniewski (2001) found that lead poisoning caused 21% of the deaths of 105 common loons examined from new York between 1972 and 1999; and Sidor et al. (2003) examined 254 dead or moribund and found that 44% died of lead poisoning. Evers (2004) reported that in New England, a 14-year study diagnosing causes of mortality in 522 common loons documented 44% of the breeding adults died from lead toxicosis. Substantial rates of lead-related mortality are also known for loons in Michigan and Minnesota. In New Hampshire, the ingestion of lead sinkers and jigs accounted for 40 to 71% of identified annual adult mortality during the breeding season.

Pokras et al. (2009) quantified the size, mass, and types of lead fishing gear regularly ingested by common loons, collecting loon carcasses from the six New England states between 1987 and 2000 and submitting them for necropsy. Of the 522 loon carcasses examined, 118 (22.6%) had ingested lead objects, and 73 of these 118 loons, 73 had more than one object in their gizzard, for a total of 222 lead objects recorded. Lead sinkers (48%) were the most frequently ingested object, followed by jigheads (19%), split shot (12%), ammunition (primarily shotgun pellets), lead wires or tapes, and unknown items. Fifty percent of loons with ingested shotgun pellets had either two or three such projectiles present. About 36% of loons with ingested lead had other fishing-related objects (mostly hooks, swivels and monofilament line) present in the gastro-intestinal tracts. All loons ingesting lead objects also had elevated liver lead levels consistent with lead poisoning. Of the 222 lead objects ingested, 94% weighed less than 10 g and the largest object weighed 25 g. Ninety-four percent of the lead objects were less than 25.4 mm in length; 44% had a length of less than 10 mm.

Lead poisoning from fishing weight ingestion was the leading cause of deaths diagnosed for common loons in eastern Canada from 1983 to 1995, in areas where loon breeding habitats overlap with sport fishing (Scheuhammer et al. 2003b). (Scheuhammer 2009) concluded from a review of available data that ingestion of small lead sinkers or jigs accounts for about 20–30% of recorded mortality of breeding adult common loons in Canada in habitats that experience high recreational angling activity. Daoust et al. (1998) found that 5 of 31 common loons collected with ingested fishing weights from the Maritime Provinces in Canada between 1992 and 1995 had died of lead poisoning.

Ensor et al. (2002) found 17% of loons examined in Minnesota died of lead poisoning; Franson and Cliplef (1992) reported lead poisoning in 7 of 77 common loons from

Minnesota and 2 of 17 loons from Wisconsin; and lead fishing tackle accounted for 52 percent of mortalities among adult and immature loons in New Hampshire from 1976 through 2000, by far the largest single cause of adult loon mortality in the state (Loon Preservation Committee). (Strom et al. 2009) reported that approximately 30% of dead loons in Wisconsin submitted for necropsy since 2006 were found to be lead-poisoned, and lead fishing gear was recovered from the gastrointestinal tracts of loons in all cases where lead toxicity was a major contributor to the cause of death.

Swans

Lead poisoning from ingesting lead fishing sinkers was documented as the cause of death for 4 of 18 trumpeter swans examined in Montana, Idaho and Wyoming from 1976 to 1987 (Blus et al. 1989).Locke and Young (1973) reported the lead poisoning related mortality of a tundra swan that ingested a lead sinker. A study in Britain found that lead poisoning from ingestion of fishing weights was the leading cause of death (up to 90%) for declining mute swans (Simpson et al. 1979; Birkhead 1982; Birkhead and Perrins 1985; Kirby et al. 1994), a trend which reversed when a ban on small lead fishing weights was implemented in Britain in 1987 (Delaney et al. 1992; Owen 1992; Kirby et al. 1994; Perrins et al. 2003; Kelly and Kelly 2004). Continued mute swan mortality in the region is thought to be from ingestion of lead weights lost prior to the ban or during illegal use after the ban (Perrins et al. 2003).

Cranes

Two sandhill cranes diagnosed with lead poisoning died after ingesting lead fishing weights (Windingstad et al. 1984) and an endangered Mississippi sandhill crane died of lead poisoning with an unidentified lead object in its gizzard (Franson and Hereford 1994).

Other Birds

Other species reported to ingest lead sinkers include redheaded ducks (*Aythya americana*), pochard (*Aythya ferina*), greater scaup (*Aythya marila*), wood ducks (*Aix sponsa*), black ducks (*Anus rubripes*), red-breasted mergansers (*Mergus serrator*), white-winged scoters (*Melanitta fusca*), double-crested cormorants (*Phalacrocorax auritus*), white pelicans (*Pelecanus erythrorhynchos*), great blue herons (*Ardea herodias*), snowy egrets (*Egretta thula*), great egrets *(Ardea alba*), black-crowned night-herons (*Nycticorax nycticorax*), white ibis (*Eudocimus albus*), laughing gulls (*Larus atricilla*), herring gulls (*Larus argentatus*), royal terns (*Sterna maxima*), and bald eagles (Mudge 1983; USEPA 1994; Scheuhammer and Norris 1995; Friend 1999; Franson et al. 2003; Scheuhammer et al. 2003b).

Reptiles

Borkowski (1997) documented a snapping turtle that ingested a lead fishing sinker, resulting in elevated blood lead, and there are reports of turtles suffering from lead toxicosis after ingesting lead fishing weights (Scheuhammer et al. 2003b).

G. Toxic Effects of Lead Ammunition and Fishing Tackle on Humans

The toxic effects of lead on humans have been known since Roman times (Nriagu 1983; Needleman 1999; Hernberg 2000; Tong et al. 2000; Nriagu 2009). Lead is an extraordinarily toxic element, and when ingested it attacks organs and many different body systems, including the blood-forming, nervous, urinary, and reproductive systems (USDHHS 1999). The effects of lead poisoning can include: damage to the brain and central nervous system; kidney disease; high blood pressure; anemia; and damage to the reproductive system, including decreased sex drive, abnormal menstrual periods, impotence, premature ejaculation, sterility, reduction in number of sperm cells, damage to sperm cells resulting in birth defects, miscarriage, and stillbirth, painful gastrointestinal irritation, diarrhea, loss of appetite, weakness and dehydration, nerve disorders, memory and concentration problems, muscle and joint pain (USDHHS 1999). In large enough doses, lead can cause brain damage leading to seizures, coma, and death (USDHHS 1999). Chronic overexposure to low levels of lead can cause health impairments to develop over time, and irreversible damage can occur without obvious symptoms (USDHHS 1999). Lead exposure can adversely affect the nervous system (resulting in impaired cognition, reduced motor coordination, and palsy), renal system, and cardiovascular system (IPCS 1977; Needleman et al. 1990; Goyer 1996; Needleman 2004; Khan 2005). Lead is especially dangerous to fetuses and young children and poisoning is even more pronounced because the lead is absorbed faster and disrupts development, causing slow growth, development defects, and damage to the brain and nervous system. Some studies link elevated bone or blood lead levels with aggression and delinquent behavior and attention deficit hyperactivity disorder (Nevin 2000; Needleman et al. 2002; Needleman 2004; Braun et al. 2006).

In humans, blood lead concentration of 10 micrograms of lead per deciliter is currently considered an elevated level, although some researchers and health professionals have advocated for a threshold of 5 micrograms or even 2 micrograms. A blood lead level of 60 micrograms per deciliter in a human would require immediate medical attention. Lead accumulates in humans mainly in bones, with lead in blood and other tissues reflecting more recent exposure.

Human exposure to lead in the United States has decreased as lead plumbing, paint, solder, toys, and gasoline have been phased out and replaced. Public health agencies have regulated lead in industrial activities and consumer products, and have to varying degrees begun to address and remediate lead exposure from shooting ranges, but have focused little attention on hunting or fishing activities that may be an important source of lead exposure in certain communities, occupations, or activities.

Hunters who use lead bullets are at risk of lead poisoning in several ways. One exposure mechanism is inhalation of airborne lead created by friction from lead slugs against the gun barrel (KDHE 2004), whereby inhaled lead enters the bloodstream and is distributed throughout the body. Hunters who handle lead bullets are also at risk of ingesting lead residue (KDHE 2004)**.** The most serious exposure is from accidental ingestion of lead shot pellets or lead bullet fragments in the meat (Carey 1977; Tsuji et al. 1997, 1999; Scheuhammer et al. 1998; Johansen et al. 2001, 2004, 2005; Bjerregaard et al. 2004; Mateo et al. 2007). Health effects in human beings following ingestion of whole lead shot pellets have been reported in many cases, and ingestion of meat tissues containing minute flakes or fragments of metallic lead from the passage of lead shot or lead bullet fragments through the tissues is also possible (Scheuhammer and Norris 1995; Khan 2005).

Published literature on lead concentrations and lead isotope patterns from subsistence hunters in the circumpolar North indicates that elevated human lead exposure is correlated with use of lead ammunition (Verbrugge et al. 2009). The mechanisms of exposure include ingestion of lead dust, ammunition fragments, and shot pellets in harvested meat, and inhalation of lead dust during ammunition reloading. Epidemiological studies and risk assessment modeling indicate that regular consumption of game meat harvested with lead ammunition and contaminated with lead residues may cause relatively substantial increases in blood lead compared to background levels, particularly in children (Kosnett 2009). A Canadian study of blood lead levels in hunters (Nieboer 2001) showed that lead pellets from wild game harvested with lead shot is a major source of exposure to lead in Native American communities in Canada. Blood lead levels were demonstrated to be higher in Native hunting communities than in a nearby reference group. Blood lead levels were also higher in men than women, consistent with greater participation of males in hunting and greater consumption of bagged wild fowl. Blood lead levels were shown to increase in male hunters during the hunting season, and one of the measured lead isotope ratios also changed in a manner consistent with exposure to lead derived from leaded ammunition. Of 132 subsistence hunters radiographed, 15% showed ingested lead pellets, with 8% located in the lumen of the digestive tract and 7% in the appendix (Tsuji and Nieboer 1997). Fifteen recent studies in Canada, Greenland, and Russia have linked lead shot found in game animals to higher levels of lead in people who eat those game animals (Carey 1977; Tsuji et al. 1997, 1999; Scheuhammer et al. 1998; Johansen et al. 2001, 2004, 2005; Bjerregaard et al. 2004; Mateo et al. 2007; Tranel and Kimmel 2009). Studies showing significantly higher lead exposure in people from hunting communities have major implications for the public health hazards of lead in ammunition (Dewailley et al. 2001; Levesque et al. 2003).

In Alaska, ammunition-related lead exposures include ingestion in shot game, use of certain indoor firing ranges and melting and casting lead to make bullets. Titus et al. (2009) quantified the population of Alaska at potential risk of lead exposure from eating game shot with lead ammunition. Alaska has 84,000 licensed resident hunters, many of whom rely on wild game for a significant part of their diet. About 29,000 hunters kill about 7,300 moose annually in Alaska. In rural Alaska, where reliance on ungulate meat is high, about 100 kg of moose and caribou meat is consumed per person annually, and small game, marine mammals, and waterfowl harvested with firearms also contribute to

the local diet. Sixty percent of households in rural Alaska harvest game animals and 86% consume wild game.

A study of lead concentrations in tissues of waterfowl killed by shotgun (Frank 1986) showed high amounts of lead (>100 mg/kg) and confirmed the presence of lead fragments by X-ray. Particles of lead ranged from irregular fragments 1–2 mm in length to very fine dust, resulting from the disruption of lead shot pellets upon collision with bone (Frank 1986). Researchers have also detected lead fragments visible by radiograph in carcasses of squirrels shot with bullets (Harmata and Restani 1995; Knopper et al. 2006). The flesh of any species of game animal killed with lead shot or lead bullets can become contaminated with high concentrations of lead through this mechanism. Studies have demonstrated that lead bullets can shatter into hundreds of fragments when fired from a high-powered rifle (Hunt et al. 2009b; Cornicelli and Grund 2009). Bedrosian and Craighead (2009) showed extensive fragmentation of lead bullets in an elk carcass shot with a .30-06 rifle. In an X-ray of the results, lead fragments appear as white shards spread throughout a large area in the elk's body. Hunt et al. (2009b) found that lead fragments in shot game spread far beyond the internal organs and can move into the meat that humans eat. X-rays of meat from a butchered game animals showed bullet fragments in steaks packaged for human consumption. While most big-game hunters discard "blood-shot" meat that's been pierced by bullet fragments, the California research shows that fragments can be packaged even by experienced butchers.

A recent study by the Minnesota Department of Natural Resources found that when lead bullets explode inside an animal, imperceptible dust sized particles of lead can infect meat up to a foot and a half away from the bullet wound (Cornicelli and Grund 2009). Cornicelli and Grund (2009) conducted a radiograph study of bullet fragmentation patterns in carcasses to determine the potential risk of lead contamination of deer meat in the Minnesota venison donation program. The study assessed lead levels in deer and domestic sheep shot using different types of bullets and firearms commonly used for hunting in Minnesota, including: a centerfire rifle with lead bullets designed to rapidly expand upon impact used for hunting mid-sized game such as deer, lead bullets designed to retain a high percentage of their weight, and non-lead copper bullets; a shotgun using a 1-ounce Foster lead slug, commonly used throughout the Minnesota shotgun-only zone; and an inline muzzleloader with two common bullets types used during Minnesota's hunting seasons.

Cornicelli and Grund (2009) showed that using bullets with no exposed lead (a copper case completely surrounds the lead core) or bullets made of copper significantly reduce (or eliminate) lead exposure. Non-exposed lead core bullets averaged nine copper fragments in the animal with an average maximum distance from the wound channel of seven inches. By design, copper bullets leave no lead and the few copper fragments that were seen on x-ray were less than an inch from the exit wound. Both of these bullet designs fragmented very little and left no lead. Ballistic tip lead bullets (rapid expansion) had the highest fragmentation rate, with an average of 141 lead fragments per carcass and an average maximum distance of 11 inches from the wound channel. In one carcass, a lead fragment was found 14 inches from the exit wound. Soft point lead bullets (rapid

expansion) left an average of 86 lead fragments at an average maximum distance of 11 inches from the wound channel. Bonded lead-core bullets (controlled expansion, exposed lead core) left an average of 82 lead fragments with an average maximum distance of nine inches from the wound. Lead shotgun slugs left an average of 28 lead fragments at an average maximum distance of five inches from the wound channel. Muzzleloader bullets (245-grain and 300-grain respectively) left an average of three and 34 lead fragments, respectively, at an average maximum distances of one and six inches, respectively. Lead fragments were found so far from exit wounds that routine trimming likely would not remove all of the fragments. Only about 30 percent of fragments were within two inches of the exit wound, and the vast majority were dispersed further from the carcass. In some cases, low levels of lead were detected as far away as 18 inches from the bullet exit hole. Rinsing of a carcass produced mixed results, tending to reduce lead around the wound channel but also transporting lead away from the wound. Lead ammunition shot into the hindquarters of a deer, where heavy bones are found, resulted in extensive fragmentation so pronounced that a hunter would likely not want to utilize this meat as there would be no way to remove all the fragments. Having venison processed at a meat processor will likely result in an increased risk of lead exposure because venison from different hunters is typically mixed during the grinding process and the vast majority of hunting bullets are made from lead. Cornicelli and Grund (2009) found that 27% of the ground venison and 2% of the whole muscle cuts tested had detectable lead fragments.

In a highly publicized recent case, packets of venison shot with lead ammunition and donated by hunters to feed the hungry tested positive for lead contamination. Cornatzer et al. (2009) studied 100 randomly selected ground venison packages donated to the Community Action Food Centers of North Dakota by hunters. The packages were studied by high resolution computerized tomography imaging and x-ray fluoroscopy for detection of metal fragments. Analysis of randomly selected ground venison samples showed 59 packages out of 100 had one or more visible lead fragments. One sample had 120 ppm lead. Cornatzer et al. (2009) concluded there is a health risk from lead exposure to humans consuming ground venison. Food banks and shelters in North Dakota pulled the meat from their shelves after the report. The Centers for Disease Control and Prevention and the North Dakota Department of Health ran a test to find out the health effects of lead-shot game. The agency compared blood-lead levels of people who regularly eat meat shot with lead bullets with the levels of those who don't eat much wild game. The results were inconclusive. Those who ate the lead-shot meat had slightly higher blood-lead levels than those who did not, but none of the 738 people in the study had levels above the government's threshold for danger. The health department recommended that children younger than 6 and pregnant women stop eating venison shot with lead bullets because those groups are at particular risk for lead poisoning, even at low levels. Avery and Watson (2009a) conducted a survey of all wild game meat donation programs throughout the United States to determine the amount of venison and other game donated annually. Venison donation programs operate in all 50 states and in at least four Canadian provinces. For the 2007/2008 hunting season 75 programs reported providing an average of 34,943 pounds of hunted game meat annually, a total of 2.6 million pounds of meat or approximately 10 million meals.

Hunt et al. (2009) radiographed 30 eviscerated carcasses of white-tailed deer (*Odocoileus virginianus*) shot by hunters with standard lead core, copper-jacketed bullets under normal hunting conditions. All deer carcasses brought to processors contained fragments (15–409 fragments counted in radiographs), and despite a high rate of removal of fragments by processors to avoid contamination, 80% were unable to do so entirely. Hunt et al. (2009) demonstrated that people risk exposure to bioavailable lead when they eat venison from deer killed with standard lead-based rifle bullets and processed under normal commercial procedures. Ten million hunters, their families, and low-income beneficiaries of venison donations in the U.S. are at risk. The evidence includes a high proportion (80%) of examined deer showing at least one bullet fragment in one or more ground meat packages, a substantial frequency of contamination (32% of all ground meat packages), a majority (93%) of assayed fragments identified as lead, isotopic homogeneity of bullet lead with that found in the meat, and increased blood lead concentrations in swine fed fragment-containing venison, meaning the lead is bioavailable to humans as well. Hunt et al. (2009) concluded that in a majority of cases, one or more consumers of a hunter-killed, commercially-processed deer will consume bullet lead.

Pain et al. (2010) found that eating the meat of animals hunted using lead ammunition can be more dangerous for health than was previously thought, especially for children and people who consume large quantities. Pain et al. (2010) analyzed the meat of six species of game birds (red partridge, pheasant, wood pigeon, grouse, woodcock and mallard) shot by hunters in the United Kingdom, and found that lead levels in cooked game meat exceeded the maximum allowances set by the European Union, due to the presence of remains of ammunition, even after lead pellets were removed. Depending on the species and type of recipe used, between 20% and 87.5% of the samples analyzed exceeded 100 parts per billion of the fresh weight of meat.

Watson and Avery (2009) assessed the numbers and proportions of state populations that hunt and may be at risk of lead exposure from lead-based ammunition, from lead handling ammunition (e.g., hunters who load their own ammunition), inhalation of vapor upon firing, or ingestion of game meat contaminated with bullet fragments and shot.. In 2006, 12.5 million people (6% of the population) aged 16 years and older in the United States hunted on 220 million days, including an estimated 1.6 million children aged 6 to 15 years.

Elevated blood lead levels resulting in biochemical effects, disease and neurotoxicity have been documented for people who frequent or work at indoor and outdoor firing ranges (Fischbein et al. 1979; Novotny et al. 1987; Chisholm 1988; Valway et al. 1989; Peddicord and LaKind 2000; Gulson et al. 2002). Exposure may be due to handling lead materials during reloading as well as inhalation of lead dust. Sportsmen who reload rifle and pistol ammunition and cast their own lead bullets are at particular risk of exposure to lead.

Watson and Avery (2009) assessed the numbers and proportions of state populations that may be at risk of lead exposure from fishing gear, from handling and making lead sinkers, and through accidental ingestion. In 2006, 30 million people (13% of the population) aged 16 years and older in the United States fished on 517 million days. As of 1994 approximately 800,000 to 1.6 million people in the United States manufactured lead fishing weights in their homes, often in enclosed garages or basements, representing approximately one-third of the lead sinkers produced in the country (USEPA 1994). Melting lead to produce fishing tackle such as lead sinkers and jigs can result in lead poisoning through inhalation of lead dust and fumes (USEPA 2004).

H. Alternatives to Lead Ammunition and Sinkers

In promulgating a rule in response to a Section 6 petition, the EPA must consider 'the benefits of such substance or mixture for various uses and the availability of substitutes for such uses" as well as "the reasonably ascertainable economic consequences of the rule, after consideration of the effect on the national economy, small business, technological innovation, the environment, and public health" (15 U.S.C. § 2605(c)(1)(C)-(D)). This petition identifies below commercially available alternatives to lead rifle bullets, rimfire bullets, shotgun pellets, fishing sinkers, and jigs containing lead. Not all products available in lead are currently available as nontoxic alternatives, but the demonstrated technology indicates that all products could be produced in nontoxic alternatives within a short period of time, if manufacturers are provided a transition period for expanding upon current designs and stocks of ammunition and fishing gear.

1. Availability, Performance, and Toxicity of Non-Lead Bullets and Shot

Stroud and Hunt (2009) reviewed basic bullet materials available to bullet manufacturers, which include lead alloys, lead with external copper wash, lead core with copper jacket, pure copper, and bismuth. Lead and bismuth are highly frangible, whereas pure copper bullets tend to remain intact after impact. Bullet fragmentation increases the degree of lead contamination in tissue ingested by scavengers feeding on hunter-killed animal remains. Modern bullet design, velocity, composition, and bone impact are significant factors in the character and distribution of lead particles in carcasses, gut piles, and wound tissue left in the field by hunters. Prior to the 1900s, bullets were made entirely of lead. Their velocities were relatively slow (<2,000 feet per second), and their tendency to fragment was accordingly lower than that of modern ammunition. Development of smokeless powder in the 1890s increased bullet speeds above 2000 feet (610 m) per second, causing lead bullets to melt in the barrels and produce fouling which reduced accuracy. Copper jacketed lead-core bullets were therefore developed, which permitted velocities that may exceed 3,000 or even 4,000 ft/sec in modern firearms. Standard hunting bullets now typically travel at 2,600 to 3,100 ft/sec, speeds highly conducive to fragmentation. Plastic-tipped "hollow-point bullets" used for varmint hunting are actually designed to completely fragment, leaving the entire mass of the lead bullet to contaminate the carcass.

Although the terms "lead-free," "non-lead" and "nontoxic" are used interchangeably, as a result of the manufacturing process, trace levels of lead can exist in any metal projectile used for bullets, including copper, resulting in ammunition that is not 100% lead-free, but that is functionally nontoxic to wildlife and humans. The Fish and Wildlife Service definition of "nontoxic" shot to be used in waterfowl hunting specifies in 50 C.F.R. 20.21(j) several alloys containing not more than 1% lead. Steel shot can be coated with metals such as zinc (which always contains lead as an impurity) as long as the coating does not exceed 1% of the weight of the shot. The California Department of Fish and Game has established a maximum amount of lead content in projectiles considered to be nontoxic at 1% by weight, given scientific consensus that this threshold for lead content will preclude risk to condors, which are typically more sensitive to lead than other taxa, from lead fragmentation. Toxicological modeling of this amount of lead impurity in bullet fragments indicates that even if condors consume major fragments of bullets, the dissolution of lead is unlikely to raise the blood lead levels above 1 μg/dL, a low level equivalent to the blood lead levels of condors being raised in Los Angeles or San Diego Zoos on a lead-free diet (Fry et al. 2009).

For all but the smallest caliber bullets (those used for varmint hunting), nontoxic ammunition is widely available. Currently available alternatives are either made completely of non-lead materials, such as copper, or designed such that a lead interior is protected from exposure upon impact. Other designs have been proposed and it is expected that the increase in demand will result in greater options of nontoxic ammunition. Non-lead bullets generally have equivalent, if not superior, performance when compared to their lead counterparts. Copper bullets were originally designed for the "premium" market not because of concerns over lead poisoning but rather for their enhanced ballistic capabilities.

Oltrogge (2009) reviewed the success of ammunition manufacturers in developing lead-free, expanding-nose centerfire bullets. The Barnes Bullet Company succeeded in 1985 in designing lead-free copper bullets that demonstrate good expansion without shedding copper particles. They have proper rotational moment of inertia, are made in traditional bullet weights, and despite the lower density, the over-all loaded cartridge lengths are within specification. These and other factors make them as capable as traditional lead-cored bullets. They are on the market as the X-Bullet series, in several varieties, chief of which are the Triple Shock and the MRX. The latter is shorthand for Maximum Range X-Bullet, which has an all-metal tungsten-composite core that is more dense than lead. It shoots further, with flatter trajectory, than any other lead-free bullet and surpasses many lead-containing bullets. Oltrogge (2009) reviewed some of the science of achieving these lead-free, centerfire bullets. Nosler and other companies are now making all-copper centerfire bullets, and availability is increasing.

Currently there are a number of lead-free copper hunting bullets produced, at least one of which—the Barnes X Bullet—is widely available. The Barnes X is made out of copper, a material that is lighter and more rigid than lead. Barnes produces a number of X-type bullets, including the X, XLC, and Triple Shock X, in a wide variety of calibers suitable for hunting game such as deer, elk, pig, and coyote. In order to promote proper

expansion, Barnes bullets are designed with a hollow point that is fluted so that the tip peels back to form a mushroom upon impact. Barnes bullets have a ballistic coefficient between .220 and .555, depending upon the caliber and cartridge used. Barnes also reports that its bullets retain close to 100% of their weight after hitting most targets. Thus, Barnes bullets are lead-free alternative ammunition that offers equivalent or superior performance to that of high-quality lead bullets.

Another alternative bullet, composed of tungsten, tin, and bismuth ("TTB") is being developed by various ammunition manufacturers and the military has been experimenting with a so-called "green" bullet that relies on the same metals to replace lead (Mikko 1999).

The California Department of Fish and Game certifies nontoxic ammunition for use while hunting big-game and non-game species in the range of the California condor in California, including deer, bear, wild pig, elk, pronghorn antelope, coyote, ground squirrels, and other nongame wildlife. Such ammunition must use a projectile or bullet which has been certified to contain ≤ 1% lead by weight. As a result of the manufacturing process, trace levels of lead likely exist in any projectile. DFG established a maximum amount of lead content in projectiles to be 1% by weight, given scientific consensus that this threshold for lead content will preclude risk to the condor from lead fragmentation. Typically, the certified nontoxic identified projectiles have far less than 1% lead content. As of April 2010, the Department had certified nontoxic ammunition from 24 manufacturers. A list of DFG approved nontoxic ammunitions can be found at http://www.dfg.ca.gov/wildlife/hunting/condor/certifiedammo.html. The Arizona Game and Fish Department also publishes a list of non-lead rifle ammunition available for big game hunters, including 120 bullets in various calibers produced by 13 ammunition manufacturers, as well as 7 manufacturers who provide custom-loaded non-lead rifle ammunition. The information can be found at http://www.azgfd.gov/pdfs/w_c/condors/Non-LeadAmmo.pdf

Both rifle bullets and .22 caliber rimfire bullets are currently marketed with non-lead alternatives. Ammunition in .22 rimfire was made available only after California required the use of nontoxic .22 ammunition in the range of California condors. Prior to that time, expert testimony was presented to the California Fish and Game Commission saying non-lead .22 caliber rimfire was impossible to produce. However, commercially available .22 caliber ammunition was available four months after the Commission decision to ban lead .22 ammunition.

In one survey, 90% of hunters and ranchers surveyed approved of the use of copper bullets (Ritter 2006). According to post-hunt survey results in Arizona, 88% of successful hunters who used non-lead ammunition said it performed as well as or better than lead bullets. In addition, 72% of all hunters said they would recommend the all-copper bullets to other hunters (Seng 2006). In general, experts appear to endorse the use of non-lead bullets (AGFD; Rees).

Nontoxic shotgun ammunition is widely available on the market, largely as the result of federal regulations requiring its use while hunting for waterfowl (50 C.F.R. § 20.134). Certification of nontoxic shot is conducted by the US Fish and Wildlife Service, and acceptable alloys must be not more than 1% lead. Shotguns, the dominant firearm used for waterfowl hunting, are also used for upland hunting of small game, such as squirrels, rabbits, and birds, and in many states are used for hunting larger game such as deer and pigs using solid slugs.

Commercially available nontoxic shotgun ammunition consists of shot composed either of steel, tungsten (including tungsten-iron, tungsten-bronze, tungsten-nickel-iron, tungsten-matrix, tungsten-nickel-iron, tungsten-tin-iron-nickel, tungsten-tin-bismuth, tungsten-tin-iron, and tungsten polymers), bismuth, or tin (WFGA 2001). It should be noted, however, that nontoxic shot is not currently available for all gauges and pellet sizes, particularly smaller shot sizes (#7½ and #8) which are popular for hunting upland game birds (WFGA 2001).

The use of nontoxic shot for hunting upland game is mandated on a variety of federal and state lands, and nontoxic shot is used by upland hunters across at least 1.33 million acres nationwide (WFGA 2001). For example, a number of individual National Wildlife Refuges require the use of nontoxic shot, as do a number of states such as South Dakota, Wisconsin, and Maine.

The performance of nontoxic shot is also roughly equivalent to that of lead shot. Non-lead shot, particularly steel, is lighter than lead and thus has reduced velocity at greater distances, whereas bismuth shot has a density almost equivalent to that of lead. Tungsten alloy shot of several compositions is superior to lead and steel shot, and can be used in double barreled shotguns and older steel barreled shotguns which would be damaged by the higher muzzle pressures created by steel shot. Tungsten alloy shot shells are currently much more expensive ($2.20-3.50 per cartridge) than either lead ($0.25-0.75 per cartridge) or steel shot ($0.40-$0.60 per cartridge).

After the federal ban on lead shot for hunting waterfowl, there were hunter complaints about the effects of non-lead shot on shotgun barrels. Older shotguns not designed for steel shot have a risk of damage to the barrels due to increased pressure of the hard steel shot which may cause “ring-bulge” deformation of the barrel at the choke of older fixed choke shotguns. Double barrel shotguns are also susceptible to barrel damage with steel shot. “Hevi-shot”®, composed of tungsten, nickel, and iron will not damage barrels of older shotguns, although it is considerably more expensive than steel shot. All modern single barreled shotguns manufactured after 1990 use interchangeable choke tubes designed for steel and other nontoxic shot, and will not be damaged by any nontoxic shot.

The U.S. Fish and Wildlife Service certifies and approves non-lead, nontoxic shot for use in waterfowl hunting (USFWS 2010). The Service has currently approved 12 nontoxic shot types. A full list can be found at
http://www.fws.gov/migratorybirds/CurrentBirdIssues/nontoxic.htm

Mandating the use of non-lead ammunition for hunting would impose some additional costs on some in the hunting community. However, the incremental cost of alternative ammunition is typically a tiny fraction of the total that hunters spend on their sport. According to the federal government, the average big game hunter in California spends just over $800 per hunting trip. Of that $800, approximately $173 dollars are spent on all "hunting equipment", with bullets representing a fraction of that cost (USDOI/DOC 2003). Likewise the cost of shot is a small portion of annual waterfowl or game bird hunting expenses (Scheuhammer and Norris 1995). The Minnesota Department of Natural Resources reports that effective nontoxic alternatives to lead shot are available, and at costs comparable to lead (Tranel and Kimmel 2009). The price of non-lead ammunition has continued to drop over time as demand has risen. As demand continues to increase and subsequent production costs fall, nontoxic bullets and shot will likely become less expensive. On a larger scale, the costs to purchase non-lead ammunition would likely be more than offset the societal costs involved in cleaning up and managing lead wastes from lead ammunition.

2. Availability, Performance, and Toxicity of Non-Lead Fishing Tackle

Fishing sinkers and jigs do not have to be made of lead. Inexpensive and ecologically sound alternatives to lead fishing weights made from non-poisonous materials such as tin, bismuth, steel, and recycled glass are available. Sinkers made of materials other than lead have gained varying levels of acceptance among anglers, with tradeoffs regarding cost and effectiveness. At least 10 substitutes for lead fishing tackle are on the market: tungsten (plastic composites and putty), stainless steel, carbon steel, tin, tin/bismuth, brass, ceramics, glass, pewter, and zinc (Scheuhammer and Norris 1995; Scheuhammer et al. 2003b; MOEA 2006). Most fishing tackle stores in the U.S. already carry alternatives to lead fishing tackle and sinkers (Scheuhammer and Norris 1995, 1996; Simpson 2001; Scheuhammer et al. 2003b; Michael 2006). Some states and non-profit organizations offer small-scale programs that exchange angler-owned lead tackle for non-lead substitutes. Fishing jigs and weights containing lead are required to carry a warning label in California (Proposition 65 warning) because lead has been identified by California as causing cancer. As a result, retailers and purchasers of fishing gear in nearly all states can currently identify gear containing lead, and can routinely avoid using lead-containing products, if they so choose. Not all states require a lead warning, but because California is a large market, most manufacturers routinely label fishing gear packages that are retailed in most states.

Metals such as bismuth and tungsten are more expensive than lead, and others such as zinc are known to be toxic to birds and other biota (Grandy et al. 1968; Zdziarski et al. 1994; USEPA 1994; Levengood et al. 1999).

Tungsten, one of the more widely used alternatives to lead fishing tackle, is sold as a tungsten-plastic composite and as tungsten putty, a specialty item marketed to flyfishers. Tungsten putty can be molded into varying shapes and sizes and affixed to fishing line, allowing anglers to vary the sink rate of their fly presentation. Tungsten is comparable to

lead in density and can be manufactured to be more dense than lead, allowing for smaller tackle. Tungsten tackle can also have noise-making attributes that may attract fish in some situations. Tungsten is more expensive than lead, and tungsten tackle requires plastic sleeves to cover sharp edges, and additional expense.

Stainless steel tackle is advertised as having fish-attracting qualities due to the noise it makes bumping along the bottom. Stainless steel tackle is larger than lead tackle of equivalent weights. Carbon steel tackle is available on the internet. Some carbon steel tackle is made from recovered waste steel mixed with resins, within a cotton sleeve. Anglers can add or subtract steel balls on a three-way swivel to adjust the sink rate to hold bait on the bottom. This gear is gaining popularity in river fisheries and steel is replacing lead in a variety of commercial traps. Iron is one of the less expensive alternatives to lead, but has the disadvantage of corroding after exposure to water.

Tin is a malleable metal that allows anglers to reuse split shot many times. The lower density of tin also allows for a slower sink rate, potentially keeping the bait in the "strike zone" longer. Tin tackle tends to be larger and more expensive than lead, but is widely available. Bismuth is a brittle metal that can be used in non-split fishing weights such as egg, worm, swivel, bullet slips, and jig heads (Scheuhammer and Norris 1995). Bismuth/tin compounds are popular among anglers who manufacture their own jigs, partly due to better paint quality on jig heads using this material. A disadvantage is that bismuth is a relatively expensive metal.

Brass fishing tackle is advertised as producing sound with fish-attracting qualities. Brass is an alloy of copper and zinc, and metallic zinc is known to be highly toxic to birds when ingested. Also, brass fishing tackle often includes lead mixed in with brass, and is not lead-free, even though the lead is bound in a state not thought to be toxic (MOEA 2006).

Fishing tackle made of glass tends to be larger and more expensive than lead. Certain types of glass can be made to "glow" after exposure to light, a quality purported to improve fish biting frequency. Glass sinkers are available primarily through the internet. Ceramic fishing tackle is also considerably larger than lead tackle.

Zinc was used as a replacement for lead sinkers until it was demonstrated that the industrial grade zinc used in the tackle was more toxic in aquatic environments than lead. Lead-free pewter tackle is another potential alternative, but pewter is not in wide use and not currently available to consumers. It is expected that pewter tackle will need to be larger than lead equivalents and more expensive.

In 1994 the EPA determined that the economic impact of switching to nontoxic fishing sinkers will be nominal (EPA 1994). Similarly, when the National Wildlife Refuge System implemented "Lead-Free Fishing Areas," they acknowledged that nontoxics sometimes cost more than lead weights but stated that as sinkers only comprise 3% of yearly equipment costs, the increase did not create a burden for anglers (Federal Regulation 50 CFR 32 and 36, proposed rule).

IV. EXISTING REGULATIONS

A. Existing Federal and State Lead Ammunition Regulations

In 1991 the USFWS banned the use or possession of lead shot while hunting waterfowl (50 C.F.R. § 20.21(j)). This regulation was passed as a result of a lawsuit brought by a coalition of environmental groups, filed under the Endangered Species Act, in response to lead poisoning of waterfowl and secondary poisoning of eagles caused by lead shot. Regulations were phased in nationally over a five year period, with additional zones designated as requiring nontoxic shot each year. By September 1, 1991, every state was designated as a nontoxic shot zone for hunting waterfowl, coots and certain other species (50 C.F.R. 20.108). While bans on the use of lead shot for hunting waterfowl will inevitably reduce the likelihood of poisoning of raptors that prey on or scavenge waterfowl, they do nothing to prevent the poisoning of raptors that feed on hunted species away from wetlands or those that feed on a range of avian and mammalian prey. Lead ammunition is still permitted for upland hunting on non-federal lands by most states. Continued lead poisoning of condors, eagles, and upland game birds, has prompted some additional restrictions on use of lead ammunition in National Parks, National Wildlife Refuges, and on public lands in some states. Other than the regulations pertaining to lead shot for waterfowl, the federal government does not regulate the method of take by hunting, deferring to state regulations on federal lands.

A 2006 survey of existing nontoxic shot regulations for hunting waterfowl contacted 50 U.S. states, 10 Canadian provinces and 2 Canadian territories (D.J. Case & Associates 2006). In 2006, 23 states had nontoxic shot regulations for dove, crane, rail, snipe, quail, and/or pheasant hunting on some state-managed lands that go beyond those required by federal law for waterfowl hunting (Alaska, California, Illinois, Iowa, Kansas, Kentucky, Louisiana, Maryland, Michigan, Minnesota, Missouri, Nebraska, New Jersey, New Mexico, New York, North Carolina, North Dakota, Ohio, Oregon, South Dakota, Utah, Washington, and Wyoming). Some of these restrictions apply to public, but not private land, and in 7 states the restrictions only apply to mourning dove and/or marsh species such as snipe and rails. In general, the regulations are more widespread for species that overlap in habitat with waterfowl (such as crane rail, and snipe), and to a lesser degree, doves, and are less restrictive for upland game birds such as grouse, quail and pheasant. Of the 40 states that allow dove hunting, 16 have some level of nontoxic shot requirements specific to dove hunting.

There have are only been a few state efforts to restrict use of lead ammunition within the range of special-status species, such as the California condor, spectacled eider and other water birds:

Alaska

Lead exposure to the threatened spectacled eider and other water birds led to regulations in Alaska in 2007 that prohibit use of lead shot “T” size and smaller for hunting small game, furbearers, and unclassified game in the Yukon-Kuskokwim Delta.

California

The Ridley-Tree Condor Preservation Act was signed into law in California in 2007, effective July 1, 2008, requiring hunters to use non-lead ammunition for hunting big game (such as deer, elk, pigs, and bighorn sheep) and shooting coyotes within the condor range, which encompasses all or portions of 13 central and southern California counties and seven deer-hunting zones. The California Fish and Game Commission approved additional regulations in 2007 expanding the non-lead requirements to hunting of non-game mammals and birds and prohibiting the use of lead .22-caliber and smaller-rimfire cartridges for non-game hunting in the condor range. In February 2010, California state Assembly member Pedro Nava proposed legislation to ban the use of lead shot in California's 627,000 acre network of State Wildlife Areas – this legislation passed the state Assembly but was rejected by the Senate in 2010.

Wisconsin

In fall of 2008 Wisconsin started requiring dove hunters on public lands to use nontoxic shot (Eisele 2008).

National Parks

In March 2009 the National Park Service announced that it would begin to develop regulations to eliminate the use of lead ammunition in all National Parks by the end of 2010, but has yet to initiate any rulemaking. Grand Teton National Park and National Elk Refuge in Wyoming asked hunters to voluntarily switch to non-lead bullets beginning in fall of 2009.

International

As part of the International Update Report on Lead Poisoning in Waterbirds in 2000, 74 of 137 responding countries had implemented regulations on the use of lead shot, and 37 more countries indicated lead shot legislation was being prepared (Beintema 2001). Restrictions range from voluntary measures to partial bans applied to certain species and areas, to outright statutory bans for all water bird hunting. Use of lead shot to hunt any water bird species is banned outright in Canada, Denmark, Finland, Norway and Switzerland (Beintema 2001). Due to extensive lead poisoning of eagles, in 2001 the Ministry of the Environment in Japan mandated use of nontoxic rifle bullets or shotgun slugs for hunting on the island of Hokkaido, Japan (Saito 2009). Canadian national regulation in 1999 prohibited the use of lead shot for hunting all migratory birds anywhere in Canada (exempting upland species such as American woodcock, mourning doves, and rock doves. (Scheuhammer 2009).

Avery and Watson (2009b) summarized international lead ammunition legislation, noting that 29 countries have implemented voluntary or legislative restrictions on the use of lead ammunition. The types of bans varied widely and ranged from partial, voluntary

restrictions of the use of lead shot to a total ban on the use and import of lead ammunition. Two counties have banned all forms of lead ammunition. Six countries have a partial ban on the use of lead bullets in addition to full bans on lead shot. Four countries have banned the use of lead shot for all hunting. Fourteen countries and Australian territories have banned the use of lead shot in wetlands or for waterfowl hunting. Two countries have voluntary or recommended restrictions in place. Eleven countries and Australian territories have a partial ban on lead shot. Seven countries have implemented increasingly strict regulations on lead ammunition over time.

B. Existing Federal and State Lead Sinkers and Fishing Tackle Regulations

Federal

In 1994 the EPA proposed a nationwide ban on the manufacture, importation, processing, and distribution of fishing sinkers less than 25 mm containing lead or zinc (USEPA 1994), but these regulations were not ratified and have been abandoned.

Lead tackle is banned on some National Wildlife Refuges that have loon and swan populations and one National Park. As of 2008, only 7 national Wildlife refuges in the U.S. had regulations prohibiting use of lead weights, sinkers, or fishing tackle: Bear Lake NWR in Idaho, Union Slough NWR in Iowa, Rachel Carson NWR in Maine, Assabet River NWR in Massachusetts, Seney NWR in Michigan, Red Rock lakes NWR in Montana, and Rappahannock River Valley NWR in Virginia (Franson et al. 2003; Michael 2006). Yellowstone National Park has banned leaded fishing tackle and weights small enough to be ingested by wildlife. In March 2009 the National Park Service announced that it would begin to develop regulations to eliminate the use of lead fishing tackle in all National Parks by the end of 2010, but has yet to initiate any rulemaking.

In 1999 the USFWS announced it would establish additional lead-free fishing areas on more National Wildlife Refuges and wilderness areas used by loons, and waterfowl production areas on federal lands in Alaska, Florida, Maine, Minnesota, and Wisconsin (USEPA 1999; USFWS 1999). However, the USFWS has not followed through on regulations, a final rule on the use of lead fishing tackle on federal refuges has yet to be made, and this effort appears to have been shelved.

A few states subsequently moved forward with banning lead associated with fishing activities. Only Maine, New Hampshire, New York and Vermont have established statewide bans on the sale and/or use of lead sinkers and jigs in the sizes that are most likely to be ingested by wildlife. Massachusetts has a partial ban, and the other 45 states have no lead fishing regulations at all.

Maine

Maine passed legislation in 2001 (effective January 1, 2002) that prohibits the sale of lead sinkers one-half ounce or less.

Massachusetts

Massachusetts prohibits the use of lead sinkers for fishing in certain reservoirs visited by loons - the Quabbin and Wachusett Reservoirs.

New Hampshire

New Hampshire became the first state to ban the use of lead sinkers. Legislation passed in 1998 (effective in 2000) prohibits the use of lead sinkers up to one ounce and lead jigs up to one inch in length, in lakes and ponds in the state. This legislation was later expanded in 2006 to include all waters of the state.

New York

New York in 2004 prohibited the sale of lead fishing sinkers (including "split shot") weighing one-half ounce or less.

Vermont

Vermont passed legislation prohibiting the use (effective January 1, 2007) and sale (effective January 1, 2006) of lead fishing sinkers weighing one-half ounce or less.

International

Restrictions on the sale and use of lead fishing sinkers and jigs are in place in Canada, Denmark, and Great Britain. In 1997, Canada amended its Wildlife Area Regulations to prohibit possession of any lead sinker or jig weighing less than 50 grams while fishing in any National Wildlife Area where sport fishing is allowed, and also amended the National Parks Fishing Regulations to prohibit the possession and use of lead sinkers or jigs while fishing in national parks. Great Britain banned the use of lead sinkers weighing less than one ounce in 1987, due to the harm lead was causing swans, diving birds, and wading birds, and after determining that voluntary efforts were ineffective. Reported cases of lead poisoning in swans from the River Thames in England dropped from a peak of 107 in 1984 to 25 in 1988, one year after the ban on sale of lead fishing weights (Sears and Hunt 1991).

V. AUTHORITY TO ACT

In adopting the Toxic Substances Contro1 Act, Congress declared its policy that (1) "adequate data should be developed with respect to the effect of chemical substances and mixtures on health and the environment" and (2) "adequate authority should exist to regulate chemical substances and mixtures which present an unreasonable risk of injury to health or the environment."

To promulgate a rule under TSCA section 2605(a), the EPA must find there is a "reasonable basis to conclude" that activities involving a chemical substance "presents or will present an unreasonable risk to health or the environment." Factual certainty of the magnitude of risk to health and environment is not required; the EPA may base its decision not only on known facts, but also on scientific theories, projections and extrapolations from available data, and modeling using reasonable assumptions (59 Fed. Reg. 11122, 11138, citing H.R. Rep. No. 1341, 94th Cong., 2d Sess. 32 (1976)).

In its determination that a risk is "unreasonable," the EPA must weigh the risks to be reduced by the Act regulation and the consequences of such regulation. Section 6(c) of TSCA outlines four basic considerations to be weighed: A) the effects of the chemical on health and the magnitude of human exposure, B) the effects of the chemical on the environment and the magnitude of environmental exposure, C) the benefits of the chemical for various uses and the ability of substitutes for such uses, and D) the reasonably ascertainable economic consequences of the rule, after consideration of the effect on the national economy, small business, technological innovation, the environment, and public health (15 U.S.C. § 2605(c)(1)).

The EPA must consider whether the risk to be addressed by regulation may be prevented or reduced to a sufficient extent under other federal laws (15 U.S.C. § 2608(a)(1)(A)). The purpose of this provision is to avoid overlap and duplication between the Act and other federal laws. The EPA has determined that other federal laws do not sufficiently prevent or reduce the risk posed by lead and that "some activities, the regulation of which could protect [health and environment] (e.g., regulation of the manufacture, processing, and distribution in commerce of chemical substances) are clearly within the purview of [the] EPA" (15 U.S.C. § 2608(a)(1)(A), at 11137-38).

In the 1994 proposal to ban lead sinkers, the EPA preliminarily determined that other laws were, in fact, insufficient to reduce the unreasonable risk of harm to health and environment (59 Fed. Reg. 11122). The EPA recognized that, in the area of wildlife protection, there was some overlap between the statutory authorities administered by the Department of the Interior and the EPA, but that "some activities, the regulation of which could protect wildlife (e.g., regulation of the manufacture, processing, and distribution in commerce of chemical substances), are clearly within the purview of the EPA" (59 Fed. Reg. 11122).

Currently, there are some limited and inconsistent regulations regarding lead ammunition and fishing tackle in some states and on some National Wildlife Refuges and National

Parks. While these measures are a good start to eliminating the risk of lead exposure to wildlife and humans, and demonstrate that regulation of lead is possible and substitute ammunition and tackle are available for fishing and hunting activities, they are no substitute for comprehensive, sweeping regulation of lead shot, bullets, and sinkers by the EPA.

A. Authority to Regulate Lead Shot and Bullets

Lead used in shot, bullets and sinkers is a "chemical substance" falling within the scope of the Act (15 U.S.C. § 2602(2)(A)).[1] Although certain substances are excluded from the definition of "chemical substances," these exclusions do not apply to lead shot or bullets (15 U.S.C. § 2602(B)). Section 2602(B)(v) excludes from Act regulation "any article the sale of which is subject to the tax imposed by section 4181 of the Internal Revenue Code of 1986." Section 4181 of the Internal Revenue Code taxes firearms, shells, and cartridges (26 U.S.C. § 4181). However, shot and bullets are not subject to this tax. In fact, a 1968 Revenue Ruling states, "The manufacturers excise tax imposed upon sales of shells and cartridges by section 4181 of the Internal Revenue Code of 1954 *does not apply* to sales of separate parts of ammunition such as cartridge cases, primers, bullets, and powder" (Rev. Rul. 68-463, 1968-2 C.B. 507 (emphasis added)). This ruling has been confirmed by subsequent administrative decisions (See, for example, Fed. Tax Coordinator ¶ W-2911(2d.)). Because shot and bullets, as separate parts of ammunition, are not taxed under section 4181 of the Internal Revenue Code, the section 2602(B)(v) exception of TSCA does not apply. Thus, lead shot and bullets are properly classified as "chemical substances" subject to TSCA regulation.

B. Authority to Regulate Lead Sinkers and Fishing Tackle

The EPA expressed its authority to regulate lead sinkers under the Toxic Substances Control Act in 1994 by proposing a ban on lead sinkers (USEPA 1994). The EPA proposed a rule under section 6(a) of TSCA to prohibit the manufacturing, processing, and distribution in commerce in the United States, of certain smaller size fishing sinkers containing lead and zinc, and mixed with other substances, including those made of brass, due to unreasonable risk of injury to human health or the environment. This proposal was issued in response to a citizens' petition to require that the sale of lead fishing sinkers be accompanied by a label or notice that such products are toxic to wildlife. The EPA recognized the risk that lead sinkers presented to wildlife, and recognized that a labeling requirement would not go far enough to reduce this risk.

To date, the rule has not been finalized. In 2005 EPA issued a proposal to withdraw the proposed rule, but to date has not done so (see 70 Fed. Reg. 27625). It is possible that the "Common Sense in Fishing Regulations Act," introduced to the Senate in 1995, prompted the EPA to hold-off on a final rulemaking as it directed the EPA not to issue the ban on

[1] "Except as provided in subparagraph (B), the term "chemical substance" means any organic or inorganic substance of a particular molecular identity, including (i) any combination of such substances occurring in whole or in part as a result of a chemical reaction or occurring in nature and (ii) any element or uncombined radical."

lead sinkers (Sen. 505, 104th Cong. (March 6, 1995)). When introducing the bill, Senator Harkin declared that the proposed ban was nothing more than "government regulation run amok" and that the risks presented by lead sinkers were not unreasonable (Sen. 505, 104th Cong. (March 6, 1995), Senator Harkin, Statements on Introduced Bills and Joint Resolutions). However, the bill was never passed and EPA has still not acted to ban lead sinkers.

VI. DESCRIPTION OF FEDERAL REGULATIONS REQUESTED

Petitioners request that the EPA issue a proposed rule under section 6(a) of TSCA to prohibit the manufacturing, processing, and distribution in commerce in the United States of lead ammunition (including bullets and shotgun pellets) and lead fishing tackle (including sinkers, jig heads, weights, and all other fishing tackle).

VII. CONCLUSION

This petition has set forth the facts establishing the indisputable toxicity of lead hunting bullets and shotgun pellets and lead fishing gear to wildlife and to humans. The scientific literature on the sources, quantities, and pathways of exposure of lead in the environment from hunting, shooting sports, and fishing is comprehensive and conclusive, as is information on the toxic effects and health risk of lead ammunition and fishing tackle on wildlife and humans. The banning of lead ammunition for hunting in wetlands has greatly reduced the massive former mortalities of waterfowl and correspondingly reduced lead consumption by predators and scavengers of waterfowl, such as bald eagles, as well as humans. However, other uses of lead ammunition have continued unabated, causing unnecessary widespread incidental mortality of many bird and mammal species.

Lead-based bullets fragment on impact, distributing toxic lead particles widely throughout carcasses, and making it impossible for scavenging animals or humans to avoid ingesting lead along with meat. Normal butchering processes do not remove this lead. This health risk potentially affects large numbers of people, particularly hunters and their families and in areas where wild game is a significant part of the diet. Lead has been shown to affect adults and children at far lower concentrations in body tissues than formerly thought, and at lower concentrations than current regulations acknowledge.

Many species of wildlife ingest spent lead shot pellets or lead fishing weights, while others ingest lead fragments from the carcasses and gut piles of shot animals on which they feed. More than 130 species of wildlife are affected by lead from these sources in this way, and in some species thousands or tens of thousands of individuals die from lead ingestion every year in North America. For most species there has been no assessment of the effect of lead-caused mortality on population levels. However, population level effects have been shown in well-studied species such as the California condor, bald eagle, trumpeter swan, sandhill crane, and spectacled eider.

The widespread poisoning of many species of wildlife requires a response from the EPA to regulate and enforce a ban on lead ammunition and lead fishing tackle. This petition presents strong evidence that lead shot, bullets and sinkers pose an unreasonable risk to health and the environment and that this risk cannot be prevented through action under other federal laws. In evaluating unreasonable risk the EPA must consider: A) the effects of the chemical on health and the magnitude of human exposure; B) the effects of the chemical on the environment and the magnitude of environmental exposure; C) the benefits of the chemical for various uses and the ability of substitutes for such uses; and D) the reasonably ascertainable economic consequences of the rule, after consideration of the effect on the national economy, small business, technological innovation, the environment, and public health (15 U.S.C. § 2605(c)(1)). Regulation under section 2605 of the Toxic Substances Control Act requires only “a reasonable basis to conclude” that a risk is unreasonable. Scientific theories, projections of trends from currently available data, modeling using reasonable assumptions, and extrapolations from limited data may help to establish risk (H.R. Rep. No. 1341, 94th Cong., 2d Sess. 32 (1976)). The data presented in the petition supports the conclusion that the risk is such that lead shot,

bullets and sinkers should be banned under the Act. The recent granting of a Section 21 petition to ban lead wheel-balancing weights is further evidence that the EPA is aware of the environmental hazards of lead. This petition demonstrates that commercially available alternatives to hunting projectiles and fishing gear are, or can be made, available to replace lead-containing sporting equipment currently on the market.

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