IN THE UNITED STATES DISTRICT COURT FOR THE DISTRICT OF COLUMBIA

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) Civ. Action No. 10-2007 (EGS
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JOINT MOTION TO HOLD CASE IN ABEYANCE PENDING RESOLUTION OF RELATED ADMINISTRATIVE PETITION

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Plaintiffs Center for Biological Diversity, Public Employees for Environmental Responsibility, and Project Gutpile (collectively, "CBD"), Defendants Lisa P. Jackson and the U.S. Environmental Protection Agency (collectively, "EPA" or "Agency"), and Intervenor-Defendants National Rifle Association of America, Safari Club International, and Association of Battery Recyclers (collectively, "Movants"), hereby jointly move to hold this case in abeyance pending EPA's resolution of a related petition for rulemaking submitted to the Agency on November 17, 2011. Counsel for Intervenor-Defendant National Shooting Sports Foundation, Inc. ("NSSF") has indicated that NSSF does not oppose this Motion. In support of this motion, the Movants state as follows:

This suit challenges EPA's denials of Plaintiffs' petition requests under section 21 of the Toxic Substances Control Act ("TSCA"), 15 U.S.C. § 2620, for EPA to initiate proceedings for the issuance of rules under section 6(a) of TSCA, 15 U.S.C. § 2605, to prohibit the manufacturing, processing, and distribution in commerce in the United States of both lead ammunition (including bullets and shotgun pellets) and lead fishing tackle (including sinkers, jig heads, weights, and all other fishing tackle). Plaintiffs submitted this petition to EPA on August 3, 2010. The Agency received more than 6,000 comments from the public regarding CBD's requests to ban lead in bullet, shot, and fishing tackle. Those comments and other documents relating to EPA's consideration of the petition are available at www.regulations.gov, Docket ID# EPA-HQ-OPPT-2010-0681. EPA denied Plaintiffs' request for a ban on lead shot and bullets on August 27, 2010, and on November 4, 2010, the Agency denied Plaintiffs' request to ban lead in fishing gear.

On November 23, 2010, Plaintiffs filed a complaint in this Court challenging EPA's denials of the Plaintiffs' petition requests. On February 8, 2011, EPA filed a partial answer

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responding to Plaintiffs' allegations about lead in fishing gear, and a partial motion to dismiss regarding Plaintiffs' challenge to the denial of the request for a ban on lead shot and bullets. Intervenor-Defendant National Sports Shooting Foundation likewise filed a partial answer and partial motion to dismiss. Intervenor-Defendants Association of Battery Recyclers, National Rifle Association of America, and Safari Club International also filed answers to the complaint.

On September 29, 2011, the Court issued an order granting the partial motions to dismiss the lead shot and bullets claim. The Court's Order also directed the Parties to file a joint recommendation for further proceedings by October 31, 2011, or if the Parties were unable to agree on a joint recommendation, to each file a separate recommendation by that date.

On October 28, 2011, the Parties filed a joint motion for extension of time to file recommendations for further proceedings until December 15, 2011, in order to allow sufficient time for the Plaintiffs to review the comments submitted to EPA regarding their petition on lead fishing gear in preparation for discussion of further proceedings in this case.

On November 17, 2011, Plaintiffs CBD and Project Gutpile, along with a third entity, Loon Lake Loon Association, filed a petition ("New Fishing Tackle Petition" or "New Petition") under TSCA section 21, 15 U.S.C. § 2620, requesting EPA to "evaluate the unreasonable risk of injury to the environment from fishing tackle containing lead . . . of various sizes and uses that are ingested by wildlife, resulting in lead exposure" and to initiate proceedings for the issuance of a rulemaking under section 6(a) of TSCA, 15 U.S.C. § 2605, "to adequately protect against such risks." New Fishing Tackle Petition at 27 (attached as Exhibit 1). "Within 90 days after filing of a petition" under TSCA section 21, "the [EPA] Administrator shall either grant or deny the petition." 15 U.S.C. § 2620(b)(3). If the Administrator denies the petition, or fails to grant or deny the petition within 90 days of filing, "the petitioner may commence a civil action in a

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district court of the United States to compel the Administrator to initiate" the request rulemaking. *Id.* § 2620(b)(4)(A). Such an action must be filed within 60 days after the petition's denial or, if the Administrator fails to act on the petition within 90 days of filing, after the expiration of that 90-day period. *Id.*

EPA has not yet acted on the New Fishing Tackle Petition. If EPA grants the New Petition, that decision may moot some or all of the issues in this litigation on Plaintiffs' remaining claim regarding regulation of lead fishing tackle. If EPA denies the New Petition, or fails to act on it within 90 days of filing (on or by February 15, 2012), Plaintiffs may seek to bring suit regarding the New Petition under 15 U.S.C. § 2620(b)(4)(A). In either event, the interests of judicial economy and consistency of judicial decisions would be served if the Court holds this case in abeyance pending EPA's resolution of the New Petition.

Therefore, the Movants respectfully move the Court to hold this case in abeyance until February 15, 2012. If the Court grants this request, the Movants further ask that the Court reset the deadline for recommendations for further proceedings to 14 days after the lifting of the abeyance.

Respectfully submitted,

Dated: December 12, 2011

IGNACIA S. MORENO Assistant Attorney General

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Counsel for Safari Club International

CERTIFICATE OF SERVICE

The undersigned hereby certifies that a true and accurate copy of the foregoing Motion was filed using the Court's electronic case filing system this 12th day of December, 2011, which results in service on all counsel of record registered on the case management/electronic case filing ("CM/ECF") system.

/s/ Madeline Fleisher____ MADELINE FLEISHER

Exhibit 1



CENTER for BIOLOGICAL DIVERSITY

November 16, 2011

via Federal Express overnight delivery

Administrator Lisa P. Jackson Environmental Protection Agency USEPA Headquarters USEPA Ariel Rios Building (AR) 1200 Pennsylvania Avenue N.W. Washington, DC 20004

Dear Administrator Jackson,

Please find enclosed a Petition for Rulemaking under Section 21 of the Toxic Substances Control Act ("TSCA"), 15 U.S.C. § 2620(b)(3). Petitioners Center for Biological Diversity, Loon Lake Loon Association, and Project Gutpile request that the EPA evaluate the unreasonable risk of injury to the environment from fishing tackle containing lead (including fishing weights, sinkers, lures, jigs and/or other tackle) of various sizes and uses that are ingested by wildlife, resulting in lead exposure. Petitioners request that the EPA initiate a proceeding for the issuance of a rulemaking under Section 6 of TSCA to adequately protect against such risks (15 U.S.C. § 2620(a); 15 U.S.C. § 2605(a)(2)(A)(i)).

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)).

We appreciate your consideration of the enclosed Petition and await your determination.

Sincerely,

Ádam Keats Center for Biological Diversity

Arizona • California • Nevada • New Mexico • Alaska • Oregon • Minnesota • Vermont • Washington, DC

PETITION TO THE ENVIRONMENTAL PROTECTION AGENCY TO REGULATE LEAD FISHING TACKLE UNDER THE TOXIC SUBSTANCES CONTROL ACT



Common loon photo courtesy of U.S. Fish and Wildlife Service

PETITIONERS CENTER FOR BIOLOGICAL DIVERSITY LOON LAKE LOON ASSOCIATION PROJECT GUTPILE

PETITION FOR RULEMAKING UNDER THE TOXIC SUBSTANCES CONTROL ACT

November 16, 2011

EXECUTIVE SUMMARY

Pursuant to the Toxic Substances Control Act ("TSCA"), 15 U.S.C. § 2601 *et seq.*, Petitioners Center for Biological Diversity, Loon Lake Loon Association and Project Gutpile formally petition the Environmental Protection Agency ("EPA") to initiate a proceeding for the issuance of a rule under Section 6 of TCSA to adequately protect against the unreasonable risk of injury to the environment from fishing tackle containing lead that are of the sizes and uses that are ingested by wildlife, resulting in lead exposure. Fishing tackle, as used in this petition, refers to and includes fishing weights, sinkers, lures, jigs, and other fishing gear.

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 humans.

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, lost or discarded lead fishing weights and tackle are uncontrolled and lead remains widely encountered and distributed in the environment from these sources. Lead fishing tackle can accumulate in aquatic habitats, where animals encounter and ingest small lead items, often mistaking them for grit or food. Lead fishing tackle such as sinkers, jigs and weights continue to cause the needless and painful lead poisoning deaths of waterfowl such as swans, ducks, geese and loons.

Tackle manufacturers now market a wide variety of non-lead, nontoxic fishing tackle that can replace lead tackle. There is no technological or commercial reason why nontoxic fishing tackle with comparable effectiveness should not be substituted for lead. Several states in the Northeast have begun to require non-lead fishing weights and lures in an effort to protect loons and other 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. 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. Eliminating lead exposure may be achieved through a range of alternatives, up to and including 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)).

States that have mandated use of nontoxic fishing gear continue to have active 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 toxic lead from fishing tackle sources from the environment can be minimal. The EPA is obligated under TSCA to grant this petition and initiate a proceeding for the issuance of a rule under Section 6 to protect wildlife and the environment from unreasonable risk of lead poisoning from lead fishing tackle.

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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 regulate lead fishing tackle under section 2605 of TSCA.

Petitioner Center for Biological Diversity 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 320,000 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 federal and state policies regulating lead, to prevent toxic lead from entering the food chain and affecting wildlife or human health. The Center has been a leading proponent of federal regulations on lead ammunition and fishing tackle to protect wildlife at risk from lead poisoning.

Petitioner Loon Lake Loon Association is a nonprofit organization concerned about the common loon in the Pacific Northwest. The association works to protect the common loon, preserve their habitat and educate the public.

Petitioner Project Gutpile is an educational organization comprised of hunters/wildlife biologists and anglers that provides educational resources for sportsmen about lead-free hunting and angling. Project Gutpile has been promoting non-lead ammunition and fishing tackle and raising awareness about lead in the hunting and angling communities since 2002.

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)).

Contact information for petitioners:

Center for Biological Diversity 351 California Street, Suite 600 San Francisco, CA 94104 Contact: Jeff Miller (415) 436-9682 x303 jmiller@biologicaldiversity.org

II. NATURE OF THE REQUESTED ACTION

Petitioners Center for Biological Diversity, Loon Lake Loon Association and Project Gutpile request that the EPA evaluate the unreasonable risk of injury to the environment from fishing tackle containing lead (including fishing weights, sinkers, lures, jigs and/or other tackle) of various sizes and uses that are ingested by wildlife, resulting in lead exposure. Petitioners request that the EPA initiate a proceeding for the issuance of a rulemaking under Section 6 of TSCA to adequately protect against such risks (15 U.S.C. 2620(a); 15 U.S.C. 2605(a)(2)(A)(i)). This Petition sets forth facts presenting a reasonable basis to conclude that such a rulemaking is necessary to protect wildlife from the ongoing threat of lead poisoning.

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. § 2601. TSCA mandates that the EPA <u>must</u> 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)). 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)). Regulations may be achieved through a range of alternatives, up to and including 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)).

Lead used in fishing tackle is a "chemical substance" falling within the scope of TSCA. As defined by TSCA, "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 fishing tackle under TSCA.

III. PREVIOUS EFFORTS TO REGULATE

The EPA expressed its authority to regulate lead fishing tackle under the Toxic Substances Control Act in 1994 by proposing a ban on smaller sized lead sinkers (USEPA 1994). The EPA proposed a rule under section 6(a) of TSCA to prohibit the manufacture, importation, processing, and distribution in commerce in the United States, of fishing sinkers less than 25 mm containing lead or 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 concluded that a labeling requirement would not go far enough to reduce this risk. The EPA preliminarily determined that other laws were, in fact, insufficient to reduce the unreasonable risk of harm to health and environment from smaller-sized lead fishing sinkers (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).

To date, the rule has not been finalized and the proposed regulations have apparently been abandoned. In 2005 EPA issued a proposal to withdraw the proposed rule, but has not done so (see 70 Fed. Reg. 27625). It is likely 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 proposed ban on smaller 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). Although the bill was never passed the EPA has still not acted to regulate lead sinkers that pose lead exposure risks to wildlife.

On August 3, 2010, the American Bird Conservancy, Association of Avian Veterinarians, Center for Biological Diversity, Project Gutpile and Public Employees for Environmental Responsibility submitted a petition to the EPA under TSCA requesting that the EPA issue a proposed rule to prohibit the manufacture, 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). The EPA informally denied the petitioners' request to ban lead in fishing gear in a November 4, 2010 letter to the petitioners.¹ The EPA published the reasons for the denial in the November 17, 2010 Federal Register, stating that the petitioners did not demonstrate that the requested ban was the least burdensome alternative. The EPA did not consider any other alternatives for regulating lead fishing

¹ EPA denied petitioners' request for a ban on lead shot and bullets on August 27, 2010.

tackle other than a blanket nationwide ban of lead fishing tackle. The EPA did not consider banning or regulating a subset of lead fishing tackle of the sizes and uses most likely to result in lead exposure. The EPA's denial of that petition is currently the subject of litigation in the D.C. federal district court.

This current petition before the EPA seeks very different relief under TSCA than the 2010 petition, is brought by a different set of petitioners, and introduces new information. This petition responds to the EPA's assertion that there is not evidence demonstrating that a uniform nationwide rule banning all lead fishing tackle is necessary. This petition requests that the EPA instead regulate a subset of lead fishing tackle - those sizes and uses of lead fishing tackle likely to be ingested by wildlife and result in lead exposure, posing unreasonable risk to the environment. This petition does not specifically ask for a ban, whether of all lead fishing tackle or a subset of lead fishing tackle most likely to result in lead exposure to wildlife. It instead asks the EPA to initiate a rulemaking for regulations that adequately protect wildlife against the unreasonable risk of injury from lead fishing tackle. The EPA has latitude under TSCA to consider a broad range of alternatives for addressing an unreasonable risk of injury, including prohibitions on an entire or certain use of a chemical substance, limitations on allowable concentration levels, labeling or recordkeeping requirements, or obligations to issue notice of risks of injury. This petition does not request a specific regulatory alternative. It is the obligation of the EPA to determine the least burdensome alternative that adequately addresses the unreasonable risk of injury.

IV. 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 fishing activities dates back thousands of years. The lead poisoning effects of lead fishing tackle ingested by wildlife have been well documented. Recognizing this problem, some jurisdictions began placing restrictions on the use of lead fishing tackle beginning in the 1970s and 1980s. These restrictions are geographically limited and significant amounts of lead continue to be deposited in aquatic habitats and enter the food chain 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, or under some environmental conditions it can be readily released and taken up by plants or animals (ATSDR 2007). Lost or discarded lead fishing weights and other lead fishing tackle of smaller sizes can be ingested by wildlife, particularly dabbling and diving water birds such as such as swans, ducks, geese and loons. 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 small lead objects or fragments directly, mistaking them for grit or food. 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; NCM 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. Lead exposure to wildlife has been widely documented yet is not adequately regulated.

As long as lead fishing tackle of the sizes and uses commonly ingested by wildlife remains available for purchase and use, numerous species of wildlife will continue to be poisoned by lead. Human health is also threatened by the use of lead fishing gear. Although the few existing state regulations regarding lead fishing tackle generally address lead weights less than 25 mm in diameter or less than one ounce in weight, there is scientific evidence that some birds can ingest lead fishing weights up to two ounces in weight and there is evidence of ingestion by loons of sinkers up to 2.75 ounces in weight; there is also evidence of ingestion of sinkers by water birds greater than 25 mm in diameter (Scheuhammer and Norris 1995; Franson et al. 2003). Observed and documented 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. The EPA must evaluate the risk of injury to wildlife from all sizes, weights and types of lead fishing tackle and initiate a rulemaking to regulate lead fishing tackle of the sizes, weights, and uses likely to cause unreasonable risk of injury.

B. 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 or fishing 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 United States. The EPA recently granted a petition to ban lead automobile wheel balancing weights, and initiated a proceeding under TSCA to investigate potential lead hazards associated with the manufacture, processing, and distribution in commerce of lead wheel weights (USEPA 2009). The EPA anticipates publishing a proposed rulemaking on regulating lead wheel weights in October 2012. A petition to phase out the largest remaining permitted use of leaded gasoline, that for piston-engine aircraft, was filed with the EPA in 2010. The EPA has already solicited comments on that petition and issued an advance notice of proposed rulemaking on lead emissions from piston-engine aircraft using leaded aviation gasoline (USEPA 2010a, 2010b).

Environmental distribution of lead from 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.

There is very little information on lead from fishing weights in aquatic habitats which could be solubilized and taken up by invertebrates or fish (Stansley et al. 1992; Hui 2002). 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.

Numerous studies describe injuries to fish incurred by tackle (often containing lead) and small fragments passing through the digestive track of fish 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. 2003). 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.

There is a large body of evidence documenting significant deposition of lead fishing tackle in aquatic environments and subsequent ingestion by numerous bird species. There is extensive documentation of direct ingestion of lead sinkers, jigs and other fishing tackle by dabbling and diving ducks, swans, loons and other water birds. Water birds 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. 2003; Pokras et al. 2009).

C. Sources and Quantities of Lead in the Environment from Fishing Activities

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 concentrations of lost or discarded 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/m² in areas of high angling pressure (Duerr and DeStefano 1999), with much higher densities (up to 190 sinkers/m²) 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 were produced each year and potentially entering the environment; and that figure may now be higher. Compare that figure to the estimated 4 million pounds of lead thought to annually enter the environment due to lead wheel weights falling off cars and trucks (USGS 2003; USEPA 2011a). Based on this amount of lead entering the environment, the EPA initiated regulations requiring non-lead wheel weight alternatives. The amount of lead entering the environment from lead fishing tackle nearly two decades ago was thought to be in the range of more than 100 times the amount from wheel weights, and in sizes and locations much more likely to cause significant lead exposure to wildlife.

D. Toxic Effects of Lead 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 lead shotgun pellets from hunting or lead 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 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 objects or fragments ingested by birds may be rapidly regurgitated, 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. For example, the Diagnostic Center for Population and Animal Health (Michigan State University, Lansing MI) defined background blood lead levels as $<35 \mu g/dL$ for eagles, while Pattee et al. (1990) defined background levels as $<20 \mu g/dL$, and Feierabend and Myers (1984) defined them as $<10 \mu g/dL$. The generally accepted blood lead levels for wild birds have been $<20 \mu g/dL$ as background; 20 to $<50 \mu g/dL$ indicating subclinical poisoning; 50 to 100 $\mu g/dL$ indicating clinical poisoning; and $>100 \mu g/dL$ representing severe clinical poisoning (Friend 1985, 1999; Franson 1996; Pain 1996; Pattee and Pain 2003). Background levels at or higher than 20ug/dl are now understood to indicate significant exposure, because animals held in captivity usually have background levels of 4 $\mu g/dL$ or less (Walters et al. 2010).

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.

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, this has the potential to skew the normal age structure toward younger and non-breeding birds and negatively influence long-term population viability. 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). Over longer periods, lead poisoning can reduce haematocrit and hemoglobin levels. 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). Finkelstein et al. (2010) found that sub-lethal concentrations of lead in blood (20 μ g/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). Lead exposure may also reduce the likelihood of birds returning to an area to breed (Mateo et al. 1999).

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). 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).

E. Lead Fishing Tackle Poisonings by Species

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 loons, swans, cranes, geese, mallards and brown pelicans (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).

1. Loons

There is a direct link between ingestion of lead fishing tackle and mortality of loons. Lead poisoning from ingesting lead fishing sinkers or jigs is a significant cause of death for adult common loons (Gavia immer) in the United States and eastern Canada (USFWS 2004). Lead poisoning from fishing tackle accounts 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). 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 ingesting either lead shot or lead fishing sinkers. 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). The U.S. Fish and Wildlife Service noted that ingestion of lead sinkers and jigs accounted for between 40 and 70 percent of mortality of dead common loons recovered in New Hampshire from 1996 to 2002 (USFWS 2004). Pokras et al (1992) examined 60 dead loons collected in New Hampshire 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 in New Hampshire 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.

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. 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 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.

Substantial rates of lead-related mortality are also known for loons in Michigan, Minnesota and Wisconsin. 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. 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 and ingesting lead shot or lead fishing sinkers on the bottom of water bodies (Eisele 2008). 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.

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. 2003). 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.

Common loons 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 tackle, including weights, 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. Water birds trapped when apparently healthy rarely show evidence of lead sinker ingestion (Franson and Cliplef 1992).

2. Swans

Efforts to restore trumpeter swans (*Cygnus buccinator*) in Wisconsin are 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 ($20 \mu g/dL$). 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 (*Cygnus olor*) (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).

3. Other Birds

Lead fishing sinkers and jigs have contributed to lead poisoning mortalities in Canada geese (Branta canadensis), mallards (Anas platyrhynchos) and brown pelicans (Pelecanus occidentalis) (Rattner et al. 2008). Two sandhill cranes (Grus canadensis) 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 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. 2003).

4. Reptiles

There is evidence of freshwater turtles ingesting lead fishing weights and suffering from lead toxicosis (Borkowski 1997; Scheuhammer et al. 2003).

F. Lead Fishing Tackle Risk of Exposure to Humans

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 fishing activities that may be an important source of lead exposure in certain communities, occupations or activities.

Watson and Avery (2009) assessed the numbers and proportions of state populations that may be at risk of lead exposure from handling and using fishing gear, from handling and making lead sinkers, and through accidental ingestion of lead fishing gear. 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). The EPA warns that anglers who cast their own sinkers, jigs or spinnerbaits at home may be exposed to potentially harmful airborne lead particles or vapors while melting and pouring lead into lead fishing sinker molds (U.S. EPA 2011b). Additionally, airborne particles from melted lead can move around and spread far distances, covering soil, dust, walls, floors, furniture, clothing, toys, stuffed animals, etc. (USEPA 2011b). Anglers also risk lead exposure from putting lead sinkers in their mouth or biting down on slip shot, as well as from handling lead sinkers or cleaning out their tackle box (USEPA 2011b).

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). Lead accumulates in humans mainly in bones, with lead in blood and other tissues reflecting more recent exposure. 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; Cecil et al. 2008). Lead is also implicated in decreased growth (Hauser et al. 2008), decreased brain volume (Cecil et al. 2006), cancer, and cardiovascular disease (Menke et al. 2006, Lustberg and Silbergeld 2002). 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 (Schnaas et al. 2006). Some studies link elevated bone or blood lead levels with aggression, delinquent behavior, attention deficit hyperactivity disorder and criminal behavior (Nevin 2000; Needleman et al. 2002; Needleman 2004; Braun et al. 2006; Wright et al. 2008).

Many studies show even very small amounts of lead can have permanent, debilitating, sub-lethal effects. In humans, blood lead concentration of 10 micrograms of lead per deciliter (μ g/dL) is currently considered an elevated level, although some researchers and health professionals have advocated for a threshold of 5 micrograms or even 2

micrograms. In the mid-20th century, the amount of lead in the bloodstream of a child considered in need of medical intervention was considered to be 60 μ g/dL, whereas today it is 10 μ g/dL. The U.S. Department of Health and Human Services has concluded that there is evidence that blood lead levels less than 10 μ g/dL are associated with adverse health effects on development in children and reproduction in adult women, such as delayed puberty, decreased postnatal growth, reduced fetal growth, spontaneous abortion and preterm birth (NTP 2011); and studies show blood lead levels at and below 2 μ g/dL are associated with adverse effects (e.g. Wu et al. 2003; Denham et al. 2005). Children sustain permanent cognitive damage when they show an average of only 7.5 μ g/dL in blood before the age of five (Lanphear et al. 2005).

A new draft assessment by the National Toxicology Program of the U.S. Department of Health and Human Services concluded that blood lead concentrations lower than the federally established levels of concerns are associated with adverse health effects and reports that even blood lead concentrations lower than 5 μ g/dL are associated with decreased academic performance and cognitive function, increased incidences of ADHD and behavioral problems in children (NTP 2011). The assessment also found that blood lead concentration levels below 10 μ g/dL are associated with delayed puberty, reduced growth, decreased IQ, and decreased hearing in children, and increased blood pressure and cardiovascular disease in adults (NTP 2011). The consensus among medical researchers is that there is no safe level of lead exposure in young children (CDC 2005).

V. AUTHORITY TO ACT

In adopting TSCA, 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" 15 U.S.C. § 2601(b). In recognizing that the EPA would not always act on its own, it authorized any person to petition the EPA to initiate a proceeding under several sections of the Act (15 U.S.C. § 2620(a)). A petition shall set forth the facts claiming that it is necessary to issue a rule under Section 6. After reviewing and granting a petition, the EPA then commences an appropriate rulemaking proceeding.

To promulgate a rule under TSCA Section 6, 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 promulgating a rule under Section 6, the EPA must consider and publish a statement with respect to:

(A) the effects of such substance or mixture on health and the magnitude of the exposure of human beings to such substance or mixture, (b) the effects of such substance or mixture on the environment and the magnitude of the exposure to the environment to such substance or mixture, (C) the benefits of such substances or mixture for various uses and the availability 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)(A)-(D). In issuing a rule, the EPA must employ the least burdensome requirements afforded by Section 6. In order to facilitate this rulemaking, the EPA shall proceed in accordance with Section 553 of the Administrative Procedure Act.

However, at the petition stage, the EPA must only review the petition and determine whether there is a reasonable basis to conclude that the manufacture, processing, distribution, use, or disposal of lead in fishing sinkers presents an unreasonable risk of injury to health or the environment, necessitating the initiation of a rulemaking proceeding under Section 6 of the Act.

VI. ALTERNATIVES TO LEAD FISHING TACKLE

In reviewing the petition, the EPA is only required to determine whether there is a reasonable basis to conclude that an issuance of such a rulemaking is necessary to protect against an unreasonable risk of injury. However, 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)). Therefore, this petition identifies commercially available alternatives to lead fishing tackle in anticipation of the promulgation of such a rule.

Fishing sinkers and jigs do not have to be made of lead. Nearly all fishing tackle products available in lead are now available in nontoxic alternative materials. Demonstrated technology indicates that all fishing tackle 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 fishing gear. Inexpensive and ecologically sound alternatives to lead fishing weights made from non-poisonous materials such as tin, bismuth, steel, and recycled glass are widely 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. 2003; MOEA 2006). However, 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).

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. 2003; Michael 2006). Some states and non-profit organizations offer small-scale programs that exchange angler-owned lead tackle for free 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.

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, at 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 eventually corroding after continued 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 also 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 is currently more expensive than its lead equivalents. 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 is also toxic in aquatic environments. 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.

The EPA has already determined that the economic impact of banning smaller-sized lead fishing tackle would 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).

VII. EXISTING REGULATIONS

While TSCA only requires the EPA to consider whether actions undertaken by the EPA under other federal laws adequately address the risk of unreasonably injury, this petition presents information on existing regulatory efforts in order to demonstrate that despite these efforts, there remains an unreasonable risk of injury to the environment.

There are an increasing number of outreach and education programs by state and federal agencies and non-governmental organizations promoting the use of non-lead fishing tackle. While these programs are important, they have not resulted in a widespread switch to non-toxic fishing tackle by anglers and there is no evidence these programs by themselves have significantly reduced lead exposure to wildlife. The fact that there are some limited state regulations addressing lead sinkers has also not ended lead poisonings of birds in those states, nor do those regulations address continued lead poisoning and likely lead exposure to birds in numerous other states where there are not even limited state regulations. TSCA does not require a showing of population-level impacts from lead poisonings to regulate toxic lead, only that there is an unreasonable risk of injury to the environment.

Federal Regulations

Currently, there are some limited and inconsistent regulations regarding lead fishing tackle on some National Wildlife Refuges and National Parks. While these measures may have begun to reduce the risk of lead exposure to wildlife and humans in limited geographic areas, and demonstrate that regulation of lead is possible and that nontoxic tackle is available for fishing activities, they are no substitute for comprehensive, sweeping regulation by the EPA of lead fishing tackle that poses lead exposure risks to wildlife and humans.

Lead tackle is banned on some U.S. National Wildlife Refuges ("NWR") that have loon and swan populations and in one National Park. Currently, the National Wildlife Refuges with regulations prohibiting use of lead weights, sinkers, or fishing tackle include Bear Lake NWR in Idaho, Union Slough NWR in Iowa, Rachel Carson NWR in Maine, Patuxtent NWR in Maryland, 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 U.S. Fish and Wildlife Service ("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, and a final rule on the use of lead fishing tackle on federal refuges has never been made.

State Regulations

A few states have moved forward with banning small-sized lead tackle associated with fishing activities. Only Maine, New Hampshire, New York and Vermont have established statewide bans on the sale and/or use of smaller lead sinkers and jigs. Massachusetts and Washington have partial bans, and the other 44 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 currently prohibits the use of lead sinkers for fishing in certain reservoirs visited by loons (Quabbin and Wachusett Reservoirs) and starting in 2012 the use of lead sinkers, weights and fishing jigs less than one half ounce will be prohibited in all inland waters.

New Hampshire

New Hampshire was the first state to ban the use of small 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 throughout 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.

Washington

The Washington Fish and Wildlife Commission in 2010 approved restrictions on the use of lead fishing tackle at 13 lakes with nesting common loons. The new regulations, which took effect May 2011, prohibit the use of lead weights and jigs measuring one and one half inches or less along the longest axis at 12 lakes; Ferry and Swan lakes in Ferry County; Calligan and Hancock lakes in King County; Bonaparte, Blue and Lost lakes in Okanogan County; Big Meadow, South Skookum and Yocum lakes in Pend Oreille County; Pierre Lake in Stevens County; and Hozomeen Lake in Whatcom County. The commission also banned the use of flies containing lead at Long Lake in Ferry County.

International

Regulations banning smaller lead fishing tackle in other countries demonstrate the potential effectiveness of regulations in the United States. Restrictions on the sale and use of smaller 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).

VIII. DESCRIPTION OF FEDERAL REGULATIONS REQUESTED

Petitioners Center for Biological Diversity, Loon Lake Loon Association and Project Gutpile formally request that the EPA:

- 1) evaluate the unreasonable risk of injury to the environment and wildlife from fishing tackle containing lead (including fishing weights, sinkers, lures, jigs and/or other tackle) of various sizes and uses that are ingested by wildlife, resulting in lead exposure; and
- initiate a proceeding for the issuance of a rulemaking under Section 6(a) of TSCA to adequately protect against such risks (15 U.S.C. § 2620(a); 15 U.S.C. § 2605(a)(2)(A)(i)).

This petition sets forth facts presenting a reasonable basis to conclude that such a rulemaking is necessary to protect wildlife from the ongoing threat of lead poisoning.

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. § 2601. TSCA also mandates that the EPA <u>must</u> 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)). 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)). Regulations may be achieved through a range of alternatives, up to and including 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)).

IX. CONCLUSION

Section 6 of the Toxic Substances Control Act requires only that the EPA find that there is "a reasonable basis to conclude" that a risk to the environment or human health 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 this petition provides a reasonable basis to conclude that the risk is such that lead fishing tackle of certain sizes and uses should be regulated under TSCA to protect against unreasonable risk of injury to the environment. This petition has set forth the facts establishing the indisputable exposure and toxicity of lead fishing tackle of certain sizes and uses to wildlife. The scientific literature on the sources, quantities, and pathways of exposure of lead in the environment from fishing tackle is conclusive, as is information on the toxic effects and health risk of fishing tackle on wildlife. Many species of wildlife, particularly water birds, ingest lost or discarded lead fishing tackle. The widespread poisoning of many species of wildlife requires a response from the EPA to regulate certain sizes and uses of lead fishing tackle.

X. REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR). 2007. Toxicological Profile for Pb. http://www.atsdr.cdc.gov/substances/toxsubstance.asp?toxid=22.

Bagchi, D. and H.G. Preuss. 2005. Effects of Acute and Chronic Oval Exposure of Lead on Blood Pressure and Bone Mineral Density in Rats. Journal of Inorganic Biochemistry 99:1155-1164.

Bates, F.Y., D.M. Barnes, and J.M. Higbee. 1968. Lead Toxicosis in Mallard Ducks. Bull. Wildl. Dis. Assoc. 4:116-125.

Bell, D.V., N. Odin, and E. Torres. 1985. Accumulation of Angling Litter at Game and Coarse Fisheries in South Wales. U.K. Biological Conservation 34:369-379.

Bellrose, F.C. 1959. Lead Poisoning as a Mortality Factor in Waterfowl Populations. Ill. Nat. Hist. Surv. Bull. 27:2335-288.

Birkhead, M. 1982. Causes of Mortality in the Mute Swan *Cygnos olor* on the River Thames. Journal of Zoology 198:15-25.

Birkhead, M. and C. Perrins. 1985. The Breeding Biology of Mute Swan *Cygnus olor* on the River Thames With Special Reference to Lead Poisoning. Biological Conservation 32:1-11.

Blus, L.J., R.K. Stroud, B. Reiswig, and T. McEneaney. 1989. Lead Poisoning and Other Mortality Factors in Trumpeter Swans. Environmental Toxicology and Chemistry 8:263-271.

Blus, L.J., C.J. Henry, D.J. Hoffman, and R.A. Grove. 1991. Lead Toxicosis in Tundra Swans Near a Mining and Smelting Complex in Northern Idaho. Arch. Environ. Contam. Toxicol. 21:549-555.

Blus, L.J. 1994. A review of lead poisoning in swans. Comparative Biochemistry and Physiology, Part C 108(3):259-267.

Blus, L.J., C.J. Henry, D.J. Hoffman, and R.A. Grove. 1995. Persistence of High Lead Concentrations and Associated Effects in Tundra Swans Captured Near a Mining and Smelting Complex in Northern Idaho. Ecotoxicology (8)2: 125-132.

Borja-Aburto, V. H., I. Hertz-Picciotto, M. R. Lopez, P. Farias, C. Rios, and J. Blanco. 1999. Blood lead levels measured prospectively and risk of spontaneous abortion. American Journal of Epidemiology 150:590-597. Borkowski, R. 1997. Lead Poisoning and Internal Perforations in a Snapping Turtle (*Chelydra serpentina*) Due to Fishing Gear Ingestion. Journal of Zoo and Wildlife Medicine 28:109-113.

Braun, J.M., R.S. Kahn, T. Froelich, P. Auinger, and B. Lamphear. 2006. Exposures to Environmental Toxicants and Attention Deficit Hyperactivity Disorder in U.S. Children. Environmental Health Perspectives 114;1904-1909.

Burger, J. and M. Gochfeld. 2000. Metals in Albatross Feathers From Midway Atoll: Influence of Species, Age, and Nest Location. Environ. Res. 82(3): 207-21.

Canfield, R. L., C.R. Henderson, Jr., D.A. Cory-Slechta, C. Cox, T.A. Jusko, and B.P. Lanphear. 2003. Intellectual Impairment in Children with Blood Lead Concentrations Below 10 micrograms Per Deciliter. New England Journal of Medicine 348:1517-26.

Carlisle, J.C., K.C. Dowling, D.M. Siegel, and G.V. Alexeeff. 2009. A blood lead benchmark for assessing risks from childhood lead exposure. J Environ Sci Health A Tox Hazard Subst Environ Eng. 2009 Oct;44(12):1200-8.

Carpenter, J.W., O.H. Pattee, S.H. Fritts, B.A. Rattner, S.N. Wiemeyerr, J.A. Royle, and M.R. Smith. 2003. Experimental Lead Poisoning in Turkey Vultures (*Cathartes aura*). J. Wildl. Dis. 39(1):96-104.

Cecil, K. M., C. J. Brubaker, C. M. Adler, K. N. Dietrich, M. Altaye, J. C. Egelhoff, S.Wessel, I. Elangovan, R. Hornung, K. Jarvis, and B. Lanphear. 2008. Decreased brain volume in adults with childhood lead exposure. PLoS Medicine 5:741-750.

Centers for Disease Control and Prevention (CDC). 2005. Preventing lead poisoning in young children.

Cheatum, E.L., and D. Benson. 1945. Effects of lead poisoning on reproduction of mallard drakes. Journal of Wildlife Management 9(1):26-29.

Cryer, M.J., J. Corbett, and M.D. Winterbotham. 1987. The Deposition of Hazardous Litter by Anglers at Coastal and Inland Fisheries in South Wales. Journal of Environmental Management 25:125-135.

Custer, T.W., J.C. Franson, and O.H. Pattee. 1984. Tissue Lead Distribution and Hematologic Effects in American Kestrels (*Falco sparverius*) Fed Biologically Incorporated Lead. J. Wildlife Dis. 20, 39-43.

Daoust, P.-Y., G. Conboy, S. McBurney, and N. Burgess. 1998. Interactive Mortality Factors in Common Loons From Maritime Canada. Journal of Wildlife Diseases 34:524-531. Delaney, S.J., J.D. Greenwood, and J. Kirby. 1992. The National Mute Swan Survey 1990. JNCC Report Number 74, Joint Nature Conservancy Council, Peterborough, UK.

Denham, M., Schell, L. M., Deane, G., Gallo, M. V., Ravenscroft, J., and DeCaprio, A. P. 2005. Relationship of lead, mercury, mirex, dichlorodiphenyldichloroethylene, hexachlorobenzene, and polychlorinated biphenyls to timing of menarche among Akwesasne Mohawk girls. *Pediatrics* **115**, e127-134.

Dorgelo, F. 1994. Alternatives for lead shot and fishing sinkers in the Netherlands. Issue Paper presented at the OECD Workshop on Lead Products and Uses, 12-15 September, Toronto, Ontario. 5 pp.

Duerr, A.E. 1999. Abundance of Lost and Discarded Fishing Tackle and Implications for Waterbird Populations in the United States. Masters thesis. School of Renewable Natural Resources, University of Arizona, Tucson, Arizona.

Duerr, A.E. and S. DeStefano. 1999. Using a Metal Detector to Determine Lead Sinker Abundance in Waterbird Habitat. Wildlife Society Bulletin 27:952-958.

Edens, F.W. and J.D. Garlich. 1983. Lead-Induced Egg Production Decrease in Leghorn and Japene Quail Hens, Poultry Sci. 62, 1757-1763.

Eisele, T. 2008. Outdoors: Time to Get the Lead Out of All Hunting, Fishing. Special to The Capital Times 3/12/2008.

Eisler, R. 1988. Lead Hazards to Fish, Wildlife and Invertebrates: A Synoptic Review. USFWS Biol. Rep. 8, 1-4.

Ekong, E. B., B. G. Jaar, and V. M. Weaver. 2006. Lead-related nephrotoxicity: a review of the epidemiologic evidence. Kidney International 70:2074-2084.

Engstad, J.E. 1932. Foreign bodies in the appendix. Minnesota Med. 15:603-6xx. Ensor, K.L., D.D. Helwig, and L.C. Wemmer. 1992. Mercury and Lead in Minnesota Common Loons (*Gavia immer*). Water Quality Division, Minnesota Pollution Control Agency, St. Paul, Minnesota. 32 pp.

Environment Canada. 1995. A review of the environmental impacts of lead shotshell ammunition and lead fishing weights in Canada. Canadian Wildlife Service. Occasional Paper No. 88. Hull, Quebec.

Evers, D.C. 2004. Status assessment and conservation plan for the Common Loon (Gavia immer) in North America. U.S. Fish and Wildlife Service, Hadley, MA.

Finkelstein, M.E., D. George, S. Scherbinski, R. Gwiazda, M. Johnson, J. Burnett, J. Brandt, S. Lawrey, A.P. Pessier, M. Clark, J. Wynne, J. Grantham, and D.R. Smith. 2010. Feather Lead Concentrations and ²⁰⁷Pb/²⁰⁶Pb Ratios Reveal Lead Exposure History of

California Condors (*Gymnogyps californianus*). Environ. Sci. Technol. 2010, 44, 2639–2647.

Finley, M.T., and M.P. Dieter. 1978. Influence of laying on lead accumulation in bone of mallard ducks. Journal of Toxicology and Environmental Health 4:123-129.

Fisher, I.J., D.J. Pain, and V.G. Thomas. 2006. A Review of Lead Poisoning From Ammunition Sources in Terrestrial Birds. Biological Conservation 131:421-432.

Franson, J.C. and D.J. Cliplef. 1992. Causes of Mortality in Common Loons. Pages 2-59 *in* W.N. Beyer, G.H. Heinz, and A.W. Redmon-Norwood, editors. Proceedings from the 1992 Conference on the Loon and its Ecosystem: Status, Management, and Environmental Concerns, August 22-24, 1992, College of the Atlantic, Bar Harbor, Maine.

Franson, J.C. and S.G. Hereford. 1994. Lead Poisoning in a Mississippi Sandhill Crane. Wilson Bulletin 106:766-768.

Franson, J.C., L. Sileo, O.H. Pattee, and J.F. Moore. 1983. Effects of Chronic Dietary Lead in American Kestrels (*Falco spaverius*). J. Wildlife Dis. 19,110-113.

Franson, J.C., N.J. Thomas, M.R. Smith, A.H. Robbins, S. Newman, and P.C. McCartin. 1996. A Retrospective Study of Post-Mortem Findings in Red-Tailed Hawks, J. Raptor Res. 30, 7-14.

Franson, J.C., S.P. Hansen, T.E. Creekmore, C.J. Brand, D.C. Evers, A.E. Duerr, and S. DeStefano. 2003. Lead Fishing Weights and Other Fishing tackle in Selected Waterbirds. Waterbirds 26:345-352.

Friend, M. 1987. Field Guide to Wildlife Diseases. USFWS.

Friend, M. 1999. Lead. Pages 317-334 *in* M. Friend and J.C. Franson, editors. Field Manual of Wildlife Diseases: General Field Procedures and Diseases of Birds. U.S. Geological Survey, Biological resources Division. Information and technology Report 1999-2001. Washington, D.C.

Goyer, R.A. 1996. Toxic Effects of Metals. Pages 691-736 *in* C.D. Klaassen, M.O. Amdur, and J. Doull, editors. Cassarett and Doull's Toxicology: The Basic Science of Poisons. 5th ed. McGraw-Hill, New York.

Grandjean, P. 1976. Possible effect of lead on egg-shell thickness in kestrels 1874-1974. Bulletin of Environmental Contamination and Toxicology 16(1):101-106.

Grandy, J.W. IV, L.N. Locke, and G.E. Bagley. 1968. Relative Toxicity of Lead and Five Proposed Substitute Shot Types to Pen-Reared Mallards. Journal of Wildlife Management 32:483-488.

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

Grinnell, G.B. 1894. Lead-poisoning. Forest and Stream 42(6):117-118.

Hanzlik, P. J. 1923. Experimental plumbism in pigeons from the administration of metallic lead. Archiv für experimentelle Pathologie und Pharmakologie 97:183-201.

Hauser, R., O. Sergeyev, S. Korrick, M. M. Leem B. Revich, E. Gitin, J. S. Burns, and P. L.Williams. 2008. Association of blood lead levels with onset of puberty in Russian boys. Environmental Health Perspectives 116:976-980.

Henny, C.J., L.J. Blus, D.J. Hoffman, R.A. Grove, and J.S. Hatfield. 1991. Lead Accumulation and Osprey Production Near a Mining Site in the Coeur d' Alene River, Idaho. Arch. Environ. Contam. Toxicol. 21, 415-424.

Hernberg, S. 2000. Lead Poisoning in Historical Perspective. American Journal of Industrial Medicine 38:244-254.

Hoffman, D.J., O.H. Pattee, S.N. Wiemeyer, and B. Mulhern. 1981. Effects of Lead Shot Ingestion on Delta-Aminolevulinic Acid Dehyratase Activity, Hemoglobin Concentration, and Serum Chemistry in Bald Eagles. J. Wildl. Dis. 17:423-431.

Hoffman, D.J., J.C. Franson, O.H. Pattee, C.M. Bunck, and A. Anderson. 1985. Survival, Growth and Accumulation of Ingested Lead in Nestling American Kestrels (*Falco sparverius*). Arch. Environ. Contam. Toxicol. 14, 89-94.

Hoffman, D.J., J.C. Franson, O.H. Pattee, C.M. Bunck, and H.C. Murray. 1985. Biochemical and Hematological Effects of Lead Ingestion in Nestling American Kestrels (*Falco sparverius*). Comp. Biochem. Physiol. 80C, 431-439.

Hohman, W. L., J. L. Moore, and J. C. Franson. 1995. "Winter survival of immature Canvasbacks in inland Louisiana." Journal of Wildlife Management 59(2):384-392.

Hui, C.A. 2002. Lead Distribution Throughout Soil, Flora and an Invertebrate at a Wetland Skeet Range. Journal of Toxicology and Environmental Health 65:1093-1107.

International Programme on Chemical Safety (IPCS). 1989. Lead Environmental Aspects. Environmental health Criteria 85. World Health Organization, International Programme on Chemical Safety, Geneva, Switzerland.

Irwin, J.C. and L.H. Karstad. 1972. The toxicity for Ducks of Disintegrated Lead Shot in a Stimulated Marsh Environment. J. Wildl. Dis. 8:149-154.

Kelly, A. and S. Kelly. 2000. Are Mute Swans With Elevated Blood Levels More Likely to Collide With Overhead Powerlines? Waterbirds 28:331-334.

Kelly, A. and S. Kelly. 2004. Fishing Tackle Injury and Blood Lead Levels in Mute Swans. Waterbirds 27:60-68.

Kendall, R.J. and P.F. Scanlon. 1981. Effects of Chronic Lead Ingestion on Reproductive Characteristics of Ringed Turtle Doves (*Streptopelia risoria*) and on Tissue Lead Concentrations of Adults and Their Progeny. Environ. Pollut. Series A 26, 203-214.

Khan, A.N. 2005. Lead Poisoning. Emedicine instant access to the minds of medicine. www.emedicine.com/radio/topic386.htm.

Kimball, W. H. and Z. A. Munir. 1971. The corrosion of lead shot in a simulated waterfowl gizzard. Journal of Wildlife Management_35(2):360-365.

Kirby, J., S. Delany, and J. Quinn. 1994. Mute Swans in Great Britain – A Review, Current Status and Long-Term Trends. Hydrobiologia 279/280:467-482.

Kramer, J.L. and P.T. Redig. 1997. Sixteen Years of Lead Poisoning in Eagles, 1980-1995: An Epizootiologic View. J.E. Cooper and A.G. Greenwood (eds.). Journal of Raptor Research 31:327-332.

Lanphear, B.P., K.N. Dietrich, and O. Berger. 2003. Prevention of lead toxicity in US children. Ambul. Pediatr. 3(1):27-36.

Lanphear, B.P., R. Hornung, J. Khoury, K. Yolton, P. Baghurst, D.C. Bellinger, R.L. Canfield, K.N. Dietrich, R. Bornschein, T. Greene, S.J. Rothenberg, H.L. Needleham, L. Schnaas, G. Wasserman, J. Graziano, and R. Roberts. 2005. Low-level environmental lead exposure and children's intellectual function: an international pooled analysis. Environ Health Perspect. 2005 Jul;113(7):894-9.

Lanphear, B.P., R. Hornung, J. Khoury, K. Yolton, and K.N. Dietrich. 2006. Lead and IQ in Children: Lanphear et al. Respond. Environ Health Perspect. 2006 February; 114(2): A86–A87.

Levengood, J.M., G.C. Sanderson, W.L. Anderson, G.L. Foley, L.M. Skowron, P.W. Brown, and J.W. Seets. 1999. Acute Toxicity of Ingested Zinc Shot to Game-Farm Mallards. Illinois Natural History Survey Bulletin 36:1-36.

Lewis, L.A., R.J. Poppenga, W.R. Davidson, J.R. Fischer, and K.A. Morgan. 2001. Lead Toxicosis and Trace Elements in Wild Birds and Mammals at a Firearms Training Facility. Arch. Environ. Contam. Toxicol. 41:208-214.

Locke, L.N., S.M. Kerr, and D. Zoromski. 1982. Lead Poisoning in Common Loons (*Gavia immer*). Avian Diseases 26:392-396.

Lustberg M. and E. Silbergeld. 2002. Blood lead levels and mortality. Archives of Internal Medicine 162:2443-2449.

Mateo, R. J. Estrada, J.Y. Paquet, X. Riera, L. Domingues, R. Guitart, and A. Martinez-Vilata. 1999. Lead Shot Ingestión by Marsh Harriers (*Circus aeruginosus*) From the Ebro Delta, Spain. Environ. Pollut. 104, 435-440.

Mateo, R., M. Taggard, and A.A. Meharg. 2003. Lead and Arsenic in Bones of Birds of Prey From Spain, Env. Poll. 126:107-114.

Menke, A., P. Muntner, V. Batuman, E. K. Silbergeld, and E. Guallar. 2006. Blood lead below 0.48 μ mol/L (10 μ g/dL) and mortality among US adults. Circulation 114:1388-1394.

Michael, P. 2006. Fish and Wildlife Issues Related to the Use of Lead Fishing Gear. Washington State: Washington Department of Fish and Wildlife, Fish Program. http://www.wa.gov/fish/papers/lead_fishing_gear/fpt_06-13.pdf.

Miller, M.J.R., M. Restani, A.R. Harmata, G.R. Bortolotti, and M.E. Wayland. 1998. A Comparison of Blood Lead Levels in Bald Eagles From Two Regions on the Plains of North America. Journal of Wildlife Diseases 34:704-714.

Minnesota Office of Environmental Assistance (MOEA). 2006. Let's Get the Lead Out! Non-Lead Alternatives for Fishing Tackle. www.pca.state.mn.us/oea/reduce/sinkers.cfm.

National Toxicology Program (NTP). 2011. Draft NTP Monograph on Health Effects of Low-Level Lead. U.S. Department of Health and Human Services. http://ntp.niehs.nih.gov/?objectid=98DAF3E2-E316-D8D9-A4F834B80E0EE1C4

Needleman, H.L., A. Schell, D.M. Bellinger, A. Leviton, and E.N. Allred. 1990. The long-term effects of exposure to low doses of lead in childhood. An 11-Year follow up report. New England Journal of Medicine 322(2):83-88.

Needleman, H.L. and D.M. Bellinger. 1991. The health effects of low level exposure to lead. Annual Review of Public Health12:111-140.

Needleman, H.L. 1999. History of Lead Poisoning in the World. International Conference on lead Poisoning Prevention and Treatment, Bangalore, February 8-10, 1999. Bangalore, India: The George Foundation. www.leadpoisoning.net/general/history.htm.

Needleman, H.L. 2004. Lead Poisoning. Annual Review of Medicine 55:209-222.

Needleman, H.L., C. McFarland, R.B. Ness, S.E. Fienberg, and M.J. Tobin. 2002. Bone Lead Levels in Adjudicated Delinquents: A Case Control Study. Neurotoxicology and Teratology 24:711-717.

Nevin, R. 2000. How Lead Exposure Relates to Temporal Changes in IQ, Violent Crime, and Unwed Pregnancy. Environmental research Section A 83:1-22.

Nordic Council of Ministers (NCM). 2003. Lead Review. Nordic Council of Ministers Report 1, Issue 4. www.norden.org/pub/miljo/miljo/sk/US20031308.pdf.

Nriagu, J.O. 1983. Lead and Lead Poisoning in Antiquity. John Wiley and Sons. New York.

Nriagu, J.O. 2009. History in lead and lead poisoning in history. Abstract *in* R. T. Watson, M. Fuller, M. Pokras, and W. G. Hunt (Eds.). Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans. The Peregrine Fund, Boise, Idaho, USA.

Pain, D.J. 1992. Lead Poisoning of Waterfowl: A Review. Pages 7-13 *in* D.J. Pain, editor. Lead Poisoning in Waterfowl. Proceedings of the International Waterfowl and Wetlands Research Bureau Workshop, Brussels, Belgium 1991. IWRB Special Publication 16, Slimbridge, U.K.

Pain, D.J. 1996. Lead in Waterfowl. *Environmental Contaminants in Wildlife: Interpreting Tissue Concentrations*. W.M. Beyer, G.H. Heinz, and A.W. Redman-Norwood (eds.), pp. 251-262.

Pain, D.J. and B.A. Rattner. 1988. Mortality and Hematology Associated With the Ingestion of One Number Four Lead Shot in Black Ducks, Anas rubripes. Bull. Environ. Contam. Toxicol. 40, 159-164.

Pattee, O.H., S.N. Wiemeyer, B. Mulhern, L. Sileo, and J.W. Carpenter. 1981. Experimental Leadshot Poisoning in Bald Eagles. J. Wildl. Manage. 45:806-810.

Pattee, O.H., P.H. Bloom, J.M. Scott, and M.R. Smith. 1990. Lead Hazards Within the Range of the California Condor. The Condor 92:931-937.

Pattee, O.H. and D.J. Pain. 2003. Lead in the Environment. Handbook of Ecotoxicology, D.J. .Hoffman, B.A. Rattner, G.A. Burton, and J. Cairns (eds.), pp. 373-408.

Perrins, C.M., G. Cousquer, and J. Waine. 2003. A Survey of Blood Lead Levels in Mute Swans *Cygnus olor*. Avian Pathology 32:205-212.

Pokras, M.A. and R. Chafel. 1992. Lead Toxicosis From Ingested Fishing Sinkers in Adult Common Loons (*Gavia immer*) in New England. Journal of Zoo and Wildlife Medicine 23:92-97.

Pokras, M.A., M.R. Kneeland, A. Major, R. Miconi, and R.H. Poppenga. 2009. Lead objects ingested by Common Loons in New England. Extended abstract *in* R. T. Watson,

M. Fuller, M. Pokras, and W. G. Hunt (Eds.). Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans. The Peregrine Fund, Boise, Idaho, USA.

Quortrup, E.R. and J.E. Shillinger. 1941. 3,000 wild bird autopsies on western lake areas. American Veterinary Medical Association Journal.

Radomski, P., T. Heinrich, T.S. Jones, P. Rivers, and P. Talmage. 2006. Estimates of Tackle Loss for Five Minnesota Walleye Fisheries. North American Journal of Fisheries Management 26:206-212.

Rattner, B.A., J.C. Franson, S.R. Sheffield, C.I. Goddard, N.J. Leonard, D. Stang, and P.J. Wingate. 2008. Sources and Implications of Leadbased Ammunition and Fishing Tackle to Natural Resources. Wildlife Society Technical Review. The Wildlife Society, Bethesda, Maryland, USA.

Redig, P. T. 1985. A report on lead toxicosis studies in bald eagles. Final Report, U. S. Dept. of Interior, Fish and Wildlife Service Project No. BPO #30181-0906.

Redig, P.T., C.M. Stowe, D.M. Barnes, and T.D. Arent. 1980. Lead Toxicosis in Raptors. J. Am. Vet. Assoc. 177:941-943.

Redig, P.T., E.M. Lawler, S. Schwartz, J.L. Dunnette, B. Stephenson, and G.E. Duke. 1991. Effects of Chronic Exposure to Sublethal Concentrations of Lead Acetate on Heme Synthesis and Immune Function in Red-Tailed Hawks. Arch. Environ. Contam. Toxicol. 21:72-77.

Reiser, M.H. and S.A. Temple. 1981. Effects of Chronic Lead Intoxication on Birds of Prey. Recent advances in the study of raptor diseases, 21-25. J.E. Cooper and A.G. Greenwood (eds.), pp. 21-25.

Ris, M. D., K.N. Dietrich, P.A. Succop, O.G. Berger and R.L. Bornschein. 2004. Early exposure to lead and neuropsychological outcome in Adolescence. Journal International Neuropsychological Society 10: 261-270.

Sanderson, G.C. and F.C. Bellrose. 1986. A Review of the Problem of Lead Poisoning in Waterfowl. Ill. Nat. Hist. Surv. Spec. Publ. 4.

Scheuhammer, A. M. 1987. The chronic toxicity of aluminum, cadmium, mercury, and lead in birds: a review. Environmental Pollution 46:263-295.

Scheuhammer, A.M. 2009. Historical perspective on the hazards of environmental lead from ammunition and fishing weights in Canada. *In* R. T. Watson, M. Fuller, M. Pokras, and W. G. Hunt (Eds.). Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans. The Peregrine Fund, Boise, Idaho, USA.

Scheuhammer, A.M. and S. L. Norris. 1995. A review of the environmental impacts of lead shotshell ammunition and lead fishing weights in Canada. Canadian Wildlife Service, Environment Canada, Ottawa.

Scheuhammer, A.M. and S.L. Norris. 1996. The Ecotoxicology of Lead Shot and Lead Fishing Weights. Ecotoxicology 5:279-295.

Scheuhammer, A.M., S.L. Money, D.A. Kirk, and G. Donaldson. 2003. Lead Fishing Sinkers and Jigs in Canada: Review of Their Use Patterns and Toxic Impacts on Wildlife. Occasional Paper 108. Canadian Wildlife Service, Environmental Canada, Ottawa, Ontario, Canada.

Schnaas, L., S. J. Rothenberg, M-F. Flores, S. Martinez, C. Hernandez, C. Osorio, et al. 2006. Reduced intellectual development in children with prenatal lead exposure. Environmental Health Perspectives 114:791-797.

Schulz, J.H., J.J. Millspaugh, A.J. Bermudez, X. Gao, T.W. Bonnot, L.G. Britt, and M. Paine. 2006. Acute Lead Toxicosis in Mourning Doves. Journal of Wildlife Management 70:413-421.

Sears, J. 1988. Regional and Seasonal Variations in Lead Poisoning in Mute Swan *Cygnus olor* in Relation to the Distribution of Lead and Lead Weights in the Thames Area, England. Biological Conservation 46:115-134.

Sears, J. and A. Hunt. 1991. Lead Poisoning in Mute Swans, *Cygnus olor*, in England. Pages 383-388 *in* J. Sears and P.J. Bacon, editors. Wildfowl. Supplement 1, Third IWRB International Swan Symposium. The Wildfowl & Wetlands Trust and the International Waterfowl and Wetlands Research Bureau, Slimbridge, U.K.

Shillinger, J. E., and C. C. Cottam. 1937. The importance of lead poisoning in waterfowl. Transactions of the North American Wildlife Conference 2:398-403.

Sidor, I.F., M.A. Pokras, A.R. Major, R.H. Poppenga, K.M. Taylor, and R.M. Miconi. 2003. Mortality of Common Loons in New England, 1987 to 2000. Journal of Wildlife Diseases 39:306-315.

Sileo, L., L. H. Creekmore, D. J. Audet, M. R. Snyder, C. U. Meteyer, J. C. Franson, L. N. Locke, M. R. Smith, and D. L. Finley. 2001. Lead poisoning of waterfowl by contaminated sediment in the Coeur d'Alene River. Archives of Environmental Contamination and Toxicology 41:364–368.

Simpson, J. 2001. Weighting Environmentally Friendly Metal Weight Alternatives for Change. Walleye In-Sider Magazine 12:56-57.

Simpson, V.R., A.E. Hunt, and M.C. French. 1979. Chronic Lead Poisoning in a Herd of Mute Swans. Environmental Pollution 18:187-202.

Snyder, N.F., H.A. Snyder, J.L. Lincer, and R.T. Reynolds. 1973. Organochlorines, Heavy Metals, and the Biology of North American Accipiters. Bioscience 23, 300-305.

Staessen, J.A., R.R. Lauwerys, C.J. Bulpitt, R. Fagard, P. Linjen, H. Roels, L. Thijs, and A. Amery. 1994. Is a Positive Association Between Lead Exposure and Blood Pressure Supported by Animal Experiments? Current Opinion in Nephrology and Hypertension 3:257-263.

Stansley, W., L. Widjeskog, and D.E. Roscoe. 1992. Lead Contamination and Mobility in Surface Water at Trap and Skeet Ranges. Bulletin of Environmental Contamination and Toxicology 49:640-647.

Stone, W.B. and J.C. Okoniewski. 2001. Necropsy Findings and Environmental Contaminants in Common Loons from New York. Journal of Wildlife Diseases 37:178-184.

Strom, S.M., J.A. Langenberg, N.K. Businga, and J.K. Batten. 2009. Lead exposure in Wisconsin birds. *In* R. T. Watson, M. Fuller, M. Pokras, and W. G. Hunt (Eds.). Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans. The Peregrine Fund, Boise, Idaho, USA.

Stutzenbaker, C.D., K. Brown, and D. Lobpries. 1983. An assessment of the accuracy of documenting waterfowl die-offs in a Texas coastal marsh. Special Report. Federal Aid Project W-106-R, Texas Parks and Wildlife Department, Austin, Tex. 21 p.

Tong, S., Y.E. von Schirnding, and T. Prapamontol. 2000. Environmental Lead Exposure: A Public Health Problem of Global Dimensions. Bulletin of the World Health Organization 78:1068-1077.

U. S. Department of Health and Human Services (USDHHS). 1999. Toxicological profile for lead. Agency for Toxic Substances and Disease Registry, July 1999. Available at http://www.atsdr.cdc.gov/toxprofiles/tp13.pdf/.

United States Environmental Protection Agency (USEPA). 1994. Lead Fishing Sinkers: Response to Citizens' Petition and Proposed Ban; Proposed Rule. Federal Register Part III, Volume 40, part 745:11121-11143.

United States Environmental Protection Agency (USEPA). 1999. 1999-2000 Refuge-Specific Hunting and Sport Fishing Regulations: Proposed Rule. Federal Register Volume 64, Number 154:43834-43854.

United States Environmental Protection Agency (USEPA). 2004. Humans and Lead Fishing Sinkers. www.epa.gov/owowwtr1/fish/humans.html.

United States Environmental Protection Agency (USEPA). 2009. Lead Wheel Weights; Regulatory Investigation. Available at http://yosemite.epa.gov/opei/rulegate.nsf/byRIN/2070-AJ64?opendocument.

United States Environmental Protection Agency (USEPA). 2010a. Request for comments on petition to phase out leaded aviation gasoline. http://www.epa.gov/otaq/aviation.htm.

United States Environmental Protection Agency (USEPA). 2010b. Advance Notice of Proposed Rulemaking on Lead Emissions from Piston-Engine Aircraft Using Leaded Aviation Gasoline: Regulatory Announcement. http://www.epa.gov/nonroad/aviation/420f10013.htm

United States Environmental Protection Agency (USEPA). 2011a. National Lead Free Wheel Weight Initiative. http://www.epa.gov/wastes/hazard/wastemin/nlfwwi.htm

United States Environmental Protection Agency (USEPA). 2011b. Humans and Lead Fishing Sinkers. http://water.epa.gov/scitech/swguidance/fishshellfish/humans.cfm

United States Fish and Wildlife Service (USFWS). 1999. Establishing "Lead Free Fishing Area" and the Prohibition of the Use of Certain Fishing Sinkers and Jigs Made With Lead on Specific Units of the National Wildlife Refuge System. Federal Register 64:17992.

United States Fish and Wildlife Service (USFWS). 2004. Status Assessment and Conservation Plan for the Common Loon in North America.

U.S. Geological Survey (USGS). 2008 Lead Shot and Sinkers: Weighty Implications for Fish and Wildlife Health. USGS Press Release 7/11/2008.

Veit, H.P., R.J. Kendall, and P.F. Scanlon. 1982. The Effect of Lead Shot Ingestion on the Testes of Adult Ringed Turtle Doves (*Streptophelia risoria*). Avian Dis. 27, 442-452.

Victery, W. 1988. Evidence for Effects of Chronic Lead Exposure on Blood Pressure in Experimental Animals: An Overview, Environmental Health Perspectives 78:71-76.

Washington Department of Fish and Wildlife (WDFW) 2000. Unpublished data. Available at http://wdfw.wa.gov/fish/papers/lead_fishing_gear/index.htm.

Watson, R.T. and D. Avery. 2009. Hunters and anglers at risk of lead exposure in the United States. *In* R. T. Watson, M. Fuller, M. Pokras, and W. G. Hunt (Eds.). Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans. The Peregrine Fund, Boise, Idaho, USA.

Windingstad, R.M., S.M. Kerr, L.N. Locke, and J.J. Hunt. 1984. Lead Poisoning of Sandhill Cranes (*Grus Canadensis*). Prairie Nat. 16, 21-24.

Woolf, A., J.R. Smith, and L. Small. 1982. Metals in livers of white-tailed deer in Illinois. Bulletin Environmental Contamination Toxicology 28:189-194.

Wright, J.P., K. N. Dietrich, M. D. Ris, R. W. Hornung, S. D. Wessel, B. P. Lanphear, et al. 2008. Association of prenatal and childhood blood lead concentrations with criminal arrests in early adulthood. PLoS Med 5:732-740.

Wu, T., Buck, G. M., and Mendola, P. 2003. Blood lead levels and sexual maturation in U.S. girls: the Third National Health and Nutrition Examination Survey, 1988-1994. *Environ Health Perspect* **111**, 737-741.

Zdziarski, J.M., M. Mattix, R.M. Bush, and R.J. Montali. 1994. Zinc Toxicosis in Diving Ducks. Journal of Zoo and Wildlife Medicine 25:438-445.