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The is a Comment on the **Fish and Wildlife Service** (FWS) Proposed Rule: **Migratory Bird Permits: Management of Conflicts Associated with Double-Crested Cormorants (Phalacrocorax auritus) Throughout the United States**

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Comment

Please see the Center for Biological Diversity's attached comment letter. I also attached an exhibit demonstrating the harm from lead ammunition. Copies of the studies cited in that exhibit can be downloaded here: https://diversity.app.box.com/folder/111814171391. I will also attempt to upload those studies as a series of separate comments.

Attachments (1)

DCCO DEIS comments by CBD 7 20 2020 with lead

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Submitted via Federal eRulemaking Portal

July 20, 2020

Public Comments Processing, Attn: FWS-HQ-MB-2019-0103
U.S. Fish and Wildlife Service; MS: PRB (JAO/3W)
5275 Leesburg Pike; Falls Church, VA 22041-3803

Dr. Eric L. Kershner
Chief, Branch of Conservation, Permits, and Regulations
FWS/DOI
5275 Leesburg Pike, MS: MB
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Re: Management of Double-crested Cormorants throughout the United States (FWS-HQ-MB-2019-0103)

Dear Mr. Kershner:

My name is Collette Adkins, and I am an attorney and biologist at the Center for Biological Diversity. For the reasons explained below, the Center urges the U.S. Fish and Wildlife Service ("FWS") to not adopt its "preferred alternative," which would establish a new permit system to allow states and tribes to kill double-crested cormorants. Instead, we offer a science-based alternative that the FWS should analyze and adopt.

The Center is a national, nonprofit organization with over 1.7 million members and supporters nationwide. Our mission is to protect and restore rare animals and their habitats through science, policy, education, advocacy, and environmental law. Our members appreciate the important ecological roles played by double-crested cormorants and enjoy opportunities to view these beautiful native birds in the wild.

I. The FWS Has Failed To Establish A Real Need For The Proposed Action

To begin, the FWS has no definitive data showing that providing additional authority to kill cormorants is necessary to reduce conflicts. Even with years of control under the previous depredation orders and permits, the agency still cannot document a feedback loop between management action and resource response, and FWS has offered no substantial evidence that killing cormorants under the public resource depredation order has helped rebuild fish populations weakened primarily by threats like overfishing and pollution. Cormorants are being unreasonably targeted for control given that gamefish constitute a small portion of their total

diet. Any "conflict" must be first established with evidence and not merely by complaint, given that myths and negative perceptions have led to irrational targeting of cormorants.[1]

Second, even if data substantiating impacts existed, killing cormorants – or any other predator – to serve narrow economic or recreational interests is inappropriate. Cormorants, like other predators, play an important biological role by eliminating prey weakened by disease or other factors. Protection of cormorants serves the interests of a wider segment of the public who enjoys birdwatching and recreating in ecosystems with functioning top predators. Moreover, the suffering that cormorants have endured under the depredation orders and permits is unethical and intolerable.

II. The DEIS Fails To Take The Requisite "Hard Look" At Impacts

While we appreciate that the FWS has prepared a DEIS, rather than an "environmental assessment," the DEIS lacks the rigorous analysis that NEPA requires. We ask that the FWS address the following deficiencies in its analysis prior to issuing a Final Environmental Impact Statement:

- Cumulative impacts of other cormorant killing, such as the planned hunting seasons in Ontario, Canada;
- Impacts on humane, aesthetic, and recreational values. FWS should analyze animal welfare issues such as the potential for animal suffering that would arise from the orphaning of chicks and the use of cervical dislocation to kill such a large bird. It should consider impacts to those of us who oppose killing cormorants, who enjoy observing them, and who might be forced to view birds harmed by the authorized control;
- Localized impacts to cormorant populations in areas where they are less abundant, such as the Western population along the West Coast. For example, the FWS did not analyze the possibility of colony collapse with information gained after control decimated the population on East Sand Island. In particular, the possibility of collapse should be analyzed given the preferred alternative would allow control of active colonies during the nesting season with invasive methods such as firehoses;
- Adverse impacts to waterbirds that may roost with cormorants or endangered species that can be found in proximity to cormorants (like streaked horned larks) that could be disturbed by control actions;
- Economic benefits of cormorants, such as in consuming invasive and predatory fish that may impact federal listed species and commercially valuable fish species.

III. The FWS Unreasonably Selected The "Preferred Alternative"

In selecting the preferred alternative, the FWS made an unreasonable decision for numerous reasons:

[1] Wires, L. R. 2014. The double-crested cormorant, plight of a feathered pariah. Yale University Press, New Haven, Connecticut, USA.

- By not adequately defining "conflict," the preferred alternative allows states to prioritize recreational fishing opportunities over the FWS's duties to protect cormorants under the Migratory Bird Treaty Act. It would allow states and tribes to kill cormorants for eating wild or stocked fish without first demonstrating the need for such action. The justification cannot be simply that cormorants were eating fish because that is what they naturally do;
- The FWS needs to analyze whether killing cormorants would help the impacted fish by addressing compensatory vs. additive predation and the complexities of introduced species in cormorant diets. The FWS justifies its selection of the preferred alternative with five case studies that cannot be relied upon – for the reasons stated in the comment letter by Linda Wires;
- The preferred alternative does not explain how states and tribes would demonstrate satisfaction of the requirement to first use nonlethal methods to address conflicts;
- Allowing states and tribes to designate "subpermittees" – without further defining the level of training and control required of these agents – could result in cormorant killing by people unable to operate in a humane, accountable, and lawful manner;
- The preferred alternative provides only a nationwide cap on the number of cormorants killed but only recommends – does not require – less liberal killing in areas with fewer cormorants. The preferred alternative would result in a large increase in the number of cormorants killed, when compared to previous years – without evidence of any increase in conflict that could justify higher kill numbers.
- The preferred alternative would allow use of toxic lead ammunition, despite abundant evidence of impacts from lead ammunition. **See Exhibit A, attached**; and
- The FWS explains that the agency will later develop a monitoring protocol but that program should be developed and analyzed now, as part of the NEPA process.

IV. The DEIS Fails To Examine A Reasonable Range Of Alternatives

The FWS's preferred alternative allows continuance of the same environmentally flawed practices that created the problems in the first place, setting up an indefinite attack on these native waterbirds. Instead, we ask that the FWS analyze and adopt the alternative proposed in the July 18, 2020 comments by Linda Wires. Under this proposed alternative, the FWS would maintain authority to issue permits on a case-by-case basis, rather than establish a permit system that would relegate authority to states and tribes. The need for control and the proper response would be assessed on a site-specific basis. Control would be authorized only exhaustion of nonlethal methods and any control efforts would have specific goals and an established protocol for monitoring progress toward that goal. The FWS would be tasked with increasing "social carrying capacity" of cormorants by educating the public about cormorants in key areas where conflicts have previously occurred. Other reasonable alternatives include (as suggested in comments submitted by Bob Sallinger of Portland Audubon): (1) lower maximum take thresholds at a national and regional scale; and (2) use of non-lethal strategies only to accomplish desired outcomes. It is very troubling that all four action alternatives set the exact same national and regional thresholds for allowed lethal take, even though commenters, including Dan Roby, have explained that the vulnerabilities of the Western population and the devasting impact of control at East Sand Island. The FWS should have also considered an alternative that would have required use of nontoxic ammunition.

* * *

Thank you for providing this opportunity to comment, and please do not hesitate to contact me with questions or concerns. Please notify me with opportunities to participate in this decision-making process.

Sincerely,

Collette Adkins, Senior Attorney
Carnivore Conservation Director
Center for Biological Diversity

Exhibit A.
Summary of Lead Poisoning in Wildlife

Authorizing hunting which allows the use of lead ammunition can result in widespread and uncontrolled introduction of toxic lead into the environment and the food chain, and significant toxic lead exposure for wildlife.

Hunting or sport shooting with lead ammunition exposes any animal that preys or scavenges on targeted wildlife to lead's toxic effects. Particularly susceptible are avian scavengers that encounter lead in carcasses left in the wild, in gut piles (viscera) from animals cleaned in the wild, and in wounded prey species that survive hunting and carry lead bullets, shot or fragments in their bodies. Raptors such as bald and golden eagles and endangered California condors are particularly sensitive to small amounts of lead. Eagles and condors are frequently killed by lead poisoning or suffer chronic sub-lethal effects of lead poisoning from scavenging meat containing lead fragments from ammunition.

Upland game hunting with lead ammunition results in widespread distribution of spent lead, which can accumulate in both terrestrial and seasonally aquatic habitats. More than 130 species of wildlife (including mammals, upland birds, raptors, waterfowl, amphibians and reptiles) have been reported in scientific literature as being exposed or killed by ingesting lead shot, bullets, bullet fragments or prey contaminated with lead ammunition.

These significant impacts led the U.S. Fish and Wildlife Service ("FWS") to enact prohibitions on the use of lead shot for waterfowl hunting. 50 C.F.R. §§ 2021(j)(2); 50 C.F.R. § 20.134 ("[w]e will not approve as nontoxic any shot type or shot coating with a lead content of 1 percent or more"). FWS more recently found that the "[e]xposure to lead ammunition and fishing tackle has resulted in harmful effects to fish and wildlife." (USFWS 2017). FWS further stated that hunting with lead ammunition poses "an ongoing risk to upland or terrestrial migratory birds and other species that ingest spent shot directly from the ground or as a result of predating or scavenging carcasses that have been killed with lead ammunition and left in the field." (USFWS 2017). Based on the finding FWS proposed programs to support nontoxic ammunition and phase out lead ammunition (USFWS 2017). While Secretary of Interior Ryan Zinke later determined to revoke the Fish and Wildlife Service's Director's order on the use of nontoxic ammunition and fishing tackle based on the determination that it was "not mandated" or following significant "coordination with affected stakeholders" the FWS did not revoke the findings regarding the ongoing dangers of lead ammunition and tackle (DOI 2017). The scientific evidence of the dangers of lead ammunition and tackle on the environment and wildlife is well established in peer reviewed research, including literature surveys conducted by FWS staff (Golden 2016, Pain 2019).

Below we have compiled a survey of some of the important scientific studies demonstrating the significant impacts of lead ammunition.

Sources and Quantities of Lead in the Environment from Hunting and Shooting Sports

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

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

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

To give an idea of the quantity of potentially lead-tainted carcasses available to scavengers, Fry and Maurer (2003) quantified hunter-shot carcasses available to condors in their California range before the California lead ammunition ban went into effect, and concluded that gut piles and whole carcasses left in the field by hunters were a highly significant source of lead within the condor range. From hunting survey data for the eight counties encompassing the condor range in California at that time, Fry and Maurer (2003) estimated an annual average of 36,000 big game animals (17,000 wild pigs, 11,000 coyotes and 8,000 deer) were taken each year by sport hunters in this area. Fry and Maurer (2003) assumed that only a very few gut piles are actually buried, hidden successfully, or removed from the field. Deer and pigs are generally field dressed and gut piles discarded in the field; coyotes are generally left in the field intact. The Fry and Maurer (2003) figures do not account for poaching, which likely significantly increases the number of deer carcasses available. The data also do not account for the thousands of pigs and deer shot by ranchers under depredation permits or small game such as ground squirrels shot by varmint hunters. The carcasses of large animals left in the field would be the primary source of hunter-shot food for condors, although condors and other scavengers will eat smaller animals as well.

Fry and Maurer (2003) estimated that almost 28,000 tree squirrel, rabbit, and ground squirrel carcasses are left in the field within the condor range annually. Even animals as small as ground squirrels shot with .22 caliber bullets can contain lead fragments at biologically relevant levels that may constitute a lead-hazard for scavenging birds of prey (Harmata and Restani 1995: Knopper et al. 2006).

Sources of Lead in the Environment from Fishing

Waterbirds are lead poisoned from ingesting lead fishing sinkers or jigs lost by anglers on the bottom of water bodies. Sport anglers attach lead weights to fishing lines to sink the hook, bait, or lure into the water. Some anglers use lead-weighted hooks, called jigs. A sinker or jig can accidentally detach from a line and fall into the water or the hook or line may become tangled and the line may break or be cut. Aquatic birds may ingest lead objects while collecting gizzard stones or by preying on live bait or escaped fish with attached fishing gear. Many ducks and other water birds forage for food in the mud at the bottom of lakes. Most of these birds also swallow small stones and grit that aid in grinding up their food. Some of the grit may contain lead from fishing tackle. The hazards and alternatives to lead fishing tackle are well known (MPCA 1, MPCA 2, MDNR 2019).

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 (USEPA 1994). 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).

Lead fishing sinkers and jigs are documented to cause lead poisoning in numerous species of water birds and wading birds, and the problem is particularly acute for mute swans (*Cygnus olor*), trumpeter swans (*Cygnus buccinator*), sandhill cranes (*Grus canadensis,*), and common loons (*Gavia immer*) (Locke et al. 1982; Windingstad et al. 1984; Pain 1992; USEPA 1994; Scheuhammer and Norris 1995, 1996; Rattner et al. 2009; Friend 1999). The U.S. Environmental Protection Agency believes that over 75 individual species are potentially at risk from exposure to lead- and zinc-containing fishing sinkers based on their feeding habits and sources of food. (USEPA 1994).

Pathways of Lead Exposure for Wildlife

Lead from shot, bullets and fragments in heavily hunted fields and shooting areas can be directly ingested or solubilized and biologically incorporated into food items (Ma 1989; Stansley and Roscoe 1996; Hui 2002).

There is extensive documentation of direct ingestion of lead shot and bullet fragments by dabbling and diving ducks, swans, loons and other water birds, and other marsh birds feeding in wetland areas that are hunted with lead ammunition can ingest spent lead, such as flamingoes, rails, shorebirds, terns and herons (Artman and Martin 1975; Kaiser et al. 1980; Maedgen et al. 1982; Custer and Mulhern 1983; Hall and Fisher 1985; Locke et al. 1991; Beck 1997; Mateo et al. 1997; Acora 2005). Numerous species of birds are at risk of lead poisoning from ingesting spent lead shot, often mistaking shot pellets for food, grit or bone fragments.

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

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

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

Reports from experimental and field observations conclude that all bird species would be susceptible to lead poisoning after ingesting and retaining shot in the gastrointestinal system (Fisher et al. 2006). Raptor and scavenger species that feed on animals killed with lead

ammunition would be at high risk for exposure to lead in this way. Animals that consume lead particles that have fragmented in hunter-killed carcasses may be at particular risk because the small size and irregular shape of fragments make them more absorbable in the digestive process (Fisher et al. 2006).

Fisher et al. (2006) listed fifty-nine terrestrial bird species worldwide that have been exposed to lead from ammunition sources, including raptors, galliforms, gruiforms, columbiforms, and gulls. Vyas et al. (2000, 2001) identified lead in songbirds resident on a shotgun trap and skeet range. Fisher et al. (2006) reviewed published literature on lead poisoning of 32 species of wild birds in the United States from spent lead ammunition. Documented cases of ingestion and poisoning by lead from ammunition in terrestrial birds globally include 33 raptor species and 30 species from *Gruiformes*, *Galliformes* and various other avian taxa, including ten globally threatened or near threatened species (Pain et al. 2009). Lead poisoning is of particular conservation concern in long-lived slow breeding species, especially those with initially small populations. A review by the Minnesota Department of Natural Resources found over 130 species of animals (including upland birds, raptors, waterfowl, and reptiles) have been reported in scientific literature as being exposed or killed by ingesting lead shot, bullets, bullet fragments or prey contaminated with lead ammunition (Tranel and Kimmel 2009). In the United States, Kendall et al. (1996) found that upland game birds ingest substantial amounts of lead shotgun pellets and deduced that raptors must incur secondary ingestion of pellets because their prey ingested it. Rifle-shot prairie dogs and ground squirrels may contain fragmented lead particles that could be ingested by scavengers or raptors (Knopper et al. 2006; Pauli and Buskirk 2007). Kramer and Redig (1997) compiled data on more than 2,000 bald eagles, demonstrating that lead shot pellets, likely from crippled waterfowl and lead fragments in offal and unrecovered deer carcasses, were responsible for elevated lead levels in more than 98% of birds admitted to a veterinary hospital and raptor center. Studies have definitively linked isotopically labeled lead in California condors with rifle bullets sold in the same region, substantiating that condors are ingesting lead from hunting sources and that condor lead poisoning deaths and sublethal effects are solely from lead bullet fragments (Scheuhammer et al. 2003a; Church et al. 2006; Chesley et al. 2009; Finkelstein et al. 2010).

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

Raptors and other scavenging birds are usually poisoned through ingesting lead shot or bullet fragments in dead or injured prey or gut piles (Friend 1987; Kendall et al. 1996). Common

ravens have been shown to have elevated blood lead levels during hunting season due to ingestion of lead in rifle-shot big game offal piles (Hatch 2006; Craighead and Bedrosian 2007, 2008). In Canada, upland game birds and mammals, the primary food source of many raptors, are now more likely to contain lead shot than waterfowl, as lead shot is prohibited for waterfowl hunting (Clark and Scheuhammer 2003).

Toxic Effects of Lead on Wildlife

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

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

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

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

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

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

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

The effects of lead toxicosis in birds commonly include distension of the proventriculus, green watery feces, weight loss, anemia and drooping posture (Hanzlik 1923; Quortrup and Shillinger 1941; Redig et al. 1980; Reiser and Temple 1981; Franson et al. 1983; Custer et al. 1984; Sanderson and Bellrose 1986; Mateo 1998). Sub-lethal toxic effects are exerted on the nervous system, kidneys and circulatory system, resulting in physiological, biochemical and behavioral changes (Scheuhammer 1987). Vitamin metabolism can be affected (Baski and Kenny 1978) and birds can go blind (Pattee et al. 1981). Lead toxicosis depresses the activity of certain blood

enzymes, such as delta aminolevulinic acid dehydratase, essential for cellular energy and hemoglobin production, and may impair immune function (Redig et al. 1991; Grasman and Scanlon 1995). Over longer periods, haematocrit and hemoglobin levels are often reduced. Finkelstein et al. (2010) found that sub-lethal concentrations of lead in blood (20 μg/dL), resulted in a 60% decrease in the levels of aminolevulinic acid dehydratase in condors.

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

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

Burger and Gochfeld (2000) found that chronic lead exposure resulted in delayed behavioral response time in both laboratory and wild herring gulls (*Larus argentatus*). Kelly and Kelly (2005) documented moderately elevated blood lead levels increased the risk of collision with overhead power lines for mute swans (*Cygnus olor*). Mallards (*Anas platyrhynchos*) experimentally fed lead exhibited hemolytic anemia during the first week of exposure and neurological impairment during the second week (Mateo et al. 2003). In experimentally fed turkey vultures (*Cathares aura*) and bald eagles (*Haliaeetus luecocephalus*), lead ingestion decreased weight and muscle mass and caused blindness (Pattee et al. 1981, 2003). Blood pressure increases and renal damage have also been observed in rodents after experimental lead exposure (Victery 1988; Staessen et al. 1994). Bagchi and Preuss (2005) found that acute lead exposure had lasting effects including lowered bone density and increased blood pressure one year after exposure in laboratory rats.

In spite of the abundance of evidence that lead is toxic to wildlife, poisoning rates are not well understood. While massive die-offs are readily visible, daily losses of individual animals are

more difficult to detect. This is because sick animals will often isolate themselves, and then are quickly predated upon after death. In one study, observers were given 30 minutes to discover 100 placed carcasses and only found 6 (Stutzenbaker et al. 1983). In another study in which researchers planted carcasses, over 60% of the carcasses were gone within 3 days and over 90% were gone within 8 days (Humburg et al. 1983; Stutzenbaker et al. 1983).

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

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

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

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

Bald Eagles (*Haliaeetus leucocephala*) and Golden Eagles (*Aquila chrysaetos*)

Bald eagles share some demographic and ecological factors with free-ranging condors that make them vulnerable to lead intoxication; they scavenge on carcasses, they are long-lived, they have low recruitment rates, and their numbers have been reduced in recent decades (Pattee et al. 1990). Bald eagles that ingest lead shot embedded in the tissues or the intestinal tract of waterfowl demonstrate acute and chronic symptoms of lead poisoning (Hoffman et al. 1981; Miller et al. 2001). The experimental intoxication of bald eagles with lead shot conducted by Pattee et al. (1981) found that it took between 10 and 133 days (median 20 days) for mortality to occur. The range of time for lead shot retention in the stomach varied between 0.5 and 48 days. Mean lead levels in dead animals were 16.6 ppm (wet weight) in liver and 6.0 ppm (wet weight) in kidney (Pattee et al. 1981). In a complementary study, Hoffmann et al. (1981) report mean blood lead levels in eagles dosed with 10 #4 lead shot (0.21g each) to be 80 µg/dl after 24 hours and 280 µg/dl after 72 hours. Mean blood lead levels as high as 270 µg/dl have been detected in apparently healthy free-ranging bald eagles but subclinical effects may be difficult to document (Reiser and Temple 1981). Foreign bodies, including lead fragments, may be regurgitated by

eagles so that fragments may not be detected in the gastrointestinal tract at the time of capture or blood tests, even if the fragments contributed substantially to elevated lead exposure levels prior to being ejected. Mateo et al. (2003) recognized the importance of accounting for this unique physiology in raptors and recommend collecting regurgitated pellets at raptor roosting sites to study the presence, frequency, seasonality and prey associated with the ingestion of lead shot.

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

Pattee and Hennes (1983) found that elevated lead levels in bald eagles corresponded well (89%) with late fall and winter waterfowl hunting seasons. However, a study attempting to trace lead poisoning in bald eagles to diet preference did not find significant differences in blood lead levels among eagles feeding on fish and eagles feeding on waterfowl in an area where waterfowl hunting was intensive (Miller et al. 1998). According to the Wisconsin Department of Natural Resources, about 15 to 20 percent of all bald eagle deaths in that state are due to lead poisoning (Eisele 2008; Strom et al. 2009), usually from eating animals that were wounded with lead ammunition or from scavenging gut piles during and after the deer hunting season. Wisconsin lead poisoning cases in bald eagles begin to increase in October, peak in December and tail off in late winter, which coincides exactly with Wisconsin's deer hunting seasons, suggesting hunter-crippled game and lead contaminated offal are the cause.

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

Golden and bald eagle feeding ecology and behaviors may expose them to some of the same factors that predispose condors to lead intoxication. In a study by Pattee et al. (1990) on the lead hazards within the California condor range, golden eagles were suggested as a model species to assess lead exposure in California condors because they are abundant in the condor range and they have been observed feeding on the same carcasses as condors. Between 1985 and 1986,

36% of the 162 golden eagles evaluated within the California condor range had elevated blood lead levels, and 2.5% had levels greater than 100ug/dl, indicative of clinical lead poisoning. This study also reported seasonal trends in lead levels in tissues of golden eagles within the California condor range which coincided with the deer hunting season (Pattee et al. 1990).

Wildlife rehabilitators in Iowa began gathering lead poisoning information on bald eagles in 2004, analyzing blood, liver, or bone samples for 62 eagles (Neumann 2009). Thirty-nine eagles showed lead levels in their blood above 0.2 ppm or lead levels in their liver above 6 ppm, which could be lethal poisoning without chelation treatment. Seven eagles showed exposure levels of lead (between 0.1 ppm and 0.2 ppm in blood samples, between 1 ppm and 6 ppm in liver samples, and between 10 ppm and 20 ppm in bone). Several of the eagles admitted with traumatic injuries showed underlying lead exposure or poisoning. Over half of the eagles being admitted to Iowa wildlife rehabilitators have ingested lead. Behavioral observations, time-of-year data analysis, and x-ray information point to lead shrapnel left in slug-shot white-tailed deer (*Odocoileus virginianus*) carcasses to be a source of this ingested lead (Neumann 2009). Thousands of bald eagles winter in Iowa, up to one fifth of the lower 48 states population.

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

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

Bedrosian and Craighead (2009) measured blood lead levels of 47 bald eagles and 16 golden eagles in the southern Yellowstone Ecosystem around Grand Teton National Park, Wyoming during and after large-game hunts for two years. They found a median blood lead level of 41.0 µg/dL (range = 3.2–523 µg/dL); 75% of all birds tested exhibited elevated lead levels (>20 µg/dL) and 14.3% exhibited levels associated with clinical poisoning (>100 µg/dL). The median blood lead levels for eagles during the hunting season was significantly higher than the non-hunting season (56.0 vs. 27.7 µg/dL, respectively; $P = 0.01$). The magnitude of lead in the blood of Wyoming eagles is extremely high and likely results in the death of some individuals (Bedrosian and Craighead 2009).

Following the ban on lead shot for waterfowl hunting, bald eagles continue to acquire elevated levels of lead from hunter-shot deer. Spent lead from ammunition is present in field residues of white-tailed deer (*Odocoileus virginianus*) (Cruz-Martinez et al. 2012). Cruz-Martinez et al. (2012) evaluated data from 1,277 bald eagles admitted for rehabilitation in Minnesota from

January 1996 through December 2009. They found that 334 bald eagles (26%) had elevated lead levels, and detected significantly increased odds for elevated lead levels based on season (late fall and early winter) and in hunting zones.

Bedrosian et al. (2012) investigated the incidence of lead exposure in bald eagles in Wyoming during the big game hunting season, and found that eagles had significantly higher lead levels during the hunt. Bedrosian et al. (2012) found 24% of eagles tested had levels indicating at least clinical exposure (>60 ug/dL) during the hunt while no birds did during the non-hunting seasons.

Franson and Russell (2014) evaluated demographic and pathologic characteristics in 484 bald eagles and 68 golden eagles diagnosed with lead poisoning at the U.S. Geological Survey National Wildlife Health Center in Wisconsin. Franson and Russell (2014) detected a distinct temporal trend in the collection date of lead poisoned bald eagle carcasses, corresponding with greater frequency during hunting season in late autumn and winter, than in spring and summer. Lead poisoning effects on eagles included emaciation, evidence of bile stasis, myocardial degeneration and necrosis, and renal tubular nephrosis and necrosis (Franson and Russell 2014). Franson and Russell (2014) found ingested lead ammunition or fragments in 14.2 % of bald eagles and 11.8 % of golden eagles.

Ecke et al. (2017) correlated lead levels in the blood of golden eagles in Sweden with progression of the moose hunting season. Based on analyses of tracking data, Ecke et al. (2017) found that even sublethal lead concentrations in blood can likely negatively affect golden eagle movement behavior (flight height and movement rate). Lead levels in liver of recovered post-mortem analyzed eagles also suggested that sublethal exposure increases the risk of mortality in eagles (Ecke et al. 2017).

Yaw et al. (2017) assessed 11 years (2004–2014) of bald eagle data from four wildlife rehabilitators in Iowa for the prevalence of elevated lead levels in blood or tissue samples. Yaw et al. (2017) found the highest blood lead levels in eagles during hunting season (October–January).

Other Raptors

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

Bobwhite, Quail, Doves, and Other Game Birds

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

Akoshegyi 1997; Keel et al. 2002; Battaglia et al. 2005; Butler 2005; Fisher et al. 2006, Schulz et al. 2006).

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

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

Franson et al. (2009) evaluated lead exposure in 4,884 hunter-harvested mourning doves from Arizona, Georgia, Missouri, Oklahoma, Pennsylvania, South Carolina, and Tennessee. The frequency of ingested lead pellets in gizzards of doves on hunting areas where the use of lead shot was permitted was 2.5%. On areas where non-lead shot was required, 2.4% of mourning doves had ingested steel shot. Doves without ingested lead pellets had lower bone lead concentrations in areas requiring the use of non-lead shot than in areas allowing the use of lead shot. Schulz et al. (2006) calculated from comparing hunting statistics and population estimates that nearly as many doves are poisoned lethally by ingesting lead shotgun pellets (8.8 million to15 million per year) as are shot by sport hunters on an annual basis. The number of mourning doves harvested in the U.S. is approximately 20 million birds annually.

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

American woodcock are exposed to lead on their breeding grounds in Wisconsin, resulting in high accumulations of lead in bone tissue (Strom et al. 2009). Bone lead concentrations considered to be toxic in waterfowl were observed in all age classes of woodcock; although

stable isotope analysis of bone samples was not able to conclusively identify the source of the lead, the data suggest a local and dietary source (Strom et al. 2009).

Cranes and Rails

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

Corvids

Scientists tested blood lead levels in 302 ravens that scavenged on hunter-killed large ungulates and their offal in and around Grand Teton National Park, Wyoming in 2004 and 2005 (Craighead and Bedrosian 2007, 2008). Blood-lead levels of ravens increased dramatically during hunting season, roughly five times higher than the rest of the year, likely due to ravens consuming lead bullet fragments left behind in gut piles of hunted elk, deer and moose. Blood samples were taken during a 15-month period spanning two hunting seasons, from mid-September 2004 to mid-December 2005. Forty-seven percent of the ravens tested during the hunting season exhibited elevated blood lead levels (≥10 µg/dL) while only 2% tested during the non-hunting season exhibited elevated lead levels. Offal is the primary food source of ravens during the time of exposure and Craighead and Bedrosian (2007) also identified un-retrieved offal piles of hunter-killed game as a point source for lead contamination in the area. These substantial increases in blood-lead levels correspond almost exactly with the open and close of hunting season. Just after the start of hunting season, blood-lead levels begin to rise. Shortly after the end of hunting season, they return to normal. Blood-lead levels show a spike again in the late spring, when melting snow uncovers gut piles left from the previous hunting season. One hundred percent of the ravens at the study site feed on gut piles at some point throughout the hunting season and get exposed to lead.

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

Craighead and Bedrosian also documented that the blood lead levels of ravens around Grand Teton dropped corresponding with increased use of non-lead ammunition by hunters on the National Elk Refuge and in Grand Teton National Park. In fall of 2009 researchers distributed 194 boxes of copper bullets to hunters with permits for the park and the refuge, captured 46

ravens (which typically scavenge the discarded gut piles) during hunting season and tested their blood for lead. An estimated 24% of hunters in the area used copper bullets in 2009, and there was a corresponding 28% drop in blood lead levels in ravens compared with what would have been expected (Hatch 2010).

Median blood lead concentrations of ravens captured during hunting season in northern California were almost 6-fold higher than those of birds captured during the non-hunting season (West et al. 2017).

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

Song Birds

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

Grizzly Bears, Black Bears, and Other Mammals

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

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