American Bird Conservancy July 2010

Petition to US EPA to regulate lead hunting and fishing components.

This factsheet is part of the data being presented by American Bird Conservancy, American Association of Avian veterinarians, Center for Biological Diversity, and Project Gutpile to the US EPA in a petition to eliminate lead bullets and shotgun pellets in sport hunting, and to require non-toxic fishing sinkers and lures. This petition is designed to halt lead deposition into the environment from hunting and fishing activities, because lost lead fishing gear and spent lead hunting ammunition cause the death of 10-20 million birds annually in the US. All of these lead containing products now have commercially available non-toxic alternatives, so that EPA can develop regulations to require non-toxic rifle bullets, shotgun pellets, and fishing weights and lures throughout the US. Several states, the National Park Service, and the Wildlife Services Branch of USDA APHIS currently have regulations or policies to use non-toxic alternatives, but no national standards are in place.

How much lead was put into the environment prior to the 1991 waterfowl lead ban, and how much lead is still being introduced?

Prior to the banning of lead shot for hunting waterfowl and coots, an estimated 2,721 metric tons of shot were deposited in United States wetlands annually (Pain 1992).

A global estimate of lead ammunition production in 2000 was 194,820 metric tons, accounting for 3% of the lead with consumer end uses (Nordic Council of Ministers 2003).

Using the annual expenditure estimate provided by the U.S. EPA, Scheuhammer *et al*. (2003b) approximated that 3,977 metric tons of lead fishing sinkers are sold in the United States annually. Scheuhammer *et al*. (2003b) also estimated that approximately 559 metric tons of lead sinkers are sold annually in Canada.

Lead consumption by waterfowl

A study by Rocke *et al*. (1997) estimated a 45% ingestion rate of lead pellets by sentinel mallards (*Anas platyrhynchos*) in a wetland enclosure containing more than 2 million shot/hectare in the upper 10 cm of sediment. In enclosures with 15,750 and 173,200 pellets/hectare, mallards exhibited ingestion rates of 4% and 34%, respectively (Rocke *et al*. 1997).

Field radiography found that up to early 12% of spectacled eider adults and 2.5% of ducklings had ingested shot, and blood lead concentrations of ≥0.5 μg/g wet weight were found in 20% of adult females and 6% of ducklings (Flint *et al*. 1997, Franson *et al*. 1998).

Does the ban on lead shot save waterfowl?

Within five to six years following the ban on use of lead shot for hunting waterfowl, a large-scale study conducted in the Mississippi flyway demonstrated dramatic reductions in the ingestion of lead shot (Anderson *et al*. 2000). Of the gizzards containing ingested pellets, 68% of mallards, 45% of ring-necked ducks *(Aythya collaris),* 44% of scaup, and 71% of canvasbacks contained only nontoxic shot. Anderson *et al*. (2000) estimated that lead poisoning of mallards was reduced by 64% in the Mississippi flyway and

projected that 1.4 million ducks of the North American fall continental flight were spared from fatal lead poisoning.

Another approach to assessing exposure to lead shot involves a threshold concentration of 0.2 ppm in blood (Friend 1985). Using this criterion, Samuel and Bowers (2000) demonstrated a 44% reduction in lead exposure of black ducks from Tennessee by comparing exposure prevalence in 1986 through 1988 to that in 1997 through 1999 after the ban in lead shot for hunting waterfowl. Samuel and Bowers (2000) suggest that conversion to nontoxic shot conservatively reduced lead exposure in waterfowl by 50%. Similarly, in Canada, substantial decreases (52% to 90%, depending on species and location) in mean bone lead concentrations in hatch-year ducklings have occurred since nontoxic shot regulations were established (Stevenson *et al*. 2005).

It was estimated that about 1.6 to 3.9 million waterfowl died each year in North America from lead poisoning before the national ban on lead shot for waterfowl hunting in 1991 (Bellrose 1959; Feierabend 1983). Lead poisoning from spent lead shot caused an estimated 2 to 3 percent of the annual losses of North American waterfowl between 1938 and 1954 (Bellrose 1959). Within six years of the ban, there was an estimated dramatic 64% decline in ingestion of lead shot by waterfowl on the Mississippi flyway (Anderson *et al*. 2000). Of examined ducks whose gizzards contained ingested pellets, 68% of mallards, 45% of ring-necked ducks, 44% of scaup, and 71% of canvasbacks contained only non-toxic shot (Anderson *et al*. 2000). Samuel and Bowers (2000) demonstrated a 44% reduction in lead exposure (defined as >0.2 ppm in blood) and of black ducks in Tennessee comparing exposure from 1986-1988 with the post-lead shot ban from 1997-1999.

Lead consumption by Loons

Pokras *et al*. (1992) examined 60 dead adults collected from 1989 to 1992, and 27 adults had ingested lead sinkers. Pokras and Chafel (1992) examined 75 dead loons of various ages from 1989 to 1990 and determined that 16 of 31 dead adult loons (52%) had ingested lead sinkers. Sidor *et al*. (2003) examined 254 dead or moribund breeding common loons and determined that 44% of loons died of lead toxicosis.

In eastern Canada, lead poisoning from lead fishing weight ingestion accounted for the largest percentage (22%) of deaths diagnosed in common loons from 1983 to 1995 in environments where loon breeding habitats and sports fishing activity overlapped (Scheuhammer *et al*. 2003b).

In the upper Midwest, Ensor *et al*. (1992) indicated that lead exposure appears to be a threat to loons in Minnesota, as 17% of those necropsied in their study died of lead poisoning, and Franson and Cliplef (1992) reported lead poisoning in 7 of 77 common loons from Minnesota and 2 of 17 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 lead shot or sinkers on the bottom of water bodies (Eisele 2008)

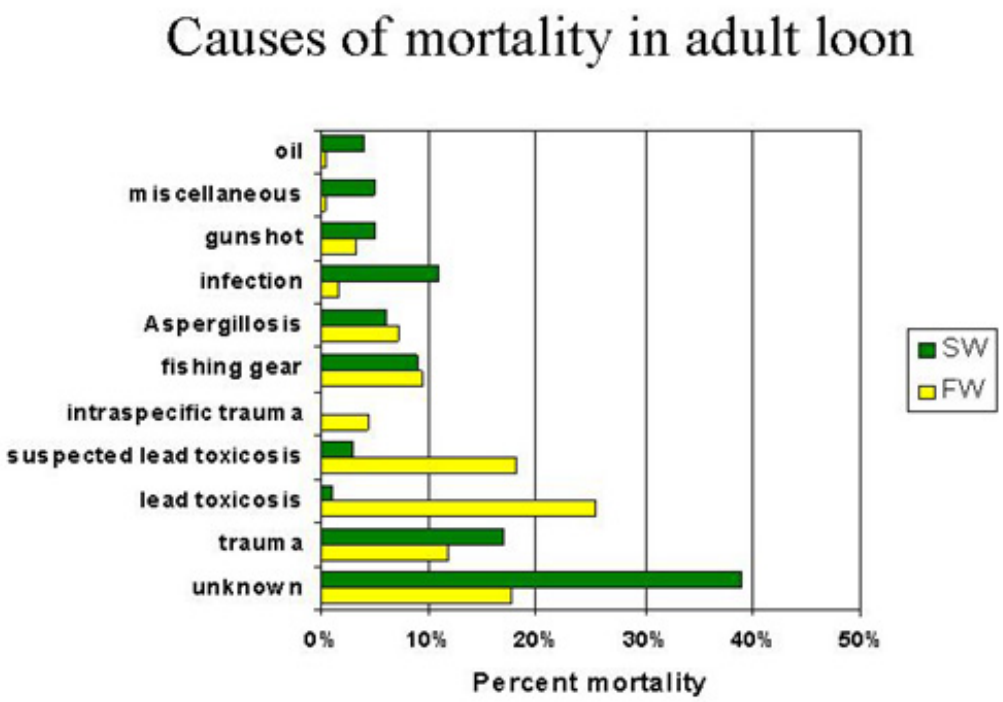


Figure from Mark Pokras, Tufts University Veterinary School. Yellow bars are birds in fresh water during the breeding season. Green bars are for wintering birds in salt water. Lead fishing sinkers are encountered in fresh water lakes.

Upland game birds and mourning dove risk from ingesting lead shot

Substantial information exists demonstrating the effects of lead poisoning on mourning doves. Reported lead pellet ingestion rates for hunter-killed mourning doves vary from 2 to 6.5 percent depending upon locale (Otis *et al*. 2008). Existing data demonstrate that some wild doves have ingested from 24 to 43 lead pellets, suggesting that doves are not *accidentally* ingesting lead (Schulz *et al.* 2002, Franson *et al.* 2008). Experimental evidence demonstrates that up to 92% of birds ingesting lead shot die acutely, and that birds not killed will be more prone to predation. Thus, every dove that ingests a lead pellet is essentially a dead dove (Schulz 2009).

Approximately 2.5% of hunter-shot doves contained lead shot in their digestive system, giving a rough estimate of the proportion of doves that ingest shot. Estimates of the 2005 U.S. dove population are 350-600 million birds (Dunks *et al* 1982, Schulz 2006), and experimental studies indicate that nearly all doves that ingest shot will die. Schulz (2006) estimates that **8.8-15 million doves** may be killed each year from ingesting lead shot pellets.

Risks of lead bullet fragments to scavenging birds

Bald and Golden Eagles:

Hunt *et al*. (2006) evaluated radiographic evidence of lead fragments in deer killed by licensed hunters using center-fire rifles with lead-based copper-jacketed, soft-point bullets. This study found 18 out of 20 (90%) offal piles contained lead fragments (mean = 160 fragments) and all five deer carcasses contained lead fragments (mean = 551 fragments). (Pertinent to condors, eagles and ravens)

Wisconsin Department of Natural Resources, about 15 to 20 percent of all bald eagle deaths are due to lead poisoning (Eisele 2008), usually from eating animals that were wounded with lead ammunition or from scavenging gut piles during and after the deer hunting season. Lead poisoning cases in bald eagles begin to increase in October, peak in December, and tail off in late winter, which coincides exactly with Wisconsin's deer hunting seasons, suggesting hunter-crippled game and lead-contaminated offal are the cause.

21% (138/654) of eagles admitted to the Minnesota Raptor Centers had evidence of lead poisoning, and only one had radiographic evidence of lead fragments in the gastro-intestinal tract (Kramer and Redig 1997).

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).

Studies by the Peregrine Fund showed that on average, 56% of all bald eagles admitted by wildlife rehabilitators in Iowa had elevated blood lead levels. Another study, the Fall Migrating Golden Eagle Lead Project, revealed that 50% of eagles tested showed high levels of lead in their blood. Domenech *et al* (2008) 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.

Scientists tested blood lead levels in 29 bald and golden eagles in and around Grand Teton National Park, Wyoming in 2004 and 2005. Eagles had an average blood lead level of 315 parts per billion during non-hunting season, compared to 871 parts per billion during hunting season. Of 29 eagles captured during hunting season, 13 showed toxic levels (> 65 ug/dl), while 12 were considered exposed (> 20 ug/dl) and four had levels of 20ug/dl or less.

Lead risks to Ravens:

Scientists tested blood lead levels in ravens (n = 302) 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). 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-September2004 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 are 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.

Lead Risks to California Condors:

From 1995 to 2002, more than 140 condors have been released in California and Arizona, and 44 are free-flying in California at the current time. Four condors have died of lead poisoning since 1997 (1 in California, 3 in Arizona), and 26 condors have received emergency chelation treatment to reduce toxic lead levels (8 in California, and 18 in Arizona).

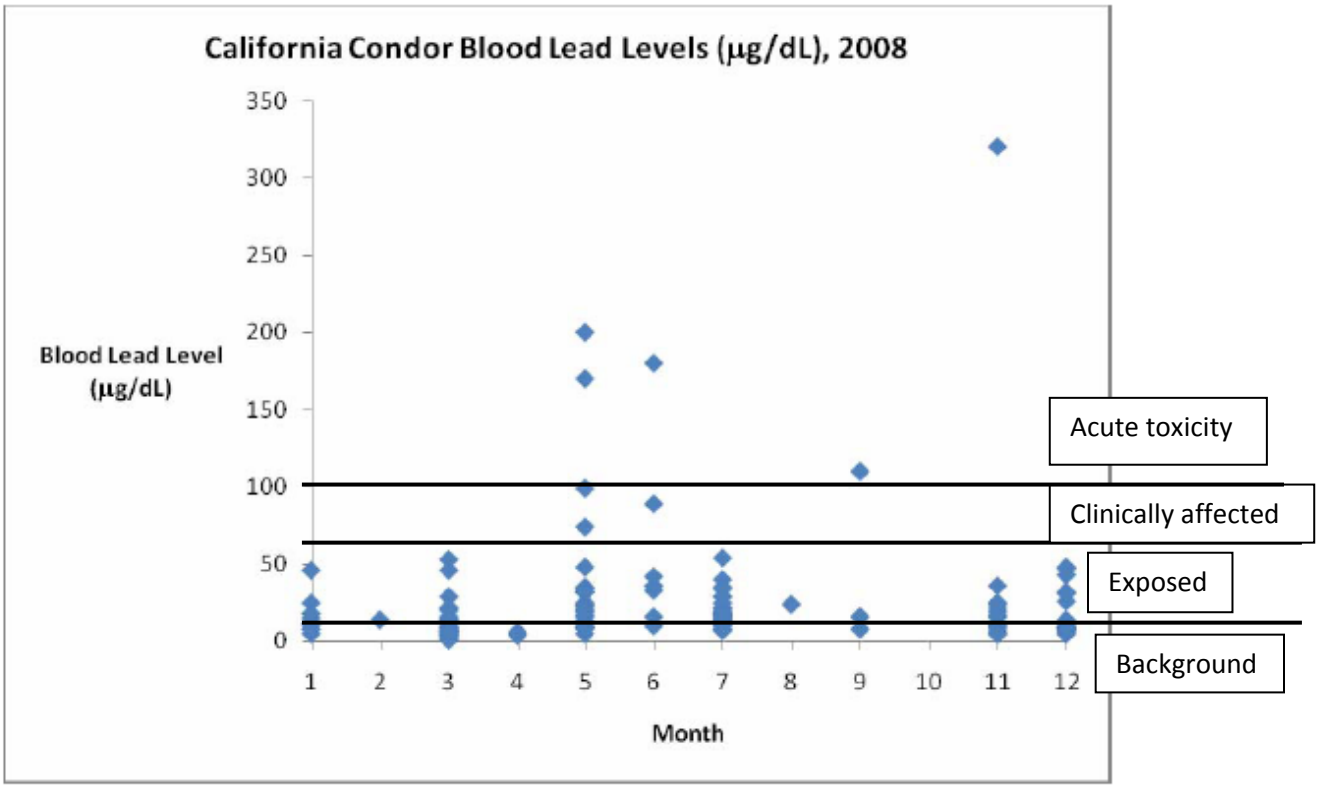
Blood lead levels were quantified for 72 individual free-flying condors in California (Southern California = 39, Central California = 33, total number of samples both populations = 148) and for five California condor nestlings (Southern California, total number of samples = 20) during 2008. Fifty-nine percent of condors sampled in the January to June sampling period exhibited blood lead levels > 10 µg/dL (background), 45% of condors sampled in the period July to December exhibited blood lead levels > 10 µg/dL (n = 72, Figure 2). A total of 35 individual condors were sampled during both sampling periods (Jan-Jun, Jul-Dec; Southern California = 29, Central California = 6). Twenty-three of these condors (66%) exhibited lower blood lead levels during the July to December sampling period compared to the January to June sampling period (Southern California = 19, Central California = 4), 12 condors (34%) exhibited higher blood lead levels during the July to December sampling period compared to the January to June sampling period (Southern California = 10, Central California = 2, Figure 1). Two of five California condor nestlings exhibited blood lead levels > 10 µg/dL (background) during 2008. Both of these nestlings were treated for lead toxicosis (chelation therapy). Seventeen free-flying condors in California (juveniles and adults) also were treated for lead toxicosis during 2008, (Jan-Jun = 9, Jul-Dec = 8).

Table 1. Information on population, reproduction, mortality, lead exposure, and treatment of California Condors in northern Arizona from 2000 through 2007.

Year	No. in Wild	No. Exposed to Lead	No. Tested for Pb	No. Treated for Pb	Blood-lead tests >15µg/dL	Blood-lead tests >65µg/dL	Deaths*	No. Birds of Breeding Age	No. Wild Young Fledged
2000	28	17 (61%)	25	9 (32%)	18	15	10(3)	7	0
2001	25	12 (48%)	25	1 (4%)	12	0	0	9	0
2002	31	23 (74%)	31	13 (42%)	29	11	4(1)	11	0
2003	40	30 (75%)	40	7 (18%)	43	7	1	14	0
2004	43	35 (81%)	43	18 (42%)	56	15	1	16	2
2005	56	29 (52%)	56	11 (20%)	40	8	6 (2)	22	2
2006	57	54 (95%)	57	40 (70%)	86	37	6 (3)	33	0
2007	61	50 (82%)	59	25 (41%)	52	18	4	40	2

*confirmed lead deaths in parentheses

Figure 2. Blood lead levels (µg/dL) among free-flying California condors (Southern and Central California populations combined) by month during 2008 (n = 72 condors, 148 samples).



Lines on figure 2 indicate approximate blood lead levels that correspond with background lead (<10 ug/dL), birds that are exposed (>10 - <60 ug/dL), birds clinically affected (>60 - <100 ug/dL) and birds

exposed to acutely toxic lead levels (>100 ug/dL). In practice, birds with blood lead levels greater than60 ug/dL are treated with chealating drugs to lower blood lead levels.

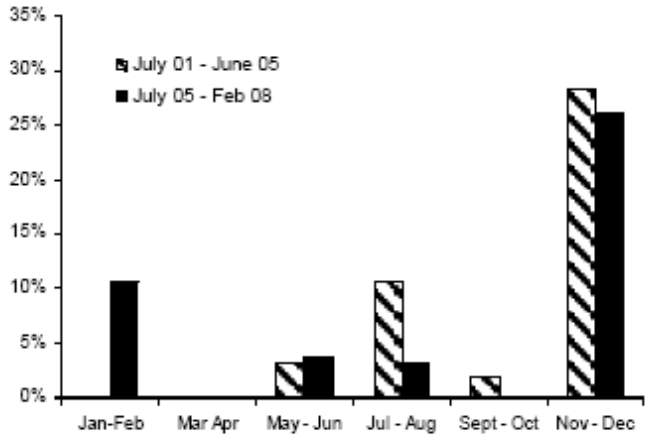


Figure 2. Annual pattern of toxic lead levels (>60 μg/dL) in condor blood from July 2001 through February 2008 in Arizona. The period July 01–June 05 was published in Parish et al. 2007. The period July 05–Feb 08 has not previously been published.

Condors in Arizona were periodically captured and monitored for blood Pb concentrations; subsets of these blood samples were analyzed for Pb isotopic ratios. To date, Pb isotopic ratios have been measured in blood in 47 birds over 3 years. Multiple measurements have been undertaken on 18 birds, including metal fragments collected at the same time from two different birds. Birds with elevated blood Pb levels were isolated, x-rayed and the excrement monitored for metal fragments. Twelve fragments were collected from 6 different birds. Analyses of the metal fragments from these birds determined that the fragments were Pb, Cu, Fe-Cr alloy and Pb-Sn alloy.

Long-term effects of lead shot remaining in the environment

Ingestion of lead shot caused the deaths of thousands of wintering trumpeter and tundra swans in northwestern Washington state and southern British Columbia (Lagerquist *et al*. 1994; Degernes *et al*. 2006). Swan mortalities continue to regularly occur, although use of lead shot was prohibited in wetland areas over ten years previously. Lagerquist *et al*. (1994) found that 35 percent of the 110 trumpeter and tundra swan carcasses collected and diagnosed from 1986 to 1992 had lead liver concentrations diagnostic of lead poisoning. Degernes *et al et al*. (2006) found that 81 percent of 400 trumpeter and tundra swan carcasses collected from 2000 to 2002 died from lead poisoning. Swan mortality could be high because swans can forage deeper into bottom sediments than other waterfowl, and be exposed to shot deposited years earlier.

Species affected by lead

Examples of affected species include bald eagles, golden eagles (*Aquila chrysaetos*), white-tailed sea eagles (*Haliaeetus albicilla groenlandicus*), Spanish imperial eagles (*Aquila adalberti*), Steller's sea eagles (*Haliaeetus pelagicus*), red-tailed hawks (*Buteo jamaicensis*), common buzzards (*Buteo buteo*), Eurasian sparrowhawks (*Accipiter nisus*), northern goshawks (*A. gentilis*), marsh harriers (*Circus aeruginosus*),

turkey vultures (*Cathartes aura*), black vultures (*Coragyps atratus*), and California condors (Janssen *et al* *et al*. 1986, Craig *et al*. 1990, Langelier *et al*. 1991, Pain and Amiard-Triquet 1993, Franson *et al*. 1996b, Pain *et al*. 1994, Kim *et al*. 1999, Wayland and Bollinger 1999, Iwata *et al*. 2000, Kurosawa 2000, Kenntner *et al*. 2001, Mateo *et al*. 2001, Clark and Scheuhammer 2003, Krone *et al*. 2004).

The risk of spent shot to upland game species, including doves and quail, is well recognized (Kendall *et al*. 1996). Lead exposure and poisoning from the ingestion of spent lead ammunition has been reported in many species of upland game birds and hunted non-Anseriform waterbirds, including 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*), sandhill crane (*Grus canadensis*), American coot, clapper rail (*Rallus longirostris*), king rail (*R. elegans*), Virginia rail (*R. limicola*), and sora (*Porzana carolina*) (Fisher *et al*. 2006).

Lead fishing sinkers and jigs have contributed to lead poisoning mortalities in aquatic birds, particularly mute swans, whooper swans (*Cygnus cygnus)*, Canada geese (*Branta canadensis*), mallards, brown pelicans (*Pelecanus occidentalis*), and common loons (Locke *et al*. 1982, Blus *et al*. 1989, Pain 1992, Pokras and Chafel 1992, U.S. EPA 1994, Scheuhammer and Norris 1995, 1996; Daoust *et al*. 1998, Friend 1999, Stone and Okoniewski 2001, Franson *et al*. 2003, Sidor *et al*. 2003).

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

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