LEAD EXPOSURE IN CALIFORNIA CONDORS AND SENTINEL SPECIES IN CALIFORNIA

This report is a critical and objective review of pertinent literature on the topic of lead toxicosis in sentinel wild birds, with special emphasis on the California condor, at the request of the California Department of Fish and Game's Wildlife Branch. The report will be updated as new information becomes available and is intended to provide information for the Department upon which to base recommendations to the California Fish and Game Commission.

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CONTENTS

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EXECUTIVE SUMMARY

Toxicosis from exposure to lead is well recognized in humans, mammals, and birds, and lead exposure is clearly an obstacle to condor recovery in both California and Arizona. Lead poisoning is the leading known cause of condor mortality in Arizona, and it is recognized as an important cause of death for condors released in southern California. Free-flying condors are exposed to sources of lead in their habitat as indicated by the elevated levels of lead found in 44% of the condors released in southern California and 21% of the condors released in Big Sur. The high levels detected in some birds indicate exposure to very concentrated sources of lead. The health effects vary according to the amount of lead ingested, and deaths have also occurred in golden eagles and bald eagles due to ingestion of lead bullet fragments. While some condors with lead poisoning have direct evidence of lead ammunition (shotgun pellets and rifle bullets) in their gastrointestinal tract, there are many occasions where the source of lead can not be found by the time birds die or are sick enough to be brought into captivity. Diagnosing lead poisoning as a cause of death in free-ranging wildlife inhabiting remote areas can be particularly difficult because carcasses need to be recovered shortly after death in fresh condition without extensive scavenging.

Elevated blood levels of lead have been found in condors during the deer hunting seasons in California and Arizona, suggesting that exposure might be caused by scavenging on deer carcasses or gut piles containing lead bullet fragments. However, lead intoxications also occur during other times of the year, and condors have been observed feeding on pigs, coyotes, rabbits and squirrels, which are also hunted with lead ammunition. While there may be other sources of lead besides ammunition that contribute to condor exposure, we currently lack data on these other sources. To improve population health and facilitate recovery of this endangered species, the most significant sources of lead in condor habitat must be prioritized and actions taken to reduce the risk of toxic exposure.

1. INTRODUCTION

Lead has been recognized as a toxic material for more than 20 centuries and many of its toxicologic actions in animals and humans are well documented. In humans, lead can have toxic effects on the cardiovascular, nervous, reproductive, blood, and renal systems (Roscoe et al. 2002). Lead exposure in pregnant women is of particular concern because of the serious central nervous system problems that may occur during fetal development (Sowers et al. 2002). Children, particularly infants, are also at high risk because of their tendency to put objects in their mouths. Over the past two decades, the campaign to eliminate lead in household and consumer products has successfully reduced the number of U.S. children with blood lead concentrations of 10 μg/dl or higher by over 80% (Lanphear et al. 2003). In 1990, the U.S. Department of Health and Human Services and the Centers for Disease Control (CDC) established a national goal to eliminate blood lead levels greater than or equal to 10 μg /dl in children and occupationally exposed adults by 2010. This effort requires improved surveillance, identification of all high-risk sources of lead in the workplace, and evaluation of existing preventive measures (Meyer et al. 2003). New data suggest that significant lead reductions may be possible; between 1998 and 2001, the mean blood lead level in adults was down to less than 3 μg/dl in the United States (Roscoe et al. 2002). While regulations to protect public health have been largely effective, lead toxicity continues to be a major public health concern, and communities that are dependent on hunter-killed game are now recognized to be at particular risk (Levesque et al. 2007).

Lead was recognized as an important cause of mortality in wildlife populations in the late 1950s (Bellrose et al. 1959; Irwin et al. 1972; Sanderson et al. 1986), when ingestion of spent hunting lead pellets or fishing sinkers was recognized to cause death in a wide range of wild waterfowl (Bates et al. 1968). 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 et al. 1981; Kramer et al. 1997; Fisher et al. 2006). Acknowledgment of the health impacts of lead shot in waterfowl and the endangered species that prey upon waterfowl prompted regulation of lead shot in waterfowl hunting. Nontoxic (steel) shot was proposed for waterfowl hunting along the most impacted flyways in 1976 and slowly phased in until a nationwide ban on the use of lead ammunition in waterfowl hunting was implemented in 1991 (USFWS, 2006). However, lead continues to be deposited in the environment in the form of lead

ammunition through upland bird and mammal hunting, target practice, and other legitimate, as well as illegal, shooting activities.

The potential effects of lead ammunition in non-waterfowl hunting practices has now received national attention in part because of recent documentation of harmful levels of lead exposure in the endangered California condor population. Biologists closely monitoring reintroduced condors have documented high levels of lead in condors in the wild (Fry 2003, Hall 2007, and Hunt 2007). The immense conservation effort to recover the California condor from the brink of extinction has galvanized public attention on the issue of lead use in ammunition.

2. HEALTH EFFECTS OF LEAD EXPOSURE IN WILD BIRDS

Lead is a cumulative metabolic poison affecting a large number of biological functions including reproduction, growth, development, behavior and survival, (Henny et al. 1991). Lead-induced lethal and sublethal effects have been documented in avian species, including passerines, waterfowl, raptors, and upland birds (Reiser et al. 1981; Lumeij, 1985; Beyer et al. 1988; Kramer et al. 1997; Vyas et al. 2001; Fisher et al. 2006).

The effects of lead intoxication can vary depending on the species and the amount and form of lead ingested. There are common predominant features among a wide range of animals such as cardiac, renal and hematologic changes, as well as non-specific signs like emaciation. The clinical profile following acute lead ingestion is difficult to interpret because blood levels may be measured prior to complete absorption and distribution to soft tissues (McKinney 2000). Therefore, the severity of clinical signs at the time of sampling does not always correlate with blood lead concentrations. A recent paper by Pattee et al. (2006) found that Andean condors experimentally dosed with lead shot exhibited normal behavior for 14 days, then developed decreased activity and alertness until dying or being euthanized 39 – 49 days post exposure. Lead absorption depends on transit time through the gastrointestinal tract, the absolute amount of lead ingested, and the surface area of the fragments. It is also likely that the mechanical action of multiple lead pellets or fragments rubbing against each other may generate smaller particles or lead powder that may be easier to absorb (McKinney 2000).

The dose required for pathologic and clinical manifestations of intoxication will vary by species and individual. Pattee et. al. (2006) proposes three interacting factors contributing to the individual and species response to lead intoxication: nutritional status, genetic predisposition, and lead availability. With chronic, low-level lead exposure, small amounts accumulate over time, especially in bone. In this situation, health changes range from no effect to general debilitation but are unlikely to cause direct mortality (Patte et al. 1981; Hoffman et al. 1981; Reiser and Temple, 1981; Beyer et al. 1988; Redig et al. 1991).

Clinical signs of lead poisoning in birds include depression, emaciation, non-regenerative anemia, vomiting, diarrhea, ataxia, blindness and seizures. Anemia is an important clinical manifestations of chronic lead toxicosis, although alteration of neurological and kidney function will also contribute to the deterioration of the intoxicated individual and, in some cases, lead to death (Lumeij 1985; Pattee et al. 1981; Moore 1988). Lead has a direct deleterious effect on the synthesis of heme (an essential component of red blood cells) and also influences other hematopoietic processes, such as globin protein synthesis and the processes that control the absorption and utilization of iron (Hoffmann et al. 1981; Miller et al. 2001). Bald eagles have been reported to show anemia when blood lead values exceed 0.6μg/ml (Hoffman et al. 1981; Redig et al. 1983), but blood lead values as low as 0.42 μg/ml have also been associated with a decrease in hematocrit (Miller et al. 2001). Gross necropsy and histopathology lesions in avian species may include impaction of the proventriculus, submandibular edema, biliary discoloration of the liver, distended gall bladder, green staining of the digestive tract and vent, renal intranuclear inclusion bodies, nephrosis, myocardial necrosis, and arterial fibrinoid necrosis (Reiser and Temple 1981; Beyer et al. 1988; Mateo et al. 2003).

3. SPECIES-SPECIFIC REPORTS ON LEAD INTOXICATION

California Condor (*Gymnogyps californianus*)

Efforts to monitor and save the California condor from extinction had been underway for nearly half a century when the controversial decision was made to bring the remaining 5 condors surviving in the wild into captivity in 1987 (Fry 2003). The subsequent captive breeding and reintroduction of condors has been a difficult task undertaken by many collaborating agencies and organizations in California and Arizona since the mid 1980's,

including the U.S. Fish and Wildlife Service, the California Department of Fish and Game, Arizona Game and Fish Department, the Utah Division of Wildlife, the Condor Recovery Team, Hopper Mountain National Wildlife Refuge, the U.S. Forest Service, the Bureau of Land Management, the National Park Service, The Ventana Wildlife Society, The Peregrine Fund, the Zoological Society of San Diego, the Los Angeles Zoo, the Phoenix Zoo, Southern Utah's Coalition of Resources Economics, the World Center for Birds of Prey, and many others.

The reintroduction of captive-reared California condors into the wild began in California in 1992, in Arizona in 1996 and in Baja California, Mexico in 2002 (USFWS, unpublished data). From 1995 to present, there have been 118 condors released in California (with 40 dead and 13 returned to captivity), 87 condors released in Arizona (with 30 dead and 3 returned to captivity) and 15 condors released in Baja California, Mexico (with 2 dead and none returned to captivity). The current free-flying population, which includes some birds that have hatched and fledged in the wild, consists of 68 condors in California, 51 condors in Arizona and 11 condors in Baja, Mexico (USFWS, unpublished data).

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

For the condors released in southern California (Ventura, Santa Barbara and San Luis Obispo counties) since 1992, blood lead concentrations were evaluated in 214 samples

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

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

A complete recount on lead intoxication events of wild and captive-reared condors in California and Arizona (1992 to 2002) was compiled by Fry in 2003 with information from the Condor Recovery Program. Currently, condors with greater than 40 μg/dl lead in blood measured with a portable lead analyzer in the field (LeadCare®, ESA Inc.) are

brought into captivity for chelation treatment to reduce blood lead concentrations. Among condors released in southern California, at least 8 have been brought back into captivity and received emergency chelation therapy (Hall et al. 2007). Four of these birds had lead levels exceeding 180 μg/dl and were likely to have died or been severely debilitated without emergency intervention (Hall et al. 2007).

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

In addition to capture and treatment of condors found with high lead levels, management practices in both California and Arizona include supplemental feeding. Several reports suggest that the condor mortality due to lead would be much higher if the free-flying population were not intensively managed by supplemental feeding and chelation treatment to minimize the impact of lead exposure events (Fry 2003, Woods et al. 2006, Pattee et al. 2006, Hall et al. 2007, Mee and Snyder 2007).

Survival of released condors has been compromised by many factors. Causes of mortality were reviewed in detail for 41 free flying condors that died in California and Arizona between 1992 and 2002 (Fry 2003). Of the 41 condor carcasses found and examined, lead toxicity was documented as the cause of death in 12% (5/41) (Meretsky et al. 2000; Fry, 2003). Predation (22%; 9/41) and power line electrocutions and collisions (20%; 8/41) were identified as the leading causes of reintroduced condor mortality in California and Arizona during this time frame (Fry 2003). While power line collisions and electrocutions were particularly problematic for the early condor releases in California, mortality due to this cause has been reduced substantially in recent years since power line aversion training was instituted as part of the reintroduction program (Mee and Snyder 2007). The coarse terrain and wide-ranging movements of the condor make it difficult to find deceased animals within a timeframe that allows for the accurate determination of causes of death. Unless carcasses are recovered in fresh post-mortem condition, it can be exceedingly difficult to identify causes of death that are only recognizable by microscopic examination of tissues or laboratory analyses of samples. It can also be difficult to distinguish whether or not disease or intoxications have altered an individual's behavior and initiated a chain of events leading to trauma or some other cause of death. Therefore, cause of death could not be accurately determined for 20% (8/41) of recovered

condor carcasses. An additional 11 condors were lost to follow up during this time period. While likely dead, these condors were never recovered.

A more recent review of the 66 condors released in southern California, found that among the 34 condors that died in this area from 1992-2005, exact cause of death could be determined for 18 birds (Hall et al. 2007). Lead toxicity is believed to be the primary cause of death in 3 of these birds (17%). Ages at death for the three condors with lead poisoning were 1.7, 2.5 and 4.8 years. Condor deaths due to lead toxicity have not been reported for the 33 condors released in Big Sur, California (Sorenson and Burnett 2007). Out of a total of 26 condor deaths observed in Arizona between 1996 and 2005, at least 6 and perhaps as many as 8 (23-31%) died from lead poisoning (Woods et al. 2006; Parish et al. 2006). Table 1 shows the most recent reported causes of mortality in California condors that have died following release to the wild (unpublished preliminary data, USFWS). Mainly because of a marked recent increase in the number of lead-related mortalities in condors released in Arizona, lead poisoning is now the leading known cause of death in free-flying condors. Data presented do not include an additional 3 condors believed to have died due to lead poisoning in Arizona over the last few months (USFWS, unpublished data).

The role lead intoxication played in the disappearance and deaths of condors with unknown cause is poorly understood. Two condors that went missing in southern California had high blood lead levels at last capture, and an additional two condors found dead in southern California had high blood lead levels at death, but cause of death has been reported as unknown (unpublished data, USFWS). If lead toxicity is suspected to have contributed to death of these condors, then the total number of deaths due to lead exposure in condors released in California would increase to 7 as opposed to the 3 shown in Table 1. Identifying unbiased estimates of cause-specific proportionate mortality in a free-ranging wildlife population can be difficult, as not all causes of death have equal detection probability, and conditions that have readily available screening tests are more likely to be detected (Kreuder et al. 2003). An association between lead exposure and lower overall condor survival is suggested by the data on southern California condors, but analyses performed to date have lacked statistical power (Hall et al. 2007). The exceedingly high blood lead levels reported in some free-flying condors suggest that these birds may have compromised abilities to avoid hazards. A thorough epidemiological investigation into the factors influencing survival in released condors

could be useful for clarifying the relative role of lead intoxication, power line hazards and predation.

Table 1: Preliminary description of causes of death in free-flying condors that have died in the wild from 1992 - August 2006 (USFWS, unpublished data).

	CALIFORNIA				ARIZONA			MEXICO		TOTAL
CAUSE OF DEATH	Adult	Juvenile	Nestling	Total	Adult	Juvenile	Total	Juvenile	Total	
missing or unknown	7	14	2	**23**	1	5	**6**	4	**4**	**33**
lead poisoning		3		**3**[a]	1	10	**11**[b]	1	**1**	**15**
powerline		10		**10**		1	**1**			**11**
predation coyote or golden eagle		1		**1**	2	6	**8**			**9**
malnutrition or starvation		4		**4**		3	**3**			**7**
West Nile Virus			1	**1**		1	**1**			**2**
shot	1	1		**2**		3	**3**			**5**
copper toxicity	1		1	**2**[c]						**2**
drowning		2		**2**						**2**
respiratory problems		1	1	**2**						**2**
cancer		1		**1**						**1**
ethylene glycol intoxication		1		**1**						**1**
head trauma			1	**1**						**1**
septicemia						1	**1**			**1**
visceral gout			1	**1**						**1**
zinc toxicity			1	**1**						**1**
TOTAL	9	38	8	**55**	4	30	**34**	5	**5**	**94**

Mortality data provided by the Condor Recovery Program (USFWS) - updated through August 2006

[a] 2 of these deaths suspected to be due to lead poisoning but unconfirmed.

[b] 6 of these deaths suspected to be due to lead poisoning but unconfirmed.

[c] Both copper toxicity cases are suspected but not confirmed

Age codes:

Nestling < 0.5 year

Juvenile > 0.5 - 6 years

Adult > 6 years

If lead exposure is not high enough to cause acute intoxication and death or to impair survival skills, lead should be eliminated from the body gradually through natural processes. Fry (2003) calculated an average depuration rate (or half-time for lead elimination from blood) of 13.34±2.87 days. This finding, along with the high proportion of samples with elevated blood levels (mentioned previously), suggest that condors are frequently exposed to lead. Similar observations of high lead levels in blood and tissues of sympatric species, such as vultures, eagles, hawks and ravens in the condor range support the conclusion that environmental lead is widely available to scavenging birds (Wiemeyer et al. 1988; Pattee et al. 1990).

Sources of Lead Exposure in Condors

Many studies have attributed lead exposure in condors to lead bullet fragment ingestion when eating hunted animal carcasses (Locke et al. 1969; Janssen et al. 1986; Meretsky et al. 2000; Fry, 2003; Hunt et al. 2006; Woods et al. 2006; Church et al. 2006). Lead from

environmental sources, such as air and water pollution, may accumulate in animals, but environmental exposure is not likely to result in levels high enough to cause mortality (Pattee et al. 1990). The very high level of lead detected in most individuals of the free-flying condor population is consistent with a highly concentrated source of exposure not typically found in air, water or soil unless in an area contaminated from lead mining and smelting activities.

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

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

Inter-annual variation and seasonal trends in lead exposure have been observed in all condor populations. The 44 condors released in southern California showed substantial inter-annual variation in blood lead concentrations with samples from 2001-2004 having a significantly lower mean than samples from 1997-2000 (Hall et al. 2007). This temporal trend has been explained by a move in release, food provisioning and trapping (sampling) location from the Sierra Madre Mountains to Hopper Mountain NWR in 2001. Condors trapped at the Sierra Madre site had a significantly higher mean lead concentration than condors trapped at Hopper Mountain. The Sierra Madre site is characterized by greater public access and hunting activity than the Hopper Mountain site (Hall et al, 2007). Increases in blood lead levels in condors tested during the deer hunting season have also been reported by Hall et al (2007), but sampling effort was not distributed evenly in all seasons. In fact, sampling between January and May was very limited (with only 20/214 samples in these months). This study noted that while mean lead concentration was significantly higher in condors sampled during deer hunting season, elevated lead exposure was detected at other times of the year with 38% (20/53) of blood samples collected in June having lead concentrations exceeding 20 μg/dl. Blood samples in the 33 condors at Big Sur also showed inter-annual and seasonal variation in lead concentration with samples obtained in 2005 and samples obtained in September and October showing the highest mean concentration of lead (Sorenson and Burnett 2007). This peak in lead concentrations in the Big Sur condor population does correspond with the time period in which hunter-shot deer are most prevalent in the coast range (Fry 2003). Systematic sampling of condors across time periods would improve evaluation of apparent seasonal trends.

The studies conducted on the introduced condor population in Arizona by Hunt et al. (2006) show correlations between increased lead exposure and foraging in deer hunting areas during and just following the hunting season. Since condors began foraging in the Kaibab Plateau in 2002, detected lead exposures have been temporally and spatially clustered and highly predictable (Hunt et al. 2007). Blood lead levels in condors visiting the Kaibab Plateau were significantly higher than condors not visiting this intensively hunted area, and evidence of lead intoxication in live and dead condors have peaked annually in November and December from 2002 to 2004, coincident with the deer hunting season (Hunt et al. 2007).

Condor lead intoxications reported in both California and Arizona during non-deer hunting seasons suggest that deer hunting practices are not the only potential source of

lead for condors. Firearms are used in the California condor range year-round for taking non-game animals, such as ground squirrels and coyotes, which are typically left in the field and available for scavenging species (Pattee et al.1990). Rabbits, squirrels, coyotes, and pigs shot with lead ammunition likely pose a similar risk for exposure as do hunter-killed deer carcasses, and these animals are more likely to be left in the field if they are not a food source or trophy for hunters. Condors have been observed feeding on hunted pigs at private dumps and piles of dead ground squirrels shot for pest control (R. Jurek personal communication, 2006). Two rifle-killed coyotes were observed as a food source for condors in Arizona, and hunted coyotes have been suggested as a potential source for summer lead exposure in condors (Parish, et al. 2006, Hunt et al. 2007).

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

Ammunition was implicated as the main source of lead exposure in released condors in a recent study by Church et al (2006). This study compared lead isotopic ($^{207}Pb/^{206}Pb$) ratios in blood samples from released free-flying condors in Central California and pre-release condors in Southern California; tissue samples from possible condor diet items (calves, road-killed deer and a sea lion); and samples from ammunition sold for bird and mammal hunting at stores within the condor range. The lead isotope ratios in free-flying condors differed significantly from those in captive pre-release condors providing further evidence that the sources of exposure for free-flying condors were different from the sources causing background levels of exposure in captive condors. Furthermore, the low background concentrations of lead detected in non-hunter killed condor diet samples had isotopic ratios similar to those described for environmental lead in rivers, lakes,

atmospheric dust and urban aerosols. These findings provided evidence that "environmental" sources of lead, such as background levels in water, soil and non-hunter killed carcasses, were not responsible for the elevated lead exposures observed in free-flying condors in California.

The lead isotopic ratios in the elevated free-flying condor samples were similar to those found in the ammunition samples tested (Church et al. 2007). This finding supports their conclusion that ammunition is the principal source of the high lead levels found in condors, but other potential point sources of lead were not available for testing. Other sources of lead could have isotope composition similar to the ammunition tested. Lead used to manufacture bullets is commonly obtained from secondary smelters where lead from batteries and other lead scrap is recycled (Peters, 2002). This continuous mixing of lead sources reflects an increased homogeneity in the lead isotopic composition of industrial lead, increasing the difficulty to differentiate between ammunition lead and other possible sources of industrial and dump waste contaminating the environment with lead (Sangster et al, 2000).

Foreign material and trash, including metal, plastic and glass fragments have been found in condor nests. Nestling condors have had foreign body impactions in their stomachs from trash fed to them by their parents, including bottle caps, spent ammunition casing and scrap metal and glass. While lead has been phased out of most industrial, commercial and consumer products in recent years, hazardous levels of lead could still be present in scrap metal and hazardous waste produced prior to regulations. Detailed descriptions of foreign material found in condor stomach contents and in condor nests have not been reported in the scientific literature and this is a gap in our understanding of potential sources of lead and other heavy metal exposure in condors. Identification and lead analysis of all foreign material found in condors (in addition to ammunition fragments) will help rule out other concentrated sources of lead exposure. While lead concentrations in condors did not correlate with elevations in other heavy metals that may also be found in trash at hazardous waste sources (Church et al. 2006), not all elements are likely to have the same half-life in condors, so it is difficult to assess co-exposure at one point in time.

The lead isotope ratio technique has been also recently applied as a forensics tool to trace lead sources involved in condor deaths. A feather from one condor (#165) released in Arizona and found dead nearly 3 years later (June 2000) from acute lead poisoning with 16-17 shotgun pellets in its stomach was analyzed for lead concentration and lead isotope

composition (Church et al. 2006). The lead isotopic ratio in the feather from this condor (in the area of the rachis and vane with most recent growth) closely matched that detected in ammunition samples from stores in California (Church et al. 2006). The authors noted in a recent press release that further analysis of concentrations of total lead in the bones of three other dead condors (#132, 175 and 181) strongly suggest that lead poisoning or debilitation induced by lead contributed to their deaths, but the isotopic ratio in tissues from condors #175 and # 181 were unlike those of the ammunition samples tested to date. However, the isotope ratio in tissues from condor #132 closely matched that of the ammunition samples tested in the study by Church et al (2006). Further lead isotopic analyses of ammunition and other potential point sources in the condor's habitat, along with bones, feathers and blood of other condors suspected to die from lead intoxication are required to attain a reasonable sample size and a more conclusive association between ammunition-derived lead intoxications and condor mortalities.

The population-level impact of condor exposure to lead may be difficult to quantify but is clearly significant. For small populations in particular, increased adult bird mortality at any measurable rate is likely to affect population dynamics (Westemeier et al. 1998; Fisher et al. 2006). Re-introduced condor populations are currently being intensively managed to reduce lead exposure, and the proportionate mortality due to lead exposure would almost certainly be higher if individual animal interventions ceased. While these practices have been an important aspect of the successful management of the condor population, these practices are not practical in the long term as the condor population grows in size. Such close monitoring and frequent recapturing are also counterproductive to the establishment of a behaviorally normal self-sustaining wild condor population (Condor Recovery Team, Lead Exposure Reduction Steering Committee, 2003). In order to ensure recovery of condors in California, an effort must be made to address all major sources of mortality in juveniles and adults. Lead ammunition from a variety of hunting practices has been implicated in some deaths and this may be the only cause of condor mortality that can be mitigated through management. However, all sources of lead in condor habitat must be identified, prioritized for risk of exposure to condors, and minimized where possible, including regulated and unregulated shooting activities, in order to improve population health and survival in this endangered population.

Bald eagles (*Haliaeetus leucocephala*)

Bald eagles share some demographic and ecological factors with free-ranging condors that make this species vulnerable to lead intoxication; they are long-lived, they have low recruitment rates, their numbers have been reduced in recent decades, and they scavenge on carcasses (Pattee et al. 1990). Bald eagles that ingest lead shot embedded in the tissues or the intestinal tract of waterfowl can 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 in some cases up to 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 (0.5 mg 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 in raptors so fragments may not be detected in the gastrointestinal tract at the time of capture or blood tests, even if they contributed substantially to lead exposure 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 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). One study by Pattee et al. (1983) found that 89% of bald eagle lead poisoning cases were reported within waterfowl hunting season. 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). A study to assess whether lead poisoning cases decreased in bald and golden eagles admitted to wildlife rescue centers in Minnesota and Wisconsin after lead shot use in waterfowl hunting was banned found that the number of cases remained steady or even increased, even though at the population level, mean blood lead concentration decreased (Kramer et al. 1997). In

this study, 21% (138/654) of eagles admitted to the centers during 15 years had evidence of lead poisoning, and only one had radiographic evidence of lead fragments in the gastro-intestinal tract (Kramer et al. 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 in this time period.

Golden eagle (*Aquila chrysaetos*)

Golden eagles share some feeding ecology and behavior with bald eagles and therefore may be exposed to some of the same factors that predispose condors to lead intoxication. In the study by Pattee et al. (1990) on the lead hazards within the California condor range, golden eagles were suggested as a model species to assess lead exposure in California condors because they are abundant in the condor range and they have been observed feeding on the same carcasses as condors. Between 1985 and 1986, 36% of the 162 golden eagles evaluated within the California condor range had been exposed to lead and 2.5% had levels indicative of clinical lead poisoning. This study also reported seasonal trends for lead levels found in tissues of golden eagles within the California condor range which coincided with the deer hunting season (Pattee et al. 1990). Although golden eagles are perhaps one of the better models for condors, there is a wide variation in species-specific responses to lead intoxication (Reiser and Temple, 1981; Beyer et. al. 1988). Therefore inter-species extrapolations should take a conservative approach.

Turkey vulture (*Cathartes aura*)

While mortality due to lead exposure in turkey vultures is not well documented, dead turkey vultures sampled within the condor range have been documented as having elevated lead exposure (Weimeyer et al. 1986). Experimental lead intoxication studies in turkey vultures suggest that vultures do succumb to lead poisoning. One experimental intoxication study, conducted by Carpenter et. al. (2003), administered turkey vultures daily oral doses of one, three or ten BB-sized lead shot (0.35 to 0.45 grams) over a six month observation period. While most measured parameters were similar to those reported for other avian species, survival time (143 to 211 days), even at the higher level of exposure, was much longer than reported for other species, suggesting turkey vultures may be somewhat more tolerant to the deleterious effects of lead ingestion (Carpenter et al. 2003). In a separate experimental trial by Reiser and Temple (1981), one turkey vulture was more susceptible to lead intoxication than two red-tailed hawks. However, it

is difficult to generalize to the species level and rule out individual responses with this sample size.

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

Other raptor species

There are several reports of elevated lead levels in other raptor species including the American kestrel, sharp-shinned hawk, Cooper's hawk, northern goshawk, northern harrier and red-tailed hawk (Fisher et al. 2006). A study that included 10 different raptors and scavenger birds, arriving at several wildlife rescue centers in Spain, found species who feed on carrion to have a higher concentration of lead in their bones (Mateo et al. 2003). They also concluded that it is difficult to assess the prevalence of mortality due to lead poisoning in raptors, but suggested that it is lower than other mortality causes such as electrocution, shooting, trauma or illegal poisoning. However, the probability of retrieving birds that have died from these other causes may be higher, because their effect is immediate and the birds tend to concentrate near power lines, roads or open fields, where they may be more easily noticed, while development of lead poisoning takes several days and birds may be inconspicuous until death (Mateo et. al., 2003).

A retrospective study on causes of morbidity and mortality evaluating 409 raptors in Colorado, Nebraska and Wyoming was conducted by Wendell et al. (2002). Known causes of toxicosis accounted for 2% of mortality in raptors between 1995 and 1998. Lead was the cause of death in two out of eight toxicity diagnoses. This study acknowledges that toxicosis may have been under-represented, because many raptors arriving to the hospital with trauma could have had an underlying toxicity that was overlooked.

Non-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). It has also been suggested that raptors intoxicated with lead may suffer from impaired hunting ability and may scavenge to a greater extent or be less selective in their choice of prey (Pain et al. 1993). Sampling methods to determine the exposure to lead intoxication in wildlife have inherent biases as with any wildlife health assessment in the field. 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). Alternatively, trapping and baiting methods may have bias in attracting birds with debilitated health status. Traps baited with carrion may have a greater chance of attracting poisoned raptors, whereas mist net trapping may have a lower chance of catching sick and debilitated birds. For example, marsh harriers captured by mist netting had lower levels of lead than those captured by carrion baited traps (Pain et al, 1993).

4. DIAGNOSTIC TESTS FOR LEAD EXPOSURE

The interpretations, comparisons and conclusions drawn from avian toxicological data are dependent on several factors, including the analytical methodology, sensitivity of equipment, and the level of detection limits (Pain et al. 1993). Studies on inter-laboratory variation on blood lead concentrations have shown small differences in reference values measured by inductively coupled plasma mass spectroscopy (ICP-MS), but the data in these studies were deemed to have comparable precision and accuracy (Subramanian 1989).

Inductively coupled plasma mass spectroscopy was developed in the late 1980s to combine quick sample analysis with accurate and low detection limits of a mass spectrometer. The mass analyzer in this technique can separate isotopes based on their mass-to-charge ratio and it can discriminate between the mass of various isotopes of an element where more than one stable isotope occurs. This technology allows measurement of specific lead isotope ratios with an extremely low detection limit of 50 to 100 parts per trillion (Bradford and Cook 2006). Lead has four stable isotopes (204, 206, 207 and 208) and isotope ratio comparisons are more discriminating if more than one isotope ratio is compared. Ideally, if there are other potential sources of lead, isotopic tracing methods

are supported by corroborating information on these other sources. This method has been used to measure lead isotopic ratios and identify specific sources of lead intoxication in humans (Gulson et. al., 1996; Gulson et. al., 2004; Gwiazda et. al., 2005; Gwiazda et. al., 2007). Levesque (2007) used ICP-MS to identify specific lead isotope ratios in umbilical cord blood from newborns in an Inuit hunter community in Canada. Lead isotope ratios were compared to possible environmental sources (water and soil) and lead shot used locally for waterfowl hunting. This study was complemented by an epidemiological investigation that showed dietary consumption of geese and ducks was significantly correlated to blood lead concentration in Inuit women of child bearing age (Dewailly et al. 2001).

The ICP-MS technique has been used more recently in environmental toxicology studies to monitor bioaccumulation of lead, zinc and cadmium in plant biomass, invertebrates and in fish from streams drained by mining waste in southeast Missouri (Besser et al. 2006). Lead isotope composition is being used increasingly to trace lead poisoning in wildlife to distinct sources of lead exposure (Scheuhammer and Templeton 1998, Svanberg et al. 2006, Finkelstein et al 2003). Church et al. (2006) used this same technique to compare the specific lead isotope ratios found in condor blood, local commercially available lead ammunition used for large and small mammal hunting, and common condor food items. Evaluation of lead isotope ratios can be an efficient tool to identify sources of lead exposure and to support the implementation of public health prevention and control measures (Chaudhary-Webb et al. 2003). This approach also holds great promise for identifying potential sources of lead likely to be exposing wildlife.

5. SOURCES OF LEAD IN THE ENVIRONMENT

The toxicity of lead in shotshell pellets and fishing sinkers directly ingested by waterfowl and raptors that prey on waterfowl has been well documented and reviewed (Fisher et al. 2006). Potential hazards from other sources have not been as widely documented as they have for lead shot used in waterfowl hunting, but reports are becoming increasingly prevalent. Most documented instances of lead exposure among terrestrial species have been associated with small contaminated areas, such as around metal smelters (Blus et al. 1991; Henny et al. 1991; Blus et al. 1995; Sileo et al. 2001), shooting ranges, or locations of intense hunting pressure (Lewis et al. 2001). Lead exposure in wildlife tends to decrease with increasing trophic level because, unlike mercury and organochlorine

pesticides (Henny et al. 1991), lead does not biomagnify in the food chain. This may be explained partially by the fact that most of the body burden of lead is distributed to bone, which is not likely to be digested by carnivorous animals (Henny et al. 1991). It seems likely that regardless of the trophic level, lead intoxications can occur when a direct source of bioavailable lead is ingested.

Lead exposure from industrial activities

Mines and smelters (where lead is recycled from used batteries and other scraps) are important sources of environmental contamination of lead. These activities produce several sources of contamination. The milled mine waste, or chat, contains lead, zinc, cadmium and other elements. Smelting also disperses metals by air, thus contaminating local soils. Metals may precipitate with iron oxyhydroxides and form highly contaminated surface crusts, a possible but unproven source of contamination for terrestrial wildlife (Beyer et al. 2004). Refuse and soils adjacent to mines and smelters may contain high concentrations of metals, much of which may be in the form of primary ores (Beyer et al. 2004). Exposure by ingestion of lead from these sources will vary among species depending on their feeding behavior. Animals feeding in aquatic environments and ingesting contaminated vegetation and sediments are likely to be exposed to the highest amounts of lead; animals feeding on invertebrates might be exposed to an intermediate amount; and animals ingesting vertebrates would be exposed to the lowest amount (Henny et al. 1991).

Long-term lead mining activities in the Viburnum Trend mining area in southeast Missouri have resulted in significantly elevated concentrations of lead, cadmium and zinc in plants and invertebrates of receiving streams, causing alterations of benthic invertebrate communities and inhibition of lead sensitive enzymes systems in fish. These levels continue to be elevated in areas where mining ceased in the 1970s (Besser et al. 2006). Metals from mining areas may enter streams by seepage from tailing or mine-water impoundments, discharge from passive treatment systems, or erosion of tailing deposits during runoff events (Besser et al. 2006). Temporal variations of metal concentrations and bioaccumulations can occur in response to seasonal patterns of precipitation and snowmelt and daily cycles of photosynthesis and respiration (Besser et al. 2006). Mining wastes in the Coeur d'Alene Valley in Idaho have been reported as a persistent cause of death in waterfowl but not in osprey, although osprey have had elevated lead levels in blood (Henny et al. 1991, Blus et al. 1999, Spears et al. 2006). Tundra swans, which feed at a lower trophic level than osprey, died from lead exposure

after feeding on the contaminated sediment near this mining and smelter complex for only a few weeks during spring migration (Blus et al. 1991). White-tailed deer mandibles collected from hunters near a metal mining district have also been shown to have elevated concentrations of lead (Conder and Lanno, 1999).

Despite recent efforts to move towards lead alternatives in commercial and consumer products because of concerns for public health, lead still has many industrial uses because of its unique properties. Most of the lead smelted and refined in the US is used for lead-acid battery production, but lead also has common uses in construction, cable sheathing, solder in electrical components, metal weights, pipes used in industry, glass and ceramic products, radiation protection products, and of course ammunition. One report documents lead poisoning in seabird chicks from exposure to leaded paint at a decommissioned military base on Midway island (Finkelstein et al. 2003) but there are still large gaps in our knowledge of wildlife exposure to potentially hazardous waste sources.

Lead exposure from hunting practices

The potential for direct exposure to lead from hunting activities was discussed here for each wildlife species of interest. The recent studies identifying significantly higher lead exposure in people from hunting communities in Canada (Dewailley et al. 2001, Levesque et al 2007) have major implications for the public health hazards of lead in ammunition. To date, public health agencies have regulated lead in industrial activities and consumer products but have focused little attention on hunting activities that may be an important source of lead exposure in certain communities. Hunting activities using lead ammunition are one of the major remaining sources of lead deposition in the environment now that the use, recycling, and disposal of lead products are heavily regulated.

Legal wildlife take activities, as well as illegal take and shooting, that use lead ammunition may directly or secondarily expose wildlife to lead and ultimately, deposit lead in the environment. Similarly, skeet/trap shooting and target practice activities deposit significant amounts of lead in geographically localized areas. Seed-eating birds are speculated to ingest lead shot at firearm training fields because they might be mistaken for berries, which may be similar in appearance after drying and falling (Lewis et al. 2001). Lead exposure has been documented in doves foraging at intensive hunting or target-shooting areas (Fisher et al. 2006). Species that forage primarily on seeds on the ground may have higher risk, but even species with very different foraging strategies,

such as woodpeckers, can acquire lead presumably by ingesting lead fragments embedded in trees or on the ground (Momer and Peterson, 1999). Lewis et. al. (2001) examined birds and mammals in a firearm shooting field and found 33% to have elevated lead tissue levels and 17% to have potential subclinical or clinical lead exposure. 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 on hunter-killed carcasses are at highest risk of encountering severely toxic concentrations of lead. A recent study by 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 5 deer carcasses contained lead fragments (mean = 551 fragments). The carcasses and gut piles from deer killed by hunters that used monolithic copper expanding bullets (or "X-bullets") showed little evidence of fragmentation with only 1 in 4 offal piles containing a single fragment and only 2 out of 4 carcasses containing 2 fragments each (Hunt et al 2006). Copper expanding bullets may be a good alternative if bullets are much less likely to fragment upon impact. While copper is substantially less toxic than lead, both copper and zinc toxicity have been reported in condors (unpublished data, USFWS). Potential toxicity as well as ballistics must be carefully evaluated when considering the potential intoxication risk to avian wildlife.

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. The probability of direct exposure to lead from hunter-killed carcasses of course varies according to the proportion of the diet comprised of hunter-killed animals, the availability of hunter-killed carcasses relative to other food sources, and the amount of lead in the animal remains consumed. With such a high degree of variability, we may expect that there would be episodes of lead exposure, especially if multiple individuals share a feeding source, when all of these factors are most likely to occur. To fully understand the impact hunting practices have on lead

exposure in wildlife, all activities that deposit lead ammunition in the environment must be evaluated.

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