Commentary

Exposure of California Condors to Lead From Spent Ammunition

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ABSTRACT The scientific evidence that California condors (*Gymnogyps californianus*) are frequently sickened and killed by lead poisoning from spent ammunition supports the conclusion that current levels of lead exposure are too high to allow reintroduced condors to develop self-sustaining populations in the wild in Arizona and, by inference, in California. The evidence for lead poisoning and its source comes from the following sorts of data: 1) 18 clinical necropsies revealing high levels of lead in body tissues and (or) presence of lead shotgun pellets and bullet fragments in digestive tracts; 2) moribund condors showing crop paralysis and impending starvation with toxic levels of lead in their blood; 3) widespread lead exposure among free-flying condors, many with clinically exposed or acute levels; 4) temporal and spatial correlations between big game hunting seasons and elevated lead levels in condors; and 5) lead isotope ratios from exposed condors showing close similarity to isotope ratios of ammunition lead but isotope ratios in less exposed condors being similar to environmental background sources, which are different from ammunition lead. Simple population models reveal harmful demographic impacts of unnatural mortality from lead on population trajectories of reintroduced condors. Recent innovations in the manufacture of nonlead shotgun pellets and bullets with superior ballistics now provide for a simple solution to the problem of lead ingestion by condors, many other species of wildlife, and human beings: substitute nontoxic forms of ammunition for traditional lead-based ammunition. The substitution of nontoxic ammunition would be highly efficacious for hunting, economically feasible, and the right thing to do. (JOURNAL OF WILDLIFE MANAGEMENT 71(7):2125–2133; 2007)

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For >2 millennia mankind has known that lead can be lethal and can cause various physiological malfunctions in human beings, other animals, and plants, but societies have continued to manufacture from lead and lead compounds a variety of products, the use of which can place human beings and other organisms in jeopardy (U.S. Environmental Protection Agency 1979, Eisler 1988). Governments have been slow to recognize and respond to the seriousness of lead's effects even after many decades of research (Eisler 1988, Fisher et al. 2006). In the United States only in the past 30 years has government played an active role in controlling the use of lead products (U.S. Environmental Protection Agency 1979). Now there are restrictions on lead in paint, gasoline, pipes, children's toys, solder and some other products, and there are clean-up requirements for indoor and outdoor shooting ranges, and proposals to eliminate the use of lead as balancing weights on vehicle wheels, fishing sinkers, and all ammunition used for hunting and for military small arms. It is now clear that the use of lead for any purpose should be carefully monitored and regulated (U.S. Environmental Protection Agency 1979).

Realization that ingested lead from spent ammunition can kill birds and other wildlife developed slowly beginning in the late 1800s with the discovery that spent shotgun pellets poison waterfowl (Bellrose 1959, Eisler 1988). The United States federal government began limited restrictions in 1979 but did not fully implement action to stop the use of lead pellets for hunting waterfowl until 1991 (Eisler 1988).

In recent decades the accelerated buildup of lead in the environment from human uses and disposal and the increased knowledge about the number of organisms affected by its unnatural presence have led to the recognition of certain sensitive species as indicators of lead pollution in the environment, species that point to especially troublesome situations that require correction (Eisler 1988, Pain 1995, Fisher et al. 2006, Johnson et al. 2007). Swans (*Cygnus* spp.) and other waterfowl have revealed the massive pollution of wetland habitats by spent shotgun ammunition (Demayo et al. 1982, Kendall and Driver 1982, Pain 1992). Ingestion of shot mistaken for grit or seeds by mourning doves (*Zenaida macroura*; Schultz et al. 2002, 2006), quail, and other upland gamebirds (Kendall et al. 1996, Fisher et al. 2006) has revealed locations, such as hunting preserves and watering tanks for livestock, where spent lead from ammunition has accumulated in hazardous concentrations. Scavengers such as eagles, vultures, condors, and ravens have shown that dangerous amounts of lead occur in the mammal and bird carcasses and offal piles that accumulate in the environment after animals have been shot, often with debilitating or lethal consequences to the scavengers (Jansen et al. 1986, Kramer and Redig 1997, Fry 2003, Garcia-Fernandez et al. 2005, Fisher et al. 2006, Johnson et al. 2007) and with clear implications for the health of people who eat game meat obtained by using lead-based ammunition (Dewailly et al. 2001, Levesque et al. 2003, Hunt et al. 2006). Because of its highly endangered status and uniqueness as a surviving example of the Pleistocene megafauna, the California condor (*Gymnogyps californianus*) has become perhaps the most notable indicator of the problems that spent ammunition lead causes to wildlife (Mee and Hall 2007).

My objective is to summarize the scientific data supporting the conclusion that reintroduced California condors are seriously exposed to lead poisoning from spent ammunition

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in the carcasses and gut-piles they eat and that this exposure causes fatalities and physiological malfunctions that—at current levels of exposure—will prevent the reintroduced condors from developing self-sustainable populations, at least in Arizona if not also in California, USA (Meretsky et al. 2000, Snyder and Snyder 2000, Cade et al. 2004, Woods et al. 2007). This summary is necessary because many conflicting statements about this problem have been expressed in popular, political, and scientific circles, including from commentators who have recently claimed that there is no solid proof that the source of lead poisoning in condors is spent ammunition in the form of shotgun pellets and bullet fragments. For example, in response to a petition to the California Fish and Game Commission to stop the use of lead ammunition in the range of the condor in California, the Governor's Office of Constituent Affairs circulated a letter stating among other things that "data collected in California during the past 10 years does not suggest that bullets used only in hunting are the main source of exposure. In fact, there is no firm evidence of the source of any of the lead ingested by condors" (Center for Biological Diversity 2006:1), a statement made a year after the landmark symposium on reintroduction of the condor held at the annual meeting of the American Ornithologists' Union in Santa Barbara, where researchers presented clear evidence to the contrary (Mee and Hall 2007). Likewise, spokesmen for the ammunition and firearms industries have attempted to diffuse the issue by pointing to other sources of manufactured lead in the environment that could affect condors adversely (e.g., R. Patterson, National Shooting Sports Foundation, oral statement to International Association of Fish and Wildlife Agencies Non-toxic Ammunition Working Group, Sep 2006).

In addition, a recent "critical and objective" scientific review of data commissioned by the California Department of Fish and Game (Johnson et al. 2007:i), presented primarily from the clinical and ecotoxicological point of view, concluded that lead from spent ammunition is, indeed, a frequent cause of death and morbidity in condors; however, the authors shied away from any conclusion about population effects, opting, instead, to recommend that ". . . all sources of lead in condor habitat must be identified, prioritized for risk of exposure to condors, and minimized where possible" (Johnson et al. 2007:14). Such scientific caution may be germane to the situation in California where actual fatalities from lead poisoning are few but not in Arizona where ammunition lead is the principal cause of deaths that limit the population growth of reintroduced condors (Woods et al. 2007).

BIOLOGICAL BACKGROUND

A quick review of some pertinent aspects of the condor's life history helps in understanding the significance of lead exposure in this species. The California condor (hereafter condor) is the largest flying bird in North America, with a normal body weight ranging from 8 kg to 10 kg. It is an obligate carrion-feeder, relying mainly on the carcasses of large mammals but also consuming animals down to the size of ground squirrels and rabbits. Like other large birds, the condor has delayed sexual maturity, first breeding successfully on average around 8 years of age, and it also has a very low rate of reproduction estimated to range between 0.25 and 0.37 young per breeding age female per year (Meretsky et al. 2000). Females lay a single egg per clutch. Young are in the nest for half a year or more and require prolonged parental care after leaving the nest. Thus, a successful pair usually reproduces only once in ≥ 2 years, although unsuccessful pairs sometimes re-lay in the same season or will lay the following year; exceptionally a pair reproduces successfully in 2 consecutive years (Snyder and Schmitt 2002).

Such life history traits require long adult lifespans for population survival. A typical pair first breeding successfully at 8 years can then continue to produce one young every 2–3 years. At a pre-adult survival rate of 0.90 and adult rate of 0.98 a breeding pair must on average produce ≥ 5 young in order to replace itself with 2 8-year-old birds, requiring a breeding life of 10–15 years and a lifespan of 18–23 years. Condors are known to live up to 40 years, and some no doubt live longer (Snyder and Schmitt 2002). This requirement for long survival means that even slight changes in mortality—especially adult mortality—can make a big difference in whether the condor population increases or decreases in number. Fatalities caused by unnatural environmental factors, for which the condors have evolved no biological defenses, are especially deleterious.

HISTORICAL PERSPECTIVE

Historically there was little recognition of lead intoxication as a possible cause of death in condors, and a connection between spent ammunition lead and dead condors could not have been made easily. Indeed, lead intoxication is unlikely to have been much of a problem prior to the development of smokeless powder and jacketed bullets in the late 1800s, producing high speed projectiles that fragment on entering an animal's body.

As early as 1976, one dead condor was recovered with very high lead residues in its bone, indicating long-term exposure to lead, although a gunshot wound actually killed it (Wiemeyer et al. 1983). It was not until the intensive fieldwork carried out on the remnant population of wild condors in California in the 1980s that biologists came to recognize lead poisoning from spent ammunition as a serious problem. From 1982 to 1985, 15 condors died or disappeared from a total population of some 24 birds, leaving only 9 remaining in the wild by 1985 (Snyder and Snyder 2000). Only 4 dead condors could be recovered for necropsy. Three of them were clinically diagnosed as dying from lead poisoning, based on high concentrations of lead in liver and kidney and on metallic fragments in the digestive systems of 2 (Scott and Jurek 1985, Jansen et al. 1986, Wiemeyer et al. 1988, Snyder and Snyder 1989). Although these reported fatalities from lead were few, the idea that lead exposure played a major role in the rapid loss of condors

in the 1980s weighed heavily in the decision of the United States Fish and Wildlife Service (USFWS) and the California Fish and Game Commission to remove all remaining condors from the wild and place them in captive breeding programs (Snyder and Snyder 2000).

LEAD AND REINTRODUCED CONDORS

Overview

Confirmation of the role that lead exposure plays in the mortality and morbidity of condors comes from the experiences of the 4 reintroduction programs, 2 in southern and central California beginning in 1995–1997, one in northern Arizona and southern Utah in 1996, and one in Baja California, Mexico, in 2002. The significance of lead poisoning can be gauged by a brief overview of results to the end of December 2006. The following account does not include condors that were released and then returned permanently to captivity.

During 12 years of work in California beginning in 1995, managers released approximately 119 condors and 2 fledged in the wild; approximately 42 died or disappeared (J. Grantham, USFWS California Condor Program, unpublished reports). Only 2 deaths were diagnosed as lead poisoning, and 2 others were judged highly likely, although 4 other condors showed indications of acute lead poisoning (Hall et al. 2007). Power-line collisions and predation were the main known causes of death, but many deaths were undetermined (Sorenson et al. 2001, Grantham 2007, Hall et al. 2007).

During 5 years in Baja California, personnel of the San Diego Zoo released 16 condors and 4 died (J. Grantham, unpublished reports). One of the 4 deaths resulted from lead poisoning, which was associated with an intact bullet from a .22 rifle found in the bird's proventriculus (M. Wallace, San Diego Zoo, personal communication).

In Arizona managers released 88 immature condors and 5 fledged in the wild over 10 years; 36 died, not including 2 released as adults (Austin et al. 2007). The Arizona population has suffered the most from lead poisoning, with 12 diagnosed cases and 2 others likely (38.9% of all fatalities and disappearances, 50% of all diagnosed fatalities). If the same proportion of undiagnosed fatalities resulted from lead poisoning, then total mortality from lead would have been 18 individuals (50% of all deaths). Of the 12 diagnosed deaths from lead 9 were of adults or near-adults >4 years old. Predation by coyotes (*Canis latrans*) and golden eagles (*Aquila chrysaetos*), mainly on immature condors during the first 5 years, was the second most important factor (Cade et al. 2004, Woods et al. 2007).

The actual number of lead-caused deaths in Arizona would doubtless have been higher without the frequent treatment of lead-poisoned condors in what has become the most intensive, hands-on management imaginable—a truly herculean effort (Parish et al. 2007; see section on management below). Without this management, adult condors would not survive long enough to replace themselves in the population, even assuming a normal rate of productivity (Woods et al. 2007).

The impact of lead exposure is, therefore, only partly revealed by fatalities. Every condor that has been in the field for ≥2 years has lead levels in its blood that are elevated above the so-called background level of about ≤10 μg/dL (Hall et al. 2007, Parish et al. 2007, Sorenson and Burnett 2007). For example, in the southern California population on 11 June 2003 one day's sampling of 17 birds revealed laboratory-determined lead levels in blood to average 36.2 μg/dL (range 18.0–66.0 μg/dL) for 9 condors exposed for an average of 82 months (range 38–100 months) after release, but 8 birds exposed for an average of 15 months (range 11–25 months) averaged only 14.5 μg/dL (range 1.0–29.0 μg/dL), based on data summarized by the Center for Biological Diversity et al. (2005). In Arizona and Utah, USA, many condors are in the exposed or subclinical range, in which there are no overt signs of toxicosis (10–59 μg/dL), others are in the clinically exposed range (60–99 μg/dL), which means that physiological malfunctions become evident, and a few condors from time to time are in the range of acute toxicity (>100 μg/dL) and are, therefore, threatened with death if this high intoxication lasts long enough (Franson 1996, Fry 2003, Parish et al. 2007).

Redig (1984) first defined these 4 stages of lead exposure based mainly on examination of bald eagles (*Haliaeetus leucocephalus*). Redig (1984) used <20 μg/dL in blood as the upper limit for background exposure, as did Pattee et al. (1990) and Fry (2003), but there is little agreement as to what the background level of exposure is for condors. Different workers use arbitrary values ranging around 10–20 μg/dL (Fry 2003, Parish et al. 2007), but captive condors that have had no contact with outdoor environments have lead levels in blood ≪10 μg/dL (Dujowich et al. 2005, Church et al. 2006), indicating that subclinical exposure likely extends below 10 μg/dL.

Death from acute toxicity frequently results from paralysis of the neuromuscular system controlling peristalsis and consequent starvation (Eisler 1988, Fry 2003, Fisher et al. 2006). This so-called crop stasis is well known in other birds poisoned by lead (Eisler 1988, Fisher et al. 2006). Fortunately, the rate at which condors eliminate lead from their blood is fairly rapid, with a half-time of about 13 days (Fry 2003). Thus, even if a condor reaches the level of acute toxicity, it may not be mortally affected if its body burden of lead can be reduced soon enough. These considerations, of course, do not address the possibility of long-term sublethal impacts upon condors, as indicated, for example, by studies of intellectual impairment in children with average lead concentrations in blood <10 μg/dL (Canfield et al. 2003).

Reproduction

The long-term reproductive rate of the reintroduced condors has yet to be determined because most recently formed pairs do not succeed in their first 2 or 3 attempts and the breeders are all still young adults; in addition, some released condors have aberrant behaviors affecting reproduction (Mee and Snyder 2007). From 2001 to 2006, of 31

eggs laid in the wild (CA and AZ combined), one was hatched in captivity and the young later released, 16 hatched in the wild (>50%) with 7 fledging in the wild, 8 dying, and 2 placed in rehabilitation, and 14 eggs failed to hatch (J. Grantham, unpublished reports; Mee et al. 2007, Woods et al 2007). Thus, the number of young fledged per laying female (1 egg = 1 F) was only 0.23 over 6 years, well below a population-sustainable level, unless survival were absurdly high; productivity would be even lower if nonlaying females of breeding age were included in the total.

California has a peculiar and unexplained problem: only 2 chicks have fledged on their own from 16 eggs, most of which hatched. The main reason is because the parents regurgitate micro-trash (objects such as metal bottle caps, shards of broken glass, bits of metal and plastic, and other manufactured items) in the food delivered to the young, which then starve as a result of impaction of the proventriculus by these objects or die from internal lesions (Grantham 2007, Mee and Snyder 2007, Snyder 2007).

Arizona's condors have reproduced somewhat better, because they ingest little micro-trash. Five young have fledged from 14 eggs, yielding a productivity of 0.36 per laying female over 6 years (C. Parish, The Peregrine Fund, unpublished data; Woods et al. 2007).

Whether or not this poor reproductive performance could be related to chronic exposure to lead remains unstudied; however, reproductive malfunctions in both males and females have been reported in many animal species clinically exposed to lead, including human beings (Eisler 1988, Henny et al. 1991, Locke and Thomas 1992, Fisher et al. 2006). Regardless of the effects of lead on survival, these reintroduced condor populations cannot become self-sustaining until productivity greatly increases (Mee and Snyder 2007).

MANAGEMENT OF LEAD EXPOSURE IN ARIZONA

Because of the problems associated with lead exposure, it has been standard practice since 2000 to bring all condors back into temporary captivity ≥2 times each year for blood sampling (from the large, medial tarsal vein of the foot) and inspection for signs of morbidity associated with lead poisoning (Parish et al. 2007). Individuals showing signs of sickness are trapped as quickly as possible any time of the year. Luckily it has proved feasible to recapture condors on a regular basis, primarily because even though they fly hundreds of kilometers across the landscape, they often return to the location where they are regularly provided with food.

The Arizona project provides an example of what it takes to manage a population of condors in an environment where lead exposure is an omnipresent hazard (Parish et al. 2007). From 2000 to 2005, 437 blood samples (excluding retests of exposed individuals) showed that 176 (40.3%) had lead levels higher than background; 82 of them were exposed (15–29 μg/dL), 55 indicated birds were in the upper exposed range of 31–59 μg/dL, and 39 exceeded 60 μg/dL in the clinically exposed range. At least 25 of the latter (14.2% of all exposures) were in the range of acute toxicity (100–400 μg/dL). Chelation therapy (2 injections of chelating agent daily for 5 d) had to be administered in 66 cases. Of 50 Arizona condors, 28 received ≥1 chelation treatment, 17 received 2 treatments, 5 were treated 4 times, and 2 had 6 treatments (60 injections). Radiographs of 7 condors (3 alive, 4 dead) revealed shotgun pellets in their stomachs; 7 others (6 alive, 1 dead) showed ingested metallic fragments consistent with those found in deer after being killed with lead bullets (Hunt et al. 2006, Parish et al. 2007).

Since these published findings, 2 other dead condors in Arizona had ingested shotgun pellets in their stomachs and 2 died from crop stasis with lead fragments in their guts after being found alive and taken into captivity. During 2005, 6 condors died or disappeared, 2 from lead poisoning; in 2006, 8 condors died or disappeared, 6 from lead poisoning during the 10 months from mid-March 2006 to mid-January 2007, including 5 condors >4 years old or 21.7% of the 23 birds in that age category alive at the beginning of 2006. In the spring of 2007, only 7 of 12 condors that had nested in prior years remained in the population (Austin et al. 2007; C. Parish, unpublished data).

All the data I summarized above show that condors in northern Arizona regularly ingest lead and that animals killed by shotguns and rifles are a major source of toxic exposure. In fact, to date the only identified source of lead in exposed condors in California and Arizona is from spent ammunition (see Pattee et al. 2006), although possible sources other than carcasses exist, such as lead items in waste dumps and landfills, contaminated ground around lead mines and smelters, contaminated water, spent ammunition at rifle and skeet ranges, atmospheric deposition, and contaminated sewage sludge used as fertilizer (Fry 2003, Johnson et al. 2007). According to Pattee et al. (2006) the only sources other than metallic lead known to poison birds are lead in sediments from mining tailings (swans) and in paint chips (albatrosses [*Diomedea* spp.]).

LEAD AND CONDOR POPULATIONS

What are the population consequences of pervasive exposure to lead from spent ammunition? As noted earlier, survival rate, especially for adults, is key to population stability and growth for long-lived, slowly reproducing animals. Meretsky et al. (2000) postulated that for the purposes of modeling population dynamics of a wild, unmanaged group of condors and of determining mortality, it would be valid to include as fatalities all birds that are saved by human interventions because they would have died under unmanaged conditions. It is not true, however, that all condors with high levels of lead in their tissues will necessarily die because they can eliminate lead from blood and some internal organs rather fast unless continually re-exposed (Fry 2003). Still, it is instructive to consider population trajectories under different assumed death rates that lie within the range of reasonable likelihood in relation to levels of lead poisoning.

In a study from December 1996 to July 2005, Woods et al. (2007) released 77 immature condors in Arizona and 26 (34%) died, including 6 from lead poisoning and 2 others likely. Estimates of annual survival proved to be difficult, because birds ranged in age from 172 days to 965 days old when first released (median 293 d) and because they also spent variable periods of time in captivity after release (64% were in captivity <100 d, 7 birds for 365–1,095 d). For the purpose of approximating annual survival based on biological age, Woods et al. (2007) assumed that the first consecutive 365 days of exposure in the field was equivalent to first year survival and so on.

Use of the Trent and Rongstad (1974) method to determine daily survival (d of exposure in the field) produced approximations of annual survival rates of 79.6% for the first year after release, 89.5% for the second through fourth years, and 97.8% for the fifth year and onward. Assuming a productivity of 0.25 for breeding age females, first breeding at 7 years of age, and a stable age distribution, these survival rates would result in population growth of 2.67% per year.

If all condors with lead levels in blood of >250 μg/dL (n = 10) died on the day encountered, adult survival would drop to 90.9%, and the population would decline at 2.8% per year. If all condors with lead levels of >100 μg/dL (n = 25) died, adult survival would be 76.9%, and the rate of decline would be 18.6% per year (Woods et al. 2007). The survival of adults in 2006 was near this calculated figure.

The impact of these hypothetical changes would have a disproportionate influence on adult and subadult survival, because lead poisoning is virtually the only factor observed to kill condors that have been exposed to field conditions for >3 years. All condors that were free-flying for >2 years had lead levels in blood indicating exposure, but those in the field for >4 years had an average level in the range of clinically exposed (Parish et al. 2007). Out of the 14 condors that died and probably died of lead poisoning from 2000 to the end of 2006, only 5 were exposed in the field for <1,000 days; the other 9 birds ranged from 1,001 days to 3,517 days; whereas, of the 22 birds that died of other causes, 18 were exposed for <1,000 days; the other 4 ranged from 1,256 days to 2,414 days, and 3 of them were in the missing or unknown categories and could well have succumbed to lead poisoning. Fatalities apparently associated with inexperience, such as collisions, depredations, starvation, and shootings, have declined as the population grew older. During the first 5 years 7 condors died from inexperience-related factors and 5 from lead poisoning, but in the second 5 years only 4 died in the first category and 9 from lead (C. Parish, unpublished data). It appears that as duration of exposure increases older condors, more adept at foraging, may become more susceptible to death by lead poisoning, a trend that would preclude development of a population with a normal age structure skewed toward older, breeding-age birds, but see Hall et al. (2007) for an apparently different, age-related trend of exposure in southern California.

On the other hand, if no condors had been killed by lead poisoning during the 9-year study adult survival would have been >98% per year, and population growth theoretically would have exceeded 3.0% (Woods et al. 2007). Thus, owing to lead exposure, this intensively managed population of condors, which had a combined artificial and natural addition of about 9 young condors per year, was delicately balanced between decline and potential growth. Our study confirms that an unmanaged, self-sustaining population probably cannot exist as long as there is a high availability of lead in the food condors eat (Cade et al. 2004, Woods et. al. 2007).

HOW DO WE KNOW LEAD FROM SPENT AMMUNITION POISONS CONDORS?

In summary, there are several lines of evidence that combine to answer this question about the source of lead poisoning.

1. Clinicians and veterinarians necropsy all condors found dead in the wild (B. Rideout, Zoological Society of San Diego, unpublished reports; K. Orr, Phoenix Zoo, unpublished data). Since the 1980s, ≥18 condors found dead or dying have been diagnosed as victims of lead poisoning. Physical and radiographic evidence consists of lead shotgun pellets and metallic fragments from rifle bullets in the digestive tracts of ≥11 condors. In carcasses that could be chemically analyzed, tissue levels of lead in liver, kidney, and blood were in the lethal ranges experimentally determined for other birds such as eagles (Redig 1984), turkey vultures (*Cathartes aura*; Carpenter et al. 2003) and Andean condors (*Vultur gryphus*; Pattee et al. 2006).
2. Observations of moribund condors with toxic lead levels in blood and showing signs of crop paralysis and starvation are indicative of approaching death. In ≥4 cases chelation, surgical removal of lead particles, and forced-feeding reversed crop stasis and the condors recovered to fly free again (J. Grantham, unpublished data; C. Parish, unpublished data).
3. Lead exposure, indicated by blood samples, is virtually ubiquitous among free-flying condors, and many of them reach clinically exposed and acute levels that require clinical intervention and chelation treatments. Furthermore, in southern California 13 dead condors not diagnosed as lead fatalities nevertheless had histories of elevated blood levels of lead and or were thought to have fed on hunter-shot deer prior to death, circumstances potentially contributing secondarily to the diagnosed cause of death (Center for Biological Diversity et al. 2005).
4. Indirect data on food and feeding habits also support the conclusion that condors encounter lead from spent ammunition. Radiographs reveal that lead bullets fragment into hundreds of tiny pieces that lodge in flesh several centimeters distant from the wound channel, so that often there is enough lead in a single deer carcass or gut-pile to poison several condors, which feed in groups (Hunt et al. 2006). In Arizona and Utah radiotelemetry tracking and blood sampling show that levels of lead in

blood increase markedly soon after condors move into a hunting area, such as the Kaibab Plateau, and they are at their lowest levels—with some exceptions—between hunting seasons (Hunt et al. 2007). The same pattern of increased exposure during the hunting season followed by a decrease afterward occurred with golden eagles studied in the California range of the condor (Bloom et al. 1989) and has been noted in the released condors there as well (Hall et al. 2007, Sorenson and Burnett 2007).

5. Church et al. (2006) measured lead concentrations and isotope ratios of lead in blood samples from 18 released condors in central California and compared the results with data from 8 prereleased condors that had minimal exposure to lead. They also measured lead isotope ratios in uncontaminated food and in lead from local soil, water, and air. These background sources had isotope ratios that were highly significantly different from isotope ratios found in a variety of lead shot and bullets commonly used in the condors' range. The captive, prereleased condors generally had low lead levels in their blood, <3.75 μg/dL, and lead isotope ratios that closely matched those from background sources; whereas, lead levels in blood of wild-ranging condors were >3.75 μg/dL and varied from <10 μg/dL to nearly 100 μg/dL in the clinically exposed range. The critical finding was that most of the exposed condors had lead isotope ratios that approximated the ratios found in ammunition lead, and the higher the levels of lead in blood, the closer the samples matched the isotope ratios in lead pellets and bullets.

These results show that elevated levels of lead in condors do not come from environmental background sources, and although the researchers did not measure isotope ratios in other objects manufactured from lead (e.g., batteries, sinkers, balancing weights for wheels, lead foil from wine bottles), their findings are consistent with the conclusion that lead from spent ammunition is a major source of the exposure that compromises the health and survival of condors. A similar study underway at the University of Arizona has produced preliminary results that seem to confirm these published findings (Sullivan et al. 2007).

When all of these lines of evidence are considered together, the conclusion that lead from spent ammunition is a serious hazard to condors becomes scientifically established with a high degree of probability. The degree of threat appears, however, to vary geographically. In California the low number of fatalities attributed to lead in the period since 1995 (Hall et al. 2007, Sorenson and Burnett 2007) indicates that there is no significant population effect by comparison with other causes of death, but the number of condors chronically exposed to clinical or near clinical blood levels of lead argue for the likelihood of long term physiological impacts that will negatively affect survival and reproduction. The lower level of exposure in condors released in the Big Sur region compared to the ones in southern California may be explained by their high consumption of marine mammals, which have low levels of lead in them (Sorenson and Burnett 2007). In Arizona lead poisoning is the principal cause of deaths, and chronic exposure is high in most individuals. The higher level of exposure in the Arizona condors may be explained by their more frequent use of hunter-shot carcasses and their lesser dependence on supplemented food compared to the California birds.

HOW CAN LEAD EXPOSURE OF CONDORS BE REDUCED TO SAFE LEVELS?

Some practices involving management of the condors themselves have potential to help reduce exposure to lead. Provision of lead-free food was tried with wild condors in California without much success (Snyder and Snyder 2000), but managers expected that captive-reared and released condors could be trained to rely principally on an artificial food subsidy (mainly discarded calves from dairies). In both California and Arizona it has been possible to keep condors coming back to established feeding stations, but they also range far enough to locate and feed on carcasses of animals that die in the field. This natural foraging has been much more frequent in Arizona than in California.

Additionally, constant monitoring of condors for signs of lead exposure, chelation, surgical removal of lead particles in the digestive system, and other emergency actions can reduce the harmful impacts of lead exposure on condors; but frequent capture may have deleterious effects on behavior. These are short-term stopgaps, not long-term solutions. The condor projects are not designed to be put-and-take game-farm operations: their purpose is to establish self-sustainable, wild populations.

The bottom line, therefore, is that the occurrence of spent ammunition lead in the environment and in the condors' food must be reduced to achieve background levels of exposure in condors of ≤10 μg/dL of lead in blood. There are 2 proposed ways to accomplish this goal. One is to promote voluntary actions by persuading firearms users to carry out their activities in ways that reduce the exposure of birds to spent ammunition lead. The other is to seek legislative or regulatory relief at the state and federal levels. In 2005–2006 there were 3 attempts in California to follow the latter course—a petition to the California Fish and Game Commission to eliminate the use of lead ammunition in the range of the condor and introduction of 2 bills in the state legislature to do the same thing. All failed, but attempts in 2007 may succeed.

In 2003 the California Condor Recovery Team established a committee to review the lead issue and come up with a set of recommendations on how to reduce the lead exposure of condors. The members of this group represented various hunting, firearms, and ammunition organizations, as well as members of the recovery team, state fish and wildlife departments, and conservation organizations (Redig et al. 2003).

This committee recommended several actions that firearms users could take voluntarily, such as removal of carcasses and gut-piles from the field, but the use of