

# 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  $\geq 2$  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  $\geq 5$  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  $\geq 2$  years has lead levels in its blood that are elevated above the so-called background level of about  $\leq 10$   $\mu\text{g}/\text{dL}$  (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  $\mu\text{g}/\text{dL}$  (range 18.0–66.0  $\mu\text{g}/\text{dL}$ ) 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  $\mu\text{g}/\text{dL}$  (range 1.0–29.0  $\mu\text{g}/\text{dL}$ ), 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  $\mu\text{g}/\text{dL}$ ), others are in the clinically exposed range (60–99  $\mu\text{g}/\text{dL}$ ), which means that physiological malfunctions become evident, and a few condors from time to time are in the range of acute toxicity (>100  $\mu\text{g}/\text{dL}$ ) 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  $\mu\text{g}/\text{dL}$  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  $\mu\text{g}/\text{dL}$  (Fry 2003, Parish et al. 2007), but captive condors that have had no contact with outdoor environments have lead levels in blood  $\ll 10$   $\mu\text{g}/\text{dL}$  (Dujowich et al. 2005, Church et al. 2006), indicating that subclinical exposure likely extends below 10  $\mu\text{g}/\text{dL}$ .

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  $\mu\text{g}/\text{dL}$  (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