

Assessment of Lead Contamination Sources Exposing
California Condors

FINAL REPORT

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Executive Summary:

In 2001 the California Department of Fish and Game requested that I conduct an independent review of the causes of mortality of California Condors and to specifically review the data on lead in the environment in California and the magnitude of the lead exposure hazard to condors. The scope of this study has included the compilation of data on mortality, movements of birds in the field, feeding, monitoring efforts, blood sampling, and the lead exposure data for other species within the condor range in California. This review also examines the potential sources of lead in the environment in California, and assesses the magnitude of the exposure risk of lead to California Condors.

The California Condor Recovery Program has overcome many hurdles in the effort to increase the condor population and return birds to successful independent survival in the wild. Captive propagation to increase numbers has been accomplished at three facilities (San Diego Wild Animal Park, Los Angeles Zoo, and the Peregrine Fund World Center for Birds of Prey, Boise Idaho) with the hatching and rearing of more than 250 chicks over the past 20 years.

Reintroduced California Condors now occupy most of the range within California that they used during the period 1940-1987. Range expansion into Monterey County has occurred with reintroductions near Big Sur. The wild-hatched condors AC8 and AC9, which were trapped in 1986-7 and held in captivity for 14 years, were reintroduced in 2000 and 2001, and they use the traditional foraging areas of the Sierra foothills.

Condors have suffered a high mortality rate in the wild. During the 1970s and 1980s approximately half of the known population disappeared from the wild, with many of the carcasses never found. During that period, at least 3 condors died of lethal exposure to lead. All remaining condors were trapped and kept in captivity until a reintroduction program was begun in the early 1990s. Since 1995, more than 140 condors have been released in California and Arizona, and 44 are free-flying in California at the current time. Four condors have died of lead poisoning since 1997 (1 in California, 3 in Arizona), and 26 condors have received emergency chelation treatment to reduce toxic lead levels (8 in California, and 18 in Arizona). AC8 nearly died in November 2002, when she ingested metallic lead near Hopper Mountain NWR. Expert veterinary care prevented her death, and several other potential lead mortalities since 2000. AC8 was released back to the wild in late December 2002, but she was shot and killed on the Tejon Ranch in early February 2003.

Field biologists have systematically monitored lead exposure in condors since 1997. In California, every bird has had detectable lead in blood samples. 62% of the blood

samples taken in Southern California have shown higher than background lead exposure (blood levels greater than 20 µg/dl blood). 13 of 87 samples (15%) have exceeded 60µg/dl, indicating the condors were clinically affected by lead poisoning, and 6 birds have had blood lead levels greater than 100 µg/dl, indicating acute toxic exposure and requiring emergency veterinary intervention. The half-time for excretion of lead from condor blood is about 13 days, demonstrating that condors will quickly depurate lead if kept in a clean environment. The high number of condors with above background blood levels suggests that the birds are frequently exposed to lead in the wild. Metallic lead fragments have been positively identified in the digestive systems of two condors in California since 1984. Two additional condors (AC3 and 132) had metal fragments in their gut at death, but chemical analysis was not performed to confirm lead. Three other condors with high blood lead levels had X-ray identification of dense particles, also not confirmed by recovery and testing, making a total of seven condors in California suspected of having lead fragments in their digestive systems. In Arizona, seven condors have either had lead pellets recovered from their gizzards, or have been diagnosed by radiography. Six of these 14 birds died of lead poisoning, and the others were successfully treated for acute toxicity. Lead exposure and intoxication continue to be a very critical problem for both the California and Arizona populations.

Seventeen of the 35 dead condors examined by pathologists have not been tested for lead exposure. High-resolution lead and copper analysis techniques should be conducted on feathers that were growing at the time of death of the archived condors to determine the extent of lead and copper exposure in all condors that have died and been recovered since 1982.

Experimental studies with lead intoxication of other species of raptors indicate that condors are not more sensitive to lead intoxication than most species of birds. Turkey Vultures, however, are very resistant to the effects of lead, and are not a good surrogate species to compare with condors. Condors and Turkey Vultures also apparently have very different responses to ingested copper. Copper levels in the livers of condors analyzed at necropsy have all exceeded 20 ppm (21-181 ppm), although the significance of copper residue levels is unknown. Copper levels should be studied in feathers, to determine whether a relationship exists with lead exposure.

Potential sources of lead within the condor range include residual atmospheric lead deposition from leaded gasoline exhaust, lead in the soil from natural deposits, from disposal of lead containing items, and lead ammunition on rifle ranges. Current industrial air emissions of lead in California are small, totally only 9301 pounds in 2001, and most emission sources are outside of the condor range and not in the prevailing wind pattern.

Hunting within the condor range is extensive, and the number of game animals taken by hunters in 2001 was estimated at 106,049 for the eight counties within the condor range. This review estimates the number of large animal carcasses and field-dressed visceral remains (gut-piles) left in the field at more than 30,000 annually. This includes the gut piles of 8,180 deer, 17,249 wild pigs, and the carcasses of 10,816 coyotes, plus unrecovered carcasses of deer and pigs crippled by hunters and depredation permit

holders. The number of ground squirrels shot within the condor range is unknown, but almost certainly represents a significant source of carrion with the potential to contain lead fragments that could be ingested by both condors and other scavenging birds and mammals. The risk of lead exposure to condors is high, because of the amount of carrion left in the field. Direct observations of condors feeding on hunter-shot carrion are few, even with intense radio telemetry and visual surveillance, and every effort should be made to document and organize the field observation data to quantify the exposure hazard from hunter-shot carrion. Every effort should be made to educate hunters and depredation permittees of this potential problem, and to encourage or require the use of commercially available lead-free and “lead-safe” rifle and shotgun ammunition.

Field and captive management efforts by the condor program have been highly professional and the overall program is very successful. The field program has concentrated on a feeding program to prevent exposure to lead, monitoring the birds to determine movements, capture of birds to keep radio transmitters functional and to collect blood samples to monitor for lead exposure, and emergency intervention to prevent the debilitation or death of birds exposed to lead. The program has become very labor intensive, but the efforts to prevent lead exposure have not been successful. If a reduction in condor lead exposure can be accomplished, the Condor Recovery Program shows every promise of successfully rebuilding a stable wild population.

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I. Introduction

In 2001 the California Department of Fish and Game requested that I conduct an independent review of the causes of mortality of California Condors and to specifically review the data on lead in the environment in California and the magnitude of the lead exposure hazard to condors. The scope of this study has included the compilation of data on mortality, movements of birds in the field, feeding, monitoring efforts, blood sampling, and the lead exposure data for other species within the condor range in California. This review also examines the potential sources of lead in the environment in California, and assesses the magnitude of the exposure risk of lead to California Condors.

Documented exposure to lead was responsible for the deaths of 3 condors in California the 1980s, and several additional birds disappeared during the winter of 1984-1985, leaving only 6 condors alive in the wild. Condor AC3 was captured in January 1986 with acute lead poisoning, and she died in captivity, leaving only 5 condors in the wild. All remaining condors were captured and brought into captivity by 1987.

The California Condor Recovery Program has overcome many hurdles in the effort to increase the condor population and return birds to successful independent survival in the wild. Captive propagation to increase numbers has been accomplished at three facilities (San Diego Wild Animal Park, Los Angeles Zoo, and the Peregrine Fund World Center for Birds of Prey, Boise Idaho) with the hatching and rearing of more than 250 chicks over the past 20 years.

The condor program began to release condors back into the wild in California in 1992, but with the setback of behavioral problems requiring the recapture and improvement of behavioral conditioning and socialization to improve the survival of released birds.

In 1995 the condor program again began releasing and carefully monitoring condors, first in California, then in Arizona near the Grand Canyon. A few behavioral problems still remain, and some accidental deaths have occurred in the wild, but the three condor field teams (Southern California USFWS, Ventura Office (referred in this report as the "Hopper team", the Ventana Wilderness Society, and the Peregrine Fund program in Arizona and southern Utah) have developed sophisticated programs to cope with the many logistical problems associated with the release and monitoring of a population that has steadily grown to more than 70 free-flying condors.

Constant monitoring of movements and behavior, the use of older birds to socialize and assist young birds with the difficult transition to living in the wild, and a supplemental

feeding program to insure a stable and wholesome food supply all have provided a fundamentally sound program to provide for the survival of condors released into former ranges in California, Arizona, and recently, Baja California, Mexico.

In spite an extensive program of managed substitution feeding to reduce the incidental exposure of birds feeding on carcasses containing lead ammunition fragments, at least 3 condors have died of acute lead poisoning in Arizona in 2000 (#s 116, 165 and 191), one died in California in 2001 (#132), and one condor was fatally poisoned in southern Utah in 2002 (#240). Additionally, 8 condors in California and 18 condors in Arizona have been treated by veterinarians for acute lead toxicity since 1997. Without these emergency measures, several additional condors would almost certainly have died of lead intoxication. Metallic lead fragments have been positively identified in the digestive systems of two condors in California since 1984 (IC1 and AC8). Two additional condors (AC3 and 132) had metal fragments in their gut at death, but chemical analysis was not performed to confirm lead. Three other condors (105, 113, and 130) with high blood lead levels had X-ray identification of dense particles, also not confirmed by recovery and testing, making a total of seven condors in California suspected of having lead fragments in their digestive systems. In Arizona, seven condors have either had lead pellets recovered from their gizzards, or have been diagnosed by radiography.

In California, all condors in the wild have been tested for lead each year since 1997, and every bird has had detectable lead in blood samples. 62% of the blood samples taken in Southern California have shown higher than background lead exposure (blood levels greater than 20 µg/dl blood). 15 of 87 samples (16%) have exceeded 60µg/dl, indicating the condors were clinically affected by lead, and 6 birds have had blood lead levels greater than 150 µg/dl, indicating acute toxic exposure requiring emergency veterinary intervention. (Section I. C presents a discussion of lead measurements and values).

Field management efforts by the condor program have concentrated on a feeding program to prevent exposure to lead, monitoring the birds to determine movements, capture of birds to keep radio transmitters functional and to collect blood samples to monitor for lead exposure, and emergency intervention to prevent the debilitation or death of birds exposed to lead. The program has become very labor intensive and the efforts to prevent lead exposure have not been successful. The sources of lead exposure are discussed in this report, and recommendations for reducing lead exposure to condors and other wildlife are offered.

Section I: The Lead Problem: Number of Birds Exposed, and Magnitude of the Problem.

I. A. History of Known Condor Lead Exposure Incidents

For most of the past 100 years, the remnant population of California Condors (*Gymnogyps californianus*) has occupied the foothills and mountains of the California Coast Range, the Tehachapi Range, and the Sierra Nevadas surrounding the San Joaquin Valley as shown in Figure 1.



Figure 1. Condor range in southern California redrawn from the Condor Recovery Plan (1984 revision). The 8 counties encompassing the condor range are identified.

The range outlined is that identified by the Condor Recovery Team (Condor Recovery Plan, 1984 revision). After the period 1987-1992, when all condors were captured and remanded to captivity, condors were reintroduced into California in 1992, then in 1995. Recently, there has been range expansion into the coastal mountains along the Big Sur

coast with the introduction of condors by the Ventana Wilderness Society (VWS), and into Arizona and southern Utah by the condor field program managed there by the Peregrine Fund.

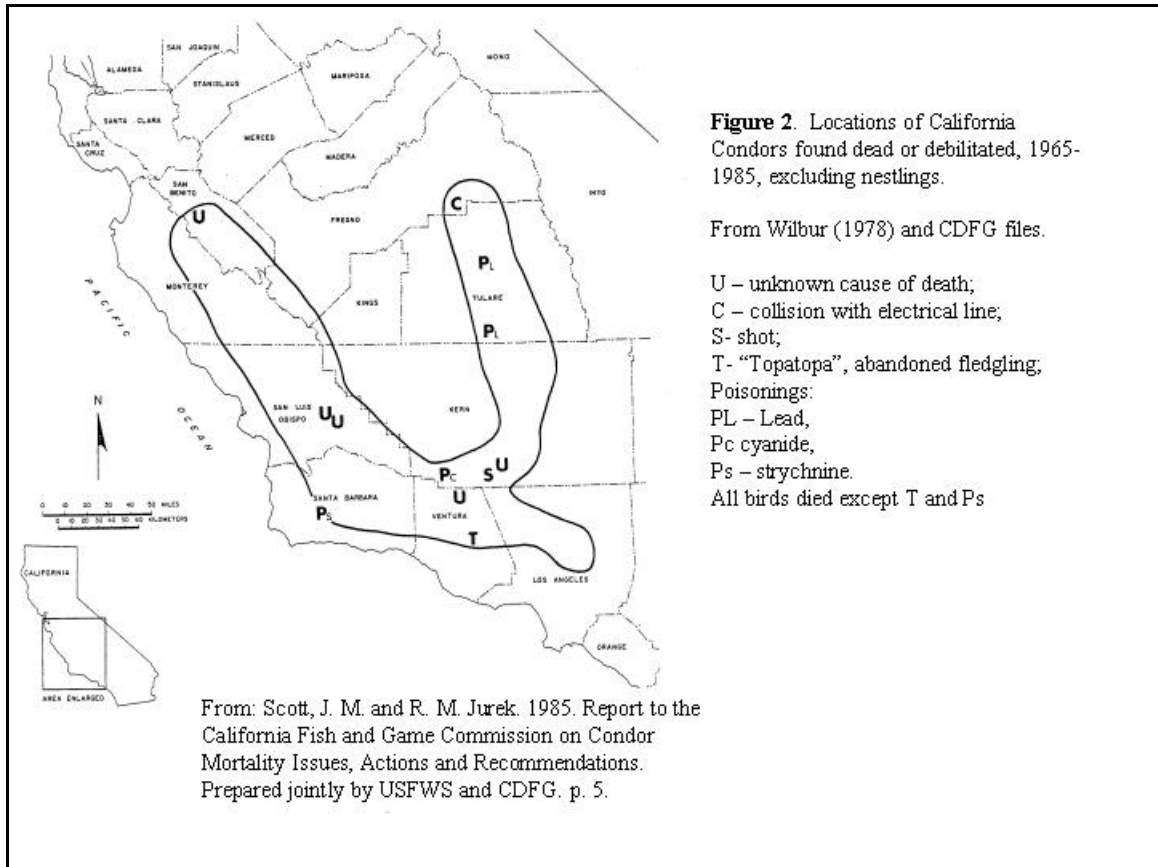
The history of condors being exposed to lead has been reviewed by Scott and Jurek (1985), and Snyder and Snyder (1989), and has subsequently been discussed at length by Snyder and Snyder (2000), and Sorenson et al (2001). Scott and Jurek (1985) reported on deaths of 19 wild condors between 1960 and 1985 (Table 1).

<u>Date</u>	<u>County</u>	<u>Age/Sex</u>	<u>Cause</u>
27 Jun 1960	Kern	Ad/M	U
Aug 1960	Ventura	U/U	U
11 Aug 1960	Kern	Imm/U	U
Fall 1960	Kern	U/U	Shot
About 1961	Kern	U/U	U
About 1961	Kern	U/U	U
23 Sept 1963	Kern	Ad/U	U
23 May 1965	Fresno	Imm/U	Collision
Aug 1965	Kern	Imm/U	U
Dec 1965	San Benito	U/U	U
27 Oct 1966	Ventura	Nestling/U	U
Fall 1972	S. L. Obispo	Ad/U	U
Fall 1972	S. L. Obispo	Ad/U	U
Fall 1974	Kern	Imm/U	U
Fall 1976	Kern	Ad/F	Shot
30 Jun 1980	Santa Barbara	Nestling/F	Handling
23 Nov 1983	Kern	Imm/F	Cyanide Poisoning
22 Mar 1984	Tulare	Imm/M	Lead Poisoning
10 Apr 1985	Tulare	Ad/M	Lead Poisoning

Key: U-Unknown, Ad-adult, Imm-immature, F-female, M-male

Table 1. Known Condor Mortalities and Causes, 1960-1985. Scott, J. M. and R. M. Jurek. 1985. Report to the California Fish and Game Commission on Condor Mortality Issues, Actions and Recommendations. Prepared jointly by USFWS and CDFG. p. 4.

Seven of the 19 could be attributed a cause of death, and 2 of these 7 were attributed to lead. Two others were shot, one nestling died of handling stress, one was an apparent overhead wire collision victim, and one bird was poisoned with cyanide after setting off an M-44 coyote control device. The locations of condors found dead or debilitated in the wild from 1965-1985 is given in Figure 2, from Scott and Jurek (1985).



Scott and Jurek also documented sub lethal lead exposures in 10 birds tested for lead residues between 1982 and 1985 (Table 2). Levels ranged up to 1.2 ppm in blood of birds that survived, and levels of 23 ppm and 35 ppm in liver samples of birds that had died.

Residues in bone of one condor (“Tehachapi”) that was shot in Kern County in 1976 measured 79 ppm (dry weight basis, Weimeyer et al. 1983), indicating long-term exposure to lead, even though shooting rather than lead poisoning was the cause of death. Bone levels in Condor “Broken Feather” measured 72 ppm at the time of death from acute lead poisoning. This high level in bone almost certainly indicates long-term, multiple sub lethal exposures, as bone accumulates lead slowly, and this amount could not have been incorporated into bone during a single acute toxic event.

Condor	Sex	Age	Date		Lead (ppm wet wt.)	
			Trapped (T) Died (D) Captured (C)	Date Sampled	Blood	Other Tissue
1	M	3-4 yr. 5-6 yr.	10/12/82 (T) 3/22/84 (D)	10/12/82 -/-/84	5.5* 24.0 (clot)	35.0-liver, 47.0-kidney
2	M	Ad	11/13/82 (T)	11/13/82	n.d. (<0.05)	
3	F	Ad	11/13/82 (T) 10/2/84 (T)	11/13/82 10/2/84	n.d. (<0.05) 0.27	
4	M	4 yr.	10/11/84 (T) 6/25/85 (C)	10/11/84	0.13	
5	M	Ad	10/19/84 (T)	10/19/84	0.78	
6	M	Ad	10/22/84 (T)	10/22/84	1.2	
7	M	Ad	11/7/84 (T)	10/7/84	0.77	
8	F	Ad	10/12/84 (T)	10/12/84	0.19	
9	M	4 yr.	12/11/84 (T)	12/11/84	0.11	
UN1	F	Ad	8/8/85 (C)	8/85	0.32	
"Paxa"	M	1 yr.	12/5/82 (C)	12/5/82 8/29/84	n.d. (<0.05) 0.09	
"Broken Feather"	M	5 yr.	4/10/85 (D)	-/-/85		23.0-liver, 72.0-bone
"Bosley"	F	1 yr.	11/23/83 (D)	-/-/83		n.d. (<0.10)-liver
Pine- hurst	U	2 yr.	5/23/65 (D)	1/14/66		0.86-liver & kidney, 0.58-crop
"Tehachapi" F		Ad	9/17/76 (C) 10/30/76 (D)	11/-/76		<0.5 ppm-kidney, <0.5 ppm-flesh; 1.6-liver, 1.0-kidney 79.0-bone, tarsus (ppm dry weight)

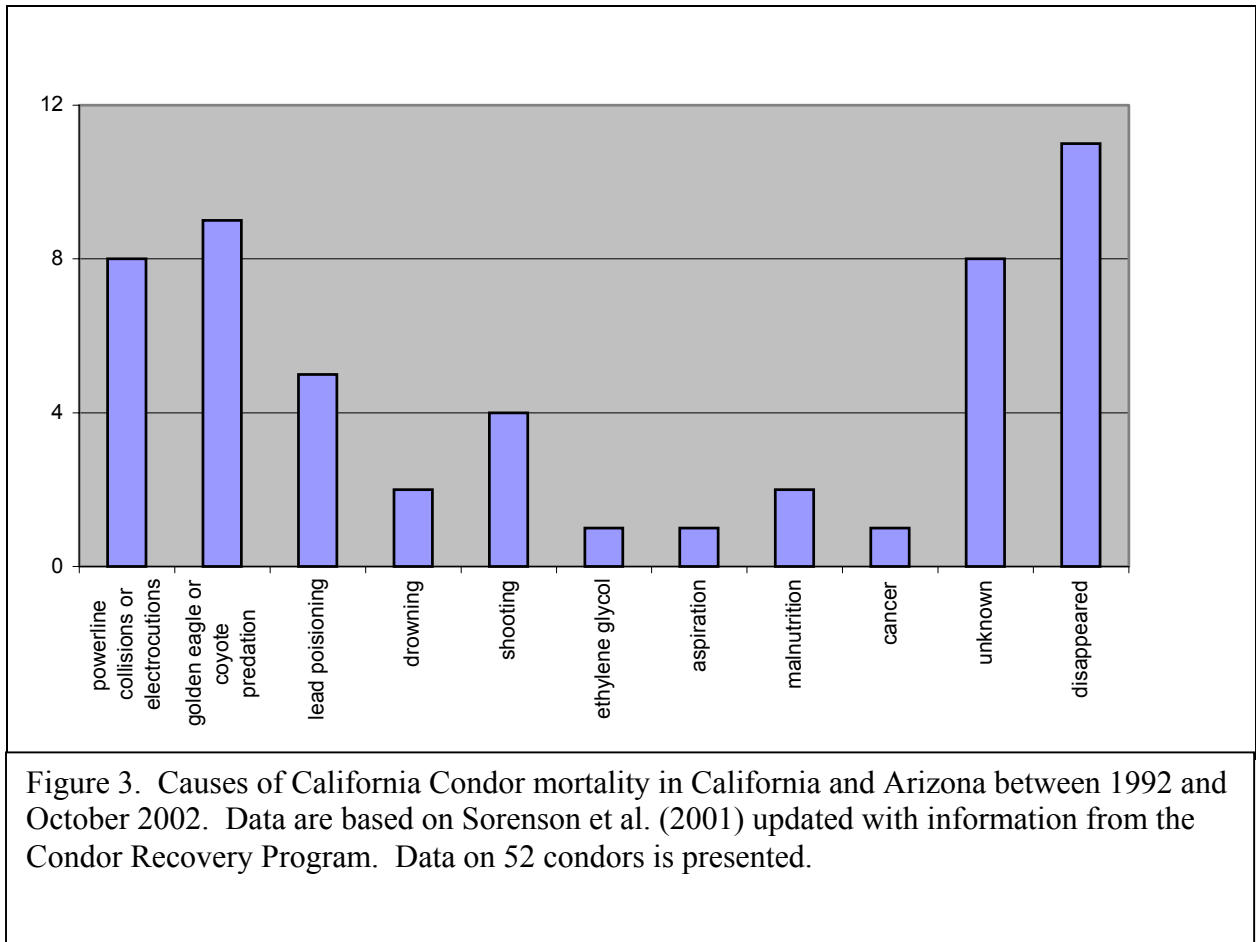
*Possible equipment contamination
n.d. - none detected

Table 2. Lead Levels in Wild Condors. From: Scott, J. M. and R. M. Jurek. 1985. Report to the California Fish and Game Commission on Condor Mortality Issues, Actions and Recommendations. Prepared jointly by USFWS and CDFG. p. 12

Snyder and Snyder (1989) documented one additional condor (AC3 or SBF) that died due to lead poisoning in 1986, and ten birds that disappeared in the wild between 1982 and 1986, before all remaining wild birds were taken into captivity by April 1987.

Since re-introductions began in January 1992, 54 condors have died in the wild in California and Arizona, not including mortalities of wild-hatched chicks. The deaths include 5 due to lead poisoning, 8 from unknown causes, and 11 that disappeared and are presumed dead.

The cause of death remains unknown in several of the cases due to the heavily scavenged or skeletonized condition of the condor carcasses when recovered. Because the internal organs were missing, toxicological analyses were not performed. Analysis of lead residues in growing feather follicles at the time of death of these birds would be necessary for an accurate assessment of whether lead was a factor in the deaths. The mortality data are summarized in the graph, Figure 3, adapted and updated from Sorenson et al. (2001), which lists the causes of death for 43 of the 116 condors released to the wild at the time of their report.



I. B. Historic Lead Exposure Deaths in California and Arizona.

The first condor to be documented with lead toxicity was IC1, a male condor discovered dead March 22, 1984, and the events are discussed at length by Snyder and Snyder (2000). Condor biologists had been tracking this bird by radio telemetry with a plane, and became concerned when he did not fly from his roosting site for the entire day. At necropsy, a bullet fragment was discovered in his stomach, and blood lead levels from clotted blood measured 24.0ppm. This extremely high blood level appeared to be responsible for a rapid death. Telemetry records have not been reviewed to determine the movements of this bird in the days prior to his death, although they could shed some light

on possible locations of lead exposure, movements during the initial exposure period, and onset of clinical symptoms.

In 1985 “Broken Feather” (BFE, Studbook #18), was observed frequently in Ventura County with a mate making nest site inspections and frequently copulating. Both birds left the area on February 25, but only UN1, the mate, returned to the nesting area on February 27. BFE was not located until April 9 (43 days later) when he was found emaciated and near death in Tulare County. He weighed 5.47 kg, about 60% of his expected weight, and he died in Tulare County just as a team from the Los Angeles Zoo was dispatched to examine and treat him. No metallic lead was found in the digestive system, but liver lead residues measured 23 ppm, a level considered lethal for a condor. The source and location of lead exposure is unknown, and the date of exposure is unknown. Because the behavior of the BFE appeared normal until February 25, and he did not return to the nest site on February 27, it is reasonable to infer that he may have ingested lead on or prior to that date, and became separated from his mate because of the initial clinical symptoms. It is possible that both birds flew to Tulare County on February 25, and UN1 returned on February 27, although the distance is more than 100 miles, and would have been a very quick trip to forage and return to Ventura County. Alternatively, the pair may have foraged somewhat nearer to Ventura County, or split up and foraged alone in different areas. BFE (#18) may have flown to Tulare County alone, ingested lead there, and remained in Tulare County, because of quickly elevated lead levels. The fact that he was still alive on April 9, 1985, however, indicates that he was exposed to lead for a long period of time, and was apparently not eating. The 43-day interval between the times he was observed with normal behavior and the time of discovery close to death makes all of the inferences as to his movements and the locale of ingestion highly speculative. The time interval between ingestion of a lead fragment and the onset of clinical symptoms is unknown.

The third case is that of “Santa Barbara Female” (SBF, AC3). This bird was fitted with conventional radio transmitters, one of which was found to be defective, and she was trapped on November 23, 1985 in southern Kern County for transmitter replacement. A blood sample was taken at the time of radio replacement, and she was released back to the wild. Subsequent to her release, residue analysis of blood revealed a lead level of 1.8 ppm (180 µg/dl), and the decision was made to recapture her for treatment. She was not recaptured until January 3, 1986 (41 days later), when she was captured by hand on the Hudson Ranch (southwest corner of Kern County). At the time of her initial capture, she was not exhibiting unusual behavior, even though she had a significantly elevated blood lead level. She remained flying in the wild for 40 days, before she became so debilitated she could be captured by hand. At the time of her capture, she had symptoms of crop paralysis, and a blood lead level of 4.2 ppm (420µg/dl). Lead chelation therapy was initiated, and her blood lead levels dropped, although the digestive system paralysis could not be reversed. AC3 (SBF) died on January 18, 1986.

In 2000, the Peregrine Fund condor program in Arizona experienced two series of lead exposure incidents with the death of 5 condors (3 confirmed and 2 suspected of lead toxicity) and 7 condors requiring chelation therapy to reduce blood lead levels.

Condor 116 was discovered dead on March 2, 2000, with a blood lead level of 320 μ g/dl. All 24 remaining condors were trapped and tested for lead by April, 2000. Only one (Condor 119) was discovered to have high blood lead levels (109 μ g/dl), and she was chelated to reduce blood lead to 10 μ g/dl and released.

SB165, a condor released to Arizona, was observed feeding with other condors on June 6, 2000, and was found dead with 17 lead shotgun pellets in its gizzard on June 12, 2000. The time from exposure to death may have been the six days from June 6 to 12, although the bird might have picked up lead earlier, and not shown clinical symptoms until just before death. Liver lead residues measured 34 ppm at necropsy. The body weight of SB165 was 7.75 kg at death, which is lower than the average body weight, but not at a starvation level.

A second Arizona bird (#191) was found alive on June 15, 2000, in very poor condition (body wt 4.9 kg), with no lead visible on X-ray. This bird died June 16, 2000 with 17 ppm liver lead residue at necropsy. Condor 182 was discovered dead on June 20, but too scavenged to provide liver samples for lead analysis. Condor 150 was presumed dead on July 25, 2000, when her radio transmitter was located motionless in an inaccessible part of the Grand Canyon. The remaining Arizona condors were re-trapped, and 6 additional birds required chelation therapy to reduce blood lead concentrations to safe levels.

In mid-August, 2002, Condor 240, released in Arizona, was found dead in southern Utah. The preliminary necropsy reports indicate lead bullet fragments in the gut. Other condors were recaptured and temporarily held in field pens, but data on lead analyses and outcome have not been obtained.

Condor #132 died in California in 2001 of apparent lead poisoning. Condor 132 was found in San Luis Obispo County near Castle Crags, and had been in the area for 3 days. It was seen flying 15 minutes before death. At necropsy, liver lead residues were 26.4 ppm, and body weight was 7.25 kg, low, but not at starvation level. A small metal object was discovered in the digestive tract at necropsy, but not analyzed.

From the data on these 8-10 condor deaths attributed to lead, it appears the clinical symptoms can vary greatly, and the time course of death may be a matter of a few days, or prolonged to as much as 40+ days. Very high blood lead levels appear to be acutely toxic, with death possibly occurring within days. Such circumstances have occurred with lead still in the stomach (proventriculus or gizzard), where acidic conditions can rapidly dissolve and mobilize the metallic lead, leading to toxic blood lead levels. Many of the birds demonstrated sequelae of prolonged duration, although no information is available as to the initial exposure, or time course of retention of lead fragments in the digestive tract, if that is, in fact, how these birds were exposed. Clinically, these birds became moribund and appeared to starve. In the case of AC3, she ate food after being captured and treated, but the food never left her crop, and she continued to lose weight. Lead induced paralysis of the digestive system and starvation appeared to be the proximate causes of death.

The flight range of condors makes it generally impossible to identify with certainty the location where individual birds have ingested lead. Two birds were located in Tulare County intoxicated, but that does not guarantee that the birds ingested lead in that county. The satellite telemetry data from California and Arizona released birds demonstrate that condors can travel more than 100 km a day. Given movements of this magnitude, a condor ingesting lead could probably move from one side of the California Condor Range to the other before it suffered any clinical signs of lead intoxication. Only after becoming moribund and immobile for several days would the field biologists be aware that the bird was in trouble. On most occasions, however, condors are discovered to be lead intoxicated upon routine trapping, and clinical measures must be taken to prevent continued intoxication.

During the 1980s only 4 of 14 condors that were missing and presumed dead in the wild were ever recovered (Snyder and Snyder, 2000). Three of these died of lead intoxication. The causes of the disappearance of the other 10 birds are unknown, but if the random causes of death were consistent among all of the individual birds, it could be expected that several of the missing birds could have died of lead exposure.

I. C. Recent California Lead Exposure Incidents

The lead poisoning cases of wild and captive-reared condors have been compiled and tabulated by the Ventura office, USFWS condor program. That table is an accounting of all cases, including veterinary intervention in cases with high blood lead levels, and is presented as Table 4.

The blood lead levels that indicate exposure or intoxication to condors are similar to values in the clinical literature and research reports such as the work of Pattee et al (1990) and the work of Redig, reported by Kramer and Redig (1997). **Background** levels of lead are expected to be less than 20µg/dl of blood (20 micrograms of lead per 100 ml of blood, which can be converted to the standard toxicological units of µg/g, (or ppm, parts per million) by multiplying by 100. Thus, 20µg/dl equals 0.2 ppm. Birds with lead levels above 20µg/dl are considered to be **exposed**, and if the levels rise above 60 µg/dl the bird is considered to be **clinically affected**. Lead toxicity reaches an **acutely toxic** level when blood lead levels rise above 100µg/dl, or 1.0ppm.

<20 µg/dl (0.2 ppm)	= Background
20-59 µg/dl (0.20-0.59 ppm)	= Exposed
60-99 µg/dl (0.60-0.99 ppm)	= Clinically Affected
>100 µg/dl (>1.00 ppm)	= Acute toxicity

The condor program uses these ranges as a guideline in evaluation of birds, and these ranges will be used throughout this report as indications of toxicity during lead exposure incidents.

Table 4: Lead Exposure Cases for California Condors: Source: USFWS, Ventura Office

Bird ID	Year	Date Sampled	Blood Lead	Liver Lead	Disposition/Other Notes
S.B. Chick	1980	06/30/80	N/A	1.0 ppm	Chick was found dead
IC 1	1982	10/12/82	5.5 ppm	N/A	Bird appeared healthy - it was suspected the sample was contaminated; bird was set free
IC 1	1984	03/22/84	24.0ppm	35.0 ppm	Bird was found dead
Broken Feather	1985	04/10/85	N/A	23.0 ppm	Bird died approximately 15 minutes after biologists found it
AC3	1982	11/82	.05 ppm	N/A	Routine check; bird was released
AC3	1984	10/84	.27 ppm	N/A	Routine check; bird was released
AC3	1985	11/85	1.8 ppm	N/A	Routine check; bird was released
AC3	1986	01/03/86	4.2 ppm	N/A	Bird exhibited signs of illness and grew progressively weaker; taken to San Diego Wild Animal Park. Evidence of crop stasis
AC3	1986	01/09/86	.81 ppm	N/A	Chelation treatment initiated. Blood lead levels dropped following treatment; crop stasis still evident
AC3	1986	01/13/86	1.08ppm	N/A	Blood lead levels increased several days after chelation; crop stasis continued
AC3	1986	01/16/86	N/A	.57 ppm	Bird died and a necropsy was performed; cause of death was determined to be related to effects of lead poisoning
64 (W64)	1992	09/22/92	1.6 ppm	N/A	This bird was set free 01/14/92. Bird appeared healthy at the time the sample was taken and was released to the wild
64 (W64)	1992	11/05/92	<.10ppm	N/A	Blood lead levels below detection limit; bird was re-released. Later recaptured and absorbed into captive breeding population
130 (Y30)	1997	09/03/97	40.2µg/dl	N/A	Bird was set free 02/13/96. **Was one of 16 free flying condors captured 09/03/97 based on concerns they fed on the carcass of an animal that was shot near Lion canyon release site

130 (Y30)	1997	09/09/97	56.2µg/dl	N/A	Field x-rays taken; questionable evidence of lead fragment in digestive tract - a second blood sample was taken; results indicated a rise in blood lead levels. Bird was taken to LA Zoo for chelation treatment, although it exhibited no signs of illness
130 (Y30)	1997	09/12/97	36.4µg/dl	N/A	Blood lead levels dropped following chelation. Bird was returned to Lion Canyon holding area until final test results were received
130 (Y30)	1997	09/18/97	5.6 µg/dl	N/A	Blood lead levels returned to normal; bird was set free at Lion Canyon
113 (R13)	1997	09/03/97	25.1µg/dl	N/A	Bird was set free 08/29/95. **See first entry for Y30
113 (R13)	1997	09/09/97	75.6µg/dl	N/A	Field x-rays taken; questionable evidence
113 (R13)	1997	09/12/97	46.8µg/dl	N/A	Radiographs taken at L.A. Zoo revealed no lead fragments. Bird was given 5 days of chelation treatment. Bird was returned to Lion Canyon holding area until final blood results were received.
113 (R13)	1997	09/18/97	12.7µg/dl	N/A	Bird was released to the wild at Lion Canyon
105 (W5)	1997	09/03/97	157.3 µg/dl	N/A	Bird was set free 02/08/95. **See first entry for Y30. Radiographs taken in the field showed the presence of a small metallic fragment in lower intestinal tract. Bird was taken to L.A. Zoo for treatment.
105 (W5)	1997	09/07/97	88.8µg/dl	N/A	Blood lead levels dropped following 5 days of chelation treatment.
105 (W5)	1997	09/09/97	15.2µg/dl	N/A	Radiographs taken while bird was undergoing treatment at the L.A. Zoo showed the fragment passed through.
105 (W5)	1997	09/13/97	11.2µg/dl	N/A	Bird was transported to holding area at Lion Canyon pending results of final blood tests. Blood lead levels returned to normal and W5 was re-released to the wild.

105 (W5)	1998	05/01/98	291.4 µg/dl	N/A	Following the 1997 lead incident, W5 behaved normally. On 04/30/98 biologists noted that the bird appeared ill - it was unable to stand upright, weight was shifted onto its hocks, and it walked with toes 'knuckled under'. The bird was captured and immediately transported to the L.A. Zoo. Blood levels revealed highest bloodlead levels recorded in a California condor.
105 (W5)	1998	05/04/98	84.9µg/dl	N/A	Aggressive chelation treatment was initiated upon its arrival at L.A. Zoo
105 (W5)	1998	05/06/98	71.7µg/dl	N/A	Chelation treatment continued
105 (W5)	1998	05/08/98	41.2µg/dl	N/A	Chelation treatment was administered for 10 days then stopped. Crop stasis was evident
105 (W5)	1998	05/11/98	28.6µg/dl	N/A	Although blood lead levels continued to decrease, crop stasis continued
105 (W5)	1998	05/13/98	23.7 µg/dl	N/A	Veterinarians at L.A. Zoo inserted a stomach tube to ensure the bird received nourishment
105 (W5)	1998	05/20/98	36.4 µg/dl	N/A	Spike in blood lead levels. W5 continued to receive nourishment via a stomach tube; crop stasis continued
105 (W5)	1998	05/28/98	31.9 µg/dl	N/A	Veterinarians continued to provide supportive care for W5
105 (W5)	1998	06/11/98	14.1 µg/dl	N/A	The stomach tube was removed and the bird was fed a ½ lb portion of meat which was passed normally through the crop. A second ½ lb of meat was fed the following day which also passed normally. A full pound was given to W5 on 06/13/98; a portion of this was digested, but some remained in the crop. Small feedings continued to encourage restoration of normal crop function
105 (W5)	1998	06/23/98	30.9 µg/dl	N/A	Blood lead levels rose again and chelation treatment was initiated for a period of 5 days
105 (W5)	1998	07/01/98	14.7 µg/dl	N/A	Bird showed continued improvement
105	1998	07/06/98	12.8 µg/dl	N/A	Continued improvement

105 (W5)	1998	07/20/98	15.0 µg/dl	N/A	Continued improvement
105 (W5)	1998	08/06/98	17.4 µg/dl	N/A	After normal crop function returned, W5 was moved from the L.A. Zoo's hospital into a flight pen with other condors
105 (W5)	1998	09/18/98	No sample taken	N/A	W5 did well in the flight pen and continued to regain its strength. It was transported to Lion Canyon on 09/18/98 and released
98	1997	09/03/97	28.8	N/A	Set free on 02/08/95. Captured on 09/03/97 based on concerns it fed on the carcass of an animal that was shot near Lion Canyon
98	1997	09/14/97	18.5	N/A	Blood lead levels returned to normal and 98 was set free. It was subsequently treated for lead poisoning in 1998 - see entry on next page
98	1998	08/31/98	194.6	N/A	This bird was set free 02/08/95. **Captured as part of a routine transmitter replacement activity on 08/31/98. Blood samples were submitted for analysis and results were received on 09/04/98. The bird was transported to the L.A. Zoo for chelation treatment although it did not exhibit signs of illness at the time of its capture.
98	1998	09/14/98	42.1	N/A	Blood levels dropped after 5 days of chelation treatment.
98	1998	09/17/98	37.1	N/A	After receiving final blood test results the bird was transported to Bitter Creek NWR and released
98	1999	10/21/99	15.0	N/A	Bird was captured on 10/21/99 for routine replacement of radio transmitters. Blood samples were submitted for analysis. Lead levels were normal. Bird was re-released to the wild.
111	1998	08/31/98	186.2	N/A	Bird was set free on 08/29/95. ** See initial entry for 98 Bird was transported to L.A. Zoo for chelation treatment.
111	1998	09/14/98	44.1	N/A	Blood levels dropped after 5 days of chelation treatment.
111	1998	09/17/98	44.1	N/A	After receiving final blood tests results, 111 was transported to Bitter Creek NWR and re-released

111	1999	10/21/99	45.0	N/A	Bird was captured on 10/21/99 for routine replacement of radio transmitters. Blood samples were submitted for analysis. Results indicated exposure to lead, but chelation treatment was not initiated; bird was re-released.
143	1997	09/14/97	26.0	N/A	Bird was set free 11/19/96. Captured on 09/14/97 to determine if it was exposed to lead after feeding on the carcass of an animal that was shot near Lion Canyon. Re-released to the wild.
132	1997	09/14/97	38.7	N/A	Bird was set free on 11/19/96. ** See entry for 143.
132	1998	08/31/98	54.0	N/A	Recaptured on 08/31/98 to replace radio transmitters. Blood samples were submitted for analysis. Results indicated exposure to lead, but chelation treatment was not initiated; bird was re-released.
106	1997	09/03/97	27.2	N/A	Bird was captured on 08/31/97 based on concerns it fed on the carcass of an animal that was shot near Lion Canyon.
106	1997	09/14/97	13.1	N/A	Blood lead levels returned to normal and 106 was re-released.
107	1998	08/31/98	46.4	N/A	Bird was set free on 08/29/95. **Captured to replace radio transmitters on 08/31/98. Bird was re-released to the wild.
107	1999	11/08/99	26.0	N/A	Bird was captured and transported to the L.A. Zoo to receive treatment for a compound fracture of the right toe on his left foot. Blood samples were taken and indicated exposure to lead but chelation treatment was not initiated. Bird was re-released on 12/15/99.
112	1998	08/31/98	45.2	N/A	**See entry for 107 on 08/31/98
102	1998	08/31/98	44.8	N/A	**See entry for 107 on 08/31/98
102	1999	10/21/99	8.0	N/A	Bird was captured for routine radio transmitter replacement on 10/21/99. Bird was re-released to the wild.
125	1999	10/21/99	27.0	N/A	Bird was released on 02/13/96. **Captured to replace radio transmitters on 10/21/99. Bird was re-released to the wild.
185	1999	10/21/99	22.0	N/A	Bird was released on 3/24/99. **Captured on 10/21/99 to replace radio transmitters. Bird was re-released to the wild. Disappeared on 11/28/99.

Several of the cases presented in Table 4 are important for the discussion of exposure cause, toxicity, and the development of treatment techniques to save exposed birds. One of the first condors to be reintroduced to the wild in 1992, Condor 64 (Xewe), was tested for blood lead in September 1992, nine months after release to the wild. Blood lead level was reported as 1.6 ppm (equivalent to 160µg/dl), with analysis conducted after the bird was released back to the wild. This bird was recaptured on November 5, 1992, and had no detectable blood lead residues (detection limit 0.1 ppm, or 10µg/dl). The source of lead exposure is unknown. This and other cases clearly indicated the need for a field portable blood lead analyzer. The high level of lead in the September sample, and the low level in November have raised questions as to the validity of the samples (I have been unable to verify either the data or the criticism). The rapid loss of lead from this bird will be discussed in Section I. C. 3., as the depuration from 160µg/dl to background in 44 days is within the range of depuration rates of other condors held in captivity.

Condor 105 was discovered twice with high lead levels in the field. On Sept 3, 1997 the bird was captured with 15 other condors, because they had been in the vicinity of a hunter killed deer carcass near Logan Potrero, San Luis Obispo County, on August 31, and could have fed on that carcass. Condor 105 had a blood level of 157 µg/dl, and was transported to the LA Zoo for chelation therapy. Blood lead level dropped to 89 µg/dl on Sept. 7, and a small metal fragment was noted in the lower GI tract by radiography, which passed through by Sept. 9, and blood levels dropped to 15.2µg/dl. Condor 105 was released to the wild at Lion Canyon on Sept 17, 1997 with a blood lead level of 11.2µg/dl.

In the second incident, Condor 105 was observed to be behaving abnormally on April 30, 1998, and was recaptured. Blood taken on May 1, 1998 had a lead level of 291 µg/dl. Chelation therapy was again initiated, and blood levels dropped to 85 µg/dl on May 4, but crop stasis became evident on May 8 at a blood lead level of 41 µg/dl. Treatment was continued along with feeding by stomach tube, and #105 recovered. After observation at the LA Zoo and Lion Canyon, #105 was released back to the wild on Sept. 18, 1998. Condor 105 disappeared in July 2000, and is presumed dead.

Condor 98 was captured with #105 in Sept. 1997, but was found to have low blood lead levels. #98 was recaptured on August 31, 1998 for routine radio transmitter replacement, and was found to have a blood lead level of 195 µg/dl, without any clinical symptoms. Condor 98 was transported to the LA Zoo for chelation therapy, which was successful, and was returned to the wild at Bittercreek NWR on Sept. 17, 1998.

Condor 111 was captured on August 31, 1998, along with Condor 98. Blood levels measured 186 µg/dl, requiring chelation therapy at LA Zoo. This bird was also released on Sept 17, 1998 at Bittercreek NWR.

In 2002 several exposure incidents have been documented. Condor 192 was captured at Hopper Mountain, Ventura County, on August 21 with a blood lead level of 62 µg/dl. Condor 155 was captured October 1, with a blood lead level of 61 µg/dl, and Condor 108 was captured November 11 with a blood lead of 70µg/dl.

On November 2, 2002, condor #199 was trapped by Ventana Wilderness Society condor biologists and found to have a blood lead level of 80µg/dl. This marks the first high lead level for a Ventana released condor. This event occurred following the coastal (Zone A) deer season and after the Ventana biologists had observed condors feeding on 2 different deer carcasses, one a road killed deer near Atascadero, and a second, believed to have been a hunter-shot carcass.

Condor AC8 (Studbook #12), one of the original adult wild condors trapped in 1986 and held in captivity for 14 years before being reintroduced to the wild, was trapped on November 12, 2002 at Hopper Mountain NWR, after having spent the months of September and October moving back and forth between Hopper Mountain and the Tejon Ranch. She was found to have an elevated blood lead level of >65 µg/dl using a field blood lead test kit, which was confirmed by laboratory analysis as a blood lead level of 101µg/dl. She was transported to the LA Zoo, retested, and her blood lead level rose to 365 µg/dl in two days and peaked at 410 µg/dl as chelation was initiated. Blood lead level dropped to 90 µg/dl by November 20, but radiographs were positive for a lead fragment and small stones in her small intestine. The lead fragment passed through by early December, and her blood lead level dropped significantly. AC8 was released back into the wild on December 23, 2002. The metal fragment recovered from her digestive system has been positively identified as lead, and is currently being analyzed for lead isotope ratios in an attempt to determine the source of the lead.

The recent California lead exposure incidents occurred in the months of August 1997, April 1998, August 1998, and August, October, and November 2002. The actual exposure timing is unknown, as condors may not demonstrate any clinical symptoms for as many as 20 days after exposure. Condors were reported feeding on a deer carcass by hunters at the August 1997 incident, and biologists found a deer carcass, but it is not known whether it was the same animal. The April 1998 incident was not during the deer season, but the season for other game (wild pigs, jackrabbits, ground squirrels, and coyotes) is year around. The August 1998 incident was during the Coastal Zone A deer season. The 2002 Zone D10 deer season in southern Kern County east of Interstate 5 extended from September 28 to October 27, 2002, and the D13 deer season west of Interstate 5 from October 12, 2002 through November 10, 2002. All of these incidents could have been associated with condors feeding on hunter shot carcasses or visceral remains (gut piles). A discussion of hunting and potential risk is included in Section IV.

I. D. Current Lead Monitoring Program for Condors

I. D. 1. Lead Analysis

All three field programs with condors (Hopper, Ventana, and Arizona) are currently monitoring blood lead levels whenever a condor is trapped for any reason. Routine transmitter replacement is scheduled annually, and individual birds may be brought into captivity for other reasons at any time. Several wild-carcass feeding incidents in California and Arizona have also prompted the capture and testing of all the condors in

an area, because of the risk of lead intoxication. Blood samples are drawn, and 50 µl samples are analyzed using a LeadCare® Blood lead analyzer (ESA, Inc. 22 Alpha Road, Chelmsford, MA 01824 (www.esainc.com)). The instrument analyzes lead by the anodic stripping voltammetry principle, by electroplating lead in the sample onto a gold anode, and then removing the lead with a stripping current and integrating the total current released during the stripping. The total current produced in the release phase is proportional to the lead concentration in the sample. The detection limit reported by the manufacturer is 1.4 µg/dl blood (0.014 ppm); range of the instrument is 1.4-65 µg/dl (equivalent to 0.014 - 0.65 ppm); analysis time is 3 minutes. The field instruments are intended to give a quick assessment of the blood lead, and if a bird has been exposed to lead, it is kept in captivity and monitored closely.

Replicate blood samples are taken for laboratory analysis of lead by a Veterinary clinical laboratory, to provide quality assurance and a definitive measure of circulating blood lead. Because the field LeadCare® instrument is temperature sensitive, measurements have not always been as consistent as laboratory analyses, and field measurements have not been able to be performed during cold weather. The speed, portability, and relative accuracy of the instruments, however, make them indispensable for field checks of condors during routine trapping.

Blood samples taken for laboratory analysis are submitted through the Los Angeles Zoo or through contract veterinarians to Antec Laboratories, or to Louisiana State University (LSU) for analysis. LSU uses graphite furnace atomic absorption spectrometry (GFAAS) with Zeeman background correction for lead analysis. Detection limits for GFAAS are generally in the low ppb range. LSU is currently using a Perkin Elmer Aanalyt 800 spectrometer, fitted with a THHGA graphite furnace. LSU is a participant in the Center for Disease Control Blood Lead Laboratory Reference System program for quality control/quality assurance (information provided 2/6/02).

Figure 4 and 5 give correlation data for the LeadCare® instruments the clinical laboratories, for blood samples from the Ventana (Fig 4) and the Hopper (Fig 5) teams.

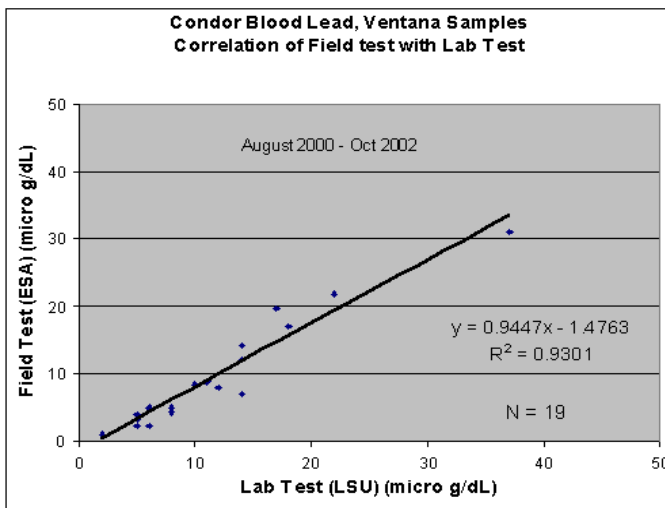


Figure 4. Correlation of LeadCare® analyzer with graphite furnace atomic absorption spectroscopic analysis performed at LSU. Data from Ventana Group.

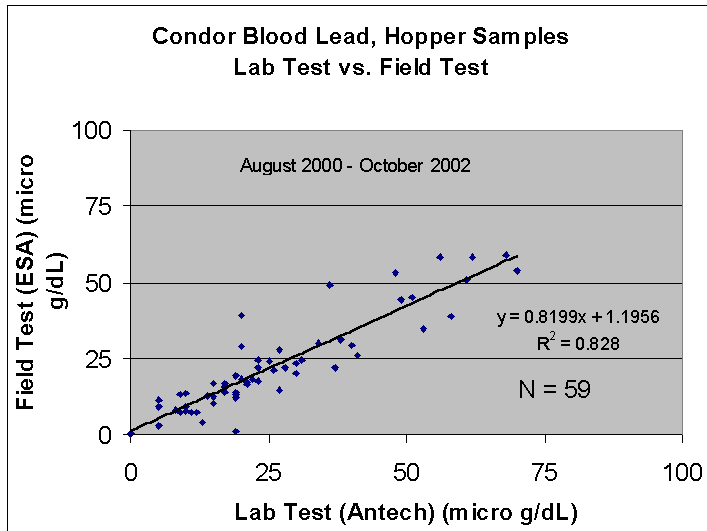


Figure 5. Correlation of LeadCare® analyzer with Antec laboratory analysis.. Data from Hopper Group.

The correlation coefficients show very good consistency and relative concentration measures between the field LeadCare® and laboratory analyses. The Ventana LeadCare® instrument consistently reads about 6% lower than the Louisiana State University analytical Lab, and the Hopper field instrument consistently reads about 16% low, compared to the Antec Lab results. The range of the LeadCare® instruments is much narrower than the analytical labs, with peak values of about 65µg/dl, but this range is extremely useful, as 60µg/dl is the threshold lead exposure level of “clinically affected” golden eagles identified by Pattee et al (1990).

In using the LeadCare® instrument, any sample at or exceeding the range of the instrument should trigger the decision to hold the bird for observation and possible therapy. Chelation therapy requires that the California birds be transported to Los Angeles or San Diego Zoo for veterinary care.

Blood lead values taken in the field are important for clinical determinations, but they cannot provide information as to the duration of lead exposure, nor whether the levels are increasing or decreasing, unless the bird is kept in captivity and retested.

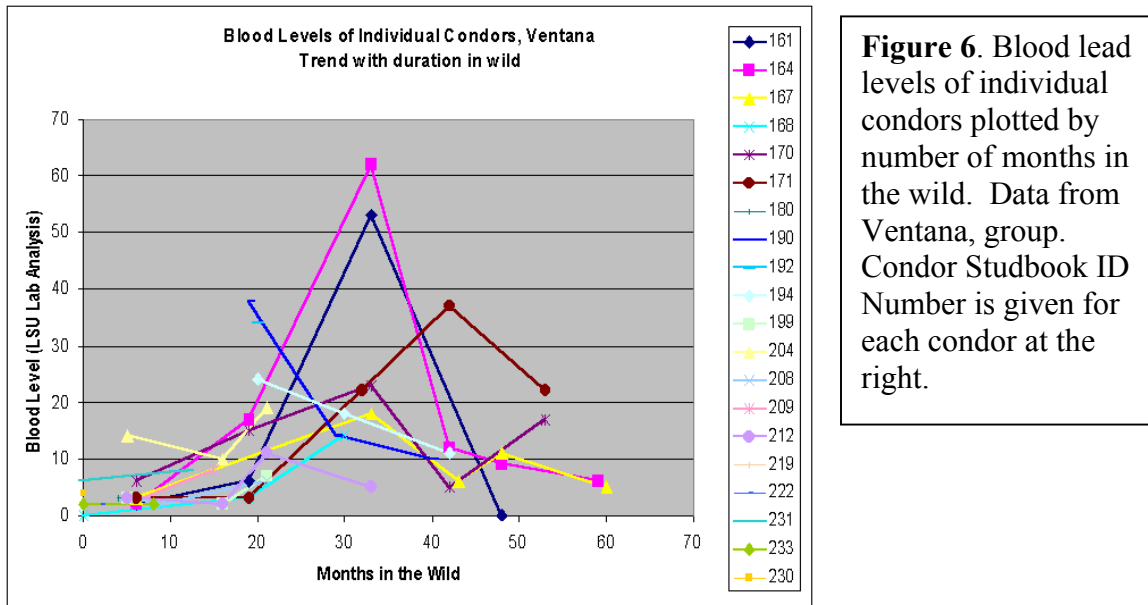
The selection of 60µg/dl (0.6 ppm) as an appropriate threshold level for monitoring and/or chelation therapy is somewhat arbitrary, and somewhat higher than threshold for chelation therapy for lead exposed human children. The US Agency for Toxic Substances and Disease Registry (ATSDR) Level of Concern for lead exposure to children is 10 µg/dl, and the Agency recommends that medical treatment may be needed for children with lead levels exceeding 45 µg/dl (ATSDR Lead toxicological profile, June 1999). Pattee et al. (1990), based on data from Redig (1984) give levels above 60 µg/dl as clinically affected. However, medical intervention is always accompanied by risk, and the dangers of bringing a condor into captivity and subjecting it to chelation therapy may be as great as the risk of low-level lead exposure. The condor program staff appears to be handling the clinical treatment of lead exposure in a very pragmatic fashion, and a review of the current procedures does not indicate any change to current policies or procedures are in order.

I. D. 2. Condor Blood Values from the Field.

In California, all condors in the wild have been tested for lead each year since 1997. Many birds have had multiple samples taken, and this review considers samples taken at least two months apart as independent samples. 87 independent samples have been taken by the Hopper team, and 62% of those blood samples have shown higher than background lead exposure (blood levels greater than 20 µg/dl blood). 13 of 87 samples (15%) have exceeded 60µg/dl, indicating the condors were clinically affected by lead, and 6 birds (7%) have had blood lead levels greater than 150 µg/dl, indicating acute toxic exposure and requiring emergency veterinary intervention.

The lead exposure situation monitored by the Ventana team shows lower exposure to lead than southern California. Only two samples of 60 (3.3%) taken since June 1998 have exceeded 60 µg/dl, and none have been in the acutely toxic range. Nine samples (15%) have been in the exposed range, above 20µg/dl. The data for both teams has been graphed in Figures 6-11.

Blood lead values from the two California field sites have been plotted in three different ways in an attempt to discern patterns in the exposure of condors in the wild. Data from the Ventana group is given in Figure 6, plotting blood lead vs. months in the wild, testing the hypothesis that birds may slowly bioaccumulate lead from environmental sources after they are released to the wild.



Values from each bird are connected to show individual trends. For many birds, there is an increase over baseline during the first 3 years after release, and then a decrease back toward baseline levels. It does not appear that condors retain and bioaccumulate lead over the long-term, as levels have decreased for almost all birds after three years in the wild. It has been hypothesized (R. Riseborough, pers. com., K. Sorenson, pers. com.) that the rise in the second and third year could be a result of young birds becoming more

exploratory, and beginning to forage more widely in search of wild, especially deer carcasses, as they get older and more experienced. The decreases cannot be explained, and may only represent random fluctuations.

The data from the Hopper group is plotted in Figure 7, also giving blood lead values vs. time in the wild. Many of the birds have been in the wild more than 7 years, and there is no overall trend of increasing lead with time. Individual birds with values over 60µg/dl have been treated to lower blood lead levels, and re-released to the wild.

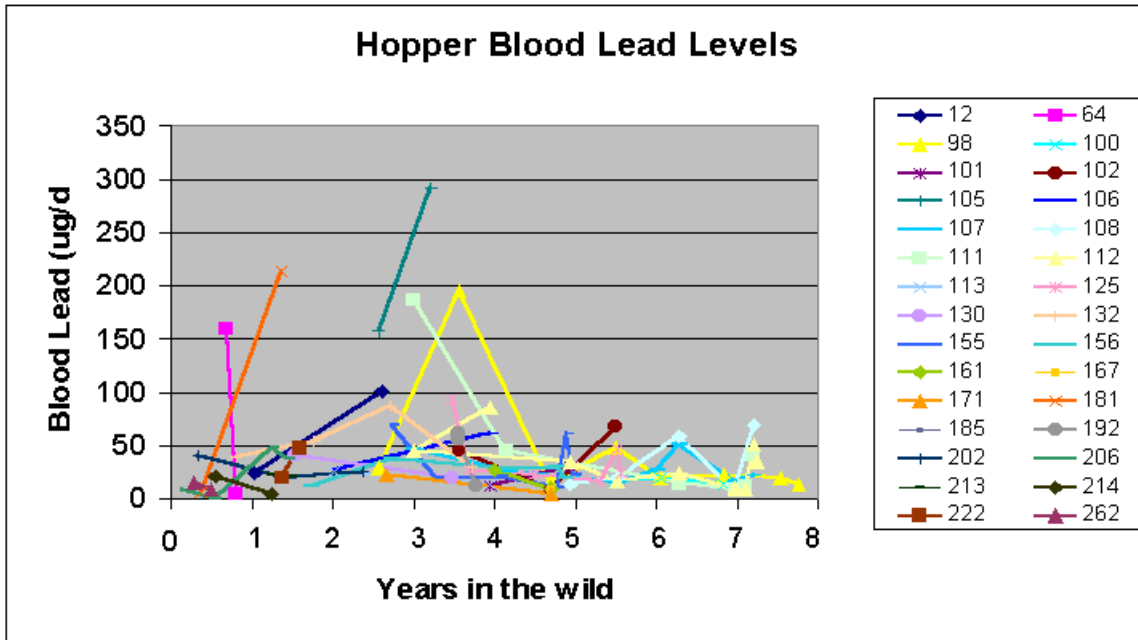
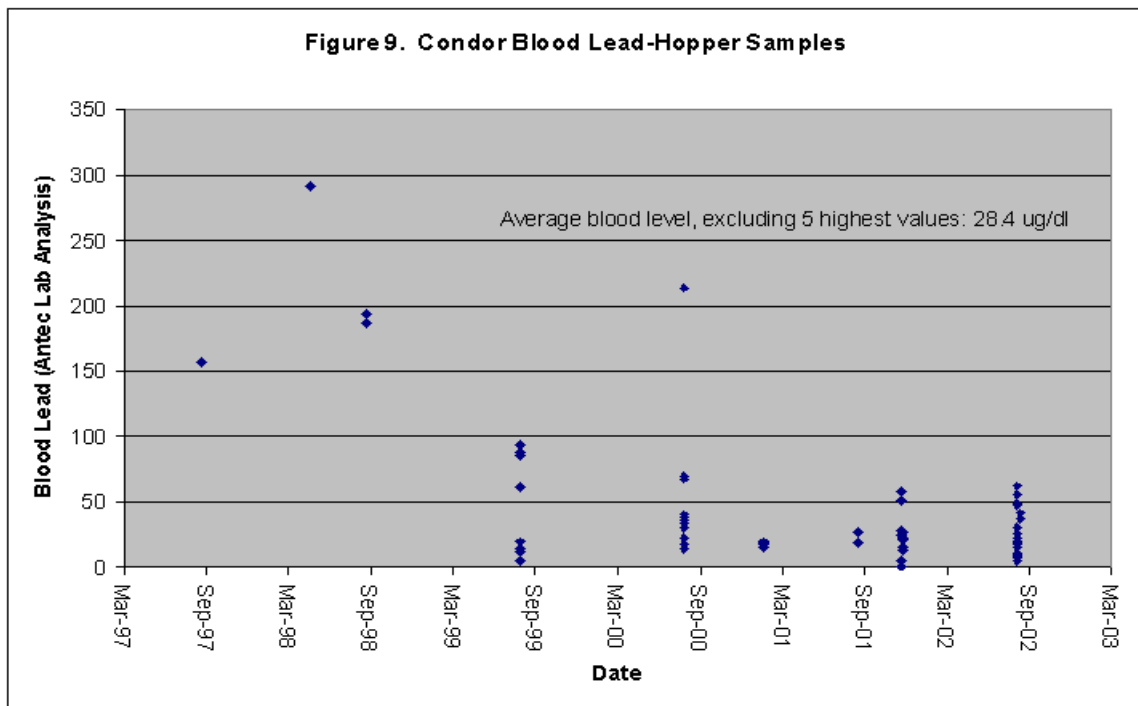
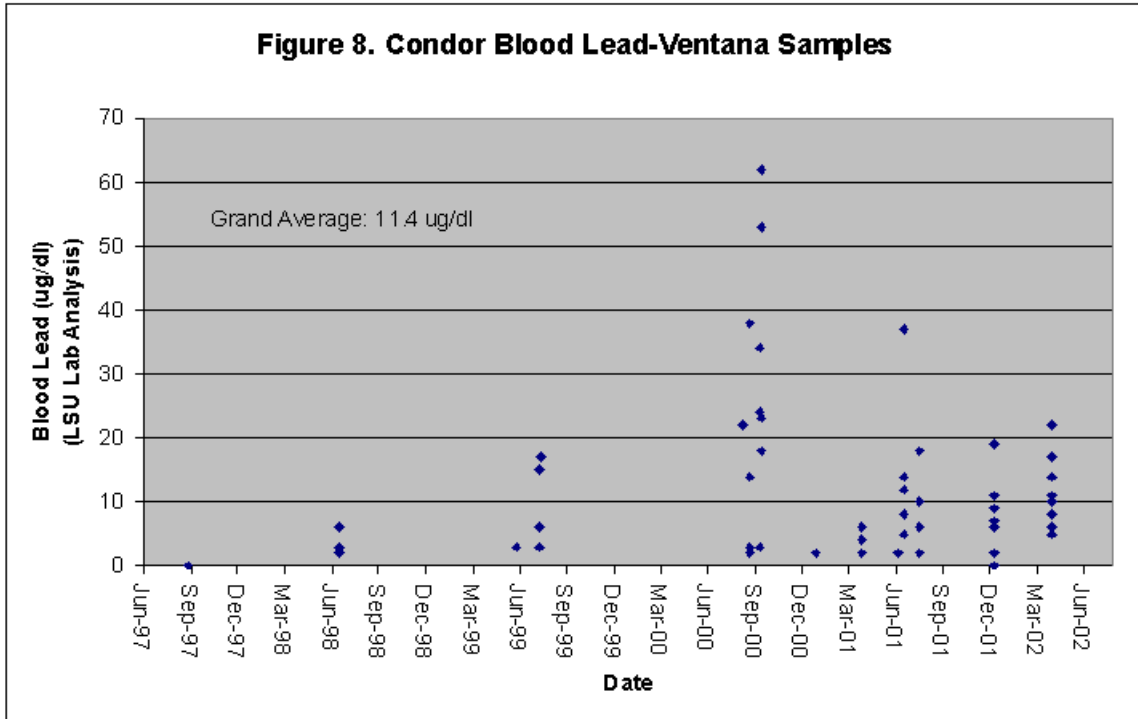
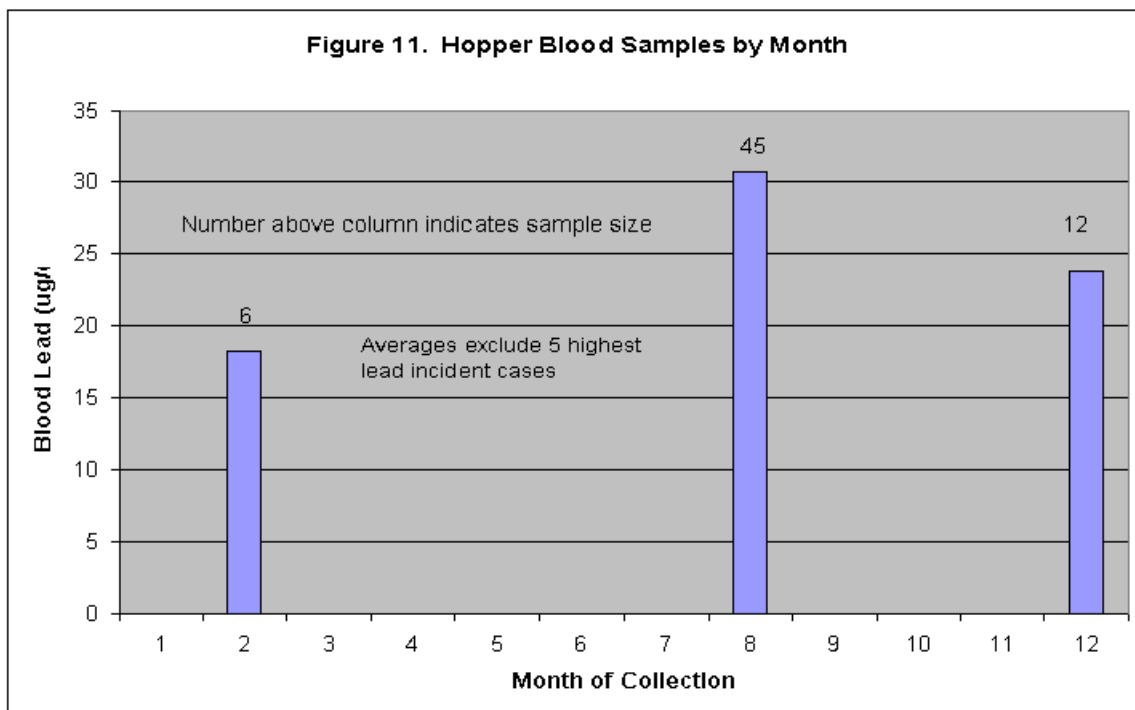
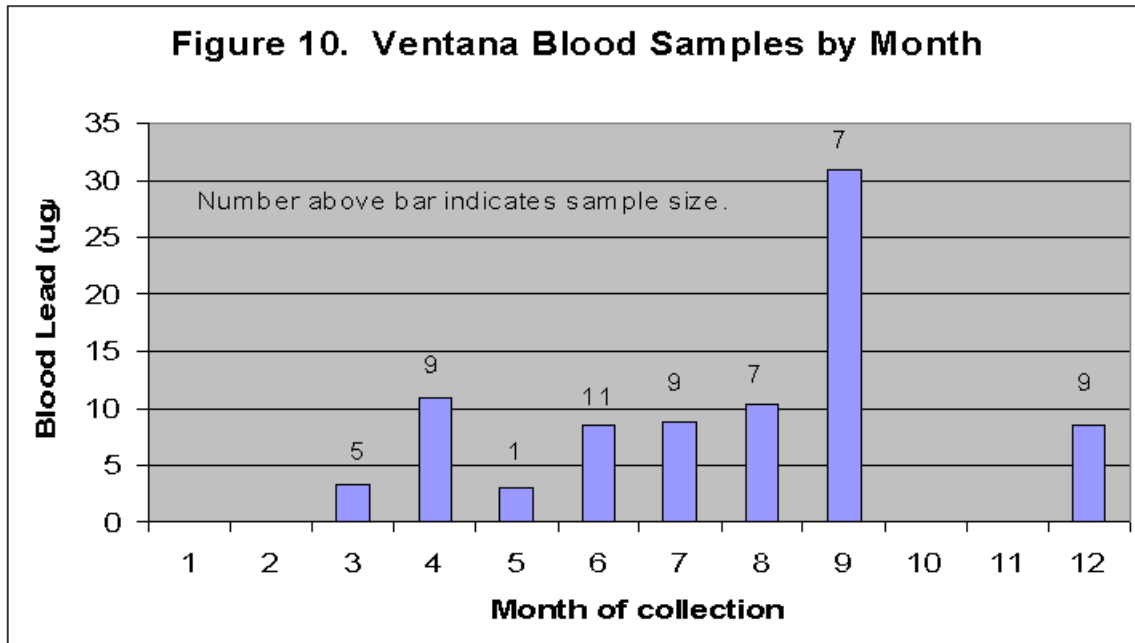


Figure 7. Blood lead levels of individual condors plotted by number of months in the wild. Data from the Hopper group. Studbook ID number is given for each condor at the right.

Blood lead levels have also been plotted against calendar date, to show the overall trend in blood lead over the past four years (1998 to October 2002) (Figures 8, Ventana, and figure 9, Hopper). The Hopper team (Figure 9) has measured lead in five condors above 150 µg/dl (1.5 ppm), indicating highly toxic levels requiring veterinary therapy to reduce the blood lead levels. These samples were taken in the months of April-May and August-Sept. The graphed data do not include the 1992 sample from Xewe (#64) or the most recent exposures of November 2002. Even when the highest lead incident cases are excluded, the samples collected by the Hopper team are significantly higher than the samples collected at Big Sur. The Hopper blood lead levels average 28.4 µg/dl and the Ventana birds average 11.4 µg/dl.



The blood lead samples have been averaged by month, to determine trends by season, and are presented in Figures 10 and 11. Figure 10 gives data from Ventana, with a significant number of samples taken during all seasons. Blood lead values are highest in August and September.

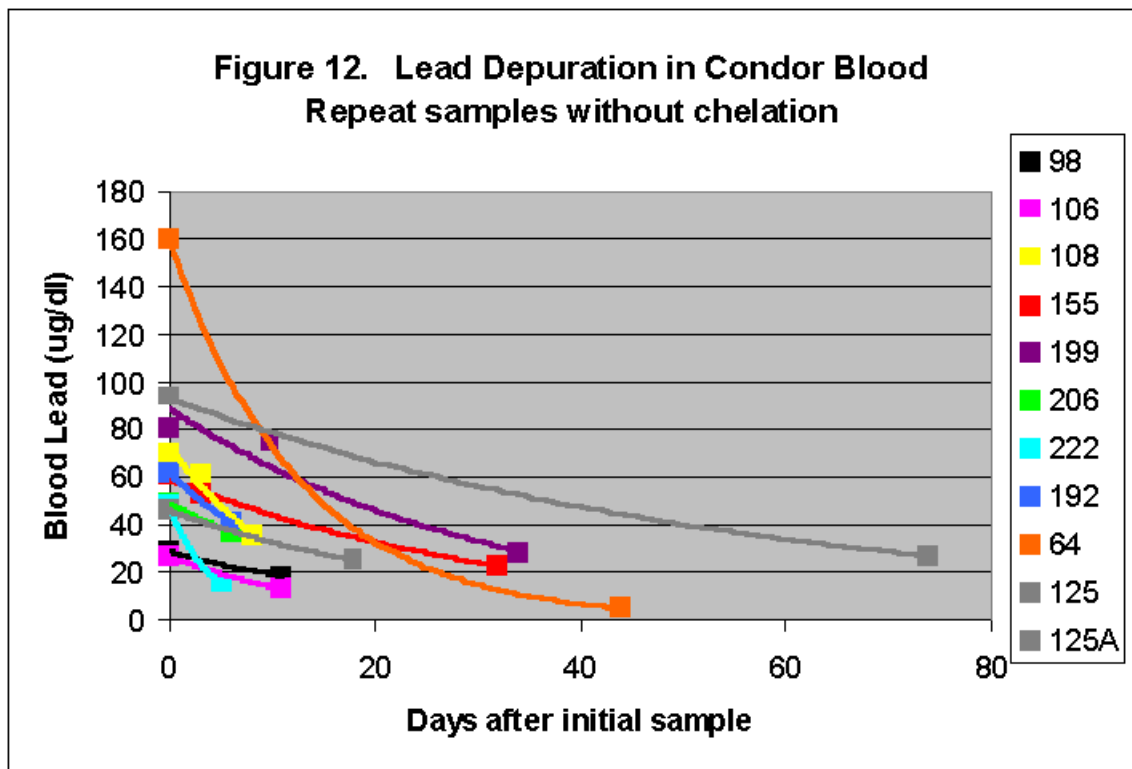


The blood lead values collected from the Ventana birds are higher than background only in the fall, with samples from other months almost always below 20 μ g/dl. This may be an indication of exposure to a specific lead source during the late summer. The majority of Hopper samples have been collected in the fall, making it impossible to determine seasonal trends in values (Figure 11).

Overall, the lead levels in the Ventana birds are close to background levels. The average of 11.4 µg/dl is only slightly above the ATSDR background level for children, and below the background level cited by Pattee et al (1990) for golden eagles in condor range in the 1980s. The Hopper samples average 28.4 µg/dl, and appear to reflect a source of lead exposure in this part of the condor range. The sources of lead are addressed in Section IV.

**I. D. 3. Fluctuations in Blood Lead Levels During Holding of Condors;
Depuration of Blood Lead.**

Occasionally condors have been held in captivity with moderate blood lead levels, or resampled twice within a short period of time, and not subjected to chelation treatments to lower blood lead levels. Two birds have been released and re-trapped after blood sampling, including #64 (Xewe) in 1992 and #125 (captured and released twice). The data have been plotted using the best-fit exponential regressions for lead depuration (Figure 12).



The half-life of lead in the blood was calculated for each bird and averaged for the 8 birds held in captivity (Table 5). Birds released to the wild and recaptured (#64, 125, and 125A) have been excluded from the depuration calculation, because of the long interval between captures and also because of the possibility of re-exposure in the wild. The data shows that blood lead depuration without chelation occurs relatively rapidly, providing there is no metallic lead in the bird's gut.

Table 5. Condor Blood Values for Birds Held in Captivity Without Chelation
(# 64 and 125 released to wild and recaptured)

Stud #	Date	Days after first sample	blood lead ug/dl	T 1/2 (Days) ^A
64	09/22/1992	0	160	9 ^C
	11/05/1992	44	5	
98	09/03/1997	0	28.8	16.2
	09/14/1997	11	18.5	
106	09/03/1997	0	27.2	11
	09/14/1997	11	13.1	
108	11/11/2002	0	70	8.5
	11/14/2002	3	61	
	11/19/2002	8	36	
125	08/08/1999	0	93	42 ^C
	10/21/1999	74	27	
125 ^B	11/02/2002	0	46	20 ^C
	11/20/2002	18	25	
155	10/01/2002	0	61	22.5
	10/04/2002	3	53	
	11/02/2002	33	23	
199	10/27/2002	0	80	20.5
	11/06/2002	10	75	
	11/30/2002	34	28	
206	08/21/2002	0	49	15
	08/27/2002	6	37	
222	11/14/2002	0	48	3
	11/19/2002	5	15	
192	08/21/2002	0	62	10
	08/27/2002	6	41	

T 1/2 Average= 13.34 +/- 2.87 days

A: T 1/2: Calculated half time for depuration, using exponential regression best fit, +/-SEM.

B: #125 was discovered twice with high blood lead.

C: T 1/2 Calculation excluded birds released and recaptured.

The data in Table 5 and Figure 12 are plotted with the blood level at initial capture, and the blood levels after the given number of days in captivity. All birds show a decrease in blood lead at every sampling, indicating lead is depurated from the condor's blood without chelation, if the birds are held in a clean environment. The series of three blood samples taken from Condor 199 indicate an initial slow loss of lead, followed by a more rapid loss. This could be due to undetected lead in the gut, but the data are included to generate the best fit exponential depuration curve. The depuration rate data for Condor 125 are uncertain, because of the long time interval between initial sampling and recapture. The calculated $T_{1/2}$ for #125 was 42 days on the first occasion, and 20 days on the second. Both could be overestimates of the depuration rate, because of the long interval between samplings. Clearly more data need to be collected to be able to analyze the lead depuration process, but it is very encouraging that all ten birds showed significant drops in blood lead without chelation.

The rates of depuration vary somewhat between birds, with an average $T_{1/2}$ (half time for exponential loss of lead from blood) of 13.34 ± 2.87 days. The range of rates varied between the rapid $T_{1/2}$ of 3 days (#222) to 22.5 days for Condor 155. Many factors could influence the depuration rate, including: prior exposure; lead deposition in tissues and bone; individual bird differences; or residual undetected lead fragments in the gut. Lead is expected to decrease slowly through fecal or urinary excretion (see section on physiology, Section II. A.), and the rate of excretion for an acute exposure is thought follow an exponential decay, proportional to the circulating blood level (Eisler 1988), ATSDR (1999).

The observed $T_{1/2}$ equal to 13 days demonstrates that the blood lead level of a condor would be expected to return to baseline relatively quickly. For example, a blood level of $100 \mu\text{g}/\text{dl}$ would drop to background ($20 \mu\text{g}/\text{dl}$) in 30 days, and a blood level of $60 \mu\text{g}/\text{dl}$ would drop to background in 21 days. Two very important conclusions can be made from the non-chelation depuration data:

- 1) Lead in blood does not appear to bioaccumulate slowly through time as condors remain in the wild, because blood levels drop quickly when exposure is terminated.
- 2) Exposure to lead must occur frequently to condors in the wild in California.

Since blood lead levels drop without chelation over the span of days or weeks, condors must be exposed frequently to lead in the wild. Random blood samples taken throughout the year indicate that more than 40% of condors in California have been recently exposed to lead. In southern California the percentage is higher, with 62% of blood samples above background. 6 birds randomly tested in southern California have had blood lead values in the "acute toxic" range (greater than $100 \mu\text{g}/\text{dl}$), which must represent very recent or continuing exposure.

The frequent lead exposure to condors must be regarded as a serious situation for the wild population. It is also important to stress that condors are not the only wildlife exposed to lead. Other raptors in the condor range have been documented with similar rates of

exposure, (Weimeyer et al (1983), Weimeyer et al (1986), and Pattee et al. (1990)). Lead exposure will also be injurious to those protected species.

I. E. Causes of Death of 35 Condors Examined by Pathologists Since 1983:

Necropsy reports were obtained from the San Diego Zoo for 35 of the 54 condors condors dying in the field between 1983 and June 2001. Table 6 provides a summary of all known deaths, and the data provided in the necropsy reports. The ID of the bird and Studbook number are given, as are the presumptive diagnosis and the final diagnosis of cause of death. The lead analysis results are included for those samples submitted. If tissues were not submitted for analysis they have been marked "NS". The weight of the bird at necropsy is included, as are the initials of the reporting pathologist. A second page of notes is included, giving other details of the death pertinent to the report.

Color coding has been used for birds dying from lead poisoning (yellow) and those of unknown cause of death, or birds that disappeared (blue). A summary of the causes of death includes: Eight deaths have been attributed to lead poisoning, 7 from electrocution or collisions, 3 birds were diagnosed as dying from emaciation or malnutrition, 8 from predation by eagle or coyote, 2 from drowning, 2 from gunshot (plus two additional birds from Arizona that are under investigation), 1 from cyanide, 1 from cancer, and one from a mixture of ethylene and propylene glycol, perhaps from discarded antifreeze solution left in the wild. One bird died from aspiration of ingesta (vomit) into the airways (ultimate cause unknown), and 10 others died of unknown causes. Eleven condors disappeared.

Several of the categories of death could have other contributing factors, including the sub-lethal effects of lead exposure, or completely unrelated factors. When multiple possibilities exist, it would be important to have lead residue analysis to rule out possible lead exposure as a cause. For example, the collision deaths could be simple accidental deaths, or could involve neural degeneration caused by lead, even though lead was not diagnosed at the time of death. Emaciation, malnutrition or rapid weight loss could be related to other illness, lack of experience of birds in the wild, or could be related to sub-lethal exposure to lead, as impaired gut mobility and anorexia are frequent symptoms of lead intoxication.

Unknown causes of death included birds without any outward signs of injury, and without histological evidence of cause of death. Some birds were heavily scavenged, and the carcasses recovered from the wild were without internal organs. No toxicology was performed on these birds, because no liver or kidney was available for residue analysis. Of 35 condors examined, lead residue analysis has been conducted on 18; 6 birds had high liver lead levels, 12 had liver levels of 1 ppm or lower. 17 birds were not analyzed. Feather analysis for lead should be performed on all of these carcasses, and would provide additional data. For the birds with no residue data, feather analysis could provide definitive data either implicating or eliminating lead as contributing.

Table 6. Condor Mortality Summary, January 1992-October 2002
 Compiled from Scott and Jurek (1985), Snyder and Snyder (1989), Sorenson et al. (2001),
 Condor Recovery Team Lead Resolution paper (2001), and recent data.

date	Nec#	stud #	sex	location at death	proximate cause of death	ultimate cause of death
11/23/83	1	"Don Victor"		CA	cyanide poisoning	M44
03/22/84	2	C1	m	CA	lead poisoning	ingestion of lead bullet
04/10/85	3	"Broken Feather"	m	CA	lead poisoning	
01/18/86	4	12(AC3)	f	CA	lead poisoning; gunshot	shot; ingestion of lead
10/07/92	5	66	m	CA	ethylene/propylene glycol poisoning	ingestion of glycols
05/28/93	6	75	m	CA	electrocution	power line electrocution
06/12/93	7	81	f	CA	trauma; collision	car or power line collision
10/30/93	8	78	m	CA	trauma; collision	power pole collision
06/24/94	9	89	m	CA	trauma; collision	car or power pole collision
07/29/94	10	87	m	CA	cancer	
08/16/96		118	m	CA	disappeared	disappeared
10/10/96		109	f	CA	disappeared	disappeared
01/10/97	11	142	m	AZ	trauma; predation	golden eagle predation
02/02/97	12	131	m	CA	emaciation	unknown
03/09/97		103	f	CA	disappeared	disappeared
05/18/97	13	151	f	AZ	trauma; collision	power line collision
07/14/97		128	f	AZ	disappeared	disappeared
08/29/97		129	f	CA	trauma; collision	power line collision
03/10/98	14	169	m	AZ	trauma, predation?	possible coyote
07/17/98	15	152	f	near Lion Canyon	drowning	drowning
07/17/98	16	143	f	near Lion Canyon	drowning	drowning
08/09/98	17	153	m	near Lion Canyon	pneumonia; gunshot	shot
12/24/98		177	m	AZ	trauma; predation	coyote predation
03/10/99	18	124	f	AZ	gunshot	shot
06/30/99	19	178	m	near Lion Canyon	unknown; emaciation	unknown
10/01/99		130	f	CA	disappeared	disappeared
11/15/99	20	175	m	CA	trauma; predation?	possible golden eagle

date	Nec#	stud #	sex	location at death	proximate cause of death	ultimate cause of death
11/28/99		185	m	CA	disappeared	disappeared
12/22/99		188	m	Lion Canyon	unknown	unknown
12/29/99		113	f	CA	disappeared	disappeared
01/15/00	21	207	m	AZ	unknown; aspiration	unknown; aspiration of ingesta
02/19/00		106	f	CA	disappeared	disappeared
03/02/00		116	m	AZ	lead poisoning	lead poisoning
04/02/00	22	197	f	AZ	trauma, predation?	possible golden eagle
04/20/00		211	f	AZ	disappeared	disappeared
06/12/00	23	165	m	AZ	lead poisoning	ingestion of lead shot
06/16/00	24	191	f	AZ	lead poisoning	
06/20/00	25	182	f	AZ	unknown	unknown
06/23/00		150	f	AZ	unknown	unknown
09/09/00	26	184	f	AZ	trauma; unknown	unknown; possible eagle predation
09/24/00	27	102	m	near Lion Canyon	unknown	unknown
10/01/00	28	181	f	near Lion Canyon	unknown	unknown
10/06/00	29	101	f	CA	unknown	unknown
12/25/00	30	82	f	CA	predation	coyote
12/29/00	31	74	m	AZ	predation	coyote
01/31/01	32	132	f	Castle Crags, SLO	lead poisoning	lead ingestion?
02/09/01	33	228	f	AZ	emaciation	unknown
05/09/01	34	230	m	Big Sur	unknown	power line electrocution or collision
06/27/01	35	215	m	CA	electrocution burn	power line electrocution or collision
07/07/01		105	m	CA	disappeared	disappeared
02/22/02		252	?	AZ	trauma; predation	predation
09/15/02		100	m	Hopper	disappeared	disappeared
late Aug 02		186	m	northern AZ	unknown	unknown
mid Aug 02		240	?	southern UT	lead poisoning	lead ingestion?
10/04/02		108/100 chick				
10/13/02		155/98 chick				
10/21/02		107/112 chick				

stud #	prior blood lead (ppm)	evidence
"Don Victor" C1		tracerite fluorescence from M44 bullet fragment in gizzard; liver Pb 35 ppm; kidney 47 ppm; blood clot heart 24 ppm
"Broken Feather" AC3	0.27 10/2/84 ^a	liver Pb 23 ppm shotgun pellets in tissue; blood lead 4.2 ppm
66		kidney ethylene glycol 502 ppm
75		observed flying into high voltage wires
81		found on roadside with femur fracture, acetabulum. wire or car collision
78		found next to power pole. wing fracture? gizzard with glass, rubber pieces
89		fractured acetabulum, pica. car or power pole collision
87		tumor; secondary aspergillosis
118		disappeared
109	no sample	disappeared
142		perforated skull, extensive hemorrhage, brain trauma
131		weak, dehydrated, malnourished
103	no sample	disappeared
151		found 1 mile from power line; transmitter under power line; legs broken, brain hemorrhage
128		
129		
169		found scavenged significantly
152	0.26 9/14/97	found w/143 in steep-sided pool
143		found w/152 in steep-sided pool
153		found alive; rt. leg broken, compound fracture, from gunshot
177		
124		multiple gunshot wounds; bullet in rt. wing
178	no sample 6/15/99	died 1 minute after retrieval; rapid weight loss; heart murmur; no lead in x-rays; Pb liver and kidney < 1ppm; Cu 75 ppm liver; Cu 77 ppm kidney; released 3/24/99; sparse feeding record
130	0.2 8/9/99	disappeared; last visual 10/1/99
175		

stud #	prior blood lead (ppm)	evidence
185	0.22 10/21/99	disappeared
188	no sample	scavenged; bones chewed upon; bear and coyote tracks nearby
113	0.756 9/9/97	disappeared;
207		found below roost ledge; vomit on roost ledge
106	0.614 8/9/99	disappeared; last visual 2/18/00
116	3.2 ppm 3/2/00	
197		
211		
165		12-17 lead shot in gizzard; liver Pb 34 ppm; kidney Pb 96 ppm
191		found alive; liver Pb 17 pm
182		found scavenged, close to road; lead?
150		
184		broken ribs, neck?, possibly postmortem. Lead?
102	0.68 8/9/00	last visual 9/9/00; found scavenged, plucked feathers; skeletonized; lead chelation therapy 8/17/00; last flight signal 9/21/00; found scavenged, decayed, maggots; bear tracks and scat at carcass
181	2.14 8/9/00	
101		found scavenged; Pb < 1 ppm; Cu 120 ppm
82		transferred from AZ 12/7/00; Pb liver < 1 ppm; Cu 21.6 ppm
74		many coyote tracks near carcass; Pb liver < 1 ppm; Cu liver 21.6 ppm
132	0.882 8/9/99; 0.310 8/9/00	fell to ground in flight; observed flying (flushed)15 min. prior to death; Pb liver 26.4 ppm
228		33% weight loss; poor body condition; Pb liver 1 ppm; Cu liver 85.6 ppm
230		found beneath power lines; Pb liver < 1 ppm; Cu liver 113 ppm
215		found beneath power lines; observed flying 1 hr earlier; Pb liver < 1 ppm; Cu liver 41.2 ppm
105	2.91 5/1/98; 0.17 8/6/98	disappeared; blood Pb 1.57 9/3/97
252		
100		disappeared; last seen 15 Sep - flew to Sierra madre ridge; brooding young
186		found dead
240		Necropsy reports lead bullet fragments in the gut.
108/100 chick		found dead below nest cave 1-2 days after fledging; no lead in liver; father disappeared two weeks earlier
155/98 chick		found dead in nest cave; radiograph: 12 ingested bottlecaps, pieces of glass, plastic, and metal (e.g., washers, electrical connectors)
107/112 chick		found dead in nest cave;

I. F. Historic Lead In Other Birds within Condor Range.

During the early period of the Condor Recovery Program, several studies were conducted to determine the extent of lead in the condor range, by measuring lead in food items, condor feathers, and in golden eagles, ravens and turkey vultures throughout the range. Weimeyer et al (1983), Weimeyer et al (1986), and Pattee et al. (1990), all present data on biological tissues collected in California. The levels in eagles, ravens and vultures are similar to the levels found in condors at capture (Table 5) or necropsy (Tables 7,), reflecting wide spread bio-available lead in the condor range. Whole condor feathers contained up to 14 ppm lead, reflecting exposure during the period of feather growth (Weimeyer et al 1986). Pattee et al. (1990) found 36% (58 of 162 samples) of golden eagles trapped within the condor range had blood lead above 20µg/dl, the “exposed” threshold above background. 9 samples (4%) were greater than 60µg/dl, and 4 of 162 samples were greater than 100µg/dl, indicating acute toxicity. Analysis of the data by month demonstrated the highest exposure to be in the months of September, November, and December, during and after the deer hunting season, and the lowest exposures in June, July and August, before the deer-hunting season. Pattee et al (1990) concluded that exposure to hunter shot carcasses was the most probable source of lead exposure to golden eagles.

In the study by Weimeyer et al 1986, deer, cattle and sheep from the condor range were collected for lead analysis, as were turkey vultures and ravens, and eggs from these species. Weimeyer et al concluded “ biologically incorporated lead, based on concentrations in these potential food items, was very low in relation to levels shown to cause a variety of adverse physiological effects...in captive falconiforms”. They also predicted, “The impact on condors of lead exposure in the form of lead fragments or pellets may be much greater than from biologically incorporated lead”.

The routine blood sample data for California Condors indicate that 43% (63 of 147 blood samples) were exposed, 10% (15/147) were clinically affected, and 4% (6/147) of condors tested were in acute toxic exposure episodes at the time of blood sampling. These data are remarkably similar to the data of Pattee et al. for golden eagle blood samples taken in 1985-86. Condors are being exposed to lead at rates at least as great as other raptors in their range in spite of a feeding program to discourage condors from feeding on wild found carcasses. Because leaded gasoline has been phased out since the Patee et al. work was completed, background levels due to atmospheric lead should also have been reduced. While the lead exposure to condors is of particular importance to the Condor Recovery Program, the potential poisoning of other species of birds and scavenging mammals should also be of concern to the Agencies responsible for protection and management of wildlife. Golden eagles, ravens and turkey vultures are all protected species.

Potential sources of environmental lead and estimates of the possible contribution of each will be addressed in Section IV.

Section II: Physiology and toxicity of Lead and Copper to Condors

II. A. Physiology of lead intoxication

Eisler (1988) summarizes the physiological and clinical aspects of lead as:

“Lead modifies the function and structure of kidney, bone, the central nervous system, and the hematopoietic system and produces adverse biochemical, histopathological, neuropsychological, fetotoxic, teratogenic, and reproductive effects. Inorganic Pb absorbed into the body enters the bloodstream initially and attaches to the red blood cell. There is a further rapid distribution of the Pb between blood, extracellular fluid, and other storage sites that is so rapid that only about half the freshly absorbed Pb remains in the blood after a few minutes. The storage sites for Pb are uncertain, although they are probably in soft tissues as well as bone; the half-time residence life (T 1/2) of inorganic Pb is estimated to be 20 days in blood, 28 days in whole body, and 600 to 3,000 days in bone. Inorganic Pb in the environment can be biologically methylated to produce alkyllead compounds. Bile is an important route of excretion; ingested Pb probably proceeds sequentially from gut, to blood, to bone and soft tissue, and by way of the bile to small intestine and fecal excretion”.

Ingested lead is both an inhibitor of blood formation and a neurotoxin, competing with calcium in neurotransmission, and additionally causing degeneration of the myelin sheaths of peripheral nerves. The toxicity profile provided by the Agency for Toxic Substances and Disease Registry (ATSDR) is an excellent review of the physiological and clinical effects of lead on humans and domestic animals. The lead profile can be found at <http://www.atsdr.cdc.gov/toxprofiles/tp13.html>

Lead accumulates in bone, feathers, hair and other tissues. Lead binds to sulfur groups in proteins, and the number of available sulfur groups affects the amount of lead that can be bound to a protein. Metallothioneine, a metal binding protein localized in liver and kidney binds lead, but the kinetics of lead binding, and the binding to other metals has not been well studied in birds.

The lead depuration from blood and from tissues each follow an exponential loss rate, but with very different time scales. Kinetics have been studied for humans and laboratory animals, but not systematically for birds. The half-life of lead in adult human blood was measured at 36 days by Rabinowitz et al. (1976), 28 days by Griffin et al. (1975b), and 19 days by Chamberlain et al. (1978). In rats, excretion of lead was biphasic following intravenous administration, with half-lives of 21 hours for a fast phase and 280 hours (11.67 days) for the slow phase (Morgan et al. 1977). Dogs excreted lead in three phases, with half-lives of 12 days (blood), 184 days (soft tissues), and 4,951 days for bone (Lloyd et al. 1975). In the limited study by Riser and Temple given in Section II. B., below, and Figure 13, the T ½ for a single turkey vulture was 7 days, and for two red-tailed hawks was 14 days, after 6 week exposures.

Blood, liver, kidney, bone and feathers have all been used as tissues for reference levels of lead in birds, including condors. The levels of lead in each tissue vary greatly, and the half-time residence life ($T_{1/2}$) in each tissue is different as cited above. Careful comparisons of tissue residues within a single individual are necessary to accurately determine the relationships between levels in different tissues, but time course of loss of lead from each tissue makes simple comparisons extremely difficult. For example, if lead has a $T_{1/2}$ of 14 days in blood and 600 days in bone, a short-term acute exposure to lead may show a significant spike in blood levels, but only a small fraction of the bone will pick up any lead. Conversely, chronic low-level lead exposure may result in substantial accumulation in bone with only low concentrations in blood. The data for condors "Broken Feather" and "Tehachapi" (Table 2, Table 7) are good examples of the lack of correlation in lead levels between tissues. "Broken Feather" liver lead was 23 ppm, and bone was 72 ppm, while "Tehachapi" liver level was 1.6 ppm, and bone was 79 ppm. The liver residues of 1.6 ppm would be considered only marginally elevated, but the 79 ppm in the bone indicates significant long-term lead exposure.

II. B. Experimental Studies of Lead Toxicity to Raptors.

Pattee, Carpenter and Fritts at Patuxent Wildlife Research Center, Maryland, (unpublished study) fed lead shot to Andean Condors over the course of several weeks, to determine the sensitivity and clinical signs of toxicity. Two Andean Condors were fed 2 #4 lead shot pellets each (approximately 418 mg/8kg body wt.) and maintained in captivity for 50 days (Table 7). One bird died at 43 days, and the other survived and was euthanized with clinical symptoms. The blood lead levels rose to 10 ppm within two weeks, and remained at about 10 ppm for the duration of the study. Liver lead residues of these birds were 45.4 and 49.8 ppm respectively. Two additional condors were fed 6 #4 shot (approx 627 mg); one bird died at 36 days, and the second was euthanized at 42 days with clinical symptoms. Blood lead rose to 15 ppm within 2 weeks, and stayed above 12 ppm. Liver lead residues were 58.5 and 109.1 ppm respectively.

Turkey vultures were also fed lead for comparison with the Andean Condor study at Patuxent. Three birds each were fed 3 or 10 #4 lead shot pellets (approximately 627 mg and 2.09 g/~1.5kg body wt). 2 of 3 turkey vultures fed three shot pellets survived 211 days and were euthanized; one bird was euthanized with clinical symptoms after 170 days. Liver lead of the two surviving birds was 2.2 and 1.5 ppm, while the residue level in the euthanized bird was 18.7 ppm. The turkey vultures fed 10 shot all developed clinical symptoms, with two birds dying at 143 and 148 days, and the third being euthanized at 182 days. Blood lead levels were 2-10 ppm throughout the study. Liver lead residues were 33.8, 20.7, and 6.8 ppm respectively.

Turkey Vultures survived much greater doses of lead than did condors (approximately 8-26 times the dose on a body weight basis), and accumulated much less lead in liver tissue. Based on this data, scaling between these closely related species does not produce meaningful data, and predicting the response of condors from turkey vulture studies also appears to be impossible.

Pattee and co-workers at Patuxent also studied the effects of lead on Bald Eagles to determine the time course, blood lead levels, and clinical symptomology of exposure and mortality (Pattee et al, 1981, Hoffman et al. 1981). Bald Eagles were exposed by feeding lead shotgun pellets, and monitoring gut passage, regurgitation, quantitative erosion of the pellets, clinical signs, and blood chemistries of the birds. Five Bald Eagles were fed 10 #4 lead shot (209mg/pellet, giving a dose of 2.09g). Most birds regurgitated pellets within 1-7 days, and were re-dosed with 10 additional pellets to maintain lead in the stomach. Regurgitated or passed pellets were weighed to determine the amount of lead eroded or dissolved from the pellet. The total lead eroded from the pellets was reported, along with the clinical outcome. One eagle died at 10 days, after mobilizing 19.4mg lead from the pellets; one died at 12 days after mobilizing 42.3 mg, and a third died in 20 days, mobilizing 37.8mg. The remaining two eagles demonstrated very different effects. One lived 125 days, after dissolving 184.9 mg lead, and the other went blind after 133 days and was euthanized. That bird mobilized 129.0 mg lead. Liver residues were measured at death, as were residues in several bones. Data are given in Table 8.

The quantitative data on lead erosion indicated that only 19-42 mg of lead were dissolved before three eagles died. At a body weight of 5 kg (eagle body wt range is 3.6-6.4 kg, and body weights were not given in the paper), 19.4 - 42 mg represent only 3.88 - 8.4 ppm respectively, if distributed evenly throughout the body. Blood lead levels were not reported for individual birds, or for birds with clinical symptoms, but blood levels at 7 days were 3.4 ± 1.4 ppm (standard error of the mean, SEM), and at 14 days (3 surviving birds) were 5.4 ppm (± 4.3 SEM). Clinical symptoms of lethargy and anorexia appeared in the birds at 7, 7, 8, 93, and 121 days, respectively.

Two very important facts are evident in this small dataset: 1) as little as 4ppm can be fatal to an eagle, and there appeared to be little excretory loss of dissolved lead from the bodies of these three eagles over the 10-20 days of exposure; and 2) other 2 eagles eroded, and presumably assimilated, 189 and 129 mg of lead, and lived for 125 to 133+ days. These birds must have excreted most of the lead during the exposure, or the liver lead levels would have been much higher at death. The data show a high degree of individual variation between birds of the same species, which must be a result of large differences in the absorption and/or excretion of lead.

Riser and Temple (1981) conducted a lead feeding trial with raptors (red-tailed hawks, rough-legged hawks, Swainson's hawks, a golden eagle and a turkey vulture), feeding measured doses of lead acetate, rather than lead shot. In one experiment, one turkey vulture and two red-tailed hawks were fed 3 mg lead/kg body wt. daily as lead acetate, and blood lead levels were measured weekly. Birds were fed lead for 6 weeks, and maintained an additional six weeks to assess depuration kinetics (Figure 13). Blood lead in the turkey vulture rose to 10.93 ppm, while the red-tailed hawk blood lead reached a plateau of about 2 ppm. The hawks showed no clinical signs throughout the study, but the turkey vulture became anorexic and depressed after 4 weeks. Following termination of dosing, the blood lead levels of the turkey vulture decreased with a $T_{1/2}$ of about 7 days, and the blood levels in the Red-tailed Hawks declined with a $T_{1/2}$ of about 14 days. Liver and bone residues of the turkey vulture were not measured.

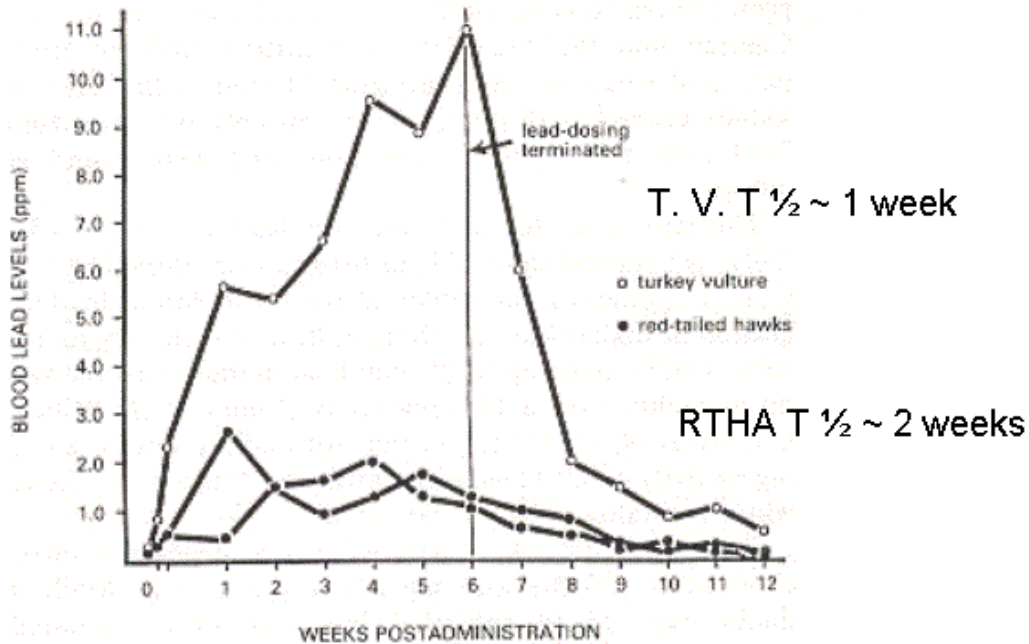


Figure 13. Comparison of lead concentrations (ppm) in blood of turkey vulture and red-tailed hawk. From: Reiser, M. H. and S. A. Temple. 1981. Effects of Chronic Lead Ingestion on Birds of Prey. *In*, J. E. Cooper and A. G. Greenwood, Recent Advances in the Study of Raptor Diseases. Chiron Publications, England. p. 23.

The experimental evidence gathered by Pattee and co-workers, and Riser and Temple (1981), with Andean Condors, Bald Eagles, hawks, and Turkey Vultures indicates large differences between individual birds of the same species, and large differences between species in sensitivity to lead and kinetics of lead excretion. From this data, it is not surprising that individual California Condors discussed above have shown different clinical scenarios with regard to survival time after exposure. “Broken Feather” and AC3 survived more than 40 days in the field with presumably high lead levels, but death may have occurred in perhaps a week or two for some of the birds exposed in Arizona.

Condors appear to have about average sensitivity to lead, based on studies with other raptors and eagles. Turkey Vultures have much greater resistance to ingestion of metallic lead, at least when measured at the gross clinical endpoints of morbidity and mortality. Turkey vultures appear to build up higher blood levels than equivalently dosed red-tailed hawks when dosed with lead acetate, but also appeared to survive much higher doses of lead than Andean Condors. Clinical symptoms were evident in one turkey vulture in the Riser and Temple study at about 9.5 ppm blood lead, about double the level for clinical symptoms in Bald Eagles and condors.

More subtle effects than depression and anorexia could be manifested in lead exposed condors, including impaired coordination and flying ability. None of the experimental studies evaluated flight, coordination, or behavior, other than anorexia and depression. To what extent any impaired flight capability could compromise a condor is unknown,

although the birds are very large, and not very maneuverable, especially when interacting with eagles or potential obstacles such as overhead power lines or towers with guy wires. The experimental studies indicate that gross clinical symptoms could be expected in a condor after 3-30 days exposure to lead shot or bullet fragments in the gizzard. Death in sensitive birds might occur in 5-20 days, although Andean Condors lived 36-50 days with 400-600 mg lead in their gizzards.

II. C. Quantity of Ingested Lead Causing Toxicity in Condors.

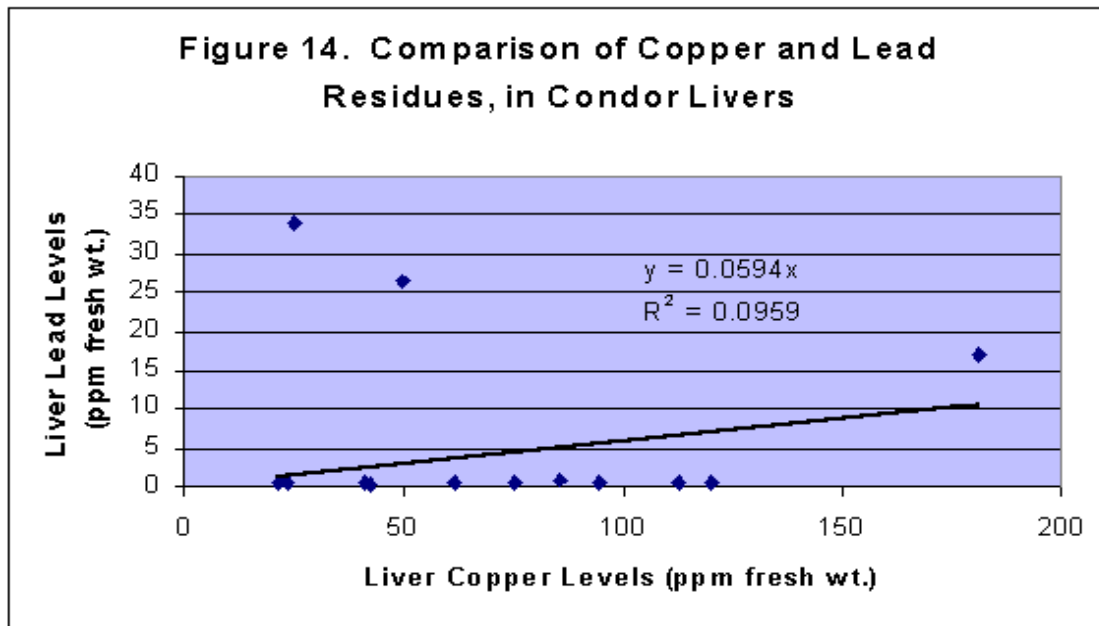
Bald Eagles exposed at Patuxent by Pattee et al. were killed after absorption of as little as 19-42 mg lead. The body weight of a condor is about 150% of a Bald Eagle, and direct extrapolation would indicate lethal lead concentrations could be achieved with as little as 30-65 mg lead. A single #4 lead shotgun pellet weighs about 209 mg, and a single #6 lead shotgun pellet weighs about 127 mg. Ingestion of either of these could, if dissolved by stomach acid, result in lead levels in the lethal range. Similarly, a 150-grain rifle bullet weighs about 9,700 mg, so that a small fragment would have sufficient lead to be lethal. The studies of Pattee and co-workers show that ingestion of a greater number of shot resulted in higher blood lead levels. The increased toxicity does not appear to be a function of the amount of lead in the stomach, as all treatment levels used potentially lethal doses. The difference was probably a function of surface area, with more shot having greater surface area, and a greater rate of dissolving due to stomach acid. It is probable that ingestion of a single small fragment that is retained in the stomach of a condor could result in morbidity and potential paralysis of the digestive system producing the symptoms observed in AC3, #105, and AC8 in California, and other condors in Arizona. These birds developed crop stasis, could not digest food, and lost weight to the point of death (except #105 and AC8 were kept alive by feeding with stomach tubes). Condors IC1, Broken Feather (BFE), and #191 also lost much weight before they were discovered dead from lead intoxication.

II. D. Copper Residues in Condors, and the Relationship with Lead.

Copper residue levels in livers of dead condors have been quite variable and inexplicably high in some birds. The levels of lead and copper residues in liver of necropsied birds are given in Table 7. Birds that died of lead toxicity had high liver levels of copper, which might be expected if they had ingested copper jacketed lead fragments from rifle bullets. In these birds the liver lead levels ranged from 17-34 ppm, with accompanying copper levels of 25-181 ppm. Other condors, however, had liver lead levels at 1 ppm or lower, but copper levels in the liver of 21-120 ppm. Figure 14 graphs the data for all condors with both lead and copper liver residues, and there is almost no correlation between the levels ($R^2 = 0.096$). No condor liver measured to date had copper levels below 21 ppm. The source(s) of copper, and the significance of copper levels in condor liver is probably not from bullet fragments, but rather from dairy calf liver, which is very high in copper.

Table 7. Comparison of lead and copper residues in condor liver samples.

BirdID	Death Date	Final Diagnosis	Liver Lead ppm	Liver Lead	Liver Copper
Condor 1	03/22/84	lead poisoning	35	35	ND
"Broken Feather"	04/10/85	lead poisoning	23	23	ND
66	10/07/82	ethylene/propylene glycol	<0.5	0.25	42.3
G78	06/30/99	Uncertain, rapid weight loss	<1.0	0.5	75
207	01/15/00	Aspiration of regurgitated ingesta, reason unknown	<1.0	0.5	23.7
197	02/04/00	UNK. Eagle predation suspect	<1.0	0.5	94.6
SB 165	06/12/00	lead poisoning	34	34	25.3
191	06/16/00	lead poisoning	17	17	181
SB 184	09/09/00	UNK.	<1.0	0.5	61.4
R1	10/06/00	UNK. High liver copper	<1.0	0.5	120
SB 082	12/25/00	Coyote Predation, possible other contributing factors	<1.0	0.5	21.6
SB 074	12/29/00	Predator attack, suboptimal health	<1.0	0.5	41.3
G132	01/31/01	lead poisoning	26.4	26.4	49.8
SB 228	02/09/01	33% wt loss, poor body condition.	1	1	85.6
SB 230	05/09/01	Probable electrocution, power line collision	<1.0	0.5	113
SB215	06/27/01	powerline electrocution, collision	<1.0	0.5	41.2
"Tehachapi"	1976	shot	1.6		87
Geometric mean of Liver Copper :					59.0



Eisler (1998) gives liver copper data for many field collected avian and mammalian species, but no raptors or vultures (data included in Table 8). Copper is an essential trace metal nutrient, and stored in the liver of many species. The range of liver copper levels is wide, with many seabirds in the range of 4-446 ppm, on a fresh weight basis (averages for species is 4-6 for gallinaceous birds and up to 80 ppm for waterfowl). Waterfowl from Poland had liver residues ranging from 11-200 ppm. Some values for avian liver are given on a dry weight (DW) basis, but Eisler gives no range of conversion factors for avian liver, fresh to dry weight basis. Livers of some canvasbacks collected in Louisiana (Custer and Hohman 1994) and livers of some mute swans (*Cygnus olor*) from England (Bryan and Langston 1992) both contained more than 2,000 mg Cu/kg DW. In the case of mute swans, several thousands of milligrams of copper per kilogram dry weight occurred in blackened livers, attributed to ingestion of flakes of copper-based antifouling paints (Bryan and Langston 1992). For values in mammalian liver given in both fresh and dry weight, the dry weight values are higher by 2.5-5 times. If these factors are approximate for avian liver, the canvasback and swan data would indicate liver copper levels of 400-800 ppm on a fresh weight basis.

Eisler reports that no data are available on copper toxicity to avian wildlife. Dietary experiments to determine nutritional requirements and toxicity with domestic birds show that growth of turkey poults is improved at 60 mg Cu/kg DW ration and inhibited at 120 mg/kg DW ration, with signs of gizzard histopathology at 500 mg/kg DW ration (Wood and Worden 1973; Poupoulis and Jensen 1976; NAS 1977). However, Supplee (1964) reported that in turkeys, natural diets with as much as 800 mg Cu/kg ration have no adverse effects on growth or survival. Normal domestic chick rations have about 4 mg Cu/kg (Carlton and Henderson 1964b). In the chicken, adverse effects of copper occur in chicks fed diets containing 350 mg Cu/kg ration for 25 days (reduced weight gain) and in adults given a dietary equivalent of more than 28 mg Cu/kg BW. Chicks fed diets of 500 mg Cu/kg ration show damage to the gizzard lining (Bremner 1979). Chicks fed copper-supplemented diets do not have elevated copper concentrations in liver or signs of liver damage (Bremner 1979).

Ducklings, unlike chicks, accumulate copper in livers when fed diets supplemented copper at dietary concentrations as low as 15 mg/kg DW ration (Wood and Worden 1973). Domesticated mallards show a dose-time dependent increase in liver copper concentrations, with a maximum concentration of 254 mg Cu/kg DW liver in ducklings fed 150mg Cu/kg diet. No growth depression or other adverse effects were observed in ducklings fed 150mg Cu/kg diet. Mallards during molt choose to drink water containing 100 mg Cu/L over distilled water (Rowe and Prince 1983).

Table 8. Copper Levels in Liver of Wildlife and Domestic Animals. From Eisler, 1998

Birds

Western gæbe, <i>Aechmophorus occidentalis</i> ;	
Puget Sound, Washington; 1985-86;	
Liver	12.7-17.6 DW
Ducks, <i>Anas</i> spp.;	
Poland; 1988-91	
Liver	58.0 (11.0-200.0) FW
Geese, <i>Anser</i> spp.;	
Poland; 1988-91	
Liver	80 (29-160) FW
Adelie penguin, <i>Pygoscelis adeliae</i>	
Liver	11.9 (11.0-12.6) DW
Chinstrap penguin, <i>Pygoscelis antarctica</i>	
Liver	12.6 (12-13) DW
Gentoo penguin, <i>Pygoscelis papua</i>	
Liver	26.5 (24.0-27.6) DW
Canvasback, <i>Aythya valisineria</i> ;	
Louisiana; 1987-88;	
liver; females	76-187 DW
Ruffed grouse, <i>Bonasa umbellus</i> ;	
liver	5.2 FW
Lesser black-backed gull, <i>Larus fuscus</i>	
Liver	17.0 DW
Albatrosses, eight species; adults vs. juveniles	
New Zealand; 1975-83	
Liver	5.0-8.6 FW vs. 12.2-225.3 FW
Gulls, <i>Larus</i> spp.; adults vs. juveniles	
Liver	5.0-6.6 FW vs. 23.8-35.0 FW
Petrels, 19 species; adults vs. juveniles	
Liver	4.45 FW vs. 8-75 FW
Shearwaters, three species of <i>Puffinus</i> ;	
liver;	6.4-7.2 FW vs. 4.6-446.3 FW
Surf scoter, <i>Melanitta perspicillata</i> ;	
San Francisco Bay, 1985; January vs. March	
liver;	37.8 (29.3-47.0) DW vs. 50.1(41.3-58.3) DW
Turkey, <i>Meleagris gallopavo</i> ;	
Poland; 1988-91	
Liver	4.7 (3.1-13.0) FW
Brown pelican, <i>Pelecanus occidentalis</i>	
Florida, Georgia, South Carolina	
Liver;	(4.3-9.0) FW
Seabirds, 19 species; pelagic;	
North Pacific Ocean; 1982-87	

Marine mammals

Gray whale, <i>Eschrichtius robustus</i> ;	
Stranded, North American west coast; 1988-91	
Liver	9.2 (0.6-25.0) FW
Grayseal, <i>Halichoerus grypus</i> ;	
British Isles and vicinity; 1988-89	
Liver	(4.0-26.0) FW
Harbour seal, <i>Phoca vitulina</i> ;	
British Isles; 1988-89;	
liver	7-21 FW
Manatee, <i>Trichechus manatus</i> ;	
Florida; 1977-81;	
Liver	175.0 (4.4-1,200.0) DW
Dolphin, <i>Tursiops geophysus</i> ;	
Argentina; found dead	
Liver	77.7 FW

Terrestrial mammals

Impala, <i>Aepyceros melampus</i> ;	
Kruger National Park, South Africa; 1989	
Liver	(3-444) FW
Moose, <i>Alces alces</i>	
Alaska	
Liver	3.1 FW
Sweden; 1979-80	
Liver	(3.2-96.0) FW
Cattle, <i>Bos</i> spp.	
Poland; 1987-91	
Liver	29.0 FW
Various locations;	
liver	38.2-156.1 DW
Waterbuffalo, <i>Bubalus</i> sp.;	
Kruger National Park, South Africa; 1989;	
Liver	Usually 18-80 FW; (6-144) FW
Dog, <i>Canis familiaris</i>	
Liver	82 FW; 336 DW
Coyote, <i>Canis latrans</i> ;	
kidney	5.2 FW
Goat, <i>Capra hircus</i> ; mother vs. newborn	
Liver	11.3 DW vs. 63.3 DW
Roe deer, <i>Capreolus capreolus</i> ;	
Poland; 1987-91	
Liver	28.0 FW
Horse, <i>Equus caballus</i> ;	
liver	10.3-51.5 DW

Risebrough (2001) fed metallic copper pellets to Turkey Vultures in a contract study for the USFWS, and measured both blood and liver residues. Liver residues after 8.5 weeks of copper feeding averaged 20.5 ppm (geometric mean, ± 3.9 S.D.) Copper levels in livers of birds fed control diets, or diets containing tungsten, tin, and bismuth averaged 17.6 ± 4.4 and 15 ± 3.9 ppm, respectively. Liver copper was slightly elevated (16%) by feeding pellets for 8.5 weeks, with no change in blood copper levels. The highest copper levels after 8.5 weeks were about equal to the lowest copper levels in livers of condors (Table 7). The blood levels of copper remained low throughout the feeding trial, and the copper levels in the copper fed group were not different from the levels in the control and tungsten-tin-bismuth groups.

Risebrough concluded that Turkey Vultures might not be good surrogates for California Condors, because the copper levels remained so low, even when birds were fed substantial doses of metallic copper. Feeding metallic copper to turkey vultures appears to be non-toxic, as blood and liver levels never rose to levels expected to clinically affect the birds. No clinical symptoms were observed, and all the birds were released to the wild as healthy birds following the study.

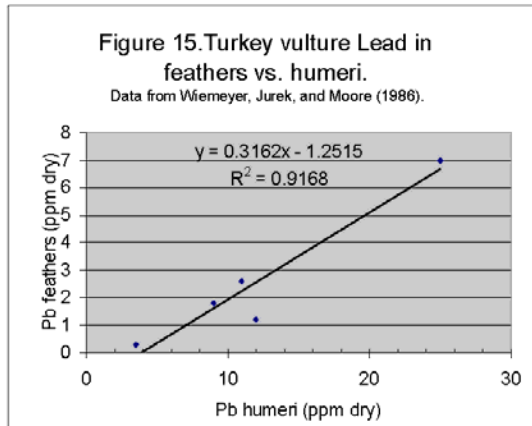
Copper is an essential trace metal nutrient, and is an essential co-factor of several enzymes critical for normal metabolism. Copper is a potent inducer of the liver metal binding protein metallothionein in some species of animals, and liver storage of copper may be a normal process accomplished by binding and sequestration by metallothionein. Poultry fed large concentrations of copper do not accumulate copper in liver, while mallards show a dose dependent increase in copper accumulation to 250 ppm (Wood and Worden, 1973). Tree swallows in high copper environments develop elevated liver copper in association with elevated metallothionein (St. Louis et al. 1993). The copper necropsy data for condors averages 59 ppm (geometric mean, range 21-181) for the 18 birds measured. It is not known whether the levels of copper in the livers of condors represent an induction of metallothionein, and/or whether this is the normal range of liver copper for this species. Measurement of metallothionein in archived samples of frozen condor liver and comparison to other species, especially Turkey Vultures, would provide substantial data. Unfortunately, the liver biopsy samples taken for the Riseborough (2001) study were consumed in analysis.

Condors obtain dietary copper from all carcasses fed upon. The normal copper levels in liver of deer (12 ppm), wild boar (16 ppm), swine (8.5 ppm), and cattle (29 ppm) as reported in Eisler (1998) would supply condors with a substantial amount of copper in their normal diets, especially since they preferentially consume the liver. ATSDR Toxicity Profile for Copper (1998) reports that calf liver contains 61 ppm Cu on a fresh weight basis. Most mammals at birth have higher liver copper than adults, probably in anticipation of rapid growth and nutritional requirements for copper during growth. Since condors readily ingest liver, it is probable that condors receive a large amount of copper from the diet provided by the field teams. No studies have been conducted to determine whether copper binding proteins in liver of condors are induced by feeding copper, as they are in ducks and tree swallows. Turkey vultures do not appear to accumulate copper in liver (Riseborough, 2001), but because of the species differences in copper metabolism and metallothionein induction, it is impossible to predict the liver physiology of condors without direct experimental evidence. If condors do induce liver copper binding proteins and bioaccumulate copper, it is probable that levels in the range of 50-150 ppm would not be toxic.

II. E. Deposition of Lead and Copper in Feathers and Other Tissues.

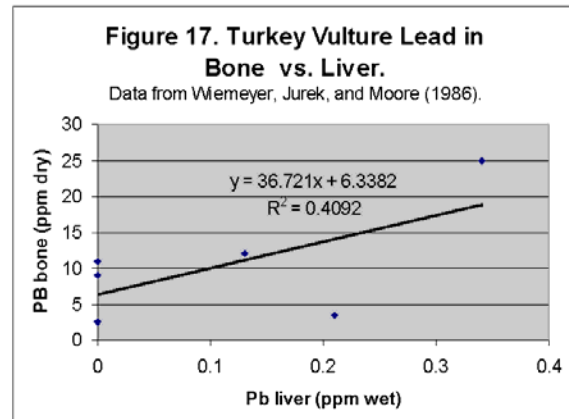
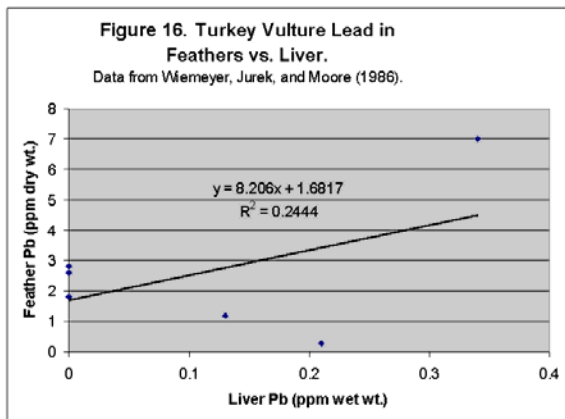
Lead in the blood is deposited in feathers during feather growth, and becomes bound to the protein matrix of the feather. Weimeyer et al (1986) quantified lead in condor feathers, finding that the average level in some feathers was as high as 14 ppm. The same study measured lead in feathers, liver and bone of ravens and turkey vultures, and

found better correlation between levels in feathers and bone than with feathers and liver or bone and liver. Figure 15 gives data plotted from their study, in which secondary feathers from one wing were ground homogeneously and levels compared to bone samples taken from the femur.



Since the secondary molt in vultures occurs on a gradual two-year cycle, homogenizing all of the feathers from one wing essentially averages the lead exposure during that period. This long-term integration can be thought of as analogous to the slow turnover of bone ($T_{1/2} > 600$ days), and the residue data from these two tissues correlate well ($R^2=0.917$), although bone accumulates about 3 times as much lead as feathers on a dry weight basis. Liver lead levels have a more rapid turnover, similar to the turnover

half-life of other soft tissues as discussed in Section II.A. The correlation between feathers and liver was poor (Figure 16 ($R^2=0.244$)) as was the correlation between bone and liver, given in Figure 17 ($R^2=0.409$).



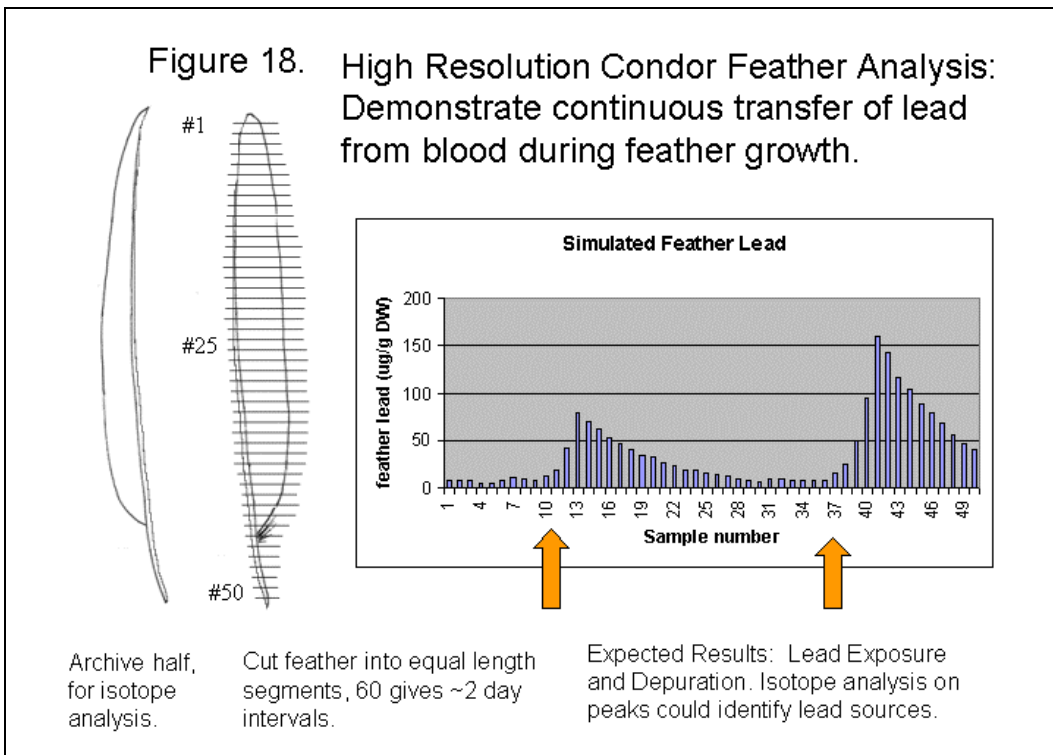
Because liver gives an indication of medium term exposure, the levels in liver may not reflect the long-term exposure reflected by levels in bone or the averaged levels in composites of many feathers.

II. F. High-Resolution Analysis of Feathers to Demonstrate Lead Exposure.

Very sensitive methods for metals analysis have been developed since the work of Weimeyer et al (1986), including inductive coupled plasma mass spectroscopy (ICP MS), which is capable of detecting metals in small fractions of feathers. Sequential analysis through the length of a feather has not been published, but if short segments of feather were analyzed, they should reflect the circulating levels of lead in the feather pulp during feather growth. Since the distal portions of feathers dry and harden as they grow, only the growth region in the feather pulp would reflect the circulating lead levels at the time

of growth. High-resolution sampling should detect the lead deposition spatially along a feather, and it would present a record of the lead circulating in the feather pulp at the time of growth. High-resolution feather analysis of lead (and copper) should, therefore, provide a record of the bird's exposure and blood levels of metals sequentially throughout the period of feather growth.

Snyder et al. (1987) have analyzed primary molt and feather growth in California Condors, and have determined that growth of a primary feather takes approximately 105-120 days (3 ½-4 months), with a growth rate of about 5mm/ day. The large size of the feathers, and the rapid (5mm) daily growth rate should provide enough material to analyze feather protein for lead and copper at a 2 or 3-day interval. Figure 18 shows a diagram for a proposed feather sample collection that would provide sufficient material for analysis, and save the remainder of the feather.



At the normal detection limit of 0.01 $\mu\text{g Pb}$ for ICP MS, the detection limit for a 20 mg sample would be about 0.5 ppm Pb in feathers, which would be equivalent to about 0.06 ppm in liver or about 1.5 ppm in bone. High-resolution analysis would provide a great deal of information for the determination of lead exposure from archived condor carcasses, if the feather follicle of a feather growing at the time of death were analyzed. This would provide a time sequence for lead accumulation in the feather correlated with circulating blood lead levels during the weeks prior to the death of the bird. Analysis of the same samples for copper could demonstrate whether there is any correlation between circulating lead and copper in condors. If the source of copper is from bullet fragments, the elevated lead and copper levels should be correlated. If, on the other hand, the source of copper is dietary, and derived from feeding on lead-free animals, there may be little

correlation between copper and lead in feathers, just as there is no correlation between copper and lead in liver samples from condors that have died.

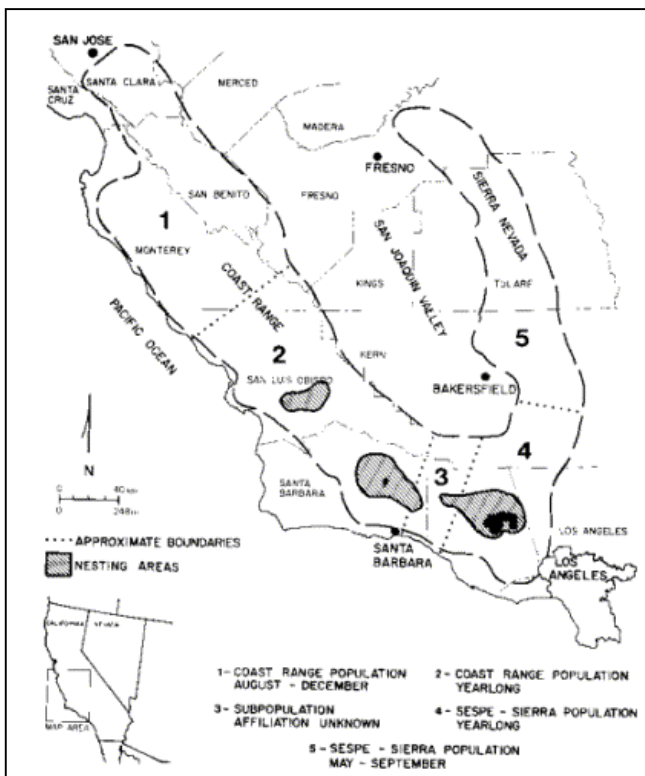
High-resolution feather analysis for lead should also provide a great deal of information on the frequency of sub-lethal lead exposure and recovery in condors feeding in the wild. Analysis of random molted condor feathers that had been grown while the bird was in the wild would also provide data on the magnitude of lead exposures of untreated condors. Since each feather takes about 3 ½-4 months to grow, analysis would document the exposure of the birds over this period of time. This study strongly recommends conducting high-resolution lead analysis on many condor feathers.

Section III: Condors in California: Habitat, Habits and foraging

III. A. Condor Habitat and Habitat Use

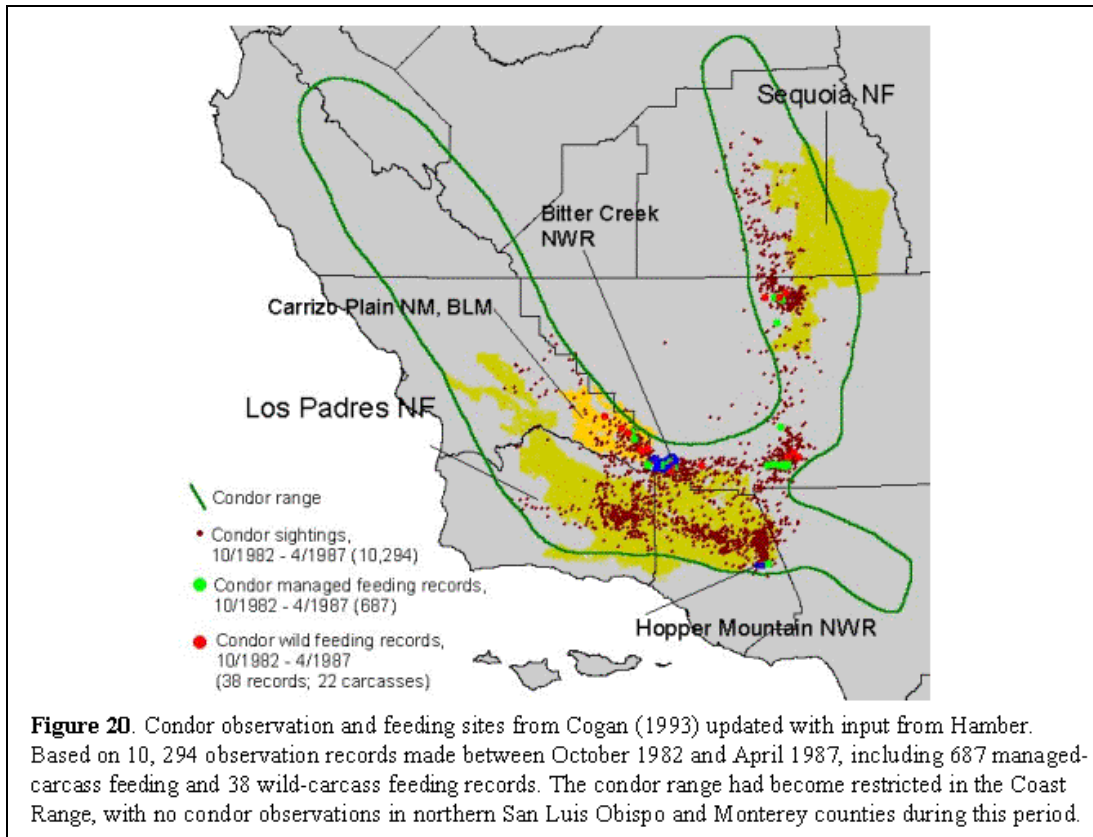
The reintroduced California population of California Condors now occupies the majority of the range of wild birds known since about 1900. Several range maps have been prepared by groups of authors, and Figure 1 outlines the range identified by the Condor Recovery Team (Condor Recovery Plan, 1984 revision). There has been some range expansion to the northwest because of the reintroductions by the Ventana Wilderness Society in the Big Sur region, and the current range along the coast is remarkably similar to the range map depicted by Wilbur (1978). (Figure19).

Figure 19. Condor Range. from Wilbur (1978).



This map also identifies the nesting areas known to be active at the time. The range has expanded into the southern Sierra Nevada over the past three years with the release and movements of condors AC8, AC9 (#21), and #171, as identified through the use of satellite telemetry. AC9 (#21) returned to the Blue Ridge National Wildlife Refuge in Tulare County, marking the first time a condor has been documented in that critical habitat since the mid 1980's. In July 2002, condor #171 was located near Breckenridge Mt. In the southern Sierras, 275 miles from her release site, and in the same area AC8 has frequented since her release in April 2000.

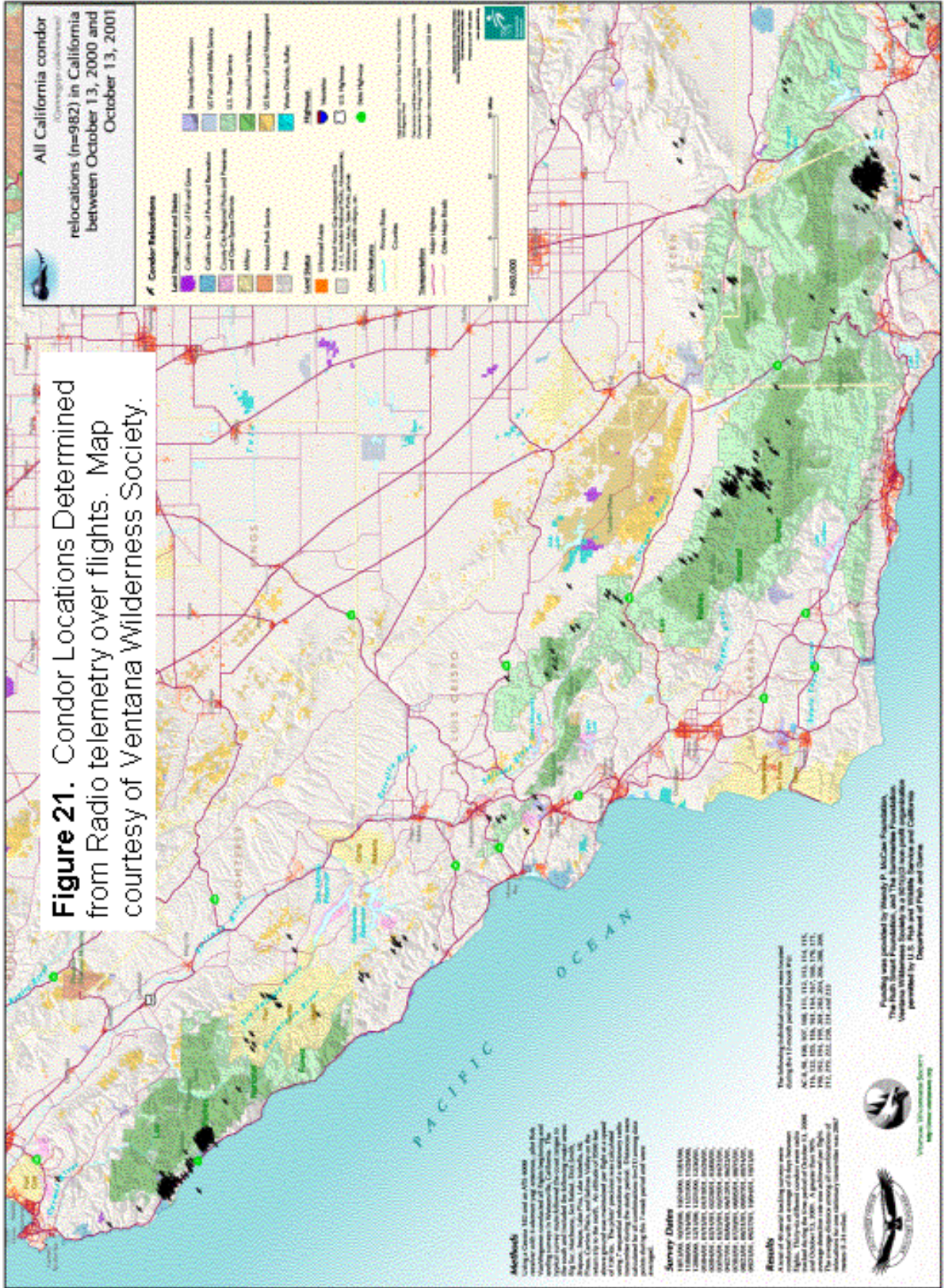
Extensive field observations during the 1980s under the direction of the Condor Recovery Program were compiled digitally by Cogan (1993), and made available on a CD ROM as an Arcview database. Figure 20 gives the locations of 10,294 records of condor sightings during the period 1982-1987, until all the birds had been remanded to captivity.



The observations include 687 records of feeding on baited carcasses, and 38 records at 22 wild carcasses. The wild feeding data was reviewed and up-dated by Jan Hamber, Santa Barbara Natural History Museum, for 48 incomplete records in the Cogan database. It is remarkable that only 22 wild carcasses were discovered in the 1980's, at a time when Snyder estimated that condors were obtaining about 80% of their food from wild carcasses, even during the period of supplemental feeding.

Condor releases in the 1990s were initially limited to young captive reared birds with no knowledge of the previous historic range. It was assumed that it would be easier to manage the feeding of free ranging condors, since they could be introduced to feeding in the wild at specific feeding stations.

The Ventana Wilderness Society (referred to as the Ventana group in this report) began releasing condors in cooperation with the Condor Recovery Team in 1997. The releases and the current feeding program are located in the Big Sur area, just inland from Julia Pfeiffer Burns State Park, Monterey County. Released birds remained in the local area for the first year or two, but since 2000 they have begun moving extensively back and forth to the Sespe Sanctuary and mingling with the birds released from Hopper Mountain NWR and monitored by the USFWS, Ventura office (Hopper team). Radio-telemetry overflights during the period October 2000-October 2001 located birds throughout the expanded range, and the data are depicted in Figure 21, kindly provided by Kelly



Sorenson, VWS. 982 recorded locations are mapped for 36 condors, shown as small bird icons. The majority of locations were in the Big Sur, Lion Canyon, and Hopper Mountain areas. All of the locations in the Coast Range have been on the coastal side of the Salinas River. During the period 10/00-10/01, none of the birds were using the Carrizo Plains Natural Area, and only a few locations were recorded for the Tejon Ranch and the Mt. Piños area. Most of these locations were for condor AC8.

III. A. 1. Analysis of Habitat Use from VWS Overflight Map:

Condor use was determined for the period October 2000-October 2001 by quantifying the location data presented in the VWS overflight map. The overflights were all made during daylight hours, and therefore represent normal diurnal activity of condors, including roosting, foraging, and exploring nesting areas. Because only single short flights approximately 8 days apart were conducted, it is apparent that condors used many areas for which locations were not detected. This is obvious, because condors must have passed over areas with no recorded locations to arrive at the locations at which they were detected.

The locations of birds detected are placed on the map as small bird icons that overlap significantly in areas of frequent condor use. Three clusters of locations contained most of the location data: two areas adjacent to Big Sur State Park, the Dick Smith Wilderness (Lion Canyon), and the area around the Hopper Mountain Refuge. Outside of these 3 locations, 118 icons could be identified as separate locations. Of the 982 locations, 864 are assumed to be included in the 3 areas of multiple overlapping icons.

The highly skewed distribution of locations is undoubtedly due to the fact that condors habitually used the feeding locations and the nesting areas the Dick Smith Wilderness preferentially to other areas. The use of other areas by condors could be quantified by counting location observations, and creating a density map of locations. Because only a hard-copy version of the overflight map was available, a physical grid was constructed, and icons were counted to quantify the distribution of condors.

A grid pattern was constructed with a cell size of 252 mi², covering the condor range depicted in the overflight map with approximately 60 grid squares. The grid was placed over the map in a random position, and the number of condor icons in each square was recorded on a datasheet. An electronic copy of the map was greatly enlarged to make the best estimate of the number of icons present in areas where icons touched or overlapped each other. The best estimate of the number of icons was written on the map, and used to assist the grid counts. Where areas of multiple overlaps occurred, the symbol “M” was used to denote “many” icons within the grid square. The grid was moved systematically from its original position by 1/3 of the grid spacing, and recounted to determine the number of icons within each grid square. This was repeated systematically for a total of nine counts to determine the best estimate of condor locations within each grid square. The number of grid squares overlapping the condor range was counted for each iteration, and recorded along with the numbers of locations within each square within the condor range included on the VWS map.

The number of condor locations in any grid square ranged from 0 to 11, plus “M” for 3-5 grid squares in each iteration. Data for all nine iterations were recorded, and the locations of maximum condor use were identified. The maximum number of individual condor locations was 10 in any one grid (excluding the three feeding location areas). The highest use area was Fort Hunter Liggett (FHL), followed by the western portion of the Dick Smith Wilderness, and the area of the coast range immediately south of FHL.

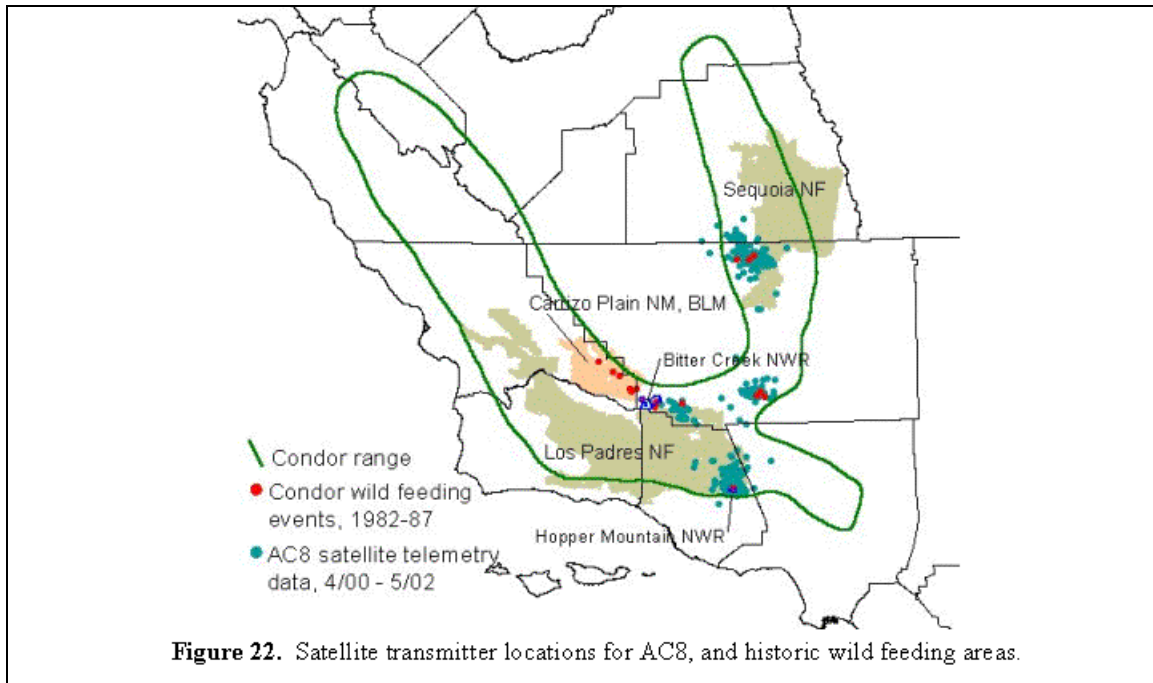
The condor range included in the VWS map was 58.1 grid squares (14,617mi²), and on each counting iteration an average of 29 (50%) of these squares had 0 locations. The distribution of locations was calculated to form a matrix, and a frequency distribution table was constructed to demonstrate the frequency of use for each area of the condor range.

No locations were ever detected over the Carrizo Plains or the Bittercreek Refuge during the period October 2000-October 2001. These are among the traditional foraging areas identified by Snyder and Snyder (2000). Only 3 locations were detected over the Tejon Ranch, and 6 over the Cuesta Pass area, known to be the flyway for birds moving from the Big Sur area to the Hopper area. The traditional foraging areas identified on page 140 of Snyder and Snyder (2000) had a total of only 12 locations for the entire year, representing only 1.2% of the observed locations. 3 locations were over the Tejon Ranch, 7 over Mt. Pinos and the San Emigdio, one in northern Kern County, and one northwest of the Carrizo Plains. Condors clearly did not frequent the traditional foraging areas during the survey year.

FHL was the highest “non-feeding site” area used by condors during the period of 2000-2001. 12 locations were observed within the boundaries of FHL, which has an area of 257.8 mi². One condor was observed feeding on an elk carcass on the base, and three other instances of probable feedings have been mentioned in field notes. This is a high concentration of the known “wild” feeding events. The Coast Range immediately south of FHL and the San Rafael Wilderness are the second highest use areas. The area to the east of Santa Clarita had the third highest use. The Mt. Pinos-San Emigdio area is still used to a moderate extent by condors. Condors remained in the vicinity of the feeding areas approximately 88% of the time during the year.

III. B. Satellite Transmitter Data for Condor Movements: Condor AC8

Condor AC8, an adult female trapped in 1986, was released back into the wild in April 2000. Figure 22 is a map of locations reported by her Argos satellite transmitter during the period April 3, 2000-May 17, 2002.



AC8 locations are mapped, as are the known historical wild feeding locations. AC8 has spent much of the time in the Sierras, with additional time at the Sespe and Hopper Ranch, and in the Tejon Ranch area. She has not been located by satellite in the Carrizo Plains, or into Santa Barbara, San Luis Obispo or Monterey Counties.

The locations and movements of AC8 concentrate around the historic feeding locations of the Sespe-Sierra subpopulation described by Wilbur (1978) on the Tejon Ranch and northern Kern and Tulare Counties in the Sierras, in addition to the feeding stations near Hopper Mountain NWR. When AC8 was released back into the wild, after 14 years in captivity, she returned to the habitats she had been using in the early 1980s, foraging for wild carcasses rather relying on the artificial feeding stations maintained by the Recovery Team. The satellite locations of condor #171 in the southern Sierras (near Breckenridge Mountain in July, 2002) indicates that other birds are exploring the Sierras, and may have accompanied AC8 on foraging trips into the Sierras, which may pose increased risk of lead exposure to those birds feeding on hunter-shot carcasses in addition to carcasses from natural mortality.

AC8 was captured November 11, 2002 and discovered to have high blood lead levels, and was taken to the LA Zoo for chelation therapy. Her blood lead rose from 1.01 ppm to 4.10 ppm in two days during holding and transport to LA Zoo, before chelation therapy was initiated. Radiography showed 3 dense fragments in her digestive tract, and her lead levels remained quite elevated until the last of the visible fragments (only one subsequently identified as lead) passed from her system by early December. Satellite transmitter data for AC8 are available through October 21, 2002, when her transmitter ceased functioning. Her conventional radio transmitter also stopped functioning in October 2002. AC8 spent the months of September and early October moving back and forth between Hopper Mountain and the Tejon Ranch, spending approximately equal

time in both areas. Visual sightings were made for AC8 at Hopper Mountain on October 27, November 1, 10, and 11, when she was trapped and taken into captivity. AC8 showed no clinical symptoms of lead intoxication on November 11. AC8 probably ingested the lead very shortly before she was captured. Her blood lead levels rose dramatically in the two days after capture, indicating that her exposure was recent. The Zone D13 deer-hunting season was open from October 12 through November 10, 2002, in the area surrounding the Hopper Mountain NWR. AC8 was visually sighted in the area on 2 occasions in early November prior to her capture. It is highly likely that AC8 consumed a bullet fragment from a deer carcass during the few days prior to her routine capture. The capture of AC8 was extremely fortunate, because she was quickly treated and saved from lead poisoning by emergency treatment from November 13-December 9, when the lead fragment was defecated. She was kept under observation until she was released into the wild on December 23, 2002. AC8 was found dead on February 13, 2003, after being shot and killed on the Tejon Ranch.

III. C. Sources of Food for Condors

The preferred traditional foods for condors before the introduction of cattle into California in the late 18th century were probably the large ungulates, deer, elk and antelope. Condors probably existed in an ecological landscape not far different from the plains of Africa, where other large vultures have evolved with herds of ungulates. When herds of game animals disappeared from California, the traditional food base of condors was marginalized. Cattle replaced the herds of native game, and condors have adapted to the new food source. Koford (1966) described five major food sources: cattle, sheep, ground squirrels, deer, and horses; which comprised more than 95% of condor diet. Many other items were also fed upon, including marine mammals, coyotes, jack rabbits, domestic dogs, goats, and domestic pigs.

Part of the scope of work for this review was to document the location of feeding sites, and identify whether the carcasses were artificial feedings, or wild carcasses found by the birds. The 687 managed feeding records in the Cogan dataset (Figure 20) concentrated in 4 areas: 1) Northern Kern County, near Glenville, where ranchers had been placing cow carcasses in prominent areas for condors at least since the 1970s; 2) the Tejon Ranch, where the Condor Program placed carcasses during 1985 and 1986; 3) the Hudson Ranch, San Emigdio Ranch, and Elkhorn Hills (Carrizo Plains Natural Area); and 4) the Hopper Ranch, adjacent to the Sespe Sanctuary. The 22 identified wild carcasses (12 calves, 5 cows, 3 deer, and 2 “unknown”) were all located in the same general four areas. It is highly likely that condors found wild carcasses in many other places, but these locations were near managed feeding locations, and observers were probably more likely to locate birds feeding in these areas.

Managed artificial feeding stations have been an important element in the condor program since Wilbur’s program in the Sespe in the 1970s. Wilbur maintained a feeding program in the Sespe Sanctuary, Ventura County, while the 1985-1986 feeding program concentrated on the Hudson Ranch (now Bittercreek Refuge), and the Tejon Ranch, both in Kern County. The program was expanded in the spring of 1985, when clean carcasses

were regularly set on the Hudson ranch to provide a source of carrion free from lead fragments that had been identified as a threat to wild condors. The program was not entirely successful, as many of the free flying birds had traditional feeding areas, and foraged widely in search of carrion. The number of wild carcasses found by observers during the 1980s is remarkably few (only 22), and Snyder and Snyder (2000) estimated that managed feeding accounted for as little as 20% of all feeding by condors during 1985. Snyder and Snyder recount in their book that estimates were made of feeding rates at the feeding stations, and compared with monitored feeding rates of condors in captivity, in an effort to estimate what proportion of their food was consumed at managed feeding sites. Data from July 1985 feedings at the Hudson Ranch were used for the estimate, because it was the most complete dataset for managed feeding, and the wild condors used the Hudson ranch extensively during July. A “very generous interpretation” of the feeding records indicated that wild condors were obtaining only 20% of their food at managed feeding sites. Even though many of the condors free-flying during the 1980s had radio transmitters, it was not possible to observe the birds feeding on wild carcasses except in the rarest of circumstances.

Until 1987, all condors in the wild were hatched in the wild, and were birds that had been foraging in the wild before either Wilbur’s Sespe feeding program, or Snyder’s program of the mid 1980s. All of these birds were habituated to searching for carcasses, and apparently used the feeding stations only occasionally.

During recent years, field crews have begun recording observations of “mystery crops”, that is, identification of condors with full crops that have not been feeding at managed feeding sites. These data are entered into field notes by the Hopper and Ventana teams, but have not been systematically organized and cataloged. Because the potential lead exposure from feeding on hunter shot carcasses is high, a review of this dataset should be done, and compared with feeding rates at managed feeding sites, to estimate the proportion of wild carcasses found and fed upon by condors.

Since May 2001, management feeding at Ventana was shifted from food carcass placement distributed within a 15-mile radius of the release site to immediately within the release site canyon to potentially reduce the risk of lead exposure to condors from feeding on shot carcasses (J. Burnett, pers. comm.) Single-site food subsidies have been proposed as a way to decrease exposure to lead in naïve condors (Meretsky et al 2000), but the data from Ventana do not indicate a difference. There was no difference ($p=0.68$) between blood lead levels taken before ($N=31$, mean = 12.3, sd = 15.7, range 0-62) and after ($N=28$, mean = 10.9, sd = 8.9, range =2-37) the date of the more centralized feeding.

In the past three years the Hopper team has visually documented an estimated 10-20 wild feeding events (Mike Barth, pers. comm.), including 2 deer and an apparent road kill wild pig on Hwy 58 in San Luis Obispo County. The Ventana program reported the following 12 wild feeding events in the past 3 years: 5 marine mammals including three sea lions fed upon by at least 8 individual condors, and 1 deer, all within 5 miles of the release site; 2 deer and one cow near Atascadero; a predator killed yearling elk on Fort Hunter Liggett

in April 2002; and one horse in Monterey County during the fall of 2002. Figure 23 is a recent example (October 9, 2002) from Atascadero, San Luis Obispo County.



This was an apparent road-kill deer, as described by the photographer, Eric Palmer, a junior high school student from Atascadero.

Older birds, such as AC8 and AC9 (wild hatched birds captured in the 1980's and recently released back into the wild) serve as examples for young birds released into the wild during their first year. The more experienced birds provide a great deal of socialization, but

also forage for wild carcasses throughout the entire condor range, including the southern Sierra Nevadas. As more experienced birds forage for wild carcasses, younger birds will be encouraged to accompany them, and the risk of lead exposure from lead fragments in hunter-shot carcasses will increase.

III. D. Estimating the Availability of Carcasses in the Wild.

Condors appear to prefer deer and calf carcasses to other animals as food. The relatively thin skin of deer and calves, compared to adult cattle or wild pigs, seems to make them more attractive, as condors have difficulty tearing the skin of adult cattle. Deer are probably one of the most common sources of traditional food with which condors co-evolved. Deer mortality in California is difficult to document, but published reports can be extrapolated to develop an estimate of deer carcass availability to condors. Vern Bleich, CA DFG, Bishop, and other DFG biologists have been radio collaring and recording mortalities of deer in the Eastern Sierras for several years. Bleich and Taylor (1998) analyzed cause specific mortality in five populations of mule deer, and determined that 83% of mortality of female deer was due to predation, while 64% of the mortality of males was due to hunting, and 36% due to predation. Mountain lions accounted for the greatest percentage of predator kills (91% of female deer, and 100% of males). Other human caused mortality (road kills, fences, etc.) were responsible for 4.8% of female deaths. 73% of mortality overall was caused by predation (92% mountain lion, 8% "other"), 13.5% hunting, 9.6% due to malnutrition, and 4% other human causes. Becky Pierce, CA DFG, Bishop Office, reported in an unpublished summary provided for an Inyo County EIS, that 20% of mortality of the Round Valley deer herd was due to road kills on Hwy 395. This may be an unusual situation, as this herd migrates across and winters adjacent to Hwy 395.

Hunting represents a small fraction of the total deer mortality, but may represent a large fraction of carrion available to condors to consume. Mountain lions cache carcasses so that they may feed on them for 2 or 3 days, and the carcasses would be hidden from visual scavengers such as condors. After multiple feedings, there may be little left for condors, and the mammalian scavengers frequently dismember the carcasses, with only limb bones and vertebrae available for birds to scavenge. Road kills, malnutrition deaths, hunter-shot cripples and gut piles represent the bulk of other deer mortality carrion available to condors. A small number of land-owner depredation permits for deer are also granted each year, but carcasses of these animals are required to be removed from the field. Of the non-predator mortality, the majority of deer carcasses would be hunter or depredation kills. The number of hunter kills within the condor range is estimated in Section IV.A.

Wild pigs, cattle, and coyotes are the other large mammals likely to be encountered by condors. No references to natural pig or coyote mortality could be located. Estimates of pig mortality have been made with large assumptions, after discussions with Doug Updike, CA DFG, Jim Matthews, and others familiar with pig activities in California. Pigs are reproductively very prolific, and populations fluctuate greatly with forage and drought conditions in California. Natural mortality is high in drought years, with young pigs being lost first. Mountain lions probably take pigs, especially young pigs, but the greatest source of mortality to adult pigs is humans or natural mortality in drought years. Pig populations have exploded in California in recent decades, with expansion of pig range throughout oak woodlands, into the coastal mountains and medium elevations in the Sierras. Pig depredation permits are issued by the DFG, and pigs are shot by ranchers and land owners. It is now legal for landowners to shoot pigs without a depredation permit when pigs are damaging or threatening to damage crops or property. The total number of reported pig depredation kills for the period 2000-2002 in the eight Counties encompassing the condor range was 980 pigs. The number taken without permits, and the number of pigs crippled or not recovered is unknown.

Pig hunting has become very popular in California, with more pigs shot than deer in recent years. Deer and pig carcasses and gut piles probably represent the greatest source of lead in the environment in the condor range, but pig carcasses may not be very attractive to condors, perhaps because of their thick skin. The Hopper team has observed condors feeding on at least one road killed pig, but the Ventana team (K. Sorenson, pers. com.) reported that a pig carcass in full view of several condors for many days was not fed upon at all. Pig and deer gut piles, however, are likely attractive to condors. There are no documented feeding records of condors using gut piles, with the exception of gut piles of domestic pigs deliberately placed in the field by the MacMillan brothers near Chalome in the 1950s (MacMillian, 1968). Condors have been observed sitting in trees above gut piles, and near remains that were probably gut piles, but direct feeding has not been observed (Pete Bloom, pers. com.). Gut piles do not remain in the field for long, as both mammalian and other avian scavengers quickly consume them.

Hunters shoot coyotes in large numbers in California, and carcasses are generally left in the field. Wildlife Services (USDA APHIS) also controls coyote populations, but

agreements made in 1985 with the USFWS have designated procedures for disposal of coyote carcasses within the condor range, and animals taken by Wildlife Services do not appear to represent a lead exposure hazard to condors.

Much small game is taken in California, and condors have been documented by Koford (1953) and Wilbur (1978) eating ground squirrels, and to a lesser extent, jackrabbits. Ground squirrel bones were found in condor nest caves by Collins et al (2000), indicating that squirrels are fed to condor chicks. Small game left in the field by varmint hunters probably represents a significant source of lead, but I could find no documentation of lead residues in small game shot by varmint hunters. Varmint hunters use high velocity center-fire rifles as well as rim-fire 22 caliber rifles, and a study should be conducted to determine whether carcasses of “varmint” are a lead hazard to scavenging birds and mammals. It is probable that ground squirrel and jackrabbit carcasses are a source of much of the lead found in golden eagles by Pattee et al (1990), and in turkey vultures and ravens by Weimeyer et al (1986).

III. E. Seasonal Mortality of Game Animals, and Availability to Condors

Natural mortality of deer occurs during harsh conditions in winter, and in drought conditions in late summer and fall. Mortality of fawns occurs in the early summer through fall, from both predation and malnutrition dieoff. Mountain lions take deer year around, but carcasses may not readily be available to condors. Most hunter shot deer within the condor range are shot in August and September in the Coastal Zone A, and in September through mid November in the Tehachapi Mountains and Sierras. Deer depredation permits are valid year around, but probably represent a small fraction of the mortality.

Wild Pig natural mortality occurs in drought conditions, when feed and water become scarce. Pig hunting is allowed year around, although the traditional hunting season is in the summer and fall. Organized pig hunts are now held on the Tejon Ranch from December through July, and hundreds of pigs have been taken off the Tejon Ranch in recent years. Pig depredation occurs primarily in summer; historical records since 1984 show that about half of the 1362 depredation permit kills in the condor range occur in June, August and September, with the remainder spread evenly throughout spring, fall and winter.

Section IV. Potential Sources of Lead Exposure to Condors in California

There are four potential sources for lead in the environment that could impact condors: 1) lead fragments in carcasses of animals shot with conventional rifle or shotgun ammunition; 2) possible lead residues in calf carcasses used in the condor feeding program; 3) atmospheric lead, from smelter emissions, battery manufacturers, and global use of leaded gasoline, which has been drastically curtailed in most countries, but still used in at least 60 developing countries, mostly in the southern hemisphere; and 4) soil lead, including natural lead deposits, residual lead from previous atmospheric deposition, and lead in soils of land fills, dump sites, and rifle ranges;

IV. A. Hunter shot carcasses and gut piles

Game take data by county and hunt zone have been obtained from the CA DFG for hunting within the condor range. Cooperation from Doug Updike of the pig-hunting program, Russell Mohr of the deer program, and Brian Archuletta, Patrick Smith and Joe Bennett of USDA APHIS Wildlife Services (formerly Animal Damage Control), is greatly appreciated.

Hunting statistics on 35 species of birds and mammals are tracked by DFG. The mammalian species hunted in California, and the annual estimated kill are presented in Table 9 and Figure 24. Accurate and precise data on hunting are not easily obtained. Deer and wild pig hunting requires a license and tags. Tags are supposed to be filled out and returned to the DFG to document hunter success. These data are tabulated as “reported kill”. The DFG estimates, from freezer checks and other monitoring, that a proportion of tags are not returned, and reports an “estimated kill” for deer and wild pigs based on the expected proportion of tags not returned.

Table 9 gives the compiled data for statewide take of game in California. The data are from several sources, given in the footnotes. Data are primarily from 2000, although the fur-bearing data is from a ten-year average. The total statewide game take is reported as 395,243 mammals, of which 377,069 are taken by hunting. Archery is included in this take, but represents a very small fraction of the total take. Wildlife Services take is reported as 15,605 mammals, about 4% of the total take.

Table 9. Statewide reported take by species

species		trapping ^a	animal damage control ^b	hunting ^c	2000 take total
badger ¹	badger	26	153	350	529
beaver ¹	beaver	379	1343	200	1922
bobcat ^{1,2}	bobcat	763	96	1753	2612
gray fox ¹	gray fox	856	134	1241	2231
raccoon ¹	raccoon	857	2901	4388	8146
black bear ³	black bear		104	3038	3142
coyote ¹	coyote	995	9302	61084	71381
wild pig ³	wild pig		210	38040	38250
deer ³	deer		8	58783	58791
jack rabbit ³	jack rabbit		11	75445	75456
cottontail ³	cottontail		11	76081	76092
tree squirrels ³	tree squirrels		5	56686	56691
ground squirrels	ground squirrels		1327	??	??

¹ - from CDFG Draft Env. Doc. Regarding Furbearing and Nongame Mammal Hunting, Jan. 31, 2002.

These figures include take reported from the CDFG 2000 Game Take Hunter Survey, Wildlife Services, and CDFG Licensed Fur Trapper and Dealers Reports.

² - Data from 2000 Game Take Hunter Survey (1753), substituted for "Hunting (2000-01)" (414) in Draft Env. Doc.

³ - from 2000 Game Take Hunter Survey

^a - 10-year average

^b - CDFG Draft Env. Doc. Furbearing and Nongame Mammal Hunting, Jan. 31, 2002, App. 1-8: Wildlife Service +33%.

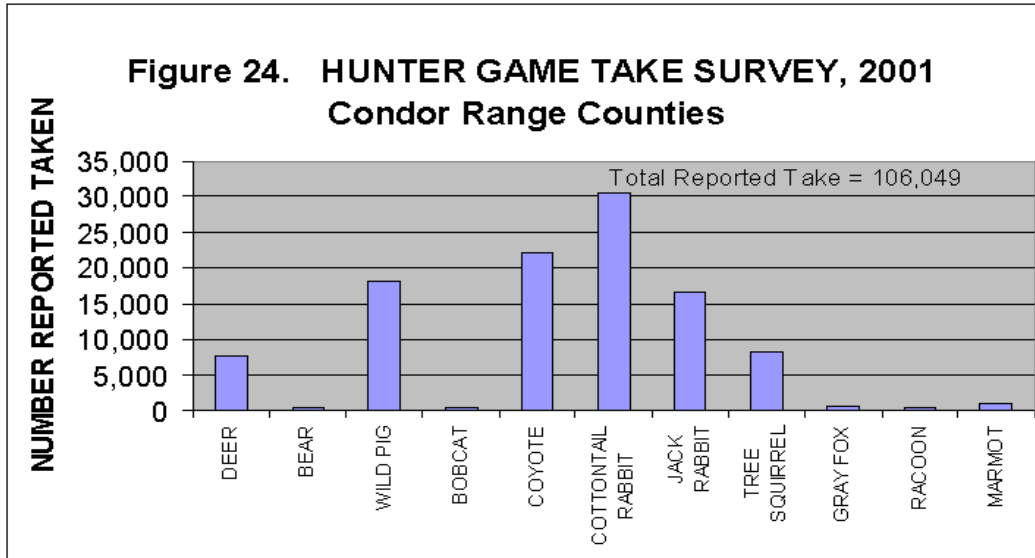
^c - from the CDFG 2000 Game Take Hunter Survey

^d - CDFG Draft Env. Doc. Regarding Furbearing and Nongame Mammal Hunting, Jan. 31, 2002, Appendix 2.

For the DFG annual Game Take Hunter Survey (GTHS), approximately 7 % of hunters are asked to anonymously fill out and return a survey form of game taken during the year. About half of these survey forms are completed and returned. The results of the GTHS are then extrapolated to the number of hunting licenses purchased by county. The Survey data are summarized in an annual DFG report, and data have been included here from the 2001 report. The DFG uses the GTHS numbers for its population modeling estimates and for game management decisions for most of the hunting program species. This dataset, although potentially flawed by inaccurate reporting, is thought to be the overall best estimate of game taken in the State.

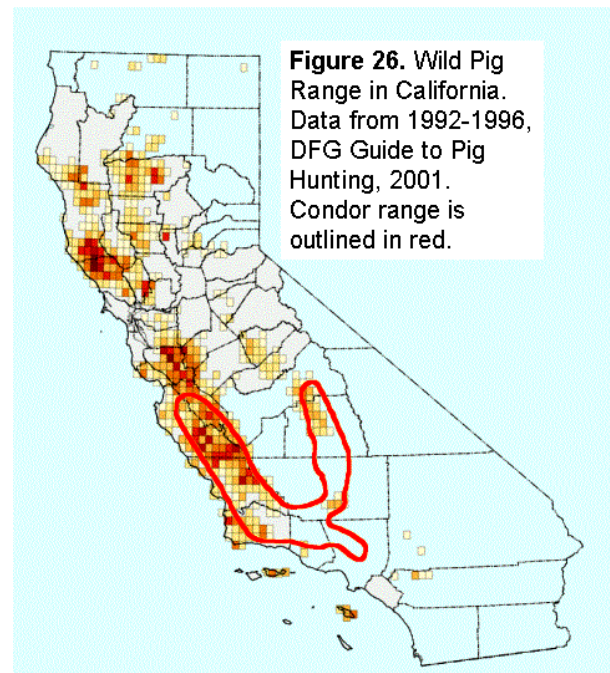
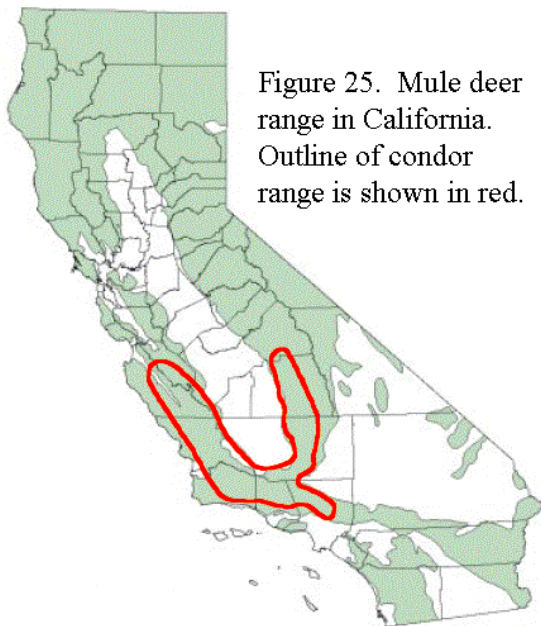
Figure 24 gives GTHS data for the eight counties encompassing the condor range. The total take for game mammals in these eight counties is reported as 106,049. Ground squirrel shooting is not included in the GTHS, and take of ground squirrels is not estimated. The lack of data on varmint hunting presents a significant data gap, as condors eat ground squirrels, and the potential for lead in these carcasses has not been investigated. Wildlife Services animal damage control numbers are only reported statewide, and are not included in this figure.

This report summarizes GTHS data from Monterey, San Benito, San Luis Obispo, Ventura, Los Angeles, Santa Barbara, Kern, and Tulare Counties. These eight counties are identified on the map in Figure 1, with the traditional condor range outline included. About half of Kern County lies outside the condor range, including parts of the Mojave



Desert and San Joaquin valley. Much of Los Angeles County lies outside the condor range, although hunting within the county is largely confined to areas included within the historic condor range, but condors now rarely use the eastern part of Los Angeles County. About half of Tulare County is within the condor range, and most of the land within the other counties is within the current condor range.

Data for deer and wild pigs for each of the counties has been included without making any adjustment for condor range within Kern and Tulare Counties, because the distribution of deer, pigs, and condors in each of these counties is nearly identical. California Mule Deer range is given in Figure 25 with the condor range superimposed on the map. Wild pig range in 1992-1996 is given in Figure 26.

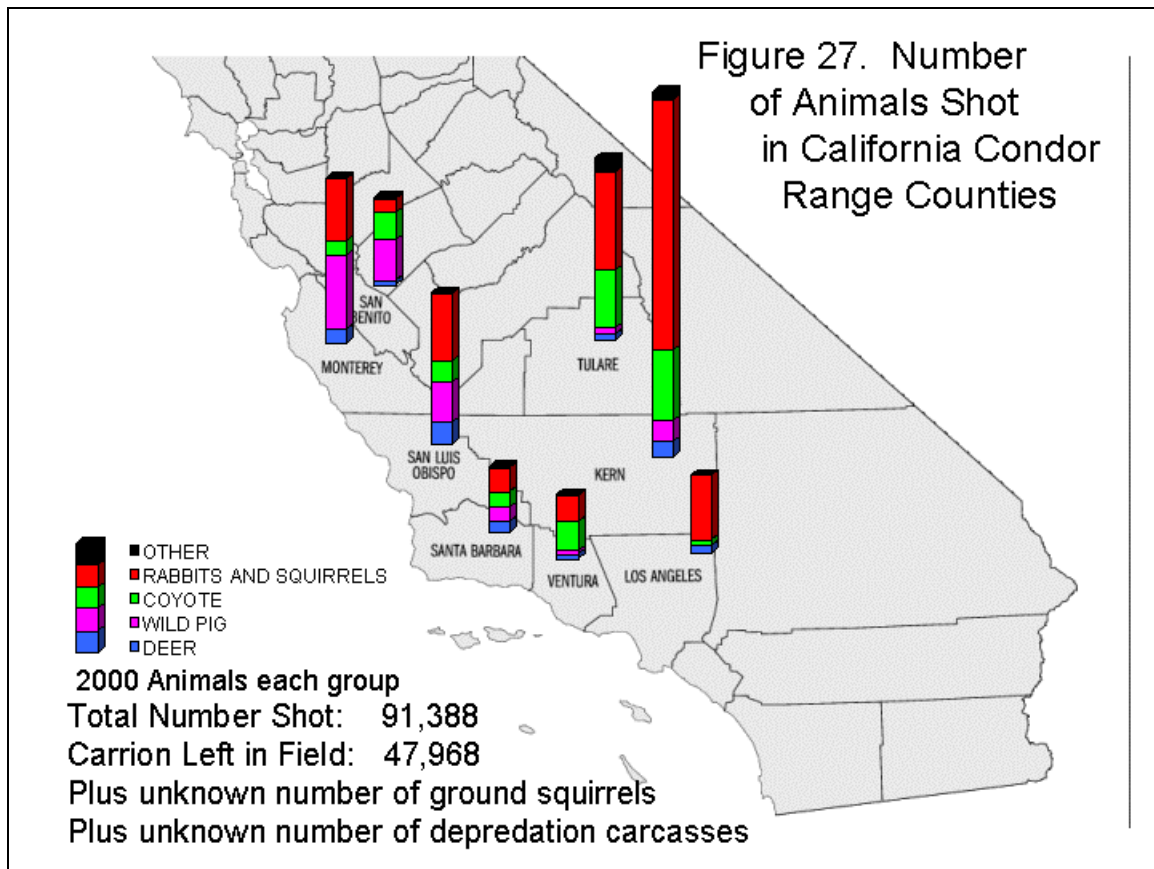


The annual deer take within the 8 condor range counties is estimated by the GTHS at 8,180. The take is graphed in Figure 27, along with other mammalian species.

Wild pig spot kill locations are reported on tag returns, but have not been mapped recently. The best location data for pig take is a four-year cumulative total tag return location map given in Figure 26. This map represents data from 1992-1996, and has also been used as the basis for the wild pig population distribution for the State. Within the condor range, the highest density of pigs is in the Coast Range counties of Monterey, San Benito, San Luis Obispo, and also Santa Clara County, which may become a range expansion with the release of condors at the Pinnacles National Monument this year.

The condor range has been superimposed on the wild pig distribution map in Figure 26. Virtually all of the pig distribution in the 8 condor counties coincides with the condor range. A significant expansion of wild pigs into the Tehachapi Mountains has occurred since this map was compiled in 1996. Pig hunting on the Tejon Ranch has increased greatly in the past 5 years.

The wild pig harvest for the 8 condor range counties represents an annual average of 17,249 animals, or 43% of the State total. The largest take in the condor range is in Monterey County, with 1,850 tags submitted, and the GTHS survey estimate of 6,488 pigs taken in the county. The data are graphed by county in Figure 27.



Data for coyote take is presented in Figure 24. The total hunter take in the 8 condor counties is 22,162 coyotes, excluding the take by Wildlife Services. Many of these animals are probably taken in the Central valley, outside of the condor range. The coyote range includes all of Kern and Tulare Counties, including the valley and desert, not just the foothills and mountains. The number of coyotes shot within the condor range is probably 1/3 of the total take for these two counties. The take within condor range for the other six counties is probably 2/3 of the county totals. The take within the condor range is estimated at 10,816, and is graphed in Figure 27.

USDA Wildlife Services uses specific guidelines for disposal of coyote carcasses within the condor range, based on two 1985 Memoranda: Within the USFWS (20-B-29: Allowable Animal Damage Control methods and their use restrictions in the range of the California condor, Portland OR, 1985 Sep 9, and 1-1-78-F(IT)72R: Final Biological Opinion on the effects of the California Animal Damage Control program on the California condor. Portland OR; 1985 Aug 26), “aerial shooting, and shooting from the ground, denning, snaring, and leg hold traps are allowable control methods as long as steel shot is used (aerial shooting) and/or animals killed by lead shot are buried or removed from the condor range.”

“Shot coyote carcasses are inspected for exit wound and if found, animals are left, if no exit wound is located carcass is either buried, dumped into a ravine, or removed from the range.” (pers. comm, Brian Archuleta, 16 Aug 2002).

The Wildlife Services take of coyotes is reported only Statewide, but an estimate of the number taken in the condor range can be made. The GTHS data for coyotes by county indicate that about 30% of the coyotes are taken in the 8 condor range counties. If that percentage is estimated for the Wildlife Services take, it is about 2700 coyotes annually within condor range. This represents about 16% of the hunter take of 22,162, a relatively small proportion. Wildlife Services currently examines each coyote carcass to minimize the lead exposure risk to condors. The WS numbers for coyote take are, therefore, not included in the hunter totals.

Ground squirrel hunting is not included in the data of the GTHS. Varmint hunters shoot many thousands of ground squirrels, with the majority left in the field for scavengers to consume. The Department should include ground squirrel hunting in the GTHS questionnaire, to assist in making an estimate of the extent of varmint hunting, and to make an estimate of the potential lead exposure to avian and mammalian scavengers consuming these carcasses.

Prior to 2000, permits were required for taking of all pigs depredating ranchlands, but the permit requirement has been relaxed, so that ranchers can take animals without a permit if pigs threaten their property. The reported pig depredation take since 2000 has averaged about 320 animals per year within the condor range, a small percentage of the hunter take.

The big game species of deer and wild pigs plus coyotes represent about 1/3 of the total number of animals shot annually within the condor range, but carcasses of these large animals would be the primary source of hunter-shot food for condors.

IV. A. 1. Hunters Leave Much Carrion in the Condor Range.

The most important question with regard to hunting and condors is: What numbers of carcasses and gut piles are left in the open by hunters in the condor range with the potential for lead ingestion by condors? The total hunter take of deer, pigs, and coyotes given in Figure 27 is 36,811 animals, excluding Wildlife Services take. Deer and pigs are generally field dressed and gut piles discarded in the field. Although a hunter education program for burying gut piles has been attempted within the condor range, DFG has made no estimate of the compliance by hunters, and this review assumes that only a very few of the gut piles are buried, hidden successfully, or removed from the field. Coyotes are generally left in the field, and this review estimates approximately 10,816 of the total 22,162 coyotes are shot in within the condor range.

The GTHS estimates that 55,365 tree squirrels, cottontail rabbits and jack rabbits are shot in the 8 condor range counties annually. No estimate is made of the number of ground squirrels. If 80 % of the trees squirrels and cottontails are taken from the field by hunters, and all of the jack rabbits are left in the field, the number of carcasses in the field of this group is estimated at 13,841. The number of ground squirrels may be at least this many.

Figure 27 compiles the GTHS data and makes estimates of the amount of carrion left in the field within the condor range. The number of carcasses and gut piles totals 36,811 big game remains (deer, pigs, and coyotes), and a total of at least 47,968 remains, plus an unknown number of ground squirrels and crippled or unrecovered deer and pig carcasses. No published data is available to estimate the prevalence of lead in carcasses and gut piles. One study is underway in Wyoming, but results are not available. If conventional lead rifle bullets are used, the prevalence may be relatively high, because lead bullets fragment upon hitting bone, and frequently leave fragments in the viscera and meat. If lead bullets continue to be used in condor habitat, a study of gut piles should be conducted. A similar study should be done on lead in varmint carcasses, both for high velocity center-fire and for rim-fire taken carcasses.

After all sources of mortality have been considered, and the number of hunter shot carcasses estimated, it must be concluded that gut piles and whole carcasses left in the field by hunters are a highly significant source of lead within the condor range. Because the use of gut piles as condor food has not been documented, and the extent of lead contamination in gut piles, ground squirrels, and unrecovered carcasses has not been investigated, this report cannot definitively state that these remains are the principle sources of lead exposure to condors. The alternative sources of lead within the condor range are discussed below, and appear to be less significant sources than lead fragments from hunting bullets.

IV. B. Other Sources of Lead in the California Condor Range

IV. B. 1. Dairy Calf Carcasses Used in Condor Feeding Programs:

Weimeyer et al (1983) (Table 10) reported on lead levels in animal carcasses fed to condors during the early years of the recovery program.

Percentage lipid and metal residues for foods of a captive California Condor at the Los Angeles Zoo and those given to wild California Condors in a supplemental feeding program.			
Food type*	% lipid of wet weight	METAL CONCENTRATION ppm WET WEIGHT	
		Lead	Mercury
<i>Captive condor foods</i>			
Horsemeat	4.5	0.43	— †
White rats – WB	4.3	1.8	0.027
<i>Supplemental feeding items</i>			
Domestic goat – M	0.7	0.10	—
Domestic goat – M	6.3	1.1	—
Domestic goat – M	2.1	0.21	—
Mule deer – M	0.3	0.34	—
Mule deer – M‡	3.3	0.43	—
Mule deer – F‡	75.5	0.77	—

*WB = whole body, skinned; M = muscle; F = fat.
 †None detected.
 ‡These two samples were obtained from the same carcass.

Table 10. Metal Residues in Food Items Supplied to Condors in 1981. From: Wiemeyer, S. N., A. J. Krynsky and S. R. Wilbur. 1983. Environmental Contaminants in Tissues, Foods, and Feces of California Condors. In, S. R. Wilbur and J. A. Jackson, eds., Vulture Biology and Management. UC Press. Chapter 28, p. 436.

Substantial quantities of lead were measured in white rats fed to condors at the LA Zoo (1.8ppm fresh wt, whole body) and in domestic goats being supplied to condors in the field (1.1 ppm in muscle). Since that time, carcasses of dairy calves have been used for the supplemental feeding program, in an effort to reduce the potential contamination of carcasses.

Dairy calf carcasses supplied to the condor program were analyzed in 1994, and found to have low lead levels (R. Riseborough, pers. com.) Over the past decade, however, disposal of sewage sludge containing heavy metal residues as fertilizer has become common practice, especially for growth of forage crops such as silage corn and bahia grass. Because dairies are dependent upon silage and hay as the primary source of food for cattle, it would be prudent to continually test dairy calves for metals. Following preliminary discussions with the condor program biologists, a testing program for dairy calf carcasses is underway. The best tissue to test would be liver samples from dairy calf carcasses when they are received from suppliers. Liver samples could be submitted to LSU, or UCD California Animal Health and Food Safety Laboratory (CAHFS) for

routine analysis. The CAHFS Heavy Metal Screen, Test 8071 analyzes for As, Cd, Fe, Cu, Hg, Mn, Mo, Pb, and Zn, at a cost of \$22.70 per sample.

[Note added in proof: Samples of dairy calf liver were submitted to CAHFS in January and February, 2003, and lead was not detected in any sample. Copper and zinc residues were also quantified, and found to be at high levels. Results of these analyses will be included in a supplemental report to be submitted at the conclusion of this study in April, 2003.]

IV. B. 2. Atmospheric Sources of Lead:

Atmospheric releases of lead have been reduced dramatically, first with the phased reduction in lead in gasoline from 1973-1986, and complete elimination of leaded gasoline for highway use in 1996. In 1996, the US atmospheric release of lead, primarily from smelters and battery manufacturers totaled 1,800,000 pounds (National Air Pollutant Emission Trends, 1900-1997, U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, 1998). The most recent data (US EPA Toxic Release Inventory (TRI), year 2000 data): US total air emissions: 1,485,833 pounds (260,040 lead, 1,225,794 lead compounds), and California: 9,301 pounds total (1,550 lead, 7551 lead compounds). All commercial users of lead who process or use more than 100 pounds of lead per year must report all emissions of lead and lead compounds to US EPA TRI (<http://www.epa.gov/tri/>). The 2000 data for air releases in California are given in Table 11, for all dischargers of greater than 100 pounds per year. The bulk of these are in the Los Angeles basin, with about 760 pounds total being discharged in Kings, Kern, and Fresno Counties.

Table 11. Lead Emissions to the Atmosphere in California, 2000. Data from USEPA Toxics Release Inventory	
Emissions in pounds per year	
Facility	Total Air Emissions
DELPHI AUTOMOTIVE SYS. ANAHEIM BATTERY PLANT, 1201 N. MAGNOLIA AVE., ANAHEIM, ORANGE	2198
MANNER PLASTIC MATERIALS, 3121 E. ANA ST., RANCHO DOMINGUEZ, LOS ANGELES	1099
EXIDE CORP., 2700 S. INDIANA ST., VERNON, LOS ANGELES	863
TAMCO, 12459 ARROW HWY. SUITE B, RANCHO CUCAMONGA, SAN BERNARDINO	652
QUEMETCO INC., 720 S. 7TH AVE., CITY OF INDUSTRY, LOS ANGELES	644
TROJAN BATTERY CO., 12380 CLARK ST., SANTA FE SPRINGS, LOS ANGELES	500
EXIDE CORP., 14500 NELSON AVE., CITY OF INDUSTRY, LOS ANGELES	316
EXIDE CORP., 5909 E. RANDOLPH ST., COMMERCE, LOS ANGELES	304
CHEMICAL WASTE MANAGEMENT INC., 35251 OLD SKYLINE RD., KETTLEMAN CITY, KINGS	255
FRESNO VALVES & CASTINGS INC., 7736 E. SPRINGFIELD, SELMA, FRESNO	255
RAMCAR BATTERIES INC., 2700 CARRIER AVE., CITY OF COMMERCE, LOS ANGELES	255
TROJAN BATTERY CO., 9440 ANN ST., SANTA FE SPRINGS, LOS ANGELES	255
KESTER SOLDER, 1730 N. ORANGETHORPE PARK, ANAHEIM, ORANGE	250
POLYONE CORP., 2104 E. 223RD ST., CARSON, LOS ANGELES	250
SAFETY-KLEEN (BUTTONWILLOW) INC., 2500 W. LOKERN RD., BUTTONWILLOW, KERN	250
TST INC. (DBA TIMCO) (DBA TANDEM INDS.), 11601 ETIWANDA AVE., FONTANA, SAN BERNARDINO	250
U.S. ARMY SIERRA ARMY DEPOT, SMASI-CO, HERLONG, LASSEN	250
Total lead air emissions of facilities releasing >100 lbs/yr:	8846
TOTAL US AIR EMISSION OF LEAD: 1,485,000 LBS/YR.	

California emissions of lead are relatively small, and outside the condor range. The prevailing winds in California are such that little lead released to the atmosphere in

California would be deposited within the condor range. Because of easterly prevailing on this continent, other US emissions are unlikely to reach the condor range. Atmospheric lead, therefore, does not now appear to be a significant source of lead to condors or other wildlife in the condor range.

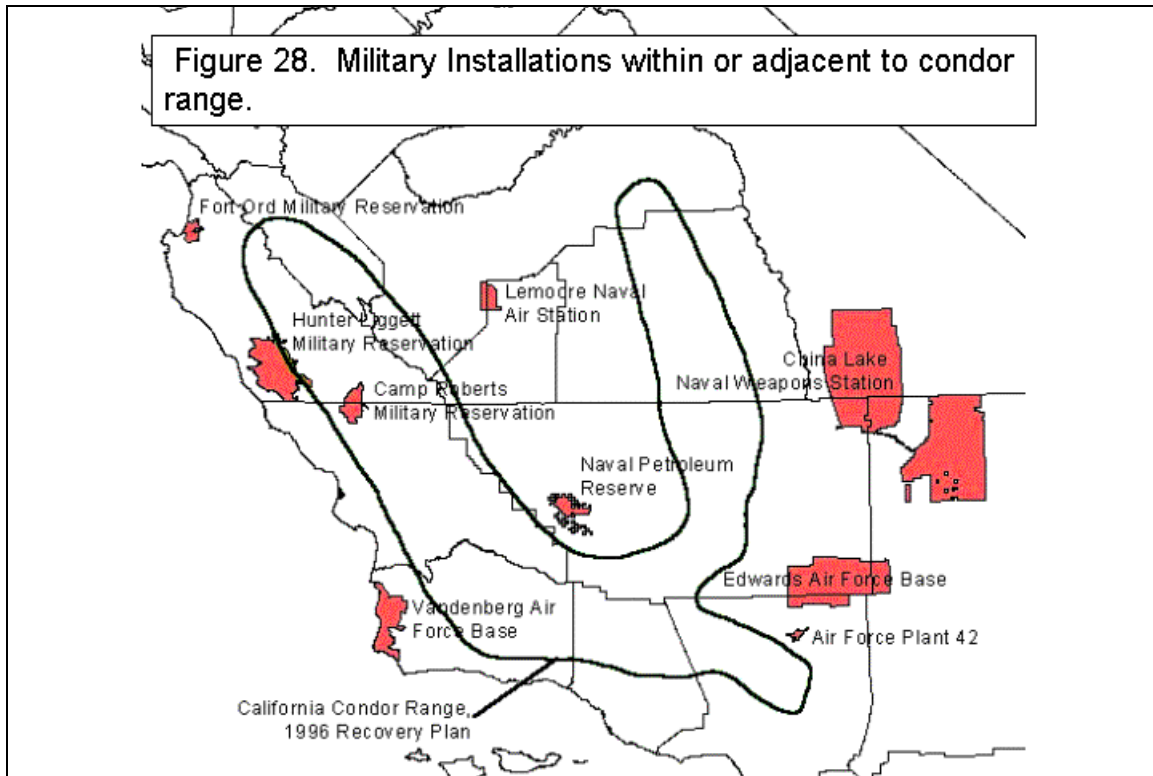
Atmospheric lead emissions from Mexico have also been reduced significantly since introduction of unleaded gasoline in 1990. The lead concentration in air samples in 1990 measured $1.2\mu\text{g}/\text{m}^3$, and by 1992 had been reduced to $0.2\mu\text{g}/\text{m}^3$. Blood lead levels in children also dropped significantly during the period 1990-1996 (Finkelman, 1996). The prevailing winds do not normally blow from Mexico into the condor range, and these data are given to indicate the source reduction throughout the continent.

IV. B. 3. Soil Lead Sources and Biologically Incorporated Lead in Wild Herbivores:

Soil lead derived from previous atmospheric deposition of lead during the approximately 50 year use of leaded gasoline is still present in the environment, and will continue to be recycled through the food web. Plant bioaccumulation and grazing by herbivores (both rodents and ungulates) would be the primary exposure risk to condors. The amount of lead available to condors in 1981 through this route was examined by Weimeyer et al. (1983, 1986) who sampled carcasses of deer, cattle and sheep. They found residues of from non-detect (0.05 ppm fresh wt) to 17.5 ppm in one sample of deer muscle. The deer muscle sample was questioned, because it was a deer head that had been frozen and stored for 6 months in a public freezer before sampling. Cow placenta averaged 0.66 ppm (range: 0.24-1.82 ppm). Current levels should be somewhat lower than samples taken in 1981, during the phase-out of leaded gasoline, but previous deposition could still be present. Leaded gasoline residues would be expected to be highest adjacent to major highways, and in the air shed of the Los Angeles basin. The present levels should be addressed by analysis of animals and plants in specific areas of the condor range. These remain a likely source for low-level lead exposure to condors. Weimeyer et al. (1986) assessed the potential risk from biologically incorporated lead in condor food sources, and concluded the risk at that time was minimal. Since that report leaded gasoline has been eliminated, thereby lowering the primary potential source for this route of exposure.

IV. B. 4. Localized Soil Lead as an Environmental Hazard to Condors.

Localized lead sources in soil are limited in the condor range in California. There have been eight mines producing and refining lead in California, and all are east of the condor range in the Sierras or Mojave Desert. Other sources of soil lead could include public outdoor rifle ranges, and almost every municipal government in California has had an outdoor police target range in the past. No complete listing of shooting ranges is available. The largest installations in the condor range are military reservations, and these are mapped in Figure 28. The two of greatest potential as sources of environmental lead are Fort Hunter Liggett and Camp Roberts.



Fort Hunter Liggett has an 11,000 acre “impact zone” that has been used continuously since 1940 for small arms and artillery training. The zone is off limits to personnel, because of unexploded ordinance, and has not been assessed for the extent of lead or other metal contamination. Camp Roberts also has an impact zone, and small arms ranges, although its size was not determined in this study. Fort Hunter Liggett has a supervised hunting program for military personnel and civilian hunters. The possible soil lead risk to condors stems from the bioaccumulation of lead in plants and the localized food chain transfer into grazing herbivores, including rodents, deer, wild pigs, and elk. Only indirect data are available for this risk.

The US military has been conducting studies to evaluate methods of environmental cleanup of small arms ranges, including assessments of “phyto-remediation” of sites by growing, harvesting, and disposing of plants that bioaccumulate lead from the soil. Studies published by Behel (1998) indicate that plants can accumulate substantial quantities of lead, although greenhouse studies and field studies have produced significantly different results.

Greenhouse studies of Behel et al. (1998) demonstrated that white mustard could accumulate as much as 1.5% lead on a dry weight basis in foliage harvested from a highly contaminated soil, if the soil was treated to make the lead bioavailable.

Field studies were conducted at the Twin Cities Army Ammunition Plant (TCAAP) in Minnesota, with mustard grown on two sites, one averaging 2,610 ppm lead, and the

other with 358 ppm lead. The field trial with corn and mustard produced crops with lower concentrations of lead (corn: 0.65% and 0.13% by dry weight; mustard: 0.083% and 0.034% by dry weight), and poor results were attributed to sub optimal growing conditions, and unsuitable soils for the plant species used. Even with sub optimal growing conditions, the field study on a site containing bullet jackets and copper scrap produced corn plants with 0.65% lead, at a corn yield of 7.4 to 12.6 tons/acre. This yield resulted in bioaccumulation into plants of 96.2-163 pounds of lead per acre. One major factor that could limit lead incorporation by plants is the availability of metallic lead to plants. In Behel (1998), areas of contaminated soil were treated with mild acid to solubilize the lead, followed by application of EDTA (ethylene-diamine-tetra-acetate, a metal chelator) to the soil to stabilize the lead into a form that could be accumulated by plant roots. Data were not presented to compare lead incorporation into plants without these treatments. Soil conditions, the native geology, soil pH, and rainfall all could influence the availability of lead from soils, and considerable research would be needed to evaluate the exposure potential of lead bioaccumulated by plants in area with lead contaminated soils.

It is unknown whether plants within the impact zones on military sites within the condor range could produce equivalent yields, although mustard is a very common plant in disturbed ecosystems in California. Rodents and other herbivores living in the area would graze upon the plants growing on abandoned rifle ranges and in impact zones. This could be a source of biologically incorporated lead available to condors foraging on wild carcasses or gut piles in these areas, although the concentrations of lead in the rodents and other herbivores would probably be low. Measurements of biologically incorporated lead in game shot on Fort Hunter Liggett could provide important data for estimating this route of potential lead exposure. The potential human health impact of biologically incorporated lead should drive this research, as hunters would be expected to consume meat from the animals taken from this installation. Condors use Fort Hunter Liggett for foraging, with a condor observed feeding on a yearling elk in April 2002.

Field studies should also be conducted on abandoned or active ranges with rodent populations feeding on vegetation growing on surface soils containing particulate lead and copper. If the phytoremediation data cited in this report reflect potential bioaccumulation under California field conditions, the lead in plants could pose a small risk through lead bioaccumulation in rodents and other herbivores foraging in areas with lead in the soil. The City of Davis has an abandoned firing range with a high population of rodents, and is willing to consider a monitoring study.

IV. C. Comparative Exposure Hazard from All Lead Sources

Atmospheric lead does not appear to pose a substantial risk to wildlife or humans in California at this time. Weimeyer et al (1986) concluded biologically incorporated lead throughout the condor range posed low risk. Atmospheric lead from leaded automobile exhaust has decreased dramatically since the progressive ban on leaded fuel in the 1980s and 1990s. Industrial sources of atmospheric lead emissions are not within the air shed of the condor range in California.

The amount of lead that could be biologically incorporated into herbivores from lead in the soils such as rifle ranges or on Fort Hunter Liggett is unknown, and should be evaluated, both for an assessment of the human health risk from eating deer or pigs that have foraged on plants growing on soils containing lead, and for condors foraging on that installation. The Ventana condors have averaged lower blood lead levels than the Hopper birds, and this may indicate that biologically incorporated lead from soils at Fort Hunter Liggett has not been a significant source of lead to condors.

The frequency and amount of lead in game and varmint carcasses and gut piles left in the field has not been studied, and is a major data gap for determining the source(s) and amount of lead exposing California Condors. It is highly probable that some proportion of carcasses and gut piles contain lead fragments, and that scavengers, including condors, quickly consume these remains. Research and monitoring to determine the frequency and amount of lead in ground squirrel carcasses and gut piles of deer and pigs should be conducted as a high priority. Gut piles need to be evaluated by X-ray, and better data are needed on wild feeding incidents by condors, to be able to quantify this potential lead exposure source.

When all potential sources of lead in the environment are compared, carcasses of ground squirrels, coyotes, deer and wild pigs, and the gut piles of deer and wild pigs appear to be the most likely sources of lead exposure to condors. Because lead exposure is currently the most difficult management issue for maintaining the health of free-flying condors, elimination of these as sources of lead exposure should be attempted on a precautionary principle basis. If future research and monitoring studies demonstrate that these remains do not present a lead exposure hazard, either to condors or other protected scavengers, the efforts to prevent exposure could be relaxed.

Section V. Minimizing Risk of Lead Ingestion from Carcasses in the Field.

Several programs could be initiated by regulatory agencies and other interested parties to minimize the number of lead containing carcasses left in the field in condor range:

1) Initiate an intensive hunter education program emphasizing the risk to scavengers from lead fragments in hunter killed game or carrion left in the field. When hunters use lead bullets, the education effort should emphasize burial or removal of gut piles from the field, rather than hiding gut piles under brush or in steep ravines. Carcasses should be examined for an exit wound, and the bullet removed from the carcass if it is still present. Gut piles should be thoroughly examined for bullet tracts through viscera, and any bullet-damaged portions should be buried or removed from the field. Scavengers readily locate gut piles by smell, and large mammalian scavengers (bears, coyotes, and foxes) may drag gut piles into the open where visual scavengers such as ravens, vultures and condors may find them.

2) Initiate a program working with specific military installations and Private Lands Management Units to require hunters to remove gut piles from the field.

Fort Hunter Liggett and Camp Roberts both have supervised hunting programs for military and civilian personnel. Access to the bases is highly regulated, and DFG personnel assist in the hunter programs. At the current time, no program to bury gut piles is in place at Fort Hunter Liggett. Fort Hunter Liggett discourages burial of gut piles, because the ground is stony, and there is the possibility of disturbance of archeological sites. This policy insures that gut piles will be left in the field, and available to scavengers, even if the gut piles are hidden under brush or placed in a steep ravine. Because Fort Hunter Liggett is one of the most heavily used foraging areas for condors, this policy presents an unnecessary exposure risk to condors. Camp Roberts is within the condor range, but no use by condors was detected in the VWS overflight survey. While the current use of Camp Roberts appears low, the base is within 30 miles of known condor nesting areas, and could receive use in the future, especially if the flight patterns of condors change when birds are released at the Pinnacles National Monument in 2003.

These military bases and the Tejon Ranch have secure access, and it would be possible to issue all hunters with a body bag, and instruct them to return the gut pile to the check out station when exiting with a successful kill. Gut piles could be X-rayed as part of a survey of lead frequency in gut piles, and subsequently discarded by the DFG or military in normal garbage, and taken to the county or military base landfill. If a hunter attempted to check out a deer or pig without a gut pile, he could be required to go back and get it. This would completely eliminate the potential gut-pile-lead-fragment hazard from two frequently used condor foraging areas.

Concurrent with a program to remove gut piles from the field, the DFG should begin a monitoring program of gut piles turned in by hunters to evaluate the lead exposure hazard. Fresh or frozen gut piles should be examined by X-ray to determine the frequency and amount of lead in the viscera of hunter killed deer and pigs. The cost of

such a program would be minimal if the Tejon Ranch and Fort Hunter Liggett would participate, and would provide much needed data on the frequency of lead in gut piles. The program would also eliminate, in a precautionary way, the potential exposure of condors to gut piles on the Tejon Ranch and Fort Hunter Liggett, two of the most important foraging areas for condors in California.

Outreach plans are in place at the Carrizo Plains Natural Area (CPNA) to encourage hunters to minimize the risk to Condors. CPNA was a traditional forage area for wild condors until 1987, but current radio-telemetry data suggest that it is not heavily used. The CPNA website (www.ca.blm.gov/bakersfield/carrizoplain.html) provides this description of the plans:

“As land was acquired in the CPNA, recreational hunting became more significant because of the new availability of public land and because of improved access. The most popular hunting areas have included the Temblor and Caliente Mountain Ranges with emphasis on wild pig, tule elk and upland game birds. Hunting is allowed and encouraged in the CPNA and will continue to be one of the principal recreational uses. However, the use of lead bullets in hunting big game, particularly wild pigs, could present a potential impact to the California condor from lead ingestion while feeding on carrion. Outreach efforts will stress the hazards of lead in animal remains. Hunter information meetings, sportsmen's clubs, and other related gatherings offer opportunities to explain the condor recovery program and emphasize techniques for preventing the loss of condors from lead poisoning.”

3) Replacement of lead containing ammunition with ammunition that will not release lead fragments into the target animal carcass. Small game taken with a shotgun should use steel or tungsten composite shot (Hevishot®, or other comparable ammunition). Hunters should use commercially available rifle ammunition such as the Barnes X® bullet, or the Failsafe® bullet marketed by Winchester. The Failsafe® bullet contains lead, but encapsulated in a steel jacket within the copper bullet, designed so that the lead will not fragment, and the lead surface will not be exposed. Tests to confirm this should be performed on this bullet, or data obtained from the manufacturer. Ingestion of the steel encapsulated lead portion of this bullet should not result in dissolved lead in the stomach and blood stream. I believe the Failsafe® bullet, if it performs as described, could be termed “lead-safe”.

The best and most immediate solution to the lead exposure risk to condors would be the voluntary use of Barnes X® or Failsafe® bullets by deer, pig and coyote hunters. This has been recommended by Jim Matthews, editor of *California Hog Hunter*, and in his syndicated outdoor newspaper column. Mr. Matthews also co-leases Tejon Ranch Hog Hunts, and actively promotes the use of “gut pile safe” ammunition for all of their hunts. Because these and other organized wild pig hunts are held on the Tejon Ranch 15 times a year, from December to July, this is an important measure to assist in preventing lead exposure to condors. If lead free bullets cannot be completely eliminated from the Tejon Ranch, hunters should remove gut piles from the field.

At the present time, no lead-free replacement for .22 caliber rim fire lead ammunition is available on the market, other than indoor target loads. This poses a real dilemma, because no data could be found documenting the amount and frequency of lead remaining in ground squirrel carcasses after being taken with .22 caliber rifles, and therefore, the exposure hazard to condors and other scavengers has not been determined.

4) A significant number of wild pigs are shot in California on depredation permits, with an unknown number of carcasses not recovered from the field. Because farmers, ranchers, and landowners with depredation permits are controlled by the CA DFG, the Department should encourage or require all permit holders to use lead free ammunition (Barnes X® or Failsafe® bullets) for taking of wild pigs. All landowners who take pigs without a depredation permit should be actively sought out and encouraged to use only lead-free or “lead-safe” ammunition.

Section VI. Summary and Conclusions:

Reintroduced California Condors now occupy most of the range within California that they used during the period 1960-1987. Range expansion into Monterey County has occurred with the reintroduction near Big Sur.

Condors have suffered a high mortality rate in the wild. During the 1970s and 1980s approximately half of the known population disappeared from the wild, with many of the carcasses never found. During that period, at least 3 condors died of lethal exposure to lead. All remaining condors were trapped and kept in captivity until a reintroduction program was begun in the early 1990s. Since 1995, more than 140 condors have been released in California and Arizona, and 44 are free-flying in California at the current time. Four condors have died of lead poisoning since 1997 (1 in California, 3 in Arizona), and 8 condors in California and 18 condors in Arizona have received emergency chelation treatment to reduce toxic lead levels. The mortality caused by lead ingestion would have been considerably higher without expert veterinary care.

Seventeen of the condors examined by pathologists have not been tested for lead exposure. High-resolution lead and copper analysis techniques should be conducted on growing feather follicles of archived condors to determine the extent of lead and copper exposure in all condors that have died and been recovered since 1982.

Experimental studies with lead intoxication of other species of raptors indicate that condors are not more sensitive to lead intoxication than most species of birds. Turkey Vultures, however, are very resistant to the effects of lead, and are not a good surrogate species to compare with condors. Condors and Turkey Vultures also apparently have very different responses to ingested copper. Copper levels in the livers of condors analyzed at necropsy have all exceeded 20 ppm (21-181 ppm), although the significance of copper residue levels is unknown. Copper levels should be studied in feathers, to determine whether a relationship exists with lead exposure.

Field biologists have carefully studied lead exposure in condors since 1995, when the current reintroduction program was initiated. In California, every bird has had detectable lead in blood samples. 62% of the blood samples taken in Southern California have shown higher than background lead exposure (blood levels greater than 20 µg/dl blood). 13 of 87 samples (15%) have exceeded 60µg/dl, indicating the condors were clinically affected by lead, and 6 birds have had blood lead levels greater than 100 µg/dl, indicating acute toxic exposure and requiring emergency veterinary intervention. Lead exposure and intoxication remain a very critical problem for both the California and Arizona populations.

Hunting within the condor range in California is extensive, and the number of animals taken by hunters in 2001 was estimated at 106,049 for the eight counties within the condor range. This review estimates the number of large animal carcasses and gut-piles left in the field may be more than 35,000 annually. This includes the gut piles of 8,180 deer, 17,249 wild pigs, and the carcasses of 10,816 coyotes, plus the carcasses of deer

and pigs not recovered after being shot. The number of ground squirrels shot within the condor range is unknown, but probably represents a significant lead hazard to both condors and to other scavenging birds and mammals. Every effort should be made to educate hunters and depredation permittees of this problem, and to encourage or require the use of commercially available lead-free and “lead-safe” ammunition.

When all potential sources of lead in the environment are compared, carcasses of ground squirrels, coyotes, deer and wild pigs, and the gut piles of deer and wild pigs appear to be the most likely sources of lead exposure to condors. However, the frequency and amount of lead in game and varmint carcasses and gut piles left in the field has not been studied, and is a major data gap for determining the source(s) and amount of lead exposing California Condors. It is highly probable that some proportion of carcasses and gut piles contain lead fragments, and scavengers, including condors, quickly consume these remains. Because lead exposure is currently the most difficult management issue for maintaining the health of free-flying condors, studies to determine the amount of lead in carcasses and gut piles are urgently needed.

This review makes recommendations based on incomplete information, as many aspects of condor foraging and habitat use are either unknown or could not be easily obtained from field notes. Condor field note data on “mystery crops” and observations of “wild” feeding events need to be systematically organized and analyzed to document the potential exposure from feeding on hunter-shot carrion.

The data gaps pertaining to condors feeding on gut piles and the frequency of lead fragments in carrion result in considerable uncertainty in assessing potential lead exposure to condors, and therefore present a difficult decision process for wildlife managers and the condor program. Because of that uncertainty, the recommendations made in this review follow a precautionary principle, recommending protection and elimination of potential lead sources, even though conclusive data on lead in some potential sources is lacking. It is important to collect the necessary data to be used in decision-making, but in the interim it is important to protect the small and highly vulnerable condor population.

Section VII. Recommendations:

This report makes several recommendations for managing the risk of lead exposure to California Condors, for evaluating possible lead sources, and assessing the historic exposure of condors to lead. These have been separated into Action items, Archive studies to evaluate unknowns in Condor carcasses, Field Monitoring Programs, and Research studies.

VII. A. Action Items:

1) Work with Fort Hunter Liggett and the Tejon Ranch to develop programs to manage gut-piles so they are not a potential risk to condors.

Fort Hunter Liggett (FHL) and the Tejon Ranch (TR) present a large potential for lead exposure to condors from a relatively small number of hunter-shot deer and wild pig carcasses, because these areas are used as forage areas by condors, and they are in close proximity to the densest congregations of condors.

Actively seek the cooperation of FHL and the TR to participate in a study to quantify lead in gut piles, by having hunters bag gut piles and remove them from the field, so that they could be X-rayed by DFG to determine the frequency and amount of lead in viscera (Recommendation 6). This program would serve two immediate purposes: 1) to acquire needed data on lead in gut piles; and 2) to prevent potential lead exposure of condors in a precautionary manner. Long-term management of gut piles should be based on the results of the study.

A combination of long-term programs could be initiated, including removal of gut piles from the field, encouraging hunting with lead free or lead safe ammunition only, and/or education to insure that all hunters remove all lead bullets and fragments from gut piles or carcasses left in the field. Lead free or non-toxic ammunition would be the preferred solution.

A study of lead in ground squirrel carcasses could also be conducted on the Tejon Ranch. If cooperating varmint hunters would collect ground squirrel carcasses, bag them and identify the type of ammunition used, an X-ray study could be conducted to determine the lead exposure hazard from ground squirrels. This study would be of importance to both condors and other scavenging wildlife.

2) Conduct active hunter education programs to “get the lead out” of all game carcasses and gut piles left in the field.

The USFWS, CA DFG, and other agencies having jurisdiction over hunting or access to hunting areas in California should actively conduct this program. Cooperation should be sought with sportsman’s groups and other interested parties to make this a broad coalition effort gaining the widest acceptance.

3) Lead free or “lead-safe” ammunition should be widely available for hunting throughout the Condor Range.

Efforts should be made by Agencies to encourage hunters and landowners with depredation permits to use lead free ammunition such as the copper Barnes X® bullet, or the Failsafe® bullet, which contains lead, but encapsulated in steel with a copper jacket, so the lead will not be exposed. Encourage retail ammunition dealers to stock supplies of these bullets.

Replacement of lead containing bullets would be the surest way of reducing lead containing carrion within the condor range. The thousands of carrion items annually left in the condor range potentially pose a high risk to condors, eagles, and other wildlife.

VII. B. Archive studies to evaluate unknowns in Condor carcasses:

4) Examine lead residues in growing feather follicles of all condors that have died since 1980. Determine the exposure of condors to lead during the months prior to death.

A research study should be instituted to measure at high resolution the lead content of blood-feather follicles growing at the time of death of all condors that have died since 1980, which are in possession of the USFWS or with access of the Agency. The uncertainty of cause of death of many birds, and the absence of toxicological data for those birds scavenged in the wild may have resulted in an underestimation of the contribution of lead exposure in the deaths of condors. Ruling out lead as a contributing factor would be as important as confirming lead as contributing. Development of methods for this study is Recommendation 10.

5) Measure feather copper levels in archived samples of California Condors.

A study should be instituted to measure the copper deposition in feathers of condors that have died, and to compare the depositional trends in single feathers with lead levels. Direct comparison of copper and lead trends in single feathers, especially of condors with high copper levels, would provide data on copper and lead toxicokinetics. This study should be done in conjunction with a study of liver metallothioneine levels in archived condor tissues.

VII. C. Field Monitoring Programs:

6) Initiate a study to quantify lead in gut piles of deer and wild pigs shot by hunters on the Tejon Ranch and Fort Hunter Liggett. Evaluate lead residues in ground squirrels.

The Department should conduct a study of the prevalence of lead fragments in gut piles of hunter shot game. This survey could be accomplished in conjunction with Recommendation 1., with the cooperation of the Tejon Ranch and Fort Hunter Liggett, if

a policy of removal of gut piles from the field were instituted as a precautionary measure to minimize the exposure risk to condors. Fresh or frozen gut piles should be X-rayed to identify lead fragments, and these data would provide answers to one of the most important questions of possible lead exposure to condors. Varmint hunters on the Tejon Ranch (and other areas, such as the annual Surprise Valley shoot) should be encouraged to recover and turn in ground squirrel carcasses for an X-ray study of lead in varmint carcasses.

7) Monitor food-chain bioaccumulation of lead in deer, elk, and wild pigs shot at Military reservations

A monitoring program for lead in deer and wild pigs shot at Fort Hunter Liggett should be conducted to determine both the ecological risk, and the risk to humans consuming liver and meat of animals shot on this base. The DFG is currently taking samples of tongue from many of the deer harvested on Fort Hunter Liggett, and this study could be conducted in conjunction with that program.

8) Compile wild feeding and mystery crop data from field notes.

Analysis of data already collected in field notes could provide much needed information on rates and general locations of wild feeding. Combining this data with satellite or conventional radio telemetry could potentially identify locations of wild carcasses. If field notes were routinely entered into pocket PCs, the analysis could be greatly facilitated.

9) Include ground squirrel hunting in the Game Take Hunter Survey.

The Department should include ground squirrel hunting in the GTHS questionnaire, to assist in making an estimate of the extent of varmint hunting within the condor range, and to make an estimate of the potential lead exposure to all avian and mammalian scavengers consuming these carcasses.

VII. D. Research Studies:

10) Develop the sampling and analysis methods to quantify lead and copper incorporated into condor feathers during feather growth.

This study would provide the basis for the evaluation of all archived condor carcasses for measurement of lead exposure. The analyses would also enable evaluation of changes in circulating levels of lead in the blood of exposed birds. This study could provide information on: 1) Time of lead exposure relative to death of individual birds; 2) “Background” lead exposure and frequency of exposure events in birds that died from other causes; and 3) Evaluation of isotope ratios in feathers from acute lead exposure, and comparison with chronic “background” exposure to determine whether the sources of lead are different. Comparison of copper and lead residues could provide data on probability of copper exposure through ingestion of copper-jacketed bullet fragments.

11) Evaluate lead accumulation in plants and rodents on rifle ranges.

Field studies should be conducted on abandoned or active ranges with rodent populations feeding on vegetation growing on surface soils containing particulate lead and copper. The phytoremediation data cited in this report should be verified at California sites. The City of Davis has an abandoned firing range with a high population of rodents, and is willing to consider a monitoring study.

12) Evaluate lead in plants, rodents, deer, wild pigs, and coyotes throughout the condor range.

A study of the extent of lead exposure to terrestrial wildlife in California should be undertaken to determine the extent of atmospheric or residual environmental lead present in the ecosystem from industrial and vehicle emissions. A comparison with data from the 1970s and 1980s should provide environmental data on reduction of lead in the environment.

Section VIII. References

- Agency for Toxic Substances and Disease Registry (ATSDR). 1999. Toxicological Profile for Lead. U.S. Department of Health and Human Services. Public Health Service July 1999
- Agency for Toxic Substances and Disease Registry. 1990. Toxicological Profile for Copper. Agency for Toxic Substances and Disease Registry. U.S. Public Health Service December 1990
- Behel, A.D., R.A. Almond, D.A. Kelly, P.A. Pier, W.J. Rogers, and D.F. Bader. 1998. Results of a Greenhouse Study Investigating the Phytoextraction of Lead from Contaminated Soils Obtained from the Sunflower Army Ammunition Plant, Desoto, Kansas. Report No. SFIM-AEC-ET-CR-98036; August 1998.
- Bleich, V. C., and T. J. Taylor 1998. Survivorship and cause-specific mortality in five populations of mule deer. *Great Basin Naturalist* 58(2): 265-272.
- Bloom P H; Scott J M; Pattee O H, and Smith M R. Lead contamination of golden eagles *Aquila chrysaetos* within the range of the California condor *Gymnogyps californianus*. Meyburg, B.-U. & Chancellor R. D. Eds. *Raptors in the modern world*. World Working Group on Birds of Prey and Owls. Berlin, London & Paris; 1989; pp. 481-482; 611 pp.
- Bremner, I. 1979. Copper toxicity studies using domestic and laboratory animals. Pages 285-306 in J. O. Nriagu, editor. *Copper in the environment. Part 2: health effects*. John Wiley, New York.
- Bryan, G. W., and W. J. Langston. 1992. Bioavailability, accumulation and effects of heavy metals in sediments with special reference to United Kingdom estuaries: a review. *Environmental Pollution* 76:89-131.
- Carlton, W. W., and W. Henderson. 1964. Studies on the copper requirement of growing chickens. *Avian Diseases* 8:227-234.
- Chamberlain A, Heard C, Little MJ, et al. 1978. Investigations into lead from motor vehicles. Harwell, United Kingdom: United Kingdom Atomic Energy Authority. Report no. AERE-9198. 1979. The dispersion of lead from motor exhausts. *Philos Trans R Soc Lond A* 290:557-589.
- Cogan, Christopher Burnell. Quantitative analysis of habitat use by the California condor: University of California, Santa Barbara; 1993.
- Collins, Paul W; Snyder, Noel F. R, and Emslie, Steven D. Faunal Remains in California Condor Nest Caves. *Condor*. 2000 Feb; 102(1):222-227.

- Custer, T. W., and W. L. Hohman. 1994. Trace elements in canvasbacks (*Aythya valisineria*) wintering in Louisiana, USA, 1987-1988. *Environmental Pollution* 84:253-259.
- Eisler, R. 1988. Lead hazards to fish, wildlife, and invertebrates: a synoptic review. U.S. Fish Wildl. Serv. Biol. Rep. 85(1.14). 134 pp.
- Eisler, R. 1998. Copper hazards to fish, wildlife, and invertebrates: a synoptic review. U.S. Geological Survey, Biological Resources Division, Biological Science Report USGS/BRD/BSR--1998-0002. 98
- Ensley, Philip K. Medical management of the California condor. Miller, R. Eric and Fowler, Murray E. *Zoo and wild animal medicine: current therapy*. 4th ed. Philadelphia, London: W.B. Saunders Company; 1999; pp. 277-292.
- Finkelman, J. Phasing out Leaded Gasoline Will Not End Lead Poisoning in Developing Countries. *Environmental Health Perspectives* 104(1): 10-11.
- Griffin TB, Couiston F, Wills H. 1975. [Biological and clinical effects of continuous exposure to airborne particulate lead.] *Arh Hig Toksikol* 26:191-208. (Yugoslavian)
- Hoffman, David J.; Pattee, O. H.; Wiemeyer, S. N., and Mulhern, B. Effects of Lead Shot Ingestion on g-aminolevulinic Acid Dehydratase Activity, Hemoglobin Concentration, and Serum Chemistry in Bald Eagles. *Journal of Wildlife Distribution*. 1981; 17(3):423-431.
- Janssen, D. L.; Oosterhuis, J. E.; Allen, J. L.; Anderson, M. P.; Kelts, D. G., and Wiemeyer, S. N. Lead-Poisoning in Free-Ranging California Condors. *Journal of the American Veterinarian Medical Association*. 1986 Nov 1; 189(9):1115-1117.
- Jurek R M. An historical review of California condor recovery programmes. *VULTURE NEWS*. 1990; 23:3-7,
- Kiff, Lloyd F; Mesta, Robert I, and Wallace, Michael Phillip. California condor recovery plan. Portland, OR: U.S. Fish and Wildlife Service; 1996 Aprix, 62 p.
- Koford, Carl B. *The California condor*. New York, Dover Publications; 1966; c1953xiii, 154 p.
- Koford, Carl B. The natural history of the California condor (*Gymnogyps californianus*): Univ. of California, Berkeley; 1950 Jun464 l. illus.
- Kramer, J. L., and P. T. Redig. 1997. Sixteen years of lead poisoning in eagles. 1980-1995. An epizootiologic view. *Jour. Raptor Research* 31: 327-332.

Lloyd RD, Mays CW, Atherton DR, et al. 1975. 210Pb studies in Beagles. *Health Phys* 28:575-583.

McMillan, Ian I. *Man and the California condor; the embattled history and uncertain future of North America's largest free-living bird.* New York: Dutton; 1968;191 p

Meretsky, Vicky J; Snyder, Noel F. R; Beissinger, Steven R; Clendenen, David a, and Wiley, James W. *Demography of the California Condor: Implications for Reestablishment.* *Conservation Biology.* 2000 Aug; 14(4):957-967.

Miller, Alden Holmes; McMillan, Eban, and McMillan, Ian L. *The current status and welfare of the California condor.* New York: National Audubon Society; 1965 61 p., Morgan A, Holmes A, Evans JC. 1977. Retention, distribution, and excretion of lead by the rat after intravenous injection. *Br. J. Ind. Med.* 34:37-42.

National Academy of Sciences (NAS). 1977. *Copper. Committee on Medical and Biologic Effects of Environmental Pollutants, National Research Council, National Academy of Sciences, Washington, D.C.* 115 pp.

National Air Pollutant Emission Trends, 1900-1997, U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, (1998).

Pattee, O. H.; Bloom, P. H.; Scott, J. M., and Smith, M. R. Lead Hazards Within the Range of the California Condor. *Condor.* 1990 Nov; 92(4):931-937.

Pattee, Oliver H.; Wiemeyer, S. N.; Mulhern, B. M.; Sileo, L., and Carpenter, J. W. Experimental Lead-shot Poisoning in Bald Eagles. *Journal of Wildlife Management.* 1981; 45:806-810.

Poupoulis, C., and L. S. Jensen. 1976. Effects of high dietary copper on gizzard integrity of the chick. *Poultry Science* 55:113-121.

Rabinowitz MB, Wetherill GW, Kopple JD. 1976. Kinetic analysis of lead metabolism in healthy humans. *J Clin Invest* 58:260-270.

Redig, Patrick T.; Stowe, C. M.; Barnes, D. M., and Arent, T. D. Lead Toxicosis in Raptors. *Journal of the American Veterinarian Medical Association.* 1980; 177:941-943.

Reiser, M. H., and S. A. Temple. 1981. Effects of Chronic Lead Ingestion on Birds of Prey. In: Cooper, JE; Greenwood, AG, eds. *Recent advances in the study of raptor diseases.* Keighley, West Yorkshire, England: Chiron Publications:

Risebrough, Robert W., 2001. Absence of demonstrable toxicity to turkey vultures (*Cathartes aura*) of copper and tungsten-tin-bismuth-composite pellets. Report to

USFWS, Sept 2001.

Rowe, B. L., and H. H. Prince. 1983. Behavioral response of mallards to contaminated drinking water. *Bulletin of Environmental Contamination and Toxicology* 30:505-510.

Scott, J. M. and Jurek, R. M. Report to the California Fish and Game Commission on Condor Mortality Issues, Actions and Recommendations. U.S. Fish and Wildlife Service and California Dept. Fish and Game; 1985:51 pp.

Snyder N F R. The California condor recovery program. Problems in organization and execution. Clark, T. W. Reading R. P. & Clarke A. L. Eds. *Endangered species recovery*: Island Press; 1994; pp. 183-204; 450 pp.

Snyder, N F R and Snyder, H A. *Biology and conservation of the California condor*. Current Ornithology. New York, New York, USA; London, England, UK: Plenum Press; 1989; pp. 175-267.

Snyder, N. F. R. and Johnson, E. V. Photographic Censusing of the 1982-1983 California Condor Population. *Condor*. 1985; 87(1):1-13.

Snyder, N. F. R.; Johnson, E. V., and Clendenen, D. A. Primary Molt of California Condors. *Condor*. 1987 Aug; 89(3):468-485.

Snyder, Noel F. R and Snyder, Helen. *The California condor : a saga of natural history and conservation*. San Diego CA ; London: Academic Press; 2000;xxi, 410 p.

Sorenson, Kelly; Burnett, Joseph L., and Davis, James R. Status of the California condor and mortality factors affecting recovery. *Endangered Species Update*. 2001; 18(4):120-123.

St. Louis, V. L., L. Breebaart, J. C. Barlow, and J. F. Klaverkamp. 1993. Metal accumulation and metallothionein concentrations in tree swallow nestlings near acidified lakes. *Environmental Toxicology and Chemistry* 12:1203-1207.

Stoms, David M; Davis, Frank W; Cogan, Christopher B; Painho, Marco O; Duncan, Brean W; Scepan, Joseph, and Scott, J. Michael. Geographic Analysis of California Condor Sighting Data. *Conservation Biology*. 1993 Mar; 7(1):148-159.

Stringfield, Cynthia E. Medical management of the free-ranging California condor. AMERICAN ASSOCIATION OF ZOO VETERINARIANS ANNUAL CONFERENCE PROCEEDINGS; 1998: 422-424.

Supplee, W. C. 1964. Observations on the effect of copper additions to purified turkey diets. *Poultry Science* 43:1599-1600.

U. S. Fish and Wildlife Service (California Condor Recovery Team; U.S. Fish and Wildlife Service). California condor recovery plan; Revised . Portland, Or. : U.S. Fish and Wildlife Service; 1984 Jul 10, [43] p

U. S. Fish Wildlife Service, Condor Recovery Program (U. S Fish Wildlife Service, Condor Recovery Program). Lead Resolution Sorenson, Kelly. California Condor Recovery Program and Recovery Team Meeting; Los Angeles Zoo. 2001 Dec 5-2001 Dec 7: Chapter 4. 9 pp

US Agency for Toxic Substances and Disease Registry (ATSDR) Level of Concern US EPA Toxic Release Inventory (TRI), year 2000 data. US EPA TRI (<http://www.epa.gov/tri/>).

Wiemeyer S N; Krynitsky A J, and Wilbur S R. Environmental contaminants in tissues, foods, and feces of California condors. Wilbur, S. R. & Jackson J. A. Eds. Vulture biology and management. Berkeley, Los Angeles & London: University of California Press; 1983; pp. 427-439; 550 pp.

Wiemeyer, S. N.; Jurek, R. M., and Moore, J. F. Environmental Contaminants in Surrogates, Foods, and Feathers of California Condors (*Gymnogyps-Californianus*). Environmental Monitoring and Assessment. 1986 Jan; 6(1):91-111.

Wiemeyer, S. N.; Scott, J. M.; Anderson, M. P.; Bloom, P. H., and Stafford, C. J. Environmental Contaminants in California Condors. Journal of Wildlife Management. 1988 Apr; 52(2):238-247.

Wilbur, S.R. 1978 The California Condor, 1966-76: A Look at its Past and Future. North American Fauna Series 72. Fish and Wildlife Service, Department of the Interior. 136 pp., illus. (A historical viewpoint)

Wood, E. C., and A. N. Worden. 1973. The influence of dietary copper concentration on hepatic copper in the duckling and the chick. Journal of the Science of Food and Agriculture 24:167-174.