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LEAD HAZARDS WITHIN THE RANGE OF THE CALIFORNIA CONDOR[1]

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Abstract. The prevalence of lead in Golden Eagles (*Aquila chrysaetos*) occurring within the recent historical range of the California Condor (*Gymnogyps californianus*) was determined by analyzing blood samples from 162 Golden Eagles captured between June 1985 and December 1986 at three sites. We found no significant differences between sex and age classes in blood lead levels nor were there differences between residents and migrants. Significant differences were found between months with the highest blood lead levels occurring during the fall/winter period. Approximately one-third (35.8%) of the Golden Eagle population sampled had elevated blood lead levels, values similar to those reported for free-flying California Condors. Given this rate of exposure, if the proposed releases of California Condors back to the wild are to succeed, whether in their former range or elsewhere, any potential for lead poisoning must be reduced. It is essential that we identify the sources of the lead, the seasonal and geographic distribution of these sources, and develop management strategies to reduce or eliminate the hazard.

Key words: *Golden Eagle;* Aquila chrysaetos; *California Condor;* Gymnogyps californianus; *blood lead levels.*

INTRODUCTION

Environmental contaminants have been frequently implicated in the decline of avian populations. Although DDT is the best known example, lead poisoning has been increasingly recognized as a significant hazard in birds and its impacts are well documented (Eisler 1988). Although the effects of lead poisoning on waterfowl (Bellrose 1964) are best known and understood, the published literature suggests that all avian species are vulnerable to the effects of lead but there are wide differences among individuals and species (Pattee et al. 1981, Beyer et al. 1988). Scavenging species appear especially vulnerable since they are more likely to encounter carcasses containing elevated lead residues and pieces of metallic lead. Lead ingestion has caused death in Bald Eagles, *Haliaeetus leucocephalus* (Pattee and Hennes 1983), Andean Condors, *Vultur gryphus* (Locke et al. 1969), and King Vultures, *Sarcoramphus papa* (Decker et al. 1979). Many of the factors suggested by Pattee and Hennes (1983) which make Bald Eagles vulnerable to lead poisoning also apply to California Condors (*Gymnogyps californianus*), i.e., long-lived, low recruitment rates, small populations, and a dependency on carcasses.

Four free-flying California Condors that died in 1981–1986 were found and necropsied; one died of presumed cyanide poisoning and three of lead poisoning (Janssen et al. 1986, Wiemeyer et al. 1988). Additionally, five of 14 wild condors captured during this period had blood lead levels greater than 0.60 parts per million wet weight (ppm), with an average for all samples of 0.69 ppm. This makes lead a significant factor in the decline of the California Condor during the 1980s, accounting for 20–25% (three of 12–15) of known

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losses (Snyder 1986) and 60% (three of five) of the confirmed mortalities (Wiemeyer et al. 1988). The three lead-poisoned California Condors were recovered in January, March, and April (Janssen et al. 1986), suggesting that the initial exposure occurred during the traditional fall-winter hunting season. Although carcasses and offal associated with hunting activities appear to be the probable source of lead affecting condors, other sources such as air pollution in the urban environment (Ohi et al. 1981) or along roadways (Grue et al. 1986) can also cause elevated tissue levels. Fourteen captive California Condors held in an urban environment had blood lead levels averaging 0.09 ppm with a maximum of 0.35 ppm (Wiemeyer et al. 1988). However, mortality in vultures and raptors from sources other than metallic lead appears unlikely (Pattee and Hennes 1983).

The above data indicate that California Condors were exposed to lead and were dying as a result of that exposure. To better understand the availability of lead to condors, Golden Eagles (*Aquila chrysaetos*) foraging in the range of the California Condor were captured and blood samples taken. Golden Eagles were chosen because they are abundant throughout the condor's range and were frequently observed feeding on the same carcasses utilized by California Condors. Although highly predatory, Golden Eagles scavenge readily and could provide a conservative estimate of lead exposure for California Condors. Turkey Vultures (*Cathartes aura*) were also considered since they are known to have elevated lead levels in southern California (Wiemeyer et al. 1986). However, they were deemed unsuitable because they are highly localized in distribution and only seasonally resident, leaving during the late winter period.

METHODS

Golden Eagles were captured in Kern and Ventura counties, California between June 1985 and December 1986 utilizing pit traps and cannon nets (Bloom 1987). The three sites (Fig. 1) were Hopper Mountain National Wildlife Refuge (Hopper), Tehachapi Mountains (Tehachapi), and Hudson Ranch (Hudson—now the Bitter Creek National Wildlife Refuge). Birds were sexed on the basis of size and mass (Bortolotti 1984) and aged on the basis of molt and plumage (Jollie 1947). Prior to release, a 10-ml blood sample was taken from the brachial vein, a numbered patagial tag was placed on the right wing, and a U.S. Fish and Wildlife Service rivet band was attached to the leg. All eagle captures were incidental to ongoing work associated with the California Condor. Therefore, sampling effort was not distributed equally between areas and over time. Biologists working on the condor project were at the three study sites regularly and recorded tag numbers of eagles as encountered.

Blood samples were preserved with 0.5-ml formaldehyde (Wiemeyer et al. 1984) and stored in vials cleaned in 10% nitric acid and rinsed with acetone, then hexane. Preserved samples were stored at −15°C prior to analysis. Lead analyses followed the methods of Wiemeyer et al. (1984) and Fernandez and Hilligoss (1982) using a Perkin-Elmer (Norwalk, Connecticut) HGA-400 graphite furnace at a wavelength of 283.3 nm for the analyses with deuterium arc background correction. The lower limit of reportable, uncorrected residues was 0.01 ppm, wet weight. Recovery of spiked samples averaged 85%.

Data were analyzed using SAS Version 6 for personal computers (SAS Institute, Cary, North Carolina). Resident birds were defined as individuals recaptured or resighted in the area 60 days or more after the initial capture. Because the lead values were not normally distributed, they were log transformed prior to statistical analysis. In samples with lead values below the detection limit, a value of one-half the detection limit (0.005 ppm) was used for all calculations and for deriving means except in those cases where more than one-half the values were below the detection limit. SAS procedures for chi-square and for ANOVA using the general linear model or *t*-tests were used with a significance level of 0.05. When significant differences in means were present, SAS procedures for Bonferonni *t*-tests of differences between means for all main-effect means were used to separate means. All results are reported as parts per million (ppm), wet weight, untransformed and uncorrected for recovery.

RESULTS

One hundred and sixty-two Golden Eagles were captured. Twenty-two birds were recaptured and three birds were captured three times. Fifty birds (30.9%) were resighted in the same general area 60 or more days after their capture and were classified as residents; 13 resident birds (26%)

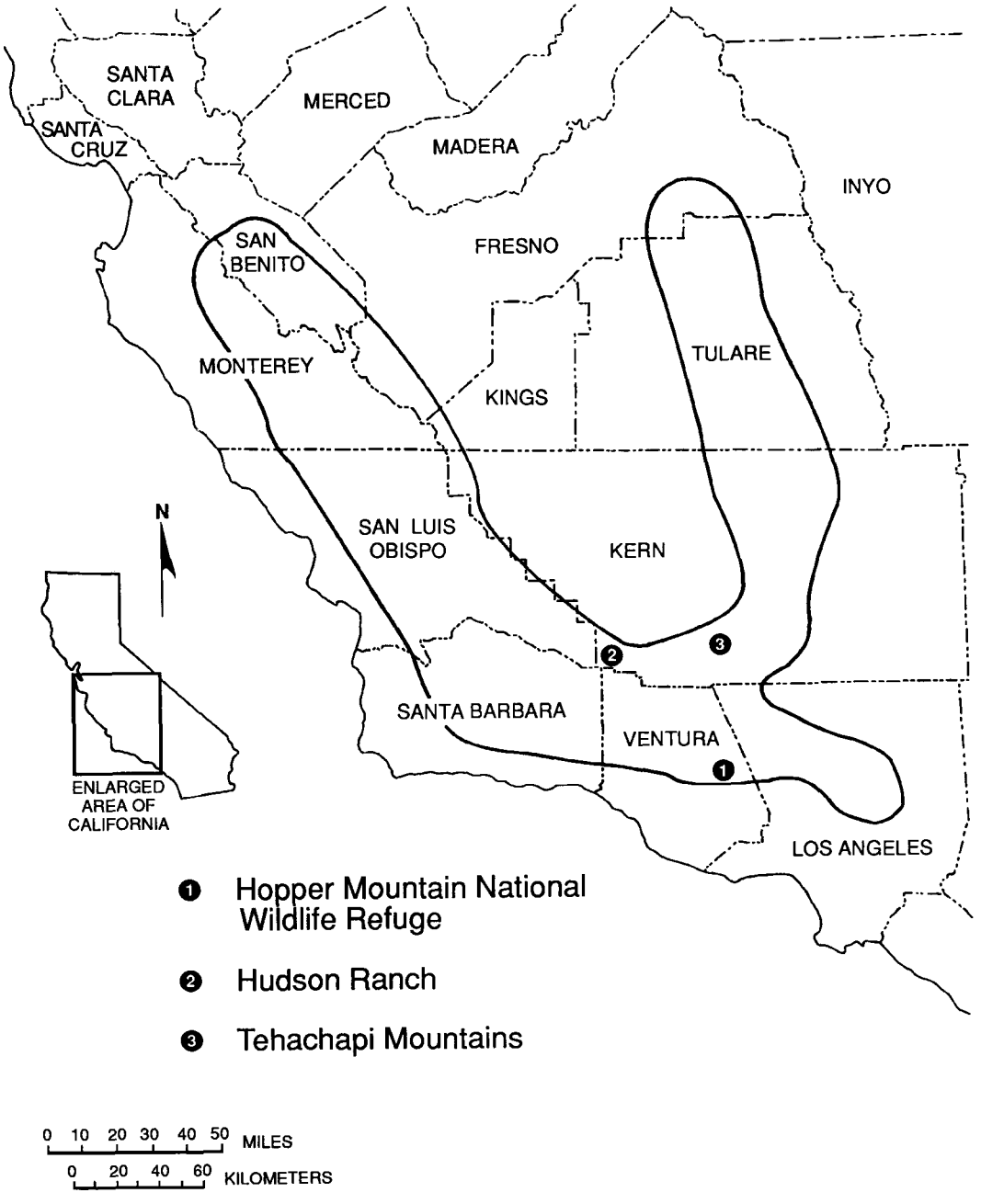

FIGURE 1. Range map of the California Condor (Wilbur 1978) showing locations of Golden Eagle capture sites.

were observed at both Tehachapi and Hudson. Age and sex ratios (two birds were not aged or sexed) were based on 160 birds (Table 1): 32% males (11 resident) and 68% females (39 resident), 22% adults (9 resident) and 78% subadults (41 resident).

There were no differences in the frequency distribution of age classes when compared to resi-

TABLE 1. Mean blood lead levels (ppm, wet weight) in Golden Eagles captured between June 1985 and December 1986 at three locations in Kern and Ventura counties, California, by sex and age.

Age	Sex	n[1]	$\bar{x}$ (ppm)	SD	Minimum	Maximum
Adult	Male	6 (2)	0.08	0.07	<0.01	0.19
	Female	29	0.40	0.99	0.01	5.49
Subadult	Male	45 (4)	0.28	0.62	<0.01	4.11
	Female	80 (3)	0.19	0.23	<0.01	1.27
All birds	Male	51 (6)	0.26	0.59	<0.01	4.11
	Female	109 (3)	0.25	0.55	<0.01	5.49

[1] Number in parentheses represents those birds whose blood lead was below the detection limit of 0.01 ppm.

dency status (df = 1, $\chi^2 = 0.639$, $P = 0.424$) or in the distribution of sexes when compared to residency status (df = 1, $\chi^2 = 3.266$, $P = 0.071$). No significant differences (df = 3, $F = 1.18$, $P = 0.32$) for lead exposure were found between sex and age classes (Table 1) nor were there significant differences (df = 1, $F = 2.42$, $P = 0.13$) between birds classified as resident and migrant (Table 2). Because no differences in blood lead levels could be attributed to age, sex, or residency, these groupings were combined for all subsequent analyses. The location of the capture site did affect the mean blood lead level (Table 2). Mean lead levels were significantly higher (df = 2, $F = 13.97$, $P = 0.0001$) at Tehachapi than at Hudson whereas Hopper was intermediate between the two. Although no significant differences were found between first and second captures ($n = 22$, $t = 1.00$, $P = 0.33$), first and third captures ($n = 3$, $t = 2.07$, $p = 0.17$), or second and third captures ($n = 3$, $t = -1.29$, $P = 0.33$), only data from first captures were used in the statistical analyses to eliminate potential bias associated with second and third captures (Table 3).

Significant differences (df = 10, $F = 5.66$, $P = 0.0001$) were found in mean lead values by month (Table 4). The lowest mean values occurred during the summer (June, July, August) whereas the highest values occurred during the winter period (November, December); March was also high. Since lead values do not follow a normal distribution (high values are rare) and because elevated values are those with the most biological significance, all blood lead values were sorted into four groups according to the convention of Redig (1984): <0.20 ppm = background; 0.20 ppm to 0.59 ppm = exposed; 0.60 ppm to 0.99 ppm = clinically affected; >1.00 ppm = acute lead poisoning. Using these criteria, 35.8% of the population had been exposed to elevated (≥0.20 ppm) environmental lead (Table 5). There were 58 blood lead values greater than or equal to 0.20 ppm, 45 (78%) of these values were from the months of September–December (September = 1, October = 7, November = 24, December = 12), six were from March, four were from January, two were from May, and one each from September and July. Nine blood lead values were greater than 0.60 ppm; eight (89%) were from the months of September–December and one was from March.

DISCUSSION

No significant differences in blood lead levels were detected that could be related to sex, age,

TABLE 2. Mean blood lead levels (ppm, wet weight) of Golden Eagles captured between June 1985 and December 1986 in Kern and Ventura counties, California, by locality and residency status.

		n[1]	$\bar{x}$ (ppm)[2]	SD	Minimum	Maximum
Status	Migrant	112 (6)	0.29	0.22	<0.01	5.49
	Resident	50 (3)	0.16	0.22	<0.01	1.27
Location	Hopper	2	0.23 AB	0.14	0.13	0.33
	Hudson	78 (7)	0.19 A	0.63	<0.01	5.49
	Tehachapi	82 (2)	0.30 BC	0.48	<0.01	4.11

[1] Number in parentheses represents those birds whose blood lead was below the detection limit of 0.01 ppm.
[2] Means with identical letters are not significantly different ($P < 0.05$). Comparisons are based on log-transformed data.

TABLE 3. Mean blood lead levels (ppm, wet weight) of Golden Eagles captured between June 1985 and December 1986 in Kern and Ventura counties, California, for first, second, and third captures.

	n[1]	$\bar{x}$ (ppm)	SD	Minimum	Maximum
First capture	162 (9)	0.25	0.56	<0.01	5.49
Second capture	22	0.32	0.66	0.02	3.22
Third capture	3	0.09	0.04	0.04	0.12

[1] Number in parentheses represents those birds whose blood lead was below the detection limit of 0.01 ppm.

or residency. Differences between areas, particularly Hudson and Tehachapi, may be an artifact. Seven of the nine birds with blood lead levels below the detection limit were from Hudson. Substantial movement between the two areas also occurred. These factors plus the nonrandom sampling scheme used to determine the trapping schedule make it impossible to determine if the reported differences are biologically significant.

Elevated blood lead levels were observed in September, October, November, and December with all but one of the clinically lead-poisoned cases occurring in these months. In Bald Eagles, 89% of the lead poisoning cases reported by Pattee and Hennes (1983) occurred between October and March, with a peak in January. They attributed the source of lead to waterfowl and the waterfowl hunting season. As with Bald Eagles, there appears to be a relationship between season and the elevated blood lead levels in Golden Eagles.

Although blood lead levels may be elevated in Golden Eagles from sources other than metallic lead, mortality is rarely associated with these sources (Pattee and Hennes 1983). Diets containing high tissue lead levels may contribute to elevated blood lead levels. American Kestrels (*Falco sparverius*) had blood lead levels of up to 9.4 ppm ($\bar{x}$ = 1.69 ppm) when fed a diet containing 448 ppm (dry weight) of biologically incorporated lead and a maximum blood lead level of 0.52 ppm ($\bar{x}$ = 0.33 ppm) when fed 0.45 ppm (dry weight) of biologically incorporated lead (Custer et al. 1984). The latter dietary level is within the ranges reported by Wiemeyer et al. (1983, 1986) for tissues collected from carcasses commonly consumed by Golden Eagles within the condor's range. This contributes to the blood lead levels but does not explain the disproportionate number of elevated blood lead levels in October, November, and December. This may be due to an increase in the number of carcasses containing metallic lead during the traditional fall hunting season.

The continued detection of elevated levels into spring may be due to a number of factors including other types of hunting activities, preservation of contaminated carcasses by cold weather, and a refractory period between initial exposure and potential effects. Pattee et al. (1981)

TABLE 4. Mean blood lead levels (ppm, wet weight) of Golden Eagles captured between June 1985 and December 1986 in Kern and Ventura counties, California, by pooled month.

	n[1]	$\bar{x}$ (ppm)[2]	SD	Minimum	Maximum
January	8	0.23 A	0.12	0.08	0.39
February	2	0.16 AB	0.06	0.11	0.20
March	9	0.30 A	0.23	0.10	0.89
April	0	—	—	—	—
May	5	0.18 AB	0.10	0.10	0.34
June	2 (1)	0.02 C	0.03	<0.01	0.04
July	4	0.14 AB	0.11	0.04	0.28
August	9 (1)	0.04 BC	0.03	<0.01	0.10
September	27 (5)	0.26 AB	1.05	<0.01	5.49
October	24 (1)	0.19 AB	0.21	<0.01	0.69
November	51 (1)	0.30 AB	0.59	<0.01	4.11
December	21	0.33 A	0.26	0.10	1.27

[1] Number in parentheses represents those birds whose blood lead was below the detection limit of 0.01 ppm.
[2] Means with identical letters are not significantly different ($P < 0.05$). Comparisons were made with log-transformed data.

TABLE 5. Mean blood lead levels (ppm, wet weight) of Golden Eagles captured between June 1985 and December 1986 in Kern and Ventura counties, California, by extent of exposure.

Exposure (ppm)	n	$\bar{x}$ (ppm)	SD
<0.20	104	0.08	0.05
0.20–0.59	49	0.33	0.09
0.60–0.99	5	0.77	0.15
>1.00	4	3.04	2.11

found that it required 10–133 days for Bald Eagles dosed with lead shot to die; Janssen et al. (1986) reported that a California Condor that died in January had a blood lead level of 1.8 ppm in November. Blood lead levels typically rise rapidly after initial exposure to lead. Hoffmann et al. (1981) found mean blood lead levels in Bald Eagles dosed with 10 number 4 lead shot to be 0.8 ppm after 24 hr and 2.8 ppm after 72 hr. Other factors besides deer hunting in the fall may be contributing to exposure to metallic lead. Firearms are used in the area year-round for target practice and for taking such nongame as ground squirrels (*Spermophilus beecheyi*) and coyotes (*Canis latrans*), species which hunters frequently leave in the field. Such activities would make carcasses containing metallic lead readily available to scavenging species.

The purpose of this work with Golden Eagles was intended to assess the potential hazard lead posed to California Condors. Beyer et al. (1988) found considerable differences in the responses of six species they dosed with lead and reported that extrapolations between species could be difficult, if not impossible. Taking these factors into consideration, we feel that Golden Eagles provide a conservative look at the exposure to lead received by California Condors. On this basis, California Condors would receive their maximum exposure to lead during the months of October, November, and December. Based on the work with Bald Eagles (Pattee et al. 1981) and the probable exposure time for the one dead California Condor (1.8 ppm blood lead in November, dead in January) reported by Janssen et al. (1986), we estimate that condors would die as long as 3 to 4 months after ingestion of metallic lead. Based on the dates when the three condors dying of lead poisoning were found (Janssen et al. 1986), the condors probably ingested lead sometime in October/November, December/January, and January/February—months quite close to those suggested by the Golden Eagle data as being most hazardous. This study does not address the issue of the source of the lead. Although there is a temporal factor that suggests a relationship between the hunting season and elevated lead levels (and lead availability), no data were collected or are available to implicate lead shot and/or bullet-killed animals as the primary source of the lead causing the elevated blood lead levels in Golden Eagles. However, eagles and condors were observed feeding on deer carcasses and offal from hunter-killed deer.

Our findings that 36% of the 162 Golden Eagles sampled had been exposed to lead and that 2.5% had levels indicative of clinical lead poisoning corroborate earlier findings by Wiemeyer et al. (1988) and Bloom et al. (1989) that carrion-feeding birds within the range of the California Condor are exposed to levels of lead sufficient to cause or contribute to their mortality. The impact of this level of lead exposure on Golden Eagle populations is unknown but could be detrimental. Even if there are no population effects, the data suggest that some Golden Eagles are exposed to sufficient dietary lead to cause death in susceptible individuals. If the proposed releases of California Condors back to the wild, whether in their former range or elsewhere, are to succeed, this threat of lead poisoning must be reduced. Wiemeyer et al. (1988) addresses this issue and suggests a number of scenarios.

Recovery of endangered species populations requires that the variables responsible for their decline be identified and controlled. In general, researchers have usually identified a number of factors believed responsible for a given species decline. This was certainly true for the California Condor (Koford 1953, Wilbur 1978). Rarely, however, have these limiting factors been quantified. With limited resources available for recovery efforts, such quantification of limiting factors is essential if managers are to direct money and actions to where they will have the greatest impact on recovery. This work and others (Wiemeyer et al. 1988, Bloom et al. 1989) have quantified one of many possible limiting factors to California Condor populations. Thus we are in a position to reduce (perhaps significantly) annual condor mortality by reducing exposure to lead. Now that lead is known to be a ubiquitous hazard, it is essential that we identify the sources of the lead, the types of carcasses it is found in,

and the seasonal and geographical distribution of those carcasses.

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