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PATTERNS OF MORTALITY IN FREE-RANGING CALIFORNIA CONDORS (*GYMNOGYPS CALIFORNIANUS*)

Bruce A. Rideout,[1,16] Ilse Stalis,[1] Rebecca Papendick,[1] Allan Pessier,[1] Birgit Puschner,[2] Myra E. Finkelstein,[3] Donald R. Smith,[3] Matthew Johnson,[4] Michael Mace,[5] Richard Stroud,[6] Joseph Brandt,[7] Joe Burnett,[8] Chris Parish,[9] Jim Petterson,[10] Carmel Witte,[1] Cynthia Stringfield,[11] Kathy Orr,[12] Jeff Zuba,[13] Mike Wallace,[14] and Jesse Grantham[15]

[1] Wildlife Disease Laboratories, Institute for Conservation Research, San Diego Zoo Global, PO Box 120551, San Diego, California 92112, USA
[2] Toxicology Laboratory, California Animal Health and Food Safety Laboratory, University of California Davis, Davis, California 95616, USA
[3] Microbiology and Environmental Toxicology Department, University of California, Santa Cruz, California 95064, USA
[4] U.S. Geological Survey, Forest and Rangeland Ecosystem Science Center, 3200 SW Jefferson Way, Corvallis, Oregon 97331, USA
[5] Bird Department, San Diego Zoo Safari Park, 15500 San Pasqual Valley Road, Escondido, California 92027, USA
[6] U.S. Fish and Wildlife Service National Wildlife Forensic Lab, Ashland, Oregon, USA
[7] U.S. Fish and Wildlife Service, Hopper Mountain National Wildlife Refuge Complex, California Condor Recovery Program, PO Box 5839, Ventura, California 93005, USA
[8] Ventana Wildlife Society, 19045 Portola Dr. Ste. F-1, Salinas, California 93908, USA
[9] The Peregrine Fund, 5668 West Flying Hawk Lane, Boise, Idaho 83709, USA
[10] National Park Service, Pinnacles National Monument, 5000 Highway 146, Paicines, California 95043, USA
[11] Moorpark College, 7075 Campus Road, Moorpark, California 93021, USA
[12] Phoenix Zoo, 455 North Galvin Parkway, Phoenix, Arizona 85008-3431, USA
[13] Veterinary Services, San Diego Zoo Safari Park, 15500 San Pasqual Valley Road, Escondido, California 92027, USA
[14] Applied Animal Ecology, Institute for Conservation Research, San Diego Zoo Global, 15600 San Pasqual Valley Road, Escondido, California 92027, USA
[15] U.S. Fish and Wildlife Service, California Condor Recovery Program, 2493 Portola Road, Suite A, Ventura, California, 93003, USA
[16] Corresponding author (email: brideout@sandiegozoo.org)

ABSTRACT: We document causes of death in free-ranging California Condors (*Gymnogyps californianus*) from the inception of the reintroduction program in 1992 through December 2009 to identify current and historic mortality factors that might interfere with establishment of self-sustaining populations in the wild. A total of 135 deaths occurred from October 1992 (the first post-release death) through December 2009, from a maximum population-at-risk of 352 birds, for a cumulative crude mortality rate of 38%. A definitive cause of death was determined for 76 of the 98 submitted cases, 70% (53/76) of which were attributed to anthropogenic causes. Trash ingestion was the most important mortality factor in nestlings (proportional mortality rate [PMR] 73%; 8/11), while lead toxicosis was the most important factor in juveniles (PMR 26%; 13/50) and adults (PMR 67%; 10/15). These results demonstrate that the leading causes of death at all California Condor release sites are anthropogenic. The mortality factors thought to be important in the decline of the historic California Condor population, particularly lead poisoning, remain the most important documented mortality factors today. Without effective mitigation, these factors can be expected to have the same effects on the sustainability of the wild populations as they have in the past.

Key words: California Condor, Cathartidae, lead toxicosis, mortality, pathology, reintroduction.

INTRODUCTION

The California Condor (*Gymnogyps californianus*) went through a severe population decline in the late 20th century (Wilbur, 1978; Snyder and Snyder, 2000). In 1980, an expanded field research effort led by the US Fish and Wildlife Service and the National Audubon Society was initiated to try and save the species. By 1982, intensive field surveys determined there were only 23 individuals left in the wild population. Between 1982 and 1987, eggs, chicks, and adults were removed from the wild for a captive breeding program. When the last bird was trapped from the wild on April 19, 1987, the total world population stood at 27 individuals, all in captivity (Grantham, 2007). The original causes for the decline were not fully elucidated. Intentional shooting, lead poisoning from ingestion of spent ammunition in carcasses, egg collecting, and strychnine poisoning had been documented (Wilbur,

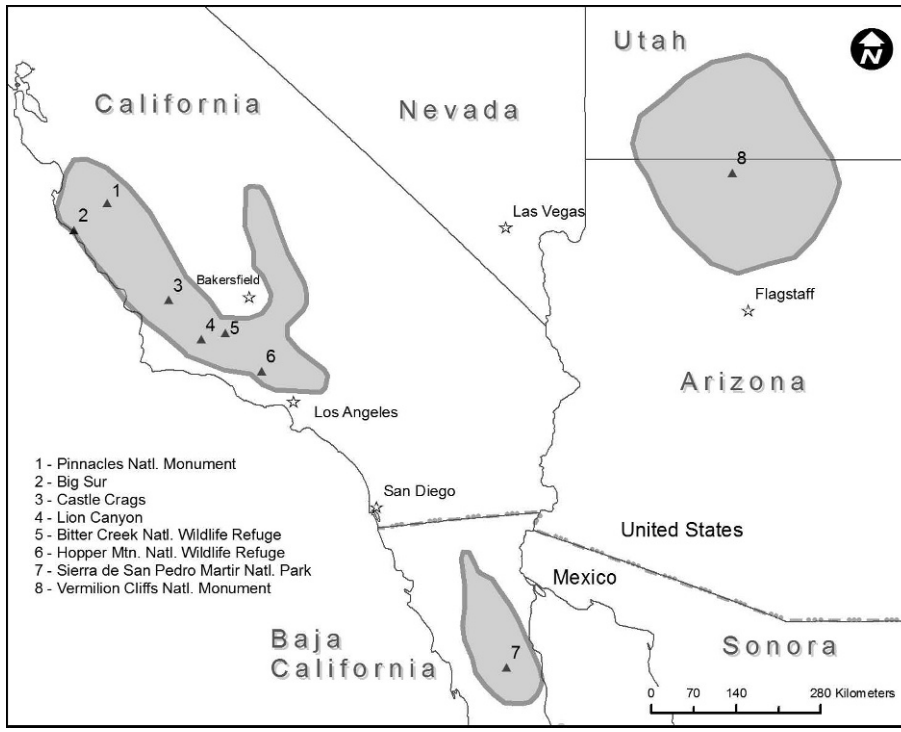

FIGURE 1. Map of California Condor release locations (numbered 1–8) and current range (shaded) in the southwestern United States and northwestern Mexico.

1978; Janssen et al., 1986; Wiemeyer et al., 1988) but their relative importance was not fully known.

The conservation breeding program that followed was very successful, producing sufficient numbers of birds for reintroductions to begin in Southern California in 1992 (Kuehler et al., 1991; Snyder and Snyder, 2000; Grantham, 2007). The reintroduction program expanded to include additional sites in Arizona in 1996, Central California in 1997, and Baja California, Mexico in 2003 (Grantham, 2007; Fig. 1).

A basic tenet of reintroduction biology is that wildlife reintroductions should not begin until the original causes of decline have been mitigated (IUCN, 1998). However, the original causes of decline may be poorly documented, as was the case with the California Condor. In these situations, establishing specific causes of death in released individuals is especially important. It not only can reveal reasons for reintroduction failures but can also provide circumstantial evidence regarding original causes of decline that might still prevail.

We documented causes of death in free-ranging California Condors at all release sites from the inception of the reintroduction program in 1992 through December 2009 to provide baseline data for future analyses of the impact of these mortality factors on the establishment of self-sustaining populations in the wild.

MATERIALS AND METHODS

All mortalities from the inception of the release program in 1992 through 31 December 2009 were evaluated. Birds were included in the study if they were found dead in the wild, were brought in from the wild for a life-threatening medical condition and died of causes directly or indirectly related to the medical problem being treated, or were missing and presumed dead. Nestlings were defined as birds <6 mo old that had not yet

fledged; juveniles as fledged birds from 6 mo through 5 yr old; and adults as birds ≥6 yr old. The maximum population at risk consisted of 49 chicks hatched in the wild and 303 juvenile or adult birds released during the study period. The cumulative crude mortality rate was defined as the total number of mortalities divided by the combined maximum population at risk for the study period. Proportional mortality rates (PMR) were calculated for each age class by dividing the number of mortalities from a specific cause by all mortalities with a known cause.

Carcasses were recovered as quickly as possible and delivered to the Wildlife Disease Laboratories, San Diego Zoo, San Diego, California, USA for complete post-mortem examinations, with the exception of cases thought to involve intentional killing, which were delivered to the US Fish and Wildlife Service Forensics Laboratory. Postmortem exams at the San Diego Zoo consisted of whole body dorsoventral and lateral x-rays, in most cases, followed by an external exam and complete necropsy. Samples of all identifiable tissues were fixed in 10% neutral-buffered formalin. Body condition was scored qualitatively as good, fair, or poor based on total body fat deposits and pectoral muscle mass.

Liver samples from each case where liver was available were frozen at −20 C for heavy-metal analysis (Toxicology Laboratory, California Animal Health and Food Safety Laboratory, Davis, California). Kidney samples were used when liver was not available (three cases). Samples were digested with nitric acid and analyzed for lead, manganese, iron, mercury, arsenic, molybdenum, zinc, copper, and cadmium by inductively coupled argon plasma emission spectrometry (ICP-AES; ARL, Accuris Model, Thermo Optek Corporation, Franklin, Massachusetts, USA). Accuracy of ICP was measured by analyzing standard reference materials (SRM) such as bovine liver (National Institute of Standards and Technology, SRM 1577b) and lobster hepatopancreas (National Research Council of Canada TORT-2). Data were accepted if analyzed standard reference material values were within two standard deviations of the certified reference value. Metal concentrations were determined and are expressed on a wet-weight basis. Feather and bone samples collected post-mortem from one bird (422) were processed using trace metal clean techniques and analyzed for lead concentrations according to the methods of Finkelstein et al. (2010). Lead concentrations were determined using a Finnigan MAT Element magnetic sector-inductively coupled plasma mass spectrometer (ICP-MS; Finnigan MAT, Inc., San Jose, California, USA), measuring masses of ^{206}Pb, ^{207}Pb, ^{208}Pb, and ^{205}Tl (the latter as an internal standard), as described (Finkelstein et al., 2010). Normal values for heavy metals in liver (in mg/kg wet weight) were based on data from California Condors in the breeding flocks (copper mean 35 mg/kg, STD 29 mg/kg, $n=8$; zinc mean 38 mg/kg, STD 17 mg/kg, $n=8$; other values not shown) as well as comparisons with Turkey Vultures (Risebrough, unpubl.) and published data from other avian species (Puls, 1994). Levels of manganese, iron, mercury, arsenic, molybdenum, and cadmium were interpreted as noncontributory and are not shown.

Fixed tissues were processed routinely, sectioned at 5 μm, and stained with hematoxylin and eosin for histopathologic examination. The cause of death in each case was defined as the causal factor or factors responsible for initiating the sequence of events that ultimately led to death. It represents the professional judgment of the pathologist based on an integration of the field history, antemortem clinical and laboratory findings (when available), results of the post-mortem examination, and any ancillary diagnostic results. It includes supporting evidence as well as evidence excluding other causes. Cases were assigned a suspected cause of death when there was historic or physical evidence pointing to a cause, but the carcass was too autolyzed to confirm the cause or the evidence did not meet the full case definition. Cases were assigned an unknown cause of death when the carcass was missing, not submitted, scavenged, too autolyzed for evaluation, or when evidence pointing to a particular cause could not be identified or was only speculative. A number of pathologists have worked on condor cases over the years of the program, but each case was reviewed by a single pathologist (B.R.) to ensure consistency. In some cases, the original cause of death was revised based on new data or on a more-comprehensive review of the data than was possible at the time the original report was written.

Lead toxicosis was diagnosed when antemortem blood lead concentrations were >50 μg/dl (determined in the field using LeadCare I or II point-of-care devices; Magellan Biosciences, Chelmsford, Massachusetts, USA) based on clinical experience and previously published reports, (e.g., De Francisco et al., 2003) or on post-mortem liver or kidney lead concentrations >6 mg/kg wet weight (Franson, 1996). Zinc toxicosis was diagnosed when hepatic zinc concentrations were >1,000 mg/kg wet weight and when (in cases with adequate cell

preservation) there was histologic evidence of individual cell necrosis in acinar regions of the pancreas, individual cell necrosis in the liver, hemosiderosis in hepatocytes and Kupffer cells, and acute tubular necrosis in the kidney (Puls, 1994; Puschner et al., 1999). Methods for diagnosis of ethylene glycol toxicosis were previously reported (Murnane et al., 1995). Power line trauma or electrocution was diagnosed when there were compatible traumatic injuries, regionally singed or discolored feathers or skin, and a history of being found in the immediate vicinity of power lines, often with feathers adhered to the power lines.

West Nile virus infection was diagnosed by immunohistochemistry on heart and brain (California Animal Health and Food Safety Laboratory) in conjunction with compatible lesions such as nonsuppurative inflammation and hemorrhage. All other causes of death relied on standard case definitions. Anthropogenic causes of death were defined as those attributable to man-made structures or human activities (lead toxicosis, environmental trash, power lines, diseases introduced by human activity [e.g., West Nile Virus], and intentional killing). Nonanthropogenic mortalities include all other causes such as predator trauma, accidental drowning, and poor body condition.

RESULTS

For the entire release program, 135 deaths occurred from October 1992 (the first post-release death) through December 2009, from a maximum population-at-risk of 352 birds, for a cumulative crude mortality rate of 38%. One-hundred carcasses were recovered and 98 were submitted for necropsy (92 to the San Diego Zoo and six to the US Fish and Wildlife Service Forensics Laboratory; two were not submitted due to poor condition of the carcasses).

A definitive cause of death was determined for 76 of the 98 submitted cases, 70% (53/76) of which were attributed to anthropogenic causes. All mortalities are presented in Tables 1–3, in chronological order by age-class and release location, but the most important mortality factors are summarized below for the released population as a whole. Figure 1 illustrates the release locations and current ranges.

Nestling mortalities

Sixteen nestling deaths occurred from an at-risk population of 49 wild-hatched nestlings (cumulative crude mortality rate 33%). A definitive cause of death was determined for 11 of 16 nestlings submitted, of which 82% (9/11) were anthropogenic.

Trash ingestion was the cause of death in eight cases, making it the most important mortality factor in nestlings, with a PMR of 73% (8/11). The trash generally consisted of bottle caps and small pieces of broken glass, plastic, and metal. In condor 285, trash ingestion was the primary problem, but the immediate cause of death was secondary zinc toxicosis. The presumed source of zinc was a galvanized metal washer (Fig. 2). Condor 308 was brought to the Los Angeles Zoo for treatment of trash ingestion but was subsequently euthanized because of secondary respiratory aspergillosis unresponsive to therapy. West Nile virus infection with secondary respiratory aspergillosis was the cause of death in one nestling that had not yet been vaccinated against West Nile Virus.

Juvenile and adult mortalities

A definitive cause of death was determined for 65 of the 85 juveniles or adults submitted, of which 68% (44/65) were anthropogenic. The most common mortality factors for juveniles and adults fell into the categories of toxicosis, trauma or accident, inanition, intentional killing, infectious disease, and trash ingestion.

There were 23 confirmed cases of lead toxicosis (PMR 35%; 23/65) and one of ethylene glycol toxicosis. In some cases, lead toxicosis was diagnosed antemortem based on blood lead levels, but chelation therapy resulted in hepatic lead levels falling below the diagnostic threshold by the time of death. Eight of the lead toxicosis cases from Arizona had metal fragments in the gastrointestinal (GI) tract on antemortem radiographs ($n=3$) or lead ammunition fragments in the GI tract

TABLE 1. California Condor mortalities in California 1992–2010.[a]

SB	Hatch date	Death date	Release location	Sex	Primary cause of death	Additional findings	Gastric foreign bodies	WNV	Liver[b] (mg/kg, ww)		
									Copper	Lead	Zinc
Nestlings (<6 mo)											
263	22-Jun-01	25-Jun-01	SoCal	F	Trauma, presumed parental	None	NP[c]	ND[d]	ND	ND	ND
271	11-Apr-02	4-Oct-02	SoCal	M	Unknown, autolysis	Elevated liver copper, terminal aspiration, mild trauma	NP	ND	531	<1	54.9
285	10-May-02	13-Oct-02	SoCal	F	Trash ingestion	Zinc toxicosis, poor body condition	Bottle caps, glass, plastic, metal spring, other metal	ND	84.8	<1	1500
288	28-May-02	21-Oct-02	SoCal	M	Trash ingestion	Visceral gout, elevated liver copper	Rubber and plastic	ND	290	<1	57
308	10-May-03	24-Sep-03	SoCal	M	Trash ingestion, euthanasia	Respiratory aspergillosis, elevated liver copper	Glass and plastic (more removed antemortem)	ND	212	<1	52.5
333	22-Apr-04	17-Aug-04	SoCal	M	Trash ingestion	Elevated liver copper	Metal nuts and washers, glass, plastic, and unidentified thick-shelled nut	ND	341	<1	48.6
386	19-May-05	25-Aug-05	SoCal	F	West Nile virus infection (unvaccinated)	Respiratory aspergillosis; ingested foreign bodies with ulcerative ventriculitis, elevated liver copper	Metal nuts, glass, and plastic	Pos	150	<1	55.5
396	25-Mar-06	16-Jun-06	SoCal	Unk	Trash ingestion	Unknown	19 bottle caps, 23 pieces of glass, aluminum pop tops, black rubber	ND	NA	NA	NA
443	3-May-07	30-Oct-07	SoCal	F	Trauma, associated with brush fire	Elevated liver copper	NP	Neg	330	<1	59
533	15-May-09	12-Jun-09	SoCal	Unk	Suspected black bear predation (only bone fragments recovered)	Unknown	NP	ND	NA	NA	NA

TABLE 1. Continued.

SB	Hatch date	Death date	Release location	Sex	Primary cause of death	Additional findings	Gastric foreign bodies	WNV	Liver[b] (mg/kg, ww)		
									Copper	Lead	Zinc
503	4-Apr-09	6-Jul-09	CenCal	Unk	Trash ingestion	Fractures, R Radius, L Humerus (Possibly postmortem)	NP	ND	NA	NA	NA
546	1-Jun-09	29-Jul-09	SoCal	F	Unknown, missing	Unknown	NP	ND	NA	NA	NA
539	24-May-09	10-Sep-09	SoCal	M	Trash ingestion	None	NP	Neg	99	<1	36
Juveniles (6 mo–5 yr)											
66	29-May-91	7-Oct-92	SoCal	M	Toxicosis, ethylene glycol	None	Styrofoam, rubber flip-flop	ND	42.3	<0.5	43.3
75	17-May-92	28-May-93	SoCal	M	Power line collision, electrocution	None	NP	ND	NA	NA	NA
81	1-Apr-92	12-Jun-93	SoCal	F	Power line collision, trauma	None	NP	ND	NA	NA	NA
78	27-May-92	30-Oct-93	SoCal	M	Power line collision, trauma	None	Glass and rubber	ND	NA	NA	NA
89	24-Apr-93	24-Jun-94	SoCal	M	Power line collision, trauma	None	Silicone rubber	ND	NA	NA	NA
87	18-Apr-93	29-Jul-94	SoCal	M	Malignant round cell tumor	Cachexia, respiratory aspergillosis	NP	ND	NA	NA	NA
118	16-Apr-95	16-Aug-96	SoCal	M	Unknown, missing	Brush fire	NP	ND	NA	NA	NA
109	14-Apr-94	10-Oct-96	SoCal	F	Unknown, missing	Unknown	NP	ND	NA	NA	NA
131	29-Mar-96	2-Feb-97	SoCal	F	Poor body condition	None	NP	ND	68.8	<1	262
103	22-Apr-94	9-Mar-97	SoCal	F	Unknown, missing	Unknown	NP	ND	NA	NA	NA
129	17-May-95	30-Aug-97	SoCal	M	Suspected Power line trauma	None	NP	ND	NA	<1	NA
143	5-May-96	17-Jul-98	SoCal	F	Entrapment–drowning	None	NP	ND	26	<1	50
152	15-Mar-97	17-Jul-98	SoCal	F	Entrapment–drowning	None	NP	ND	56	<1	44
153	18-Mar-97	9-Aug-98	SoCal	M	Gunshot	Idiopathic interstitial pneumonia	NP	ND	ND	ND	ND
178	30-Mar-98	1-Jul-99	SoCal	M	Poor body condition	None	NP	ND	**75**	**<1**	**38.4**
130	24-May-95	2-Oct-99	SoCal	F	Unknown, missing	Unknown	NP	ND	NA	NA	NA
175	18-Mar-98	15-Nov-99	SoCal	M	Toxicosis, lead	Trauma of unknown origin, elevated liver copper	NP	ND	266	21	72.3

TABLE 1. Continued.

SB	Hatch date	Death date	Release location	Sex	Primary cause of death	Additional findings	Gastric foreign bodies	WNV	Liver[b] (mg/kg, ww)		
									Copper	Lead	Zinc
185	13-Apr-98	28-Nov-99	SoCal	M	Unknown, missing	Unknown	NP	ND	NA	NA	NA
188	27-Apr-98	22-Dec-99	SoCal	M	Unknown, no carcass submitted	Unknown	NP	ND	NA	NA	NA
113	9-Jun-94	29-Dec-99	SoCal	F	Unknown, missing	Unknown	NP	ND	NA	NA	NA
106	18-Mar-94	19-Feb-00	SoCal	F	Unknown, missing	Unknown	NP	ND	NA	NA	NA
181	2-Apr-98	1-Oct-00	SoCal	F	Unknown, skeletal remains	Unknown	NP	ND	NA	NA	NA
132	24-Apr-96	31-Jan-01	SoCal	F	Toxicosis, lead	None	Piece of wood	ND	49.8	26.4	22.4
230	29-Apr-00	9-May-01	CenCal	M	Power line collision, electrocution	None	NP	ND	113	<1	44.1
201	17-Apr-99	17-Jun-01	SoCal	M	Unknown, missing	Unknown	NP	ND	NA	NA	NA
215	30-Mar-00	27-Jun-01	SoCal	M	Power line collision, electrocution	None	NP	ND	41.2	<1	37.4
233	9-May-00	17-May-02	CenCal	F	Unknown, missing	Unknown	NP	ND	NA	NA	NA
212	25-May-99	30-Nov-02	CenCal	F	Power line collision, electrocution	None	Rubber weather stripping	ND	25.9	<1	50.3
254	13-May-01	19-Feb-03	CenCal	M	Power line collision, electrocution	None	NP	ND	33.4	<1	34.1
260	1-Jun-01	20-May-03	CenCal	M	Poor body condition associated with poor feeding response post-release	None	NP	ND	69.4	<1	124
267	5-Apr-02	19-Jul-03	SoCal	M	Trauma, predator (possible canid)	Poor body condition, elevated liver copper	NP	ND	141	<1	77.1
202	18-Apr-99	26-Oct-03	SoCal	M	Unknown, missing	Brush fire	NP	ND	NA	NA	NA
268	6-Apr-02	26-Oct-03	SoCal	F	Unknown, missing	Brush fire	NP	ND	NA	NA	NA
256	13-May-01	15-Nov-03	CenCal	F	Unknown, scavenged	None	NP	ND	NA	NA	NA
179	30-Mar-98	20-Dec-03	SoCal	M	Unknown, missing	Brush fire	NP	ND	NA	NA	NA
277	24-Apr-02	5-Jan-06	SoCal	F	Unknown, missing	Unknown	NP	ND	NA	NA	NA
376	3-May-05	6-Jul-06	CenCal	M	Power line collision, electrocution	None	NP	ND	57.7	<1	55
363	14-Apr-05	14-Jul-06	CenCal	M	Poor body condition associated with poor feeding response post-release	Elevated liver lead	Cellophane	Neg	57.1	1.5	219

TABLE 1. Continued.

SB	Hatch date	Death date	Release location	Sex	Primary cause of death	Additional findings	Gastric foreign bodies	WNV	Liver[b] (mg/kg, ww)		
									Copper	Lead	Zinc
417	9-May-06	12-May-07	CenCal	F	Trauma, predator (USFWS Forensic Lab)	Possible hemolytic anemia	NP	ND	NA	<0.-2-5	NA
301	20-Apr-03	16-May-07	CenCal	M	Power line collision, trauma and electrocution	None	NP	ND	24	<1	47.8
307	5-May-03	16-May-07	CenCal	F	Rattlesnake bite	None	NP	ND	45.3	<1	62.6
356	25-Mar-05	1-Oct-07	CenCal	F	Unknown, scavenged	Unknown	NP	ND	NA	NA	NA
429	8-Apr-07	4-Dec-07	CenCal	F	Unknown, missing	Unknown	NP	ND	NA	NA	NA
402	14-Apr-06	8-Jan-08	SoCal	F	Trauma, predator, mountain lion	None	NP	ND	3.6	<1	21
377	4-May-05	25-Jun-08	CenCal	F	Unknown, missing	Brush fire	NP	ND	NA	NA	NA
336	28-Apr-04	7-Sep-08	CenCal	F	Toxicosis, lead	Poor body condition	NP	ND	110	11	80
450	15-May-07	12-Dec-08	SoCal	F	West Nile virus infection (vaccinated twice)	Aspergillosis, elevated liver copper	NP	Pos	210	<1	40
475	20-Apr-08	19-Dec-08	CenCal	M	Trauma, presumed conspecific	Elevated liver copper	NP	Neg	150	<1	78
474	21-Apr-08	7-Mar-09	SoCal	F	Trauma, conspecific	Handling for fluid therapy	NP	Neg	98	<1	41
422	14-May-06	9-Jul-09	CenCal	F	Toxicosis, lead (see figure 3)	Unknown	NP	ND	NA	NA	NA
358	30-Mar-05	11-Jul-09	SoCal	M	Accidental strangulation with 8 mm rope	None	NP	ND	14	<1	37
Adults (6 yr and older)											
105	18-Apr-94	30-Jul-00	SoCal	M	Unknown, missing	Unknown	NP	ND	NA	NA	NA
102	28-Mar-94	24-Sep-00	SoCal	M	Unknown, scavenged	Unknown	NP	ND	NA	NA	NA
101	28-Apr-94	6-Oct-00	SoCal	F	Unknown, autolysis	None	NP	ND	120	<1	44.9
100	27-Mar-94	19-Sep-02	SoCal	M	Unknown, missing	Unknown	NP	ND	NA	NA	NA
12	1-Jan-76	13-Feb-03	SoCal	F	Gunshot (USFWS Forensics Lab)	Unknown	NP	ND	NA	NA	NA
170	23-May-97	15-Jun-03	CenCal	M	Toxicosis, lead (antemortem diagnosis, under treatment)	Visceral gout associated with chelation therapy	NP	ND	12	<1	19.8
6	1-Jan-76	27-Sep-05	SoCal	M	Poor body condtion	Unknown	NP	ND	NA	NA	NA
164	19-Apr-97	30-Sep-05	CenCal	M	Unknown, scavenged	Unknown	NP	ND	NA	NA	NA

Table 1. Continued.

SB	Hatch date	Death date	Release location	Sex	Primary cause of death	Additional findings	Gastric foreign bodies	WNV	Liver[b] (mg/kg, ww)		
									Copper	Lead	Zinc
245	25-Apr-01	14-Aug-07	SoCal	F	Toxicosis, lead (antemortem diagnosis, under treatment)	Visceral gout associated with chelation therapy	NP	ND	53.8	2	23.1
238	28-Mar-01	10-May-08	SoCal	M	Toxicosis, lead (antemortem diagnosis, under treatment)	Visceral gout associated with chelation therapy	One nonlead metallic pellet in antemortem x-ray	ND	11	<1	24
278	24-Apr-02	25-Jun-08	CenCal	M	Unknown, missing	Brush fire	NP	ND	NA	NA	NA
286	16-May-02	11-May-09	CenCal	M	Toxicosis, lead (antemortem diagnosis, under treatment)	Pneumonia, endocarditis, poor body condition; gunshot.	NP	ND	12	<1	18
303	22-Apr-03	23-Oct-09	CenCal	F	Toxicosis, lead (antemortem diagnosis, under treatment)	None	NP	ND	24	3.7	2.1

[a] SB = Studbook number; SoCal = Southern California; CenCal = Central California; M = male; F = female; Unk = unknown; USFWS = United States Fish and Wildlife Service; NA = not applicable; ND = not done; ww = wet weight; Pos = positive; Neg = negative.

[b] Results in bold from kidney.

[c] NP = not present.

[d] ND = not done.

TABLE 2. California Condor mortalities in Arizona 1996–2010.[a]

SB	Hatch date	Death date	Sex	Primary cause of death	Additional findings	Gastric foreign bodies	WNV	Liver[b] (mg/kg, ww)		
								Copper	Lead	Zinc
Nestlings (<6 mo)										
527	7-May-09	21-Jul-09	Unk	Unknown, missing	Unknown	NP[c]	ND[d]	NA	NA	NA
Juveniles (6 mo–5 yr)										
142	29-May-96	7-Jan-97	M	Trauma, golden eagle	None	NP	ND	29	<1	52
151	2-Jun-96	18-May-97	F	Power line collision, trauma	None	NP	ND	39.6	<1	64.1
128	19-Apr-95	14-Jul-97	F	Unknown, missing	Unknown	NP	ND	NA	NA	NA
169	20-May-97	3-Oct-98	M	Unknown, scavenged	Unknown	NP	ND	NA	NA	NA
177	28-Mar-98	24-Dec-98	M	Unknown, missing, suspected coyote predation	Unknown	NP	ND	NA	NA	NA
124	4-Apr-95	13-Mar-99	F	Gunshot	Unknown	NP	ND	ND	ND	ND
207	4-May-99	15-Jan-00	M	Aspiration pneumonia and airsacculitis, idiopathic	Septicemia (bacterial rods)	NP	ND	23.7	<1	27.9
197	24-Mar-99	4-Feb-00	F	Suspected golden eagle attack	None	NP	ND	94.6	<1	29.6
116	13-Apr-95	2-Mar-00	M	Toxicosis, lead (USFWS Forensics Lab)	Unknown	NP	ND	NA	32	NA
211	23-May-99	21-Apr-00	Unk	Unknown, missing	Unknown	NP	ND	NA	NA	NA
165	20-Apr-97	12-Jun-00	M	Toxicosis, lead	None	12 Lead shotgun pellets (2 sizes)	ND	**25.3**	**34**	**35.8**
191	10-May-98	16-Jun-00	F	Toxicosis, lead	Anemia, poor body condition, elevated liver copper	NP	ND	**181**	**17**	**178**
182	2-Apr-98	22-Jun-00	F	Unknown, scavenged	Unknown	NP	ND	NA	NA	NA
150	29-May-96	23-Jun-00	F	Unknown, missing	Unknown	NP	ND	NA	NA	NA
184	11-Apr-98	9-Sep-00	F	Unknown, scavenged	Unknown	NP	ND	61.4	<1	52.7
228	28-Apr-00	9-Feb-01	F	Poor body condition	Elevated liver zinc and lead	NP	ND	85.6	1	116
252	11-May-01	22-Feb-02	M	Unknown, scavenged	Unknown	NP	ND	NA	NA	NA
240	11-Apr-01	26-Aug-02	M	Unknown	Unknown	Corroded lead pellets in body cavity (USFWS Forensics Lab)	ND	NA	NA	NA

TABLE 2. Continued.

SB	Hatch date	Death date	Sex	Primary cause of death	Additional findings	Gastric foreign bodies	WNV	Liver[b] (mg/kg, ww)		
								Copper	Lead	Zinc
186	15-Apr-98	31-Aug-02	M	Shot by arrow (USFWS Forensics Lab)	Unknown	NP	ND	NA	NA	NA
258	25-May-01	25-Oct-02	M	Gunshot (USFWS Forensics Lab)	Unknown	NP	ND	NA	NA	NA
198	31-Mar-99	16-Sep-03	M	Unknown, autolysis	None	Piece of black rubber hose	ND	45.9	<1	36.6
176	19-Mar-98	11-Feb-04	F	Unknown, missing	Unknown	NP	ND	NA	NA	NA
235	18-May-00	12-Jan-05	F	Toxicosis, lead	None	Asphalt or rock	ND	49.2	24	28.2
249	9-May-01	23-Jan-05	M	Toxicosis, lead	None	2 lead shotgun pellets	ND	30.7	48	40.6
305	3-May-03	29-Mar-05	M	Poor body condition	Elevated liver lead	NP	ND	49.7	2.8	65.3
347	17-May-04	9-Apr-05	M	Poor body condition (possible poor foraging skills or feeding response)	None	NP	ND	88.6	<1	162
300	18-Apr-03	20-May-05	F	Unknown, missing	Unknown	NP	ND	NA	NA	NA
291	27-Mar-03	1-Nov-05	M	Unknown, autolysis	Unknown	NP	ND	14.8	<1	37.8
304	24-Apr-03	16-Mar-06	M	Toxicosis, lead (antemortem diagnosis, under treatment)	Poor body condition, crop stasis, elevated liver copper	1 metallic pellet in antemortem x-ray	ND	133	2.7	127
353	7-Jun-04	10-Jun-06	F	Unknown, missing	Unknown	NP	ND	NA	NA	NA
248	8-May-01	10-Jan-07	F	Toxicosis, lead	None	Lead ammunition fragment	ND	27.6	32	39.5
343	13-May-04	March 07	F	Unknown, missing	Unknown	NP	ND	NA	NA	NA
281	2-May-02	23-Apr-08	F	Trash ingestion with secondary zinc toxicosis	Possible renal gout and poor body condition	8 pennies, 2 penny fragments, one dime, and one quarter	ND	2.2	<1	1800
384	15-May-05	7-Dec-08	M	Unknown, missing, suspected predation by coyote	Unknown	NP	ND	NA	NA	NA
372	28-Apr-05	23-Mar-09	F	Unknown, scavenged, suspected coyote predation	Unknown	NP	ND	NA	NA	NA
134	2-Apr-96	Spring '09	M	Unknown, missing	Unknown	NP	ND	NA	NA	NA
391	3-Jun-05	2-May-09	F	Unknown, not submitted, suspected coyote predation	Unknown	NP	ND	NA	NA	NA
515	18-Apr-09	26-Dec-09	Unk	Unknown, missing	Unknown	NP	ND	NA	NA	NA

TABLE 2. Continued.

SB	Hatch date	Death date	Sex	Primary cause of death	Additional findings	Gastric foreign bodies	WNV	Liver[b] (mg/kg, ww)		
								Copper	Lead	Zinc
Adults (6 yr and older)										
82	4-Apr-92	29-Dec-00	F	Trauma, predator, coyote	None	NP	ND	21.6	<1	59.3
74	20-May-92	29-Dec-00	M	Poor body condition, ventricular candidiasis	Predator trauma, coyote	NP	ND	41.3	<1	94.7
149	7-May-96	20-Mar-06	F	Toxicosis, lead (antemortem diagnosis, under treatment)	Poor body condition, crop stasis	NP	ND	100	5.2	47.4
196	20-Mar-99	17-Jul-06	F	Unknown, missing	Unknown	NP	ND	NA	NA	NA
119	15-Mar-95	29-Dec-06	F	Toxicosis, lead (antemortem diagnosis, under treatment)	Crop stasis	Latex glove, 10–12 metallic pellets in antemortem x-ray	ND	36.5	5	21.6
232	30-Apr-00	3-Jan-07	F	Toxicosis, lead (antemortem diagnosis, under treatment)	Crop stasis, mild aspiration pneumonia	NP	ND	30.5	3	21.7
227	28-Apr-00	12-Jan-07	M	Toxicosis, lead (antemortem diagnosis, under treatment)	Aspiration pneumonia	15 metallic pellets in antemortem x-ray	ND	25.2	3	16.8
136	12-May-96	3-May-07	F	Trash ingestion with secondary zinc toxicosis	Visceral gout, hemocoelom, poor body condition	Penny fragments and glass	ND	3.2	<1	1770
276	23-Apr-02	Spring '09	M	Unknown, missing	Unknown	NP	ND	NA	NA	NA
127	31-Mar-95	23-Dec-09	F	Toxicosis, lead	Possible visceral gout	NP	ND	31	62	25

[a] SB = Studbook number; M = male; F = female; Unk = unknown; USFWS = United States Fish and Wildlife Service; NA = not applicable; ND = not done; ww = wet weight; Pos = positive.

[b] Results in bold from kidney.

[c] NP = not present.

[d] ND = not done.

TABLE 3. California Condor mortalities in Baja California 2003–2010.[a]

SB	Hatch date	Death date	Sex	Primary cause of death	Additional findings	Gastric foreign bodies	Liver (mg/kg, ww)		
							Copper	Lead	Zinc
Nestlings (<6 mo)									
437	20-Apr-07	21-May-07	Unk	Unknown, missing	Unknown	NP[b]	NA	NA	NA
531	14-May-09	20-Oct-09	Unk	Trash ingestion	Unknown	NP	NA	NA	NA
Juveniles (6 mo–5 yr)									
279	28-Apr-02	Nov. 2004	M	Unknown, missing	Unknown	NP	NA	NA	NA
338	4-May-04	7-Feb-06	Unk	Unknown, scavenged	Unknown	NP	NA	NA	NA
259	26-May-01	7-Jun-06	M	Toxicosis, lead	Focal encephalitis	22-caliber lead bullet	18.5	98	33
315	19-May-03	22-Jul-06	M	Unknown, scavenged	Unknown	NP	NA	NA	NA
390	28-May-05	14-Nov-07	Unk	Unknown, scavenged	Unknown	NP	28	<1	39
325	9-Apr-04	6-Dec-07	M	Toxicosis, lead (antemortem diagnosis, under treatment)	Visceral gout associated with chelation therapy	NP	7.9	<1	24
407	22-Apr-06	13-Dec-08	F	Unknown, scavenged	Unknown	NP	NA	NA	NA

[a] SB = Studbook number; M = male; F = female; Unk = unknown; NA = not applicable; ND = not done; ww = wet weight.

[b] NP = not present.

post-mortem ($n=5$). Condor 259 from Baja California died of lead toxicosis during treatment after accidental ingestion of a 22-caliber bullet not known to be present in a donkey carcass fed to the free-ranging birds. This bird also had evidence of focal inflammation of the brain, but West Nile virus testing was inconclusive due to advanced autolysis. In condor 422, feather analysis indicated at least four lead exposure events since release approximately 180 days earlier with one of the exposure events producing an estimated peak blood lead concentration of 300 μg/dl (Fig. 3). The final lead exposure event immediately prior to death resulted in an estimated blood lead concentration of 60 μg/dl. Three of the confirmed lead toxicosis mortalities from California and one from Baja California were complicated by visceral gout associated with chelation treatment. The only consistent histologic finding associated with lead toxicosis was Kupffer cell erythrophagocytosis and hemosiderosis.

There were 22 confirmed cases of trauma or other accident (PMR 29%; 22/76) with power line collision trauma or electrocution the most important cause (11 confirmed; 7 electrocutions and 4 collisions). Other causes of trauma included five confirmed predator trauma cases (two thought to be a coyote [*Canis latrans*] or other canid and one each of mountain lion (*Puma concolor*), Golden Eagle (*Aquila chrysaetos*), and undetermined).

Nine birds died from inanition, two of which had a history of poor feeding response after release. A third was killed by a coyote, but poor body condition and secondary ventricular candidiasis were

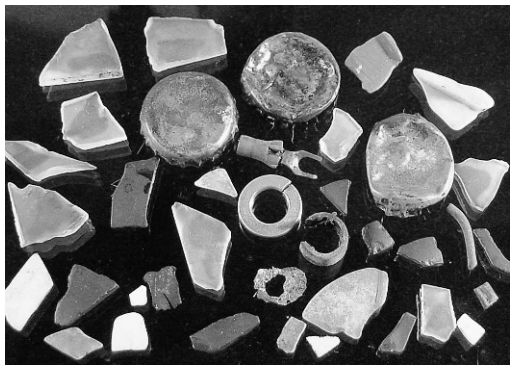

FIGURE 2. Gizzard content from condor 285. The presumed source of zinc toxicosis was the galvanized split-metal washer at center.

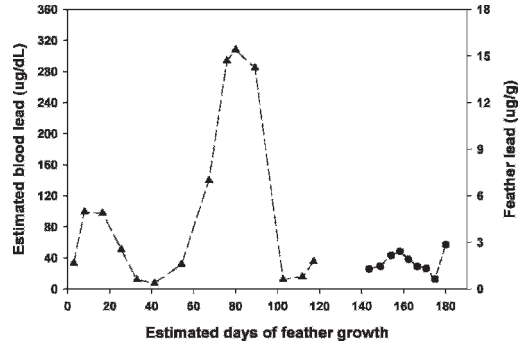

FIGURE 3. Condor 422's feather lead and estimated blood lead concentrations, versus estimated days of feather growth following release into the wild, for two primary feathers collected post-mortem, left primary 4 (LP4; triangles) and left primary 10 (LP10; circles). Feathers were estimated to have grown over different time periods following release into the wild and, combined, reflect ~160 days of growth and document four lead exposure events with one exposure resulting in an estimated blood lead of ~300 μg/dl (LP4). LP10 appeared to be growing at the time of death, and the most-recent feather section indicates condor 422 was exposed to lead just prior to death with a blood lead estimated at ~60 μg/dl. Blood lead concentrations were estimated from measured feather lead concentrations using a blood:feather lead concentration relationship of ~20:1 (Finkelstein et al., 2010). Days of feather growth were determined as described by Finkelstein et al. (2010).

considered the primary factors. Four birds were killed by gunshot and another by arrow. One of the gunshot birds died of idiopathic interstitial pneumonia while being treated for a severe, open, comminuted fracture of the distal right tarsometatarsus caused by the gunshot. One additional bird had evidence of nonfatal gunshot, with shotgun pellets embedded in the body and one wing. The only confirmed infectious disease was a West Nile virus infection, complicated by secondary respiratory aspergillosis, that was the cause of death in one subadult (450) that had been vaccinated against West Nile virus (see Chang et al., 2007). Two females had ingested a variety of small trash items (see Table 2), including zinc-core pennies, which led to secondary zinc toxicosis as the immediate cause of death. Nine birds, three of them nestlings, had hepatic copper concentrations more than four standard deviations above the mean for the captive flock, but none had lesions suggestive of copper toxicosis (e.g., inanition and bile stasis in the liver). Two of them had ingested metallic trash. One bird (260) had evidence of zinc exposure but with no ingested foreign bodies present at necropsy.

DISCUSSION

The leading causes of death at all California Condor release sites were anthropogenic and include lead toxicosis, power line collisions and electrocutions, trash ingestion, intentional killing, and West Nile virus infection. The most important mortality factor for the combined free-ranging populations was lead toxicosis with a PMR of 26% (13/50) for juveniles and 67% (10/15) for adults. The evidence that the principal source of exposure was lead ammunition is overwhelming and includes the recovery of lead shotgun pellets and bullet fragments from the upper GI tract where lead is readily absorbed (Mautino and Bell, 1986), tissue lead isotope signatures that match lead ammunition and not other sources of lead (Church et al., 2006; Finkelstein et al., 2010), confirmed exposures from a pig carcass containing spent ammunition (Finkelstein et al., 2010),

exposures coinciding with the hunting season and foraging activity in popular hunting areas (Hunt et al., 2007; Green et al., 2009; Parish et al., 2009), the high prevalence of bullet fragments in hunter-killed carcasses and gut piles from field-dressed kills (Hunt et al., 2006), a lack of other plausible sources of ingestible lead that would occur across such diverse habitats (e.g., Eisler, 1988), and the fact that mortality is rare from exposure to nonmetallic sources of lead (such as paint chips and ceramic glazes; Pattee et al., 1990). Although birds from California lacked recoverable lead ammunition in the GI tract post-mortem, lead ammunition has been recovered antemortem from two California birds that survived lead toxicosis with treatment (and are, therefore, not part of this study) and from another fatal lead toxicosis case after this study was completed. Three Arizona birds with metallic densities in the GI tract on antemortem radiographs had passed the fragments by the time of the post-mortem exam. Taken together, these findings suggest that many birds probably ingest minute lead fragments, found along wound channels (Hunt et al., 2006), which are then completely absorbed or partially absorbed and passed by the time the bird dies. A nonammunition source of lead remains a possibility in some cases, but no such source has yet been confirmed.

The number of lead-associated deaths would undoubtedly be higher if not for supplemental provision of lead-free calf carcasses at the California and Arizona release sites and intensive monitoring and repeated chelation treatment of the wild population for lead exposure. A specific chelation protocol was associated with four cases of visceral gout in this study. As a result, this protocol has been discontinued. In spite of these losses during therapy, chelation has been very successful overall as a treatment for acute lead toxicosis in California Condors. However, prerequisite monitoring and treatment requires substantial investment of resources that is not in accord with establishment of self-sustaining populations.

Power line collisions and electrocution were a frequent problem in California. Because of that, power pole aversion training began in 1994 in an attempt to keep birds away from power lines and to reduce perching on man-made objects (Mee and Snyder, 2007). Power pole aversion training gives birds a mild shock when landing on power pole replicas. Visual bird-flight diverters were also installed on power lines in high risk areas, but the effectiveness of visual diverters might be species-dependent (Martin and Shaw, 2010). One collision has occurred at a power line with diverters, but the number of deaths attributable to power line collisions and electrocution has declined significantly and no fatal collisions or electrocutions have occurred since 2007. The low number of power line fatalities noted in Arizona and Baja California is presumably due to the presence of fewer power lines in the release areas. Whether power line collisions and electrocutions contributed to the decline of the original wild population remains uncertain.

Parent-feeding of trash to nestlings was the most important cause of death in this age class. Clearing nest caves of trash prior to hatching, and periodically throughout the nestling stage, has reduced but not eliminated the occurrence of foreign body ingestion, which suggests that the majority of foreign material is brought by the adults to be fed to the nestlings (Mee et al., 2007). The reason(s) for this aberrant behavior remain open to speculation, but one plausible hypothesis is that it reflects misdirected attempts to provide bone or mollusk shell fragments as a calcium source for nestlings (Mee et al., 2007). Others have proposed that trash is fed to neonates as a substitute for small stones and sticks ("rangle") normally fed as an aid to digestion (Benson et al., 2004; Houston et al., 2007). Polished bone fragments are

now being provided as a calcium source for adults to feed to nestlings. Historic accounts suggest that this problem did not occur to this extent prior to the release program, although some trash was found in nests of the original wild population and the problem has been reported to occur in some Old World *Gyps* spp. vulture populations (Snyder et al., 1986; Mee et al., 2007). Trash ingestion by nestlings is, therefore, not likely to have been a factor in the original population decline.

West Nile virus infection is an emerging mortality factor for young, wild-hatched birds. Mitigating this risk is an ongoing challenge. An experimental DNA vaccine against West Nile virus has been used successfully in the institutional breeding flocks (Chang, 2007) and in wild birds, but it is not protective in all individuals. Although offspring of vaccinated adult females with high antibody titers have passive maternal immunity through yolk antibodies, such passive immunity would be waning in many nestlings before the peak of mosquito activity in summer, making them vulnerable to infection. Vaccination of nestlings will, therefore, continue to be required indefinitely at nest sites where West Nile virus is prevalent unless other prevention strategies become available.

The significance of the apparent elevation of hepatic copper concentrations in some birds also remains to be clarified. There was no definitive evidence of copper toxicosis in any individual, but the signs of copper toxicosis can be vague and nonspecific. One possible copper source would be livers from neonatal dairy calves provided as supplemental food in California and Arizona, as neonates tend to have higher hepatic copper and zinc concentrations than do adult cattle (Puschner et al., 2004a; Puschner et al., 2004b). However, if dairy calves were the source of copper, we would have expected to see a correlation between hepatic copper and zinc concentrations in these condors, and this was not observed. Another possible source of copper is ponds and water troughs for beef cattle, as these water sources are frequently treated with copper sulfate in California to control algal growth; but this would not explain the two juvenile birds from Arizona with apparent elevations in hepatic copper. Two nestlings with elevated hepatic copper had ingested metallic foreign bodies, some of which could have contained copper. However, metallic copper is poorly absorbed from the gastrointestinal tract in birds (Ledoux et al., 1991). This, combined with the lack of correlation between hepatic copper and lead, suggests that copper jacketing on bullets is not a likely source. Additional monitoring will be required to determine the sources and significance of hepatic copper accumulation in free-ranging condors.

The cause of death in one condor in California was ethylene glycol toxicosis (Murnane et al., 1995). The most common source of ethylene glycol is automobile antifreeze. However, propylene glycol was also present in the kidney sample from this individual. Because propylene glycol is not used in combination with ethylene glycol by any antifreeze manufacturer (Dow Chemical technical support, pers. comm., 2009), finding both compounds together suggests that a possible source of exposure was an industrial motor using ethylene glycol as a coolant and propylene glycol as a hydraulic fluid. Alternatively, exposure might have occurred through a carcass intentionally laced with ethylene glycol and pesticides (which might have used propylene glycol as a vehicle) intended to kill coyotes.

The number of birds reported missing or with unknown causes of death due to poor carcass condition at recovery is high, but may be unavoidable due to the remote locations and rugged conditions of the release sites, particularly the Baja California release site. These carcass-recovery difficulties introduce a sampling bias that could cause over-representation of mortality factors that operate in less-remote areas. Power line collisions and electrocutions, for

example, are far more likely to be encountered than most poisonings because of the proximity of power lines to roads. Determining whether additional mortality factors are operating in more-remote locations will require more intensive field monitoring and carcass recovery efforts.

In conclusion, the mortality factors thought to be important in the decline of the historic California Condor population, particularly lead poisoning, are still important. Because the risk of lead exposure from ammunition was likely just as great during the decline of the original wild population (see, for example, California Department of Fish and Game, 2010), these findings provide additional circumstantial evidence that lead toxicosis was the primary cause of the decline of the historic California Condor population through the mid- to late 20th century. The cumulative crude mortality rate for the free-ranging population was 38% during this study. In comparison, the cumulative crude mortality rate for the North American captive population was 8% (28 nestling and 10 juvenile or adult mortalities from a maximum population-at-risk of 485). Additional investigations will be required to determine annual population-specific mortality rates and their projected impact on population sustainability but, without effective mitigation, the principal anthropogenic mortality factors will likely continue to have the same impacts on population sustainability as they have in the past.

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LITERATURE CITED

Benson, P. C., I. Plug, and J. C. Dobbs. 2004. An analysis of bones and other materials collected by Cape Vultures at the Kransberg and Blouberg colonies, Limpopo Province, South Africa. Ostrich 75: 118–132.

California Department of Fish and Game. 2010. Hunting license statistics by decade, http://www.dfg.ca.gov/licensing/statistics/statistics.html. Accessed August 2011.

Chang, G. J., B. S. Davis, C. Stringfield, and C. Lutz. 2007. Prospective immunization of the endangered California Condors (*Gymnogyps californianus*) protects this species from lethal West Nile virus infection. Vaccine 25: 2325–2330.

Church, M. E., R. Gwiazda, R. W. Risebrough, K. Sorenson, C. P. Chamberlain, S. Farry, W. Heinrich, B. A. Rideout, and D. R. Smith. 2006. Ammunition is the principal source of lead accumulated by California Condors reintroduced to the wild. Environmental Science and Technology 40: 6143–6150.

De Francisco, N., J. D. Ruiz Troya, and E. I. Agüera. 2003. Lead and lead toxicity in domestic and free living birds. Avian Pathology 32: 3–13.

Eisler, R. 1988. Lead hazards to fish, wildlife, and invertebrates: A synoptic review. US Fish and Wildlife Service Biological Report 85 (1.14), 94 pp.

Finkelstein, M. E., D. George, S. Scherbinski, R. Gwiazda, M. Johnson, J. Burnett, J. Brandt, S. Lawrey, A. P. Pessier, M. Clark, J. Wynne, J. Grantham, and D. R. Smith. 2010. Feather lead concentrations and (207)Pb/(206)Pb ratios reveal lead exposure history of California Condors (*Gymnogyps californianus*). Environmental Science and Technology 44: 2639–2647.

Franson, J. C. 1996. Interpretation of tissue lead residues in birds other than waterfowl. *In* Environmental contaminants in wildlife: Interpreting tissue concentrations, W. N. Beyer, G. H. Heinz and A. W. Redmon-Norwood (eds.). CRC Press, Boca Raton, Florida, pp. 265–279.

Grantham, J. 2007. Reintroduction of California Condors into their historical range: The recovery program in California. *In* California Condors in the 21st Century, A. Mee, L. S. Hall and J. Grantham (eds.). Series in Ornithology, No. 2. American Ornithologists Union, Washington,

DC, and Nuttall Ornithological Club, Cambridge, Massachusetts, pp. 123–138.

GREEN, R., W. G. HUNT, C. N. PARISH, AND I. NEWTON. 2009. Effectiveness of action to reduce exposure of free-ranging California Condors in Arizona and Utah to lead from spent ammunition. *In* Proceedings of the conference: Ingestion of lead from spent ammunition: Implications for wildlife and humans, R. T. Watson, M. Fuller, M. Pokras and G. Hunt (eds.). The Peregrine Fund, Boise, Idaho, pp. 240–253.

HOUSTON, D. C., A. MEE, AND M. MCGRADY. 2007. Why do condors and vultures eat junk?: The implications for conservation. Journal of Raptor Research 41: 235–238.

HUNT, W. G., W. BURNHAM, C. N. PARISH, K. K. BURNHAM, B. MUTCH, AND J. L. OAKS. 2006. Bullet fragments in deer remains: Implications for lead exposure in avian scavengers. Wildlife Society Bulletin 34: 167–170.

———, C. N. PARISH, S. C. FARRY, T. G. LORD, AND R. SEIG. 2007. Movements of introduced California Condors in Arizona in relation to lead exposure. *In* California Condors in the 21st Century, A. Mee, L. S. Hall and J. Grantham (eds.). Series in Ornithology, No. 2. American Ornithologists Union, Washington, DC, and Nuttall Ornithological Club, Cambridge, Massachusetts, pp. 79–96.

INTERNATIONAL UNION FOR THE CONSERVATION OF NATURE (IUCN). 1998. Guidelines for reintroductions. Prepared by the IUCN/SSC reintroduction specialist group. IUCN, Gland, Switzerland and Cambridge, UK, 10 pp.

JANSSEN, D. L., J. E. OOSTERHUIS, J. L. ALLEN, M. P. ANDERSON, D. G. KELTS, AND S. N. WIEMEYER. 1986. Lead poisoning in free ranging California Condors. Journal of the American Veterinary Medical Association 189: 1115–1117.

KUEHLER, C. M., D. J. STERNER, D. S. JONES, R. L. USNIK, AND S. KASIELKE. 1991. Report on captive hatches of California Condors (*Gymnogyps californianus*): 1983–1990. Zoo Biology 10: 65–68.

LEDOUX, D. R., P. R. HENRY, C. B. AMMERMAN, P. V. RAO, AND R. D. MILES. 1991. Estimation of the relative bioavailability of inorganic copper sources for chicks using tissue uptake of copper. Journal of Animal Science 69: 215–222.

MARTIN, G. R., AND J. M. SHAW. 2010. Bird collisions with power lines: Failing to see the way ahead? Biological Conservation 143: 2695–2702.

MAUTINO, M., AND J. U. BELL. 1986. Experimental lead toxicity in the Ring-necked Duck. Environmental Research 41: 538–545.

MEE, A., AND N. F. R. SNYDER. 2007. California Condors in the 21st Century—Conservation problems and solutions. *In* California Condors in the 21st Century, A. Mee, L. S. Hall and J. Grantham (eds.). Series in Ornithology, No. 2. American Ornithologists Union, Washington, DC, and Nuttall Ornithological Club, Cambridge, Massachusetts, pp. 243–279.

———, B. A. RIDEOUT, J. A. HAMBER, J. N. TODD, G. AUSTIN, M. CLARK, AND M. P. WALLACE. 2007. Junk ingestion and nestling mortality in a reintroduced population of California Condors *Gymnogyps californianus*. Bird Conservation International 17: 119–130.

MURNANE, R. D., G. MEERDINK, B. A. RIDEOUT, AND M. P. ANDERSON. 1995. Ethylene glycol toxicosis in a captive-bred released California Condor (*Gymnogyps californianus*). Journal of Zoo and Wildlife Medicine 26: 306–310.

PARISH, C. N., W. G. HUNT, E. FELTES, R. SEIG, AND K. ORR. 2009. Lead exposure among a reintroduced population of California Condors in northern Arizona and southern Utah. *In* Proceedings of the conference: Ingestion of lead from spent ammunition: Implications for wildlife and humans, R. T. Watson, M. Fuller, M. Pokras and G. Hunt (eds.). The Peregrine Fund, Boise, Idaho, pp. 259–264.

PATTEE, O., P. BLOOM, J. SCOTT, AND M. SMITH. 1990. Lead hazards within the range of the California Condor. Condor 92: 931–937.

PULS, R. 1994. Mineral levels in animal health. Sherpa International, Clearbrook, Canada, pp. 294–296.

PUSCHNER, B., J. ST. LEGER, AND F. D. GALEY. 1999. Normal and toxic zinc concentrations in serum/plasma and liver of psittacines with respect to genus differences. Journal of Veterinary Diagnostic Investigation 11: 522–527.

———, Y. K. CHOI, J. H. TEGZES, AND M. C. THURMOND. 2004a. Influence of age, sex, and production class on liver zinc concentration in calves. Journal of Veterinary Diagnostic Investigation 16: 278–282.

———, M. C. THURMOND, AND Y. K. CHOI. 2004b. Influence of age and production type on liver copper concentrations in calves. Journal of Veterinary Diagnostic Investigation 16: 382–387.

SNYDER, N., AND H. SNYDER. 2000. The California Condor. A saga of natural history and conservation. Academic Press, San Diego, California, 410 pp.

———, R. R. RAMEY, AND R. C. SIBLEY. 1986. Nest-site biology of the California Condor. Condor 88: 228–241.

WILBUR, S. 1978. The California Condor, 1966–1976: A look at its past and future. United States Department of the Interior, Fish and Wildlife Service. North American Fauna 72: 1–142.

WIEMEYER, S. N., J. M. SCOTT, M. P. ANDERSON, P. H. BLOOM, AND C. J. STAFFORD. 1988. Environmental contaminants in California Condors. Journal of Wildlife Management 52: 238–247.

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