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ACUTE ORAL TOXICITY OF SODIUM CYANIDE IN BIRDS

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ABSTRACT: Sensitivities of six avian species, black vulture (*Coragyps atratus*), American kestrel (*Falco sparverius*), Japanese quail (*Coturnix japonica*), domestic chicken (*Gallus domesticus*), eastern screech-owl (*Otus asio*), and European starling (*Sturnus vulgaris*), to acute poisoning by sodium cyanide (NaCN) were compared by single dose LD50's. Three species, domestic chickens, black vultures, and turkey vultures (*Cathartes aura*), were dosed with NaCN to determine cyanide residues in those that died and also in survivors, in addition to postmortem fate. Three flesh-eating species (black vulture, American kestrel, and eastern screech-owl; LD50's 4.0–8.6 mg/kg) were more sensitive to NaCN than three species (Japanese quail, domestic chicken, and European starling; LD50's 9.4–21 mg/kg) that fed predominantly on plant material. Elevated concentrations of cyanide were found in the blood of birds that died of cyanide poisoning; however, concentrations in birds that died overlapped those in survivors. Blood was superior to liver as the tissue of choice for detecting cyanide exposure. No gross pathological changes related to dosing were observed at necropsy.

INTRODUCTION

Cyanides are highly toxic compounds. They are readily absorbed and cause death by preventing the use of oxygen by tissues (Guatelli, 1964; Ballantyne, 1974; Egekeze and Oehme, 1980). Deaths of wild birds from cyanide poisoning appear to have occurred through several routes of exposure. Dead burrowing owls (*Athene cunicularia*) were found at entrances of black-tailed prairie dog (*Cynomys ludovicianus*) burrows treated with calcium cyanide (Wade, 1924). Bald eagles (*Haliaeetus leucocephalus*) (Kaiser et al., 1980; Reichel et al., 1984) and golden eagles (*Aquila chrysaetos*) (Reidinger and Crabtree, 1974) have died from cyanide poisoning. Sodium cyanide (NaCN) from coyote getters and M-44's (cyanide ejector mechanisms used in predator control) has caused the death of several avian species including magpies (*Pica* sp.), ravens and a crow (*Corvus* spp.), vultures (species not given), wild turkeys (*Meleagris gallopavo*), eagles, and an unidentified hawk (Robinson, 1943; Beasom, 1974; Matheny, 1976; U.S. Fish and Wildlife Service, 1979). More recently, a California condor (*Gymnogyps californianus*) was exposed to NaCN from an M-44 (U.S. Fish and Wildlife Service, Patuxent Wildlife Research Center, Laurel, Maryland, unpubl.). Cameron (1972) reported a bird-kill that was possibly caused by the ingestion of cyanogenic plant material. Cyanide poisoning of chickens from ingestion of cherry pits has been implied (Smith and Jones, 1957).

The lethal toxicity of several forms of cyanide to birds has been reported; however, exposure details and LD50's were usually not provided. Hunt-Boston (1923) reported the fatal dose of hydrogen cyanide (HCN) in rock doves (*Columba livia*) to be 1.5 mg/kg. Spector (1956) summarized the toxicity of various forms of cyanide via several routes of exposure. The lethal dose of HCN to birds via subcutaneous exposure was 0.1 mg/kg, whereas the minimum lethal dose of potassium cyanide (KCN) to rock doves via intramuscular (i.m.) and intravenous (i.v.) injection was 4 mg/kg. Barcroft (1931) exposed chickens, rock doves, and canaries to HCN in the air and plotted the relationship between dosage and length of exposure. When all three species were exposed to

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TABLE 1. Single-dose lethal oral toxicity of reagent grade sodium cyanide to adult birds.

Species	Median weight (g)	Sex	No. dosages tested	Birds/ dosage	LD50[a,b]	95% CI	Slope[c]	SE
Black vulture (BV)	2,215	M and F	4	3	4.8	4.4–5.3	23.2	15.0
American kestrel (AK)	118	M and F	4	5	4.0	3.0–5.3	20.6	10.8
Japanese quail (JQ)	130	M and F	5	20	9.4	7.7–11.4	4.1	0.7
	124	M	5	10	10.3	7.5–14.1	4.2	1.0
	148	F	5	10	8.5	5.9–12.2	4.0	0.9
Domestic chicken (DC)	1,610	F	4	3	21	12–36	3.8	1.8
Eastern screech-owl (SO)	185	M and F	4	5	8.6	7.2–10.2	7.1	3.3
European starling (ES)	75	M and F	5	10	17	14–22	4.4	1.0
	78	M	5	5	17	9–32	5.2	1.7
	72	F	5	5	18	11–30	3.8	1.3

[a] LD50: mg NaCN per kg body weight in a single oral dose calculated to kill 50% of the test population.
[b] Statistical separation: DC = ES > JQ = SO > BV = AK ($P < 0.05$).
[c] Slope: probit on log dose.

0.12 mg/liter, chickens survived for 60 min (the maximum time recorded), pigeons died in 10 min, and canaries died in 3 min. Davis (1981) found that a dose of 1.5 mg/kg KCN was lethal to anesthetized chickens when administered intravenously. The LD50 or repellency (R50) of other chemicals containing cyanide or thiocyanates, including phenyl cyanide and hexyl cyanide, was reported for several species of birds by Schafer et al. (1983).

The death of the California condor exposed to NaCN, the lack of acute toxicity data for NaCN in birds, and the absence of data on cyanide residues, including its postmortem fate, in tissues of birds led us to conduct the studies reported herein.

MATERIALS AND METHODS

A range-finding study was conducted with domestic chickens to determine the approximate acute toxicity of NaCN to a species similar in weight to the turkey vulture, the primary experimental model. This study also established a basis for subsequent acute tests with diverse captive avian species. Test doses of reagent grade sodium cyanide (99.4% AI) were administered to three birds each at 6, 12, 24, and 48 mg/kg body weight via gelatin capsule delivered to the proventriculus. Chickens were observed until death or for 30 min, bled by cardiac venipuncture, and blood and livers were analyzed for cyanide residues. Analyses were initiated on the day of exposure and death. The killing time of 30 min was arbitrarily assigned to approximate a reasonable time to death after acute exposure, and to minimize alteration and excretion of absorbed cyanide. An LD50 was estimated from observed deaths and projected deaths based on clinical signs at time of killing (Table 1).

Two black vultures and two turkey vultures were dosed with NaCN using similar methods. Bird weights varied from 1.94 to 2.30 kg. Dose rates for black vultures were 16 and 25 mg/kg and for turkey vultures were 25 and 36 mg/kg. Dose rates were established with the intent that they would be lethal. The response of vultures was observed at these exposures to ensure that a lethal rate would be selected for the subsequent dosing of turkey vultures. Blood samples from the heart and livers were collected immediately following death, frozen, and analyzed for cyanide residues 3 wk later.

Fifteen turkey vultures trapped from the wild were assigned randomly to one of three treatments. Weights of birds varied from 1.92 to 2.26 kg. Birds were deprived of food for a minimum of 16 hr before experimentation. The birds in two treatments were given NaCN at 36 mg/kg body weight via gelatin capsule to the proventriculus. A blood sample was collected from the brachial vein of each bird immediately before treatment and analysis was initiated the same day. In the first treatment, blood (from the heart) and liver were obtained from the birds immediately following death by cyanide poisoning and analysis for cyanide was initiated the same day. In the second treatment,

birds killed with cyanide were stored for 12 hr at 20 C followed by 12 hr at 2 C. Blood samples (from heart and thoracic cavity) and liver were removed and frozen at −20 C for analysis 45 days later. These storage conditions were established to mimic storage of the dead California condor that was exposed to an M-44. Birds in the third treatment served as controls. They were asphyxiated by CO_2; storage conditions were the same as for the second treatment. One bird from each treatment was killed on each of 5 days. Each dosed bird was placed in a small holding cage immediately postexposure where it was observed for the time of onset and progression of signs of poisoning. Birds were recorded as dead when respiration ceased and no corneal response was detected. Samples were collected for analysis and sex was determined at the time gross necropsies were conducted. A one way analysis of variance was used to determine if there were significant differences among or between treatment means. Statistical significance was set at $\alpha = 0.05$.

Thirteen additional wild-trapped black vultures were given graded doses of NaCN via gelatin capsule in the proventriculus to estimate an LD50 in this species and to obtain data on cyanide concentrations in blood in relation to exposure and survival or death. Three birds each were given 3, 7, and 36 mg/kg, and four were given 4.5 mg/kg. The highest dose was used to compare times to death with turkey vultures given the same dose as described above. A blood sample was taken from the brachial veins from three birds immediately before dosing and from the heart shortly following death, or from the brachial vein of survivors after 1 hr. Analyses of blood were initiated on the day of sampling. Birds were observed as in the previous turkey vulture study. Necropsies were performed and sex was determined. Survivors were killed with CO_2 2 hr postexposure.

American kestrels, Japanese quail, eastern screech-owls, and European starlings were subjected to acute NaCN poisoning for determination of single dose LD50 values. Kestrels, quail, and owls were produced in captive colonies and starlings were trapped from the wild. The quail were reproductively active at the time of dosing, whereas other species were not. The experimental methods were similar to those described by Hill and Camardese (1984). The number of dose concentrations and birds per dose are given in Table 1. All birds were observed until death or until remission of clinical signs of toxicosis. The LD50 values and associated statistics (95% confidence interval; slope and standard error of the probit regression curve) were derived by probit analysis (Finney, 1971). LD50 values were considered significantly different ($P < 0.05$) when the 95% confidence intervals did not overlap (Finney, 1971).

Blood samples were collected from two captive California condors (controls) at the Los Angeles Zoo on 29 August 1984. They were frozen and shipped to the Patuxent Wildlife Research Center for cyanide analysis. Samples were held at −20 C for 3 wk, thawed, and analyzed on 19 September 1984.

Chemical methodology

A gas-liquid chromatographic method (Valentour et al., 1974) was modified to quantify unmetabolized (free) cyanide in blood and liver (Krynitsky et al., 1986). Cyanide was separated from homogenized blood and liver using an acidified microdiffusion cell with sodium hyroxide solution in a central reservoir of the cell as an absorbant. Cyanide was converted to cyanogen chloride. A Hewlett-Packard Model 5713 gas chromatograph equipped with a ^{63}Ni electron capture detector, and a 1.83-m × 6-mm i.d. Halcomid M-18 on 100/120-mesh Supelcoport glass column was used in quantifications. The average percent recovery of cyanide was 91% (SD = 8.9) for blood fortified with 1 ppm NaCN.

RESULTS

All NaCN dosed chickens showed signs of toxicosis. At 6 mg/kg the responses commenced about 10 min postexposure, were comparatively mild (e.g., panting, eye-blinking, salivation and lethargy), and were clearly sublethal. Signs of toxicosis were observed also in birds dosed with 48 mg/kg beginning about 10 min postexposure, but the signs intensified over time. Birds dosed with 12 and 24 mg/kg responded predictably as intermediate dosages when compared to 6 and 48 mg/kg. The estimated LD50 was 21 mg/kg (Table 1). Concentrations of cyanide in blood were similar among dose concentrations (Table 2), ranging from 0.70 to 1.6 ppm. The maximum concentration of cyanide in liver was generally one-hundredth of the exposure rate; however, many samples had no detectable cyanide.

In the preliminary study with vultures,

black vultures died in 11 (16 mg/kg) and 8 (25 mg/kg) min, whereas turkey vultures died in 27 (25 mg/kg) and 9 (36 mg/kg) min. Based on the exposure rates and times to death, an exposure rate of 36 mg/kg was selected for the major residue study with turkey vultures. Residues of cyanide in blood from these birds frozen for 3 wk were 4.2 and 6.6 ppm for black vultures, and 3.9 and 4.8 ppm for turkey vultures. Only one liver sample, the black vulture dosed with 16 mg/kg, contained a detectable concentration (0.51 ppm) of cyanide.

All turkey vultures in the residue study died between 8 and 41 min after dosing (Table 3). The time to death between the two dosed groups was not significantly different. Although the signs of toxicosis following exposure were somewhat variable, a general pattern was observed. An early period characterized by slight incoordination, rapid eye-blinking, head-bowing, and wing-droop, was followed by loss of coordination and convulsions resulting in the birds lying in various positions and exhibiting tail fanning and opisthotonos. Breathing became increasingly deep and labored and was followed by gasping, shallow intermittent breathing, and death. All birds were in good flesh with moderate to heavy deposits of subcutaneous and abdominal fat. Weights of birds were not significantly different among treatments. Sex ratios were similar among treatments. No gross pathological changes related to dosing were seen at necropsy.

Cyanide was not detected in pretreatment blood samples or in postmortem blood from control turkey vultures. A significant difference occurred between the two dosed groups in postmortem cyanide concentrations in blood; the mean concentration in stored samples was nearly five times that in samples analyzed immediately postmortem. Cyanide concentrations in livers were not significantly different among treatments or between the two dosed groups.

TABLE 2. Cyanide concentrations in blood and liver of chickens dosed with sodium cyanide.

Dose (mg/kg)[a]	Cyanide residues (ppm wet weight)	
	Blood	Liver
0	n.d.[b]	n.d.
6	0.95 (0.84–1.1)[c]	0.04 (n.d.–0.06)
12[d]	1.3 (1.1–1.6)	0.04 (n.d.–0.12)
24[e]	1.1 (0.70–1.4)	0.10 (n.d.–0.24)
48[f]	1.2 (0.90–1.5)	0.26 (n.d.–0.56)

[a] n = 3 per dosage.
[b] n.d. = none detected.
[c] Range in parentheses.
[d] Bird with highest concentration in blood died during cardiac venipuncture.
[e] Bird with highest concentration in blood died of cyanide poisoning at 6 min post-dosage.
[f] All birds had advanced signs of acute poisoning and probably would have died if not killed at 30 min.

The black vultures, kestrels, owls, and quail reacted much more violently to NaCN exposure than did the chickens or the starlings. The first signs of toxicosis on nearly all dosages occurred between 30 sec and 5 min postexposure. Death usually followed in 15–30 min. Birds alive at 1 hr usually recovered. The order of species sensitivity to acute NaCN poisoning was American kestrel = black vulture > eastern screech-owl = Japanese quail > European starling = domestic chicken (Table 1). There were no sex differences to acute NaCN poisoning for quail and starlings; sex differences were not tested in other species.

The sequence of clinical signs of cyanide poisoning in black vultures was similar to that in turkey vultures. The severity of signs in black vultures that survived was variable. In some cases the birds appeared to be near death with extremely labored breathing, whereas, in at least one other case, signs were very mild and only involved slight incoordination and tremors. The severity of signs of poisoning in sur-

TABLE 3. Time to death in turkey vultures dosed with 36 mg/kg sodium cyanide and cyanide concentrations in samples from control and dosed birds.

Treatment	Time to death (min)	Cyanide residues (ppm wet weight)		
		Blood		Liver
		Pre-dose	Postmortem	Postmortem
Dose-analyzed-immediately	17 A[a] (8–40)[c]	n.d.[b]	2.3 A (0.95–4.1)	0.02 A (n.d.–0.11)
Dose-stored 45 days[d]	21 A (8–41)	n.d.	11 B (2.2–21)	1.2 A (n.d.–2.8)
Control-stored 45 days	—[e]	n.d.	n.d.	n.d. A

[a] Means within columns that share a common capital letter were not significantly different from one another ($\alpha = 0.05$). $n = 5$.
[b] n.d. = none detected; a value of 0.05 ppm was assigned for statistical purposes where cyanide in liver was not detected.
[c] Range in parentheses.
[d] Unable to place cyanide capsule in proventriculus of one bird; left in esophagus.
[e] Killed with CO_2; time to death not applicable.

vivors appeared to be correlated positively to cyanide concentrations in blood collected 1 hr after exposure. Survivors were standing and reasonably alert 1 hr postdosage. The LD50 of NaCN for black vultures was estimated to be 4.8 mg/kg (Table 1). Mortality, time to death, and cyanide concentrations in postmortem blood from black vultures are reported in Table 4. Time to death was related inversely to dosage. Although cyanide residues in postmortem blood samples declined with lowered dosages, these changes were related also to time to death. There was a significant ($P < 0.05$) negative correlation between time to death (for all black vultures) and cyanide concentration in blood at death ($r = -0.83$; $n = 7$). Cyanide concentrations in postmortem blood from birds overlapped those in survivors. All birds that were necropsied were in good flesh. No gross abnormalities were noted in relation to exposure.

Cyanide was not detected in the blood in one of two living California condors at the Los Angeles Zoo. Blood from the other condor contained 0.06 ppm cyanide.

DISCUSSION

The progression of signs of toxicity leading to death in these birds was generally similar to those reported for mammals (Ballantyne, 1975, 1983; Egekeze and Oehme, 1979, 1980). Rock doves exposed to HCN via inhalation vomited before death whereas canaries and chickens did not (Barcroft, 1931); however, neither passerines nor galliforms have well developed vomiting reflexes. Even though vomiting was noted in two black vultures that eventually survived exposure, its prevalence was difficult to ascertain in these studies because birds were deprived of food before exposure.

Cyanide concentrations in blood or liver of dosed mammals at death have been reported in a number of studies. Laboratory rabbits dosed orally with 10–15 mg/kg NaCN had about 4 ppm cyanide in heart blood at death (Yamamoto et al., 1979). Rabbits given i.m. doses of 8.3 mg/kg HCN or 20 mg/kg KCN had 6.9 and 4.5 ppm cyanide in blood and 1.5 and 0.8 ppm cyanide in liver (Ballantyne et al., 1972). Rabbits given i.m. doses of 8 mg/kg cyanide as HCN (Ballantyne et al., 1973) or 8 mg/kg cyanide as KCN (Ballantyne et al., 1974) had 16 μg/ml and 5–6 μg/ml cyanide in blood, respectively, at death, and 0.1–0.4 ppm cyanide in liver in the latter study. Ballantyne (1975) reported a mean of 3.3 μg/ml cyanide in

TABLE 4. Body weights, dose rates, mortality, and postmortem cyanide concentrations in blood of black vultures dosed with sodium cyanide.

Dose (mg/kg)	n[a] M	n[a] F	Body weight (kg)	No. died	Time to death (min)	Cyanide residues (ppm wet weight) Died	Cyanide residues (ppm wet weight) Survived[b]
3	2	1	2.31	0	—	—	0.69 (0.30–0.94)[c]
4.5[d]	2	2	2.22	1	30	0.74	1.2 (1.1–1.3)
7		3	2.18	3	15.7 (14–18)	2.0 (1.8–2.1)	—
36		3	2.15	3	11.3 (8–14)	2.8 (1.9–4.2)	—

[a] Sample size; M = male, F = female.
[b] Samples collected 1 hr after dosage.
[c] Range in parentheses.
[d] Blood from only three birds analyzed.

blood of domestic sheep that were killed with KCN, after i.m. exposure of 10 mg/kg cyanide. Blood from laboratory rats, collected at death from cyanide poisoning, had means of 4.5–5.0 μg/ml cyanide following oral exposure to NaCN and 2.9–3.1 μg/ml cyanide following inhalation of HCN (Yamamoto et al., 1982). Egekeze and Oehme (1979) dosed rats orally with KCN at 6, 10, and 14 mg/kg body weight. The minimum concentration of cyanide in blood indicative of lethal exposure (individuals were killed when respiration ceased) was 2.6–2.9 μg/ml. The maximum concentration in the blood of a rat that died was 5 μg/ml. Liver concentrations were highly variable in those that died (0.5–6.1 ppm) and were attributed to the varying efficiency of individuals to detoxify cyanide. Liver concentrations of cyanide were considered unreliable for diagnostic purposes, because of the variability and the overlap in concentrations between those that died and those that survived. The endogenous blood cyanide concentration was 0.3 μg/ml. Cyanide concentrations in blood of rabbits at death were lowest when exposure was by inhalation rather than for five other routes, and five mammalian species had similar cyanide concentrations in blood at death when given lethal i.m. injections of KCN (Ballantyne, 1983); liver concentrations generally exceeded 0.5 ppm.

Cyanide concentrations in blood of vultures at death (samples analyzed immediately) generally were slightly lower than the concentrations found in blood of mammals at death. Cyanide concentrations in livers of our turkey vultures (analyzed immediately) at death were low in relation to those reported for mammals. No detectable cyanide was found in the pre-dose blood samples, whereas clearly detectable concentrations were found in non-exposed rats (Egekeze and Oehme, 1979) and man (Ballantyne, 1977 and others).

Marked differences in toxicity of cyanide among species of birds were found. Species differences in sensitivity to NaCN in these studies were not related to body size, but seemed to be associated with diet. The three predominantly flesh eating species gave lower LD50's than those that fed predominantly on plant material. To illustrate: the general order of body size of these test species was black vulture > chicken > eastern screech-owl > Japanese quail > American kestrel > Euro-

pean starling, whereas the order of sensitivity to cyanide was kestrel > vulture > owl > quail > starling > chicken (Table 1). The three flesh eaters had the steepest dose-response lines of the six species tested. Such relationships generally indicate a high degree of susceptibility to poisoning, because proportionally smaller increases of exposure are associated with increased reaction. Black vultures appeared to be more sensitive than turkey vultures based on time to death when both species were exposed to 36 mg/kg.

Cyanide concentrations in the blood of black vultures that died increased with higher dose rates; however, this relationship was not noted in chickens, perhaps because most samples were taken before death. Although an inverse relationship between time to death and cyanide concentration at death in black vultures (possibly confounded by dose rates) was noted, this relationship was not observed in turkey vultures, sheep (Ballantyne, 1975) or rats (Egekeze and Oehme, 1979; Yamamoto et al., 1982).

The rapid recovery of some birds exposed to cyanide, even those showing extreme signs of exposure, may be due to the rapid metabolism of cyanide to thiocyanate and its subsequent excretion. Davis (1981) found a rapid rise in urinary thiocyanate excretion following a single i.v. dose of cyanide in anesthetized chickens where urine flow was increased by a diuretic. The peak excretion rate occurred 30 min following dosage, with a recovery of 50% of the dose over 6 hr. Less than 0.1% of the dose was excreted as cyanide. Rats that survived exposure from KCN exhibited normal behavior within 1 hr after dosing (Egekeze and Oehme, 1979), similar to the surviving black vultures.

The higher concentrations of cyanide detected in blood and liver of turkey vultures in the dose-stored treatment over those observed in the dose-analyzed-immediately treatment may be due to several factors. First, difficulty was encountered in obtaining adequate blood samples from the heart of birds in the dose-stored treatment; therefore, blood found in the thoracic area also was collected. Yamamoto et al. (1982) suggested that there may have been postmortem diffusion of cyanide from the stomach of orally dosed rats. Postmortem diffusion of cyanide into liver and blood of turkey vultures cannot be discounted. Second, thiocyanate may be converted to cyanide ". . . a reaction which is catalyzed by free hemoglobin liberated as a consequence of mechanical hemolysis due to the freeze-thaw process" (Ballantyne, 1983). However, the increase in the turkey vultures was far higher than expected in blood from the freeze-thaw process alone as shown by Krynitsky et al. (1986), where increases in samples frozen once did not exceed 67% and repeated refreezing and thawing did not result in increases greater than 150%. The mean concentration of cyanide in blood of the dose-stored samples was more than 4.5 times the concentration in the dose-analyzed-immediately samples from the turkey vultures. Cyanide concentrations in human blood from individuals not exposed to cyanide have increased markedly (two to four times) following freezing (Ballantyne, 1976, 1977); however, such increases were not noted following freezing in blood to which cyanide had been added (Pettigrew and Fell, 1973; Ballantyne, 1976). Unfortunately, data on the effects of freezing on concentrations of cyanide in blood from dosed animals other than those of Krynitsky et al. (1986) are unknown. The low concentrations of cyanide in liver samples from dosed birds may be due to the presence of blood in the liver (Ballantyne et al., 1972).

The California condor that was exposed to NaCN from an M-44 in November 1983 had 0.09 ppm cyanide in its blood and none in its liver; samples were removed from the bird about 24 hr following death,

stored for 45 days at −20 C and analyzed. The concentration in blood was similar to that found in blood of one of two living California condors and only slightly above the lower limit of detection (0.05 ppm). This amount was far below concentrations in dosed vultures that died, and somewhat below those that survived a low dose. No cyanide was found in fresh pretreatment blood from the vultures in these studies or in two samples that were reanalyzed following freezing (Krynitsky et al., 1986). Although a moderate loss (<33%) of cyanide occurred in blood of rabbits dosed with cyanide following 1 day of storage at 10–15 C, either when samples were removed at death and stored 1 day or when carcasses were stored and samples removed 1 day later, the concentrations (>3 ppm) were still clearly elevated and indicative of cyanide exposure (Ballantyne et al., 1974). The low concentration of cyanide in the blood of the exposed California condor may represent either a sublethal exposure or postmortem cyanide formation from the freeze-thaw process. However, cyanide as the cause of death of the California condor cannot be entirely ruled out, because the route of exposure (possible inhalation rather than ingestion) may have been different than in these studies, and the dose and time to death were unknown.

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