

Supporting Information

Finkelstein et al. 10.1073/pnas.1203141109

SI Results and Discussion

Condors Have High Blood Lead Exposure and Poisoning Rates Compared with Young Children. National blood lead screening data from children < 72 mo of age show that the percentage of tested children that exhibited an elevated blood lead level (i.e., > 100 ng/mL) or lead poisoning indicating chelation treatment (i.e., blood lead > 450 ng/mL) declined from 7.6% and 0.085%, respectively, in 1997 to 0.83% and 0.011%, respectively, by 2008 (1). Thus, median lead exposure prevalence rates in condors have exceeded median national rates in children over a similar period by ~30-fold, whereas the median annual prevalence rates of lead poisoning in condors have exceeded the median lead poisoning rates in children by over 400-fold (i.e., 71% vs. 2.42% and 20% vs. 0.045%, respectively).

Factors Influencing Blood Lead Concentrations in Condors. Uncontaminated condor diet items (e.g., mule deer, dairy calves, and California sea lions) have been shown to be very low in lead (2–68 ng/g dry weight) and possess lead isotopic signatures consistent with background environmental lead in California (2); thus, they cannot account for even the low to moderate blood lead levels (e.g., 100–200 ng/mL) observed in free-flying condors. These moderately elevated blood lead levels most likely reflect a relatively minor exposure to specific point sources of lead, such as minute ammunition fragments, or small amounts of undepurated lead remaining from a prior, more severe exposure event (2–4). Consistent with this finding, the work by Hunt et al. (5) examined offal piles of hunter-killed deer; they reported that lead-based ammunition fragmented into a range of sizes from very small (~0.5 mm and smaller) to large (>5 mm) and was distributed widely around the wound channel (5). In a subsequent study, Hunt et al. (6) showed that pigs fed venison contaminated with small amounts of lead ammunition fragments had ~3.6-fold higher blood lead levels than pigs fed uncontaminated venison (22.9 vs. 6.3 ng/mL). These studies support our suggestion that the full range of blood lead levels observed in free-flying condors (i.e., ~40 to >5,500 ng/mL) can be explained by condors eating noncontaminated tissue or tissue contaminated with varying amounts of lead dependent on the ammunition fragmentation pattern, the distance from the wound channel that the condor fed, and the amount of contaminated tissue consumed.

We consider it unlikely that exposure to lead in prey animals (carrion) that metabolically incorporated lead into their tissues would contribute significantly to elevated exposures in condors, because lead is biologically depleted and not biomagnified relative to its biologic analog calcium with each trophic transfer (e.g., carrion to scavenger in the case of condors) (7); also, dietary lead absorption rates across the gastrointestinal tracts of adult vertebrates are typically low (3–10%). Finally, although inhalation is also a plausible route of low to moderate lead exposures that has been well-documented in children living in lead-contaminated urban environments (8, 9), free-flying condors would not be expected to be at any significant risk of inhalation exposure to suspended urban particulates enriched in lead or exhaust from small aircraft or other vehicles (e.g., small boat engines) that may still use leaded gasoline.

Cessation of Ammunition-Related Mortalities. In addition to running demographic models which eliminated all lead-related mortalities (see main text), we ran models of reduced lead-caused mortality that specifically account for the fraction of highly lead-poisoned birds whose lead isotope ratios clearly link the source of lead

poisoning to ammunition. Isotope ratios have not been characterized for most poisoning events and the blood samples for which we have lead isotope data are not a random sample of all blood samples with high lead levels. Nonetheless, we can use the data available to make a rough estimate of the fraction of high exposure events that are best linked to ammunition. All four of the blood samples we have with lead >3,000 ng/mL have isotope ratios that are well matched to ammunition ($P > 0.10$ for ammunition, and higher for ammunition than paint or pre-release sources). Of the 30 blood samples with lead >1,000 ng/mL, 23 (77%) are similarly well-matched to ammunition. Finally, of the 48 blood samples with lead >500 ng/mL, 39 (81%) are well-matched to ammunition. We used this last, intermediate value (81%), to estimate the minimum fraction of all lead-based mortality events [27% of all mortalities (10), see also *SI Materials and Methods*] that would be eliminated by a ban on lead-based ammunition. Using this estimate in our demographic model predicts an increase in condor population growth of ~1.4% per year over current growth ($\lambda = 1.0142$ versus 1.0003). Accounting for uncertainty in baseline demographic estimates, this predicted effect results in a decline in the chance of a $\lambda < 1$ from 52.5% under current conditions to 20.2%. As with our other predictions we incorporated this effect into a model that accounts for age differences in lead-based mortality (10) and determined the reduced mortality from a ban on lead ammunition is predicted to yield a λ of 1.024, and a risk of $\lambda < 1$ of only 4.3%.

SI Materials and Methods

Sample Collection. Condor blood and feather samples were collected during standard monitoring events of condors in California. Whole-blood samples (~2 mL) for determination of lead concentrations were collected by field biologists and sent to a certified commercial laboratory (ANTECH Diagnostics, Louisiana Animal Disease Diagnostic Laboratory, and/or California Animal Health and Food Safety Laboratory, University of California at Davis). Whole-blood samples (~3 mL) for δ -amino-levalulinic acid dehydratase (ALAD) activity were collected in low-lead Vacutainers with heparin anticoagulant (Fisher Scientific), placed immediately on dry ice, and stored frozen at -70°C until analysis.

Blood and feather samples collected for lead isotopic composition and associated lead concentrations were collected and processed as previously described (2, 11). Briefly, whole-blood samples (1–3 mL) were collected in low-lead Vacutainers, placed on ice, and stored frozen at -20°C until analysis. Feather vane samples from primary flight feathers were collected while birds were restrained for blood collection, or if collected postmortem, intact whole growing feathers were collected by pulling the feather out of the feather follicle or cutting the feather at the base (i.e., adjacent to the skin). Feathers were stored in polyethylene bags at room temperature until processed for analyses.

Ammunition samples were obtained through an ammunition exchange program in central California, United States, in which sportsmen voluntarily exchanged their lead-based ammunition for copper-based ammunition ($n = 41$), opportunistically from hunters in central California ($n = 4$), by removal from shot carcasses ($n = 3$), or from information previously published in the work by Church et al. (2) ($n = 18$). Lead shot or lead-containing fragments were recovered from lead-poisoned condors ($n = 10$) (Table S2B). Samples of deteriorating lead-based paint ($n = 9$) (Table S2C) were collected from and around an inactive fire

lookout tower and associated structures within Pinnacles National Monument, placed into plastic bags, and stored at room temperature until analysis.

Lead Isotopic Compositions and Associated Concentrations. Whole-blood and feather samples were processed and analyzed using established trace metal clean techniques under high-efficiency particulate arresting-filtered air laboratory conditions using procedures previously described (11–13). A subset (~15%) of bullets from each box of ammunition was processed, and therefore, the frequency of collected brands, calibers, and caliber subtypes of ammunition was reflected in the analyzed samples; each subsample of bullets was processed as a single sample. Ammunition and fragments were cleaned by rinsing sequentially with HPLC grade methanol, 1% trace metal grade HNO₃, and ultrapure water, and then, they were leached in 2 mL 1% HNO₃ for 30 s. Paint was leached as previously described (14) in 0.5 N HCl at room temperature for 24 h to approximate the chemically (and possibly, biologically) available fraction of ingested paint lead. Leachates were diluted in 1% HNO₃ and assessed for lead isotopic composition. Separate aliquots of paint samples were digested in concentrated HNO₃ to produce a near total measure of paint lead levels (Table S2C); paint containing >0.5% lead (5,000 µg/g) is classified as lead-based (15).

Lead concentrations and isotopic ratios were determined using a Finnigan MAT Element or Finnigan XR magnetic sector inductively coupled plasma mass spectrometer measuring masses of ²⁰⁶Pb, ²⁰⁷Pb, and ²⁰⁹Bi or ²⁰⁵Tl (the latter two as internal standards) as previously described (14, 16). The within-run precision [2× relative SD (RSD)] of ²⁰⁷Pb/²⁰⁶Pb ratio measurements was typically ~0.10% based on repeated measurements (≥3) of condor blood samples. Long-term ²⁰⁷Pb/²⁰⁶Pb ratio measurement precision, determined using a previously digested blood sample(s) measured over the course of the sample analysis (2001–2010), was typically <0.20% (2 RSD).

ALAD Activity. To determine inherent (i.e., nonactivated) and activated ALAD activity, a 20-µL aliquot of heparinized whole blood was lysed in 80 µL 1% saponin/0.1% Triton X-100 and mixed with 100–150 µL 0.5 M MES buffer (pH 6.6), and the reaction was started with the addition of 50 µL 60 mM ALA to produce porphobilinogen product (inherent ALAD activity). Activated ALAD activity was determined with an addition of 25 µL 0.8 mM Zn + 25 µL 60 mM DTT (with 30 min incubation) before starting the reaction with ALA. The reaction was stopped with the addition of 200 µL 0.4 M trichloroacetic acid/60 mM mercury. Ehrlich's reagent (*p*-Dimethylaminobenzaldehyde, glacial acetic acid, and perchloric acid) was then added to each sample mixture for measurement on a spectrometer at 555 nm. ALAD activity (in nanomoles porphobilinogen per minute per milliliter blood) was determined using a separately prepared porphobilinogen standard curve.

Blood Lead Data Analysis. Blood samples were collected for routine monitoring as well as suspected lead poisoning events; there was no measurable bias from the purpose of collection on the blood lead results. If no laboratory blood lead level value was reported ($n = 43$; <4% of samples), the field-based value measured using the LeadCare Blood Lead Testing System (ESA) was used. Blood lead data from condors in California used in analyses included samples collected during routine monitoring as well as samples from birds suspected to have been lead poisoned based on observational data from field biologists. To determine if inclusion of blood lead data from suspected lead-poisoned birds introduced a bias in the dataset to higher blood lead levels, we compared the annual median blood lead values using the complete dataset with values generated from a subdataset that excluded data from birds that were collected because of suspected

lead poisoning or if the reason for trap-up was not specified (Fig. S5B). Only data from 2005 to 2010 was compared, because information on why blood samples were collected was not routinely recorded before 2005. For each dataset, the annual median blood lead concentration exceeded or was equal to 100 ng/mL (Fig. S5B), and the annual percent of blood samples with elevated lead levels was comparable between datasets (Table S4). These results indicate that no significant bias was incurred in our analysis of the full dataset that included data from birds targeted because of suspected lead poisoning.

Isotopic Fitting Models. The ²⁰⁷Pb/²⁰⁶Pb ratio in the blood of any free-flying condor will reflect a mixture of the isotopic signatures of the sources of lead exposure, with the contribution of each source to blood determined by the magnitude of exposure, the elapsed time since the exposure occurred, and the toxicokinetics of lead in condor blood. After an elevated lead exposure (e.g., through ingestion of lead fragments in a carcass shot with lead ammunition), the majority of lead in blood will likely reflect that particular source of exposure. Therefore, we used the statistical characteristics of the two most likely sources of lead to condors [background uncontaminated food sources, represented by the ²⁰⁷Pb/²⁰⁶Pb ratios in prerelease condors fed uncontaminated food, and the ²⁰⁷Pb/²⁰⁶Pb ratio in lead-based ammunition and lead-containing fragments, represented by the distribution of ²⁰⁷Pb/²⁰⁶Pb ratios in ammunition and fragments (the latter recovered from lead-poisoned condors)] to determine the probability that the lead in each blood sample collected from free-flying condors came just from background lead or lead-based ammunition/fragments.

We used the ²⁰⁷Pb/²⁰⁶Pb ratios for a total 22, 110, and 76 samples from prerelease condors, free-flying condors, and ammunition, respectively, to determine the likelihood that the lead in a given free-flying condor blood sample came from the same source as in prerelease birds or ammunition. First, we used prerelease samples to estimate the mean and variance of a normal distribution of ²⁰⁷Pb/²⁰⁶Pb ratios of prerelease condors and determined the one-tailed probability of sampling a value as or more extreme as the value found in each free-flying condor blood sample. We performed the same analysis to determine the probabilities of each free-flying sample arising only from lead ammunition. Samples of both ammunition and prerelease blood were relatively small, making any testing for the normality of the distribution of isotope ratios uncertain.

We tested the importance of this assumption by repeating the analysis but fitting the ²⁰⁷Pb/²⁰⁶Pb ratios as generalized λ -distributions (GLDs) with the Freimer, Mudholkar, Kollia, and Lin parameterization (17) rather than to normal distributions. Using the parameters of a GLD fit to each potential lead source, we then estimated the one-tailed probabilities of observing the isotope ratios seen in each blood sample collected from a free-flying bird or a value more extreme. GLDs are extremely flexible distributions that can take on many nonstandard aspects of empirical probability distributions (17–19), and thus, they provide a good test for artifactual results that could arise from our use of normal distributions in the text (Fig. 3B). We ran these results two times: one time using starship and one time using maximum likelihood algorithms to fit GLDs for ammunition and prerelease ²⁰⁷Pb/²⁰⁶Pb ratio distributions (all fitting and probability estimation uses the GLDEX package in R).

Results of these GLD models are shown in Fig. S6. For both fits of the GLD, the essential pattern of results is the same as for the assumption of normally distributed ²⁰⁷Pb/²⁰⁶Pb ratios (Fig. 3B), with 83% and 77% of all blood samples from free-flying condors being better matched to the distribution of ammunition ²⁰⁷Pb/²⁰⁶Pb ratios than the ²⁰⁷Pb/²⁰⁶Pb ratios from prerelease birds using the maximum likelihood and starship fitting methods, respectively.

Demographic Model. To the greatest extent possible, we projected future population trends based on empirical data taken from 1994 to 2010 from 182 free-flying condors released or wild-fledged in California, which together, include a total of 703 condor-years of data. Survival rates were estimated from logistic regression of observed annual survivorship as influenced by year and age up to 8 y, after which time birds were modeled as having a constant average adult survival rate. Although condors may gain in annual survival as they become older adults, very few birds in our sample were of even moderate age for a long-lived species. Because of the young age of released birds, of the 703 y of individual survival data, only 8% of observations were for birds older than 10 y. More strikingly, there are only 3 y of data for birds older than 15 y, and no data at all for birds older than 20 y, making fitting of age-varying survival for older age classes impossible. If a bird was captured and not subsequently released, we counted its last year in the wild as survival, and we did not consider its loss a death. Both age and time dependence are supported by Akaike information criterion (AIC) model selection, with the best predictive model including year, age, and age² (ΔAICc of next best model = 2.20). We used the estimated age-dependent survival estimates for the last year in our dataset (2010) as the estimates of survival rates in our subsequent modeling. Because of changes in management practices (20), survival rates have risen modestly through time, and we, thus, used the last estimates to make the most optimistic assumptions possible based on the observed data. Coefficients for the factors included in the logistic regression to estimate survival are constant = -125.9 , age = 1.12 , age² = -0.0943 , and year = 0.0627 .

Because of the rarity of breeding events in the wild population, we estimated a single average annual rate of production of successful fledglings based on all observations of breeding attempts of female birds. This estimate results in a mean observed field-based reproductive rate of 0.21 fledglings per adult female per year or 0.105 female fledglings per adult female per year. Finally, from a total of 24 wild-fledged birds, we used observed mortality data to estimate a postfledging survival rate of 83.4% over the rest of the first year of life.

We used these estimates of actual survival and reproduction in the current condor population in California to build a simple, deterministic, age-based demographic model for female condors assuming no future releases of captive-reared birds. We assumed that birds could start breeding at age 6 y; field data support this assumption, and at 6 y of age, all birds should have their full breeding plumage (21). We also assumed that all birds reach reproductive senescence at age 65 y and have a lifespan of 75 y, similar to the assumptions in the work by Meretsky et al. (22). We used this model to determine λ , the annual population growth rate. We note that, like all estimates for deterministic population growth, our estimates are biased to a higher population growth rate than will actually occur, because they do not include the effects of environmental or demographic stochasticity (23, 24).

Modeling Cessation of Actions to Prevent Lead Deaths. We estimated the effects of more limited management to prevent lead poisoning

effects by calculating revised age- and year-dependent survival rates. For this calculation, we again used real records of survival and death but modified these records by assuming that the first time that a condor's observed blood lead level exceeded a dangerous threshold, it would die (rather than be clinically treated for lead poisoning, which in fact, happens under current management). We used two thresholds of blood lead levels to approximate lethal lead poisoning: 1,000 and 3,000 ng/mL; we counted a bird as dead in the year that it was first reported to have a blood lead at or greater than these thresholds. With the modified records of survival vs. mortality, we refit annual survival rates, keeping the best supported model structure from the original analyses (age, age², and year). Finally, we rebuilt our demographic model using these revised survival rates along with unmodified reproductive rates to estimate annual population growth.

Modeling the Cessation of Any Lead-Caused Mortalities. To predict changes in condor survival if lead poisoning ceased, we began with our empirically estimated survival rates but then estimated the increase in survival that would result from elimination of lead-caused mortalities. The work by Rideout et al. (10) estimated that 27% of all condor mortalities are lead-related. We thus, decreased each age-dependent mortality rate by this percentage and then used the resulting survival rates to estimate population growth in the absence of lead poisonings. In estimating the proportion of deaths caused by lead, we did not include mortalities between 1992 and 1994, because this time was before the adoption of power line aversion training for all released birds (10). Because the work by Rideout et al. (10) found that juveniles and adults had very different lead-related mortality rates (juveniles = 15% and adults = 71%), we also performed an analysis of population growth in which we altered survival rates with these separate proportional adjustments for the two age classes and found that λ increased by $\sim 1\%$ ($\lambda = 1.0296$ vs. 1.0174).

Uncertainty Analysis. For each of the demographic scenarios described above, we estimated the uncertainty in projected population growth rates. We built 10,000 matrix models for each scenario, with each model constructed using a separate, randomly generated set of parameter estimates that reflects the uncertainty in the distribution of each demographic rate. For survival rates, we used the parameter covariance matrix estimated from our logistic regressions to draw multivariate normal estimates of each regression parameter, and we used these estimates to estimate the age-specific survival rates that were used in the matrix model. For the remaining two demographic rates (production of fledglings and survival over the remainder of the first year of life after fledging), we estimated variance around the mean probabilities using the binomial distribution, and then, we used the mean and variance to draw random values from a β -distribution. We summarize the distributions of λ -values for each scenario using box plots (Fig. S4) and various quantiles (Table S3), and we also calculate the probability under each scenario of not achieving a stable or growing population (annual growth rate < 1) (Fig. S4).

1. Center for Disease Control (2009) *Managing Elevated Blood Lead Levels Among Young Children: Recommendations from the Advisory Committee on Childhood Lead Poisoning Prevention* (US Department of Health and Human Services, Public Health Service, Bethesda).
2. Church ME, et al. (2006) Ammunition is the principal source of lead accumulated by California condors re-introduced to the wild. *Environ Sci Technol* 40:6143–6150.
3. Gwiazda RH, Smith DR (2000) Lead isotopes as a supplementary tool in the routine evaluation of household lead hazards. *Environ Health Perspect* 108:1091–1097.
4. Church ME, et al. (2008) Ammunition is the principal source of lead accumulated by California condors re-introduced to the wild—Reply. *Environ Sci Technol* 42:1809–1811.
5. Hunt WG, et al. (2006) Bullet fragments in deer remains: Implications for lead exposure in avian scavengers. *Wildl Soc Bull* 34:167–170.

6. Hunt WG, et al. (2009) Lead bullet fragments in venison from rifle-killed deer: Potential for human dietary exposure. *PLoS One* 4:e5330.
7. Smith DR, Niemeyer S, Flegal AR (1992) Lead sources to California sea otters: Industrial inputs circumvent natural lead biodepletion mechanisms. *Environ Res* 57:163–174.
8. Hayes EB, et al. (1994) Long-term trends in blood lead levels among children in Chicago: Relationship to air lead levels. *Pediatrics* 93:195–200.
9. Laidlaw MAS, Mielke HW, Filippelli GM, Johnson DL, Gonzales CR (2005) Seasonality and children's blood lead levels: Developing a predictive model using climatic variables and blood lead data from Indianapolis, Indiana, Syracuse, New York, and New Orleans, Louisiana (USA). *Environ Health Perspect* 113:793–800.
10. Rideout BA, et al. (2012) Patterns of mortality in free-ranging California Condors (*Gymnogyps californianus*). *J Wildl Dis* 48:95–112.

11. Finkelstein ME, et al. (2010) Feather lead concentrations and (207)Pb/(206)Pb ratios reveal lead exposure history of California Condors (*Gymnogyps californianus*). *Environ Sci Technol* 44:2639–2647.
12. Smith DR, Osterloh JD, Flegal AR (1996) Use of endogenous, stable lead isotopes to determine release of lead from the skeleton. *Environ Health Perspect* 104:60–66.
13. Gwiazda R, Campbell C, Smith D (2005) A noninvasive isotopic approach to estimate the bone lead contribution to blood in children: Implications for assessing the efficacy of lead abatement. *Environ Health Perspect* 113:104–110.
14. Finkelstein ME, Gwiazda RH, Smith DR (2003) Lead poisoning of seabirds: Environmental risks from leaded paint at a decommissioned military base. *Environ Sci Technol* 37:3256–3260.
15. EPA (1998) *Proposed Rule on Identification of Lead-Based Paint Hazards* (Environmental Protection Agency, Government Printing Office, Washington DC), Federal Register 30302, June 3, 1998.
16. Gwiazda R, Woolard D, Smith D (1998) Improved lead isotope ratio measurements in environmental and biological samples with a double focusing magnetic sector inductively coupled plasma mass spectrometer (ICP-MS). *J Anal At Spectrom* 13: 1233–1238.
17. Freimer M, Mudholkar G, Kollia G, Lin C (1988) A study of the generalized Tukey Lambda family. *Comm Statist Theory Methods* 17:3547–3567.
18. Ramberg JS, Tadikamalla PR, Dudewicz EJ, Mykytka EF (1979) A probability distribution and its uses in fitting data. *Technometrics* 21:201–214.
19. Lakhany A, Mausser H (2000) Estimating the parameters of the generalized lambda distribution. *Algo Res Q* 3:47–58.
20. Mee A, Snyder N (2007) California condors in the 21st century—conservation problems and solutions. *California Condors in the 21st Century*, Series in Ornithology, eds Mee A, Hall LS (American Ornithologists Union, Nuttall Ornithological Club, Washington, DC), Vol 2, pp 243–279.
21. Snyder NFR, Schmitt JN (2002) California condor (*Gymnogyps californianus*). *The Birds of North America Online*, ed Poole A (Cornell Laboratory of Ornithology, Ithaca, NY).
22. Meretsky VJ, Snyder NFR, Beissinger SR, Clendenen DA, Wiley JW (2000) Demography of the California condor: Implications for reestablishment. *Conserv Biol* 14:957–967.
23. Caswell H (2001) *Matrix Population Models: Construction, Analysis, and Interpretation* (Sinauer, Sunderland, MA).
24. Morris WH, Doak DF (2002) *Quantitative Conservation Biology: Theory and Practice of Population Viability Analysis* (Sinauer, Sunderland, MA).

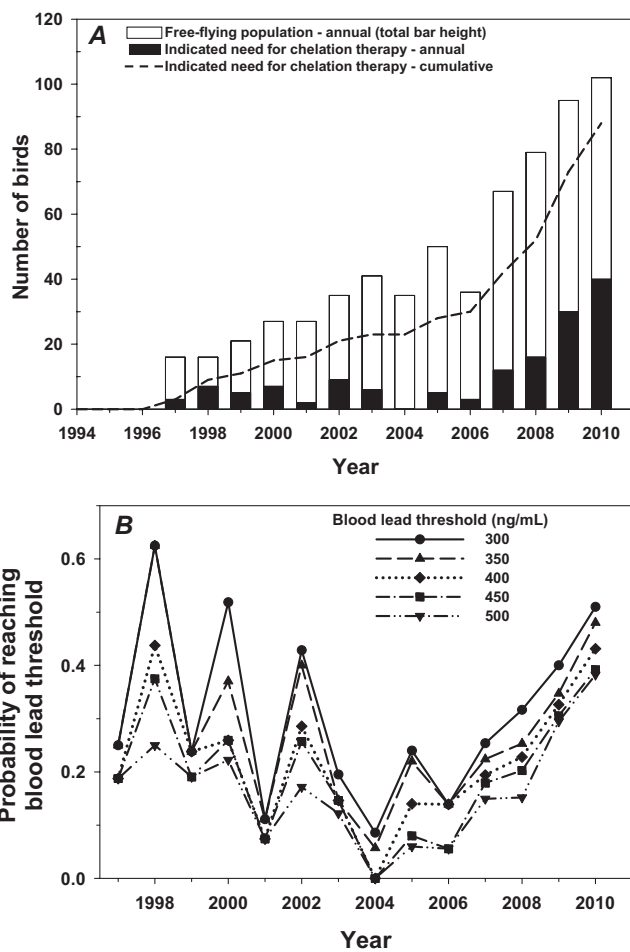


Fig. S1. Free-flying California condors are undergoing intensive management to mitigate the effects of lead poisoning. (A) Over the course of the release program in California, 88 condors have had a blood lead level ≥ 450 ng/mL, which indicated the need for clinical intervention and lead chelation therapy. On an annual basis, between 0 (2004) and 40 (2010) birds per year had a blood lead level that indicated the need for chelation therapy. Compared with the number of free-flying condors that year, an average of ~20% of birds per year had a blood lead value that indicated the need for chelation therapy. (B) Because for the years 1997–2010, there was no standardized blood lead threshold to indicate clinical chelation treatment for condors in California, we considered additional blood lead thresholds to more broadly reflect health risks to condors associated with lead poisoning as well as the clinical resources associated with managing lead poisoned condors. For all years combined, the probability that a condor would present with a blood lead level of 300, 350, 400, 450, or 500 ng/mL was 0.33, 0.29, 0.24, 0.22, and 0.19, respectively. On a yearly basis, the average probability that a condor would present with a blood lead of 300, 350, 400, 450, and 500 ng/mL was 0.31 (range = 0.09–0.63 across years), 0.28 (range = 0.06–0.63 across years), 0.22 (range = 0–0.44 across years), 0.19 (range = 0–0.39 across years), and 0.17 (range = 0–0.38 across years), respectively. Note that the variation in exposure probabilities in the first half of the reintroduction program is most likely attributed to chance and confounded by low sample size, although it is not necessarily unusual compared with the distribution of all blood lead data (Fig. 2A). It is noteworthy that, from 2006 to 2010, the probability of a condor presenting with one of these five blood lead threshold levels seems to be increasing over time (a finding also indicated in Fig. 2A).

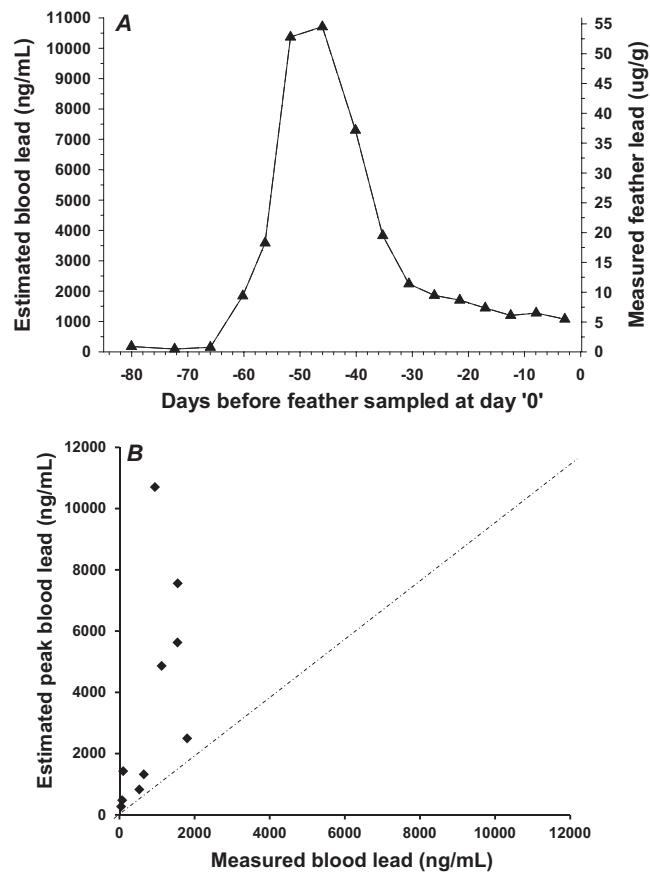


Fig. S2. Sequential feather analysis can be used to examine the magnitude and duration of lead exposure events that a condor experiences over the timeframe of feather growth (~3 mo) (1). (A) Measured feather lead and corresponding estimated blood lead levels vs. estimated days of feather growth for a primary feather collected postmortem from condor ID 336 that died of lead poisoning in 2008. Blood lead concentrations were estimated from measured feather lead concentrations using a blood (nanograms per milliliter) to feather (micrograms per gram) lead concentration relationship of ~200:1 (2). Modified from Finkelstein et al. (2). (B) Using 10 feathers collected from eight condors, we found a significant relationship between a condor's peak exposure blood lead level, estimated from the peak feather lead level, and the measured blood lead level ($R = 0.827$, $n = 10$, Pearson's correlation on log-transformed data). Furthermore, measured blood lead values consistently underestimated the blood lead level at the peak of exposure (estimated from the feather lead levels) by a range of 1.4- to 14.4-fold (geometric mean = 4.3, SE = 1.3) (Table S1). The dashed line indicates the idealized 1:1 relationship between measured and estimated (from feather analyses) blood lead levels.

- Snyder NFR, Johnson EV, Clendenen DA (1987) Primary molt of California condors. *Condor* 89:468–485.
- Finkelstein ME, et al. (2010) Feather lead concentrations and (207)Pb/(206)Pb ratios reveal lead exposure history of California Condors (*Gymnogyps californianus*). *Environ Sci Technol* 44:2639–2647.

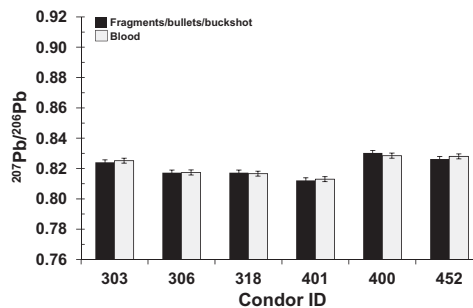


Fig. S3. Lead isotopic ratio match between recovered lead-containing fragments/ammunition and blood of lead-poisoned condors in California indicates that fragments/ammunition are the source of lead poisoning to these condors. Six case studies comparing the $^{207}\text{Pb}/^{206}\text{Pb}$ ratio of fragments/buckshot/bullets recovered from condors (fragments: condor ID 303, 400, and 452; buckshot: condor ID 401) or carcasses on which condors fed (bullets: condor ID 306 and 318) with the lead isotopic ratio of the condor's blood at the time of lead poisoning (Table S2B). Error bars represent long-term analytical precision of the $^{207}\text{Pb}/^{206}\text{Pb}$ ratio measurements (0.2%, 2 RSD) (SI Materials and Methods).

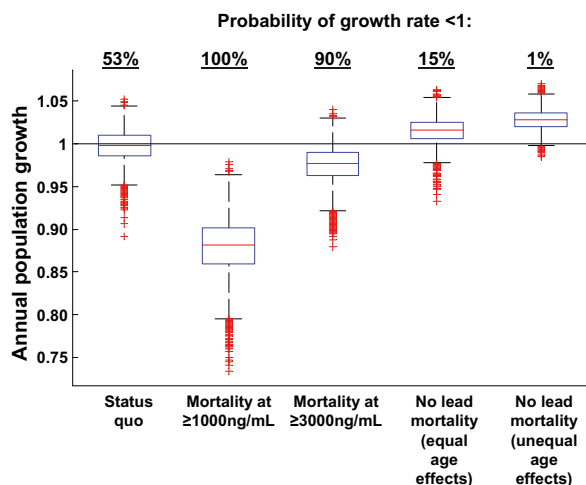


Fig. S4. Box plots showing distributions of projected annual condor population growth rates accounting for parameter uncertainty for different scenarios of lead management and mortality effects. Probabilities that each management/mortality scenario would lead to declining population numbers are shown above each box at top of figure. Scenarios are status quo (current interventions to mitigate lead poisoning continued), mortality at $\geq 1,000$ ng/mL (cessation of interventions to mitigate lead poisoning with mortality occurring at blood lead $\geq 1,000$ ng/mL), mortality at $\geq 3,000$ ng/mL (cessation of interventions with mortality occurring at blood lead $\geq 3,000$ ng/mL), no lead mortality (equal age effects; no lead-related mortalities assuming equal mortality rates between juveniles and adults), and no lead mortality (unequal age effects; no lead-related mortalities assuming mortality rates unequal between juveniles and adults). The horizontal line indicates a growth rate of one meaning stable population numbers. Lead mortality rates are based on the work by Rideout et al. (1). Assumptions behind each scenario are described in the text and *SI Materials and Methods*.

1. Rideout BA, et al. (2012) Patterns of mortality in free-ranging California condors (*Gymnogyps californianus*). *J Wildl Dis* 48:95–112.

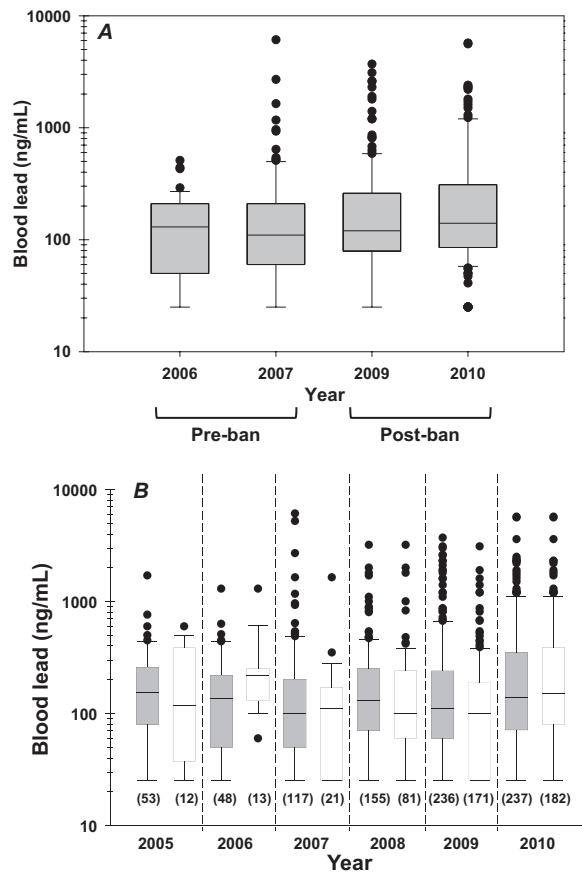


Fig. S5. Blood lead levels in condors in California do not seem to have been reduced by recent regulatory actions. (A) On July 1, 2008, regulatory action to reduce the use of lead-based ammunition in condor habitat in California went into effect (1–3). Blood lead values from the free-flying condor population in 2009 and 2010 (Fig. 2A) provide no indication that condor blood lead levels decreased as a result of these regulations. We compared blood lead levels between the same set of birds ($n = 60$) sampled preban (2006, $n = 42$ blood lead measurements; 2007, $n = 101$ blood lead measurements) and then sampled again postban (2009, $n = 156$ blood lead measurements; 2010, $n = 131$ blood lead measurements). To quantitatively investigate if condor blood lead levels decreased postban, using log-transformed blood lead values, we generated two different mixed linear models that had random year and bird ID effects but differed in the inclusion of a fixed pre- vs. postban time period effect. AICc comparisons of the two models provided no support for a pre- to postban time period effect (AICc for the model without time period is 1.9 lower than for model with time period). Note, however, that the slight trend is for higher blood lead levels after the ban than before it; however, more detailed analysis of longer-term trends is needed to fully evaluate the ban's efficacy. Box indicates median, upper and lower are 75th and 25th percentiles, whiskers represent 10th and 90th percentiles, and y axis is log scale. (B) Comparison of blood lead levels of condors in California from 2005 to 2010 by year using two datasets: the complete dataset, which includes samples obtained from birds targeted because of suspected lead poisoning (filled boxes), and a subdataset, which excludes data from birds targeted because of suspected lead poisoning (nontargeted; open boxes) or when the reason for trap-up was not recorded. Box indicates median, upper and lower are 75th and 25th percentiles, whiskers represent 10th and 90th percentiles, (number of samples), and y axis is log scale.

1. Ridley-Tree Condor Preservation Act (2008) *Bill No. 821* (California State Assembly, Sacramento, CA). Available at <http://www.leginfo.ca.gov/bilinfo.html>.
2. California Department of Fish and Game (2008) *Methods Authorized for Taking Big Game*, Section 353, Title 14, CCR (Office of Administrative Law, Sacramento, CA). Available at <http://www.oal.ca.gov/Publications.htm>.
3. California Department of Fish and Game (2008) *Methods of Take for Nongame Birds and Mammals*, Section 475, Title 14, CCR (Office of Administrative Law, Sacramento, CA). Available at <http://www.oal.ca.gov/Publications.htm>.

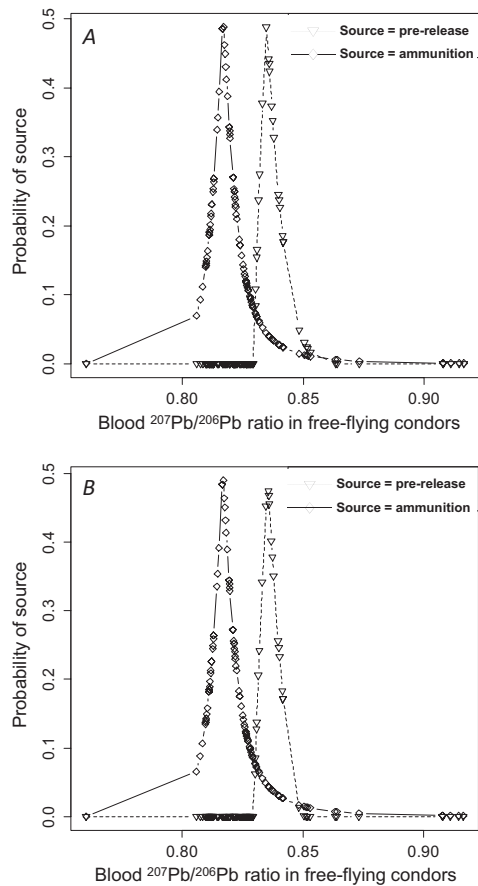


Fig. S6. Probabilities of observing $^{207}\text{Pb}/^{206}\text{Pb}$ isotope ratios in free-flying condors in California if they arose solely from either background lead exposure, represented by the distribution of $^{207}\text{Pb}/^{206}\text{Pb}$ ratios in prerelease condors, or from lead-based ammunition, represented by the distribution of $^{207}\text{Pb}/^{206}\text{Pb}$ ratios in ammunition and lead fragments (the latter recovered from lead-poisoned condors). (A) Results from maximum likelihood fitting of generalized λ -distributions. (B) Results from starship fitting of generalized λ -distributions.

Other Supporting Information Files

[Table S1 \(DOCX\)](#)

[Table S2 \(DOCX\)](#)

[Table S3 \(DOCX\)](#)

[Table S4 \(DOCX\)](#)