Wildlife Society Bulletin; DOI: 10.1002/wsb.762

Original Article

Guidelines for Evaluation and Treatment of Lead Poisoning of Wild Raptors

JESSE A. FALLON,[1,2] *Department of Fish and Wildlife Conservation, Virginia Tech, Blacksburg, VA 24061, USA*
PATRICK REDIG, *Department of Clinical Veterinary Sciences, University of Minnesota, The Raptor Center, 1920 Fitch Avenue, St Paul, MN 55108, USA*
TRICIA A. MILLER, *Division of Forestry and Natural Resources, West Virginia University, Morgantown, WV 26506, USA*
MICHAEL LANZONE, *Cellular Tracking Technologies, 2405 North Center Avenue, Suite B, Somerset, PA 15501, USA*
TODD KATZNER, *U. S. Geological Survey, Forest and Rangeland Ecosystem Science Center, 970 Lusk Street, Boise, ID 83706, USA*

ABSTRACT Lead poisoning is a threat to birds, particularly scavenging birds of prey. With the availability of portable lead-testing kits, an increasing number of field researchers are testing wild-caught birds, *in situ,* for lead poisoning. We describe guidelines for evaluation of lead toxicity in wild raptors by outlining field testing of blood-lead concentrations, presenting criteria for removing a lead-poisoned bird from the wild for treatment, and suggesting strategies for effective treatment of lead intoxicated raptors. Field testing of birds is most commonly accomplished via portable electrochemical analysis of blood; visual observation of condition alone may provide insufficient evidence upon which to make a decision about lead poisoning. Our intended audience is not only the avian research community, but also rehabilitation facilities that may receive apparently uninjured birds. Best practices suggest that birds whose blood-lead levels are <40 μg/dL be released back to the wild as soon as possible after capture. The decision to release or treat birds with blood-lead levels between 40 μg/dL and 60 μg/dL should be made based on the presence of clinical signs of poisoning and relevant biological characteristics (e.g., breeding status). Finally, birds with blood-lead levels >60 μg/dL are potentially lethally poisoned and best served if removed from the wild for appropriate treatment at a licensed rehabilitation facility and later released. We present guidelines for decision-making when treating lead poisoning of wild raptors. Future work based on experimental studies will clarify the role of lead poisoning for specific species and be important to refine these guidelines to improve effectiveness.

KEY WORDS chelation, ecotoxicology, lead toxicity, raptor, scavenger, wildlife rehabilitation.

Lead poisoning is a significant threat faced by birds. Scavenging birds are exposed to lead primarily through ingestion of spent lead ammunition, especially fragments from rifle bullets used for big game hunting or rodent shooting (Scheuhammer and Templeton 1998, Garcia-Fernandez et al. 2005, Church et al. 2006, Pain et al. 2007, Finkelstein et al. 2010). In places where lead shotgun ammunition is used for waterfowl and upland game, wounded birds carrying lead pellets have increased mortality risk from raptors and other predators. For some species, predation on wounded game birds is a common route of exposure (Helander et al. 2009). Nearly all birds of prey scavenge, including eagles, owls, hawks, falcons, and vultures; therefore, there is potential for these and other species to be poisoned by lead (T. Katzner, personal observation; Jackowski et al. 2015). Likewise, upland game birds and waterfowl ingest spent shotgun shot as grit (e.g., Keel et al. 2002, Walter and Reese 2003, Newth et al. 2013, Haig et al. 2014). Many species of waterbirds can be exposed to lead via intentionally or unintentionally discarded fishing tackle (e.g., Locke et al. 1982). Less commonly, birds may also be exposed to lead via other sources including paint, coal-fired power plant emissions, fuel, lead mining, improper disposal of lead-acid batteries, and smelting (Pacyna 1987, Thomas and Spiro 1994, Johnson et al. 2007, Pain et al. 2009, Walker et al. 2012). Records from rehabilitation organizations suggest that ≥ 75 species of birds have been admitted for lead toxicity (Pain et al. 2009).

Visual observation of external condition is often insufficient to make a diagnosis of lead poisoning. However, with the development of field-ready lead-testing kits (e.g., LeadCare®; ESA Biosciences, Chelmsford, CT, USA), there is potential for immediate evaluation of lead poisoning of wild birds captured by field biologists. If, in the process of capture for research, a bird was discovered with a broken wing or leg,

Received: 8 January 2016; Accepted: 8 December 2016

[1]*E-mail: jfallon@vt.edu*
[2]*Present address: 60 Fairchance Road, Morgantown, WV 26508, USA*

most researchers would not immediately release it back to the wild. Likewise, it seems doubtful that scientists would knowingly release to the wild a lead-poisoned bird with obvious evidence of neurological or other organ impairment. However, the extent of lead poisoning as a specific clinical diagnosis may not be readily apparent through casual observation, and it can be challenging to make educated decisions about when it is appropriate remove a bird from the wild for treatment for lead poisoning.

We describe guidelines for evaluation of lead toxicity in wild raptors, outline field testing of blood-lead levels, and present criteria for removing a lead-poisoned bird from the wild for chelation therapy. Our intended audience is not only the avian research community, but also rehabilitation facilities that may receive apparently uninjured birds from researchers. We provide detail on 4 main points: 1) the basics of lead ingestion and toxicity; 2) details on how to test wild birds for lead poisoning; 3) suggested best practices for when to remove a bird from the wild for chelation and where to bring such a bird; and 4) suggested treatment options for rehabilitators.

Lead Ingestion by Raptors

Lead rifle ammunition is specifically designed to fragment when it hits a target. This process facilitates transfer of kinetic energy from the projectile to the animal being shot and increases the likelihood that the impact will be fatal. As a consequence of these processes, lead fragments, some microscopic, can be found dispersed in animal tissue far from the primary path of the bullet. Lead fragments have been reported as far as 15 cm (6″; Hunt et al. 2009) and 46 cm (18″; Grund et al. 2010) from the center of the bullet's exit hole.

Wild raptors are exposed to lead when they scavenge shot rodents or ungulates not recovered by the shooter or gut piles left by hunters (Haig et al. 2014). The ubiquity and number of ungulates harvested is widely recognized and available indirectly through hunting records (e.g., WY, USA, hunt records are available at https://wgfd.wyo.gov/Hunting/Harvest-Reports/). However, the number of rodents shot is less well-understood and estimates are only available from some states. For example, in South Dakota, USA, it is estimated that every year 1.05 million prairie dogs (*Cynomys ludovicianus*) are shot (Huxoll 2012). Stauber et al. (2010) suggest firearm-killed coyotes (*Canis latrans*) as another source of lead. Nongame carcasses, especially rodents, are rarely retrieved by the shooter; therefore, many remain available for consumption by avian scavengers. Although nonlead ammunition is available to consumers, it is more expensive, and the majority of ammunition used for shooting in the United States is lead-based.

Raptors may ingest lead through other sources. Wounded game birds carrying lead pellets or weakened by lead poisoning serve as prey for raptors. Ingestion of fishing tackle is a potential mechanism of lead poisoning for raptors near water (e.g., osprey [*Pandion haliaetus*] and fish eagles [*Haliaeetus* spp], Haig et al. 2014). In rare cases, there are other mechanisms of exposure (e.g., eating lead paint; Finkelstein et al. 2012), although isotope analysis suggests that these other mechanisms are infrequent.

Pathophysiology and Diagnosis of Lead Poisoning

Once ingested, lead can remain in the body for days, weeks, or longer (Bates et al. 1968). In the acidic environment of the stomach, lead is worn down by the muscular action of the ventriculus, solubilized, and actively absorbed through a calcium transport mechanism (Fullmer 1991). The rate and degree of absorption depends on several factors including physical form of the lead, particle size, species-specific gastrointestinal physiology and transit time, and individual-specific nutritional status and age (Redig and Arent 2008, Pokras and Kneeland 2009). Diurnal raptors maintain a relatively low stomach acid compared with other species of birds, which likely increases the rate of absorption and may increase risk of lead intoxication (Duke 1986). Once absorbed, lead is distributed to many tissues throughout the body. The half-life of lead *in vivo* is highly variable, with detectible concentrations present for weeks in blood (Redig and Arent 2008, Pokras and Kneeland 2009), months in soft tissue organs (Scheuhammer 1987, Pokras and Kneeland 2009), and years in bone (Rabinowitz 1991, Pokras and Kneeland 2009).

Lead poisoning can cause lethargy, gastrointestinal stasis, anorexia, vomiting, anemia, disturbance of cellular function, and neurologic injury potentially leading to blindness, seizures, weakness, and death (Redig et al. 1980, Franson et al. 1983, Custer et al. 1984, Pain et al. 1993). Even at low doses, lead exposure causes a number of sublethal effects such as neurological impairment, organ dysfunction, immune system disturbance, and reproductive impairment (Burger and Gochfeld 2000). Despite the myriad effects of lead poisoning, it is important to note that not all of these consequences will necessarily be expressed in any one animal. Neurological impact, including head tilt, loss of balance, pelvic limb weakness, and loss of conscious proprioception (positional awareness) are the most widely reported visible effects (Benson et al. 1974, Redig et al. 1980, Lumeij 1985, Fisher et al. 2006). However, this same suite of symptoms can be triggered by other conditions such as head trauma, encephalitis (e.g., West Nile virus), botulism, nutritional deficiency, and other toxicities (e.g., zinc poisoning; Dierenfeld 1989, Mautino 1997, Jones 2006, Redig and Arent 2008, Gamino and Höfle 2013).

Because there is strong selection pressure on birds not to show external signs of illness and these clinical (i.e., recognizable symptoms on examination) signs associated with lead poisoning are nonspecific, diagnosis in the live bird relies on measurement of lead in blood. Background concentrations of lead in the blood of birds have been reported to be $<20\,\mu g/dL$ (<0.2 parts per million [ppm]; Pain et al. 2009, Stauber et al. 2010) although recently some authors have suggested that blood lead concentrations should be considered background only when $<10\,\mu g/dL$ (<0.1 ppm; Church et al. 2006, Cade 2007). There is general consensus that for most raptor species, blood lead concentrations $\geq 20\,\mu g/dL$ (>0.2 ppm) clearly represent

elevated blood lead (Kramer and Redig 1997, Pain et al. 2009, Stauber et al. 2010, Harmata 2011). However, there is little experimental evidence to support these thresholds. In the case of human children, lead levels >5 μg/dL (>0.05 ppm) are considered to indicate exposure and may require case management (Centers for Disease Control 2014). Bald eagles (*Haliaeetus leucocephalus*) grounded and admitted for rehabilitation with blood lead ≥60 μg/dL (>0.6 ppm) express clinical signs of poisoning, whereas concentrations >120 μg/dL (>1.2 ppm) regularly result in mortality (Kramer and Redig 1997). The clinical effect of blood lead concentrations varies among species. For example, swans (*Cygnus* spp.) have been reported to show clinical signs at ≥40 μg/dL and be lethally poisoned at values >200 μg/dL (2 ppm; Sears et al. 1989, Degernes et al. 2002). Further, turkey vultures (*Cathartes aura*) experimentally dosed with lead shot survived longer with elevated blood-lead concentrations than did other species of birds (Carpenter et al. 2003). Additional experimental work is needed to more fully understand how individual species respond to lead exposure. However, raptor blood-lead concentrations >20 μg/dL but <60 μg/dL typically are considered to be elevated but subclinical (i.e., with few obvious recognizable manifestations of poisoning and only sometimes requiring treatment; Kramer and Redig 1997). Such birds are typically admitted to rehabilitation centers with traumatic injury, with elevated blood lead detected as part of the routine clinical diagnostic testing. Whether the lead burden contributed to the occurrence of the traumatic event is not known.

Severity of clinical signs depends in part on the intensity and duration of exposure to lead. A single blood-lead concentration measurement between 20 μg/dL and 60 μg/dL in and of itself cannot accurately be interpreted as requiring or not requiring treatment. This is because concentrations of circulating lead can change rapidly, as the toxin moves to and from other tissues (e.g., liver, bone) into and out of the bloodstream or from ongoing exposure from lead fragments in the gastrointestinal tract. It is therefore important for field biologists and rehabilitators to understand that 1) blood lead concentrations are dynamic; 2) a single blood test does not characterize lead exposure or degree of poisoning; and 3) the decision to treat a wild bird should be based on a thorough evaluation of all signs of exposure (see below).

Testing Wild Birds for Lead Poisoning

Reference laboratory methods for measuring lead include graphite-furnace atomic absorption spectrophotometry and inductively coupled plasma mass spectrometry (ICP-MS; Murthy et al. 1973, Ting and Janghorbani 1988). However, these methods require expensive equipment and time, and are not practical for quick assessment in the field or at a treatment facility. Inexpensive "point of care" lead analyzers developed to assess human exposure to lead have been used in a variety of wildlife settings to obtain a rapid test result. Although such lead analyzers may be less accurate than ICP-MS, they are fast, only require a small volume of blood, and come with a degree of error generally considered acceptable for clinical decision-making. The most frequently used of these analyzers rely on electrochemical anodic stripping voltammetry (LeadCare®). Within their operating ranges, these devices are correlated with ICP-MS values for samples from humans (e.g., Counter et al. 1998, McMillin and Bornhorst 2008, Sobin et al. 2011) and birds (Domenech and Langner 2009, Rodriguez-Ramos et al. 2009, Langner et al. 2015; Redig and Risebrough, unpublished data). These systems require only 50 μL of blood, providing the opportunity to safely quantify blood lead concentration in all species of raptors, regardless of body size. Disadvantages of these clinical testing units include greater among-sample variability in results than those from ICP-MS (averaging 1.0 μg/dL in human blood with lead concentrations <10 μg/dL, Sobin et al. 2011) and processing constraints to only one sample at a time. Furthermore, some of the most commonly used devices have a maximum quantifiable concentration of lead of 65 μg/dL; greater concentrations are reported as "high." Prior to sampling birds in the field, it is important to calibrate any portable lead analyzer with laboratory measurements to define the accuracy and precision of the field test. We recommend that field test kits be calibrated with laboratory based methods whenever a new lot of test strips is opened or at the beginning of a new research project or field season.

Portable radiographic units are occasionally used in the field to check for gravidity (presence of eggs) and metal foreign bodies. Most metals appear with the same density on radiographs, and thus it is impossible to use a radiograph to distinguish between lead and other metals. Elevated blood-lead concentration combined with radiographic evidence of metal in the gastrointestinal tract is a clear indication for removing a bird from the field for decontamination. It is also critical to recognize that many individuals (especially raptors and corvids) with lead poisoning do not have metal objects present in the gastrointestinal tract at the time of diagnosis. Lead fragments can pass in the feces or be egested via pellet egestion (Pattee et al. 1981). Birds can remain poisoned long after the metal passes (Kramer and Redig 1997). Therefore, although radiographic evidence of metal in the gastrointestinal tract can occur in wild birds, blood results are necessary to further assess the animal for possible exposure to lead.

Best Practices for When to Remove a Bird From the Wild

Because of the increased accessibility and use of portable lead analyzers in the field and prevalence of lead exposure in wildlife, field scientists may encounter birds that they know, based on the field test results, have elevated blood-lead concentrations. When this situation arises, researchers have only a limited time to evaluate and determine whether the animal should be immediately returned to the wild or brought into captivity for decontamination and chelation. Furthermore, within the United States, members of the general public, including federally permitted bird banders, are allowed (under 50 C.F.R. 21) to immediately transfer an exhausted, ill, or injured bird to a federally permitted migratory-bird rehabilitator (B. Peterjohn, U.S. Geological

Survey, personal communication). Therefore, it is important that a clearly defined and succinct set of criteria be established to address this medical situation and take action appropriate to the medical condition of the bird. There is overwhelming evidence that untreated lead toxicity in raptors can result in morbidity and mortality (e.g., Pattee et al. 1981, Kramer and Redig 1997, Deem et al. 1998, Fisher et al. 2006, Finkelstein et al. 2012).

When interpreting blood lead concentration in the field, we recommend separating blood lead concentration into 3 categories: ≤40 μg/dL (low), 40–60 μg/dL (elevated), and ≥60 μg/dL (toxic). We identify these categories based primarily on published clinical observations of eagles admitted to rehabilitation centers that were analyzed for blood lead concentration regardless of admission cause (Kramer and Redig 1997). These categorical values may be extrapolated with caution to other raptors, although species-dependent variability should be considered. If a bird's blood-lead level is ≥40 μg/dL, we recommend repeating the measurement immediately on a freshly collected blood sample to confirm the result, prior to evaluating the bird closely for signs of toxicity that may require treatment (i.e., clinical signs).

There are a multitude of organ system effects of lead toxicity; therefore, clinical signs can be nonspecific and may include dull mentation (e.g., slow or weak response to stimulation or handling), weakness, lack of coordination, poor body condition, or green biliverdin staining in the mouth (Table 1 and Fig. 1). Head tilting, rhythmic head twitching, nystagmus (rhythmic eye movement), and head drooping are commonly reported clinical signs in lead poisoned birds encountered in the field. It is important to note that a bird with modestly elevated blood-lead concentration may exhibit obvious clinical signs and a bird with high blood-lead levels may exhibit no readily apparent clinical signs at all (Harmata 2011). Therefore, both blood lead concentration and clinical signs should be considered when assessing toxicity, because even an apparently asymptomatic bird with high blood-lead concentration is likely suffering from lead toxicity.

Table 1. Clinical signs that can occur in birds with lead poisoning.

Neurological	Gastrointestinal	Hematological
Weakness	Vomiting or regurgitation	Nonregenerative anemia
Ataxia (loss of coordination)	Diarrhoea	
Blindness	Green excreta	
Seizures	Green staining on feathers	
Nystagmus (involuntary eye movement)		
Head tilt		
Clenched toes		
Drooping wings		
Closed eyelids		
Tremors		
Instability or unable to stand		
Abnormal mentation		

Figure 1. Biliverdin staining on feathers near the beak of a bald eagle (*Haliaeetus leucocephalus*).

We describe a proposed set of best practices for evaluating potentially lead-poisoned raptors that can be used to aid decision-making (Fig. 2):

1. When blood lead concentrations are ≥60 μg/dL then, regardless of other symptoms, it is appropriate to transfer the bird as rapidly as possible to a licensed rehabilitator experienced with treating lead toxicity and able to further evaluate the clinical condition of the bird.
2. If the blood lead concentration is >40 μg/dL and <60 μg/dL and clinical signs are not present, then the most reasonable approach is to return the bird to the wild as soon as possible. If logistically feasible, properly trained field researchers should consider providing fluid administration (subcutaneous and oral) to support diuresis. Although the efficacy of a one-time fluid bolus in treating elevated blood-lead concentration is not clear, fluid diuresis is an important component of treating heavy metal intoxication and may be beneficial to a bird with subclinical lead poisoning (DeFrancisco et al. 2003).
3. If the blood lead concentration is >40 μg/dL and <60 μg/dL and clinical signs are present, then the bird should be transferred as rapidly as possible to a licensed rehabilitator for treatment of lead intoxication. It is possible that, in case-specific situations, removal from the wild for treatment would not be warranted, but we recommend that researchers err on the side of caution when handling birds with clinical signs of intoxication.
4. If lead concentration <40 μg/dL and the bird otherwise appears healthy, then it should be returned to the wild as quickly as is possible.

Suggested Treatment Options for Rehabilitation Facilities

Treatment for lead poisoning has been described previously (e.g., Redig and Arent 2008). Here, we briefly review the steps that are appropriate and provide citations for those readers interested in more details. Lead poisoning may occur with or without the presence of lead visible in radiographs

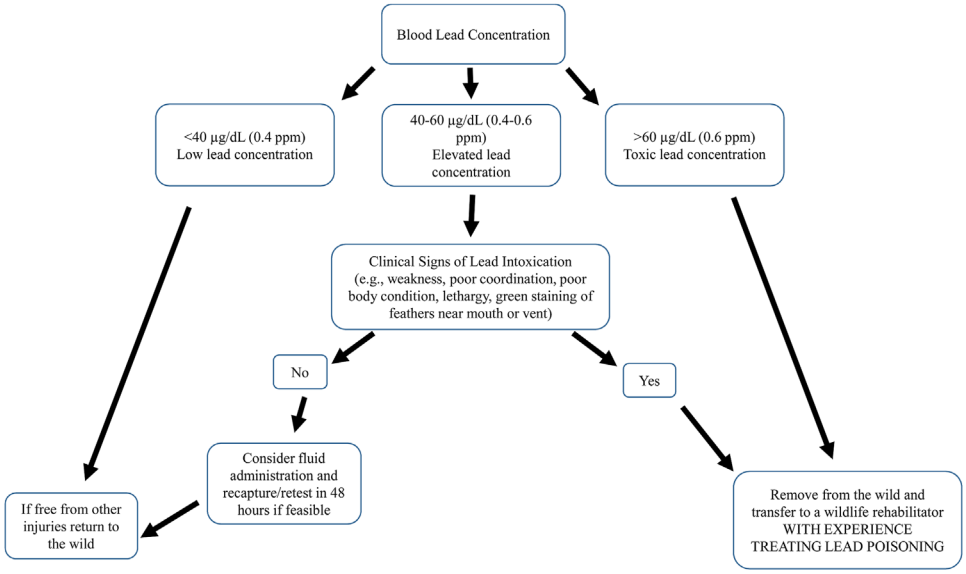

Figure 2. Flowchart showing a suggested decision-making process for wild raptors with possible lead toxicosis.

(Redig and Arent 2008). If radiographs indicate metal objects within the gastrointestinal tract, initial therapy should consist of removing ongoing exposure with, in order of most to least frequently applied, gastric lavage, emollient laxatives, carthartics, endoscopy, or proventriculotomy. Without removal of lead fragments, exposure continue, and chelation treatment is less effective. However, if circumstances dictate a delay in the implementation of any of these removal procedures, chelation therapy should be instituted immediately to reduce circulating blood levels. Prior to initiating therapy, we recommend rehabilitators repeat a measurement of blood lead to establish a pretreatment concentration.

Following decontamination or in the absence of ongoing exposure, chelation therapy is the mainstay of treatment for avian lead poisoning (DeFrancisco et al. 2003). Chelation agents that can effectively bind lead include D-penicillamine, succimer (Dimercaptosuccinic acid), British Anti-lewisite (BAL), and $CaNa_2$ ethylene diamine tetra acetic acid (CaEDTA). Of these, succimer and CaEDTA are the treatments of choice (Redig and Arent 2008). Prior to treating birds with chelation therapy, it is important for the veterinarian and rehabilitator to understand the possible risks that may be associated with different chelation agents and dosing protocols.

Calcium EDTA is most commonly used to treat heavy metal toxicity; it can be administered intravenously, intramuscularly, or subcutaneously, and is effective at binding lead in soft tissues. Dosing protocols vary, but CaEDTA is typically given at 35–100 mg/kg every 12 hr for 5 days. Although it has not been reported for raptors treated with this dosing protocol, calcium EDTA has the potential to be nephrotoxic (Schwartz et al. 1966). Falcons with lead intoxication treated daily at doses of 100 mg/kg for >3 weeks showed no adverse effects and had significantly reduced blood-lead concentrations (Samour and Naldo 2002). Still, because of the potential for kidney insult, it is important to maintain appropriate hydration of patients undergoing treatment, and patients on extended courses of CaEDTA should be monitored for kidney function. Five-day courses of CaEDTA can be repeated for individual patients as necessary to return blood lead levels to <0.2 ppm. Rest days are helpful between rounds for the patient to replenish divalent cations that are also chelated by the agent. Blood-lead concentration measurements should be made at the end of these rest periods because the presence of the chelation agent may interfere with results. The ultimate determination to cease treatment should be based on resolution of clinical signs and repeated measurements of blood lead concentration.

Succimer is orally administered, making it a poor choice for chelation of a vomiting bird. However, it is very effective at decreasing lead in soft tissues and believed to not be nephrotoxic. Successful treatment has used dosages of succimer in a range from 10 to 40 mg/kg, but there is limited information on an optimal duration of treatment (Hawkins et al. 2013). Succimer can be given concurrently with CaEDTA. Again, treatment duration should be based on resolution of clinical signs and repeated measurements of blood lead concentration. Birds have been treated with succimer at 30 mg/kg every 12 hr for up to 10 days without apparent side effects and with successful reduction in blood lead concentrations (Hoogesteijn et al. 2003). However, experimentally repeated administration of extremely high doses of succimer (80 mg/kg every 12 hr for 21 days) in the absence of lead intoxication resulted in regurgitation and morbidity or mortality in up to 66% of cockatiels (*Nymphicus hollandicus*), although the pathophysiology of this result is unknown (Denver et al. 2000). Therefore, succimer should be used at appropriate doses because the safety margin may be narrow.

Regardless of the method of chelation, supportive care including administration of fluids, benzodiazepine (e.g., diazepam) for seizure control, and treatment for concurrent disease (e.g., aspergillosis, endoparasitisim, hemoparasites) is critical to successful treatment. Although chelation has been

used for decades to treat acute lead toxicity in human and veterinary medicine, treatment protocols have been based largely on observational data. Chelation therapy itself may carry risks, so it is important that rehabilitators rely on veterinarians with expertise in treating lead toxicity. Following the recommendations outlined above, side effects associated with chelation are unlikely.

CONCLUSIONS

These guidelines are suitable for treatment of wild-caught raptors regardless of whether or not they display symptoms of lead poisoning. The physiology of lead toxicity is complex and there are occasions where birds present with modest blood-lead levels and clear indications of lead toxicity or, in contrast, with high blood-lead levels and few signs of toxicity. It is thought that the relationship between an individual's condition and its blood lead concentration is determined by factors such as length of exposure, size of the original dose, and duration of exposure (e.g., Pattee et al. 2006). Furthermore, any decision to remove a bird from the wild must be weighed against the biological circumstances in the field and availability of suitable resources for treatment. Although removal from the wild of a bird without apparent symptoms may be difficult to contemplate, it is not a new idea; wild and endangered California condors (*Gymnogyps californianus*) are managed in exactly this way (Finkelstein et al. 2012). However, the material presented in this manuscript is for information purposes only and not intended to be a protocol for treatment of lead poisoning. Finally, researchers and rehabilitators conducting any work with raptors should always be sure to operate within the appropriate local and national permits as required where they work.

ACKNOWLEDGMENTS

This work was funded by the authors' institutions. We thank the authors' institutions for support, and the Associate Editor and a number of reviewers for comments that improved the quality of the manuscript. Any use of trade, product, or firm names is for descriptive purposes only and does not imply endorsement by the U.S. Government.

LITERATURE CITED

Bates, F. Y., D. M. Barnes, and J. M. Higbee. 1968. Lead toxicosis in mallard ducks. Journal of Wildlife Diseases 4:116–125.

Benson, W. W., B. Pharaoh, and P. Miller. 1974. Lead poisoning in a bird of prey. Bulletin of Environmental Contamination and Toxicology 11:105–108.

Burger, J., and M. Gochfeld. 2000. Effects of lead on birds (*Laridae*): a review of laboratory and field studies. Journal of Toxicology and Environmental Health, Part B: Critical Reviews 3:59–78.

Cade, T. J. 2007. Exposure of California condors to lead from spent ammunition. Journal of Wildlife Management 71:2125–2133.

Carpenter, J. W., O. H. Pattee, S. H. Fritts, B. A. Rattner, S. N. Wiemeyer, J. A. Royle, and M. R. Smith. 2003. Experimental lead poisoning in turkey vultures (*Cathartes aura*). Journal of Wildlife Diseases 39:96–104.

Centers for Disease Control. 2014. What do parents need to know to protect their children? http://www.cdc.gov/nceh/lead/acclpp/blood_lead_levels.htm. Accessed 30 Sep 2014.

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 re-introduced to the wild. Environmental Science and Technology 40:6143–6150.

Counter, S. A., L. H. Buchanan, G. Laurell, and F. Ortega. 1998. Field screening of blood lead levels in remote Andean villages. Neurotoxicology 19:871–877.

Custer, T. W., J. C. Franson, and O. H. Pattee. 1984. Tissue lead distribution and hematologic effects in American kestrels (*Falco sparverius* L.) fed biologically incorporated lead. Journal of Wildlife Diseases 20:39–43.

Deem, S. L., S. P. Terrell, and D. J. Forrester. 1998. A retrospective study of morbidity and mortality of raptors in Florida: 1988–1994. Journal of Zoo and Wildlife Medicine 29:160–164.

DeFrancisco, 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.

Degernes, L., S. Kittelson, M. Linck, and P. Manthey. 2002. Survival and reproductive success of trumpeter swans after treatment for lead poisoning. Waterbirds 25:368–374.

Denver, M. C., L. A. Tell, F. D. Galey, J. G. Trupkiewicz, and P. H. Kass. 2000. Comparison of two heavy metal chelators for treatment of lead toxicosis in cockatiels. American Journal of Veterinary Research 61:935–940.

Dierenfeld, E. S. 1989. Vitamin E deficiency in zoo reptiles, birds, and ungulates. Journal of Zoo and Wildlife Medicine 20:3–11.

Domenech, R., and H. Langner. 2009. Blood-lead levels of fall migrant golden eagles in west-central Montana. Pages 221–222 *in* R. T. Watson, M. Fuller, M. Pokras, and W. G. Hunt, editors. Ingestion of lead from spent ammunition: implications for wildlife and humans. The Peregrine Fund, Boise, Idaho, USA.

Duke, G. E. 1986. Alimentary canal: secretion and digestion, special digestive functions, and absorption. Pages 289–302 *in* P. D. Sturkie, editor. Avian Physiology. Springer, New York, New York, USA.

Finkelstein, M. E., D. F. Doak, D. George, J. Burnett, J. Brandt, M. Church, J. Grantham, and D. R. Smith. 2012. Lead poisoning and the deceptive recovery of the critically endangered California condor. Proceedings of the National Academy of Sciences 109:11449–11454.

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 207Pb/206Pb ratios reveal lead exposure history of California condors (*Gymnogyps californianus*). Environmental Science and Technology 44:2639–2647.

Fisher, I. J., D. J. Pain, and V. G. Thomas. 2006. A review of lead poisoning from ammunition sources in terrestrial birds. Biological Conservation 131:421–432.

Franson, J. C., L. Sileo, O. H. Pattee, and J. F. Moore. 1983. Effects of chronic dietary lead in American kestrels (*Falco sparverius*). Journal of Wildlife Diseases 19:110–113.

Fullmer, C. S. 1991. Intestinal calcium and lead absorption: effects of dietary lead and calcium. Environmental Research 54:159–169.

Gamino, V., and U. Höfle. 2013. Pathology and tissue tropism of natural West Nile virus infection in birds: a review. Veterinary Research 44.1:39.

Garcia-Fernandez, A. J., E. Martinez-Lopez, D. Romero, P. Maria-Mojica, A. Godino, and P. Jimenez. 2005. High levels of blood lead in griffon vultures (*Gyps fulvus*) from Cazorla Natural Park (southern Spain). Environmental Toxicology 20:459–463.

Grund, M. D., L. Cornicelli, L. T. Carlson, and F. A. Butler. 2010. Bullet fragmentation and lead deposition in white-tailed deer and domestic sheep. Human-Wildlife Interactions 4:43–50.

Haig, S. M., J. D'Elia, C. Eagles-Smith, J. M. Fair, J. Gervais, G. Herring, J. W. Rivers, and J. H. Schulz. 2014. The persistent problem of lead poisoning in birds from ammunition and fishing tackle. Condor 116:408–428.

Harmata, A. R. 2011. Environmental contaminants in tissues of bald eagles sampled in southwestern Montana, 2006–2008. Journal of Raptor Research 45:119–135.

Hawkins, M. G., H. W. Barron, B. L. Speer, C. Pollock, and J. W. Carpenter. 2013. Birds. Pages 183–437 *in* J. W. Carpenter, editor. Exotic animal formulary. Elsevier Health Sciences, St. Louis, Missouri, USA.

Helander, B., J. Axelsson, H. Borg, K. Holm, and A. Bignert. 2009. Ingestion of lead from ammunition and lead concentrations in white-tailed sea eagles (*Haliaeetus albicilla*) in Sweden. Science of the Total Environment 407:5555–5563.

Hoogesteijn, A. L., B. L. Raphael, P. Calle, R. Cook, and G. Kollias. 2003. Oral treatment of avian lead intoxication with meso-2, 3-dimercaptosuccinic acid. Journal of Zoo and Wildlife Medicine 34:82–87.

Hunt, G., W. Burnham, C. Parish, K. Burnham, B. Mutch, and J. L. Oaks. 2009. Bullet fragments in deer remains: implications for lead exposure in scavengers. Pages 254–258 *in* R. T. Watson, M. Fuller, M. Pokras, and W. G. Hunt, editors. Ingestion of lead from spent ammunition: implications for wildlife and humans. The Peregrine Fund, Boise, Idaho, USA.

Huxoll, C. 2012. 2011 prairie dog shooting survey. South Dakota Wildlife Report No. 2012-08, South Dakota Department of Game, Fish and Parks, Pierre, USA.

Jackowski, D. S., T. Katzner, J. L. Rodrigue, and W. M. Ford. 2015. Monitoring landscape-level distribution and migration phenology of raptors using a volunteer camera trap network. Wildlife Society Bulletin 39:553–563.

Johnson, M. S., W. T. Wickwire, M. J. Quinn, D. J. Ziolkowski, D. Burmistrov, C. A. Menzie, C. Geraghty, M. Minnich, and P. J. Parsons. 2007. Are songbirds at risk from lead at small arms ranges? An application of the spatially explicit exposure model. Environmental Toxicology and Chemistry 26:2215–2225.

Jones, M. P. 2006. Selected infectious diseases of birds of prey. Journal of Exotic Pet Medicine 15:5–17.

Keel, M. K., W. R. Davidson, G. L. Doster, and L. A. Lewis. 2002. Northern bobwhite and lead shot deposition in an upland habitat. Archives of Environmental Contamination and Toxicology 43:318–322.

Kramer, J. L., and P. T. Redig. 1997. Sixteen years of lead poisoning in eagles, 1980-95: an epizootiologic view. Journal of Raptor Research 31:327–332.

Langner, H. W., R. Domench, V. A. Slabe, and S. P. Sullivan. 2015. Lead and mercury in fall migrant golden eagles from western North America. Archives of Environmental Contaminants and Toxicology 69:54–61.

Locke, L. N., S. M. Kerr, and D. Zoromski. 1982. Lead poisoning in common loons (*Gavia immer*). Avian Diseases 26:392–396.

Lumeij, J. T. 1985. Clinicopathologic aspects of lead poisoning in birds: a review. Veterinary Quarterly 7:133–138.

Mautino, M. 1997. Lead and zinc intoxication in zoological medicine: a review. Journal of Zoo and Wildlife Medicine 28:28–35.

McMillin, G. A., and J. A. Bornhorst. 2008. Toxic element testing with clinical specimens. Pages 263–282 *in* A. Dasgupta, editor. Handbook of drug monitoring methods. Humana Press, Totowa, New Jersey, USA.

Murthy, L., E. E. Menden, P. M. Eller, and H. G. Petering. 1973. Atomic absorption determination of zinc, copper, cadmium, and lead in tissues solubilized by aqueous tetramethylammonium hydroxide. Analytical Biochemistry 53:365–372.

Newth, J. L., R. L. Cromie, M. J. Brown, R. J. Delahay, A. A. Meharg, C. Deacon, G. J. Norton, M. F. O'Brian, and D. J. Pain. 2013. Poisoning from lead gunshot: still a threat to wild waterbirds in Britain. European Journal of Wildlife Research 59:195–204.

Pacyna, J. M. 1987. Source-receptor relationships for trace elements in northern Europe. Water Air Soil Pollution 30:825–835.

Pain, D. J., C. Amiard-Triquett, C. Bavoux, G. Brurneleau, L. Eon, and P. Nicolauguillaumet. 1993. Lead poisoning in wild populations of marsh harrier (*Circus aeruginosus*) in the Camargue and Charente-Maritime, France. Ibis 135:379–386.

Pain, D. J., I. Carter, A. W. Sainsbury, R. F. Shore, P. Eden, M. A. Taggart, S. Konstantinos, L. A. Walker, A. A. Meharg, and A. Raab. 2007. Lead contamination and associated disease in captive and reintroduced red kites *Milvus milvus* in England. Science of the Total Environment 376:116–127.

Pain, D. J., I. J. Fisher, and V. G. Thomas. 2009. A global update of lead poisoning in terrestrial birds from ammunition sources. Pages 99–118 *in* R. T. Watson, M. Fuller, M. Pokras, and W. G. Hunt, editors. Ingestion of lead from spent ammunition: implications for wildlife and humans. The Peregrine Fund, Boise, Idaho, USA.

Pattee, O. H., J. W. Carpenter, S. H. Fritts, B. A. Rattner, S. N. Wiemeyer, J. A. Royle, and M. R. Smith. 2006. Lead poisoning in captive Andean condors (*Vultur gryphus*). Journal of Wildlife Diseases 42:772–779.

Pattee, O. H., S. N. Wiemeyer, B. M. Mulhern, L. Sileo, and J. W. Carpenter. 1981. Experimental lead-shot poisoning in bald eagles. Journal of Wildlife Management 45:806–810.

Pokras, M. A., and M. R. Kneeland. 2009. Understanding lead uptake and effects across species lines: a conservation medicine based approach. Pages 1–16 *in* R. T. Watson, M. Fuller, M. Pokras, and W. G. Hunt, editors. Ingestion of lead from spent ammunition: implications for wildlife and humans. The Peregrine Fund, Boise, Idaho, USA.

Rabinowitz, M. B. 1991. Toxicokinetics of bone lead. Environmental Health Perspectives 91:33–37.

Redig, P. T., and L. R. Arent. 2008. Raptor toxicology. Veterinary Clinics of North America: Exotic Animal Practice 11:261–282.

Redig, P. T., C. M. Stowe, D. M. Barnes, and T. D. Arent. 1980. Lead toxicosis in raptors. Journal of the American Veterinary Medical Association 177:941–943.

Rodriguez-Ramos, J., V. Gutierrez, U. Höfle, R. Mateo, L. Monsalve, E. Crespo, and J. M. Blanco. 2009. Lead in griffon and cinereous vultures in central Spain: correlations between clinical signs and blood lead levels. Pages 235–236 *in* R. T. Watson, M. Fuller, M. Pokras, and W. G. Hunt, editors. Ingestion of lead from spent ammunition: implications for wildlife and humans. The Peregrine Fund, Boise, Idaho, USA.

Samour, J. H., and J. Naldo. 2002. Diagnosis and therapeutic management of lead toxicosis in falcons in Saudi Arabia. Journal of Avian Medicine and Surgery 16:16–20.

Scheuhammer, A. M. 1987. The chronic toxicity of aluminium, cadmium, mercury, and lead in birds: a review. Environmental Pollution 46:263–295.

Scheuhammer, A. M., and D. M. Templeton. 1998. Use of stable isotope ratios to distinguish sources of lead exposure in wild birds. Ecotoxicology 7:37–42.

Schwartz, S. L., J. R. Hayes, R. S. Ide, C. B. Johnson, and P. D. Doolan. 1966. Studies of the nephrotoxicity of ethylenediaminetetraacetic acid. Biochemical Pharmacology 15:377–389.

Sears, J., S. W. Cooke, Z. R. Cooke, and T. J. Heron. 1989. A method for the treatment of lead poisoning in the mute swan (*Cygnus olor*) and its long-term success. British Veterinary Journal 145:586–594.

Sobin, C., N. Parisi, T. Schaub, and E. de la Riva. 2011. A Bland-Altman comparison of the Lead Care® System and inductively coupled plasma mass spectrometry for detecting low-level lead in child whole blood samples. Journal of Medical Toxicology 7:24–32.

Stauber, E., N. Finch, P. A. Talcott, and J. M. Gay. 2010. Lead poisoning of bald (*Haliaeetus leucocephalus*) and golden (*Aquila chrysaetos*) eagles in the US inland Pacific Northwest region—an 18-year retrospective study: 1991–2008. Journal of Avian Medicine and Surgery 24:279–287.

Thomas, V., and T. Spiro. 1994. Emissions and exposure to metals: cadmium and lead. Pages 297–320 *in* R. Socolow, C. Andrews, F. Berkhout, and V. Thomas, editors. Industrial ecology and global change. Cambridge University Press, Cambridge, United Kingdom.

Ting, B. T., and M. Janghorbani. 1988. Optimisation of instrumental parameters for the precise measurement of isotope ratios with inductively coupled plasma mass spectrometry. Journal of Analytical Atomic Spectrometry 3:325–336.

Walker, L. A., J. S. Chaplow, A. J. Lawlor, M. G. Pereira, E. D. Potter, A. W. Sainsbury, and R. F. Shore. 2012. Lead (Pb) concentrations in predatory bird livers 2010 and 2011: a Predatory Bird Monitoring Scheme (PBMS) report. CEH Project no. C04288. NERC/Centre for Ecology & Hydrology, Lancaster, United Kingdom.

Walter, H., and K. P. Reese. 2003. Fall diet of chukars (*Alextoris chukar*) in eastern Oregon and discovery of ingested lead pellets. Western North American Naturalist 63:402–405.

Associate Editor: Hernandez.