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CHARACTERIZING GOLDEN EAGLE RISK TO LEAD AND ANTICOAGULANT RODENTICIDE EXPOSURE: A REVIEW

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ABSTRACT.—Contaminant exposure is among the many threats to Golden Eagle (*Aquila chrysaetos*) populations throughout North America, particularly lead poisoning and anticoagulant rodenticides (AR). These threats may act in concert with others (e.g., lead poisoning and trauma associated with striking objects) to exacerbate risk. Golden Eagles are skilled hunters but also exploit scavenging opportunities, making them particularly susceptible to contaminant exposure from ingesting tissues of poisoned or shot animals. Lead poisoning has long been recognized as an important source of mortality for Golden Eagles throughout North America. More recently, ARs have been associated with both sublethal and lethal effects in raptor species worldwide. In this review, we examine the current state of knowledge for lead and AR exposure in Golden Eagles, drawing from the broader raptor contaminant ecology literature. We examine lead and AR sources within Golden Eagle habitats, exposure routes and toxicity, effects on individuals and populations, synergistic effects, and data and information needs. Continued research addressing data needs and information gaps will help with Golden Eagle conservation planning.

KEY WORDS: *Golden Eagle*; Aquila chrysaetos; *bullet fragments*; *first generation anticoagulant rodenticide*; *lead poisoning*; *second generation anticoagulant rodenticide*.

CARACTERIZACIÓN DEL RIESGO DE EXPOSICIÓN DE *AQUILA CHRYSAETOS* AL PLOMO Y A LOS RODENTICIDAS ANTICOAGULANTES: UNA REVISIÓN

RESUMEN.—La exposición a contaminantes se encuentra entre las muchas amenazas a las poblaciones de *Aquila chrysaetos* a lo largo de América del Norte, particularmente el envenenamiento con plomo y rodenticidas anticoagulantes (RA). Estas amenazas pueden actuar en conjunto con otras (e.g., envenenamiento con plomo y trauma asociado con objetos contundentes) para exacerbar el riesgo. Los individuos de *A. chrysaetos* son hábiles cazadores, pero también aprovechan las oportunidades para alimentarse de carroña, haciéndolos particularmente susceptibles a la exposición a contaminantes por la ingesta de tejidos de animales envenenados o muertos por disparos. El envenenamiento por plomo ha sido reconocido como una importante fuente de mortalidad para *A. chrysaetos* en toda América del Norte. Más recientemente, los RAs han sido asociados a efectos subletales y letales en especies de aves rapaces a nivel mundial. En esta revisión, examinamos el estado actual del conocimiento sobre la exposición al plomo y a los RAs de individuos de *A. chrysaetos*, a partir de la literatura sobre la ecología de los contaminantes en aves rapaces. Examinamos las fuentes de plomo y de RAs en los hábitats de *A. chrysaetos*, las rutas de exposición y toxicidad, los efectos sobre los individuos y sobre las poblaciones, los efectos sinérgicos y la necesidad de datos e información. Se requieren investigaciones adicionales que se enfoquen en la necesidad de datos y en los vacíos de información, para contribuir con los planes de conservación de *A. chrysaetos*.

[Traducción del equipo editorial]

The predatory and scavenging behaviors of Golden Eagles (*Aquila chrysaetos*), along with their high trophic position in terrestrial food webs, make them particularly susceptible to exposure and bioaccumulation of environmental contaminants (Bedrosian et al. 2012, Harmata and Restani 2013, Franson and Russell 2014, Langner et al. 2015). Lead in particular can impair survival (Finkelstein et al. 2012) and

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nestling growth (Hoffman et al. 1985), and is routinely found in tissues of scavenging eagles (see Bedrosian et al. 2012, Cruz-Martinez et al. 2012, Hunt 2012, Golden et al. 2016). Additionally, Golden Eagles may be exposed to other contaminants such as anticoagulant rodenticides (ARs) because they commonly consume agricultural pests and other animals that may have been poisoned by rodenticides (Langford et al. 2013, Kelly et al. 2014; Fig. 1).

Research on contaminant exposure and effects on Golden Eagles has been largely dominated by studies of lead in free-ranging Golden Eagles, primarily in migrating birds (see Pattee et al. 1990, Harmata and Restani 1995, Langner et al. 2015) or in salvaged carcasses of birds that were found dead with unknown history (see Wayland and Bollinger 1999, Franson and Russell 2014). Although these studies indicate potential lead exposure in free-ranging birds, little is known about lead exposure during the reproduction and nestling stages, which are among the most critical periods for exposure to contaminants in many bird species (ATSDR 2007). Additionally, knowledge of species-specific toxicological endpoints for lead are limited for raptors, complicating interpretation of tissue concentrations in Golden Eagles given the substantial range in sensitivity that has been documented among other species (Buekers et al. 2009, Haig et al. 2014). Even greater limitations exist in the understanding of contaminants such as ARs, which are a contaminant of emerging concern, particularly for raptor species (Rattner et al. 2014a). The goal of our review was to summarize the current understanding of lead exposure and toxicology in Golden Eagles, and raptors in general, as well as to review available information regarding potential threats from ARs. We examine lead and AR sources within Golden Eagle habitats, exposure routes and toxicity, impacts on individuals and populations, synergistic effects, and data and information needs.

Although avian lead exposure and toxicology have been studied for decades, the substantial variation in interspecific sensitivity to lead exposure confounds direct, tissue concentration-based quantification of toxicological risk in Golden Eagles (Haig et al. 2014). To facilitate an understanding of potential lead effects to Golden Eagles, we draw from the broad literature base across multiple avian species, particularly focusing on other raptor species. For ARs, the toxicological science is relatively limited in comparison to that of lead, and data on AR exposure in Golden Eagles are scarce. Recent efforts to characterize data gaps and synthesize the state of knowledge regarding AR risk to wildlife (Rattner et al. 2014a) can serve as a framework for understanding how this applies to Golden Eagles and provide guidance on future research and conservation needs.

Lead Exposure. Lead poisoning in birds has long been an important conservation issue (Bellrose 1959, Pattee et al. 1990, Scheuhammer and Norris 1996, Rattner et al. 2008, Franson and Russell 2014, Haig et al. 2014, Russell and Franson 2014, Golden et al. 2016). After ingestion, lead is assimilated within the gastrointestinal tract and transported throughout the body via the circulatory system (Fisher et al. 2003, Redig and Arent 2008). Lead exposure in birds can result in a wide range of physiological and neurological responses, including mortality (Haig et al. 2014). Numerous sources of lead occur in the landscape, including legacy lead-based paint (Finkelstein et al. 2003), soil and sediment lead, and mining and smelting activities (Henny et al. 1994, Legagneux et al. 2014). However, lead from hunting and recreational shooting activities remains one of the most important sources (Church et al. 2006, Finkelstein et al. 2010, Legagneux et al. 2014, Haig et al. 2014, Golden et al. 2016; Fig. 1). Avian scavengers are often exposed to spent lead shot and bullet fragments because of their propensity to feed on carcasses and offal remaining in the field (Church et al. 2006, Finkelstein et al. 2010, Legagneux et al. 2014). To date, lead exposure has been measured in more than 120 bird species (Haig et al. 2014). Traditionally, waterfowl shot with lead pellets were considered an important vector for many species of scavenging raptors (Pattee and Hennes 1983, Scheuhammer and Norris 1996). Since the ban of lead shot for waterfowl hunting in 1991, both Bald (*Haliaeetus leucocephalus*) and Golden eagles continue to be secondarily exposed to lead (Stauber et al. 2010, Bedrosian et al. 2012, Cruz-Martinez et al. 2012, Jenni et al. 2015). The ongoing exposure of both Bald and Golden eagles has been at least partially attributed to ingestion of lead bullet fragments associated with big-game hunting (Bedrosian et al. 2012, Franson and Russell 2014), as well as the use of lead shot in upland habitats (Stauber et al. 2010, Kelly et al. 2011). For species like Golden Eagles that are opportunistic scavengers (Kochert et al. 2002, Legagneux et al. 2014), feeding on carrion increases their likelihood of lead exposure and the

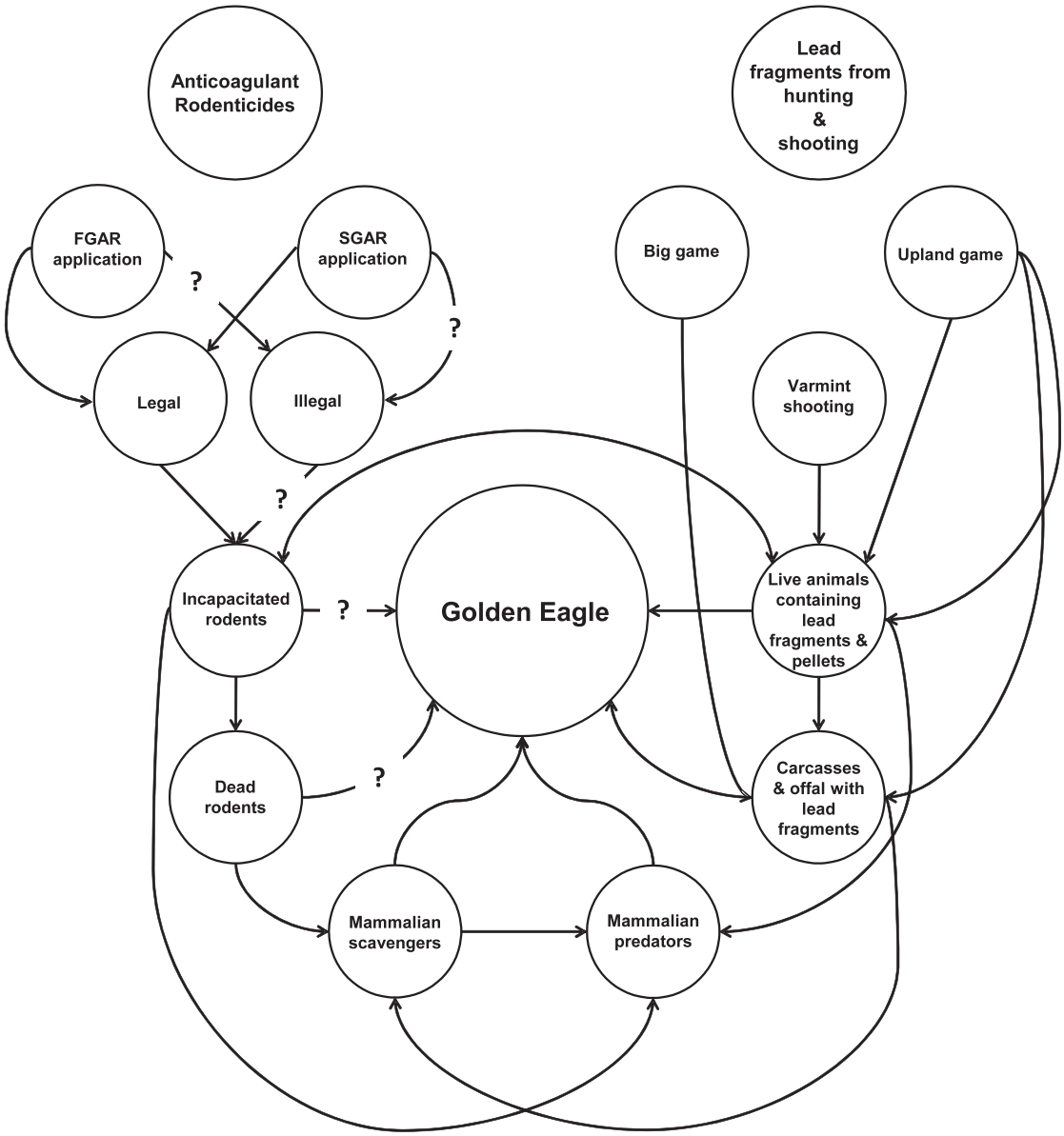

Figure 1. Conceptual model of potential Golden Eagle exposure to anticoagulant rodenticides and lead, with plausible and hypothetical linkages.

potential effects of ingesting lead fragments from carcasses.

Anticoagulant Rodenticide Exposure. Anticoagulant rodenticides have been used globally since the 1940s to manage rodent populations in urban and rural landscapes (Rattner et al. 2014a.). They inhibit vitamin K-epoxide reductase at two points in the vitamin K cycle, and limit or prevent the activation of blood clotting factors required for hemostasis (Rattner et al. 2014a). First generation ARs (FGARs; e.g., warfarin, chlorophacinone, and diphacinone) require multiple ingestions of bait by a target species over several days to cause coagulopathy that results in mortality (Rattner et al. 2014a). First generation

ARs remain in wide use despite evidence of genetic resistance in some species and settings (Buckle et al. 1994, Rattner et al. 2014a). As a result, more potent second generation ARs (SGARs) such as brodifacoum, bromadiolone, difenacoum, and difethialone were developed beginning in the 1970s. Second generation ARs are more acutely toxic at lower doses than FGARs, and in many cases SGARs require only a single bait ingestion to be lethal to the target species (Rattner et al. 2014a). Second generation ARs are also more persistent in vertebrate livers (Parmar et al. 1987, Stone et al. 2003, Erickson and Urban 2004). Consequently, raptors that prey on SGAR-poisoned animals may have a higher likelihood of bioaccumulation and secondary poisoning compared to raptors exposed to FGARs (Rattner et al. 2014a).

Recently, new regulations on the use of SGARs in both the United States and Canada were enacted to limit or reduce exposure of humans, pets, and nontarget predatory birds to SGARs (Health Canada 2012, U.S. EPA 2012). As the new regulations take effect, an increase in the use of some FGARs such as chlorophacinone (Rozol®) may occur (Vyas et al. 2013, Rattner et al. 2015). The mechanism of AR toxicity (vitamin K inhibition and failure to carboxylate clotting factors) results in a lag time between ingestion, coagulopathy, and death for both FGARs and SGARs. The lag time between ingestion and coagulopathy occurs because several days are needed for clearance of existing active clotting factors from the blood (Rattner et al. 2014a). First generation ARs require consecutive days of intake in target rodents to accumulate a lethal dose and subsequent accumulation in tissues of target and nontarget animals is lower (Eason and Ogilvie 2009). The half-life of most FGARs in both target and nontarget wildlife is generally measured in hours to days and is much shorter than SGARs (Eason and Ogilvie 2009). Second generation ARs have the same mechanism of action as FGARs, but they have an increased affinity for the target enzyme (vitamin K epoxide reductase), increased ability to disrupt the vitamin K-epoxide cycle at more points, and significantly longer half-lives in blood and liver (Watt et al. 2005). Because of the time lag between ingestion and mortality associated with SGARs and their greater half-life than FGARs of months to a year versus days to weeks, target pest species may consume multiple lethal SGAR doses prior to death, resulting in greatly elevated concentrations of SGARs in their organs (Stone et al. 2003). The longer half-lives of SGARs and their greater tissue persistence (Rattner et al. 2014a) mean predators and scavengers are at a higher risk of becoming exposed via secondary or tertiary poisoning after consuming SGAR-laden prey (Riley et al. 2007, Lima and Salmon 2010, Tosh et al. 2011, Serieys et al. 2015; Fig. 1).

The prevalence of avian AR exposure may be widespread for many species, in particular avian raptors (Stone et al. 2003, Murray 2011, Sanchez-Barbudo et al. 2012). Anticoagulant rodenticides (chiefly SGARs) have been detected in raptors in the United States (Stansley et al. 2014), Canada (Albert et al. 2010, Thomas et al. 2011), Denmark (Christensen et al. 2012), France (Lambert et al. 2007), Norway (Langford et al. 2013), Spain (Sanchez-Barbudo et al. 2012, Ruiz-Suárez et al. 2014, López-Perea et al. 2015), United Kingdom (Newton et al. 1990, Walker et al. 2008), and elsewhere (Rattner et al. 2014a). Although there have been field studies to assess AR exposure for many species of raptors (e.g., Golden Eagles), more comprehensive research is needed on the potential effects and frequency and magnitude of exposure from these compounds.

To examine published records of Golden Eagle exposure to ARs, we searched the Thompson Reuters ISI Web of Science® on 29 June 2016, using all possible combinations of these topic words: anticoagulant, FGAR, Golden Eagle, raptor, rodenticide, and SGAR, and found a total of six published studies that attempted to measure ARs in Golden Eagles (Hosea 2000, Stone et al. 2003, Thomas et al. 2011, Sanchez-Barbudo et al. 2012, Langford et al. 2013, Kelly et al. 2014). Four of these studies evaluated both FGARs and SGARs in Golden Eagles and in all cases only SGARs were detected. Across all Golden Eagles, 67% (32 of 48) of the birds sampled were exposed and at least 17% (8 of 48) of the birds exceeded concentrations (0.1 ppm wet weight [ww]) thought to represent toxic concentrations in the liver for Great Horned Owls (*Bubo virginianus*; Thomas et al. 2011). Where the type of AR was reported, brodifacoum was detected in four of the studies ($n = 26$), bromadiolone and flocoumafen in two of the studies ($n = 10$ and $n = 3$, respectively), and difethialone in one study ($n = 1$). Although the results implicate SGARs as the primary rodenticides associated with Golden Eagle exposure, the short half-lives of FGARs may preclude their detection. Of the 48 sampled Golden Eagles, over half of those carcasses originated from populated urbanized areas not typical of their preferred habitat (see Hosea

2000, Langford et al. 2013, Kelly et al. 2014), where FGAR applications would not typically occur. The prevalence of SGAR-exposed Golden Eagles in urbanized areas across these studies may simply be an artifact of animal detectability, with sick or deceased eagles recovered more regularly in urbanized areas.

Sources and Distribution of Lead and ARs in the Environment

Lead. Upon impact, lead rifle bullets typically shatter into several hundred fragments distributed throughout the carcass and viscera of a target animal. Estimates indicate that a single big-game bullet (e.g., 100–180 grains) impact results in an average of 235 fragments in the eviscerated carcass and 170 in the viscera (Hunt et al. 2006, 2009, Knott et al. 2010). This can substantially increase the risk of lead exposure to scavenging Golden Eagles feeding on shot carcasses and offal during the hunting season (Kelly et al. 2011, Bedrosian et al. 2012, Cruz-Martinez et al. 2012, Legagneux et al. 2014). Increased numbers of small lead fragments in carcasses result in lead being easily ingested because of their larger surface area, resulting in lead being readily absorbed into the blood stream by scavengers (Barltrop and Meek 1979).

The relationship between blood lead concentrations in eagles and availability of lead bullet fragments in offal or shot game carcasses has been reported in a number of studies associated with big-game hunting. Across all four North American migratory flyways, the likelihood of lead poisoning in Bald Eagles (but not Golden Eagles) was greater during the big-game hunting season than the nonhunting season (Franson and Russell 2014). In Minnesota, during the white-tailed deer (*Odocoileus virginianus*) hunting season, lead concentrations in Bald Eagles increased on average 7.6 times compared to the nonhunting season (Cruz-Martinez et al. 2012). In southern California, Golden Eagle blood lead concentrations were approximately 1.8 times higher during the fall big-game hunting season than during the remainder of the year (Pattee et al. 1990). Likewise, in the Pacific Northwest (ID, OR, and WA), the prevalence of moribund Golden Eagles increased, along with lead exposure, from January to March, during a time when big-game carcasses remained on the landscape although the big-game hunting season had ended (Stauber et al. 2010).

Aside from big-game hunting, other shooting activities also provide a potential source of lead to Golden Eagles. For example, both ground and aerial predator management programs may provide a substantial source of lead in the environment. In 2013, Federal aerial gunning programs killed at least 20,800 coyotes (*Canis latrans*) in the western United States (AZ, CA, CO, ID, MT, NW, NV, OR, UT, WA, WY) as part of the Wildlife Damage Program within the U.S. Department of Agriculture's (U.S.D.A.) Wildlife Services program (U.S.D.A. 2014). Aerial coyote gunning is typically conducted using shotguns with lead-based buckshot (U.S.D.A. 2009) because lead shot travels at considerably slower speeds than higher velocity rifle bullets and has a lower potential for ricocheting and fragmenting (U.S.D.A. 2009). Therefore, coyote carcasses may contain fewer lead fragments relative to animals shot with rifle ammunition. The proportion of shot coyotes that are left on the landscape due to predator management activities is unreported and currently unclear. However, these carcasses could be a potential source of lead to Golden Eagles and other scavengers, depending on when predator management occurs, particularly given the propensity of Golden Eagles to scavenge carrion during the nesting season (Boag 1977) and because they will readily scavenge coyotes (Kochert et al. 2002). Recreational or non-agency management of coyotes also results in large numbers of coyote carcasses on the landscape and potential lead exposure in Golden Eagles (Fig. 1). Typically, those coyotes are shot with high velocity rifles and ammunition that produces large numbers of lead fragments (Stauber et al. 2010).

Another source of ammunition-derived lead to Golden Eagles is associated with the shooting of ground squirrels and prairie dogs (Sciuridae), which can be important agricultural pests. Individual recreational shooters can kill over 100 animals per day (Pauli and Buskirk 2007, Herring et al. 2016), and almost all shooters routinely use lead-based bullets that fragment to result in quantities of small lead fragments sufficient to be lethal to raptors (Knopper et al. 2006, Pauli and Buskirk 2007, Stephens et al. 2008, Herring et al. 2016). The link between ground squirrel shooting and the potential for elevated lead exposure in Golden Eagles was postulated as long ago as the 1980s (Pattee et al. 1990). Although research evaluating the availability of lead to scavengers from ground squirrel and prairie dog hunting is limited, two studies (Pauli and

Buskirk 2007, Herring et al. 2016) estimated that 47% and 7% of the black-tailed prairie dog (*Cynomys ludovicianus*) and Belding's ground squirrel (*Urocitellus beldingi*) carcasses respectively had sufficient mass of lead to be lethal to nestling raptors based on dosing studies of American Kestrels (Hoffman et al. 1985). Similarly, Knopper et al. (2006) found that 20% of shot Richardson's ground squirrels (*Urocitellus richardsonii*) contained sufficient quantities of lead to be lethal to adult Swainson's Hawks (*Buteo swainsoni*) and to Ferruginous Hawks (*B. regalis*) if they consumed at least 6.5 ground squirrels per bird. Based on their dietary requirements, this would take 23 d if they fed exclusively on shot Richardson's ground squirrels (Knopper et al. 2006).

One limitation to these lead-exposure studies in Golden Eagles and other raptors is that all were focused on adult/subadult (hereafter "free-flying") and most studies did not occur during the breeding season. A recent study in the Columbia Basin in Washington found 65% of the breeding adult Golden Eagles had blood lead concentrations elevated above background levels (0.21–0.50 ppm) and 24% had concentrations (0.51–1.00 ppm) representing chronic exposure (Watson and Davies 2015). To our knowledge, there have been only two studies (Craig and Craig 1998, Stephens et al. 2008) that have examined lead exposure in Golden Eagle nestlings, and both were limited in scope. During 1991 and 1992, blood lead concentrations in six of 12 nestling Golden Eagles sampled by Craig and Craig (1998) in east-central Idaho just prior to fledging, exceeded the detection limit of 0.01 ppm, ranging from 0.01–0.06 ppm (mean = 0.03 ppm; T. Craig and E. Craig pers. comm.). More recently, seven Golden Eagle nestlings sampled in the Thunder Basin National Grassland in Wyoming had blood lead concentrations ranging from 0.02 to 0.07 ppm (mean = 0.03 ppm; Stephens et al. 2008). Although both studies reported relatively low concentrations in Golden Eagle nestlings, neither study had adequate sample sizes or spatial coverage to broadly characterize lead exposure in nestlings across a wide geographic range.

Anticoagulant Rodenticides. Source attribution for ARs is seemingly less complicated than for lead because ARs specifically originate from treatment activities to control rodents. Anticoagulant source attribution to a site, region, or specific rodenticide application could be difficult depending on the time of year (e.g., migration versus breeding seasons) or if the use of ARs was related to illegal applications. Although SGARs are registered for legal commensal rodent control in and around structures, more recently their use has been reported with illegal application associated with illicit marijuana cultivation practices (Gabriel et al. 2012). Although the use of ARs connected with illegal marijuana cultivation may not affect Golden Eagles because the associated habitats are so different, it does illustrate that ARs used illegally have the potential to increase exposure for wildlife. Anticoagulant rodenticides are not target-specific in their toxicity and can result in primary exposure of many nontarget animals and potential secondary poisoning of raptors that depend on these species as their main prey (Brakes and Smith 2005, Geduhn et al. 2016). Anticoagulant rodenticide exposure in rodents decreases their ability to escape from predators (Cox and Smith 1992). Additionally, exposed rodents forage more frequently in the open and suffer more mortality aboveground than unexposed rodents, enhancing their risk to predation by raptors (Cox and Smith 1992, Howald et al. 1999, Vyas et al. 2012, Elliott et al. 2014). These symptoms would make exposed rodents easier prey for predators such as Golden Eagles (Fig. 1). Tertiary poisoning of scavenging birds can also occur from eating mammals that were secondarily poisoned by SGARs (Ebbert and Burek-Huntington 2010, Lee et al. 2013). Many SGARs accumulate in soft tissues of scavenging animals such as coyotes (Poessel et al. 2015), and exposed animals shot and left in the field could also be a source of SGARs to avian scavengers such as Golden Eagles (Fig. 1). A facultative scavenger like a Golden Eagle could be exposed to ARs either from hunting or scavenging AR-exposed prey.

Important Assumptions Used to Identify Primary Sources of Lead and AR Stressors

There are two primary assumptions associated with an animal's exposure to lead and ARs; one assumption is common to both lead and ARs, the other assumption only applies to lead. In the case of lead, researchers may assume exposure occurred within the area where a bird was sampled (e.g., home range). However, if birds are migrating or dispersing, this assumption may not be valid (Bedrosian et al. 2012). The ability of Golden Eagles to migrate hundreds of kilometers in short periods of time (Broduer et al. 1996, Kochert et al. 2002) means that lead measured in blood during migration (recent exposure) cannot always be attributed to local lead sources. Although the half-life of lead in the blood of

Golden Eagles is unknown, the half-life in California Condors (*Gymnogyps californianus*) is 14 d (Fry et al. 2009). Thus, a migrating Golden Eagle could travel approximately 238–910 km during this 14-d period (assuming an average daily migration flight of 17–65 km; R. Domenech and B. Bedrosian pers. comm., Broduer et al. 1996). As a result, studies sampling blood lead from raptors captured during migration may not accurately reflect local exposure, which has important ramifications for management if not evaluated at the appropriate scale.

Similarly, for AR exposure, liver concentrations may not reflect the local area from which a bird was recovered, due to lack of knowledge on travel distance from AR source, time elapsed since ingestion of AR-contaminated prey, and the spatial-temporal mosaic of AR use in the region. In contrast, blood lead concentrations in resident breeding birds and nestlings would reflect more recent and localized exposure, whereas lead measured in avian livers or bone may reflect older exposure. Similarly, liver AR concentrations could reflect local exposure in resident or breeding birds, particularly for FGARs because of their short half-lives (Erickson and Urban 2004, U.S. EPA 2007, Rattner et al. 2011).

The second assumption associated with exposure is unique to lead rather than ARs, and has to do with its origins. Lead detected in avian blood or any other tissue matrix is commonly assumed to be associated with spent ammunition (see statements in Redig and Arent 2008, Stauber et al. 2010), and studies using lead isotopes often provide support to this assertion (Church et al. 2006, Finkelstein et al. 2012, Lambertucci et al. 2011). However, other sources of lead in the environment require more accurate accounting to depict lead exposure in eagles (Henny et al. 1994, Scheuhammer and Templeton 1998, Haig et al. 2014), as some avian scavengers are exposed to lead from sources other than ammunition (see Finkelstein et al. 2012, Legagneux et al. 2014). For instance, some lead in California Condors has been linked to lead-based paint at contaminated sites (Finkelstein et al. 2012), and a small proportion of lead in exposed Common Ravens (*Corvus corax*) was associated with soil lead (Legagneux et al. 2014). Blood lead concentrations considered to be background (<0.10 ppm) had lead isotope ratios that did not differ from the isotopic signature associated with soil or lichens (Legagneux et al. 2014). In contrast, isotope ratios in blood with elevated lead concentrations (>0.10 ppm) differed significantly from background sources and were generally associated with isotope ratios of lead-based ammunition (Legagneux et al. 2014). However, this observation may not hold true for all lead exposure across bird species or in other geographic regions.

Toxicity

Assessing the risk of either lead or AR exposure to Golden Eagles is particularly difficult because species-specific toxicological endpoints are not well defined, and interspecific variability in sensitivity can be large (Buekers et al. 2009, Haig et al. 2014, Rattner et al. 2014b). A number of studies have assessed toxicological responses of birds to lead exposure (Franson and Pain 2011, Golden et al. 2016), but the variability in toxicity among species makes it difficult to draw inference to other species where no toxicity data exist (Buekers et al. 2009). To illustrate the difficulty in assessing the potential effects of lead on Golden Eagles, we draw inference from recent reviews in the literature. A summary of results from nine repeated-dosing studies (three laboratory and six field studies) for nine avian species (six waterbirds, three raptors) found a 50-fold range in estimates of no observed–effect concentrations (NOEC) for lead (Buekers et al. 2009). Because toxicity endpoints do not exist for Golden Eagles, we infer from studies on other avian species that there would be considerable differences in the observed effects of lead on growth, survival, behavior, and reproduction of Golden Eagles. An important step in reducing lead exposure in Golden Eagles would be the development of a standardized vulnerability assessment that considers life history traits, exposure likelihoods, and species-specific sensitivities to the toxic effects of lead (see Golden and Rattner 2003).

Much of the peer-reviewed literature associated with dosing studies has relied on the use of NOECs and lowest observed effect concentrations (LOECs). However, these approaches can be flawed because these values are not determined from regression of the dose-response curve and do not incorporate the full set of data from an experiment. Rather, NOEC and LOEC values are determined by selecting the experimental treatment concentration at which either no effect or the lowest effect occurred (Landis and Chapman 2011). Additionally, because NOEC and LOEC values are determined for a given dataset based on study dosages used, they are often inconsistent among studies (Fox 2008, Landis and Chapman 2011). Although there has been a movement toward using curve-fitting models, we

Table 1. Generalized criteria for classifying lead exposure in birds across blood, liver, and bone matrices developed from Franson (1996), Pain (1996), and Pattee and Pain (2003).

TISSUE TYPE	CONCENTRATION PPM WET WEIGHT	DESCRIPTIVE TOXICITY
Blood	<0.2	Background
	0.2–<0.5	Subclinical
	0.50–1.0	Clinical poisoning
	>1.0	Severe clinical poisoning
Liver	<2.0	Background
	2.0–<6.0	Subclinical
	6.0–15.0	Clinical poisoning
	>15.0	Severe clinical poisoning
Bone	<10.0	Background
	10.0–20.0	Subclinical
	>20.0	Severe clinical poisoning

were limited in selecting studies from which we could draw inference, and the only values available for comparison were derived based on the NOEC/LOEC approach.

Estimates of NOECs can be difficult to compare because of the high variability associated with species-specific sensitivities to lead and different endpoints measured in studies. Other approaches such as hazardous concentrations for the 5th percentile (HC_5; Buekers et al. 2009) broaden the risk assessment to include groups of species. Buekers at el. (2009) used NOEC data from nine repeated dosing studies to determine the HC_5 across species' (the 5^{th} percentile that will protect 95% of the species), using growth, reproduction or blood chemistry as endpoints. The resulting HC_5 for birds was 0.71 ppm ww (90% confidence interval = 0.26–1.16 ppm) in blood, the threshold below which 95% of the birds are thought to be protected from lead toxicity (Buekers et al. 2009). One caveat to this study is that raptors made up only three of nine species used in the model, American Kestrel, Osprey, and Turkey Vulture (*Cathartes aura*), and many raptors have been shown to be more sensitive to lead exposure than other bird species (Hoffman et al. 1985). In fact, the geometric mean NOEC value for non-raptor species was more than two times higher than that for raptors. Therefore, the HC_5 value may be higher than a level that would be protective of just raptor species.

Lead contaminant exposure in early life stages is often more deleterious due to the sensitivity of development to contaminants compared with adults across species (ATSDR 2007). American Kestrel nestlings dosed with a range of lead concentrations (metallic lead powder) experienced decreased growth rates at exposures of 125 and 625 ppm body weight/d, and nestling masses were 16% and 39% lower, respectively, than controls after 10 d (Hoffman et al. 1985). Overt mortality was only observed at the 625 ppm body mass/d dose after six d of dosing, with an overall mortality rate of 40% (Hoffman et al. 1985). Thus, mortality occurred within the first six doses, after a total consumption of 60–70 mg of lead (Hoffman et al. 1985). Shot rodents such as ground squirrels and black-tailed prairie dog carcasses can contain between 39 and 228 mg of lead, and 7–40% of all carcasses in recent studies had a sufficient mass of lead to be lethal to raptors (Pauli and Buskirk 2007, Stephens et al. 2008, Herring et al. 2016). However, the rate at which ingested lead fragments are eroded and absorbed into the blood of birds is poorly understood and has important ramifications on the effects manifested in birds eating the carcasses, particularly for nestlings feeding on lead-laced ground squirrels provided by adults. Although scavenging birds can detect large lead particles in prey and avoid them or regurgitate them, smaller lead particles are more likely to be completely digested (Nadjafzadeh et al. 2015).

To facilitate a simplified understanding of measured lead concentrations in tissues, generalized toxicity thresholds have been developed (see Franson 1996, Pain 1996, Kramer and Redig 1997, Pattee and Pain 2003; Table 1). Routinely used throughout the published literature (see Harmata and Restani 2013, Jenni et al. 2015, Watson and Davies 2015), toxicity thresholds provide an understanding of the potential for toxicological effects based upon tissue concentrations. One minor constraint associated with using toxicity thresholds is the concentration associated with background, which in blood is considered to be <0.20 ppm (Franson 1996, Pain 1996, Pattee and Pain 2003). Recent data from blood samples of nestling avian scavengers suggest that background concentrations may be lower by an order of magnitude (e.g., median = 0.01 ppm [Craighead and Bedrosian 2008], mean = 0.003 ppm [Bedrosian et al. 2012]). Regardless of constraints and species-specific differences, these toxicity thresholds allow researchers and avian health care providers to rapidly characterize lead exposure and potential risk.

Sublethal and lethal effects resulting from AR exposure are not well-defined for most avian species.

Table 2. Toxicity categories for avian species relative to LD_{50} and LC_{50} values used by the United States Environmental Protection Agency (U.S. EPA 2004).

DESCRIPTIVE CATEGORY	AVIAN LD_{50} PPM	AVIAN LC_{50} PPM
Relatively non-toxic	>2000	>5000
Slightly toxic	500–2000	1000–5000
Moderately toxic	50–500	500–1000
Highly toxic	10–50	50–500
Extremely toxic	<10	<50

We are aware of only one potential toxicity threshold referenced for liver tissue of 0.1–0.2 ppm ww for the sum of three ARs (bromadiolone + brodifacoum + difethialone), which was associated with mortality in barn owls (Newton et al. 1999). Liver is the most common tissue for measuring anticoagulant rodenticide exposure because it is the primary site for vitamin K reprocessing, which is needed for clotting factor carboxylation in hemostasis (Rattner et al. 2015). Although this toxicity benchmark is applied to other species (Albert et al. 2010, Thomas et al. 2011, Langford et al. 2013), there appears to be little consistency in the response of birds based upon their liver concentrations as measured at death. A recent analysis of published data on hepatic SGAR concentrations across 270 individuals of four species of raptors estimated that the probability of toxicosis spanned a wide exposure range, occurring below the 0.1 ppm ww threshold in some species (Thomas et al. 2011). Furthermore, data pooled across four raptor species including the Barn Owl, Barred Owl (*Strix varia*), Great Horned Owl, and Red-tailed Hawk (*B. jamaicensis*), revealed that 5% of all birds showed signs of toxicosis when liver SGAR concentrations were only 0.02 ppm ww, and 20% of birds showed signs of toxicosis when liver concentrations reached 0.08 ppm ww (Thomas et al. 2011). These findings suggest that there may be substantial variability in fatal liver SGAR concentrations among species, making it difficult to interpret results from most studies including those on Golden Eagles.

Similarly, the FGAR diphacinone had 20-fold higher toxicity for American Kestrels in comparison to Northern Bobwhites (*Colinus virginianus*; Rattner et al. 2010, 2011), illustrating that the traditional use of bobwhite quail in toxicity studies may be inappropriate for extrapolating to raptor species because of the differences in their sensitivity. Because of this variability, diagnosis of AR poisoning is typically based upon liver concentrations coupled with internal or external signs of excessive bleeding in live animals (and subsequent reversal of symptoms when treated with vitamin K) or evidence of hemorrhaging in tissues during necropsy of dead animals (Albert et al. 2010, Ebbert and Burek-Huntington 2010, Murray 2011). However, diagnosis is dependent on having access to live birds or recently dead carcasses (Albert et al. 2010, Ebbert and Burek-Huntington 2010, Murray 2011). None of the published studies on Golden Eagle AR exposure reported necropsy data to look for evidence of hemorrhaging.

Although there is limited information regarding Golden Eagle toxicity to ARs, two studies conducted in the 1970s dosed a limited number of Golden Eagles ($n = 8$) with either an SGAR (brodifacoum; Marsh and Howard 1978) or an FGAR (diphacinone; Savarie et al. 1979). No deaths occurred after 3 d of dosing with rats fed 0.005% brodifacoum bait, although two of the birds presented with external bleeding. The Golden Eagles dosed with meat containing 2.7 ppm diphacinone did not die but did exhibit behavioral effects (Savarie et al. 1979). Additionally, both external bleeding and increased coagulation times were observed in all birds. No data were reported on AR concentrations in the Golden Eagles for either study.

Chemical registrants and the U.S. EPA have developed both the lethal $dose_{50}$ (dose at which 50% of the test population is killed given a period of time; LD_{50}) and the lethal $concentration_{50}$ (concentration required to kill 50% of the test population; LC_{50}) estimates for a number of FGARs and SGARs. Using those estimates, the U.S. EPA developed descriptive categories to help put these wide-ranging toxicities into perspective (Table 2; Erickson and Urban 2004, U.S. EPA 2011). Although this U.S. EPA guidance is helpful in categorizing risk, there can be extreme differences in sensitivity, leading to widely differing LD_{50}s among species. For instance, the most sensitive LD_{50} for brodifacoum is 0.26 ppm in Mallard (*Anas platyrhynchos*), whereas the Ring-necked Pheasant (*Phasianus colchicus*) has an LD_{50} over 38 times higher at 10 ppm (Godfrey 1986, Erickson and Urban 2004). Table 3 lists the LD_{50}s and the descriptive toxicities (based on the U.S. EPA's Pesticide Assessment Guidelines) for seven ARs for the most sensitive birds (Erickson and Urban 2004, U.S. EPA 2007, Rattner et al. 2011).

These data illustrate that the SGARs brodifacoum and difethialone are extremely toxic to sensitive avian species like raptors, whereas SGARs broma-

Table 3. Most sensitive LD_{50} and descriptive toxicity for birds based on dosing studies derived from Erickson and Urban (2004), U.S. EPA (2007), and Rattner et al. (2011).

TYPE OF RODENTICIDE	RODENTICIDE	AVIAN LD_{50} (PPM)	DESCRIPTIVE TOXICITY
SGAR	Brodifacoum	0.26	Extremely toxic
	Bromadiolone	138.00	Moderately toxic
	Difenacoum	66.00	Moderately toxic
	Difethialone	0.26	Extremely toxic
FGAR	Chlorophacinone	>100.00	Moderately toxic
	Diphacinone	96.80	Moderately toxic
	Warfarin	620.00	Slightly toxic

diolone and difenacoum tend to be only moderately toxic. Similarly, FGARs appear to have low to moderate toxicity in sensitive species like raptors. In the case of the FGARs, the appropriateness of using LD_{50} estimates for FGARs has come into question (Ashton et al. 1986, Jackson and Ashton 1992, Vyas and Rattner 2012) primarily due to exposure scenarios. FGARs require multiple doses to reach lethality in pest species. Thus, the standardized single dose avian acute toxicity test is not a good model for FGARs or the time course for those effects to occur as they would in the field (Vyas and Rattner 2012). Accordingly, avian acute oral toxicity testing underestimates of the toxicity of FGARs. In the case of rats dosed with chlorophacinone or diphacinone, the traditional LD_{50} single dose might underestimate the toxicity by 21 or 41 times, respectively (Ashton et al. 1986, Jackson and Ashton 1992). A similar assessment has not been conducted on birds to date. Ultimately this could result in underestimating the risk of FGARs to raptor species such as Golden Eagles. Currently, there are no published accounts of Golden Eagle mortalities associated with FGARs, but there are published accounts of Bald Eagles dying from diphacinone poisoning (U.S.F.W.S. 2012).

Recent efforts using dose-response curves to estimate risk associated with exposure to the FGAR chlorophacinone found that birds are much more sensitive than previously believed (Rattner et al. 2015). Although LD_{50} values for sensitive species provide an understanding of the relative lethality of these individual ARs and non-ARs, they are the result of laboratory studies and do not reflect the actual conditions in which wild birds live. Additional stressors such as low prey availability and inclement weather can result in interactions in the wild that make it challenging to identify and interpret adverse effects (Rattner et al. 2014a).

There are considerable differences in the half-lives of FGARs and SGARs. The half-lives of the SGARs in livers derived from rat and mouse toxicity studies range from months to nearly a year, contrasting with those of the FGARs, which are on the order of days (Table 4). It should be noted that half-life estimates in liver tissue differ considerably among taxa, and

Table 4. Half-life (d) of a single dose of rodenticides in blood or liver of rats and mice derived from Morrow (2001), Erikson and Urban (2004), Fisher et al. (2003), U.S. EPA (2007), Spaulding and Spannring (1988), and Vandenbrouke et al. (2008).

TYPE OF RODENTICIDE	RODENTICIDE	DOSE (AI[a]) (PPM)	HALF-LIFE (D) IN BLOOD	HALF-LIFE (D) IN LIVER
SGAR	Brodifacoum	0.02–0.35	6.5–91.7	113.5–350
	Bromadiolone	0.2–3.0	1.0–2.4	170–318
	Difenacoum	1.2	NA[b]	118.0
	Difethialone	0.5	2.3	126.0
FGAR	Chlorophacinone	4.0–5.0	0.4	<2.0
	Diphacinone	0.32	NA	2.0–3.0
	Warfarin	1.0	0.7–1.2	7.0–26.2

[a] AI = active ingredient.
[b] NA = not available.

most estimates are developed from rats. Rattner et al. (2014b) reported that the half-life of diphacinone in Eastern Screech-Owl liver (*Megascops asio*) was 11.7 d, as compared to 2–3 d in rat livers. Regardless of taxa, SGARs remain in the liver tissue for much longer periods of time. Subsequently, there is an increased likelihood of bioaccumulation and secondary poisoning in raptors that consume rodents poisoned with SGARs.

Potential Impacts to Individuals or Populations

Lead and Physiology.

Upon ingestion, lead fragments are eroded within the acidic environment of the gastrointestinal tract (Redig and Arent 2008) and then absorbed into the blood through the small intestine (Fisher et al. 2006). For diurnal raptors such as Golden Eagles, digestion and absorption may occur rapidly because raptors have more caustic stomach acidity (pH 0.7–2.5) relative to other bird groups (Welty 1982, Duke 1986). Upon absorption, lead is bound to red blood cells and plasma proteins where it circulates through the body (Redig and Arent 2008). Following absorption and circulation, lead poisoning can result in a cascade of effects; detailed reviews on physiological effects in raptors can be found in Redig and Arent (2008). Typical external signs of chronic and acute exposure often include lethargy and anorexia, breast-muscle wasting, loss of strength and coordination, and sagging or drooping wings, and decreased mental faculties (Redig and Arent 2008).

In all taxa including birds, exposure to lead can result in impairment of the nervous system (ATSDR 2007, Sanders et al. 2009). Lead alters calcium homeostasis while obstructing cholinergic nerve cells, consequently inhibiting signal transmission across nerve synapses (ATSDR 2007, Sanders et al. 2009). One important consequence of lead impairment of the nervous system is reduced cognitive abilities. Experimental dosing studies with environmentally relevant lead acetate concentrations on wild and captive Herring Gull (*Larus argentatus*) nestlings revealed that lead impairment resulted in less vigorous food acquisition behaviors, poor coordination, and decreased ability to learn (Burger and Gochfeld 1994, 2005).

The erythropoietic system, which produces red blood cells, is associated with one of the most common physiological impairments of lead exposure in birds. Lead can impair the production of delta-aminolevulinic acid dehydratase (ALAD), which is a precursor for heme synthesis (Hoffman et al. 1981). Elevated lead concentrations resulting in decreased heme synthesis can cause anemia (Hoffman et al. 1985, ATSDR 2007, Redig and Arent 2008). However, studies on raptors illustrate that ALAD responses to lead exposure are strongly dependent on the lead dose. For instance, Red-tailed Hawks dosed with 1.5 mg lead/kg body mass/d for 24 d experienced an 83% reduction in ALAD activity during the 3-wk experiment in which blood lead concentrations reached a maximum of 0.75 ppm (Redig et al. 1991). In these studies, ALAD activity did not return to pre-dosing levels for 5 wk after cessation of lead dosing (Redig et al. 1991). Similarly, ALAD activity in Turkey Vultures rapidly declined when repeatedly dosed with 0.19, 0.57, or 1.92 g lead/kg body mass (maximal blood lead concentrations ranged from 1.87 to 29.56 ppm; Carpenter et al. 2003). These two examples illustrate a severe response when lead concentrations are elevated, yet at lower lead exposure the effects are often muted or even nonexistent. No relationship was found between lead and ALAD activity in free-flying Cooper's Hawks (*Accipiter cooperii*) when blood lead concentrations ranged from 0.03 to 0.09 ppm (McBride et al. 2004). To date, the only study evaluating lead and ALAD in Golden Eagles was in nestlings, but the sample size for ALAD was only five individuals at one site and too limited for statistical analyses (Stephens et al. 2008).

Anticoagulant Rodenticides and Physiology. Target rodents and nontarget predators and scavengers may exhibit substantial blood loss and fatal hemorrhaging after exposure to ARs (Rattner et al. 2011, 2012, 2014a). Although rodenticide poisoning is often associated with extreme effects (e.g., acute hemorrhaging), smaller microscopic hemorrhages can also result in ischemia, hypoxia, and cell death at critical organ sites (Rattner et al. 2011, 2012). Hemorrhaging can be spontaneous in rodenticide-poisoned animals, but it can also originate from a trauma and be potentially lethal to the animal because of their diminished blood coagulation capacity (Rattner et al. 2014a). For birds, the specifics of hemorrhaging have not been documented as comprehensively as they have for mammals (Rattner et al. 2014a).

A number of studies have documented AR poisoning in birds, and there is a wide range of observable effects (Berny et al. 1997, Stone et al. 2003, Murray 2011, Rattner et al. 2011, 2012). These include external bleeding from the cloaca, mouth,

and nares (Savarie et al. 1979), pale blue skin and mucous membranes, bruising, and blood in excreta (Redig and Arent 2008, Murray 2011, Rattner et al. 2014a). Even small scratches incurred during prey capture can be the site of lethal bleeding in raptors (Redig and Arent 2008). Numerous studies have also documented behavioral effects such as irregular posture and lethargy (Savarie et al. 1979, Redig and Arent 2008, Murray 2011, Rattner et al. 2011). Currently, broad differences in responses among and within species complicate ascription of one specific symptom attributed to rodenticide poisoning. Despite potential overt signs of SGAR poisoning, Redig and Arent (2008) found no signs of coagulopathy indicative of SGAR poisoning in 7426 raptors admitted for emergency care at the University of Minnesota Raptor Center. In contrast, two separate studies (Murray and Tseng 2008, Murray 2011) documented overt clinical signs of AR poisoning in four species of raptors presented to a wildlife clinic. Identifying birds that have been exposed to ARs is quite difficult, with most diagnoses of AR poisoning not confirmed until the bird is dead, a necropsy conducted, and liver tissues analyzed (Redig and Arent 2008, Albert at al. 2010, Murray 2011). Unless there are overt symptoms or other reasons to suspect AR poisoning, tissues from ailing Golden Eagles captured alive and brought to a rehabilitation center are unlikely to be evaluated for AR residues because of the expense.

Population Effects. Quantifying population-level effects due to contaminant exposure is exceedingly difficult. The only raptor species in North America for which population effects have been attributed to lead exposure is the California Condor (Cade 2007, Walters et al. 2010, Finkelstein et al. 2012). In other avian groups, such as waterfowl, annual population level losses due to direct lead poisoning from ingesting spent lead pellets were estimated to be 2–3% of the annual fall migration or in excess of a million birds (Bellrose 1959). In Golden Eagles, little is known about how lead exposure may influence populations at local or regional scales. Focused studies at smaller regional scales might help resolve the effects of lead exposure on reproduction and survival from the influence of other stressors such as low prey density, inclement weather, and habitat loss (Steenhof et al. 1997, McIntyre and Adams 1999, Kochert et al. 2002). Improving our understanding of which life stages (nestling or free-flying) may experience the greatest effects from lead exposure would also be beneficial for parameterizing estimates of population effects.

Like lead, ARs have not been associated with population reductions in predatory or scavenging raptors (Smith 1999, Rattner et al. 2014a). Using a probabilistic approach, Thomas et al. (2011) recently calculated a predicted mortality rate of 11% for Great Horned Owls exposed to SGARs across six Canadian provinces (BC, AB, SK, MB, ON, QC). Owl samples came largely from developed regions of Canada with elevated human population densities; thus, the predicted mortality rate of 11% is only applicable to these specific regions and is not reflective of SGAR exposure and toxicological risk at the province-wide scale.

Additionally, AR exposure may be additive with other sources of mortality (Brakes and Smith 2005). As a long-lived K-selected species, Golden Eagles could experience local population declines if AR poisoning resulted in the death of reproductive birds, exceeding the compensatory mortality threshold (Rattner et al. 2014a). Further, the likelihood of mortality could be exacerbated with the combination of lead, ARs, and other anthropogenic effects occurring simultaneously. Golden Eagles are long-lived (Kochert et al. 2002) and generally do not breed until their fourth to seventh year (Steenhof et al. 1984). One difficulty in assessing potential effects of ARs on a raptor population is the confounding effect the rodenticides may have on the prey base. Salim et al. (2014) recently studied the effects of both FGAR and SGARs on the breeding performance of Barn Owls in Malaysia. The authors found a negative relationship between concentrations of rodenticides and breeding performance. However, it is difficult to ascribe a cause for the owls' population decline without knowing the threshold at which local prey populations influence breeding success in the Barn Owls.

Although current data are limited, one valuable approach to understanding how lead and ARs might influence Golden Eagle populations may be through the use of adverse outcome pathway (AOP) models. Adverse outcome pathway models are an emerging ecotoxicology technique designed to provide a mechanistic representation of critical toxicological effects that can span different levels of biological organization (Ankley et al. 2010, Kramer et al. 2011). Adverse outcome pathway models can provide a structure for summarizing and organizing existing data, facilitating the identification of the key data gaps and research priorities, and can

contribute to the development of predictive models for linking early toxic exposure with more complex adverse effects (Ankley et al. 2010, Kramer et al. 2011). Notably, Rattner et al. (2014a) developed an AOP to describe AR effects for nontarget wildlife. Specifically, this AOP model for ARs identifies each of the established linkages: e.g., FGAR/SGAR→Macromolecular Interaction→Cellular Response→Multiple Organ Response→Organism Response→Population Response (Rattner et al. 2014a). The AOP model also identifies plausible and hypothetical linkages, as well as biomarkers for assessing AR exposure and diagnostic tools to verify if fatalities are related to ARs (Rattner at el. 2014a).

Synergistic Effects of Lead and ARs and the Likelihood of Collisions with Structures

Lead and AR exposure and accidental trauma are three common threats to raptors in the western United States (Smallwood and Thelander 2008, Stauber et al. 2010, Kelly et al. 2014). Evidence indicates that Golden Eagles in the western United States may be continually exposed to lead throughout the year (Watson and Davies 2015), and limited evidence suggests that eagles may also be regularly exposed to ARs (Kelly et al. 2014). Additionally, Golden Eagles suffer injury and mortality from collisions with motor vehicles, power lines, and wind turbines (Smallwood and Thelander 2008, Kelly et al. 2014). Turbine strikes are a growing concern as utility companies develop wind power in the west, because many landscape features suitable for producing wind energy are also in areas that Golden Eagles favor for nesting and foraging (Watson et al. 2014). Although contaminant exposure and physical traumas are two risk factors that independently threaten Golden Eagle mortality in the west, they may also act synergistically, resulting in a more pronounced effect than either threat independently. Because lead is a neurotoxicant, sublethal exposure could impair flight coordination or spatial recognition in Golden Eagles, increasing the susceptibility of eagles to accidental trauma and the likelihood of a fatal encounter with vehicles or structures. Mute Swans (*Cygnus olor*) with only moderate blood lead levels had an increased likelihood of collision with large fixed objects such as power lines (Kelly and Kelly 2005). Similarly, 32% of lead-exposed White-tailed Eagles (*Haliaeetus albicilla*) either died from collisions with objects or had wing injuries suggestive of collisions (Helander et al. 2009). In contrast, Franson and Russell (2014) found no evidence that lead exposure in Golden and Bald eagles (combined samples) predisposed them to other causes of mortality including trauma associated with collisions. Anticoagulant rodenticide exposure may result in collision fatalities by causing weakness and impaired flying ability (Savarie et al. 1979, Redig and Arent 2008, Murray 2011, Rattner et al. 2011). The sensorial capacity of AR-exposed rodents is reduced (Cox and Smith 1992, Vyas et al. 2012), and they become more prone to accidents. Although similar data are lacking for birds, at least one study of FGAR poisoning on Golden Eagles reported behavioral effects associated with diphacinone poisoning (Savarie et al. 1979). Similarly, diphacinone-poisoned Northern Bobwhites exhibited lethargic behaviors (Rattner et al. 2010). If both are present, then lead and SGARs may act in concert to further influence Golden Eagle mortality, such as in agricultural fields where rodents are managed with recreational shooting and rodenticides.

Data Gaps and Information Needs

To date, almost all Golden Eagle studies of lead and AR exposure and effects have focused exclusively on free-flying birds. Thus, even a basic understanding of lead or AR exposure and effects in Golden Eagle nestlings is lacking. Additionally, most of the data on adult lead exposure comes from studies of migrating eagles (Miller et al. 1998, Harmata and Restani 2013, Langner et al. 2015), and very few studies have focused on either adults or nestlings during the breeding season. These data gaps are critical in understanding the potential effects of both of these contaminants to Golden Eagles.

Data on Golden Eagle AR exposure in the wild are very limited; across six peer-reviewed studies, only 48 birds have been assessed for AR exposure. Nearly all that is known about potential AR exposure and effects comes from species other than Golden Eagles. Applying data from other species to understand AR exposure and effects requires the assumption that exposure levels in other species reflect what would be expected in Golden Eagles (or is scalable in some known way), and that toxicity thresholds determined in other species are appropriate for Golden Eagles. These assumptions may not be applicable because of the degree of variability in species-specific responses to ARs (Thomas et al. 2011, Rattner et al. 2010, 2011). Golden Eagles may be more likely exposed to FGARs in these areas if

their territories overlap with agricultural lands, although uncertainty remains as to whether exposure would be due to secondary or tertiary sources (Fig. 1). Exposure to FGARs is likely underdiagnosed in Golden Eagles due to the short half-lives of FGARs, and rapid degradation of organs in carcasses that are critical to diagnosing cause of death. Conservation and ethical concerns with conducting dosing studies in Golden Eagles may also preclude the ability to determine relative sensitivity to AR exposure. Developing an improved understanding of blood AR concentrations may enhance exposure detection or interpretation for markers of pathology, as long as they are correlated with the appropriate spatial or temporal frame.

Although we have identified potential sources and mechanisms for Golden Eagles to become exposed to lead and ARs on the landscape (Fig. 1), research addressing multiple stressors under field conditions will be critical for assessing the combined effects of lead and ARs to Golden Eagles in association with environmental factors. Establishing a link between either lead or ARs and demographic effects may be exceptionally difficult because it requires mortality to be assigned to specific factors, which can only be obtained from intensive studies. More intensive studies at smaller regional landscape scales may be effective at estimating the effects of lead or ARs on demographic parameters. Although not necessarily reflective of what the entire Golden Eagle population may be experiencing, these estimates could serve as a basis for understanding whether and how lead and ARs influence regional Golden Eagle populations. Additionally, these studies may help define more specifically how Golden Eagles are exposed to ARs (e.g., secondary or tertiary exposure and from which species of prey), which would be beneficial to management and conservation programs.

Specific information needs for ARs were highlighted by Rattner et al. (2014a), acknowledging that there are large data gaps in relation to exposure pathways, comparative species sensitivity, consequences of sublethal effects, hazards of increased exposure to poisoning due to higher survival of genetically resistant prey, effects of low-level exposure to multiple ARs, and quantitative data on the magnitude of nontarget wildlife mortality. For Golden Eagles, the focus for further study should include further elucidation of exposure pathways, sublethal effects, species sensitivity, and the effects of multiple rodenticide exposures. Direct comparisons between FGAR and SGAR toxicity to birds using environmentally appropriate concentrations would be particularly valuable (Rattner et al. 2015). Research addressing any of these knowledge gaps will improve understanding of the risk to Golden Eagles from AR poisoning, and where management actions might be focused to mitigate that risk.

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