Review: Diseases and ectoparasites of golden eagles in western North America

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In a summary of causes of mortalities in golden eagles (*Aquila chrysaetos*) analyzed at the National Wildlife Health Center between 1975 and 2013, Russell and Franson (2014) reported that diagnosed diseases were responsible for 2.7% (n = 39) of all causes of death. Diseases such as trichomonosis, West Nile virus, and aspergillosis have been reported in golden eagles and are reviewed below. In addition to disease, endo- and ecto-parasites can affect eagle health and sometimes lead to death. Here we review commonly reported diseases and parasites, and report on an expert review of 12 golden eagle researchers regarding disease and parasites that affect golden eagle nestlings.

Trichomonosis Trichomonosis is a disease caused by the protozoan *Trichomonas gallinae* [family Trichomonadidae]. *T. gallinae* lives as a parasite in the upper digestive tract of birds and occurs commonly in North America, Europe, Japan, and South Africa (Stone & Nye 1981). The worldwide distribution of *T. gallinae* is attributed to the spread of its primary host, the rock pigeon (*Columba livia*), and the protozoan now commonly occurs in several species of columbids (Stabler 1954). In pigeons and doves, transmission of *T. gallinae* occurs when infected adults slough the epithelial cells in their crop to produce "crop milk" for young birds, or through the sharing of infected watering or feeding areas. Hawks, eagles, falcons, and owls that feed on columbids infected with *T. gallinae* can develop trichomonosis (sometimes termed "frounce" in raptors) (Stabler 1947; Krone et al. 2005).

Fig. 1. Caseous ("cheese-like") plaques of trichomoniasis (in white circle) in the mouth of a golden eagle nestling from SW Idaho.

The pathology of *T. gallinae* varies from mild inflammation of the mucosa to large, caseous lesions, or plaques, within the esophagus (Stabler 1954, Figure 1). Plaques typically begin to develop 5-6 days post infection and they can grow large enough to completely block the esophageal lumen, leading to decreased feeding and, eventually starvation or suffocation (Amin et al. 2014). The severity of the disease depends on the strain of *T. gallinae* and the susceptibility of the infected bird (Cooper & Petty 1988). Girard et al. (2014) found that during non-epidemic periods, band-tailed pigeons (*Patagioenas fasciata monilis*) were infected with a different strain of *T. gallinae* than during epidemic outbreaks that killed hundreds of birds. When trichomonosis is diagnosed early and the plaques are relatively small, it can be successfully treated with a single dose of Spartrix (generic carnidazole), an antiprotozoal drug effective at reducing the development of oral lesions. Unfortunately, widespread dosing of watering or feeding areas with anti-protozoans has led to resistant strains of *T. gallinae* and harmful effects in non-target bird species (Amin et al. 2014).

Trichomonosis was recently recognized as an emerging infectious disease when it

caused the death and decline of two common songbirds in the United Kingdom, the greenfinch (*Carduelis chloris*) and chaffinch (*Fringilla coelebs*) (Robinson et al. 2010). Within 2 years of detection of *T. gallinae* in wild birds, more than half a million birds died resulting in a 35% decline in greenfinches and a 21% decline in chaffinchs. Since then, *T. gallinae* infection of songbirds has spread with migration patterns (Lawson et al. 2011) and several outbreaks were recorded in northern Europe (Lehikoinen et al. 2013). In addition, the number of species infected with *T. gallinae* has increased, including other Passeriformes, owls, and cuckoos (Park 2011). A recent study from Northern California documented *T. gallinae* in house finches (*Carpodacus mexicanus*) and corvids and a novel trichomonad in northern mockingbirds (*Mimus polyglottos*) (Anderson et al. 2009). *T. gallinae* is a major concern for vulnerable species with small population sizes, such as pink pigeons (*Nesoenas mayeri*) (Bunbury et al. 2007) and Pacific Coast band-tailed pigeons (Girard et al. 2014).

T. gallinae infection has been noted in golden eagles historically, particularly during the nestling stage. A single nestling fatality, attributed to trichomonosis, was reported by Boeker & Ray (1971) in the Front Range of the Rocky Mountains. In the early 1970s, Beecham & Kochert (1975) and Kochert (1972) reported that at least 4 of 129 nestling (3%) eagles died from trichomonosis in Idaho's Morley Nelson Birds of Prey National Conservation Area (NCA). In 2014, 3 of 19 nestlings (16%) in the NCA showed physical signs of trichomonosis (Heath unpub. data). Two of the nestlings were treated with 30 mg of Spartrix and recovered from the infection; the third was treated too late (the nestling had developed large plaques) and died. Siblings of infected birds were treated with a preventative dose and did not show signs of the disease. In 2015, Dudek et al. (*in prep A*) collected samples from 32 eagle nestlings from 19 nests in the NCA and adjacent Upstream Comparison Area (UCA). *Trichomonas gallinae* was detected in 41% (n = 13) of nestlings, and 42% of nests (n = 8) had at least one nestling that developed infection. The mean nestling age, for which *T. gallinae* infection was detected in culture from oral swabs, was 23.5 ± 11.0 days (range 8 – 38 days). The mean age of detection of oral lesions was 30.3 ± 13.5 days (range 12 – 49 days). The development of oral lesions were observed 7.2 ± 7.0 days after detecting presence of *T. gallinae* in culture from oral swabs. Twelve of 13 (92%) nestlings that tested positive for *T. gallinae* in culture subsequently developed oral lesions suggestive of *T. gallinae* infection. Nestlings that developed oral lesions indicative of trichomonosis were treated with a 30 mg dose of Spartrix (Janssen, Brussels, Belgium) after oral sampling. In all cases in which oral lesions were observed and antiprotozoal medicine was administered, the treatment resulted in the disappearance of oral lesions within 8 – 10 days. Moreover, *T. gallinae* was not detected in cultured swabs taken on the subsequent visit. Re-infection was observed in three cases, both in culture and through the presence of oral lesions within 16, 24 and 25 days of initial treatment, respectively. All three birds were successfully treated a second time. Two nestlings that tested positive for *T. gallinae* in culture developed small oral lesions just prior to fledging and were left untreated. One nestling presumably fledged successfully, whereas the other was found dead in the nest after its sibling had fledged. Decomposition was too advanced to determine whether oral lesions contributed to this individual's death.

In 2014 and 2015, we conducted phone interviews of 12 researchers that had worked with golden eagles for more than 8 years. Two of the 12 researchers that were interviewed recalled cases of trichomonosis. A clinically diagnosed case of trichomonosis occurred in 2014 in Oregon at Smith Rock State Park resulting in a nestling mortality (J. Buck pers. comm.). Another apparent case of trichomonosis in an eagle nestling was reported from the Four Corners region (D.W. Stahlecker pers. comm.). The nestling was administered

treatment, but fate of the bird was uncertain. Approximately half of the eagle researchers have noted that eagles feed on columbids, at least occasionally. Most researchers have never noted a case of trichomonosis, but some mentioned they have not specifically been looking for signs of the disease. Nor does the disease always produce clinical symptoms of infection. Culturing or wet-mount microscopy must be performed to accurately assess *T. gallinae* prevalence. Symptomatic trichomonosis can be confused with aspergillosis, avian pox, Vitamin A deficiency, or nematode infection (Kocan & Herman 1970). In 2015, Dudek et al. (*in prep A*) collected oral swab samples from 96 eagle nestlings ranging in age from 21 days old to 59 days old, from 62 nests, across 10 western states outside of Idaho. Prevalence of *T. gallinae* in non-Idaho eagles was 6.2% (n = 6) of nestlings and 9.7% (n = 6) of nests had at least one nestling that developed infection. Positive samples came from Kern and Siskiyou counties in California, Crook and Lake counties in Oregon, and Tooele County, Utah.

T. gallinae infection has been reported in several other eagle species, and is a significant mortality factor in at least one sensitive species (Real et al. 2000). Trichomonosis has been reported in bald eagles (*Haliaeetus leucocephalus*) on several occasions, over time and in different locations. The first case was reported in an adult bald eagle in 1977 in New York (Rettig 1978). A second case was reported in New York in 1978 in an immature bald eagle (Stone & Nye 1981). Another case of trichomonosis was seen in an adult bald eagle in Montana years later (Bates et al. 1999). After months in a rehabilitation facility, an asymptomatic hatch-year bald eagle on Prince Edward Island, Canada cultured for *T. gallinae* in 2011 (Kelley-Clark et al. 2013).

Unfortunately, little is known about the factors that affect eagle infection with *T. gallinae* or susceptibility to trichomonosis. Beecham & Kochert (1975) and Kochert (1972) speculated that eagles were exposed to *T. gallinae* from eating rock pigeons. In the cases reported in 2014 in Idaho, rock pigeons were the primary prey item for two of the nest sites and at the third site rock pigeons were second most common item after ground squirrels (Heath unpub data). At 9 other sites where trichomonosis was not detected in Idaho, rock pigeons were infrequent or absent from the pellet and prey remains samples. In another study, mourning doves (*Zenaida macroura*) were prey items of a nestling that died from trichomonosis (Boeker & Ray 1971). These results support the hypothesis that foraging on columbids significantly increases risk of developing trichomonosis. During the 2015 breeding season in southern Idaho, the proportion of rock pigeons in nestling diet predicted *T. gallinae* infection. As the proportion of rock pigeons in the diet increased, so did the probability of developing *T. gallinae* infection; chance of infection approached 100% when rock pigeons accounted for at least 10% of nestling diet (Dudek et al. *in prep A*).

The relationship between consumption of columbids and infection with *T. gallinae* is consistent with research on Cooper's hawks (*Accipiter cooperii*) in Arizona. In the urban areas of Tucson, where columbids make up 83% of Cooper's hawk diets, 85% of nestlings have *T. gallinae*; whereas in rural areas, where columbids are only 10% of the diet, 9% of the nestlings have *T. gallinae* (Boal 1997; Boal et al. 1998). A similar pattern was observed for goshawk nestlings (*Accipiter gentilis*) close to urban areas in Poland (Wieliczko et al. 2003) and Germany (Krone et al. 2005). A change in land use or habitat that results in diet shifts towards columbids can increase exposure to *T. gallinae*, and may increase risk of infection.

Western European Bonelli's eagles (*Aquila fasciatus*) are a vulnerable species that regularly feed on rock pigeons, especially in the absence of rabbits or partridges (Palma et al. 2006). Trichomonosis has been identified as the most important factor affecting nestling survival, with 22% of nestling mortality attributed to *T. gallinae* (Real et al. 2000). In northeast Spain, the proportion of rock pigeons in the diet was positively associated with the

probability of infection with the parasite (Real et al. 2000).

Like Bonelli's eagles, declines in preferred prey species (e.g., black-tailed jackrabbits in the Great Basin) may lead to increase predation on rock pigeons and exposure to *T. gallinae* in golden eagles. Further, land use change and agricultural practices that provide large, common feeding areas for rock pigeons may facilitate transmission of *T. gallinae* among rock doves. In addition, other drivers of global change, such as changes in climate may increase outbreaks. Warmer temperatures and lower rainfall has been associated with the emergence of the disease in finches in the UK (Simpson & Molenaar 2006) and the prevalence of *T. gallinae* infection in doves in Mauritius (Bunbury et al. 2007). Therefore, warmer temperatures and reduced precipitation, both of which are possible outcomes of future climate change, could increase *T. gallinae* viability, thereby leading to higher prevalence in columbid species (Rogers et al. 2016).

The research summarized here indicates that *T. gallinae* may become a disease with population-level consequences in coming years, in a wider variety of avian species then previously seen. It is important to note that increased vigilance and surveillance likely accounts for some of this pattern, as it was uncommon to clinically test for, or even visually note, *T. gallinae* symptoms before the last decade. *T. gallinae* also varies greatly in virulence and its associated symptoms (Amin et al. 2014), confusing the issue of whether risk of mortality from *T. gallinae* has changed over time.

West Nile Virus. West Nile is a virus in the family Flaviviridae (genus *Flavivirus*). This virus is most commonly transmitted by feeding mosquitoes and, like other Flavivirus, West Nile Virus (WNV) can quickly replicate and cause infection once established inside a host cell (Center for Disease Control and Prevention 2016). WNV can persist in many hosts including mammals and reptiles, but birds are the most commonly affected host group (Hayes et al. 2005).

WNV was first isolated in the West Nile District of Uganda in 1937 (Smithburn et al. 1940), but has since spread around the world. A large scale outbreak of WNV in 1999 occurred in New York City, marking the first introduction in recent history of the Old World Flavivirus into the New World (Petersen and Roehrig 2001). During this outbreak, the severe impact of WNV on birds was brought to light. Following the outbreak, tens of thousands of birds have been infected, representing over 300 different species (Center for Disease Control and Prevention 2016).

Corvids have been shown to be one of the most susceptible families, but infections have also been found in many other passerine families, as well as in raptors (Nemeth et al. 2006). Although relatively few documented cases of infected golden eagles exist, infection has been shown to lead to encephalitis resulting in a variety of abnormal behavioral activities and neurological failure. Lesions on the brain, heart, and eyes were common symptoms among a group of bald and golden eagles from across the U.S. that died from WNV infections (Wünschmann et al. 2014). Other, less severe, symptoms in birds include anorexia, weight loss, pinching off of blood feathers, unawareness of surroundings, weakness in legs, and seizures (Jones 2006, Marra et al. 2004).

Current research on WNV as it pertains to eagles, especially wild individuals, is relatively sparse and generally is limited to localized infection accounts. International findings include detections of WNV in golden eagles in Austria (Wodak et al. 2011), as well as golden and the endangered imperial eagle in Spain (Jiménez Clavero et al. 2008). In one of the only known WNV prevalence studies for any eagle species, 80% of Spanish imperial eagles (*Aquila adalberti*) sampled had detectable levels of WNV (Höfle et al. 2008). In the

United States, intensive WNV monitoring took place across the country after the 1999 outbreak and over the course of the first 5 years a total of 26 golden eagles were reportedly found dead due to WNV infection (Nemeth et al. 2006). Specific accounts include several captive birds along with records from state wildlife agencies and rehabilitation centers. The virus first hit the Rocky Mountain region in 2002, and by 2003 large numbers of raptors were being brought to rehabilitation centers in Colorado, Wyoming, Nebraska, and Idaho (McLean 2006, Nemeth et al. 2007). The Idaho Department of Fish and Game's Wildlife Health Lab also confirmed 5 golden eagle deaths from WNV between 2004 and 2008 (M. Drew pers. comm.). A golden eagle tested positive for WNV in Utah and was being treated at the Best Friends Animal Society in Kanab, UT in August of 2016, making it one of the most recent known cases in the West (C. Smith pers. comm.). Although accounts such as these help to determine the range and spread of the virus, the impact at the population level is difficult to assess, and at this time it appears that the prevalence rate of WNV in golden eagles is unknown. However, a 2005 study of Swainson's hawks (*Buteo swainsoni*) in Idaho showed a significant decline in the population due to WNV, and while golden eagles were not studied with the same intensity, the trends in Swainson's hawk numbers may have been mirrored by eagles given that eagles were found to be more susceptible to fatal infection than most other raptors in the region (M. Drew pers. comm.). Indeed, data from breeding surveys in southwestern Idaho indicate that golden eagles experienced declines in both the number of occupied territories and the number of young fledged in the 4 years following the 2005 outbreak (U.S. Geological Survey, Unpublished Data).

With all the unknowns surrounding this virus, it is difficult to determine its potential future impacts. However, some concern surrounds the fact that birds are not only common hosts of WNV, but may also play a role in the geographical spread of the virus. In migratory species, birds that are infected but have low levels of viremia may be capable of transmitting the virus between breeding and wintering areas (Rappole et al. 2000). With both migratory and resident golden eagle populations across the western U.S., it is therefore possible that localized outbreaks could be spread during migrations. It is also possible that populations which were affected by the outbreak in the early 2000's have built up antibodies that increase tolerance of the virus and reduce the severity of individual infections, allowing the virus to persist in the system with minimal impacts (M. Drew pers. comm.). However, these antibodies typically only last for a single year which could leave birds highly susceptible to another mass die-off if the current low intensity occurrence of WNV changes (M. Drew pers. comm.).

Aspergillosis. *Aspergillus spp* are fungal organisms that thrive naturally in soil organic layers, but are commonly found in a variety of areas with poor ventilation. The microscopic fungal spores (conidia) are released into the atmosphere through physical disturbance and are light enough to stay airborne for an extended period of time, increasing the chances that these conidia will be inhaled by animals (Latgé 1999).

Inhalation of *A. fumigatus* is the most common form of infection, and can lead to the disease aspergillosis (Latgé 1999). In birds, aspergillosis is most commonly contracted in captive settings where unchanged bedding material, old fecal matter, moldy feed, and poor air ventilation all can contribute to a thriving *Aspergillus* population (Beernaert et al. 2010). Plaques or granulomas in the nasal sinuses, trachea, syrinx, bronchi, lungs, and air sacs are common symptoms of aspergillosis in birds, and in advanced cases necrosis and hemorrhaging of the lungs can occur (Joseph 2000). Three stages of aspergillosis are known

to exist in birds. The acute stage can be fatal within a week of infection, whereas a chronic condition can take months to develop (Joseph 2000).

Many raptors are considered high risk species for contraction of aspergillosis; these include gyrfalcons (*Falco rusticolus*), northern goshawks (*Accipter gentilis*), red-tailed hawks (*Buteo jamaicensis*), and golden eagles (Joseph 2000, Redig et al. 1980, Russell and Franson 2014). Although raptors have been shown to be highly susceptible, very few cases of aspergillosis in wild birds have been documented. Only 15 of 1,427 golden eagles brought to the National Wildlife Health Center for necropsy were diagnosed with aspergillosis (Russell and Franson 2014). In a study of free-ranging raptors of Northern California, 409 individual birds representing 25 species were necropsied and 7.5% (n = 31) were determined to have died from aspergillosis (Morishita et al. 1998).

It is not likely that major landscape changes due to climate shifts or increases in anthropogenic activity will have a significant influence on aspergillosis prevalence in wild golden eagles or other raptors. There have been human-induced cases of large die-offs in waterfowl near major farm lands where moldy feed produced an abundance of *Aspergillus spp* conidia in the surrounding area (Bowes 1990). However, the level of exposure to these anthropogenic conditions is relatively low for raptors compared to waterfowl, and other landscape changes such as installation of wind turbines, power poles, or transmission lines do not create conditions that are conducive to the growth of *Aspergillus spp* (Leishangthem et al. 2015). As other threats to golden eagles do increase, and with them the potential to have more eagles placed in captive rehabilitation facilities, it is crucial to keep aviaries clean and well ventilated to reduce the chance of *Aspergillus spp* growth and infection in captive birds (Joseph 2000).

Blood Parasites. Avian blood parasites impact a large variety of species and have varying levels of pathological effect in their hosts (Remple 2004). Hundreds of species of parasites have been documented in birds around the world, but in raptors *Haemoproteus spp* and *Leucocytozoon spp* (Haemoproteidae family) are the most commonly documented (e.g., Remple 2004, Smith et al. 1998, Ziman et al. 2004). These parasites can attack red blood cells alone (*Haemoproteus*) or both red and white blood cells (*Leucocytozoon*), depending on the species (Remple 2004). Vectors of *Haemoproteus* and *Leucocytozoon* are thought to be common biting insects including louse flies, biting midges, and black flies, making these parasites easily transmissible around the world (Remple 2004).

Generally, Haemoproteidae species are fairly innocuous in raptors, and can often persist in a host without any noticeable symptoms (Remple 2004). In fact, the prevalence of parasites in many species is known to be extremely high (>90%), and yet there are often little to no measurable impacts at the population level (e.g., Hanel et al. 2016, Ishak et al. 2010, Jeffries et al. 2015). However, infected birds may be more susceptible to other diseases and illnesses due to a compromised immune system and may even experience increasing parasitemia when suffering from other ailments (Remple 2004). Indeed, studies of captive raptors show that added stress may be a contributing factor to increased infection within a population and increased parasitemia in an individual, leading to more severe symptoms and increases in mortality rates (Ziman et al. 2004).

There is a growing number of described Haemoproteidae species, and the degree of host-specificity is extremely variable among different taxa (Murdock et al. 2015). Some studies have suggested that infection status was not related to age, or sex of the host species (Ishak et al. 2010, Matthews et al. 2016), yet specific accounts of parasite related nestling mortality exist for red-tailed hawks (Smith et al. 1998), and great horned owls (Hunter et al.

1997). Nestlings may be more susceptible to mortality from blood parasites when infection occurs simultaneously with unusually high black fly infestations of raptor nests, resulting in a combination attack of parasite infection along with physical harassment including direct loss of blood (Hunter et al. 1997, Smith et al. 1998). Similar to the impacts of ectoparasites, this harassment can lead to fatal "premature" fledging (see 'Ectoparasites' section).

Few hematological studies have been published on eagles, and there are currently no known publications on blood parasitism in golden eagles. *Leucocytozoon spp* have, however, been observed in bald eagles in Michigan and Minnesota (Stuht et al. 1999), and both *Haemoproteus* and *Leucocytozoon spp* were found in nestlings of two eagle species (imperial eagles, and white-tailed sea eagles) studied in Kazakhstan (Leppert et al. 2004). Unpublished accounts of golden eagle infections do exist in the western U.S. For example, in 2016 a rescued fledgling eagle died at a rehabilitation center in Wyoming after being diagnosed with high levels of *Leucocytozoon spp* (S. Ahalt pers. comm.). Current research on golden eagles from Idaho, Oregon, and California aims to test for blood parasites, yet no conclusive results have been reported (C. Downs, unpublished data). Further studies on golden eagles are needed to assess prevalence of blood parasites at the population level and to isolate the impact that these parasites may have on individual birds.

As with many vector-transmitted diseases, there is concern that a changing climate could increase the spread of these already widely distributed avian blood-parasites (Gage et al. 2008, Lafferty 2009). Complex interactions between shifting landscapes, parasite vectors, and host species have been modeled to show that events such as deforestation can result in new parasite emergence or increasing severity of avian infection (Sehgal 2010). Longer and hotter summers may also result in increased heat stress on nestlings, which in turn could result in a suppressed immune system making already infected young birds more vulnerable (Møller et al. 2013).

Ectoparasites Other potential mortality factors that may increase in prevalence with changes in climate may be the incidence and negative effects of ectoparasites in golden eagles. Hematophagous parasites have been documented to decrease nestling fitness in many avian species, and in some cases, cause mortality (McFadzen & Marzluff 1996). Multiple golden eagle collaborators noted at least some presence of ectoparasites while climbing nests and handling nestlings. A few noted particularly heavy loads of nest parasites, particularly hematophagous parasites like cimicids and ticks. Repeated exposure to bedbugs (Cimicidae family) causes inflammation and irritation to the host's skin. In particular, bedbugs tend to feed around the host's eyes and face (Philips 2007). For nestlings that are unable to fly, repeated biting from ectoparasites can lead to chronic stress and escape movements that could be fatal. "Premature" fledging associated with exposure to high ectoparasite loads has been reported for purple martins (*Progne subis*, Loye & Zuk 1991), cliff swallows (*Petrochelidon pyrrhonota*, Brown et al. 1995), California condors (*Gymnogyps californianus*, Brown & Amadon (1968), and prairie falcons (McFadzen & Marzluff 1996). Half of the eagle researchers we interviewed have documented premature fledgling at nests with high ectoparasite loads. Most often these cases resulted in death, but some fledglings successfully fledged from the nest in poor condition. Additionally, nestlings have been observed branching early or have been captured on the ground under nests with high parasite loads. Parasite-induced anemia (from ticks and/or bedbugs), has been reported as the suspected cause the death for nestling bald eagles (Grubb et al. 1986) and cactus ferruginous pygmy owls (*Glaucidium brasilianum cactorum*, Proudfoot et al. 2005).

In 2014, high ectoparasite loads were noted in 3 golden eagle nests in the NCA: one where a nestling died in the nest, another where the nestling left the nest at night and died, and another that successfully fledged in extremely poor condition (i.e., low body mass, Heath unpub data). At the Owyhee Mountains study site in southwestern Idaho, young from at least 4 nests "prematurely" fledged, and post-failure nest climbs revealed high ectoparasite load. Other anecdotal reports from Montana suggest that young eagles may jump from nests at an early age in response to ectoparasites (Anderson pers comm). In the golden eagle *Birds of North America* species account, Kochert et al. (2002) report on several species of ectoparasitic arthropods that occur in eagle nests including: "2 species of ticks (*Ornithodoros concanensis* and *Haemaphysalis leporispalustris*; Hickman 1968, Knight & Marr 1983), 3 species of cimicids (Mexican chicken bugs [*Haematosiphon inodorus*]; Lee 1954, McFadzen et al. 1996; human bed bugs [*Cimex lectularius*]; and cliff swallow bugs [*Oeciacus vicarius*]; Hickman 1968), and a biting midge (*Leptoconops herteszi*; Hickman 1968)." Mexican chicken bugs have been reported in raptor nests in the American southwest (Grubb et al. 1986, D. Driscoll, pers. comm.), but only recently described in Idaho (McFadzen et al. 1996). McFadzen and Marzluff (1996) found that young prairie falcons (*Falco mexicanus*) in nests with "moderate to high load" infestations of Mexican chicken bugs had lower hematocrit and lower weights compared to nestlings from nests with "none to low" parasite load. Grubb et al. (1986) reported concentrations of 0.2 Mexican chicken bugs/sq. cm were found in two successful golden eagle nests within 1 week after fledging. An updated assessment on the diversity of ectoparasites and the factors associated with ectoparasite infestation would yield useful information for understanding changes over time and costs of ectoparasites.

In 2016, Dudek (2017) completed a survey of arthropods in golden eagle nests in the NCA and UCA. Using pitfall traps in nests, insects from ten families representing six orders were collected, as well as spiders, ticks, and a scorpion, were collected from the pitfall traps placed within the 16 golden eagle nests. Most of the arthropods collected were either larval or adult scavenging Coleopterans, including dermestids, histerids, clerids, and staphylinids, that feed primarily on decaying prey remains and detritus in the nest. Other common insects included leaf beetles, plume moths, ants, and grasshoppers. The two most common ectoparasites found in eagle nests were blowflies (Diptera: Calliphoridae) and Mexican chicken bugs. Myiasis, caused by blowflies, was observed typically within the first 5 weeks in the nestling period, at which point larvae exited nestlings, pupated, and emerged as adult flies. Dudek (2017) observed myiasis in 9 of 26 (35%) nestlings from 7 of 16 (44%) nests.

Mexican chicken bugs were detected in 14 of 16 eagle nests while nestlings were present, and in one additional nest after the nestlings fledged. Although median date of first chicken bug detection in the nest was 12 May, dates of first detection ranged substantially (21 April – 22 May). Dudek (2017) collected 2,712 bugs from pitfall traps in nests, 85.8% (n = 2,327) of which were nymphs. Relative abundance varied by nest, and ranged from 0.03 - 13.5 bugs $trap^{-1}$ $night^{-1}$. These values were consistent with categorical infestation levels assigned during each visit (either no bugs, low infestation, or high infestation). Mexican chicken bug abundance was best predicted by nest aspect, nest phenology, and previous nest use. Higher abundance was found in south-facing nests compared to north-facing nests and nests with a median hatch date later in the season had higher *H. inodorus* abundance than nests with earlier hatch dates. Nests reused within the previous three years were less infested than nests that had not been used in the previous years, which was opposite of the prediction that recent nest use would lead to increased bug abundance. These results suggest that eagles may reuse nests with lower ectoparasite abundance, potentially to avoid selecting nests with high ectoparasite loads (Dudek 2017).

The physiological effects of Mexican chicken bugs on 57 eagle nestlings from 35 nests during the 2015 and 2016 breeding seasons were measured by Dudek et al. (*in prep B*). Increased ectoparasitism reduced nestling mass and hematocrit, both of which suggest that ectoparasitism created an energetically expensive cost to nestling development. Relative ectoparasite infestation levels predicted circulating concentrations of corticosterone in eagle nestlings, and heavily parasitized nestlings had higher corticosterone levels compared to non-parasitized nestlings. Increased corticosterone can result in a range of deleterious effects, including reduced immune function (Wingfield et al. 1997), reductions in cognitive capabilities, and increased locomotor activities which can facilitate nest departure (Heath 1997). In addition, increased ectoparasitism also increased the probability that nestlings either fledged early or died in the nest. In total, the death of 18% (n = 10) of all nestlings in the study occurred in, or below, highly infested nests and can likely be attributed to Mexican chicken bug parasitism (Dudek et al. in prep B).

Although the presence of aromatic green plant material to nests had no effect on the abundance of Mexican chicken bugs, higher proportions of aromatic plants at nests had a positive effect on nestling hematocrit (Dudek 2017). These results suggest that the addition of aromatic plants in nests by golden eagles may not directly reduce ectoparasites in nests, but may instead disrupt the cues these ectoparasites use to locate hosts, inhibit feeding by ectoparasites, or delay reproduction or development of the ectoparasites (Clark and Mason 1988). Additionally, Mennerat et al. (2009) suggested aromatic plants might stimulate host immune systems to the physiological benefit of eagle nestlings. Developing young birds face a trade-off between growth and immune function (Brommer 2004), therefore mechanisms that improve immune function could improve nestling growth rates and hematocrit.

The long-term consequences of these physiological costs on survival (after birds leave the nest) remain unknown. More work is necessary to understand the prevalence and risk factors for disease and ectoparasites and whether there is evidence that changes in risk factors may lead to population-level impacts.

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