Assessment of Foot and Leg Abnormalities in the Burrowing Owl at Naval Air Station Lemoore

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Executive Summary

The occurrence of the morphological abnormalities at NAS Lemoore (NASL) is consistent with multiple causes, including injuries, infection that may have set in following an injury, viral infection, and mites. Injury, disease, or injury followed by secondary infection, as well as infestations of parasitic mites, appears to be the most plausible explanation for the observed abnormalities at NASL. All of these causes can result in similarly appearing abnormalities, and require laboratory testing for conclusive diagnosis. Based on a review of toxicological studies, the abnormalities of the feet and legs of burrowing owls noted at NASL are not likely a direct result of exposure to toxic compounds such as agricultural pesticides. Immunosuppression has often been observed following contaminant exposure, and therefore it is possible that there is an indirect link between contaminants and the abnormalities noted at NASL. However, similar abnormalities have been found on burrowing owls in areas with limited or no agricultural influence. Most of the potential causes of the observed abnormalities at NASL are most likely of natural origin, and we do not recommend changes in management, nor treatment of individuals based on our current state of knowledge. It would be desirable to determine if avian pox virus is the causative agent in the abnormal talons and external growths. This virus has not yet been recorded in burrowing owls, although it has been found in other raptor species. The demographic work conducted during 1996-2001 (Rosenberg et al. 2007) demonstrated that owls at NASL have similar or higher survival and reproduction than in other areas in California, suggesting a reasonably healthy population.

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Introduction

Burrowing owls with foot, talon, and leg abnormalities were observed at Naval Air Station Lemoore (NASL) in the spring of 2006 during research on migration patterns in burrowing owls. These findings motivated the Environmental Management Division at NASL and Naval Facilites Engineering Command Southwest to request assistance in evaluating the possible causes for the abnormalities and identifying appropriate research and management actions.

In this report, we synthesize existing information on the occurrence of morphological abnormalities in burrowing owls at NASL and elsewhere. We reviewed data records compiled during several years of demographic studies in California, and in particular at NASL, made inquiries to other burrowing owl researchers in the western United States, and reviewed the scientific literature for information on possible causes of abnormalities, including agricultural pesticides. We then used these data to evaluate the need for further research or management action on morphological abnormalities in burrowing owls at NASL.

Observations of Injuries and Deformities of Burrowing Owls

Morphological structure and incidence of talon and leg deformities at NAS Lemoore

Studies conducted from 1996-2001

Rosenberg et al. (2007) conducted studies of the burrowing owl at NASL from 1996 to 2001 to investigate their demography, space use patterns, and risk of exposure to agricultural pesticides. During this study, a total of 542 burrowing owls were captured, examined, and marked at NASL. Of the 542 owls, 370 were juveniles and 172 were first captured as adults. Owls were observed and captured at nests throughout the station, with most individuals nesting near the airfields (Figure 1). We reviewed all of the original data forms from the captures and recaptures of the 542 burrowing owls. There were no deformities noted; however, leg and foot injuries and swollen talon pads were noted in 7 cases out of the 542 individuals captured during the study, resulting in a 1.3% abnormality rate. In one of these 7 cases, a foot was slightly injured during trapping with metal box traps. Swollen talon pads were noted in 3 of the 7 cases, and in the remaining 3 cases, we observed leg injuries, including scabbing.

Studies conducted from 2006-2007

As part of a study on migration patterns of burrowing owls in western North America, Conway and his colleagues captured burrowing owls at NASL during 2006 (Figure 2) when the talon and foot abnormalities were noted at NASL. Field investigations continued in 2007. They captured a total of 71 juveniles and 32 adults in 2006, and noted no juveniles with abnormalities, but 7 adults from 6 nests were captured with growths or deposits on the feet and legs (Figure B-1). The observer noted that the abnormalities were not extreme; feet were most affected. One earlier email to the Navy from the 2006 research team referred to an owl with a missing talon. This was later corrected with a note explaining that this owl was captured at Edwards Air Force

Base (see below). In 2007, Conway and colleagues captured 8 juveniles and 26 adults, and noted 7 owls from 5 nests with some abnormalities or injuries to the foot, talon, and/or leg. These 5 nests were located throughout the area searched, predominately near the airfields (Figure 3). One of the abnormalities was a thickened toe pad. In one case the foot was swollen such that the owl could not close it completely (Figure B-2). In another case, one of the talons of an owl was irregular in shape and embedded in the toe pad. There have not been any findings of missing talons. However, scabbing and flakiness of feet and legs (Figures B-3 and B-4) were commonly observed, and growths on the foot (e.g., Fig. B-1), were seen sufficiently frequently (about 7% of owls) that the researchers thought these observations were much more common at NASL than at other sites where they had captured burrowing owls.

Review of Talon Abnormalities in Burrowing Owls Outside of Naval Air Station Lemoore

To review the type and incidence of talon and leg deformities in burrowing owls, we requested information from researchers studying burrowing owls. We contacted various burrowing owl researchers directly and/or through an email list serve (BURROWINGOWL@LISTSERV.UNL.EDU). Appendix A is a copy of the written request. We received several responses to these requests, which we summarize below.

California

Jack Barclay, San Jose Airport

Two owls were reported with abnormalities from a total of 802 captures of burrowing owls in northern California from 1992-2006. One of these was a 3-week old nestling with no right foot. This owl had a well-developed callus at the end of the tarsus. No photo taken. The observer suspected an injury, not a malformation. There was a smooth and uniform callus, as if a wound had been cauterized.

The other abnormality was of a recapture 1 year after the initial capture. No notes were taken about a deformity at time of first capture, suggesting a later injury. On the recapture 1 year later, the owl was apparently in poor condition and had a deformed beak.

Byron Buckley, Edwards Air Force Base

A juvenile was captured in 2006 that was missing a talon, which appeared as an undeveloped talon rather than an injury (Figure C-3). This was not observed in the siblings. The report of the missing talon was incorrectly reported from NASL, but was later corrected to have been observed at Edwards Air Force Base.

Dan Catlin, Sonny Bono Salton Sea National Wildlife Refuge, Imperial County

Dan Catlin investigated burrowing owls in the Imperial Valley from 2002-2003 All three young from one nest were missing part of a single toe. The deformed toe resulted from the

lack of the ultimate distal joint anterior to the talon. USFWS staff collected the three young in hopes of learning the cause of the deformations. Unfortunately, the specimens were never submitted to the laboratory for necropsy.

Jeff Kidd, Imperial and Riverside Counties

Four of thirty young banded outside of the nest in YEAR had a malformed toe, as shown in Figure C-1. This was not found in the adult population. Whether or not the four young with the malformed toe were related is unknown.

Christopher Nadeau, Imperial County

An owl was observed in the Imperial Valley in or adjacent to the Sonny Bono Salton Sea National Wildlife Refuge, with a foot missing and a large bulbous "callus" at the end of the tarsus. This female was nesting, and she appeared to be functioning normally. It is unknown if this was an injury or a deformity at birth, but the nature of the "callus" suggests an injury (Figure C-2).

Alberta, Canada

Ray Poulin

Several owls were observed with what were considered old injuries. However, the researchers noted that in some cases, it was possible that the abnormalities were deformities and not healed injuries (Figure C-3). The researchers also reported an owl with a missing talon, the distal end of the toe ending bluntly; this owl also had dark growths on the pad of the toe (Figure C-3). Researchers also observed owls with broken legs that were apparently healed (Figure C-3).

Literature Review of Burrowing Owl Foot and Talon Abnormalities

There are several potential causes of abnormal feet in birds that are not due solely to an underlying traumatic injury. As a first step in exploring whether the individual owls observed at NASL and elsewhere are symptomatic of a widespread problem that should be actively addressed, we discuss classes of possible mechanisms below. These range from strictly anthropogenic in nature (exposure to pesticides or other chemicals released into the environment), to mechanisms that are much less directly related to human activity, such as parasites and disease.

Developmental pathways may be disrupted by a variety of mechanisms, but still lead to the same result. For example, beak deformities in wild birds are probably most strongly associated with *in ovo* exposure to PCBs (e.g., Yamashita et al. 1993, Ludwig et al. 1996), but beak malformations in domestic chickens can result from incubation temperature fluctuations, physical shaking of the egg, temporary hypoxia during incubation, and nutrient deficiencies such as biotin, riboflavin, and manganese (Sunde et al. 1978, Stevens et al. 1984, Hurwitz 1992). Vitamin D deficiency in young chicks can also lead to deformities (Nichols et al. 1983, Kuiken et al. 1999). Determining the cause of deformities of wild birds without diagnostic work is unlikely. The discussion below highlights potential causes of skeletal abnormalities in burrowing owls, but clearly many different factors may be at work, and it is impossible to anticipate every potential interaction. If knowledge of specific causation is desired, then further diagnostic work is necessary.

Vitamin Deficiency

Vitamin deficiency may be a result of actual lack of the appropriate nutrients in the diet, or deficiency due to disruption of either the uptake or synthesis of a nutrient by a contaminant. Great blue heron embryos near a pulp mill were exposed to dioxins in the eggs, and reduced growth, beaks, and overall mass were associated with heron embryos closest to the contaminated site (Hart et al. 1991). The authors speculated that reduced levels of circulating calcium in the blood of these birds was possibly due to reduced synthesis of vitamin D, although the mechanism of reduced bone growth was not clear (Hart et al. 1991). There was no report of deformed limbs, however.

Bill development has been demonstrated to be sensitive to a variety of factors, including riboflavin deficiency (Kuiken et al. 1999 and references therein). Cormorants taken into captivity at 1-3 days of age developed deformed beaks at 2-3 weeks of age, apparently due to vitamin D_3 deficiency in the diet coupled with a lack of exposure to sunlight (Kuiken et al. 1999). Given that the causes of the malformed bones were related to the conditions of captivity, it seems unlikely that wild birds would be similarly deficient in this vitamin. This is supported by the fact that the nestlings of the captive birds, left in their natal nests, developed only one twisted beak among the 19, whereas the captive birds' rate was 8 in 20 (Kuiken et al. 1999). In addition, organochlorine exposure was considered an unlikely mechanism based on liver and kidney tissue samples taken from wild cormorants from the same colony at 4 weeks of age. The authors did note that the relationship between diet, sunlight, and beak deformities was tentative, and noted that very similar deformities are correlated with PCB exposure in cormorants

(Yamashita et al. 1993, Ludwig et al. 1996). The potential for PCBs and 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) to cause these deformities is discussed below.

Nichols and coworkers (Nichols et al. 1983) noted that bone deformities caused by vitamin D deficiency may also occur generally due to calcium deficiency or improper calcium-phosphorus ratios, either from inadequate diet or due to disease (Nichols et al. 1983). Rickets in captive cormorants manifested itself as bowed bones in both the wings and legs, and bill abnormalities. Radiographs indicated multiple fractures, suggesting that these animals would not have survived to adulthood in the wild. In this case, it was believed that dietary inadequacy from feeding on frozen fish was the cause (Nichols et al. 1983). To the extent that contaminants or disease interfere with proper bone development, similar malformations may occur in wild birds.

Genetic Abnormalities and Trans-generational Effects

Although this would not seem to be very prevalent due to the obvious fitness costs associated with malformed limbs or beak, there is at least one case where a heritable defect did in fact result in deformities of the feet and legs in wild birds. A female screech owl (*Otis asio*) captured on a superfund site in South Carolina possessed an extra leg-like appendage attached to the joint between the femur and tibiotarsus on her right leg (Albers et al. 2001). This female was taken into captivity and bred to healthy screech owls in the raptor colony at Patuxent Wildlife Research Center. Her offspring were also bred to normal individuals. This experiment demonstrated that the defect was both heritable and heterozygous dominant. Most of the malformed offspring did not survive very long, and likely could not have reached adulthood in the wild; however, clearly the less severely deformed birds were capable of surviving and breeding successfully despite the abnormality. Although genetic mutations arising from contaminants such as ionizing radiation are possible, it seems that other explanations for deformities are far more likely in most locations.

In addition, considerable work was carried out by Fernie and colleagues, examining the transgenerational effects of *in ovo* PCB exposure (Fernie et al. 2001, 2003a, 2003b). The offspring of kestrels exposed to PCBs through their diet demonstrated altered development and growth, including more rapid growth, reduced fledging mass, and earlier fledging dates. However, responses were gender-specific (Fernie et al. 2003b). Kestrels in this first generation were then bred to unexposed individuals, so that both maternal and paternal effects could be examined in the second-generation hatchlings. Patterns of growth in the maternally exposed F2 generation were similar to those shown in the exposed F1 generation , and in addition, there were gender-specific alterations in thyroid function for both paternally and maternally exposed F2 young (Fernie et al. 2003a). No skeletal abnormalities were noted.

Organochlorine Contaminants

Organochlorine contaminants include both those compounds released intentionally into the environment, such as the pesticides DDT and dieldrin, and those that are released due to manufacturing processes, such as polychlorinated dibenzo-*p*-dioxins (PCDDs), the most well-

known of which is 2,3,7,8'-tetrachlorodibenzo-*p*-dioxin or TCDD, polychlorinated dibenzofurans (PCDFs), and polychlorinated biphenyls or PCBs. These compounds frequently occur together in avian tissues, and thus separating effects has been problematic; however, negative effects including deformities have been documented. Because of the number of chlorinated hydrocarbon compounds with similar modes of action which may be present in a single sample, many studies calculate a toxicity equivalency (TEQ) with TCDD, as a means of assessing the net effects of a given mixture (Newman and Unger 2003 p. 187). It is not clear from previous work whether sublethal effects may differ among the compounds, as they are typically only evaluated from the standpoint of relative acute toxicity. The precise cogeners responsible for malformations are thus not known.

PCBs as a group have been correlated with increased egg mortality by some investigators (Tillit et al. 1992). In addition, some deformities have also been documented, and associated with levels of PCBs and related compounds (Yamashita et al. 1993, Ludwg et al. 1996). These deformities include crossed bills, edema, and less commonly, polydactyly or other deformities of the feet (Yamashita et al. 1993, Ludwig et al. 1996). However, other researchers did not find correlations between PCBs and TCDD in sample eggs and nestling fate (Larson et al. 1996, Rykman et al. 1998, Custer et al. 1999). There are many difficulties with inferring causation in uncontrolled field studies, including the possibility that the putative cause is simply correlated with the real causative agent, and thus the relationship observed by the investigators is spurious. Injecting eggs in the laboratory with TCDD and PCBs led to no increases in abnormalities in the embryos (Powell et al. 1997, 1998). Thus, although these compounds may well compromise the viability of the eggs, they do not by themselves seem to lead to deformities in nestlings.

Of the organochlorine pesticides, most of the compounds whose use was discontinued due to concerns regarding environmental persistence have now declined to the point where they are rarely detected in tissue samples of birds. The major exception to this is dichorodiphenyl-tricholroethane, or DDT. DDT itself is occasionally detected, but it is not considered as toxic as its metabolic breakdown products, particularly the ortho-para and para-para isomers of dichorodiphenlydichlroethene (DDE). The most common metabolic product is *p,p*'DDE, and it has remained in soils and in tissues of organisms despite the fact that the last legal application of DDT in the US was in 1971. DDT is still used in other countries to control mosquitoes in regions with malaria, and there is evidence that global transport can carry these compounds around the globe (Simonich and Hites 1995, Shen et al. 2005).

DDE is well known for its negative effects on reproduction, beginning with the documentation of eggshell thinning and subsequent breakage in numerous species of birds in the late 1960's and early 1970's (e.g., Porter and Wiemeyer 1969, Enderson and Burger 1970, Cade et al. 1971). Because of the link between p,p'DDE and eggshell thickness, eggshell thinning has usually been assumed to be the mechanism of injury to exposed birds. It is now understood that other mechanisms of injury occur, including endocrine disruption, immune suppression, and interactions with natural stressors to cause lowered survival or reproductive success (e.g., Fry and Toone 1981, Grasman et al. 1996, Gervais and Anthony 2003, Gervais et al. 2006). However, neither DDT nor its metabolites have been clearly associated with deformities in birds.

Organochlorines are most frequently associated with embryo mortality, as the damage is great enough to prevent young from surviving to hatching. One exception is o,p'DDE, another metabolic breakdown product of DDT. This particular isomer has been implicated in endocrine disruption, where normal sexual development is derailed and affected individuals show feminization of male reproductive tracts and secondary sexual characteristics (Fry and Toone 1981). However, this particular isomer of DDE was not found in nearly 100 sampled burrowing owl eggs from NAS Lemoore collected between 1996 and 2001 (Gervais et al. 2000, Gervais and Anthony 2003, Gervais unpublished data), and as previously noted, it is not associated with structural deformities such as malformed feet.

Heavy Metals

Selenium

The metal most well known for its effects on birds is selenium, as a result of the disastrous use of recycled irrigation drain water at Kesterson National Wildlife Refuge (e.g., Ohlendorf et al. 1986, 1988a, Hoffman et al. 1988). Selenium, especially in the form of selenomethionine (Hoffman and Heinz 1988) can cause multiple deformities in embryos, including those of the eyes, beak, wings, legs, and feet in addition to organ defects, with multiple defects frequently occurring within the same embryo (Ohlendorf et al. 1986, Hoffman et al. 1988, Ohlendorf et al. 1988a). Specific leg and foot deformities included contorted legs, swollen joints, contorted, incomplete, or missing metatarsi, missing toes, and fused toes (Ohlendorf et al. 1986, Hoffman et al. 1988). However, these individuals did not survive; much of the mortality occurs prior to hatching with selenium toxicosis (Ohlendorf et al. 1989).

Selenium is a well-characterized contaminant of drainwater within the San Joaquin Valley (Presser and Ohlendorf 1988). Selenium in living tissues is dynamic, varying seasonally and by location in a study of bioaccumulation of selenium in waterbird tissues (Ohlendorf et al. 1990). The authors concluded that species differences in bioaccumulation were not due to food habits, but were better explained by residence time in the contaminated site, and the foraging range of individuals (Ohlendorf et al. 1990).

The realization that agricultural drain water contained toxic levels of selenium and caused clear adverse effects in waterfowl led to management changes of wildlife areas, most notably use of fresh water in wetlands. However, it appears that many years must pass before concentrations of selenium in the livers of over-wintering waterfowl decline below those associated with impaired reproduction (Pavelglio et al. 1997).

Although it is primarily considered a contaminant of aquatic birds, there is some evidence that it may be incorporated into upland food webs and aquatic vertebrates, as indicated by tissue concentrations in gopher snakes and bullfrogs (Ohlendorf et al. 1988b). Selenium biomagnification in a grassland system led to tissue concentrations in grasshoppers great enough to lead to detectable tissue concentrations in their predators, in this case praying mantises, of up to 52 ug/g dry weight (Wu et al. 1995). Concentrations of selenomethionine above 3 ppm in eggs are considered indicative of selenium toxicosis in eggs (Heinz 1996). However, risks of selenium uptake in terrestrial systems appear to be far less than those in aquatic systems (Wu et al. 1995).

A feeding study with mallards suggested that enlarged foot pads, joint swellings, and calluses, in addition to claw and feather abnormalities, might be suitable biomarkers of chronic selenium toxicity (Albers et al. 1996). However, given that many of these outcomes can be the result of processes other than selenium toxicosis, tissue sampling should be used to verify the presence of selenium.

Burrowing owls at NAS Lemoore consume terrestrial prey almost entirely in contrast to owls at the Salton Sea National Wildlife Refuge, whose pellets also contained fish scales apparently from scavenged fish, and bivalve shells (Gervais et al. 2000, Gervais unpublished data). This difference in their diets suggests selenium concentrations would not be great at NASL. Eggs taken from NAS Lemoore and the Sonny Bono Salton Sea National Wildlife Refuge in 1996 were tested for selenium concentrations, but these were found to be normal (Gervais et al. 2000), and no further scans for selenium were performed on later samples. Selenium toxicosis in adult birds includes emaciation and loss of feathers on the head and neck (Ohlendorf et al. 1988); although some owls captured at Lemoore were emaciated, this appeared to be associated with low rodent densities (Rosenberg and Gervais unpublished data). No abnormal plumage was noted during any of the years nor locations of the demographics study. In addition, reduced reproductive output was only noted for owls whose eggs contained organochorine contaminants and whose pellets contained relatively few rodent remains (Gervais and Anthony 2003). However, at study sites other than NASL, owls with reduced plumage on the head were observed (C. Conway, pers. obs.).

Agricultural drain water can contain a variety of other metal contaminants as well as selenium, and some of these metals are also known to be toxic to birds. Boron was also present in tissues of birds contaminated with selenium, but boron was not considered likely to cause malformations (Hoffman et al. 1988). The authors noted that interactive effects were possible but unknown. Arsenic was found to be antagonistic to the effects of selenium in a controlled study using mallards, however; deformity rates were reduced in mallards fed both arsenic and selenium diets (Stanley et al. 1994). This highlights one of the greatest difficulties with interpreting tissue concentrations of samples collected from free-living birds: multiple contaminants are frequently documented, but the effects of their interactions with each other are rarely understood.

Cadmium

Cadmium has been implicated in abnormal bone formation in ptarmigan in Colorado as a result of dietary exposure. Willow accumulates cadmium to concentrations twice that in the soil, which can be quite high when associated with mining sites. Willow is a major food source for the ptarmigan. Skeletal calcium was found to be reduced, leading to lower bone calcium content and greater potential for bone fractures in ptarmigan in Colorado (Larison et al. 2000). Bones deficient in calcium could be expected to break more easily, and healed fractures in wild birds are likely to lead to malformed feet or legs. However, there is no history of industrial usage within the area of NASL consistent with elevated cadmium levels, and so it seems unlikely that burrowing owls are at risk from this metal at NASL.

Other metals

Strontium and barium may interfere with calcium uptake in the developing embryo, and thus the presence of these metals in the eggshell may lead to deformities due to improper calcium absorption and deposition in growing bones, leading to beak abnormalities (Mora et al. 2007). The potential for other skeletal abnormalities was not discussed.

Many studies of the effects of contaminants on birds find multiple contaminants in tissue samples, thus assigning probable causation is difficult. Multiple metals were present in eggs from clapper rails in the San Francisco Bay region, including strontium, chromium, barium, boron, cadmium, lead, and mercury, and embryos with deformed feet (extra digits or stunting) were found (Schwarzbach et al. 2006). Embryos with deformities were associated with wetlands in the San Francisco Bay region that contained elevated concentrations of aluminum, boron, chromium, lead, and barium, although mercury was present at elevated concentrations at all sampling sites (Schwarzbach et al. 2006). Interestingly, selenium was present at concentrations well below those believed to be toxic. Aluminum, barium, chromium, and strontium were statistically associated with the deformities (Schwarzbach et al. 2006). Earlier sampling of black-crowned night-herons (*Nycticorax nycticorax*) and snowy egrets (*Egretta thula*) within San Francisco Bay revealed DDE and PCBs in all of the eggs collected, and a wide variety of metals was also detected including chromium, strontium, selenium, and barium (Hothem et al. 1995). Eggs also contained DDE and PCBs, so it is not possible to separate effects of metals from effects of organochlorine contaminants. Two deformed night-heron embryos were found, both with abnormalities of the mandible, and one young egret and night heron each were seen with shortened and deformed mandibles (Hothem et al. 1995).

Disease

Avian pox

Avian pox, or *avipoxvirus*, is a member of the Poxviridae family of viruses. It can be spread through vectors such as mosquitoes or other blood sucking arthropods, or though direct contact with a pox lesion or contact between broken skin and pox particles in the environment. The virus is extremely resistant to environmental degradation. This disease has been recognized in the literature since the 1850s, and has been recorded in 232 species of birds representing 23 orders (Bolte et al. 1999). However, it is most well known from outbreaks in captive bird facilities. Raptors are not considered especially susceptible, but captive hawks, eagles, and falcons have been infected (Wheeldon et al. 1985, Krone et al. 2004). The virus can lie dormant for many months and infection can be spread through the use of cages or other equipment that came into contact with infected birds.

In the wild, disease prevalence is linked with mosquito abundance (Van Riper et al. 2002, Buenestado et al. 2004, Vander Werf et al. 2006), and clusters of cases have been reported in family groups of red-legged partridges (Buenestado et al. 2004), reflecting the contagion of this virus through direct contact with active lesions or contact with contaminated soil. Wild birds vary greatly in susceptibility by species (Bolte et al. 1999).

Pox virus is best known in wild birds for the epidemics it has caused in avifauna that had not previously been exposed (Kleindorfer and Dudaniec 2006). The most unfortunate example comes from Hawaii, where avian pox has spread rapidly through native bird populations due to introduced mosquitoes. Up to 10% of captured native birds showed signs of pox infection in one study (Atkinson et al. 2005). In research involving ‘elapaio, an average of 20% of all captured birds had active lesions, and an additional 16% had foot deformities or missing toes associated with healed lesions (Vander Werf et al. 2006).

Pox exists on mainland North America, and apparently has been here for quite some time; it has evolved to be genetically distinct from European strains (Adams et al. 2005). In particular, it has been noted in mourning doves in the Southeast (Gerhold et al. 2007). Cases of wild raptors contracting the disease are uncommon, but cases involving bald eagles, several hawk species, barred owls, and eastern screech owls have been reported (Deern et al. 1997). In these larger birds, including red-legged partridges, reports have indicated that avian pox left scars from healed lesions, but abnormalities such as loss of toes were not mentioned (Buenestado et al. 2004, Wheeldon et al. 1985). Disease susceptibility may be increased in individuals stressed by chemical exposure and shortage of food or other environmental factors (e.g., Porter et al. 1984). The type of lesions and growths caused by pox are consistent with the abnormalities observed and photographed (e.g., Figures B-1 and B-2) in the feet of wild burrowing owls at NASL (R. Bildfell, Oregon State University; pers. comm.).

Injury and Subsequent Pododermatitis (bumble foot)

Disease may secondarily be an agent of odd swellings on feet and legs following injury. Secondary infection of pox lesions has been suggested to be a major factor in the presence of severe scarring or possible digit loss (Vander Werf et al. 2006). Secondary infection of a cut or other wound on a wild burrowing owl’s foot might lead to swelling or the eventual development of scar tissue. Cage birds and captive raptors in particular, frequently suffer from a condition known as ulcerative pododermatitis, or “bumblefoot”, in which pressure sores or small wounds in the feet lead to abscesses. In captive birds, vitamin A deficiency, inactivity, and poor perch design have been linked to the development of this condition. Wild raptors may also develop pododermatitis, when an injury to the foot becomes infected. Photographs of bumblefoot (and pox) in captive birds are very similar to some of the deformities seen in the feet of wild burrowing owls, so it seems possible that this is the cause in at least some cases. Furthermore, although contaminants per se do not have a direct link to the development of these infections, their role in immunosuppression may make individuals with low or moderate body burdens of contaminants more susceptible to infection (e.g., Smits et al. 2002).

Parasites

Foot and leg abnormalities can also be caused by scaly face and leg mites. In pet birds the species of mite is typically *Knemidocoptes mutans* and in the wild, there are several species *Knemidocoptes* that are likely to occur, but difficult to identify. These mites bury into the tissue, and are only visible under a microscope. Mites tend to leave crusty, scaly lesions, somewhat similar to what was observed at NASL (Figures B-3 and B-4, but also external growths as shown

in B-1). The growths are a proliferative response of the epidermal cells (R. Bildfell, DVM, Oregon State University, pers. commun.), the same response as with Pox. Recovery is less likely if secondary infection results (R. Bildfell, pers. commun.). As with Pox, immunosuppression increases the severity of the infliction.

Conclusions and Recommendations

Burrowing owls at NAS Lemoore are exposed to a range of agricultural pesticides and legacy contaminants including DDE and PCBs (Gervais et al. 2000, Gervais and Anthony 2003, Gervais et al. 2006), but there are no indications that contaminant exposure is directly responsible for the observed deformities in the feet and legs of the owls at NASL. PCBs in particular have been associated with malformed embryos in birds, but these individuals typically die before hatching. No deformed embryos were observed in the 96 burrowing owl eggs collected 1996-2001 from NASL (J. Gervais, pers. obs.). Selenium is also linked to teratogenesis in developing embryos, but it was not found at elevated concentrations at NASL (Gervais et al. 2000), and it also would be expected to cause at least some malformed embryos. Similarly, there is no evidence that heavy metals are affecting reproduction at NASL. However, exposure to low levels of organochlorine contaminants has been linked to immune dysfunction, which may leave affected individuals more prone to disease or infection.

Injury, disease, or injury followed by secondary infection, as well as infestations of parasitic mites, appears to be the most plausible explanation for the observed abnormalities. All of these causes can result in similarly appearing abnormalities, and require laboratory testing for conclusive diagnosis. Some of the abnormalities observed are most likely direct injuries, such as a dislocation of a joint or a small fracture, as shown in Figures C-1 and C-3. Injuries to the feet and legs followed by infection lead to swellings that are very consistent with some of the observations in the field, as is Pox. Infection giving rise to the abnormalities observed at NASL is often associated with pododermatit*is* (bumblefoot), an infection that can penetrate deeply and cause loss of appendages, such as shown in Figure C-2, C-3 (upper photo), and C-4. This syndrome is well known to keepers of captive birds, particularly raptors. Given that burrowing owls spend so much of their time standing on the ground, it is entirely likely that wounds obtained while hunting subsequently become infected. Due to the poor circulation in the feet and legs, these injuries are slow to heal. Low levels of organochlorine contaminants are well documented in the NASL burrowing owl population, so it is also possible that the owls at NASL may be more susceptible to these infections than owls from populations with less of a contaminant legacy.

Our findings from our review suggest that the abnormalities observed at NASL are "natural" in the sense that they are expected with some varying frequency in healthy owl populations and that no further management actions are necessary. A conclusive diagnosis of avian pox may be useful, as this disease has not been previously documented in burrowing owls. To document that the external growths and toe-pad deformities are caused by avian pox and/or mites, we recommend that samples of the growths be collected and sent to a veterinarian diagnostic laboratory for testing. The primary cost is the capture of the owls. Diagnostic testing is likely to cost approximately $50 per sample (R. Bildfell, Oregon State University, pers. commun.). Owls to be sampled should have growths similar to that shown in Figure B-1 and B-

2. To sample the growths, the tissue should be flaked off without causing bleeding but sufficient to allow the crust and abnormal growth tissue to be collected. The samples should be stored in chemically clean vials at room temperature and sent to a veterinary diagnostic laboratory equipped to test for avian pox. The Fresno office of the California Animal Health and Food Safety Laboratory would be the most efficient laboratory given the location near NASL. Alternatively, other labs, such as that available at Oregon State University (College of Veterinary Medicine) could conduct the tests

Regardless of whether the mites or avian pox are one of the causes for the abnormal talons, feet, and legs of burrowing owls at NASL, such parasites and diseases occur naturally, and no treatment would be recommended at this time. The frequency of the occurrence of the abnormalities is not extraordinarily high and cycles in infection do occur (R. Bildfell, DVM, Oregon State University). Documentation of the occurrence of these abnormalities at NASL would be useful during any future research on burrowing owls at NASL that involved capture of burrowing owls. If the prevalence of these abnormalities continues to increase, then further diagnostic testing and investigation into possible immuno-suppression might be considered.

Acknowledgements

We thank John Crane and Rob Palmer at NAS Lemoore and Naval Facilities Engineering Command, Southwest, respectively, for their continued interest in the conservation and management of burrowing owls at Naval installations, and in particular, for their support of this project to evaluate field observations that suggested a potential for concern over the health of the burrowing owl population at NAS Lemoore. We are also very grateful for the observations of Byron Buckley that led to this evaluation, and to the observations and photographs made by the following, all of whom allowed us to include their observations and/or photos in this report: Jack Barclay, Dan Catlin, Jeff Kidd, Chris Nadeau, Ray Poulin, and Meg Schwender. We thank Dr. Rob Bildfell (DVM, OSU College of Veterinary Medicine) for very helpful discussions on avian diseases.

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Figure 1. Map of NASL showing location of all burrowing owl nests located from 1997-2001. The yellow line demarks the Station boundaries; each circle denotes the location of an owl nest, many of which were located in more than a single year.

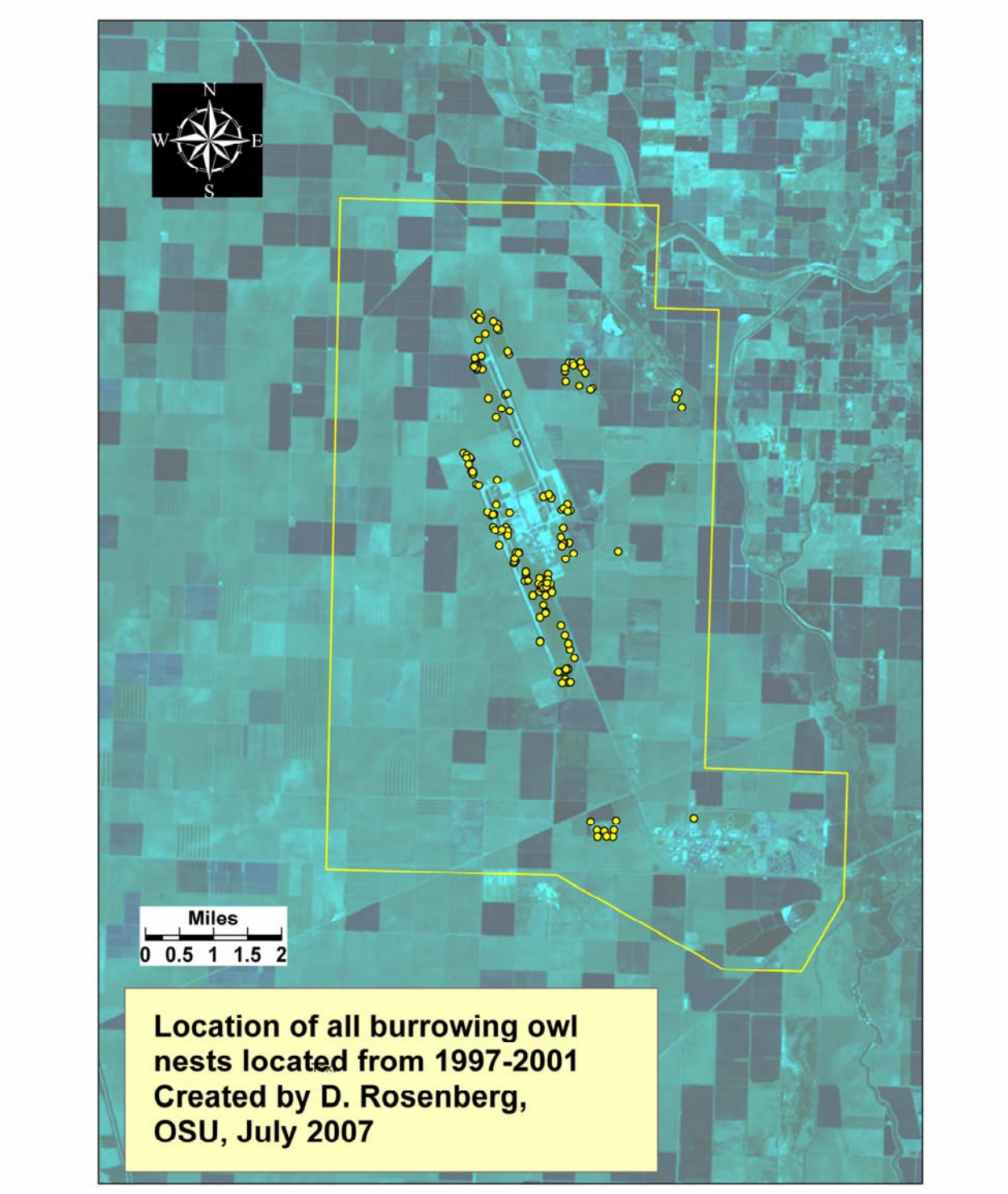


Figure 2. Map of NASL showing location of all burrowing owl nests located during 2006. The yellow line demarks the Station boundaries; each circle denotes the location of an owl nest.

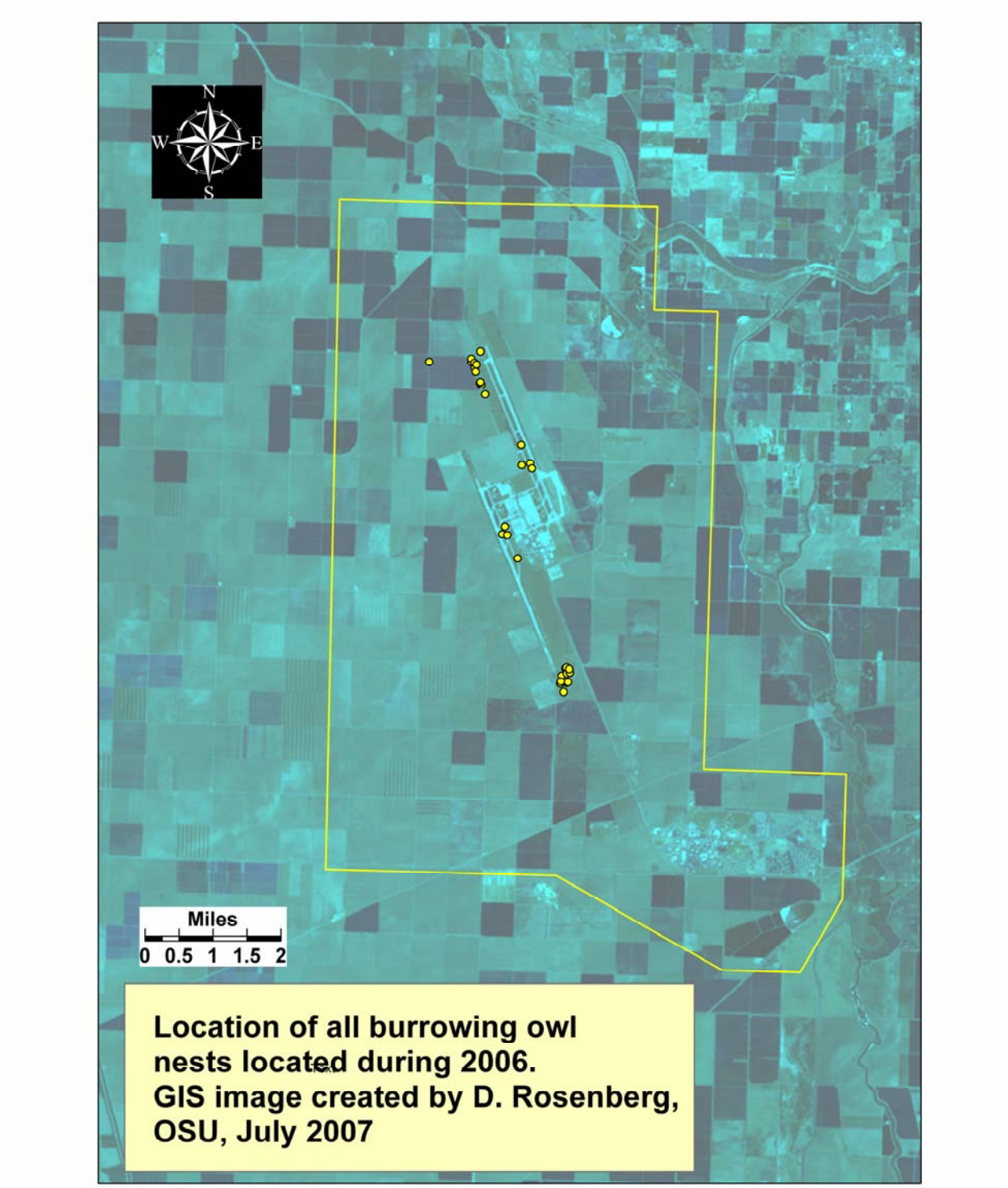


Figure 3. Map of NASL showing location of burrowing owl nests located during 2006 with owls observed with foot or leg abnormalities. The yellow line demarks the Station boundaries; each circle denotes the location of an owl nest; however, each cluster contains 3 nests, two of which are too close to see on this figure separately.

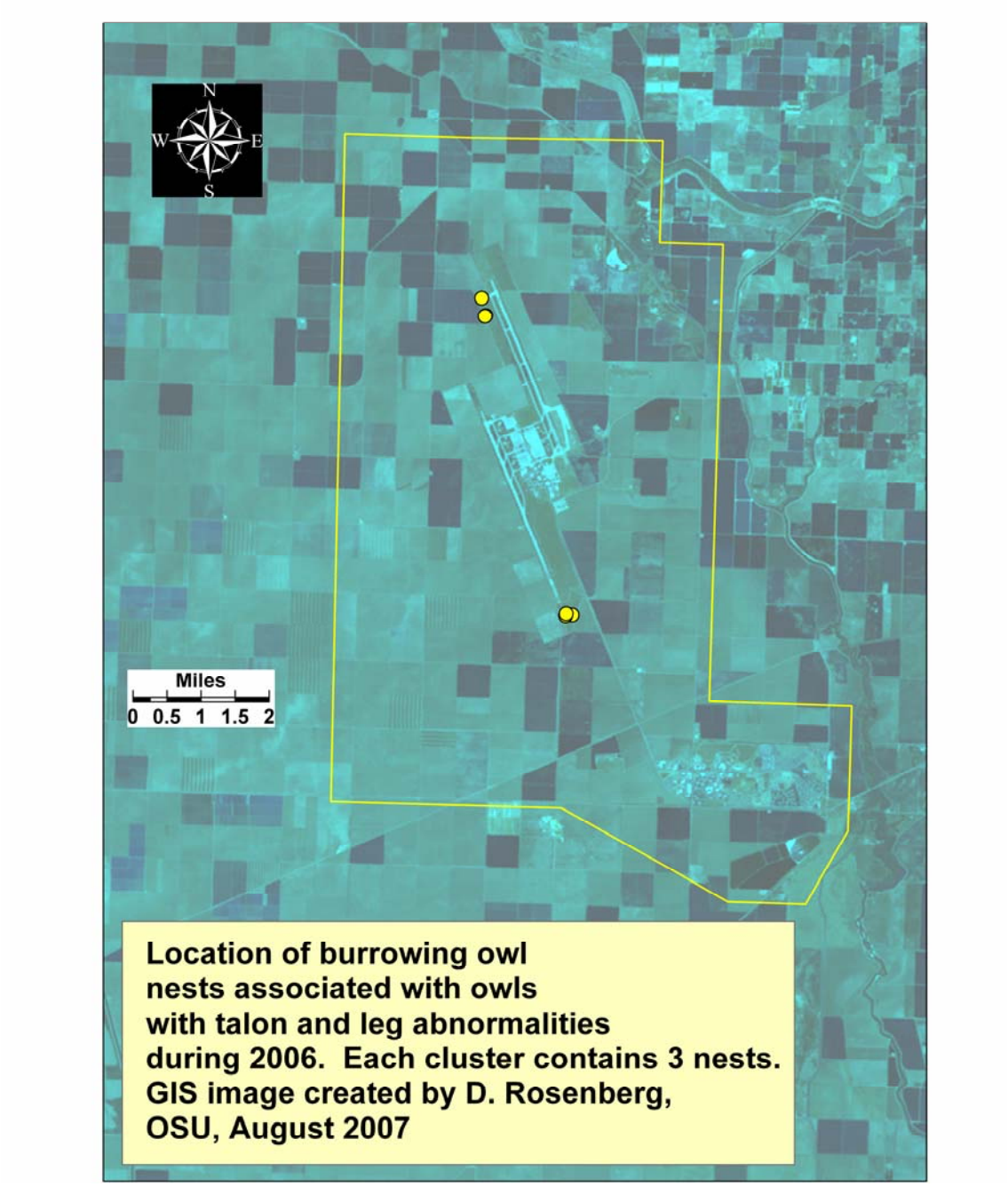
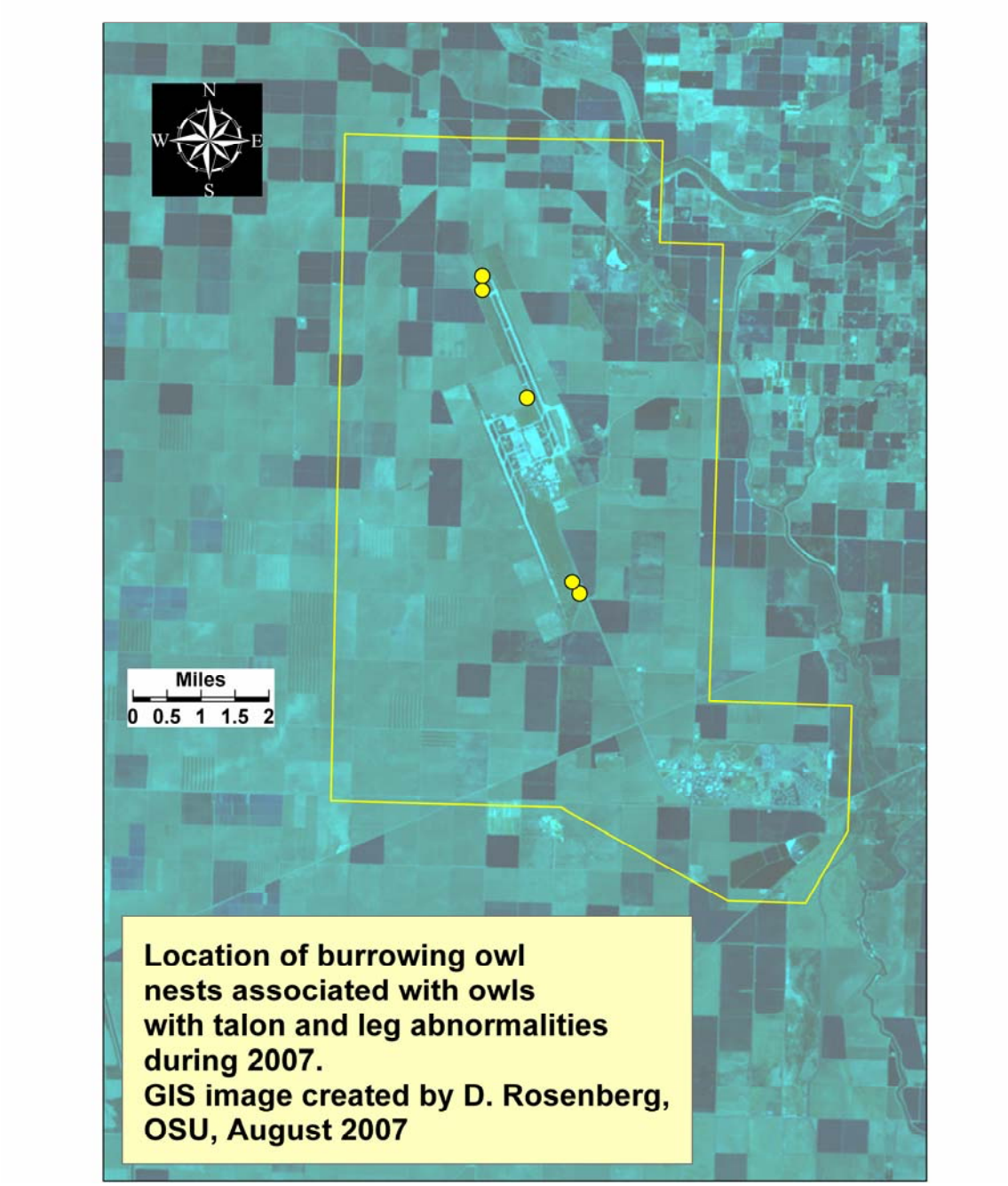


Figure 4. Map of NASL showing location of burrowing owl nests located during 2007 with owls observed with foot or leg abnormalities. The yellow line demarks the Station boundaries; each circle denotes the location of an owl nest.



Appendix A. Request submitted to Burrowing Owl List-serve.

Rosenberg, Dan

From: BurrowingOwl Discussion List on behalf of Rosenberg, Dan **Sent:** Tue 3/13/2007 5:31 PM
To: BURROWINGOWL@LISTSERV.UNL.EDU
Cc:
Subject: Request for observations of deformities on burrowing owls
Attachments:

Hi Folks,

Deformities of the talons and abnormalities on the legs of Burrowing Owls have been observed at a few locations where we have been banding. Courtney Conway, Jennifer Gervais, and I are seeking information on observations of deformities in burrowing owls from other sites. We would appreciate any information you have regarding your observation of any apparent deformities in either chicks or adults. If you have been capturing large number of owls, the following information would be helpful even if you have not observed such deformities:

1. years in which you have been capturing owls
2. general location
3. approx. number of chicks and adults (counted separately) captured
4. deformities noted - if none, please state so
5. IF deformities were noted, please provide details (number of owls, approx. age, description of deformity, nature of the landscape surrounding nest (urban, agriculture, etc.) and any photos or sketches that you may have.
6. If you did observe deformities, please provide contact info so we can follow up in more details.

Thank you very much for your assistance.

Cheers
dan

Daniel K. Rosenberg, Ph. D.

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AND

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OWI Web: http://oregonwildlife.org

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Appendix B. Photos and descriptions of owls noted with abnormalities at NAS Lemoore.

Figure B-1. Photo of adult owl with external growths on talon pads. Photo by B. Buckley.

Figure B-2. Photo of adult owl with swollen talon pad. Photo by M. Schwender.

Figure B-3. Photo of adult owl with scaling/lesions on toe. Photo by M. Schwender.

Figure B-4. Photo of adult owl with scabbed leg. Photo by M. Schwender.

Appendix C. Photos and descriptions of owls noted with abnormalities outside of NASL.

C-1. Photo showing deformed talon; Imperial Valley, CA.. Photo from J. Kidd.

Fig. C-2. Adult owl with presumed injured foot or loss of foot from infection. From Imperial Valley, CA. Photo taken by C. Nadeau.

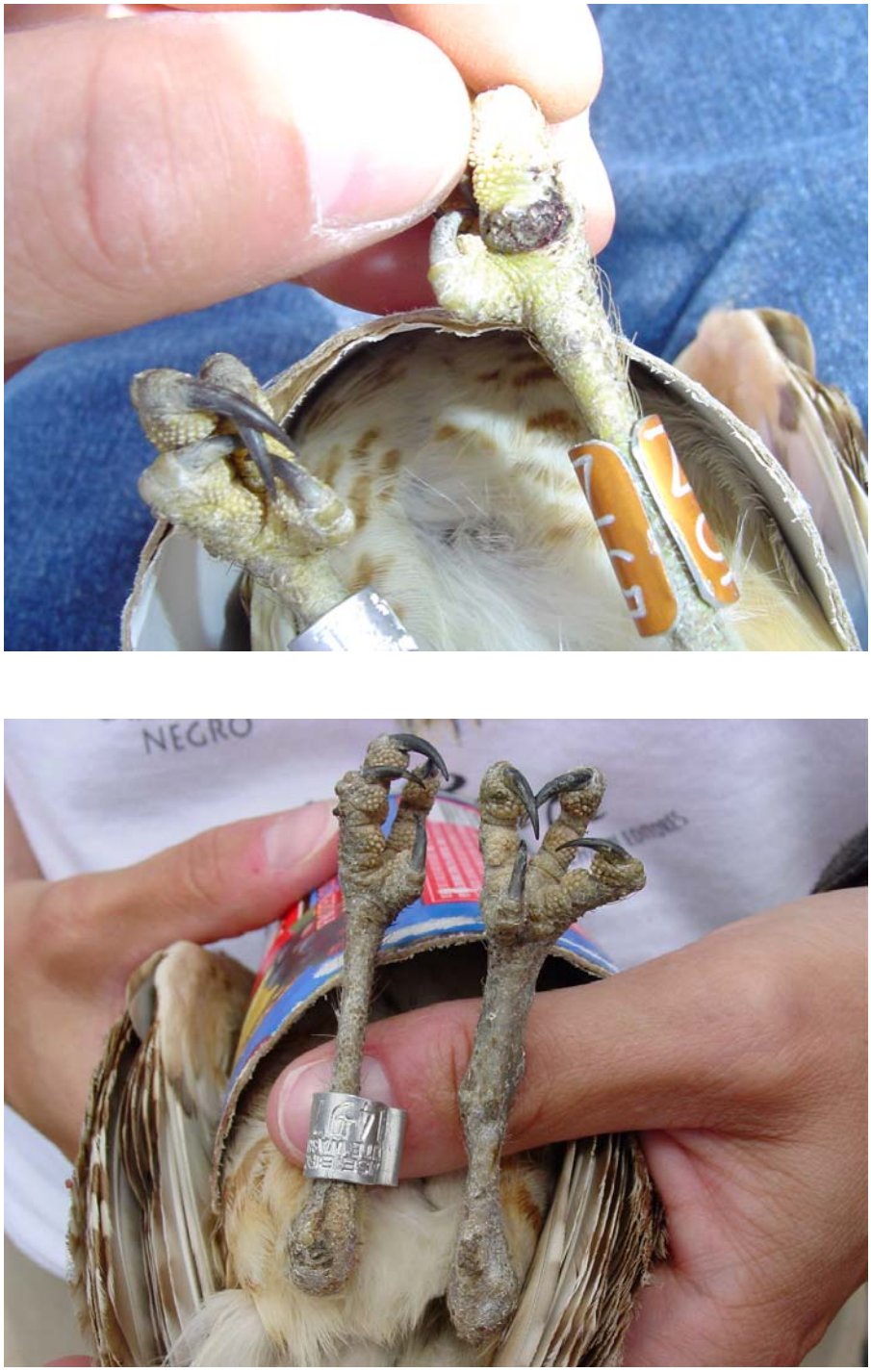

Figure C-3. Photograph of burrowing owls with (upper) toe with a missing talon, either injured or from malformation, and (b) injured leg. Owls captured in Alberta, Canada. Photo by R. Poulin.

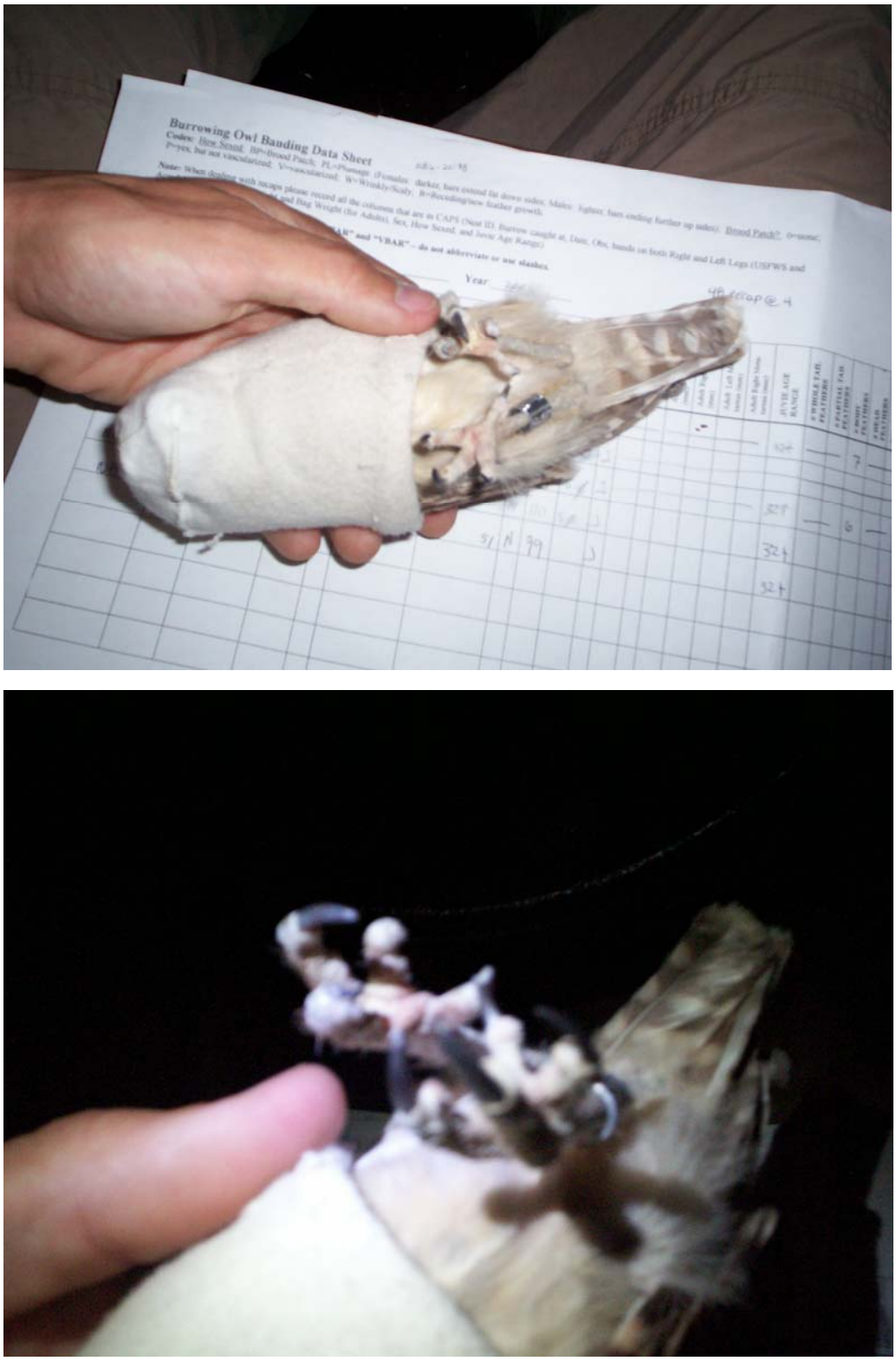

Figure C-4. Juvenile owl at Edwards Air Force Base showing the missing talon, left-most toe, left foot. Upper photo: dorsal view; Lower Photo: closeup of missing talon. Photo by Byron Buckley.