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Abstract: One hundred and fifteen Florida sandhill crane (*Grus canadensis pratensis*) chicks were captured in Osceola and Lake Counties, Florida in 1998 - 2000 and examined for evidence of disease. Evidence of *Eimeria gruis* and/or *E. reichenowi* infection was found in 52% of chicks examined. Ten chicks were positive for antibodies to St. Louis encephalitis virus and 1 of these chicks was also positive for antibodies to eastern equine encephalitis virus. Predation was the most commonly identified cause of mortality. An unidentified microfilaria, and an unknown protozoan were detected in blood smears from crane chicks. A number of other disease conditions were also encountered, including: ant bites, chigger infestations, helminth infections, bacterial infections, leg problems associated with capture, and a bill deformity.

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Many disease agents have been reported in sandhill cranes (*Grus canadensis*). Forrester and Spalding (2003) reported more than 40 disease agents in sandhill cranes in Florida. Antibodies to two arboviruses, eastern equine encephalitis (EEE) and St. Louis encephalitis (SLE), were also identified in cranes from Florida (Forrester and Spalding 2003). Avian pox was identified in Florida sandhill cranes (Simpson et al. 1975, Forrester and Spalding 2003). Suspected poisoning due to *Fusarium* mycotoxins from peanuts resulted in extensive sandhill crane mortality in Texas and New Mexico and was noted in migratory sandhill cranes in Florida (Forrester and Spalding 2003, Roffe et al. 1989, Windingstad et al. 1989), although, this disease has not been recognized as an acute cause of death in Florida sandhill cranes (Forrester and Spalding 2003). Trauma due to power line collisions and vehicle strikes also results in mortality (Forrester and Spalding 2003, Windingstad 1988).

In studies undertaken to identify causes of disease and mortality in prefledged sandhill crane chicks, predation has been identified as a major source of mortality (Littlefield and Linstedt 1992, DesRoberts 1997, Ivey and Scheuering 1997, Nesbitt and Schwikert 1999). The most commonly identified predator in these studies is the coyote (*Canis latrans*). Other conditions identified that resulted in disease or mortality in sandhill crane chicks during these studies include tracheal worms (*Syngamus trachea*), an unidentified bacterial infection, drowning, and intraspecific aggression. The goal of this study was to identify disease conditions and causes of mortality in pre-fledged sandhill crane chicks in central Florida.

STUDY AREA

Sandhill crane chicks were captured in Osceola and Lake Counties of central Florida (Fig. 1). Five areas were used as primary study sites; Escape Ranch (27° 53’ N, 80° 57’ W), Hayman’s 711 Ranch (27° 50’ N, 80° 59’ W), Overstreet Rd. (27° 57’ N, 81° 12’ W), Gardner-Cobb Marsh (28° 2’ N, 81° 18’ W), and Pruitt Ranch (28° 44’ N, 81° 56’ W). Three areas were used as incidental study sites: Crescent J Ranch (28° 4’ N, 81° 3’ W), Disney Wilderness Preserve (28° 8’ N, 81° 26’ W), and Kissimmee Park Rd. (28° 14’ N, 81° 19’ W). Incidental study sites were visited only once or twice during this study, whereas primary study sites were visited multiple times per week during the years they were used. All areas had extensive improved cattle pastures with scattered shallow seasonal and semipermanent marshes. Intermixed around and through the middle of pastures were pine flatwoods, oak woodlands, and cypress domes of varying sizes. Cattle grazing was conducted on all primary study sites; sod harvest was conducted on some (Escape, Haymans, Overstreet); and secondary uses included hunting, fishing, and birdwatching. Two of 3 incidental sampling areas were primarily cattle ranches; the third (The Nature Conservancy’s Disney Wilderness Preserve) was being man-

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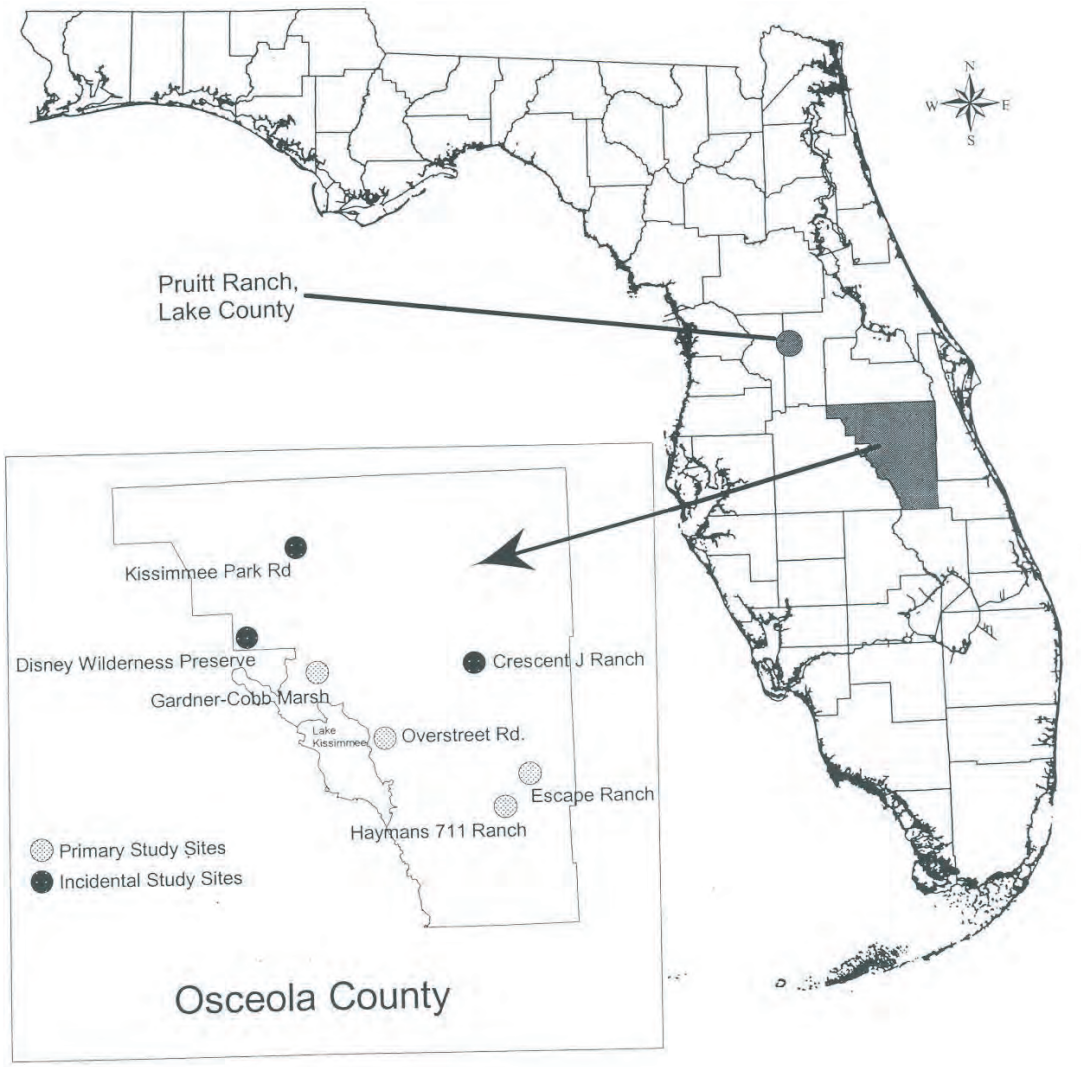

Fig. 1. Location of primary and incidental study sites of disease factors in Florida sandhill cranes in the state of Florida.

aged for native ecosystems. Bishop (1988) conducted a detailed review of land-use patterns and ecological traits of these and other Osceola County sites.

Study areas were not used in all years of this study. In 1998 only Escape Ranch and Hayman's 711 Ranch were sampled. Overstreet Road, the Pruitt Ranch, and the Gardner-Cobb Marsh were sampled in 1999 and 2000. The 3 incidental study sites were only sampled in 2000.

METHODS

Primary study sites were surveyed thoroughly from a vehicle 3 to 4 times weekly throughout the sandhill crane breeding season. When chicks were encountered they were chased by the observer on foot until they were captured by hand. Chick captures were made throughout the day. During 1998, chicks were not marked, and all captures were made on an opportunistic basis. DNA obtained from red blood cells was used to identify individuals (Jones 1998). In 1999 and 2000 all chicks encountered were marked with a uniquely coded transponder (Trovan Electronic Identification Systems, Trovan, Ltd., United Kingdom). The transponders were inserted subcutaneously in a dorsal location between the wings. To facilitate recapture of specific individuals and recover carcasses, a subset of these birds was also fitted with radio-transmitters (Advanced Telemetry Systems, Isanti, MN) as per Spalding et al. (2001). Chicks were sampled approximately every 2 weeks. Captured birds were held for up to 30 min before being released back to their

parents or at the site of capture. All chick carcasses that were recovered were necropsied.

Blood Sampling

Approximately 1 ml of blood was drawn from the jugular vein using a 1 ml insulin syringe with a 27-gauge, 13 mm needle. Blood smears were made in the field immediately following blood collection. Blood smears were fixed with methanol, stained with Leucostat (Fisher Diagnostics, Pittsburgh, PA) or Giemsa (E. M. Science, Gibbstown, NJ), and examined with a compound microscope for blood parasites. The remainder of the blood was centrifuged and the serum was tested for evidence of infection by selected mosquito-borne viruses including SLE virus (Flaviviridae: *Flavivirus*) and EEE virus (Togaviridae: *Alphavirus*).

Serum samples obtained in 1998 and 1999 were frozen immediately after centrifugation of whole blood samples, held for up to 1 month, and then transferred to a -60ºC freezer until submission for antibody testing to the Centers for Disease Control laboratory in Fort Collins, Colorado. Specific antibodies were detected using the plaque-reduction neutralization test (PRNT) (Beaty et al. 1995) using reference strains of SLE virus (TBH-28) and EEE virus (NJ/60). In 2000, serum samples for antibody testing were frozen immediately after centrifugation of whole blood samples and then submitted bi-weekly for analysis to the Florida Department of Health and Rehabilitative Services, Tampa Branch Laboratory, Virology Section. Sera were first screened using a hemagglutination-inhibition (HI) antibody assay using the same reference strains as above (Beaty et al. 1995). Sera were then tested using a PRNT antibody assay using reference strains of SLE virus (SLE-P15) and EEE virus (D64-837) (Beaty et al. 1995).

Oral Examination

The oral cavity of chicks was visually examined for the presence of granulomas as described by Carpenter and Gardiner (1979) and Novilla et al. (1981). Oral lesions matching these descriptions were assumed to be a result of disseminated visceral coccidiosis (DVC) but other causes could not be ruled out by gross examination.

Fecal Samples

Fecal samples were collected opportunistically and put in a small jar. In the lab, 2% potassium dichromate was added and samples were stored at room temperature until they were analyzed. Samples were analyzed for the presence of sporulated coccidial oocysts using a fecal flotation method (Sloss et al. 1994).

Determination of Predation

Only chicks with radio-transmitters attached were found after death. Predation was determined to be the cause of death if a carcass was recovered and evidence of trauma found at necropsy was consistent with predation. In many cases no carcass was left for recovery, in these instances, predation was judged to be the cause of death if evidence of a predator was observed, such as feathers or feces, at a site where a crane chick was killed.

RESULTS

Predation

Evidence to indicate predation was available in 8 cases. Bobcat (*Felis rufus*) predation was identified as the cause of death for 3 chicks based on method of kill, disposition of the carcass, and the presence of bobcat feces at the location the radio-transmitters were recovered. Coyote predation was suspected in the cause of death of 1 chick based on bite marks present on the carcass and the observation of coyotes nearby. Avian predation was suspected as the cause of death for 2 chicks. One chick was found whole with only a single puncture wound in its back and a great horned owl (*Bubo virginianus*) feather lying next to it. In the second case, flesh had been removed from the bones without damage to the bones, except that the skull was crushed, and there was no evidence from 2 previous captures to indicate disease. Unknown predators were suspected of killing two chicks. In these two cases, feathers and some tissues were found at the sites the radio-transmitters were recovered.

Disseminated Visceral Coccidiosis

Sixty-six of 160 (41%) examinations of the oral cavities of 114 live sandhill crane chicks were positive for granulomas. Of 44 fecal samples from 31 individual crane chicks, 7 (16%) (representing 7 individuals, 23%) were positive for oocysts of *Eimeria gruis* and/or *E. reichenowi*, one or both of which are etiologic agents of DVC. Three chicks captured multiple times were positive for oral granulomas at one capture but negative at subsequent captures. Two of 5 (40%) carcasses of sandhill crane chicks examined at necropsy were positive for DVC.

There were 27 pairs of siblings that were examined of the 114 sandhill crane chicks examined. Three of these pairs also had fecal samples examined for oocysts of *Eimeria* spp. No evidence of DVC infection was observed in 12 pairs of chicks, both siblings were positive for oral granulomas or fecal oocysts of *Eimeria* spp. in 10 pairs of chicks, and in 5 pairs of chicks, 1 individual was positive and 1 individual was negative for oral granulomas or fecal oocysts of *Eimeria* spp. Based on these data, siblings were considered subsequently to be a single sample for determination of prevalence data. With siblings combined, of 86 chicks examined, 52% were positive for signs of DVC. In all cases of oocyst shedding, either the chick or its sibling had oral granulomas.

Oocysts of either *E. gruis* or *E. reichenowi* were found in fecal samples obtained from 7 crane chicks. Three chicks had both *E. gruis* and *E. reichenowi* fecal oocysts, 2 had just *E.*

gruis fecal oocysts, and 2 had just *E. reichenowi* fecal oocysts. Three of these chicks also had oral lesions, 2 with just *E. reichenowi* fecal oocysts and 1 with both *E. gruis* and *E. reichenowi* fecal oocysts.

Arboviruses

From the 172 serum samples obtained from 115 chicks, 10 were positive for antibodies to SLE virus and 1 was also positive for antibodies to EEE virus. Virus was not isolated from any of the 45 samples collected and analyzed in 2000. All antibody-positive individuals had 90% neutralization titers of 1:10. All positive chicks were estimated to be younger than 9 days of age. For SLE virus, 4 pairs of siblings were both seropositive (positive for antibodies to SLE virus), 1 pair of siblings had a single seropositive chick, and a single chick (with no sibling observed) was seropositive. For EEE virus, 1 pair of siblings sampled in 1998 had a single seropositive chick while both siblings were also seropositive for antibodies to SLE virus. For all subsequent analyses, pairs of siblings were treated as a single sample, if 1 chick was positive or if both chicks were positive this counted as a positive sample, if both chicks were negative they counted as a negative sample. Across all years and in chicks younger then 9 days of age, 25% (6 of 24) samples were positive for SLE antibodies and 4% (1 of 24) were positive for EEE antibodies. Antibodies to SLE virus were detected from crane chicks in both Lake and Osceola Counties. Antibodies to EEE virus were detected only in Osceola County.

Blood Parasites

Blood smears from 114 individual birds were examined during this study. *Leucocytozoon grusi* (10%), *Haemoproteus antigonis* (7%), and *H. balearicae* (3%) were detected in blood smears and reported elsewhere (Dusek et al. 2004). An unidentified protozoan and an unidentified microfilaria were detected in 1 bird each in 1998 and 1999 respectively.

Miscellaneous Conditions

Presence or absence of ant bites was noted during 127 captures, 13 of which had lesions present (10%). Ant bites were assumed when small, approx. 1 mm diameter, raised lesions were observed on the feet and/or legs. As with DVC, other causes could not be ruled out by gross examination. Number of bites counted ranged from 1 to 42 (median = 4, n = 12). Of these 13 chicks, 12 were estimated to be less than 21 days of age. Four of these chicks were subsequently recaptured at 7, 13, 14, and 40 days later at which time no lesions were noted.

A single chigger (*Blankaartia sinnamaryi*) and associated lesion was detected on 1 of 2 siblings at their initial capture (less than 9 days of age) on the Pruitt Ranch. During the second capture (7 days later), chiggers were detected on the second sibling but not on the first. Fourteen days later, at the third capture, both chicks had chiggers present. Numbers of chiggers were described as 'numerous' on the second chick at the second capture, and 'moderate' on both chicks at the third capture.

Helminths were found in three chicks. Of 6 carcasses examined, 1 chick (50-60 days of age) had 4 *Brachylaima fuscatum* in the lower small intestine and 1 *Strongyloides* sp. in the duodenum. A second chick had a single nematode found in the proventriculus but it was not identified further. An unidentified microfilaria was detected by blood smear examination for a third chick. Seven days after capture this chick was found dead and a complete necropsy was performed but attempts to locate adult nematodes were unsuccessful.

A single chick had an unknown extra-cellular *Sarcocystis*-like protozoan parasite present on examination of its blood smear. This chick on external examination also had numerous 3 mm x 3 mm (estimated) rounded, raised nodules around the hocks of both legs.

Phagocytized rod-shaped bacteria were detected in the white blood cells of a single crane chick during this study via blood smear examination. This chick disappeared 4 days after capture and it could not be relocated using the radio-transmitter that was attached. The radio-transmitter was recovered 21 days later, but no remains of the chick were observed and it was believed to be dead. Adult sandhill cranes were observed in and around the wetland where this bird was captured, but no chicks were observed with them. A second crane chick had numerous rod-shaped bacteria detected in a swollen area on 1 foot. This chick was also infected with *H. balearicae* and was anemic and moribund. Diagnosis of the cause of death of this chick was linked primarily to the *H. balearica*e infection (Dusek et al. 2004).

Capture-related injuries were seen in 4 individuals. Injuries were confined to the legs and did not coincide with radio-transmitter attachment. In all cases chicks were unable to stand well at release. In 1 instance, 2 siblings were both afflicted with this problem. The chicks were captured quickly with no evidence of injury during capture. Both of the chicks were observed the day following capture with parents, but both birds were limping. The problem recurred in 1 of the chicks after handling 2 weeks later with no apparent leg problems. Chick feathers found at the release site the following day indicated that this chick probably did not survive, and it is unlikely that its sibling, not seen at the second capture, survived.

In another instance, a single chick very close to fledging, was chased on foot for approximately 50 m before it fell to the ground and was captured. After normal processing, the chick was unable to stand and died after about an hour. Death was most likely brought on by heat stress and possibly ant harassment following heat stress development. At necropsy, the cause of the leg problem and death was not discovered.

In the final case, a chick (but not its sibling) developed leg problems during capture or handling. On release, it was able to sit on its hocks but could not rise to its feet or walk. After about an hour of trying, the parents started to walk away with its

sibling. At that point the chick was recaptured. The legs were immediately cooled in a nearby wetland as the bird was panting and the bare parts of the legs were very warm. The chick was held overnight and fed mealworms and water. The following day the chick was able to walk although somewhat unsteadily. It was returned to its parents and sibling about 24 hr after capture. Based on observations by individuals that worked on the property this chick and its sibling were believed to have fledged.

One physical deformity was observed. The upper bill of one chick was curved to the left at an approximate 40° angle from the lower bill, which appeared straight and normal. On initial capture, the upper bill measured 55 mm (straight-line measurement, not along the curve of the bill) and a lower bill measurement of 62 mm (as measured from the top of the upper bill where the feathering begins). The distance between tips of the upper bill and lower bill was 42 mm. Tarsus length at this capture was 171 mm. Twenty-two days later, this chick was recaptured. At this time the upper bill was the same length but the lower bill was 80 mm and the distance between the tips of the upper bill and lower bill was 60 mm. Tarsus length at this time was 197 mm. The weight of the chick increased from 915 gm at the first capture to 1185 gm at the second capture.

DISCUSSION

Predation

Predation has been the most frequent cause of death reported for cranes in studies utilizing radio-telemetry techniques (Nesbitt et al. 1997, Nesbitt and Carpenter 1993). This is especially true for studies involving sandhill crane chicks (DesRoberts 1997, Ivey and Scheuering 1997, Nesbitt and Schweikert 1999). Of the 11 cases for which the cause of death was known or suspected in this study, 8 were linked to predation.

While bobcat predation was determined as the cause of death in 3 cases, it is likely that bobcats killed many of the 20 other crane chicks that disappeared and were suspected dead during this study. Bobcats accounted for the deaths of 31 of 32 released whooping cranes that died between 1993 and 1995 (Nesbitt et al. 1997), as well as for almost half the mortality of subadult greater sandhill cranes experimentally released into central Florida in 1986 and 1987 (Nesbitt and Carpenter 1993). In addition, 1 of 2 whooping crane chick deaths in central Florida in 2000 was attributed to a bobcat (Nesbitt et al. 2001).

Coyotes have colonized Florida recently and appear to be continuing their expansion into the state (Brady and Campell 1983, Wooding and Hardisky 1990). Coyote predation on crane chicks has been well documented in the western U.S. (DesRoberts 1997, Ivey and Scheuering 1997, Littlefield and Lindstedt 1992) and was first documented in Florida in 1993 (Nesbitt and Badger 1995). As coyotes become more common in Florida, it is likely they will account for a greater number of crane chick deaths.

With the exception of 1 study, avian predation on sandhill crane chicks seems to be rarely reported. In Oregon, U.S.A., avian predation accounted for 25 of 64 predation cases documented (Ivey and Scheuering, 1997). Golden eagles (*Aquila chrysaetos*) and great horned owls accounted for 9 and 10 of the cases, respectively. In 3 other studies, avian predation of crane chicks was not observed or was minimal (DesRoberts 1997, Littlefield and Lindstedt 1992, Nesbitt and Schweikert 1999). In all of these studies there were numerous unknown fates of individual birds. At least some of these unknowns could have been due to avian predation. In this study, only 2 birds were suspected of being killed by avian predators.

In all studies of chick survival, predation has played an important role as a mortality factor. Crane chicks, due to their relatively long prefledging period, precocial nature, and tendencies to feed in upland pastures, seem to make themselves suitable and easily available prey items. Numerous additional factors, including infectious and noninfectious diseases, may also play a role in the high predation mortality. One chick in this study had 2 preexisting disease conditions, DVC and infection with *H. balearicae* (Dusek et al. 2004). This chick was found moribund and abandoned by its parents. Likely the death of this chick would have been attributed to predation had it been first found by a predator. In a second chick that was killed by a bobcat, DVC was noted at necropsy and could have predisposed this individual to predation. Predation is an important component of chick mortality, but the role of disease in predation may be difficult to assess. Most studies of crane chicks have not addressed health parameters of marked birds until after a carcass is recovered. This method may not yield the primary causes of mortality and may lead to an over estimate of how important predation is to a population of birds.

Disseminated Visceral Coccidiosis

In one chick that was necropsied, DVC was severe enough that it may have predisposed the chick to predation. Sandhill crane chicks are reported to suffer mortality after experimental exposure to high doses of oocysts of *E. gruis* and/or *E. reichenowi* (Novilla et al. 1989). While many of the chicks that survived beyond approximately 3 weeks of age and examined in this study developed oral lesions consistent with DVC, they were likely not exposed to as high a dose of oocysts of *Eimeria* as experimental chicks have been. High dose *Eimeria* exposure in wild chicks is probably very rare under natural conditions or else seriously ill chicks are difficult to find.

Arboviruses

Antibodies to both SLE and EEE viruses were detected only in crane chicks estimated to be less than 9 days of age, and were suspected to be from maternal transmission of antibodies and not antibodies acquired through natural exposure. If mosquito borne transmission had occurred, antibodies should have

been detected throughout the age range of captured chicks. Additionally, antibody titers were barely detectable and did not increase with age or persist longer than 9 days after hatch. Maternal transmission of arbovirus antibodies has been reported in other avian species (Kissling et al. 1954, Reeves et al. 1954).

Antibodies believed to have developed from natural infection with EEE and SLE viruses have been detected previously in near-fledged (approximately 60 to 70 days of age) sandhill crane chicks in Osceola and Lake counties from 1992 to 1994 (Forrester and Spalding 2003). Antibodies to EEE and SLE were detected in 19% and 2%, respectively, of chicks sampled and contrasts with the lack of antibodies in chicks older than 9 days of age in this study. EEE and SLE virus transmission to sentinel chickens occurs sporadically from year to year and also varies in the month of onset from year to year (Day 1989, Day and Stark 1996). Additionally, yearly variation in rainfall may influence mosquito populations, which could subsequently affect transmission patterns of arboviruses. Drought conditions, as seen throughout most of this study, may severely limit arboviral transmission. This variation may account for the dissimilar results between this study and the 1992-94 study.

Blood Parasites

A single infection of a *Sarcocystis*-like protozoan was detected in the peripheral blood of one crane chick. Protozoan organisms that fit this description have not been reported before from sandhill cranes, although species of *Eimeria* can produce a similar looking merozoite that may be found in peripheral blood. Other blood parasites are discussed elsewhere (Dusek et al. 2004).

Miscellaneous Disease Conditions

A number of other etiologic agents of disease and disease conditions were observed during this study. These included ant bites, chigger infestation, helminth infection, and bacterial infection, as well as a bill deformity and leg problems associated with capture.

Littlefield (1987) suggested that harassment by the stinging ant *Myrmica incompleta* led to the death of sibling sandhill crane chicks in Oregon as the adults were unable to brood comfortably in unseasonably cool temperatures and as a result the chicks died from exposure. Chiggers and their associated lesions were first reported from four Florida sandhill cranes by Spalding et al. (1997). Helminths are found commonly in adult sandhill cranes in Florida (Forrester and Spalding 2003). Bacterial infection has been reported rarely in sandhill crane chicks. *Staphylococcus aureus* was indicated as a cause of death in California (DesRoberts 1997) and Forrester and Spalding (2003) have identified several species of bacteria from sandhill cranes. Little information is available, however, on the effects of any of these conditions on sandhill crane chicks.

Leg problems are common in crane chicks in captivity (Olsen and Langenberg 1996). Leg problems were reported in crane chicks captured just prior to fledging that were handled in a similar way as chicks in this study, but were much older (M. G. Spalding, unpublished data). In that case it was believed that nutritional deficiencies associated with eating mole crickets might have made those birds susceptible to leg injury. Florida sandhill cranes can average growth in their legs of up to 1 cm per day and this may make leg joints, tendons, and muscles susceptible to injury. Injury to chicks in this study appeared directly related to capture and handling of chicks, although it is not known if other preexisting conditions may have predisposed the chicks to leg injuries. Exertional myopathy has been previously reported in sandhill cranes (Hayes et al. 2003) and may explain the injuries observed. Muscle weakness following the stress of capture may also be the cause of the observed responses in these chicks rather than physical injury.

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