Journal of Wildlife Diseases, 46(3), 2010, pp. 1005–1011

Exposure to *Toxoplasma gondii* in Galapagos Penguins (*Spheniscus mendiculus*) and Flightless Cormorants (*Phalacrocorax harrisi*) in the Galapagos Islands, Ecuador

Sharon L. Deem,[1,2,3,7] Jane Merkel,[1] Lora Ballweber,[4] F. Hernan Vargas,[3,5] Marilyn B. Cruz,[6] and Patricia G. Parker[1,2] [1]WildCare Institute, Saint Louis Zoo, One Government Drive, Saint Louis, Missouri 63110, USA; [2]University of Missouri–Saint Louis, 8001 Natural Bridge Road, Saint Louis, Missouri 63121, USA; [3]Charles Darwin Foundation, Puerto Ayora, Santa Cruz, Galapagos, Ecuador; [4]Veterinary Diagnostic Laboratory, Colorado State University, 300 West Drake, Fort Collins, Colorado 80523-1644, USA; [5]The Peregrine Fund, 5668 West Flying Hawk Lane, Boise, Idaho 83709, USA; [6]Laboratorio de Epidemiología, Patología, y Genética–Proyecto Iniciativa Darwin, Galapagos National Park, Ecuador; [7]Corresponding author (email: deem@stlzoo.org)

ABSTRACT: *Toxoplasma gondii* is one of the most common protozoan parasites of humans and warm-blooded animals. Members of the family Felidae are the only definitive hosts of this parasite and, thus, important in the epidemiology of the disease. Previous studies on Pacific islands have found *T. gondii* infections in a number of avian species where domestic cats (*Felis catus*) have been introduced. Little is known about *T. gondii* in the Galapagos Islands, although introduced domestic cats in the archipelago are known to be *T. gondii* antibody-positive. In this study, we quantified prevalence of antibody to *T. gondii* in two threatened avian marine species, Galapagos Penguins (*Spheniscus mendiculus*) and Flightless Cormorants (*Phalacrocorax harrisi*), and tested the hypothesis that this parasite is more prevalent on Isabela Island (with cats) than on Fernandina Island (without cats). Overall, antibody prevalence was 2.3% in both Galapagos Penguins and Flightless Cormorants from samples collected during 2003–2005, and in 2008. In Galapagos Penguins (n=298), a significantly higher antibody prevalence was found in penguins on Fernandina Island (free of cats) than on Isabela Island (with cats; Fisher's exact test; P=0.02). In Flightless Cormorants (n=258), there was a higher antibody prevalence in cormorants living on Isabela than on Fernandina, although this difference was not statistically significant (Fisher's; P=0.19). This study is the first to show exposure to *T. gondii* in endemic avian species in the Galapagos Islands, providing evidence for disease-related risks associated with the feral cat population in the archipelago. We provide possible explanations for these findings and recommendations for future studies towards a better understanding of the epidemiology of *T. gondii* in the Galapagos Islands.

Key words: Antibody prevalence, feral cats, Flightless Cormorant, Galapagos Penguin, *Phalacrocorax harrisi*, *Spheniscus mendiculus*, *Toxoplasma gondii*.

Galapagos Penguins (*Spheniscus mendiculus*) and Flightless Cormorants (*Phalacrocorax harrisi*) are endemic bird species of Galapagos. Both species have limited geographic distributions and are considered endangered on the IUCN red list (http://www.redlist.org) due to their small ranges and major fluctuations in adult population sizes. The current population estimates for Galapagos Penguins and Flightless Cormorants are 1,500 and 1,600, respectively (Jiménez-Uzcátegui and Vargas, 2008). Recent studies have identified a number of pathogens of potential conservation concern for these populations, although the general health status of both species was rated good in 2006 (Travis et al., 2006a, b; Merkel et al., 2007; Levin et al., 2009).

Ninety-five percent of Galapagos Penguins, and all Flightless Cormorants, are found on Fernandina and Isabela Islands. The remaining 5% of the Galapagos Penguin population resides in small groups on Floreana, Santiago, and Bartolomé Islands. Fernandina is one of the most pristine islands in the world and has no cats (*Felis catus*), whereas Isabela has a long history of human presence with the resulting introduction of cats and other vertebrate species. The negative impact of invasive vertebrates in Galapagos has long been appreciated and has led, for instance, to one of the most successful island goat-eradication programs in the world (Cruz et al., 2009). The impact of feral cats on the wildlife of Galapagos has been less studied. However, in one study, vertebrate species were detected in more than 50% of prey items and in the biomass of cat scats sampled (Konecny, 1987). In 2005, cats were confirmed killing adult Galapagos Penguins on Isabela (Steinfurth and Merlen, 2005). The current distribution of feral cats on Isabela coincides with areas of high concentrations of Galapagos Penguins and Flightless Cormorants (Vargas, 2006).

The grave consequences of domestic cats as invasive species in island systems are well accepted, although studies have generally focused on the predatory behavior of cats (Alterio et al., 1998; Nogales et al., 2004). Studies linking cat introductions to diseases in island species, including humans, are less common (Wallace et al., 1972); however, as the only definitive hosts of *T. gondii*, feral cats have been linked to clinical *T. gondii* infections in a number of island bird species (Work et al., 2000; Dubey, 2002; Work et al., 2002).

Toxoplasma gondii is found throughout the world and is one of the most common protozoan parasites of humans and warm-blooded animals (Dubey and Beattie, 1988). Island species are often more severely affected following exposure to *T. gondii* (Dubey and Beattie, 1988), presumably due to their naïveté to previous exposure. Clinical signs associated with *T. gondii* in avian species are often nonspecific and include anorexia, diarrhea, and respiratory distress, and may result in death (Dubey, 2002, 2008). Mortality rates can be high in birds, as was best exemplified during an attempt to reintroduce the 'Alala (*Corvus hawaiiensis*) in Hawaii; approximately 20% of the wild population succumbed to *T. gondii* upon exposure at the release site (Work et al., 2000).

In Galapagos, there are no published studies on the epidemiology of *T. gondii* in domestic or wild species, with the exception of an antibody-prevalence study in which 63% of the domestic cats tested in Puerto Villamil, Isabela Island had demonstrable antibodies to *T. gondii* (Levy et al., 2008). Additionally, there is one report in Galapagos of a domestic chicken infected with *T. gondii* (Gottdenker et al., 2005). Based on these data, along with the wide feral cat distribution in the archipelago, we hypothesized that *T. gondii* exposure, resulting from direct and indirect contact with cats living near penguin and cormorant colonies, poses a disease threat to the conservation of Galapagos Penguins and Flightless Cormorants.

Recent findings of other parasites in both Flightless Cormorants and Galapagos Penguins (e.g., microfilariae), and in Galapagos Penguins (e.g., *Plasmodium* sp.), suggest these populations are exposed to parasitic agents that may have fitness costs and may cause direct mortality (Merkel et al., 2007; Levin et al., 2009). Therefore, the objectives of this study were to determine the prevalence of antibodies to *T. gondii* in Galapagos Penguins and Flightless Cormorants and to test the hypothesis that prevalence is significantly higher on Isabela, an island with feral cats, than on Fernandina, an island free of cats.

Galapagos Penguin and Flightless Cormorant colonies were visited in 2003–2005 and in 2008. Colonies were studied on the coasts of Isabela (0°25′30″S, 91°7′17″W) and Fernandina (0°22′0″S, 91°31′20″W; Fig. 1). Blood samples, collected in 2003–2005 from Flightless Cormorants ($n=258$), and in 2003–2005 plus 2008 from Galapagos Penguins ($n=298$), were stored at −80 C until tested in January 2009. Capture methods and blood processing have been described (Travis et al., 2006a, b). Briefly, both species are relatively sedentary on land and allow net- or hand-catches after initial approach in a small dinghy or when walking on land. General physical condition, as well as age and sex based on morphologic characteristics, were recorded as previously described (Snow, 1966; Boersma, 1977). Blood was collected by jugular venipuncture using a 20- or 22-gauge needle and a 12-ml syringe for penguins and a 20- or 22-gauge needle and 6-ml syringe for

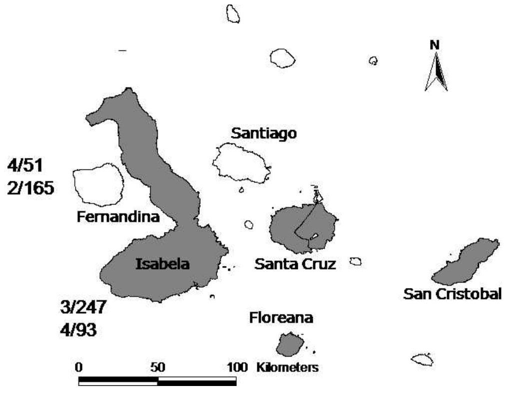

FIGURE 1. Map of the Galapagos Islands, Ecuador with number of *Toxoplasma gondii* antibody-positive Galapagos Penguins (upper) and Flightless Cormorants (lower) over individuals tested on Fernandina and Isabela Islands. Islands with human and feral cat populations are indicated in grey.

cormorants. Blood samples collected in serum separator tubes (Vacutainer PST gel, Becton Dickinson, Franklin Lakes, New Jersey, USA) were kept cool for 1–4 hr, centrifuged (Mobilespin, Vulcan Technologies, Grandview, Missouri, USA) for 20 min, and placed in cryogenic vials. Sera were stored in liquid nitrogen in the field, in a −80 C freezer, until transported to the USA. Samples were kept in a −80 C freezer at the Saint Louis Zoo for 6 mo–6 yr, until sent to the Colorado State University laboratory on dry ice.

The modified agglutination test (MAT) was performed using whole *T. gondii* tachyzoites of the RH strain fixed in formalin and mercaptoethanol, as described (Dubey and Desmonts, 1987). Positive and negative avian control sera were included in each run of the MAT. A titer ≥1:50 was considered positive.

Data were recorded for species, age, and sex (if known), year, antibody titer, and island. Fisher's exact tests (Petrie and Watson, 2006) were performed to compare antibody prevalences in penguins and cormorants between an island with cats (Isabela) and an island without cats (Fernandina).

Seven of the 298 Galapagos Penguins and six of 258 Flightless Cormorants (2.3%) were antibody-positive to *T. gondii*. All of the antibody-positive penguins and cormorants were clinically healthy at the time of sampling. Positive birds were from colonies along the coasts of both Isabela and Fernandina Islands. Three of 247 (1.2%) of the Isabela penguins and 8% (4/51) of the Fernandina penguins were antibody-positive. Titers in penguins ranged from 1:50 to 1:512, with Isabela penguins having titers of 1:50 ($n=1$) and 1:512 ($n=2$) and Fernandina penguins having titers of 1:50 ($n=3$) and 1:128 ($n=1$). In the cormorants, 4% (4/93) of the Isabela birds and 1.2% (2/165) of the Fernandina birds were antibody-positive. Titers differed in each of these six cormorants and ranged from 1:128 to 1:4,096, with Isabela cormorants having titers of 1:128, 1:1,024, 1:2,048, and 1:4,096 and Fernandina cormorants having titers of 1:512 and 1:1,024. Penguins on Isabela (with cats) were significantly less likely to have detectable antibody than those on Fernandina (without cats; Fisher's exact test; $P=0.02$). Cormorants on Fernandina (without cats) were less likely to be antibody-positive than those on Isabela, although the difference was not statistically significant (Fisher's exact test; $P=0.19$). Since neither species travels outside the archipelago, these data support that *T. gondii* exposure occurred in the Galapagos Islands.

Our original hypothesis, that higher antibody prevalence would be detected in both species on Isabela than in those on Fernandina, was not supported by the data. Explanations for this observation may include: 1) movement patterns of penguins and cormorants in Galapagos; 2) we were incorrect in our original assumption that no cats are present on Fernandina; 3) oocysts from cats on Isabela are dispersed by ocean currents across the canal between the islands; 4) the route of exposure to these marine bird species is not dependent on the definitive hosts (cats) but, rather, may be from another source (e.g., intermediate hosts); or 5) the

MAT resulted in false positives in some of the birds.

Previous studies based on genetic assessments have shown that penguins move throughout the archipelago and that the species should be regarded as one panmictic population (Nims et al., 2008). This could help explain the significant difference in antibody prevalence, with more penguins on Fernandina being positive although cats are not present on this island, as these penguins may have been exposed on Isabela. Conversely, the Flightless Cormorants do not move between islands and have low movement between colonies (Duffie et al., 2009), consistent with the higher (though nonsignificant) prevalence of exposure in cormorants on Isabela, where cats are present. However, the finding that two of 165 cormorants on Fernandina had antibody, even though this species does not move between islands, suggests that explanations 2, 3, 4, and 5 must also be considered.

The second possibility (cats are present on Fernandina) is unlikely, as cats have never been reported there by visiting park guards, scientists, or tourists. An islandwide ecologic inventory conducted in late 2009 provided further evidence that cats are not likely to be present on this island (G. Merlen, pers. comm.).

The third possible explanation, concerning movement of oocysts from cats on Isabela to Fernandina Island, is supported by previous studies. *Toxoplasma gondii* oocysts can move through freshwater drainages into marine systems, where they can accumulate in marine invertebrates that may serve as vectors if consumed by vertebrate predators (Miller et al., 2002; Arkush et al., 2003; Miller et al., 2008). Additionally, because *T. gondii* oocysts can sporulate and survive in seawater for months, they would remain infectious during dispersal in the water between Isabela and Fernandina (Lindsay et al., 2003; Shapiro et al., 2009). Galapagos Penguins and Flightless Cormorants may be exposed to *T. gondii* oocysts suspended in water or in invertebrate prey.

The fourth possibility of exposure not related to cat presence is supported by recent studies in the Arctic in which a high *T. gondii* antibody prevalence, in Arctic species on islands free of felid species, has been documented (Prestrud et al., 2007). Those authors suggested that migratory birds from Europe carry the parasite to the Arctic (Prestud et al., 2007). In Galapagos, few migrant species arrive in sites where penguins are encountered (e.g., southern Isabela; Harris, 1974). However, this mode of transmission is unlikely, as the Galapagos Islands are not located on a major flyway, few migrants visit western Isabela and eastern Fernandina where the majority of penguins and cormorants occur, and their dietary preference for small fish, and small fish and crustaceans, respectively, would suggest that this is not a mode of exposure for these two marine bird species.

Lastly, we cannot rule out false positives on the MAT, as this test has not been validated in Galapagos Penguins and Flightless Cormorants, and there could be a possible inaccuracy or cross-reacting substances in these avian hosts (Greiner and Gardner, 2000). This explanation is unlikely because the MAT has been used in a number of avian species, and the titers we used in this study are in accordance with accepted standards (Dubey and Desmonts, 1987; Dubey et al., 2000; Dubey, 2002).

Pathogens and parasites have become an increasingly recognized threat to the conservation of endemic birds in Galapagos (Wikelski et al., 2004; Parker et al., 2006; Deem et al., 2008) at a time of unprecedented growth rates in tourism throughout the archipelago and in the human population living on the inhabited islands (González et al., 2008). The introduction of invasive invertebrates, vertebrates, and pathogens are listed as top threats to the conservation of endemic species (Causton, 2007; Jimenez-Uzcategui et al., 2007; Deem et al., 2008; Bataille et al., 2009). An example of this threat is the recent discovery, and probable recent arrival, of *Plasmodium* spp. in the Galapagos Penguin population (Levin et al., 2009). This finding, along with the new evidence of exposure to *T. gondii*, may be additive with other previously documented threats, including El Nino events and oil spills, all of which challenge the long-term survival of this species (Vargas et al., 2006).

In addition to the threats associated with the disease agents identified in these two marine species in Galapagos, penguins may act as marine sentinels, thereby providing valuable data on the health of the marine ecosystem (Boersma, 2008). Thus, the finding of *T. gondii* in this species in Galapagos provides evidence of pathogen pollution (Cunningham et al., 2003) in the archipelago and may serve as a warning of marine environmental degradation within the archipelago.

In conclusion, this is the first study to show exposure to *T. gondii* in endemic avian species in Galapagos. *Toxoplasma gondii* may be pathogenic in Galapagos Penguins and Flightless Cormorants, due to their naïve status and lack of diverse natural immunity (Bollmer et al., 2007), and is most likely a recent introduction from feral cats living in the archipelago. However, to better understand the epidemiology of this pathogen in the Galapagos, we recommend that future studies include the determination of: 1) *T. gondii* prevalence in feral cats; 2) whether oceanic transport of oocysts occurs; 3) other potential sources of *T. gondii*; 4) the validity of the MAT *T. gondii* serologic assay for use in Galapagos Penguins, Flightless Cormorants, and other endemic Galapagos bird species; and 5) the possible fitness costs to Galapagos Penguins and Flightless Cormorants. Pending the results from further studies, this study provides evidence that, in addition to their predatory behavior, the feral cat population in the archipelago puts Galapago's species at risk of infection with *T. gondii*.

This study was funded by the Saint Louis Zoo Field Research for Conservation Program. Complementary funding was also provided by the Des Lee Collaborative Vision in Zoological Research and the WildCare Institute, Saint Louis Zoo. The authors thank the Galapagos National Park and Charles Darwin Foundation for supporting studies on the Galapagos Penguins and Flightless Cormorants. Additionally, the authors thank Sally Zemmer, Erika Travis, Nicole Gottdenker, R. Eric Miller, and Gustavo Jiménez-Uzcátegui for logistical support on this project. Laurie A. Baeten and David Lindsay were instrumental in diagnostic testing. Karl Campbell and Michelle Verant provided valuable comments on this manuscript.

LITERATURE CITED

Alterio, N., H. Moller, and H. Ratz. 1998. Movements and habitat use of feral house cats, *Felis catus*, stoats *Mustela erminea* and ferrets *Mustela furo*, in grassland surrounding Yellow-eyed Penguin *Megadyptes antipodes* breeding areas in spring. Biological Conservation 83: 187–194.

Arkush, K. D., M. A. Miller, C. M. Leutenegger, I. A. Gardner, A. E. Packham, A. R. Heckeroth, A. M. Tenter, B. C. Barr, and P. A. Conrad. 2003. Molecular and bioassay-based detection of *Toxoplasma gondii* oocyst uptake by mussels (*Mytilus galloprovincialis*). International Journal of Parasitology 33: 1087–1097.

Bataille, A., A. A. Cunningham, V. Cedeño, M. Cruz, G. Eastwood, D. M. Fonseca, C. E. Causton, R. Azuero, J. Loayza, J. D. Cruz Martinez, and S. J. Goodman. 2009. Evidence for regular ongoing introductions of mosquito disease vectors into the Galapagos Islands. Proceedings of the Royal Society B. doi: 10.1098/rspb.2009.0998.

Boersma, P. D. 1977. An ecological and behavioural study of the Galapagos Penguin. Living Bird 15: 43–93.

———. 2008. Penguins as marine sentinels. BioScience 58: 597–607.

Bollmer, J. L., F. H. Vargas, and P. G. Parker. 2007. Low MHC variation in the endangered Galápagos Penguin (*Spheniscus mendiculus*). Immunogenetics 59: 593–602.

Causton, C. 2007. Riesgos asociados con las rutas aereas actuales y propuestas hacia Galapagos. Informe Galapagos 2006–2007. F. C. D. Parque Nacional Galapagos, INGALA, pp. 55–59.

CRUZ, F., V. G. CARRION, K. J. CAMPBELL, C. LAVOIE, AND C. J. DONLAN. 2009. Bio-economics of large-scale eradication of feral goats from Santiago Island, Galápagos. Journal of Wildlife Management 73: 191–200.

CUNNINGHAM, A. A., P. DASZAK, AND J. P. RODRÍGUEZ. 2003. Pathogen pollution: Defining a parasitological threat to biodiversity conservation. Journal of Parasitology 89 (Suppl): S78–S83.

DEEM, S. L., M. CRUZ, G. JIMÉNEZ-UZCÁTEGUI, B. FESSL, R. E. MILLER, AND P. G. PARKER. 2008. Pathogens and parasites: An increasing threat to the conservation of Galapagos avifauna. *In* Galapagos Report 2007–2008. Puerto Ayora, Galapagos, Ecuador, pp. 125–130.

DUBEY, J. P. 2002. A review of toxoplasmosis in wild birds. Veterinary Parasitology 106: 121–153.

———. 2008. Toxoplasma. *In* C. T. Atkinson, N. J. Thomas and D. B. Hunter (eds.). Parasitic diseases of wild birds. Wiley-Blackwell, Ames, Iowa, pp. 204–222.

———, AND C. P. BEATTIE. 1988. Toxoplasmosis of animals and man. CRC Press, Boca Raton, Florida, pp. 1–220.

———, AND G. DESMONTS. 1987. Serological response of equids fed *Toxoplasma gondii* oocysts. Equine Veterinary Journal 19: 337–339.

———, W. B. SCANDRETT, O. C. H. KWOK, AND A. A. GAJADHAR. 2000. Prevalence of antibodies to *Toxoplasma gondii* in ostriches (*Struthio camelus*). Journal of Parasitology 86: 623–624.

DUFFIE, C. V., T. C. GLENN, H. VARGAS, AND P. G. PARKER. 2009. Genetic structure within and between island populations of the flightless cormorant (*Phalacrocorax harrisi*). Molecular Ecology 18: 2103–2111.

GONZÁLEZ, J. A., C. MONTES, J. RODRÍGUEZ, AND W. TAPIA. 2008. Rethinking the Galapagos Islands as a complex, social-ecological system: Implications for conservation and management. Ecology & Society. 13. www.ecologyandsociety.org/vol13/iss2/. Accessed April 2010.

GOTTDENKER, N., T. WALSH, H. VARGAS, M. DUNCAN, J. MERKEL, G. JIMÉNEZ-UZCÁTEGUI, R. E. MILLER, M. DAILEY, AND P. G. PARKER. 2005. Assessing the risks of introduced chickens and their pathogens to native birds in the Galápagos Archipelago. Biological Conservation 126: 429–439.

GREINER, M., AND I. A. GARDNER. 2000. Epidemiologic issues in the validation of veterinary diagnostic tests. Preventive Veterinary Medicine 45: 3–22.

HARRIS, M. 1974. A field guide to the birds of Galapagos. Collins, St. James's Place, London, United Kingdom, pp. 39–40.

JIMÉNEZ-UZCÁTEGUI, G., AND F. H. VARGAS. 2008. Censo del Pingüino de Galápagos y Cormorán no volador 2008. Informe técnico para la Fundación Charles Darwin y Servicio Parque Nacional Galápagos. Estación Científica Charles Darwin. Puerto Ayora, Galápagos, 19 pp.

———, V. CARRÍON, J. ZABALA, P. BUITRÓN, AND B. MILSTEAD. 2007. Estado de los vertebrados introducidos en Galápagos. *In* Informe Galápagos 2006–2007. F.C.D., Parque Nacional Galápagos, INGALA, pp. 140–145.

KONECNY, M. J. 1987. Food habits and energetics of feral house cats in the Galapagos Islands. Oikos 50: 24–32.

LEVIN, I. I., D. C. OUTLAW, F. H. VARGAS, AND P. G. PARKER. 2009. *Plasmodium* blood parasite found in endangered Galapagos Penguins (*Spheniscus mendiculus*). Biological Conservation. doi:10.1016/j.biocon.2009.06.017.

LEVY, J. K., P. C. CRAWFORD, M. R. LAPPIN, E. J. DUBOVI, M. G. LEVY, R. ALLEMAN, S. J. TUCKER, AND E. L. CLIFFORD. 2008. Infectious diseases of dogs and cats on Isabela Island, Galapagos. Journal of Veterinary Internal Medicine 22: 60–65.

LINDSAY, D. S., M. V. COLLINS, S. M. MITCHELL, C. N. WETECH, A. C. ROSYPAL, G. J. FLICK, A. LINDQUIST, AND J. P. DUBEY. 2003. Sporulation and survival of *Toxoplasma gondii* oocysts in sea water. Journal of Eukaryotic Microbiology 50: S687–S688.

MERKEL, J., H. I. JONES, N. K. WHITEMAN, N. GOTTDENKER, H. VARGAS, E. K. TRAVIS, R. E. MILLER, AND P. G. PARKER. 2007. Microfilariae in Galápagos Penguins (*Spheniscus mendiculus*) and Flightless Cormorants (*Phalacrocorax harrisi*): Genetics, morphology and prevalence. Journal of Parasitology 93: 495–503.

MILLER, M. A., I. A. GARDNER, C. KREUDER, D. M. PARADIES, K. R. WORCESTER, D. A. JESSUP, E. DODD, M. D. HARRIS, J. A. AMES, A. E. PACKHAM, AND P. A. CONRAD. 2002. Coastal freshwater runoff is a risk factor for *Toxoplasma gondii* infection of southern sea otters (*Enhydra lutris nereis*). International Journal of Parasitology 32: 997–1006.

———, W. A. MILLER, P. A. CONRAD, E. R. JAMES, A. C. MELLI, C. M. LEUTENEGGER, H. A. DABRITZ, A. E. PACKHAM, D. PARADIES, M. HARRIS, J. AMES, D. A. JESSUP, K. WORCESTER, AND M. E. GRIGG. 2008. Type X *Toxoplasma gondii* in a wild mussel and terrestrial carnivores from coastal California: New linkages between terrestrial mammals, runoff and toxoplasmosis of sea otters. International Journal of Parasitology 38: 1319–1328.

NIMS, B. D., F. H. VARGAS, J. MERKEL, AND P. G. PARKER. 2008. Low genetic diversity and lack of population structure in the endangered Galapagos Penguin (*Spheniscus mendiculus*). Conservation Genetics 9: 1413–1420.

NOGALES, M., A. MARTÍN, B. R. TERSHY, C. J. DONLAN, D. VEITCH, N. PUERTA, B. WOOD, AND J. ALSONSO. 2004. A review of feral cat eradiation on islands. Conservation Biology 18: 310–319.

PARKER, P. G., N. K. WHITEMAN, AND R. E. MILLER. 2006. Perspectives in ornithology. Conservation medicine in the Galápagos Islands: Partnerships among behavioural, population and veterinary scientists. Auk 123: 625–638.

PETRIE, A., AND P. WATSON. 2006. Statistics for veterinary and animal science, 2nd Edition. Blackwell Publishing Ltd., Oxford, United Kingdom, 299 pp.

PRESTRUD, K. W., K. ASBAKK, E. FUGLEI, T. MORK, A. STIEN, E. ROPSTAD, M. TRYLAND, G. W. GABRIELSEN, C. LYDERSEN, K. M. KOVACS, M. J. J. E. LOONEN, K. SAGERUP, AND A. OKSANEN. 2007. Serosurvey for *Toxoplasma gondii* in arctic foxes and possible sources of infection in the high Arctic of Svalbard. Veterinary Parasitology 150: 6–12.

SHAPIRO, K., J. LARGIER, J. A. K. MAZET, W. BERNT, J. R. ELL, A. C. MELLI, AND P. A. CONRAD. 2009. Surface properties of *Toxoplasma gondii* oocysts and surrogate microspheres. Applied and Environmental Microbiology 75: 1185–1191.

SNOW, B. K. 1966. Observations on the behaviour and ecology of the Flightless Cormorant *Nannapterum harrisi*. Ibis 108: 265–280.

STEINFURTH, A., AND G. MERLEN. 2005. Predación de gatos salvajes (*Felis catus*) sobre el pingűino de Galápagos (*Spheniscus mendiculus*) en Caleta Iguana, Isla Isabela. Report to the Charles Darwin Research Station and Galapagos National Park Service. Charles Darwin Research Station, Puerto Ayora, Isla Santa Cruz, Galápagos, pp. 1–8.

TRAVIS, E. K., F. H. VARGAS, J. MERKEL, N. GOTTDENKER, R. E. MILLER, AND P. G. PARKER. 2006a. Hematology, plasma chemistry, and serology of flightless cormorant (*Phalacrocorax harrisis*) in the Galápagos Islands, Ecuador. Journal of Wildlife Diseases 42: 133–141.

———, ———, ———, ———, ———, AND ———. 2006b. Hematology, serum chemistry, and serology of Galápagos Penguins (*Spheniscus mendiculus*) in the Galápagos Islands, Ecuador. Journal of Wildlife Diseases 42: 625–632.

VARGAS, F. H. 2006. The ecology of small populations of birds in a changing climate. PhD Thesis. University of Oxford, Oxford, England, 353 pp.

———, S. HARRISON, S. REA, AND D. W. MACDONALD. 2006. Biological effects of El Niño on the Galápagos Penguin. Biological Conservation 127: 107–114.

WALLACE, G. D., L. MARSHALL, AND M. MARSHALL. 1972. Cats, rats, and toxoplasmosis on a small Pacific island. American Journal of Epidemiology 95: 475–482.

WIKELSKI, M., J. FOUFOPOULOS, H. VARGAS, AND H. SNELL. 2004. Galápagos birds and diseases: Invasive pathogens as threats for island species. Ecology and Society 9: 5. www.ecologyandsociety.org/vol9/iss1/art5/. Accessed April 2010.

WORK, T. M., J. G. MASSEY, B. A. RIDEOUT, C. H. GARDINER, D. B. LEDIG, O. C. H. KWOK, AND J. P. DUBEY. 2000. Fatal toxoplasmosis in free-ranging endangered 'Alala from Hawaii. Journal of Wildlife Diseases 36: 205–212.

———, ———, D. S. LINDSAY, AND J. P. DUBEY. 2002. Toxoplasmosis in three species of native and introduced Hawaiian birds. Journal of Parasitology 88: 1040–1042.

Submitted for publication 10 November 2009.
Accepted 16 February 2010.