J. Parasitol., 94(3), 2008, pp. 584–590

PATTERNS OF PARASITE ABUNDANCE AND DISTRIBUTION IN ISLAND POPULATIONS OF GALÁPAGOS ENDEMIC BIRDS

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ABSTRACT: Parasite life-history characteristics, the environment, and host defenses determine variation in parasite population parameters across space and time. Parasite abundance and distribution have received little attention despite their pervasive effects on host populations and community dynamics. We used analyses of variance to estimate the variability of intensity, prevalence, and abundance of 4 species of lice (Insecta: Phthiraptera) infecting Galápagos doves and Galápagos hawks and 1 haemosporidian parasite (Haemosporida: Haemoproteidae) infecting the doves across island populations throughout their entire geographic ranges. Population parameters of parasites with direct life cycles varied less within than among parasite species, and intensity and abundance did not differ significantly across islands. Prevalence explained a proportion of the variance (34%), similar to infection intensity (33%) and parasite abundance (37%). We detected a strong parasite species-by-island interaction, suggesting that parasite population dynamics is independent among islands. Prevalence (up to 100%) and infection intensity (parasitemias up to 12.7%) of *Haemoproteus* sp. parasites varied little across island populations.

The abundance and distribution of parasites have received little attention despite their pervasiveness, diversity, and potential impacts on host population and community dynamics (Windsor, 1998; Poulin, 1999; Mouritsen and Poulin, 2005). Parasite life-history characteristics, together with their biotic and abiotic environments and the antiparasite defenses of their hosts, determine variation in parasite abundance, prevalence in host populations, and intensity of infection in individual hosts across space and time (Poulin, 1998; Krasnov et al., 2006). Unlike prevalence, parasite infection intensity and abundance per host have been found to vary less within, than among, parasite species (Arneberg et al., 1997; Krasnov et al., 2006; Poulin, 2006). Whether these patterns can be generalized to all parasites will depend on additional observations over a range of parasite and host species. Parasites analyzed previously have complex life cycles, wherein some free or vectored life stages might be affected by environmental conditions, which subsequently influence encounter rates between parasites and hosts (Poulin, 2006). In contrast, species with direct life cycles, such as lice (Insecta: Phthiraptera), are less likely to face the challenges imposed by free-living stages and, as such, the variation in population parameters might differ from that presented by parasites with indirect life cycles.

We studied 4 louse species and 1 vector-borne haemosporidian parasite (*Haemoproteus* sp.). Lice have direct life cycles, i.e., they complete all their developmental stages on the host, but they vary in feeding strategies, host specificity, and mobility. Of the 2 louse species that parasitize endemic doves (*Zenaida galapagoensis*), *Columbicola macrourae* (Ischnocera: Philopteridae) is commonly found on wing and tail feathers, and it is more mobile than *Physconelloides galapagensis* (Ischnocera: Philopteridae), which feeds on body feathers. The better dispersal capacity of *C. macrourae* lice has been confirmed by straggling events, (movement of parasites between species), population genetics analyses, and coevolutionary studies (Johnson et al., 2002; Clayton and Johnson, 2003; Whiteman et al., 2004). The other 2 louse species parasitize endemic Galápagos hawks (*Buteo galapagoensis*). *Colpocephalum turbinatum* (Amblyceran: Menoponidae) feeds on epidermal tissues and blood over most regions of the host's body, and it has a strong dispersal capacity (Whiteman and Parker, 2004). *Degeeriella regalis* (Ischnoceran: Philopteridae) feeds mainly on keratin of feathers and dead skin, and disperses poorly. Host defenses against these lice are restricted to preening in the case of *D. regalis*, but they include immune defenses in the blood-feeding *C. turbinatum* (Marshall, 1981; Whiteman and Parker, 2004; Whiteman et al., 2006). Haemosporidian parasites infect the endemic dove, but they have not been reported from hawks (Padilla et al., 2006; Parker et al., 2006). These parasites have an indirect life cycle, which includes dipteran vectors, in which the macrogametes undergo fertilization to form zygotes, and meiosis takes place with subsequent development of infective sporozoites. Hippoboscid flies are implicated in the transmission of *Haemoproteus* spp. infecting Columbiformes (Valkiūnas, 2005).

The Galápagos dove is the only columbiform species in the archipelago. It occurs on all the major islands, including the 2 northern and the somewhat isolated islands of Darwin and Wolf (*Z. g. exsul*), and as the main part of the island group (*Z. g. galapagoensis*) (Santiago-Alarcon and Parker, 2007). Genetic evidence suggests a high degree of historical gene flow among populations of the southern subspecies, which is supported by low and not significant F_{ST} values (Santiago-Alarcon et al., 2006). F_{ST} values also indicate some degree of genetic structure between southern and northern populations (data not shown). The Galápagos hawk is widely distributed among the larger islands, and it is the top predator in the archipelago. Unlike the endemic dove, hawk populations are highly structured, and they have low levels of genetic diversity, as well as behavioral and morphological differences across islands (Bollmer et al., 2003, 2005, 2006; Whiteman et al., 2006).

The Galápagos Islands represent the only Pacific Ocean archipelago that still preserves its entire avifauna (Tye et al., 2002). Some bird populations are declining, however, and we have detected several infectious agents, e.g., *Haemoproteus* sp. and *Chlamydophyla psittaci*, with interspecific transmission potential, infecting the endemic Galápagos dove (*Z. galapagoensis*) (Padilla et al., 2004). In addition, a vector of avian malaria

Received 6 June 2007; revised 11 September 2007; accepted 3 October 2007.

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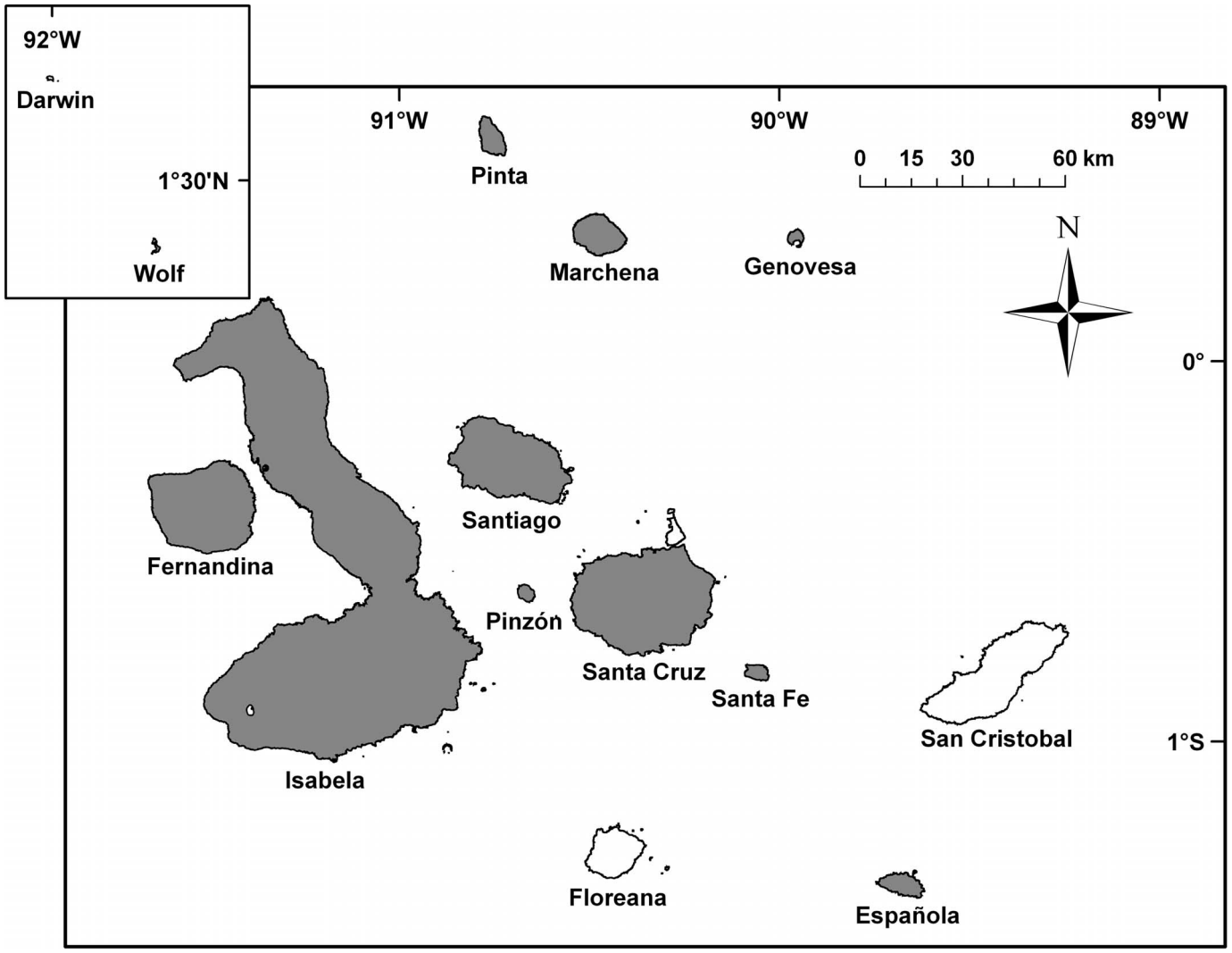

FIGURE 1. Map of the Galápagos archipelago, with islands on which Galápagos doves (*Zenaida galapagoensis*) and Galápagos hawks (*Buteo galapagoensis*) were sampled shaded in gray. Wolf and Darwin islands are located 186 and 239 km, respectively, northwest of the northern tip of Isabela Island.

parasites (*Plasmodium* sp.), *Culex quinquefasciatus*, has been reported on the islands (Whiteman et al., 2005), which is worrisome due to the negative impacts that this blood parasite has had on Hawaiian endemic birds (van Riper et al., 1986; Atkinson et al., 2000; Atkinson, Dusek, and Lease, 2001; Atkinson, Lease et al., 2001). Furthermore, we have previously shown that lice can straggle from doves to hawks when the later preys on the former (Whiteman et al., 2004). Thus, the possibility of interspecific transmission of infectious agents among Galápagos endemic birds highlights the importance of analyzing the variability of parasite population parameters across the archipelago.

In the present study, we analyzed the spatial variation of intensity, abundance, and prevalence of 4 species of lice (Insecta: Phthiraptera) infecting Galápagos doves and Galápagos hawks and 1 haemosporidian parasite (Haemosporida: Haemoproteidae) infecting the doves across island populations throughout their entire geographic ranges. ''Intensity is defined as the number of conspecific parasites living in (or on) an infected host, and abundance is defined as the number of conspecific parasites living in (or on) any host individual (intensity > 0, abundance ≥ 0)'' (Rózsa et al., 2000). Prevalence refers to the proportion of the host population that is infected (Bush et al., 1997). These measures are related by abundance = intensity $\times$ prevalence. Thus, abundance is the infection intensity averaged over all individuals in the host population, whether infected or not.

Parasite species were selected because of previously reported negative effects of closely related parasites on the condition of individuals of other host species such as rock pigeons (*Columba livia*; Booth et al., 1993; Brown et al., 1995) and honeycreepers (Drepanididae) from Hawaii (Atkinson et al., 2000, Atkinson, Dusek, and Lease, 2001; Atkinson, Lease et al., 2001). We also collected mites and hippoboscid flies, but these were not included here because of low sample sizes.

MATERIALS AND METHODS

We live-captured 199 Galápagos hawks from 8 islands (Fig. 1; May–August 2001: Española, n = 8; Isabela, n = 25; Marchena, n = 26;

Santa Fe, n = 13. May–July 2002: Santiago, n = 58. May–July 2003: Fernandina, n = 28; Pinta, n = 31; Pinzón, n = 10) and 139 Galápagos doves from 6 islands (Fig. 1; May–July 2002: Santiago, n = 27; Santa Cruz, n = 23; Santa Fe, n = 24; Española, n = 24. June–July 2004: Genovesa, n = 21. July 2005: Wolf, n = 20). We sampled endemic doves using hand nets and mist nets following the guidelines in Ralph et al. (1996); hawks were captured using techniques described in Bollmer et al. (2005).

For haemosporidian parasites, we took blood samples (50 μl) by venipuncture from 25 doves each from Santa Cruz, Santa Fe, and Española Islands, 30 each from Santiago and Genovesa Islands, and 29 from Wolf Island (Fig. 1). We visited San Cristobal during 2002 and Darwin during 2005, but, due to small sample sizes (n = 2 and 4, respectively), these islands were not included in our analysis. We prepared 2 thin blood smears from each sampled Galápagos dove. Smears were air-dried, fixed in methyl alcohol, and stained with Giemsa. Intensity of infection in blood parasites was quantified from blood smears by counting the number of parasites observed in 10,000 red blood cells for each individual (Valkiūnas, Bensch et al., 2006).

Ectoparasites were quantitatively sampled using the dust-ruffling technique (Walther and Clayton, 1997) by applying pyrethroid insecticide (Zema® Flea and Tick Powder for Dogs, St. John Laboratories, Harbor City, California). Ectoparasites were subsequently stored in vials containing 70% ethanol. Dust-ruffling is the method of choice for ectoparasite quantitative sampling when hosts cannot be killed. This method is known to predict parasite abundances well (Clayton and Drown, 2001).

To determine parasite spatial variation in population parameters, we conducted model III analyses of variance (ANOVAs) (SPSS 14.0, SPSS Inc., Chicago, Illinois) with parasite species as random factor and island as fixed factor. To determine the variation explained among groups, we followed the procedures described in Underwood (1997). Intensity and abundance values were $\log_{10}$ transformed, and prevalence was arcsine$\sqrt{}$ transformed to fulfill assumptions of parametric tests. Prevalence after transformation was not normally distributed and, due to the unbalanced sample sizes, we decided to use a categorical model using the general linear model (glm) procedure in R version 2.4.1 with a quasibinomial error structure (Crawley, 2005).

Intensity of blood parasite infection was analyzed by means of a glm with negative binomial errors (Wilson et al., 1996) using the glm.nb procedure of the MASS library of Venables and Ripley (2002) in R version 2.4.1. We report prevalence, mean, and median intensity, and mean abundance (Bush et al., 1997) for the different island populations of the 4 louse species calculated using the program Quantitative Parasitology 3.0 (Rózsa et al., 2000). We provide 95% confidence intervals estimated by ≥2,000 bootstrap replicates for the different population parameters.

RESULTS

Our results indicate that population parameters of louse parasites are less variable within, than among, parasite species. The amount of variation explained among parasites for intensity was 33.2% ($F_{3,568} = 7.863$, $P = 0.002$), for abundance was 36.7% ($F_{3,648} = 8.16$, $P = 0.002$), and for prevalence was 34.4% ($z = 5.24$, $P < 0.001$). We identified a significant interaction effect between parasite species and island for both intensity and abundance ($F_{14,568} = 6.4$, $P < 0.001$ and $F_{14,648} = 6.7$, $P < 0.001$), which suggests independent parasite–host interactions among island populations. Island alone did not have a significant effect on lice intensity and abundance ($P \geq 0.14$). There was a significant effect of island on parasite prevalence, however, but this was driven by low values from Genovesa Island ($z = -4.83$, $P < 0.001$) represented by the 2 louse species (*C. macrourae* and *P. galapagensis*) infecting doves.

Both prevalence and intensity of *Haemoproteus* sp. infections differed significantly among islands ($z = 2.86$, $P < 0.01$ and $z = 17.0$, $P < 0.001$, respectively). This effect was produced by low values of both prevalence (37%) and intensity (0.009–0.84%) on Genovesa Island ($z = -3.2$, $P < 0.01$ and $z = -6.32$, $P < 0.001$, respectively), which is the same pattern observed for the 2 louse species infecting doves. Intensities of blood parasites ranged from 0.008 to 12.7% (Table I).

DISCUSSION

In the present study, we have analyzed the variation of population parameters of 5 parasite species (4 lice [Insecta: Phthiraptera] and 1 haemosporidian blood parasite [Sporozoa: Haemosporida]) infecting the endemic Galápagos dove (*Z. galapagoensis*) and the endemic Galápagos hawk (*B. galapagoensis*) across their entire geographic range. Our results revealed no significant differences in parasite population parameters among island populations (excluding Genovesa), although a significant island × species interaction indicated independent variation in the populations of both louse species and the haemosporidian parasite among islands. Also, parameter values from different populations of the same parasite species are more similar to each other than to those of other parasite species. Previous studies have shown that infection intensity and abundance of parasites can be considered parasite species-specific traits to the extent that these parameters vary less within than among parasite species, i.e., they are repeatable across parasite populations (Poulin, 2006). Although the variability explained by parasite infection parameters in our system was lower than that reported previously for other parasite systems, i.e., metazoan parasites of fishes and mammals (Arneberg et al., 1997; Krasnov et al., 2006; Poulin, 2006), our analysis corroborates that intensity and abundance are less variable within, than among, parasite species. Previous studies have suggested that prevalence is too variable to be considered a species-specific trait (Arneberg et al., 1997; Poulin, 2006) because it depends on the transmission of parasites among hosts and is sensitive to variable environmental conditions affecting free-living or vectored stages (Poulin, 2006). In contrast, when the life cycle of the parasite species occurs entirely on the host, as in louse species (Insecta: Phthiraptera), environmental variability would be more likely have less effect on the transmission dynamics of the parasite. Accordingly, our results showed that 34% of the variation in prevalence was explained by differences among louse species, which is similar to the 36 and 33% explained for abundance and intensity, respectively. This suggests that intensity, prevalence, and abundance in parasites with direct life cycles are equally variable, in contrast to previous studies conducted on parasites with indirect life cycles, where parasite species explained less variation in prevalence than in intensity and abundance. The results of studies by Arneberg et al. (1997), Poulin and Mouritsen (2003), and Poulin (2006) have shown that repeatability of parasite infection parameters among host species is rather weak, or insignificant, meaning that infection population parameters represent traits of parasite species and not of host species. Longitudinal data are necessary, however, to generalize these patterns across parasite species.

Haemosporidian parasites from Caribbean Islands infecting several host species showed the same pattern of variation in prevalence as the blood parasite studied here, where there was no, or little, island effect, and there was a significant island × species interaction (Apanius et al., 2000; Fallon et al., 2003). However, some parasite lineages presented significant differ-

TABLE I. Estimated population parameters for 5 parasite species (*Columbicola macrourae, Physconelloides galapagensis, Colpocephalum turbinatum, Degeeriella regalis*, and *Haemoproteus* sp.) infecting either the endemic Galápagos dove (*Zenaida galapagoensis*) or the endemic Galápagos hawk (*Buteo galapagoensis*). Values in parentheses are 95% bootstrap confidence intervals estimated by ≥2,000 bootstrap replicates.

Island	Parasite species	Mean intensity*	Median intensity	Mean abundance	Prevalence (%)
Santa Cruz	*Haemoproteus* sp.	0.008–9.4			95 (76–99)
	C. macrourae	20.05 (13.38–31.76)	11 (7–24)	18.3 (11.74–28.22)	91 (72–98)
	P. galapagensis	9.35 (6–16.7)	7 (4–10)	8.13 (5–14.7)	87 (67–96)
Santiago	*Haemoproteus* sp.	0.01–6.9			95 (75–99)
	C. macrourae	31.5 (23.77–40.65)	30 (19–42)	30.33 (22.44–39.19)	96 (81–99)
	P. galapagensis	34.12 (24.56–47.24)	25 (17–41)	31.6 (22.44–44.41)	92 (76–98)
	C. turbinatum	23.85 (18.85–30.18)	17 (10–27)	22.62 (17.48–29)	94 (85–98)
	D. regalis	6.48 (4.52–10)	3 (1–5)	4.69 (3.17–7.22)	72 (59–83)
Española	*Haemoproteus* sp.	0.017–3.8			90 (68–98)
	C. macrourae	16.22 (12.52–22.8)	16 (10–19)	15.8 (11.88–22.46)	95 (79–99)
	P. galapagensis	22 (16.95–33.25)	20.5 (13–25)	18.38 (12.67–29.1)	83 (62–94)
	C. turbinatum	161.63 (75.5–350.5)	55.5 (26–211)	161.3 (75.5–350.5)	100 (63–100)
	D. regalis	55.63 (31.63–87.38)	56.5 (4–82)	55.63 (30–84)	100 (63–100)
Santa Fe	*Haemoproteus* sp.	0.01–1.6			92 (73–98)
	C. macrourae	13.9 (9.73–19)	12 (4–19)	12.75 (8.58–17.46)	91 (73–98)
	P. galapagensis	16 (10.45–23.15)	13.5 (6–20)	13.13 (8.5–20.54)	83 (62–94)
	C. turbinatum	206.46 (120.3–353)	128 (57–171)	206.46 (120.3–353)	100 (77–100)
	D. regalis	21.5 (12.75–32.1)	16 (6–35)	19.85 (11.15–30.54)	92 (65–99)
Genovesa	*Haemoproteus* sp.	0.009–0.84			36 (21–55)
	C. macrourae	10.83 (6.5–17)	9.5 (2–19)	6.19 (3.24–10.86)	57 (35–76)
	P. galapagensis	6.71 (3–10.43)	8 (1–15)	2.24 (0.81–4.62)	33 (15–55)
Fernandina	*C. turbinatum*	62.6 (37.5–115.1)	23.5 (11–50)	62.6 (37.5–115.1)	100 (88–100)
	D. regalis	22.39 (12.6–37.26)	12 (2–19)	18.39 (10.29–32.68)	82 (64–92)
Isabela	*C. turbinatum*	48.56 (32.4–76.52)	27 (23–53)	48.56 (32.4–76.52)	100 (86–100)
	D. regalis	7.79 (5.25–12.1)	5 (3–7)	7.48 (5–11.28)	96 (80–99)
Marchena	*C. turbinatum*	114.88 (65.32–195.32)	46 (16–104)	110.46 (63–184.58)	96 (81–99)
	D. regalis	13.65 (9–20.1)	10 (5–19)	12.1 (7.65–17.62)	88 (69–96)
Pinta	*C. turbinatum*	89.9 (56.13–154.77)	36 (13–63)	87 (52.9–143.5)	96 (82–99)
	D. regalis	20 (11.96–36.18)	5.5 (2–10)	18 (9.45–36.58)	90 (74–97)
Pinzón	*C. turbinatum*	101.8 (69–141.4)	98 (30–166)	101.8 (69–141.4)	100 (70–100)
	D. regalis	30.7 (19.2–43)	28 (10–57)	30.7 (19.2–43)	100 (70–100)
Wolf	*Haemoproteus* sp.	0.02–12.7			100 (88–100)
	C. macrourae	50.5 (38.75–64.75)	39 (28–66)	50.5 (38.75–64.75)	100 (83–100)
	P. galapagensis	60.44 (36.6–92.56)	42 (12–75)	48.35 (28.3–78.65)	80 (57–92)

* *Haemoproteus* sp. intensity was measured as the number of erythrocytes infected in 10,000 examined red blood cells per dove individual for each island. We report the percentage of infected cells of the 10,000 examined as is conventionally done for haemosporidians. Blood parasites were analyzed only in Galápagos doves because to this date, we have not detected haemosporidian infections in endemic hawks (see Parker et al., 2006).

ences across islands (Fallon et al., 2003). The lack of strong island effects is unexpected due to the indirect life cycle of haemosporidians. The presence of a vector may increase the susceptibility of these parasites to environmental conditions, thus making infection parameters more variable across populations. In fact, other studies reported a high degree of heterogeneity among populations of blood parasites infecting the same host (e.g., Freeman-Gallant et al., 2001; Valkiūnas and Lezhova, 2001). The significant interaction effect detected between parasite species and island suggests that infection parameters undergo independent dynamics among islands (see Fallon et al., 2003 for an example of the same pattern in the Caribbean Islands). This could be explained by differences in both biotic and abiotic conditions among islands of the Galápagos, where extreme effects on populations can be observed over short distances (e.g., Wikelski and Trillmich, 1997). For example, the degree of inbreeding within island populations of the Galápagos Hawk positively co-varies with louse abundances and intensities and negatively co-varies with variation in host natural antibody responses (Whiteman et al., 2006). However, because these parameters are less variable within, than among, parasite species, biological features of parasites seem to override local environmental conditions to some degree and maintain fluctuations within narrow species-specific limits (Poulin, 2006). Only if long-term studies confirm the specific nature of parasite population parameters, can we hope to use such parameters as predictive tools for population and community analyses. In particular, when different parasite species are infecting the same host, dynamics can be complex depending on the nature of the interaction, and the outcome is not always intuitive (Schjørring and Koella, 2003; Pedersen and Fenton, 2006). Moreover, using parasite species that infect different host species will allow us to determine if these population parameters can represent host–parasite interaction characters (Poulin, 2006).

Genovesa Island was an extreme sample for parasites infecting doves in that it had a significant impact on the results of our analyses. The fact that Genovesa was sampled 2 yr later than the other southern islands and 1 yr before Wolf Island

might explain its extreme deviation from the other samples due to seasonal or interannual variation in environmental conditions influencing lice or hippoboscid vectors. The range of relative humidity on all the islands during the sampling periods (>64%; data obtained from the Charles Darwin Research Station) was substantially above the level considered to impact lice infecting rock pigeons (*C. livia*), mourning doves (*Zenaida macroura*), and Inca doves (*Columbina inca*), i.e., 40% (Moyer et al., 2002). Thus, relative humidity cannot explain the low abundance of lice on Genovesa Island. In the case of haemosporidian parasites, Sol et al. (2000) have demonstrated that vector abundance is the main factor influencing the spatial variation in prevalence, but not intensity, of blood parasites infecting rock pigeons in populations of western Europe. It is interesting to note that Sol et al. (2000) found a parallel geographic pattern to the one reported here. Prevalence at 4 of 5 localities was 100%, and the single sample with low prevalence (14.8%) had few vectors present in that population. Two experiments confirmed that vector abundance limits parasite transmission, ruling out host individual variation, i.e., susceptibility, as an alternative factor (Sol et al., 2000). Hence, lower abundance of vectors for parasite transmission on Genovesa Island might have caused the low parasite prevalence. Unfortunately, we do not have data to support this hypothesis. Alternatively, the low prevalence observed in Genovesa blood parasites might represent sampling during a low point in a seasonal cycle of parasite prevalence, common to many haemosporidians (e.g., Bensch et al., 2007, but see Fallon et al., 2004 for an example of temporal stability of a community of insular blood parasites). This still would not explain the observation that the 3 parasite species infecting endemic doves showed the same pattern of variation across islands, which suggests that these parasites, despite their different life cycles, might be responding in parallel to the same factors. In addition, this temporal variability further supports the need for longitudinal studies that can confirm the specific nature of parasite population parameters.

Finally, we observed high prevalence and parasitemia of *Haemoproteus* sp. in the dove host (Table I). This suggests that doves are highly susceptible to the local blood parasites. Experimental infections on Hawaiian endemic birds have shown that endemics are highly susceptible and present high parasitemias (>40%) of *Plasmodium relictum* (mitochondrial cytochrome *b* gene lineage GRW4), leading in most cases to host death (Atkinson et al., 1995, 2000; Yorinks and Atkinson, 2000; Atkinson, Dusek, and Lease, 2001; Atkinson, Lease et al., 2001). Species of *Haemoproteus* are often considered to be relatively benign in their avian hosts (Bennett et al., 1993). However, some haemoproteids have been reported to cause diseases in birds (Miltgen et al., 1981; Atkinson et al., 1986, 1988; Cardona et al., 2002) and to affect their fitness (Nordling et al., 1998; Merino et al., 2000; Marzal et al., 2005; Valkiūnas, 2005; Valkiūnas, Zickus et al., 2006). High parasitemias as those presented by the endemic Galápagos dove are uncommon in the wild (Valkiūnas, 2005). There are some examples of wild birds with high parasitemias, but these are the exception rather than the rule (Valkiūnas, 2005). Therefore, experimental studies on the fitness effects of these parasites on the endemic dove are desirable.

Summarizing, our data suggest (1) that infection parameters of louse parasites are, for the most part, homogeneous across islands; (2) that prevalence has the same degree of variation as intensity and abundance in louse parasites; (3) that intensity and prevalence of *Haemoproteus* sp. parasites across island populations are similar, with the exception of Genovesa Island; and (4) that the endemic Galápagos dove is susceptible to the *Haemoproteus* sp. parasite, with prevalence up to 100% in some island populations and with intensities up to 12% in some individuals.

ACKNOWLEDGMENTS

We thank all the people who provided help during the different stages of the field season, particularly G. Buitron, A. Iglesias, I. Rose, J. Bollmer, J. Merkel, and J. Rabenold. We are grateful to the staff of the Charles Darwin Research Station for invaluable help and logistical support during the course of this study, especially P. Robayo. N. Cuevas, and J. Freire who helped with dove sampling at Tortuga Bay, Santa Cruz. Members of Dr. Parker's lab group commented on earlier versions of the manuscript. Two anonymous reviewers provided comments that improved the quality of the manuscript. Permits for sample collection were provided by the Galápagos National Park authorities. Financial support was provided by The Whitney R. Harris World Ecology Center, The Saint Louis Zoo FRC 05-2, and E. Des Lee Collaborative Vision in Zoological Research.

LITERATURE CITED

APANIUS, V., N. YORINKS, E. BERMINGHAM, AND R. E. RICKLEFS. 2000. Island and taxon effects in parasitism and resistance of lesser Antillean birds. Ecology **81:** 1959–1969.

ARNEBERG, P., A. SKORPING, AND A. F. READ. 1997. Is population density a species character? Comparative analyses of the nematode parasites of mammals. Oikos **80:** 289–300.

ATKINSON, C. T., R. J. DUSEK, AND J. K. LEASE. 2001. Serological responses and immunity to superinfection with avian malaria in experimentally-infected Hawaii Amakihi. Journal of Wildlife Diseases **37:** 20–27.

———, ———, K. L. WOODS, AND W. M. IKO. 2000. Pathogenicity of avian malaria in experimentally-infected Hawaii Amakihi. Journal of Wildlife Diseases **36:** 197–204.

———, D. J. FORRESTER, AND E. C. GREINER. 1988. Pathogenicity of *Haemoproteus meleagridis* (Haemosporina: Haemoproteidae) in experimentally infected domestic turkeys. Journal of Parasitology **74:** 228–239.

———, E. C. GREINER, AND D. J. FORRESTER. 1986. Pre-erythrocytic development and associated host responses to *Haemoproteus meleagridis* (Haemosporina: Haemoproteidae) in experimentally infected domestic turkeys. Journal of Protozoology **33:** 375–381.

———, J. K. LEASE, B. M. DRAKE, AND N. P. SHEMA. 2001. Pathogenicity, serological responses, and diagnosis of experimental and natural malarial infections in native Hawaiian thrushes. Condor **103:** 209–218.

———, K. L. WOODS, R. J. DUSEK, L. S. SILEO, AND W. M. IKO. 1995. Wildlife disease and conservation in Hawaii: Pathogenicity of avian malaria (*Plasmodium relictum*) in experimentally infected Iiwi (*Vestiaria coccinea*). Parasitology **111:** S59–S69.

BENNETT, G., M. A. PEIRCE, AND R. W. ASHFORD. 1993. Avian Haematozoa: Mortality and pathogenicity. Journal of Natural History **27:** 993–1001.

BENSCH, S., J. WALDENSTRÖM, N. JONZÉN, H. WESTERDAHL, B. HANSSON, D. SEJBERG, AND D. HASSELQUIST. 2007. Temporal dynamics and diversity of avian malaria parasites in a single host species. Journal of Animal Ecology **76:** 112–122.

BOLLMER, J. L., R. T. KIMBALL, N. K. WHITEMAN, J. H. SARASOLA, AND P. G. PARKER. 2006. Phylogeography of the Galápagos Hawk (*Buteo galapagoensis*): A recent arrival to the Galápagos Islands. Molecular Phylogenetics and Evolution **39:** 237–247.

———, T. SANCHEZ, M. D. CANNON, D. SANCHEZ, B. CANNON, J. C. BEDNARZ, T. DE VRIES, M. S. STRUVE, AND P. G. PARKER. 2003. Variation in morphology and mating system among island populations of Galápagos Hawks. Condor **105:** 428–438.

———, N. K. WHITEMAN, M. D. CANNON, J. C. BEDNARZ, T. DE VRIES, AND P. G. PARKER. 2005. Population genetics of the Galápagos Hawk: Genetic monomorphism within isolated populations. Auk **122:** 1210–1224.

BOOTH, D. T., D. H. CLAYTON, AND B. A. BLOCK. 1993. Experimental demonstration of the energetic cost of parasitism in free-ranging hosts. Proceedings of the Royal Society of London, Series B **253:** 125–129.

BROWN, C. R., M. B. BROWN, AND B. RANNALA. 1995. Ectoparasites reduce long-term survival of their avian host. Proceedings of the Royal Society of London, Series B **262:** 313–319.

BUSH, A. O., K. D. LAFFERTY, J. M. LOTZ, AND A. W. SHOSTAK. 1997. Parasitology meets ecology on its own terms: Margolis et al. revisited. Journal of Parasitology **83:** 575–583.

CARDONA, C. J., A. IHEJIRIKA, AND L. MCCLELLAN. 2002. *Haemoproteus lophortyx* infection in bobwhite quail. Avian Diseases **46:** 249–255.

CLAYTON, D. H., AND D. M. DROWN. 2001. Critical evaluation of five methods for quantifying chewing lice (Insecta: Phthiraptera). Journal of Parasitology **87:** 1291–1300.

———, AND K. P. JOHNSON. 2003. Linking coevolutionary history to ecological process: Doves and lice. Evolution **57:** 2335–2341.

CRAWLEY, M. J. 2005. Statistics: An introduction using R. John Wiley & Sons Ltd., London, U.K., 327 p.

FALLON, S. M., E. BERMINGHAM, AND R. E. RICKLEFS. 2003. Island and taxon effects in parasitism revisited: Avian malaria in the Lesser Antilles. Evolution **57:** 606–615.

———, R. E. RICKLEFS, S. C. LATTA, AND E. BERMINGHAM. 2004. Temporal stability of insular avian malaria parasite communities. Proceedings of the Royal Society of London, Series B **271:** 493–500.

FREEMAN-GALLANT, C. R., K. D. O'CONNOR, AND M. E. BREUER. 2001. Sexual selection and the geography of Plasmodium infection in Savannah sparrows (*Passerculus sandwichensis*). Oecologia **127:** 517–521.

JOHNSON, K. P., B. L. WILLIAMS, D. M. DROWN, R. J. ADAMS, AND D. H. CLAYTON. 2002. The population genetics of host specificity: Genetic differentiation in dove lice (Insecta: Phthiraptera). Molecular Ecology **11:** 25–38.

KRASNOV, B. R., G. I. SHENBROT, I. S. KHOKHLOVA, AND R. POULIN. 2006. Is abundance a species attribute? An example with haematophagous ectoparasites. Oecologia **150:** 132–140.

MARSHALL, A. G. 1981. The ecology of ectoparasitic insects. Academic Press, London, U.K., 459 p.

MARZAL, A., F. DE LOPES, C. NAVARRO, AND A. P. MØLLER. 2005. Malarial parasites decrease reproductive success: An experimental study in a passerine bird. Oecologia **142:** 541–545.

MERINO S., J. MORENO, J. J. SANZ, AND E. ARRIERO. 2000. Are avian blood parasites pathogenic in the wild? A medication experiment in blue tits (*Parus caeruleus*). Proceedings of the Royal Society of London, Series B **267:** 2507–2510.

MILTGEN, F., I. LANDAU, N. RATANAWORABHAN, AND S. YENBUTRA. 1981. *Parahaemoproteus desseri* n. sp.; Gamétogonie et schizogonie chez l'hôte naturel: *Psittacula roseata* de Thailande, et sporogonie expérimentale chez *Culicoides nubeculosus*. Annales de Parasitologie Humaine et Comparée **56:** 123–130.

MOURITSEN, K. N., AND R. POULIN. 2005. Parasites boosts biodiversity and changes animal community structure by trait-mediated indirect effects. Oikos **108:** 344–350.

MOYER, B. R., D. M. DROWN, AND D. H. CLAYTON. 2002. Low humidity reduces ectoparasite pressure: Implications for host life history evolution. Oikos **97:** 223–228.

NORDLING, D., M. ANDERSON, S. ZOHARI, AND L. GUSTAFSSON. 1998. Reproductive effort reduces specific immune response and parasite resistance. Proceedings of the Royal Society of London, Series B **265:** 1291–1298.

PADILLA, L. R., D. SANTIAGO-ALARCON, J. MERKEL, E. MILLER, AND P. G. PARKER. 2004. Survey for *Haemoproteus* spp, *Trichomonas gallinae*, *Chlamidophila pssitaci*, and *Salmonella* spp. in the Galápagos islands Columbiformes. Journal of Zoo and Wildlife Medicine **35:** 60–64.

———, N. K. WHITEMAN, J. MERKEL, K. P. HUYVAERT, AND P. G. PARKER. 2006. Health assessment of seabirds on Isla Genovesa, Galápagos Islands. Ornithological Monographs **60:** 86–97.

PARKER, P. G., N. K. WHITEMAN, AND E. MILLER. 2006. Conservation medicine on the Galápagos islands: Partnerships among behavioral, population, and veterinary scientists. Auk **123:** 625–638.

PEDERSEN, A. B., AND A. FENTON. 2006. Emphasizing the ecology in parasite community ecology. Trends in Ecology and Evolution **22:** 133–139.

POULIN, R. 1998. Evolutionary ecology of parasites: From individuals to communities. Chapman and Hall, London, U.K., 212 p.

———. 1999. The functional importance of parasites in animal communities: Many roles at many levels? International Journal for Parasitology **29:** 903–914.

———. 2006. Variation in infection parameters among populations within parasite species: Intrinsic properties versus local factors. International Journal for Parasitology **36:** 877–885.

———. AND K. N. MOURITSEN. 2003. Large-scale determinants of trematode infections in intertidal gastropods. Marine Ecology Progress Series **254:** 187–198.

RALPH, C. J., G. R. GEUPEL, P. PYLE, T. E. MARTIN, D. F. DESANTE, AND B. MILA. 1996. Manual de métodos de campo para el monitoreo de aves terrestres. USDA General Technical Report PSW-GTR-159. USDA Pacific Southwest Research Station, Forest Service, Albany, California, 44 p.

RÓZSA, L., J. REICZIGEL, AND G. MAJOROS. 2000. Quantifying parasites in samples of hosts. Journal of Parasitology **86:** 228–232.

SANTIAGO-ALARCON, D., AND P. G. PARKER. 2007. Sexual size dimorphism and morphological evidence supporting the recognition of two subspecies in the Galápagos Dove. Condor **109:** 132–141.

———, S. M. TANKSLEY, AND P. G. PARKER. 2006. Morphological variation and genetic structure of Galápagos Dove (*Zenaida galapagoensis*) populations: Issues in conservation for the Galápagos bird fauna. Wilson Journal of Ornithology **118:** 194–207.

SCHJØRRING, S., AND J. C. KOELLA. 2003. Sub-lethal effects of pathogens can lead to the evolution of lower virulence in multiple infections. Proceedings of the Royal Society of London, Series B **270:** 189–193.

SOL, D., R. JOVANI, AND J. TORRES. 2000. Geographical variation in blood parasites in feral pigeons: The role of vectors. Ecography **23:** 307–314.

TYE, A., H. L. SNELL, S. B. PECK, AND H. ADSERSEN. 2002. Outstanding terrestrial features of the Galapagos archipelago. *In* A biodiversity vision for the Galapagos Islands. Charles Darwin Foundation and World Wildlife Fund, Puerto Ayora, Galapagos, Ecuador, 147 p.

UNDERWOOD, A. J. 1997. Experiments in ecology: Their logical design and interpretation using analysis of variance. Cambridge University Press, Cambridge, U.K., 504 p.

VALKIUNAS, T. A. 2005. Avian malaria parasites and other haemosporidia. CRC Press, Boca Raton, Florida, 346 p.

———, S. BENSCH, T. A. LEZHOVA, A. KRIŽANAUSKIENĖ, O. HELLGREN, C. V. BOLSHAKOV. 2006. Nested cytochrome B polymerase chain reaction diagnostics underestimate mixed infections of avian blood haemosporidian parasites: Microscopy is still essential. Journal of Parasitology **92:** 418–422.

———, AND T. A. LEZHOVA. 2001. A comparison of the blood parasites in three subspecies of the yellow wagtail Motacilla flava. Journal of Parasitology **87:** 930–934.

———, T. ZICKUS, A. P. SHAPOVAL AND T. A. IEZHOVA. 2006. Effect of *Haemoproteus belopolskyi* (Haemosporida, Haemoproteidae) on body mass of the blackcap *Sylvia atricapilla*. Journal of Parasitology **92:** 1123–1125.

VAN RIPER III, C., S. G. VAN RIPER, M. L. GOFF, AND M. LAIRD. 1986. The epizootiology and ecological significance of malaria in Hawaiian land birds. Ecological Monographs **56:** 327–344.

VENABLES, W. N., AND B. D. RIPLEY. 2002. Modern applied statistics with S-PLUS, 4th ed. Springer-Verlag, New York, New York, 520 p.

WALTHER, B. A., AND D. H. CLAYTON. 1997. Dust-Ruffling: A simple method for quantifying ectoparasite loads of live birds. Journal of Field Ornithology **68:** 509–518.

WHITEMAN, N. K., S. J. GOODMAN, B. J. SINCLAIR, T. WALSH, A. A. CUNNINGHAM, L. D. KRAMER, AND P. G. PARKER. 2005. Establishment of the avian disease vector *Culex quinquefasciatus* Say, 1823 (Diptera: Culicidae) on the Galápagos Islands, Ecuador. Ibis **147:** 844–847.

———, K. D. MATSON, J. L. BOLLMER, AND P. G. PARKER. 2006. Disease ecology in the Galápagos Hawk (*Buteo galapagoensis*): Host genetic diversity, parasite load and natural antibodies. Proceedings of the Royal Society of London, Series B **273:** 797–804.

———, AND P. G. PARKER. 2004. Effects of host sociality on ectoparasite population biology. Journal of Parasitology **90:** 939–947.

———, D. SANTIAGO-ALARCON, K. P. JOHNSON AND P. G. PARKER. 2004. Differences in straggling rates between two genera of dove lice (Insecta: Phthiraptera) reinforce population genetic and cophylogenetic patterns. International Journal for Parasitology **34:** 1113–1119.

WIKELSKI, M., AND F. TRILLMICH. 1997. Body size and sexual size dimorphism in marine iguanas fluctuate as a result of opposing natural and sexual selection: An island comparison. Evolution **51:** 922–936.

WILSON, K., B. T. GRENFELL, AND D. J. SHAW. 1996. Analysis of aggregated parasite distributions: A comparison of methods. Functional Ecology **10:** 592–601.

WINDSOR, D. A. 1998. Most of the species on earth are parasites. International Journal for Parasitology **28:** 1939–1941.

YORINKS, N., AND C. T. ATKINSON. 2000. Effects of malaria on activity budgets of experimentally infected juvenile Apapane (*Himatione sanguinea*). Auk **117:** 731–738.