3 Evolution of pathogens and parasites on the Galápagos Islands

Patricia G. Parker and Noah K. Whiteman

The history of life on Galápagos has been viewed from the perspective of the islands themselves as ever-changing physical entities (e.g. Geist *et al.* 2002; Munro and Rowland 1996), and from the perspective of the living residents. The highly endemized lineages of birds, mammals, and reptiles have been studied extensively and estimates made for their colonization dates and patterns of diversification, which form the backbone of modern evolutionary studies (see Chapter 2, Valle and Parker). However, the identity, prevalence, impact, and general biology of the parasites and pathogens that inhabit Galápagos species are largely unknown. Here we propose a general framework for considering these organisms that have been ignored, possibly because they are so small. We have begun to appreciate that they, too, are evolving, undergoing adaptive radiations, and invading unoccupied niches. We will leave the applied concerns of surveillance and potential corrective measures for these parasites and pathogens to another chapter (see Chapter 10, Parker and Deem), and explore using them as study species. Going forward, we will use the word "parasite" to refer collectively to viruses, bacteria, and other organisms that live in or on the body of another organism known as the host, from which they draw their nutrition at the expense of the host's fitness.

We have measured the distribution and prevalence of parasites among 26 native and three introduced bird species on 16 islands (Parker *et al.* 2006; Parker 2009b). Across islands, we have documented parasites (1) with long histories with their avian hosts; (2) that have jumped from one host species to another; and (3) that are recent arrivals, some with major impacts. We will discuss each of these categories in turn.

Colonization of parasites with hosts

All biological lineages inhabiting Galápagos are descendants of ancestors that colonized the island system and successfully reproduced. Undoubtedly, there were many unsuccessful colonists. Before human inhabitants complicated the natural life on Galápagos, colonizing parasites arrived exclusively with naturally arriving organisms, such as migratory birds that were blown off course, arthropods caught up in airstreams, or collections of seeds, seedlings, and animals

rafting on the ocean's surface from coastal tectonic disturbances. These animals had parasites. If the colonist did not survive, its parasites likely did not survive, unless they jumped to a new host. Even if the colonist did survive, the survival of the parasite depended on the ecosystem or on the colonist, including abiotic features often critical to parasite survival such as seasonal or diurnal patterns in temperature or humidity. Some disease-causing agents may eliminate individuals not healthy enough to survive the colonization. Because of this filtering, far fewer parasites colonize than hosts do, and biological lineages on islands are exposed to fewer parasites than those on continents.

Parasites that successfully establish with the colonizing host lineage may experience environments dramatically different from their ancestral environment. Some may attempt to colonize a new host if necessary, which may allow them to persist until a better alternative appears, or they may adapt to the new host. Thus, the rate at which new host–parasite relationships are formed accelerates on islands, where simplified communities present few options. We will present one detailed example of the co-evolutionary relationships that can result as both host and parasite adapt to their new environments by focusing on the Galápagos hawk (*Buteo galapagoensis*) and the ectoparasite lineages that accompanied its colonization.

The Galápagos hawk (*B. galapagoensis*) (Figure 3.1) is the apex diurnal predator in Galápagos on the islands of Española, Fernandina, Isabela, Marchena, Pinta, Pinzón, Santa Fe, and Santiago. The hawk is endangered because of its small population size and because hawks were extirpated from three large islands (Floreana, Santa Cruz and San Cristobal) by human persecution. The hawk is famous for its unusual mating system, i.e. cooperative polyandry, in which one female and up to eight males cooperate to rear chicks in territories they occupy year-round (de Vries 1975; Parker 2009a). Ecological and population genetic studies on the hawk (de Vries 1975; Faaborg *et al.* 1995; Bollmer *et al.* 2005, 2006) inspired a detailed comparative study on the ecology and evolutionary genetics of the hawk's co-colonizing parasite community.

B. galapagoensis is among the youngest lineages of endemic vertebrates in Galápagos. Molecular phylogenetic evidence (Riesing *et al.* 2003; Bollmer *et al.* 2006) indicated that *B. galapagoensis* is most closely related to *B. swainsoni* (Swainson's hawk) and that the two recently separated from a common ancestor. Using a molecular clock, the split between *B. galapagoensis* and *B. swainsoni* was estimated to have occurred less than 250,000 years ago (Bollmer *et al.* 2006) and, in fact, *B. swainsoni* is paraphyletic with respect to *B. galapagoensis*. Before the advent of modern phylogenetic methods, *B. swainsoni* was deemed unlikely to be the closest relative of *B. galapagoensis* (de Vries 1975). Interestingly, Theresa Clay, a louse systematist, reported the feather louse *Degeeriella regalis* exclusively from the Galápagos and Swainson's hawks (Clay 1958), suggesting a close relationship between these two bird species that was confirmed by DNA sequence data decades later.

When the ancestors of the Galápagos hawk (*B. galapagoensis*) colonized Galápagos, they brought with them a subset of the parasite community living

Figure 3.1 Photograph of a Galápagos hawk on Isla Fernandina in 2004 (source: J. Rabenold).

within the mainland population (Parker *et al.* 2006; Palma 1994). At least five ectoparasite species have been regularly found on the Galápagos hawk, including a fly *Icosta nigra* (Diptera: Hippoboscidae) that feeds on blood; the feather louse *D. regalis* (Ischnocera: Philopteridae) mentioned above (Figure 3.2); the amblyceran louse *Colpocephalum turbinatum* (Amblycera: Menoponidae); an undescribed feather louse in the genus *Craspedorrhynchus*, which is restricted to the head region of the host; and a skin mite *Myialges caulotoon* that uses *I. nigra* as a vector and parasitizes that fly during part of its lifecycle. Each of these species has been reported from *Buteo swainsoni* or other raptors on the mainland, although each now probably represents a cryptic species unique to the Galápagos hawk. We next discuss their natural history, as well as the ecological and evolutionary interactions between the Galápagos hawk and these parasites.

The lice *Colpocephalum turbinatum* and *D. regalis* differ in several natural history traits, including dispersal ability (Marshall 1981). Transmission is primarily vertical between parents and offspring during brooding in *D. regalis*, while

Figure 3.2 Photograph of the louse *Degeeriella regalis*, which parasitizes the Galápagos hawk.

C. turbinatum primarily transmits horizontally, during interactions such as copulation or fighting. However, both species are capable of reproducing on a single host and are relatively permanent parasites. The ecology of the fly *Icosta nigra* is less studied, but it is highly vagile and is found on several falconiform hosts (Maa 1969). All of these parasites are restricted to *B. galapagoensis* in the Galápagos Islands, but host species specificity is generally inversely related to dispersal abilities (Clayton *et al.* 2004). Given the variation across the three parasite species in dispersal, we predicted that *D. regalis* would have the highest degree of population genetic structure, followed by *C. turbinatum* and *I. nigra*. Due to its vertical transmission, we also predicted that only *D. regalis* would track the host's evolutionary history across the archipelago. We used the evolutionary histories of these parasites to create a hypothesis of the host's evolutionary history in the archipelago (Whiteman and Parker 2005; Nieberding and Olivieri 2007).

This system presented a context in which to study host–parasite co-speciation in its earliest stages. Whereas lice and their hosts have served as excellent examples of co-speciation (Hafner *et al.* 1994), there was very little information on whether macroevolutionary patterns, such as co-speciation of parasites and hosts, were recapitulated at the microevolutionary scale. This was a useful system because (1) island populations of the host are genetically and phenotypically differentiating but the overall lineage is young, and (2) a stable parasite community occurs throughout the distribution of the hawk, and variation in life history and population dynamics allows a comparative approach across parasite species.

The patterns

Gene flow of *B. galapagoensis* is restricted by distance between islands and is specifically restricted by water (Bollmer *et al.* 2005, 2006), as *Buteo* species avoid flying over bodies of water. Several Galápagos hawk populations are among the least genetically variable wild bird populations in the world; each small population is nearly fixed for different alleles, including at the most genetically variable neutral markers (minisatellites). Thus, fixation indices between islands approached unity in some cases, indicating fixed differences among monomorphic island populations. Mitochondrial DNA (mtDNA) sequences are typically used for phylogeographic studies and we sequenced >3 kb of mtDNA from all island populations of the Galápagos hawk and from Swainson's hawks. The few genetically variable sites that we found were typically fixed within island populations; either islands had private haplotypes or there were shared haplotypes between islands with no variation within islands. Thus, for both nuclear and mitochondrial genomes, almost no intra-island variation and only slight inter-island variation existed, consistent with the hawk's small population sizes and recent colonization of the islands, and demonstrating the effectiveness of water as a barrier to gene flow for soaring flight requiring thermals or updrafts.

We genotyped one parasite individual of each species from each hawk individual and sampled multiple hawks (at least nine) on each island, usually >20 hawks per island. Using mitochondrial DNA sequence data for four ectoparasite species (*Colpocephalum turbinatum, Craspedorrhynchus* sp., *D. regalis*, and *Icosta nigra*), we found highly structured populations among islands and significant patterns of isolation by distance (Whiteman *et al.* 2006a, 2007, 2009). However, the patterns of gene flow and population history in the parasite tracked those in the host only in *D. regalis*, which has the highest degree of vertical transmission. Modern coalescent methods (Nielsen and Wakeley 2001) allow decoupling of population structure from population history and allow estimates of migration rates and population divergence times. First, we found that the population structure (using the fixation index) between islands of the louse *D. regalis* was positively and significantly related to that of the host as estimated by nuclear markers, even after correcting for geographic distance between islands. Second, we found that population divergence times of the Galápagos hawk and the louse *D. regalis*, based on mtDNA data, were positively and significantly related after correcting for geographic distance between islands (Figure 3.3). This suggests that *D. regalis* moves with hawk genes in contemporary (ecological) time in the form of recent inter-island gene flow, so the colonization histories of the two species are linked. Divergence time between populations and inter-population migration rate of *D. regalis* lice are correlated, but not in a linear way (Figure 3.4). Why is this? Both migration rate and divergence time between populations of hawks and *D. regalis* lice depend on geographic distance; for migration rate the relationship is asymptotic, as the likelihood of successful migration declines with distance. This suggests that there is still some gene flow at relatively short geographic distances between populations that recently split, but that gene flow is negligible for distant populations that split >20,000 years ago.

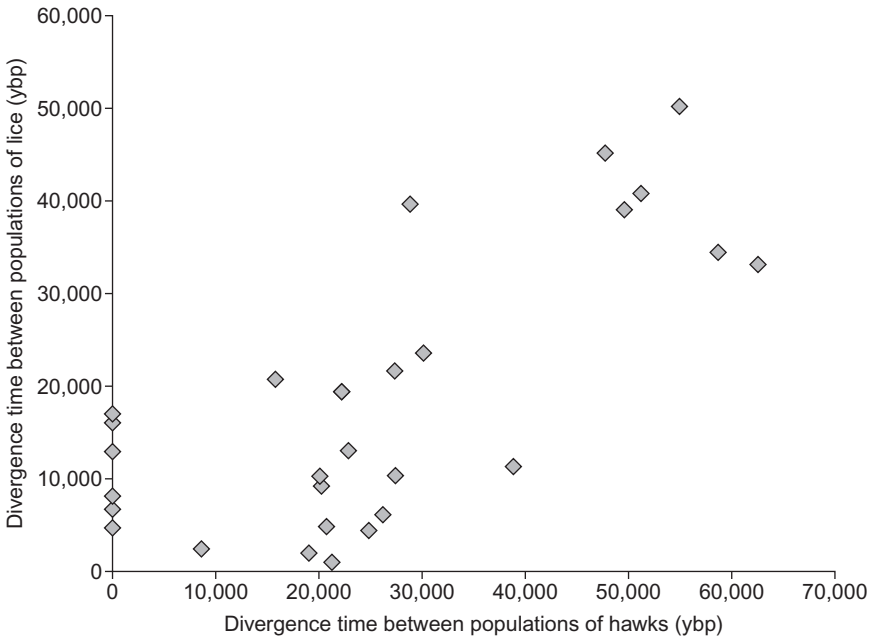

Figure 3.3 Plot of population divergence times for the Galápagos hawk and the louse *Degeeriella regalis*. Data was estimated using the MDIV program. We assumed ten generations/year for the louse and a minimum breeding age of the hawks of three years. We assumed a mtDNA mutation rate of 9×10^{-8} per base pair per generation for both hawks and the lice.

As *D. regalis* individuals move from mother to baby hawk, so lice move from mother to daughter island populations, as new populations of hawks are founded through metapopulation dynamics. This pattern was not found for any of the other parasite species and we speculate that this is due to their higher probability of moving between islands without host genes. We also showed that because *D. regalis* tracked host population structure, its phylogeographic history could be used as a proxy for the host (Whiteman and Parker 2005; Whiteman *et al.* 2007) because there simply was not enough variation in the host's mtDNA dataset to provide information on how four of eight hawk populations were related to one another.

For the two parasites that were not the focus of detailed study we found that while morphological differences between *Craspedorrhynchus* lice on Galápagos and Swainson's hawks were small, they were highly divergent genetically (Whiteman *et al.* 2009). So, while morphological change in parasites is conservative compared to their hosts, neutral genetic change can proceed much more rapidly. Within the Galápagos, our limited sampling showed significant population genetic structure among islands. For the mite *Myialges caulotoon*, we found two cryptic species,

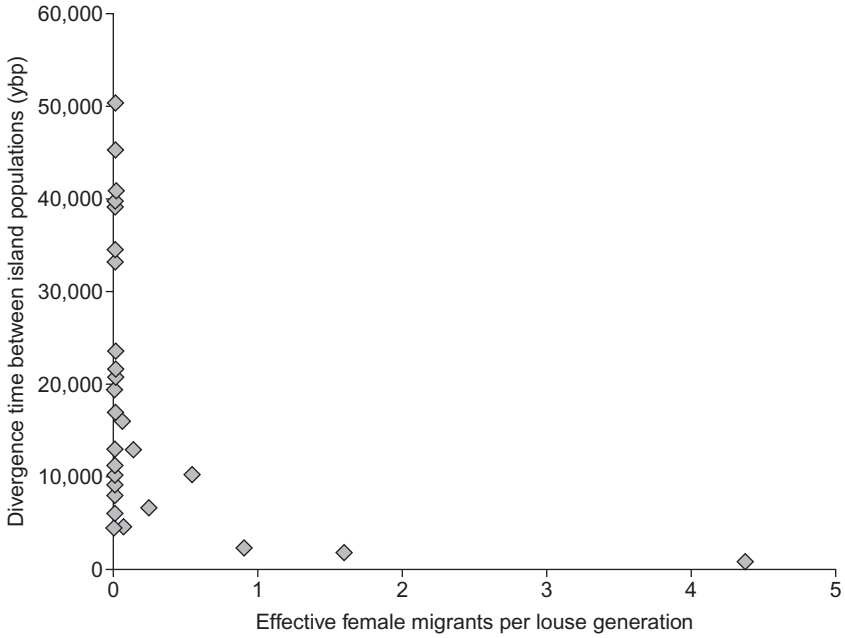

Figure 3.4 Population divergence times versus migration rate of *Degeeriella regalis* lice across eight island populations. Data was estimated using the MDIV program.

one found on the hippoboscid fly species that feeds on flightless cormorants and one found on the hippoboscid species that feeds on hawks. Although hawks and cormorants often occupy the same habitat in the Galápagos, particularly on Fernandina, there is no mitochondrial gene flow between the two mite lineages (Whiteman *et al.* 2006b). In both cases, small morphological changes in parasite lineages mask enormous amounts of neutral genetic divergence.

In summary, the Galápagos hawk and its ectoparasite community provided a rich framework to explore the early stages of host–parasite co-diversification. It showed that ecological and life history traits believed to underlie macroevolutionary patterns, such as co-speciation in hosts and parasites, are important at the microevolutionary scale, providing a link between microevolutionary processes and macroevolutionary patterns. This also underscores the importance of preserving co-evolutionary units in the Galápagos, including native symbionts and parasites, for their own sakes and for what they can tell us about their hosts (Whiteman and Parker 2005).

Host switching

It would normally be difficult to trace the history of the relationship of parasite and host lineages, partly due to variation in host species diversity in time and

space. On islands, we predict this to be simplified by the limited number of host species available.

One example of a host switch is the microfilarid nematode infecting the Galápagos penguin (*Spheniscus mendiculus*) and Galápagos flightless cormorant (*Phalacrocorax harrisi*) (Merkel *et al.* 2007); genetics and morphology reveal the same parasite infecting both host species. The geographic ranges of these two Galápagos endemic birds overlap almost completely along the coasts of Isabela and Fernandina, and they are almost certainly bitten by the same hemophagous arthropods. Microfilaria of similar characteristics have been described from a large number of Phalacrocoracidae (cormorants and allies) species throughout the world (e.g. Cleland and Johnston 1911) but none from many studies of wild penguin populations. Thus, we speculate that the cormorant is the primary host, and that the nematode "jumped" to the penguin through shared arthropod vectors. We have surmised, by having found cormorant blood meals in individual mosquitoes, that the vector may be the black salt-marsh mosquito *Aedes taeniorhynchus* found commonly along these same shores (Bastille *et al.* 2009a; Siers *et al.* 2010).

The susceptibility of Galápagos penguins to this parasite may be attributed to its being the only tropical penguin; other penguins may also be susceptible, but live in climates that may not sustain an annual infection cycle. In addition, we expect the Galápagos penguin to be particularly susceptible due to its lack of exposure to pathogens in general (Travis *et al.* 2006) and its extremely reduced major histocompatibility complex (MHC) variability (Bollmer *et al.* 2007). The MHC is responsible for identifying and presenting pathogen peptides on cell surfaces to cytotoxic "killer" T-cells (Hughes and Nei 1993). Therefore, selection favors diversity across loci, making MHC genes the most genetically variable loci. In a single 157 base pair region of one MHC gene, seven penguin species showed between 16 and 23 variable positions per species, while Galápagos penguins showed only three variable positions, indicating a striking lack of polymorphism that likely leaves them unprotected from many pathogens that continental penguins could resist (Bollmer *et al.* 2007).

The geographic spread of this parasite would result from movement of infected birds or vectors from point to point. Significant geographic clustering of *A. taeniorhynchus* genetic groups suggests limited cross-archipelago movements (Bataille *et al.* 2009a), and the cormorants also show profound sedentariness associated with restricted gene flow (Duffie *et al.* 2009). However, the Galápagos penguin shows low genetic structure, suggesting regular large movements among sites (Nims *et al.* 2008). Taken together, this suggests a parasite of cormorants has undertaken a host shift to include penguins, vectored by their shared mosquito pest *A. taeniorhynchus*, and that the new penguin host is responsible for moving the parasite around the archipelago, where it now occurs in sites favoring populations of *A. taeniorhynchus* (Siers *et al.* 2010). However, although this conjecture may nicely describe the transmission dynamic operating today, it cannot explain its history.

The history must be more complex because both avian hosts arrived on the archipelago millions of years before the mosquito. The most recent common ancestor of the Galápagos penguin and its sister species the Humboldt penguin (*S. humboldti*) is estimated to have lived about four million years ago (4 Mybp; Baker *et al.* 2006), making this the estimated colonization date for what is now the Galápagos penguin. Likewise, the most recent common ancestor of the Galápagos flightless cormorant and its sister species the double-crested cormorant (*P. auritus*) is estimated to have lived ~2 Mybp (Kennedy *et al.* 2009). In contrast, the mosquito *A. taeniorhynchus* has an estimated colonization date only 0.2 Mybp (Bataille *et al.* 2009a). If the nematode itself is a long-term resident of Galápagos, there must have been a different vector species (which may still be involved in transmission); alternatively, the parasite is a more recent arrival than *A. taeniorhynchus*, arriving since the establishment of the penguin, the cormorant, and the mosquito, and it was introduced through an additional bird species not yet sampled. Either way, this means we do not yet fully understand the history of this host switch and the conditions under which it occurred. However, the broadening of the host range from cormorants to successful infections in penguins seems clear.

Recent arrivals

Arrivals of exotic organisms have increased explosively over the last five decades with human population expansion (Snell *et al.* 2002). Although parasites have not been part of analyses of Galápagos threats until recent years, it is certain that their arrival has been accelerated by the same pathways by which invasive plants, vertebrates, and arthropods arrived in recent years. Arrivals of parasites that are novel to the archipelago are a grave concern because of the expectation that their impact on immunologically naïve hosts would be more serious than in a host lineage with an evolutionary history involving continual or periodic interactions with the same parasites. We will develop this idea next.

Dove Haemoproteus species

One example that spans the categories of host switching and recent arrivals is the *Haemoproteus* blood parasite species found in the Galápagos doves (Padilla *et al.* 2004; Padilla and Parker 2006). Although considered relatively nonpathogenic, *Haemoproteus* blood parasites infect a broad range of bird species causing fitness-related consequences, such as reduced reproductive potential or lifespan (e.g. Zehtindjiev *et al.* 2008). An index of how well a population is resisting a particular pathogen is the proportion of individuals infected (prevalence), or the proportion of blood cells containing parasites within an infected individual (intensity). Higher values of these indices tend to be associated with recent infections to which the host has limited immune defense.

Rock pigeons (*Columba livia*) were introduced to Galápagos in several pulses during the 1970s (Harmon *et al.* 1987) and were eradicated by 2002.

The only other columbiform bird in Galápagos is the endemic Galápagos dove, *Zenaida galapagoensis*. Padilla and colleagues (2004) found extremely high *Haemoproteus* prevalences (100% on most islands) in Galápagos doves, with intensities as high as 12% (Santiago-Alarcon *et al.* 2008). Genetic analysis shows that the *Haemoproteus* parasites residing in the Galápagos dove populations comprise diverse lineages that are found in other dove species on mainland South America, with no indication of Galápagos-specific differentiation (Santiago-Alarcon *et al.* 2010). This suggests that the multiple introductions of rock pigeons also brought the diverse dove-specific *Haemoproteus* parasites found in South America, and that the eradication of rock pigeons in 2002 occurred after these lineages had already "jumped" into highly susceptible Galápagos doves.

Avipoxvirus

Recent arrivals also include the avipox virus (Figure 3.5; Thiel *et al.* 2005), *Culex quinquefasciatus* (mosquito vector of avian malaria in Hawaii; Whiteman *et al.* 2005) and most recently *Plasmodium* sp. in 2008 (Levin *et al.* 2009), each of which has significantly altered the disease transmission dynamics within the Galápagos biological community.

Avian pox is a mild to severe disease of birds (Figure 3.5) diagnosed in 278 bird species from 70 families in 30 orders worldwide (van Riper and Forrester 2007), caused by a DNA virus of the genus *Avipoxvirus* of the family Poxviridae; 13 strains have been identified (Francki *et al.* 1991; Tripathy 1993) that vary in virulence and host specificity. The most common recognized strains are fowlpox, pigeonpox, and canarypox viruses. We showed that the poxvirus in chickens in Galápagos was identical to that in chickens elsewhere, while the Galápagos passerines were infected with two variants of canarypox virus that differ significantly from fowlpox virus (Thiel *et al.* 2005); similar results were found in Hawaii (Jarvi *et al.* 2008). The genetic stability of avipox viruses is

Figure 3.5 Cutaneous lesions on Galápagos mockingbirds (*Mimus parvulus*) caused by the avipoxvirus.

poorly understood, and the potential for recombination of poxviruses among the two canarypox and one fowlpox strains known to occur in Galápagos can continue to generate recombinants of unknown pathogenicity. Again, the immunological naïveté of most host lineages in Galápagos may leave them more susceptible to new combinations than their mainland relatives, presenting an evolutionary blank slate for viral recombinants that may not survive on the mainland due to widespread host resistance.

An important overarching, but untested, hypothesis is that lineage age (time in isolation since colonization) correlates positively with susceptibility to new pathogens. This hypothesis predicts that immune function should decline with time without exposure, predicting that the older (earlier colonizing) lineages will be more vulnerable to new diseases. Only two avian lineages in Galápagos have radiated into multiple species: the finches (colonization 2.7 Mybp; Sato *et al.* 1999, 2001) and the mockingbirds (>2 Mybp; Arbogast *et al.* 2006). These two lineages have the highest prevalences and only known deaths from recently arrived Avipoxvirus (Figures 3.5 and 3.6; Gottdenker *et al.* 2008; Jimenez-Uzcategui *et al.* 2007; Parker *et al.* 2006). While these results are consistent with the predictions of our hypothesis, a test will require comparing disease resistance capability and estimated colonization dates across diverse Galápagos avian lineages. Other colonization estimates for terrestrial Galápagos endemic birds are more recent (<0.25 Mybp for Galápagos hawks, Bollmer *et al.* 2006; ~0.8 Mybp for Galápagos flycatchers, Sari *et al.*, in prep.). Endemic avian lineages and their pathogens for which colonization estimates have been made are shown in Table 3.1. Testing this hypothesis will require many more estimates of this kind, and the development of a standardized measure of impact. Moreover, it will require estimates of colonization times for both hosts and parasites, as hosts that brought their parasites with them have not lacked exposure; we predict greatest impact following extensive periods without exposure (Figure 3.6).

There have been no extinctions of bird species in Galápagos, but island-population-level extinctions have increased 100-fold since the arrival of humans 200 years ago (Steadman 2006). Island populations harbor lower levels of genetic variation than their mainland counterparts (Frankham 1998). One possible cause of extinction of genetically depauperate island populations is that they are more susceptible to new diseases (Dobson and May 1986; O'Brien and Evermann 1988). Our studies of genetic diversity in Galápagos birds have revealed extremely low neutral (microsatellites; Duffie *et al.* 2009; Nims *et al.* 2008) and functional (MHC; Bollmer *et al.* 2007) diversity. However, in this chapter we have tried to take the perspective of the parasite in these interactions, pointing out examples of co-evolution of colonizing parasites with their host lineages; examples of rapid host switching; and opportunities for rapid evolution by recombination made possible by the availability of an array of susceptible hosts representing open niches. There are great opportunities to extend studies of parasites within highly co-evolved communities on islands in comparison to those on continents.

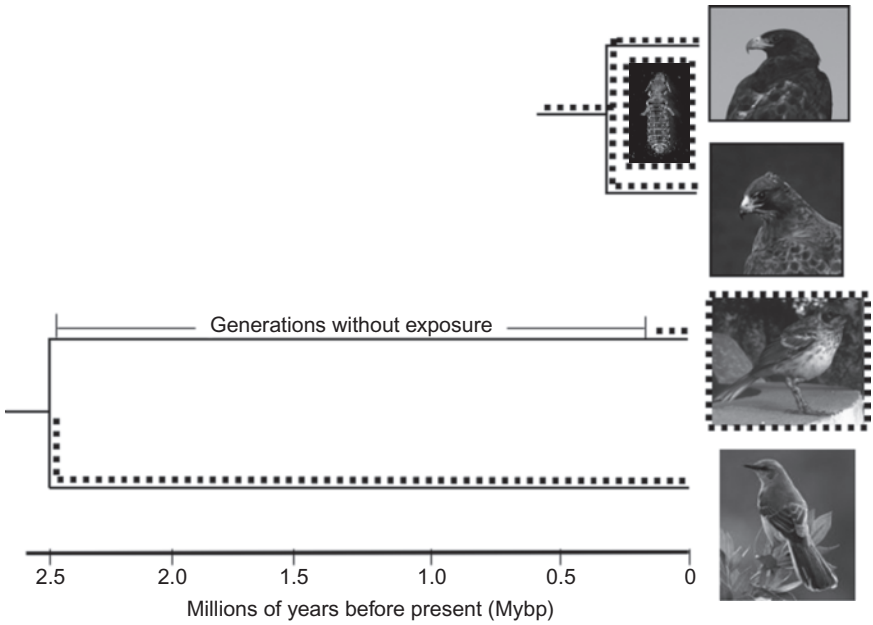

Figure 3.6 Illustration of the hypothesis that time without exposure results in high levels of susceptibility to new pathogens and greater intensity of infection. The lower branches (solid black lines) represent the colonization of the Galápagos >2 Mybp by the ancestral Galápagos mockingbird. The lower lineage remaining on the mainland had continued exposure to the avian poxvirus (dashed line), which "missed the boat" for colonization of Galápagos. The Galápagos mockingbirds on the upper branch were not exposed to this pathogen again until its arrival within the last 120 years and display dramatic infections. The upper branches (solid black) depict the more recent colonization of the archipelago by the hawk that became the Galápagos hawk we know today. Its ectoparasites (dashed lines) came with the colonists and have evolved alongside the hawk (see details in text). The feather louse *Degeeriella regalis* is shown in inset.

Acknowledgments

Our work in Galápagos is facilitated by the Charles Darwin Foundation and permitted by the Galápagos National Park. We particularly appreciate the support of Washington Tapia, Felipe Cruz, and Sonia Cisneros. The University of Missouri–St. Louis and the Saint Louis Zoo have participated in all of this work, a significant part of which was led by Jennifer Bollmer, Diego Santiago-Alarcon, Luis Padilla, and Jane Merkel. Our collaborators in the work on the hawks include Tjitte de Vries and students at Pontificia Católica Universidad del Ecuador, particularly Pablo Sanchez and Paolo Piedrahita, and current UMSL students Jose Luis Rivera and Mari Cruz Jaramillo. On Galápagos, we are frequently joined by Marilyn Cruz in our work on parasites and pathogens, and we

Table 3.1 Galápagos hosts and parasites with estimated colonization dates

Species	*Colonization estimate*	*Reference*
Yellow warbler *Dendroica petechia aureola*	~130 Kybp	Chavez et al., in prep
Galápagos hawk *Buteo galapagoensis*	<250 Kybp	Bollmer *et al.* 2006
Galápagos flycatcher *Myiarchus magnirostris*	~800 Kybp	Sari *et al.*, in prep
Galápagos cormorant *Phalacrocorax harrisi*	2 Mybp	Kennedy *et al.* 2009
Galápagos dove *Zenaida galapagoensis*	2 Mybp	Johnson and Clayton 2000
Galápagos mockingbirds *Mimus* spp.	>2 Mybp	Arbogast *et al.* 2006
Galápagos finches *Geospiza, Camarhynchus, Certhidia* spp.	~2.3 Mybp	Sato *et al.* 1999, 2001; Burns *et al.* 2002
Galápagos penguin *Spheniscus mendiculus*	~4 Mybp	Baker *et al.* 2006
Pathogens or vectors		
Avian malarial blood parasite *Plasmodium* sp.	<20 ybp?	Levin *et al.* 2009
Southern house mosquito *Culex quinquefasciatus*	< 50 ybp	Whiteman *et al.* 2005; Bataille *et al.* 2009b
Avian pox virus Avipoxvirus	<150 ybp	Parker *et al.*, in prep.
Body louse and feather louse of Galápagos hawk *Colpolcephalum turbinatum* *Degeeriella regalis*	~200 Kybp with hawk colonization	Whiteman *et al.* 2007
Black salt-marsh mosquito *Aedes taeniorhynchus*	200 Kybp	Bataille *et al.* 2009a

appreciate having used the facilities under her supervision at the Laboratorio Epidemiologico Patologico y Genetico (LEPG) in Puerto Ayora, as well as the input and collaboration of Virna Cedeno.

References

Arbogast, B.S., S.V. Drovetski, R.L. Curry, P.T. Boag, G. Seutin, P.R. Grant, B.P. Grant and D.J. Anderson. 2006. The origin and diversification of Galapagos mockingbirds, *Evolution* 60:370–382.

Baker, A.J., S.L. Pereira, O.P. Haddrath and K.A. Edge. 2006. Multiple gene evidence for expansion of extant penguins out of Antarctica due to global cooling, *Proc. R. Soc. Lond. B.* 273:11–17.

Bataille, A., A.A. Cunningham, V. Cedeno, L. Patino, A. Constantinou, L.D. Kramer and S.J. Goodman. 2009a. Natural colonization and adaptation of a mosquito species in Galapagos and its implications for disease threats to endemic wildlife, *Proc. Nat. Acad. Sci. USA* 106:10230–10235.

Bataille, A., A.A. Cunningham, V. Cedeno, M. Cruz, G. Eastwood, D.M. Fonseca, C.E. Causton, R. Azuero, J. Loayza, J.D. Cruz Martinez and S.J. Goodman. 2009b. Evidence for regular ongoing introductions of mosquito disease vectors into the Galapagos Islands, *Proceedings of the Royal Society B* 276(1674):3769-3775.

Bollmer, J.L., RT Kimball, N.K. Whiteman, J Sarasola and P.G. Parker. 2006. Phylogeography of the Galápagos Hawk: a recent arrival to the Galápagos Islands, *Molecular Phylogenetics and Evolution* 39:237–247.

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

Bollmer, J.L., N.K. Whiteman, M.D. Cannon, J.C. Bednarz, T. de Vries and P.G. Parker. 2005. Population genetics of the Galápagos Hawk (*Buteo galapagoensis*): Genetic monomorphism within isolated populations, *Auk* 122:1210–1224.

Burns, K.J., S.J. Hackett and N.K. Klein. 2002. Phylogenetic relationships and morphological diversity in Darwin's finches and their relatives, *Evolution* 56:1240–1252.

Clay, T. 1958. Revisions of Mallophaga genera: *Degeeriella* from the Falconiformes. *Bulletin of the British Museum of Natural History, Entomology* 7: 121–207.

Clayton D.H., S.E. Bush and K.P.Johnson. 2004. Ecology of congruence: Past meets present. *Systematic Biology* 53: 165–173.

Cleland, J.B. and T.H. Johnston. 1911. The haematozoa of Australian birds II. *J. Roy. Soc. New South Wales* 45:415–444.

Dobson, A.P., R.M. May. 1986. Disease and conservation, in M.E. Soulé (ed.), *Conservation Biology: The Science of Scarcity and Diversity*, pp. 345–365 (Sinauer Associates: Sunderland, MA).

Duffie, C.V., T.C. Glenn, F.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.

Faaborg, J., P.G. Parker, L. DeLay, T. de Vries, J.C. Bednarz, S. Maria Paz, J. Naranjo and T. Waite. 1995. Confirmation of cooperative polyandry in the Galápagos Hawk (*Buteo galapagoensis*), *Behavioral Ecol. Sociobiol*. 36:83–90.

Fain, A. 1965. A review of the family Epidermoptidae Trouessart parasitic on the skin of birds (Acarina: Sacroptiformes), *Konink VI Acad. Wetensch. Let. Schone Kunst. Belg.* 84 (I-II), 1–176, 1–144.

Francki, R.B., C.M. Fauguet, D.L. Knudson and F. Brown (ed.) 1991. Classification and nomenclature of viruses. Fifth report on the International Committee on the Nomenclature of Viruses, *Archives of Virology*, Supplement 2:94–95 (Springer Verlag: Wien, Austria; New York, NY).

Frankham, R. 1998. Inbreeding and extinction: Island populations, *Conserv. Biol.* 12:665–675.

Geist, D., W.M. White, F. Albarede, K. Harpp, R. Reynolds, J. Blichert-Toft and M.D. Kurz. 2002. Volcanic evolution in the Galapagos: The dissected shield of Volcan Ecuador, *Geochem. Geophys. Geosyst.* 3(10): 1061. doi:10.1029/2002GC000355.

Gottdenker, N.L., T. Walsh, G. Jimenez-Uzcategui, F. Betancourt, M. Cruz, C. Soos, R.E. Miller and P.G. Parker. 2008. Causes of mortality of wild birds submitted to the Charles Darwin Research Station, Santa Cruz, Ecuador from 2002–2004, *J. Wildlife Diseases* 44:1024–1031.

Hafner, M.S., P.D. Sudman, F.X. Villablanca, T.A. Spradling, J.W. Demastes and S.A. Nadler. 1994. Disparate rates of molecular evolution in cospeciating hosts and parasites, *Science* 265:1087–1090.

Harmon, W.H., W.A. Clark, A.C. Hawbecker and M. Stafford. 1987. *Trichomonas gallinae* in Columbiform birds from the Galapagos Islands, *J. Wildlife Diseases* 23:492–494.

Hughes, A.L. and M. Nei. 1993. Evolutionary relationships of the classes of major histocompatibility complex genes, *Immunogenetics* 37:337–346.

Jarvi, S.I., D. Triglia, A. Giannoulis, M. Farias, K. Bianchi and C.T. Atkinson. 2008. Diversity, origins and virulence of Avipoxviruses in Hawaiian forest birds, *Conservation Genetics* 9:339–348.

Jimenez-Uzcategui, G., D.A. Wiedenfeld and P.G. Parker. 2007. Viruela aviar en especies silvestres (Passeriformes) en la isla Santa Cruz, Galapagos, Ecuador, *Brenesia* 67:29–34.

Johnson, K.P. and D.H. Clayton. 2000. A molecular phylogeny of the dove genus Zenaida: Mitochondrial and nuclear DNA sequences, *Condor* 102:864–870.

Kennedy, M., C.A. Valle and H.G. Spencer. 2009. The phylogenetic position of the Galapagos cormorant, *Mol. Phylogenetics Evol.* 53:94–98.

Levin, I.I., D.C. Outlaw, F.H. Vargas, P.G. Parker. 2009. *Plasmodium* blood parasite found in endangered Galapagos penguins (*Spheniscus mendiculus*), *Biological Conservation* 142:3191–3195.

Maa, T.C. 1969. A revised checklist and concise host index of Hippoboscidae (Diptera), *Pacific Insects Monographs* 20:261–299.

Marshall, A.G. 1981. *The Ecology of Ectoparasitic Insects* (Academic Press: London, UK).

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, *J. Parasitology* 93:495–503.

Munro, D.C. and S.K. Rowland. 1996. Caldera morphology in the western Galápagos and implications for volcano eruptive behavior and mechanisms of caldera formation, *J. Volcanol. Geotherm. Res.* 72:85–100.

Nieberding, C. and I. Olivieri. 2007. Parasites: Proxies for host genealogy and ecology? *Trends Ecol. Evol.* 22:156–165.

Nielsen, R. and J. Wakeley. 2001. Distinguishing migration from isolation: A Markov chain Monte Carlo approach, *Genetics* 158: 885–896.

Nims, B., H. Vargas, N. Gottdenker and P.G. Parker. 2008. Low genetic diversity and lack of population structure in the endangered Galápagos penguin (*Spheniscus mendiculus*), *Conservation Genetics* 9:1413–1420.

O'Brien, S.J. and J.F. Evermann. 1988. Interactive influence of infectious disease and genetic diversity in natural populations, *Trends Ecol. Evol.* 3:254–259.

Padilla, L.F. and P.G. Parker. 2006. Monitoring Avian Health in the Galápagos Islands: Current Knowledge, in M. Fowler and R.E. Miller (ed.), *Zoo and Wild Animal Medicine 6: Current Therapy*, pp. 191–199 (Saunders Elsevier: St. Louis, MO).

Padilla, L.R., D. Santiago, J.F. Merkel, R.E. Miller and P.G. Parker. 2004. Survey for *Trichomonas gallinae, Chlamydophila psitacci, Salmonella* spp. and *Haemoproteus* organisms in Columbiformes from the Galápagos Islands, *J. Zoo Wildlife Medicine* 35:60–64.

Palma, R.L. 1994. The identity of *Nirmus obtusus* and other *Quadraceps* species (Phthiraptera: Philopteridae) from Clipperton Island and the Galápagos Islands, J. *Roy. Soc. New Zealand* 24:267–276.

Parker, P.G. 2009a. A most unusual hawk: One mother and several fathers, in T. de Roi (ed.), *Galapagos: Preserving Darwin's Legacy*, pp. 130–137 (Firefly Books: Ontario).

Parker, P.G. 2009b. Parasites and Pathogens: Threats to native birds, in T. de Roi (ed.), *Galapagos: Preserving Darwin's Legacy*, pp. 177–183 (Firefly Books: Ontario).

Parker, P.G., N.K. Whiteman, R.E. Miller. 2006. Perspectives in Ornithology: Conservation Medicine in the Galápagos Islands: Partnerships among Behavioral, Population and Veterinary Scientists. Auk 123: 625–638.

Riesing, M.J., L. Kruckenhauser, A. Gamauf and E. Haring. 2003. Molecular phylogeny of the genus *Buteo* (Aves: Accipitridae) based on mitochondrial marker sequence, Molecular Phylogenetics Evol. 27:328–342.

Riper, C. van, III and D.J. Forrester. 2007. Avian Pox, in N.J. Thomas, D.B. Hunter and C.T. Atkinson (ed*.), Infectious Disease of Wild Birds*, pp. 131–176 (Blackwell Publishing: Oxford, UK).

Santiago-Alarcon, D., D.C. Outlaw, R.E. Ricklefs and P.G. Parker. 2010. Phylogenetic relationships of haemosporidian parasites in New World Columbiformes, with emphasis on the endemic Galapagos dove, *International J. Parasitology* 40:463–470.

Santiago-Alarcon, D., N.K. Whiteman, R.E. Ricklefs, P.G. Parker and G. Valkiunas. 2008. Patterns of parasite abundance and distribution in island populations of Galapagos endemic birds, *J. Parasitology* 94:584–590.

Sato, A., C. O'hUigin, F. Figueroa, P.R. Grant, B.R. Grant, H. Tichy and J. Klein. 1999. Phylogeny of Darwin's finches as revealed by mtDNA sequences, *Proc. Natl. Acad. Sci. USA* 96:5101–5106.

Sato, A., H. Tichy, C. O'hUigin, P.R. Grant., B.R. Grant and J. Klein. 2001. On the origin of Darwin's finches, *Mol. Bol. Evol.* 18:299–311.

Siers, S., J.F. Merkel, A. Bataille, F.H. Vargas and P.G. Parker. 2010. Ecological correlates of microfilarial prevalence in endangered Galapagos birds, *J. Parasitology* 96:259–272.

Snell, H.L., A. Tye, C.E. Causton and R.B. Bensted-Smith. 2002. Current status of and threats to the terrestrial biodiversity of Galapagos, in B. Bensted-Smith (ed.), *A biodiversity vision for the Galapagos Islands*, pp. 30–47 (Charles Darwin Foundation and World Wildlife Fund: Puerto Ayora, Galapagos).

Steadman, D.W. 2006. *Extinction and Biogeography of Tropical Pacific Birds* (University of Chicago Press: Chicago, IL).

Thiel, T., N.K. Whiteman, A. Tirape, M.I. Maquero, V. Cedeno, T. Walsh, G. Jimenez and P.G. Parker. 2005. Characterization of Canarypox-like Viruses Infecting Endemic Birds in the Galápagos Islands, *J. Wildlife Disease* 41:342–353.

Travis, E.K., F.H. Vargas, J. Merkel, N. Gottdenker, R.E. Miller and P.G. Parker. 2006. Hematology, serum chemistry, and serology of the Galápagos Penguin in the Galápagos Islands, Ecuador, *J. Wildlife Diseases* 42:625–632.

Tripathy, D.N. 1993. Avipox viruses, in J.B. McFerran and M.S. McNulty (ed.), *Virus infections of birds*, pp. 5–15 (Elsevier: New York, NY).

Vries, T. de. 1975. The breeding biology of the Galapagos Hawk, *Buteo galapagoensis*, *Le Gerfaut* 65:29–57.

Whiteman, N.K., V.S. Dosanjh, R.L. Palma, J.M. Hull, R.T. Kimball, P. Sánchez, J.H. Sarasola and P.G. Parker. 2009. Molecular and morphological divergence in a pair of closely related birds and their ectoparasites, *J. Parasitology* 95:1372–1382.

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.

Whiteman, N.K., R.T. Kimball and P.G. Parker. 2007. Co-phylogeography and comparative population genetics of the Galápagos Hawk and three co-occurring ectoparasite species: Natural history shapes population histories within a parasite community, *Molecular Ecology* 16:4759–4773.

Whiteman, N.K., K.D. Matson, J.L. Bollmer and P.G. Parker. 2006a. Disease ecology in the Galápagos Hawk (Buteo galapagoensis): Host genetic diversity, parasites, and natural antibodies, *Proc. Roy. Soc. London B* 273:797–804.

Whiteman, N.K. and P.G. Parker. 2005. Using parasites to infer host population history: A new rationale for parasite conservation, *Animal Conservation* 8:175-181.

Whiteman, N.K., P. Sanchez, J.F. Merkel, H. Klompen and P.G. Parker. 2006b. Cryptic host specificity of an avian skin mite (Epidermoptidae) vectored by louse flies (Hippoboscidae) associated with endemic Galápagos bird species, *J. Parasitology* 92:1218–1228.

Zehtindjiev, P., M. Ilieva, H. Westerdahl, B. Hansson, G. Valkiūnas and S. Bensch. 2008. Dynamics of parasitemia of malaria parasites in a naturally and experimentally infected migratory songbird, the great reed warbler *Acrocephalus arundinaceus*, *Experimental Parasitology* 119:99–110.