Chapter 6
Host-Switching: How It Starts

Maricruz Jaramillo and José Luis Rivera-Parra

Abstract A parasite depends, during its entire life or at least part of it, on other organisms, but parasites often "jump" from one host species to another and may be able to colonize new host species. The chances of parasite spillover, the first step in such a host switch, may be influenced by factors such as the local ecosystem, community composition, and modes of transmission, among others. In Galapagos, for example, seabirds show a spatially clustered community, with several species that are related and/or nest in close proximity, a seemingly perfect scenario for host switching. However, only one instance of a straggling ischnoceran louse and larva (indicating successful reproduction on the new host) was found on a different host species, suggesting that the specifics of ectoparasite body size and host feather interbarbular space may prevent lice from readily switching hosts. On the other hand, the haemosporidian parasite, *Haemoproteus multipigmentatus*, of the Columbiform-specific sub-genus *Haemoproteus*, was found in significant numbers of Galapagos passerines. The spillover events occur where Galapagos doves (*Zenaida galapagoensis*), a widespread endemic, are present or abundant enough; however, there is no evidence of parasite development in the passerine birds. Thus, the Galapagos archipelago provides an exceptional host-parasite system to investigate details of parasite spillover and its implications for host health and survivorship.

Keywords Avian health • Galapagos • Host switching • Host-parasite interactions • Spillover

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6.1 Spillover

When a parasite finds itself on a host individual that is not of its typical host species, we may call this a host switch. If successful, it could lead to speciation in the parasite lineage, a process that could increase biodiversity in healthy ecosystems (Hudson et al. 2006). When a parasite switches hosts, it changes branches in the tree of life, occupying a different niche and potentially expanding its range. If the isolation from the previous host is relatively strong, it can lead to genetic differentiation and speciation (Ogden and Thorpe 2002; Johnson et al. 2002a, b; Clayton and Johnson 2003; Schluter 2009; Feder et al. 2012). But parasites can jump to other hosts and not establish a viable population. It can be a single individual that jumps and cannot reproduce alone, or the intricacies of host-parasite interaction may hamper establishment of the parasite on the new host; these more temporary relationships are called straggling events (Rozsa 1993; Paterson and Gray 1997; Norton and Carpenter 1998; Ricklefs et al. 2004).

Straggling events may be the starting point of a successful host switch. Parasites that continuously end up in a different host are more likely to end up in enough numbers to establish a population, competing with the native parasites and even evolve to "tweak open" the lock of the host immune system or defense mechanisms (Rozsa 1993; Ricklefs et al. 2004). A major challenge when studying host switching has been to draw a line between a straggling or a host switching event (Rozsa 1993; Whiteman et al. 2004). For the purposes of this chapter, we will define a host switch as having occurred when there is evidence of reproduction (or reproductive stages) in the novel or atypical host.

The chances of parasite spillover, from one host species to another one, are influenced by various ecological and life history traits. Aspects such as niche similarity among host species, modes of transmission, and vector dietary preferences are only a few of the most relevant ones (Rozsa 1993; Johnson et al. 2002a, b; Clayton and Johnson 2003; Whiteman et al. 2004; Bush et al. 2006). We will continue to discuss in detail these and other aspects that may explain the spillover (or straggling) events observed in Galapagos and the ecological and biological factors that explain them. Galapagos is a great laboratory to understand parasite spillover.

6.1.1 Host Community Structure and Transmission

Host-parasite interactions are present throughout the tree of life. The specifics of those interactions depend on the specific host and parasite species involved (Price et al. 2003; Koh et al. 2004; Whiteman and Parker 2005). For example, avian malaria parasites interact directly with the host immune system and need very specific surface proteins to infect the host red blood cells (Valkiūnas 2004). Moreover, these parasites are vector-borne, so they also need a set of proteins that let them infect the arthropod

vector, moving through different organs and reproductive phases. In contrast, ectoparasitic avian lice are directly transmitted and barely interact with the host immune system; what they need to be worried about is host preening, which is the main defense mechanism of the host (Price et al. 2003; Whiteman and Parker 2005).

Community composition, its phylogenetic clustering, and similarity of niches among hosts and potential host species define the chances for spillover (Johnson et al. 2003; McCoy et al. 2005; Whiteman and Parker 2005; Hughes et al. 2007; Whiteman et al. 2007). Communities of species that are very distinct phylogenetically or for which related species have very divergent niches, present lower opportunities for parasites to colonize a novel host (Ricklefs et al. 2004). Galapagos shows a very clustered community, with adaptive radiations in the Darwin finches (Lamichhaney et al. 2015), and several species of seabirds that are related and/or nest in close proximity and have significant ecological and social interactions (Baião and Parker 2012; Rivera-Parra et al. 2014).

Having a clustered community is not the only requirement; there must also be real chances for host switching. For example, ectoparasitic lice cannot survive long off the body of the host (Price et al. 2003), so the typical and potential host species must interact physically for the lice to jump from one to the other (Rivera-Parra et al. 2014). Vector-borne parasites such as *Haemoproteus* or *Plasmodium* depend on the dietary preference of the biting insect vector to move across hosts (Valkiūnas 2004; Njabo et al. 2011). Thus, even when there are many potential hosts that have similar niches, there must be opportunities for host switching, through generalist vectors or physical interactions. Depending on the specifics of the transmission mode, there might be even bigger challenges not only for host switching but for parasite survival. For example, if an infected host colonizes a novel environment but there is no competent vector or other competent hosts for the parasite, then the parasite will die off (Telfer and Bown 2012; Inbar et al. 2013; Levin et al. 2013).

Therefore, the way parasites are transmitted across individuals (and potentially across species) is crucial for understanding parasite diversity, specificity, evolutionary history, and chances for spillover (Whiteman and Parker 2005; Rivera-Parra et al. 2015). Roughly, parasites can be classified depending on their transmission as either directly transmitted or vector-borne.

6.1.1.1 Directly Transmitted

Parasitism is a complicated way of life. Parasites depend, during their entire life cycle or part of it, on another organism (Price et al. 2003; Valkiūnas 2004). This makes them vulnerable to stochasticity (e.g., the death of a host before transmission) and even co-extinction (Koh et al. 2004; Whiteman and Parker 2005). Parasites are said to be directly transmitted when they do not rely on other organisms to be vectored from one host to another (Price et al. 2003). Thus, parasites use their own means or their hosts' habits to colonize another individual.

Directly transmitted parasites can take advantage of social interactions to be transmitted (Whiteman et al. 2006). They can be transmitted among independent individuals, which is called horizontal transmission, or they can be transmitted from parents to offspring (vertical transmission; Clayton et al. 1992, Whiteman and Parker 2004). Parasites that are more mobile and/or inhabit social host species or hosts that interact regularly and directly with other potential host species are more likely to spread to novel hosts.

The Galapagos hawk (*Buteo galapagoensis*) is an endemic and diurnal predator of the Galapagos Archipelago (Fig. 6.1). As predators, they interact intimately with their prey, and there is evidence of parasite spillover from their prey to the hawks. Whiteman et al. (2004) found Galapagos dove (*Zenaida galapagoensis*) and introduced goat (*Capra hircus*) ectoparasites on a Galapagos hawk. As the authors suggest, this seems like an example of a parasite straggling. Thus, parasites will survive only for a short period of time and not establish a viable population. The intricacies at play in a host-parasite interaction, such as specific defense mechanisms (like preening) or the host immune system, may prevent a successful colonization, but represent how the host habits create opportunities for parasite spillover.

Fig. 6.1 Group of the polyandrous Galapagos hawk (*Buteo galapagoensis*) and their potential prey, the Galapagos dove (*Zenaida galapagoensis*)

6.1.1.2 Indirectly Transmitted

Indirect transmission of parasites usually brings another player into action, the vector. Although this complicates the parasite's life cycle, it also enhances the possibility of transmission as direct contact between hosts is no longer necessary. In the past few decades most emergent infectious diseases that involved wildlife were exotic to the environment in which the epidemic occurred (Daszak et al. 2000; Dobson and Foufopoulos 2001). Even though we would generally expect host-parasite introductions to be greater for parasites with direct life cycles, various co-introduction studies involve parasites with indirect life cycles, the majority of which resulted in host-switches to native hosts (Lymbery et al. 2014). Once parasites are introduced, the potential for pathogen spillover will depend on the host community structure and the presence or co-introduction of alternative hosts or vectors (e.g., Warner 1968; van Riper et al. 1986; Gaither et al. 2013; Novak and Goater 2013).

Spillover occurs when the disease dynamics in one or multiple host populations are driven by transmission from a reservoir host in which the pathogen is highly prevalent, regardless of the mode of transmission (Daszak et al. 2000; Power and Mitchell 2004). Introduced species are often the reservoirs of these pathogens in naive native communities (Lymbery et al. 2014). For this reason, various research efforts in Galapagos have focused on assessing the risk that the poultry industry or backyard chickens pose to endemic wild birds, as introduced chickens may serve as reservoirs for important infectious diseases (Gottdenker et al. 2005; Soos et al. 2008; Deem et al. 2012).

The first evidence of possible spillover of disease from domestic to wild birds in Galapagos was found during a study that assessed pathogens and parasites in chickens and wild birds on Floreana Island, to determine disease risks prior to a possible re-introduction of the endangered Floreana mockingbird (*Mimus trifasciatus*, see Fig. 4.4) (Deem et al. 2012). Thirty percent of chickens presented antibodies against paramyxovirus-1 and 11.3% presented antibodies against adenovirus-2, while for wild birds, prevalence was much lower with only 3% presenting antibodies against paraxymovirus-1 and 2.4% against adenovirus-2, suggesting the direction of transmission from chickens to wild birds. Paramyxovirus-1 and adenovirus-2 are viruses that are transmitted via airborne particles (direct) but transmission can also occur from contaminated surfaces or material or even from fecal matter (indirect). Thus, the potential for indirect transmission of these viruses may increase the risk of transmission from introduced chickens to the endemic wildlife.

Another example of possible spillover from an introduced species to the endemic Galapagos avifauna involves the common protozoan, *Toxoplasma gondii*. Exposure to *T. gondii* has been shown in Galapagos penguins (*Spheniscus mendiculus*) and Flightless cormorants (*Phalacrocorax harrisi*) (Deem et al. 2010). Prior to this study, there had been a single report of a domestic chicken infected with *T. gondii* (Gottdenker et al. 2005). Introduced cats (*Felis catus*) are likely the major reservoir for infection as they are the only host in which sexual reproduction of the parasite is known to occur. Domestic cats on Isabela have been found to have an antibody prevalence of 65% (Levy et al. 2008). Furthermore, it appears that the spillover of

disease occurs not only on islands where cats are present, like Isabela, but also on Fernandina, one of the most pristine islands in the archipelago where there are no introduced cats (Deem et al. 2010). Plausible explanations for this observation include but are not limited to: widespread movement of Galapagos penguins (Nims et al. 2008) and dispersal of oocysts by ocean currents (Dubey 2004); attempts to evaluate this mode of dispersal in Galapagos have not been conclusive (Verant et al. 2013). Although *T. gondii* infections are common in many avian species, pigeons and canaries can be severely affected and it can even cause blindness (Dubey 2002). Moreover, *Toxoplasma gondii* poses a significant threat to isolated island avifauna as it has been associated with mortality in several Hawaiian endemics (Work et al. 2000, 2002).

Native species can also become reservoirs for introduced pathogens (Woodworth et al. 2005). In Galapagos, this appears to be the case of the Haemosporidian parasite *Haemoproteus multipigmentatus* and the endemic Galapagos dove (*Zenaida galapagoensis*) (Santiago-Alarcon et al. 2008). *H. multipigmentatus* belongs to the subgenus *Haemoproteus*, thought to be transmitted by hippoboscid flies and previously recorded only in columbiform birds (Valkiūnas 2004; Valkiūnas et al. 2010). Two other species within the subgenus *Haemoproteus* have since been described in Galapagos hosts, *H. iwa* from frigatebirds and vectored by *Olfersia spinifera* (Levin et al. 2011), and *H. jenniae* from swallow-tailed gulls (Levin et al. 2012) (Fig. 6.2); these two species form a deeply divergent sister clade to the hippoboscid-transmitted dove-specific species.

H. multipigmentatus is highly prevalent in Galapagos doves (Santiago-Alarcon et al. 2008) and is transmitted between doves by the endemic hippoboscid fly (*Microlynchia galapagoensis*) (Valkiūnas et al. 2010). *H. multipigmentatus* seems to have a wide distribution in the American continent as it has been found in Mexico, Guatemala, and Peru (Valkiūnas et al. 2010). A phylogenetic study of *H. multipigmentatus* recovered from Galapagos doves and from continental doves suggested that there were multiple events associated with the colonization of the parasite (Santiago-Alarcon et al. 2010, Chap. 7 this volume). The pathogen was likely brought to the Galapagos Islands via domestic rock pigeons (*Columba livia*) which were repeatedly introduced to the archipelago (Harmon et al. 1987; Padilla et al. 2004). Furthermore, sampling of nine pigeons, before they were completely eradicated in 2002, revealed that several individuals were in fact infected with *H. multipigmentatus* (Levin and Parker pers. comm.).

The first report of *Haemoproteus* (*Haemoproteus*) infection in a passerine bird was by Sari et al. (2013), during an effort to elucidate the origin of parasites infecting Galapagos flycatchers, *Myiarchus magnirostris*. Five flycatchers from Santa Cruz Island were infected with *Haemoproteus multipigmentatus* out of a total of 254 Galapagos flycatchers sampled from six different islands in the archipelago. The presence of *H. multipigmentatus* in these birds was detected by molecular methods and examination of the infected blood smears presented no evidence of parasite development (gametocytes were absent), indicating that Galapagos flycatchers may not be competent hosts. Thus, it appeared that the parasites detected in *M. magnirostris* were acquired in the Galapagos Islands by spillover from their reservoir host, the Galapagos dove (Sari et al. 2013).

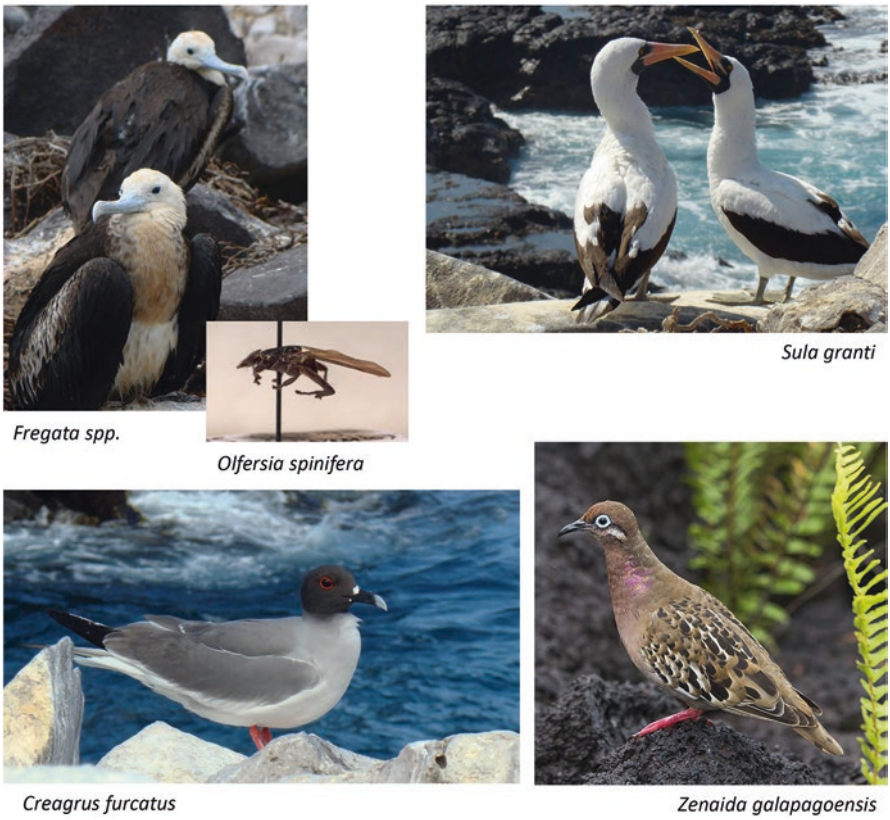

Fig. 6.2 Galapagos hosts reported infected with *Haemoproteus* (*Haemoproteus*) spp. (Photo credits. *Olfersia spinifera*: Manuel Mejía; *Zenaida galapagoensis*: Jeisson Andrés Zamudio; *Fregata* spp., *Sula granti*, *Creagus furcatus*: Maricruz Jaramillo)

An ongoing large-scale avian disease survey that began in 2001 detected *Haemoproteus* PCR signals in passerines but they were not reported because the numbers were usually too small and too scattered to determine the cause of infection (Parker and collaborators, unpublished data). Infected species included a small tree finch (*Camarhynchus parvulus*), a yellow warbler (*Setophaga petechia*), a large cactus finch (*Geospiza conirostris*), seven common cactus finches (*Geospiza scandens*), three small ground finches (*Geospiza fuliginosa*), two large ground finches (Geospiza magnirostris), four Galapagos flycatchers and a vegetarian finch (*Platyspiza crassirostris*) on the islands of Santa Cruz, Isabela, Santiago, Floreana, and Pinta in a span of 6 years.

The most recent avian haemosporidian survey in the archipelago sampled 2254 individuals of 19 endemic and three introduced bird species along an altitudinal gradient in the islands of Isabela, Santa Cruz and Santiago (Jaramillo et al. 2017). The survey revealed 90 PCR positive birds in all years (2013–2015), 89 of which

occurred on Santiago. Of these, 31 were Galapagos doves, and the other 58 included small ground finches, medium ground finches (*G. fortis*), large ground finches, a large tree finch (*C. psittacula*), Galapagos mockingbirds, and yellow warblers. These clusters of PCR-positive birds appeared only in locations where doves were also captured and all captured doves were infected (100% prevalence) (Fig. 6.3). Infection intensity in Galapagos doves was generally high, averaging 357 (±307) gametocytes per 10,000 erythrocytes, whereas Galapagos passerines presented no evidence of intraerythrocytic development. This suggests the role of Galapagos doves as reservoir hosts for *Haemoproteus multipigmentatus* in multiple spillover events (Jaramillo et al. 2017).

Although *Haemoproteus* infections have been considered to be relatively benign to their bird hosts (Bennett et al. 1993) or even positive for their lifetime reproductive success (Zylberberg et al. 2015), numerous field and experimental studies have shown the negative effects these parasites can have on birds' fitness (Valkiūnas 2004; Marzal et al. 2005; Møller and Nielsen 2007; Atkinson 2008) and have also been found to be lethal in adapted (Earle et al. 1993) and non-adapted birds (Atkinson et al. 1988; Cardona et al. 2002; Donovan et al. 2008; Olias et al. 2011; Cannell et al. 2013).

Some scientists propose that pathogen spillover from single key host species may be the main source of the parasitic fauna in evolutionarily recent bird communities

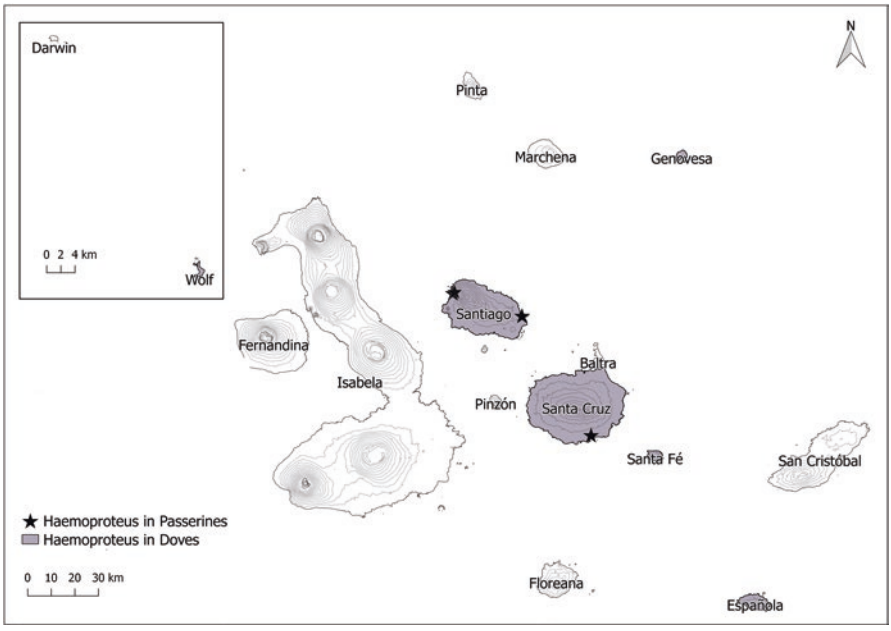

Fig. 6.3 Map of the Galapagos Islands indicating islands (in *grey*) where a study found *Haemoproteus multipigmentatus* in 100% of sampled doves, and sites (*stars*) where it has been found in passerine birds. Galapagos doves are present in all major islands of the archipelago and show high infection at all sampled sites (Adapted from Jaramillo et al. 2017)

(Hellgren et al. 2011). We have reviewed a few examples in which introduced species are likely to be the source for various pathogenic agents found in wild birds in Galapagos (Deem et al. 2010, 2012), and an example of a vector-borne parasite that was likely brought to Galapagos by an introduced dove and whose current reservoir is a widespread Galapagos endemic (Jaramillo et al. 2017). The presence of native alternative hosts and vectors has enabled the spillover of disease to a native community of susceptible hosts. Spillover is the preceding step to host switching, but even if a host switch never occurs, there still might be important effects for the non-adapted hosts and the possibility that these parasites are in turn shaping their hosts' population dynamics.

6.2 Opportunities for Host-Switching

The chances of moving from one host species to another depend on the opportunities the local ecosystem presents. For a successful host-switch to happen there has to be a suitable potential host. This means that the host needs to offer similar "environmental" conditions and similar defense mechanisms (that can be dealt with in a similar way as in the typical host). In addition, there should be enough chances for a parasite to be transmitted across species, so if the parasite is vector-borne, the vector should be more generalist; if the parasite is directly transmitted, the hosts must interact in some way (Whiteman et al. 2004; Whiteman et al. 2005; Rivera-Parra et al. 2015).

Communities that share phylogenetically related species may be more susceptible to host switching, assuming that related hosts maintained similar mechanisms against parasites and share similar niches (Johnson et al. 2003). Niche similarity is relevant because it means more interaction among species. For example, in Galapagos, Darwin's finches are closely related phylogenetically and share the same ectoparasitic lice species (*Brueelia interposita* and *B. chelydensis*; Price et al. 2003). Thus, it seems likely that populations of these two parasites on their hosts have not been sufficiently isolated to allow speciation.

6.2.1 Mixed Species Colonies of Seabirds and Their Lice

Among the rich seabird fauna of the Galapagos archipelago, there are two frigatebirds, magnificent (*Fregata magnificens*) and great (*F. minor*), and three species of boobies, Nazca (*Sula grantii*), blue-footed (*S. nebouxii*), and red-footed (*S. sula*). These five species of seabirds present specific local combinations and degree of spatial overlap. Each seabird species has one specific species of ischnoceran louse, the frigatebirds share an amblyceran louse (*Fregatiella aurifasciata*) and Nazca and blue-footed boobies share another amblyceran (*Eidmaniella albescens*) (Fig. 6.4). In this context, where hosts species nest in close proximity and the lice are

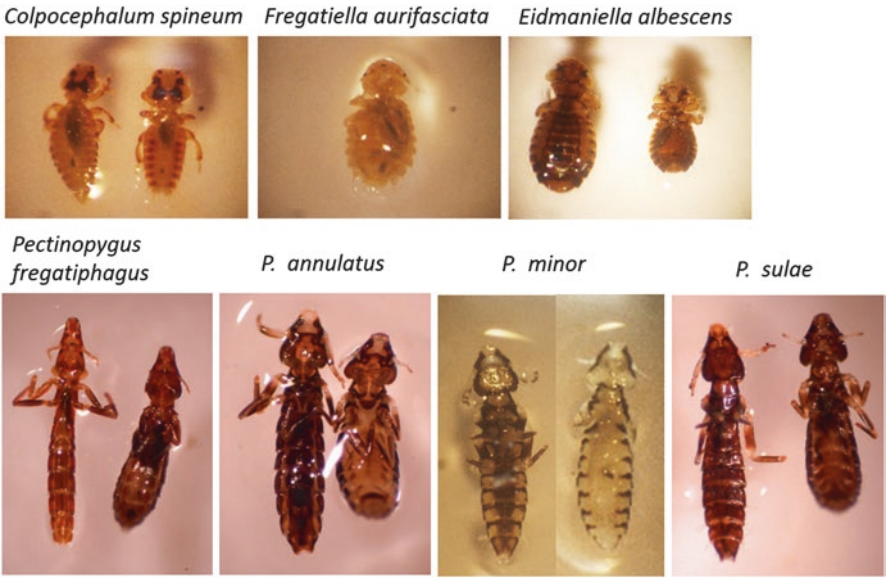

Fig. 6.4 Ischnoceran and Amblyceran lice infecting the three species of boobies and two frigatebirds from the Galapagos Islands. Amblycerans: *Colpocephalum spineum* (commonly infects Magnificent frigatebirds), *Fregatiella aurifasciata* (ex. Magnificent and Great frigatebirds), *Eidmaniella albescens* (ex. Blue-footed and Nazca boobies). Ischnocerans: *Pectinopygus fregatiphagus* (ex. Magnificent frigatebird), *P. annulatus* (ex. Nazca booby), *P. minor* (ex. Blue-footed booby) and *P. sulae* (ex. Red-footed booby)

phylogenetically related or shared, we expected to find a high degree of host switching (Rivera-Parra et al. 2014).

Fregatiella aurifasciata, which was thought to be a single species, showed evidence of genetic differentiation, suggesting lineage sorting, even on the islands where great and magnificent frigatebirds nest together (Rivera-Parra et al. 2015). Similarly, *Eidmaniella albescens* shows two distinct lineages, one in Nazca boobies and the other in blue-footed boobies (Rivera-Parra et al. 2015). Amblyceran lice tend to be highly mobile and transmit horizontally or vertically, but even in this scenario where they could jump from one host to another, they do not seem to do it regularly (Rivera-Parra et al. 2015).

The ischnoceran lice seemed to be extremely specific as well. This system with closely related hosts and parasites seemed perfect for finding host switches, but only a single adult individual and some larvae were found straggling on a different species. Even the effect of neighbor identity did not increase the likelihood of host-switch (Rivera-Parra et al. 2017). It seems plausible that the differences between parasite size (body width) and feather interbarbular space are preventing lice from establishing on a different host. Ischnoceran lice insert themselves in the interbarbular space of the feather as a mechanism of defense against the host's preening; if the parasite is too big, they do not fit and are more easily dislodged (Bush et al. 2006). Boobies plunge dive to fish (del Hoyo et al. 1992), so their ectoparasitic lice

have to withstand not only preening by the host, but the forces exerted during the plunge. Thus, any sub-optimal attachment to the feather may result in the lice falling from the host which would prevent the establishment of a viable population.

6.3 Implications for Avian Health

6.3.1 The Immune System of Island Endemics

Biologists frequently believe that isolated island parasite communities are small and impoverished (Wikelski et al. 2004), thus theoretically reducing the number of interactions that occur between parasites and hosts (Hochberg and Møller 2001). The costs associated with maintenance of immune function (Sheldon and Verhulst 1996; Norris and Evans 2000) also suggest that reduced selective pressures, due to low parasite diversity, would result in weakening of the immune system function of hosts through time (Van Riper and Scott 2001; Jarvi et al. 2001). In Hawaii, for example, endemic honeycreepers have been shown to be highly susceptible to introduced pathogens such as *Plasmodium relictum*. The susceptibility of these birds to avian malaria appears to be related to the low genetic diversity of their major histocompatibility complex (MHC) which in turn may reduce antigen recognition and antibody production by the host's immune system (Jarvi et al. 2001).

Loss of MHC and neutral genetic diversity is perhaps an inevitable result of genetic drift for small populations (Sutton et al. 2011) like those found on isolated archipelagos. The Galapagos penguin's (*Spheniscus mendiculus*) population size, for example, was last estimated at 1,500 individuals and it has undergone repeated bottlenecks of about 50% reduction in size every time there is an El Niño event (Vargas et al. 2006). It exhibits low levels of genetic diversity throughout its entire population in the archipelago and presents a lack of population structure among subpopulations (Nims et al. 2008). This low genetic variability can also be expressed at immunological loci that are fundamental in host resistance to disease. Compared to eight other species of penguins, including the Magellanic penguin (*S. magellanicus*) and the king penguin (*Aptenodytes patagonicus*), the Galapagos penguin had the lowest MHC diversity (Bollmer et al. 2007). Hence, the Galapagos penguin has been classified as Endangered (Birdlife International 2016) due to the risks presented by its demographic factors and the genetic monomorphism at loci involved in immune resistance.

Similarly, the endemic Galapagos hawk (*Buteo galapagoensis*) also presented reduced MHC and neutral genetic diversity related to a founder event and subsequent genetic drift, compared to its closest mainland relative the Swainson's hawk (*B. swainsoni*) (Bollmer et al. 2011). Unlike the penguin, the Galapagos hawk exhibits a significant genetic population structure that increases as distance between islands increases (Bollmer et al. 2005; Koop et al. 2014). This structure provided the context for Whiteman et al. (2006) to examine the association between genetic diversity, inbreeding, and disease resistance in the Galapagos hawk. Island populations of

hawks with higher degrees of inbreeding presented higher ectoparasite abundance and lower and less variable natural antibody (Nab) levels, demonstrating, for the first time in a wild island endemic, the link between genetic diversity, the innate immune system, and parasitic load.

The relationship between parasite abundance, immunity, and population size has also been investigated for Darwin's finches. Lindström et al. (2004) compared four island populations of small ground finches (*Geospiza fuliginosa*) and found that as parasite prevalence and/or intensity increased with island size, concentrations of natural antibodies and the speed of specific antibody responses also increased with island size. However, the strength of the cell-mediated immune response decreased with increasing island size, presenting an opposite pattern that suggested a tradeoff between antibody and cell-mediated immunity. In environments where parasites are more abundant, it may be more cost-effective to combine the presence of natural antibodies and a rapid production of specific antibodies than to invest in cell-mediated immunity.

A different shift in immune defense strategy of insular versus continental birds was suggested by Matson (2006). His comparison of eight indices of immune function between insular and continental species of birds found that island birds had increased innate and inducible immune responses. Insular birds presented higher concentrations of plasma haptoglobin and elevated levels of two innate leukocytes (heterophils and eosinophils) than continental birds but showed no differences in agglutination and lysis titers (acquired responses). However, Matson warns, the increase in innate responses may be a way to compensate for aspects of insular life such as reduced genetic variation and could possibly intensify the disease risks. In whole, it appears that the relationship between the host's immune system and parasite diversity in island populations is too complex to expect only a simple reduction in immune response in insular birds. Development of the immune system of isolated populations may depend not only on the diversity of parasites present but also on the specific parasites encountered and the stochasticity of mutation and genetic drift (Beadell et al. 2007).

6.3.2 Mortality

Island bird species have shown high vulnerability to introduced parasites. A clear example of this comes from Hawaii, where endemic honeycreepers experimentally infected with *Plasmodium relictum* have been shown to be extremely susceptible to the pathogen, with high mortality rates after a single mosquito bite (Jarvi et al. 2001). Other examples from islands include *Plasmodium* sp. parasites and mortality of native captive birds in New Zealand (Tompkins and Gleeson 2006), and reduced survivorship of endangered pink pigeons (*Columba mayeri*) infected with *Trichomonas gallinae* in Mauritius (Bunbury et al. 2008), among others (Wikelski et al. 2004).

In Galapagos wild birds, documented pathogenic causes of mortality include *Philornis downsi*, avian pox (genus *Avipoxvirus*: Poxviridae), and schistosomiasis (Gottdenker et al. 2008). An experimental approach attributed 27% of nestling mortality to *P. downsi* infestation given that pathogen-reduced nests had three times the nesting success of control parasitized nests (Fessl et al. 2006, see Chap. 9 this volume). *P. downsi* has been found in the nests of 12 introduced, native and endemic species in the archipelago (Fessl and Tebbich 2002) and has been associated with nestling morality in the small (*Geospiza fuliginosa*) and medium ground finches (*Geospiza fortis*) and in the critically endangered medium tree finch (*Camarhynchus pauper*) in Floreana (Fessl et al. 2006; Huber 2008; O'Connor et al. 2010). Avian pox is a prevalent disease affecting a wide variety of Galapagos endemic birds that has been present in Galapagos for at least a century (Parker et al. 2011). High mortality rates had been suggested for young Galapagos mockingbirds (*Mimus parvulus*) given the low recapture rates exhibited by infected individuals (Vargas 1987). Even though *P. downsi* and avian pox are highly prevalent pathogens, these examples constitute the only evidence of disease-related mortality in the avifauna of Galapagos.

Until now, no reports of Haemosporidian infection-related mortality have been documented for any Galapagos bird. Mortality associated with blood parasites in Galapagos wild birds may be underreported or hard to find as most of the Galapagos National Park is uninhabited; moreover, passerine carcasses may be rapidly scavenged by raptors or by feral dogs and cats. However, the potential risks that the parasites reported in the archipelago represent are great as these parasites can be lethal in non-adapted hosts (Atkinson et al. 1988; Jarvi et al. 2001; Cardona et al. 2002; Ferrell et al. 2007; Donovan et al. 2008; Olias et al. 2011; Cannell et al. 2013).

6.4 Concluding Remarks and Future Directions

The Galapagos archipelago provides an exceptional system to investigate the intricacies of parasite spillover. Its simplicity, or low number of host-parasite interactions, compared to continental systems, provides a natural laboratory to determine where the line falls between spillover and host-switching. Future research efforts should focus on determining the effects and risks that each of these events has on host health and survivorship. Furthermore, the link between genetic diversity, the immune system, and disease risk has only been touched and continues to pose very interesting questions about the ecology and evolution of hosts and parasites in isolated ecosystems. The degree of isolation of the archipelago declines with its increasing popularity as a travel destination, which in turn will increase the likelihood for introduced species and pathogens to arrive to the islands and bring ever-increasing opportunities for spillover. Thus, it is of great importance to continue to monitor avian health and pay close attention to ectoparasites and potential vectors of disease.

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