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Temporal stability of insular avian malarial parasite communities

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Avian malaria is caused by a diverse community of genetically differentiated parasites of the genera *Plasmodium* and *Haemoproteus*. Rapid seasonal and annual antigenic allele turnover resulting from selection by host immune systems, as observed in some parasite populations infecting humans, may extend analogously to dynamic species compositions within communities of avian malarial parasites. To address this issue, we examined the stability of avian malarial parasite lineages across multiple time-scales within two insular host communities. Parasite communities in Puerto Rico and St Lucia included 20 and 14 genetically distinct parasite lineages, respectively. Lineage composition of the parasite community in Puerto Rico did not vary seasonally or over a 1 year interval. However, over intervals approaching a decade, the avian communities of both islands experienced an apparent loss or gain of one malarial parasite lineage, indicating the potential for relatively frequent lineage turnover. Patterns of temporal variation of parasite lineages in this study suggest periodic colonization and extinction events driven by a combination of host-specific immune responses, competition between lineages and drift. However, the occasional and ecologically dynamic lineage turnover exhibited by insular avian parasite communities is not as rapid as antigenic allele turnover within populations of human malaria.

Keywords: avian malaria; temporal stability; community; turnover; *Plasmodium*; *Haemoproteus*

1. INTRODUCTION

Parasites are under continual selection to evade the immune systems of their hosts, while hosts are under selection to resist or control infection. A common assertion in the host–parasite literature contends that parasitism should evolve towards avirulent commensalism and coexistence since parasites that do not harm their hosts have the best chance of survival (Dubos 1965; Hoeprich 1977; Alexander 1981; Holmes 1982; Palmieri 1982). By contrast, theoretical and experimental evidence suggests that hosts and parasites are under continual selection to increase their resistance and virulence in a coevolutionary arms race (Ball 1943; Andrewes 1960; Anderson & May 1982; Levin 1983). Vector-mediated parasites, such as malaria, are predicted to evolve greater virulence than those transmitted without vectors because their transmission success does not depend on the mobility of the infected host (Ewald 1983, 1994; Herre 1993). Additionally, malaria parasites have highly variable antigenic surface proteins, which help them to evade the immune systems of their hosts. Antigen turnover has been examined both genetically and serologically within the most virulent species of human malaria parasite, *Plasmodium falciparum*. In some regions, this parasite exhibits significant allelic turnover both seasonally (Forsyth *et al.* 1988) and between years (Babiker *et al.* 1995; Silva *et al.* 2000; but see Conway *et al.* 1992). Estimates of between-population variation given in these studies suggest that antigen turnover in parasite populations of *P. falciparum* may be the result of a combination of mutation, frequency-dependent selection and random genetic drift (Forsyth *et al.* 1988; Babiker *et al.* 1995; Silva *et al.* 2000).

Within a multi-species community of parasites, host-mediated selection, combined with indirect negative interactions between parasite species resulting from cross resistance, could lead to highly variable and dynamic species compositions, with competitive exclusion and stochastic local extinctions of some parasites being followed by re-introduction or the colonization by new parasites. Laboratory and field studies of the interactions of malarial parasites have reported mixed results varying from positive through neutral to antagonistic interactions (Schall & Bromwich 1994; Staats & Schall 1996; Richie 1988 and references therein). Surveys of the prevalence of malaria in four avian host species in the Lesser Antilles revealed negative relationships between the prevalences of lineages on different islands, despite uniform total infection prevalence across islands, suggesting that parasites may compete with one another (Fallon *et al.* 2003*a*). Thus, selective pressures from both hosts and other parasites, in combination with stochastic genetic drift, could reasonably result in dynamic species compositions of parasite communities over time.

Alternatively, the frequency of parasite lineages may vary, perhaps owing to allelic variation specific to the host–parasite interaction, while the community composition of parasite lineages is stabilized over the long term by frequency- or density-dependent factors. Whether a community of parasites is temporally stable or experiences high turnover rates is important to our understanding of the nature and permanence of host–parasite associations. A recent study of two malaria parasite lineages within one avian host species in Sweden revealed a continuous regional presence of both lineages, but local variation in

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their distribution (Bensch & Akesson 2003). Temporal and spatial variations in malaria infections have also been demonstrated in two temperate lizard species (Jordan & Friend 1971; Schall & Marghoob 1995), while infections by two types of malarial parasite remained stable over a decade in a tropical lizard species (Schall *et al.* 2000). None of these studies, however, has examined parasite lineage composition within an entire community of hosts and their parasites.

Communities of birds are infected by a diverse assemblage of malarial blood parasites (*Haemoproteus* spp. and *Plasmodium* spp.) whose lineage diversity is of the same order as the number of host species (Ricklefs & Fallon 2002). We use a gene that is presumably independent of the host–parasite interaction (cytochrome *b*) to examine the temporal stability of the lineage composition of avian malarial blood parasites within two insular host communities across various time-scales. Specifically, we explore seasonal, annual and decadal variations in parasite infections within the bird communities of the islands of St Lucia and Puerto Rico in the West Indies.

2. MATERIAL AND METHODS

As part of an ongoing study of the bird communities of the West Indies, we sampled birds on the Lesser Antillean island of St Lucia during July 1991 and July 2000. Birds in Puerto Rico, in the Greater Antilles, were sampled during October 1993, January 2001, June 2001, October 2001 and January 2002. This sampling allows for an 8 or 9 year comparison for both islands and additional seasonal and 1 year comparisons for one location, the Guanica Forest in Puerto Rico. Sampling sites on both islands were either identical or similar throughout the sampling periods.

We caught birds using mist nets and took 5–10 µl of blood by wing or carotid venipuncture. Smears were prepared using *ca*. 2–3 µl of blood. Slides were air-dried, fixed in absolute methanol and stained with modified Giemsa stain solution (Sigma, St Louis, MO, USA). The remaining blood sample was stored in buffer prior to DNA extraction. Samples collected in 2000 and 2001 were stored in Puregene cell lysis buffer. All other samples were stored in Queen's lysis buffer (Seutin *et al*. 1991). Samples were collected and transported under the appropriate permits and licences from local governments following protocols approved by the University of Pennsylvania and the University of Missouri, St Louis.

DNA from blood samples in Puregene cell lysis buffer was extracted by salt precipitation according to the manufacturer's protocol (Gentra systems, Minneapolis, MN, USA). In all other samples we performed a standard phenol–chloroform extraction with dialysis in 1× Tris EDTA buffer as described in Seutin *et al.* (1993). All samples were screened for infection using a PCR assay based on a conserved RNA region of the 6 kb mtDNA genome for avian malaria (Fallon *et al.* 2003*b*). Blood smears were examined to confirm infection when a sequence could not be obtained.

We amplified positive samples using original PCR primers that target approximately 320 base pairs of the mitochondrial cytochrome *b* gene: 621F, 5′-AAAAATACCCTTCTATCC AAATCT-3′, and 983R, 5′-CATCCAATCCATAATAAA GCAT-3′ (Richard *et al.* 2002; Fallon *et al.* 2003*a*). PCR reactions were run in 10 µl volumes that contained the following components in their final concentrations: 2.5 mM of MgCl, 0.2 mM of each dNTP, 0.4 µM of each primer and 0.5 units of Taq polymerase. Aliquots of 1 µl of the extracted DNA were used for amplification. Thermal cycling conditions were as follows: 1 min at 94 °C, followed by 35 cycles of 0.5 min denaturation at 94 °C, annealing at 48 °C for 1 min and elongation at 72 °C for 1.5 min. After the 35 cycles, there was a final elongation step at 72 °C for 3 min. We purified the amplified product by gel extraction. Sequencing was carried out on an automated sequencer (ABI Prism 377; Applied Biosystems, Foster City, CA, USA) according to the manufacturer's protocol. The cytochrome *b* sequences were edited and aligned using DNASTAR software and are available through GenBank (accession numbers AY167239–AY167250 and AY455656–AY455663).

Because the RNA-based PCR assay used to screen for infections amplifies a region of the parasite mitochondrial genome that is more conserved than cytochrome *b* (Fallon *et al.* 2003*b*), we were unable to amplify the cytochrome *b* region for all of the identified infections. The discrepancy in amplification success between the two assays is the combined result of increased variability in cytochrome *b* priming sites and a decrease in sensitivity to low parasitaemia levels. Additionally, we did not include samples that produced unclear chromatograms owing to either mixed parasite infections or poor amplification.

The generic identity of the parasite lineages (*Haemoproteus* versus *Plasmodium*) was determined by the placement of the cytochrome *b* sequences within the phylogenetic trees of Ricklefs & Fallon (2002) and Perkins & Schall (2002). We arbitrarily named each lineage by assigning a letter in sequential alphabetic order following either an H (for *Haemoproteus*) or a P (for *Plasmodium*). Two lineages named 'Cpa' were recovered from common ground doves (*Columbina passerina*) and are basal in their phylogenetic position, but cannot currently be placed with confidence in either genus. The regional distributions of lineages HA–HE and PA–PC are described in Fallon *et al.* 2003*a*.

Lineages that we recognize as distinct differ from one another by between 1.2 and 14% sequence divergence and are considered evolutionarily independent. The rate of nucleotide substitution in the cytochrome *b* gene of malaria parasites appears to be only one-third as rapid as that in the avian hosts (Ricklefs & Fallon 2002), and so the lower end of this range is consistent with species-level mtDNA divergence in birds. Additionally, a cytochrome *b* sequence divergence as low as 1.0% has been observed between named species of malaria parasites of mammals (Escalante *et al*. 1998). However, true 'species' boundaries of avian malarial parasite lineages are yet to be defined, and await detailed analyses of nuclear as well as mitochondrial markers.

Distributions of parasite lineages were evaluated using standard goodness-of-fit tests (*G*-tests). When a lineage was recovered from only one host, temporal heterogeneity for that lineage was assessed within the single host. For lineages recovered from more than one host, temporal heterogeneity was assessed over individuals of all host species pooled.

3. RESULTS

Sample size, host composition and parasite prevalence (*ca*. 30%) were similar for the July 1991 and 2000 sampling periods in St Lucia (see electronic Appendix A, available on The Royal Society's Publications Web site). In total, we sampled 416 individuals representing 28 host species and detected 128 infections. We produced unambiguous sequences for 119 of these. Fourteen parasite

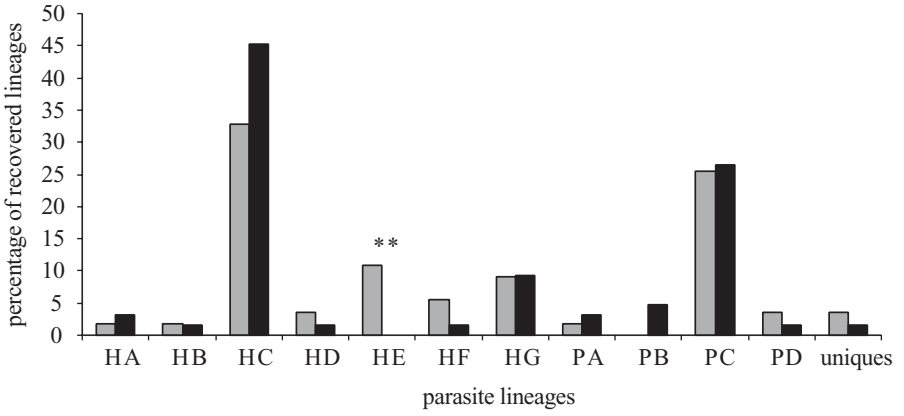

Figure 1. Prevalence of parasite lineages for two sampling periods in St Lucia (1991, grey bars; 2000, black bars). Asterisks represent a significant change in the frequency of a parasite lineage ($^{**}p < 0.01$).

lineages were recovered in July 1991 and 11 lineages were recovered in July 2000 (figure 1). Out of these, nine lineages were present during both sampling periods, and 11 lineages were recovered from more than one host individual. Four lineages were recovered only once; three of these were detected in 1991 and one in 2000. Among the lineages recovered more than once, lineage PB was detected only during the second sampling period and lineage HE only during the first. Lineage PB was found in three out of six *Vireo altiloquus* infections in 2000. Because only four *V. altiloquus* were sampled in 1991 (see electronic Appendix A), the probability that the 2000 results represent the acquisition of a novel parasite between sampling periods rather than random variation is not significant ($p = 0.063$). By contrast, lineage HE was recovered from five out of 22 *Loxigilla noctis* and one out of 35 *V. altiloquus* in 1991. Given a sample of 36 *L. noctis* in 2000, it is unlikely ($p = 0.001$) that we would not have detected this lineage if it were present at its 1991 frequency of 0.23 in that species. To have a probability of less than 0.05 of being detected in 36 *L. noctis* in 2000, the frequency of the parasite in the population would have to have been less than 0.08. Therefore, the absence of lineage HE from the second sampling period in St Lucia represents either the loss or a significant reduction in the frequency of this lineage from the island population. Overall, however, the relative frequencies of parasite lineages did not differ significantly between the two sampling periods ($\chi^2 = 13.6$, d.f. = 10, $p > 0.05$); lineages recovered only once (i.e. unique lineages) were not included in this analysis.

In Puerto Rico, 1743 individuals representing 66 host species yielded 357 infections (see electronic Appendix B). We successfully sequenced only 205 of these infections. Overall, parasite prevalence (20%) was similar between the 1993 and 2001 sampling periods. Seasonal samples (January, June and October 2001 and January 2002) were obtained in the Guanica Forest Reserve in southwestern Puerto Rico. Samples from January 2001 at this site exhibited significantly higher parasite prevalence than the remaining three seasonal samples ($G = 16.5$, d.f. = 3, $p < 0.005$). However, the fact that the January 2002 sample did not reveal a similarly high parasite prevalence suggests that the cause of the high prevalence in 2001 was not seasonal variation, but rather a factor specific to that sampling period. Moreover, the high parasite prevalence

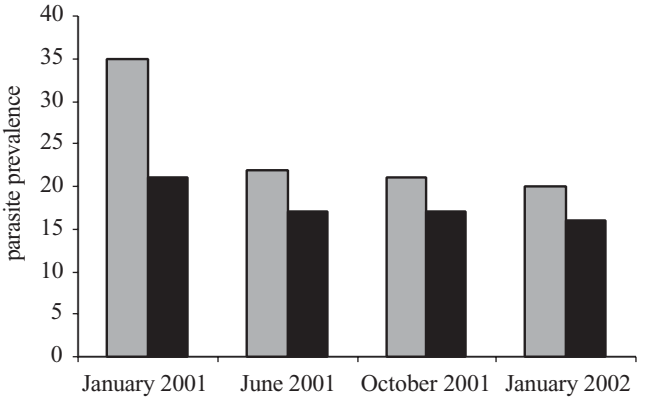

Figure 2. Seasonal prevalence of parasite infections in Guanica Forest including (grey bars) and excluding (black bars) *Coereba flaveola*. Levels of infections in *C. flaveola* were elevated in January 2001.

in January 2001 resulted largely from an elevated prevalence in one host, *Coereba flaveola* (75% in January 2001, but averaging 27% in the remaining three sampling periods; figure 2). When *C. flaveola* was removed from the analysis, parasite prevalence in the remaining 15 infected host species did not vary seasonally or annually ($G = 1.89$, d.f. = 3, $p > 0.05$).

The lineage composition of the parasite community in the Guanica Forest Reserve also did not vary seasonally between January 2001 and January 2002. Nine lineages were recovered overall and five of these lineages were present in all seasonal samples. The remaining four lineages were present in low numbers and their absence from one or more of the sampling periods did not represent a significant variation in prevalence (table 1). In order not to count January twice in the seasonal comparison, we considered only one of the January samples at a time. There was no evidence of a seasonal effect on the relative prevalence of the common parasite lineages between January 2001 and October 2001 ($G = 10.9$, d.f. = 8, $p > 0.05$), or between June 2001 and January 2002 ($G = 8.3$, d.f. = 8, $p > 0.05$).

A comparison of the two January samples (2001 and 2002) demonstrated heterogeneity in the annual prevalences of the five well-represented parasite lineages ($G = 10.57$, d.f. = 4, $p < 0.05$). A partitioned *G*-test

Table 1. Probabilities that parasite lineages not present in one or more seasonal samples result from chance sampling rather than a true absence.
(Probabilities determined by $(1-f)^n$. In cases where the lineage is found in more than one host species (generalist), sample size is determined by the total number of all sampled hosts. Where lineages are recovered from only one host, sample size is restricted to the sampling of that host species.)

parasite	generalist	frequency (f) when detected	sample size (n) when not detected	probability
lineage HB	no	0.500	1	0.50
lineage PA	yes	0.005	470	0.095
lineage PC	yes	0.004	390	0.21
lineage Cpa2	no	0.100	10	0.35

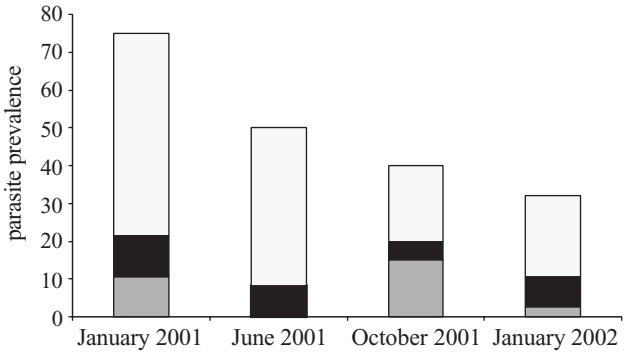

Figure 3. Total parasite prevalence and relative prevalence of individual parasite lineages (HD, open bars; HC, grey bars; HH, black bars) recovered from *Coereba flaveola* in the Guanica Forest Reserve, Puerto Rico 2001–2002.

analysis revealed that only lineage HD contributed significantly to the observed heterogeneity ($G = 14.9$, d.f. = 1, $p < 0.001$). Because lineage HD was recovered almost exclusively from *C. flaveola* (37 out of 41 infections), the between-years difference in the prevalence of this lineage reflects the elevated level of infections of *C. flaveola* in January 2001. When we removed *C. flaveola* from the analysis, the relative prevalence of the remaining parasite lineages did not differ significantly between years ($\chi^2 = 7.5$, d.f. = 4, $p > 0.05$). Within *C. flaveola*, the relative parasite prevalence of lineages, including HD, also did not vary, despite a change in total prevalence ($\chi^2 = 1.9$, d.f. = 2, $p > 0.05$; figure 3).

Because the presence and prevalence of parasite lineages showed no significant seasonal variations, samples taken throughout the year 2001–2002 were combined for the 8 year comparison. Eleven parasite lineages were recovered in 1993 and 17 lineages were recovered in 2001–2002. Out of these, eight lineages were present during both sampling periods and 13 lineages were recovered from more than one individual. Seven lineages were detected only once; two of the unique lineages were recovered in 1993 and five in 2001. Among the lineages found in more than one individual, four were detected only in 2001 and one only in 1993.

The overall distribution of common parasite lineages varied significantly between the 8 year sampling periods (figure 4; $\chi^2 = 46.3$, d.f. = 11, $p < 0.001$). Contributing to this difference were changes in the presence and relative frequency of a number of lineages. Lineage HD, which was not detected in 1993, was the most prevalent lineage in 2001. Again, this pattern is largely the result of the elevated level of infections in the lineage's primary host, *C. flaveola*, during January 2001. In addition, lineage HD was recovered from only one location in Puerto Rico (Guanica Forest) and may represent a habitat-specific as well as host-specific parasite lineage. Guanica Forest was sampled less intensively in 1993 and few *C. flaveola* were sampled ($n = 6$). Therefore, the 'absence' of this lineage in 1993 may be a sampling artefact rather than indicative of the acquisition of a new lineage.

Parasite lineage PE is also restricted to *C. flaveola* and declined island-wide between sampling years ($G = 12.8$, d.f. = 1, $p < 0.001$). In 1993, it was detected in mid- to high-elevation locations, both in eastern and western portions of Puerto Rico, and was one of two sequenced *C. flaveola* infections in Guanica Forest. By contrast, lineage PE was detected in only one mid-elevation eastern location (El Yunque) in 2001–2002, and was absent from 55 sequenced infections in Guanica Forest. Given the extensive sampling of *C. flaveola* during 2001–2002, the observed decline in this lineage both island-wide and within western areas of the island represents a significant change in the prevalence of this lineage. That this decrease was accompanied by the dominating appearance of lineage HD in the same host is notable.

Lineage HC also showed a marginally significant decline island wide ($G = 3.95$, d.f. = 1, $p < 0.05$). The remaining lineages individually showed no significant differences between sampling periods. Thus, differences in the relative prevalence of particular lineages of parasites between 1993 and 2001 can be attributed to the presence of lineage HD in the 2001 sample and the declines in the prevalences of lineages HC and PE.

4. DISCUSSION

The avian malarial parasite community on Puerto Rico appears to be relatively stable over periods of up to at least 1 year. The parasite community showed no seasonal variation in lineage composition or prevalence over a single year. A single host species, *C. flaveola*, demonstrated an unusually high level of infections during one sampling period (January 2001), but the remaining host species and parasite lineages showed little to no variation in patterns of infection throughout 2001–2002.

That parasite prevalence did not vary seasonally contrasts with a relatively well-established literature that has documented an increase in infections during the breeding season (Garnham 1966; Weatherhead & Bennett 1991,

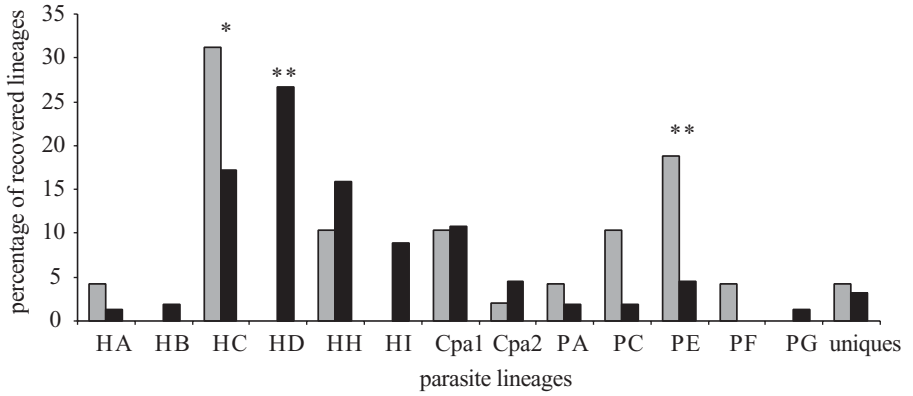

Figure 4. Prevalence of parasite lineages for two sampling periods in Puerto Rico (1993, grey bars; 2001, black bars). Asterisks represent a significant change in the frequency of a parasite lineage ($^{*}p < 0.05$; $^{**}p < 0.01$).

1992; Hatchwell *et al.* 2000; Deviche *et al.* 2001; Schrader *et al.* 2003). Breeding in the Guanica Forest Reserve is concentrated between April and June. Therefore, our June sample should accurately estimate parasite prevalence during the breeding season. It is possible, though unlikely, that a peak in infections could have preceded our sample. In addition, previous studies of seasonal variation of blood parasite prevalence have relied on visual inspection of blood smears rather than PCR techniques, which are more effective at detecting infections at low levels (Richard *et al.* 2002; Fallon *et al.* 2003*b*). Therefore, these changes may reflect fluctuations in the intensity of infection (parasitaemia) rather than differences in the presence and absence of infections. Though PCR is also sensitive to parasitaemia (Fallon *et al.* 2003*b*), seasonal fluctuations would be less pronounced in studies employing molecular techniques than in those using visual inspection of blood smears. Additionally, temporal (and spatial) variation in malarial parasite prevalence is more pronounced in temperate faunas than in tropical ones (e.g. Schall & Marghoob 1995; Schall *et al.* 2000; Freeman-Gallant *et al.* 2001; Fallon *et al.* 2003*a*). Thus, a combination of improved detection techniques and a relatively stable tropical environment may explain the lack of seasonal variation in parasite prevalence.

While our study revealed no differences in parasite prevalence or lineage composition seasonally or over the course of a year, sampling intervals approaching a decade demonstrated marked changes in both St Lucia, where a lineage was apparently lost from the avian malaria parasite community, and Puerto Rico, where a lineage may have been gained. The loss of parasite lineage HE from the sample of *L. noctis* on St Lucia was not accompanied by significant changes in the prevalences of other parasite lineages (figure 1). However, because overall parasite prevalence for the community did not vary, the decrease (or loss) of lineage HE must have been compensated for by increases in the prevalence of infections of one or more other lineages of parasites. Although not significant, the prevalences of lineages HC and PC did increase between samples. Similarly, in Puerto Rico, the appearance of lineage HD as the dominant parasite of *C. flaveola* was coincident with significant decreases in the prevalences of two other lineages (HC and PE; figure 4). These patterns further support the idea of an upper limit to the proportion of hosts that avian blood parasites can infect as a community (Fallon *et al.* 2003*a*). Within this limit, the composition of parasite communities and the frequencies of lineages vary not only spatially, but also temporally.

Changes in community composition when total parasite prevalence remains constant suggest that some parasites are able to exploit additional hosts in the absence or diminished prevalence of other parasite lineages. This pattern is consistent with parasite lineages competing with each other for infection opportunities. For example, parasite lineages HD and PE are virtually restricted to *C. flaveola* (*ca*. 90% of infections for both). On Puerto Rico, between 1993 and 2001, we observed what could be interpreted as a near displacement of lineage PE by lineage HD in some parts of the island. While we cannot assess statistically whether lineage HD was absent from Guanica Forest in 1993, lineage PE, which was originally present there, was conspicuously absent in 2001, at a time when lineage HD was dominant. This possible turnover event is similar to one observed over a 4 year period between two *Haemoproteus* lineages in a population of willow warblers (*Sylvia borin*) in southern Sweden (Bensch & Akesson 2003). While both lineages remained regionally present, locally they demonstrated temporal variation suggestive of displacement.

Variation in the regional presence of parasite lineages HD and PE in *C. flaveola* populations provides some insight into local colonization and the extinction dynamics of parasites. Lineage HD exhibits a broad but sporadic presence among *C. flaveola* in the West Indies. It was recovered from two northern Lesser Antillean islands (Monserrat and Barbuda) in 1993 and from Jamaica in 1995; the lineage is also sparsely represented on other Lesser Antillean islands (Fallon *et al.* 2003*a* and S. M. Fallon, unpublished data) and accounted for 20 out of 29 infections in *C. flaveola* in Venezuela. Because birds typically colonize islands in the West Indies sequentially in stepping-stone fashion, gaps in their distributions can be interpreted as local extinctions (Ricklefs & Cox 1972; Ricklefs & Bermingham 1999). Similarly, the presence of gaps in the distribution of parasites, such as those observed for lineage HD, may indicate which lineages experience frequent local extinctions. Lineage PE has been recovered in only small numbers outside Puerto Rico: two individuals in the Dominican Republic and three individuals in a large North American sample (data

not shown). The lineage is absent from the Lesser Antillean islands. Regional persistence of sparsely distributed lineages such as these would depend on their ability to colonize or recolonize island populations of hosts (Gilpin & Hanski 1991).

A mitochondrial phylogeographic study of *C. flaveola* in the Caribbean region suggests that this host species displays historical periods of invasiveness followed by relative geographical stasis (Seutin *et al.* 1994). Individuals of *C. flaveola* on Puerto Rico and Jamaica are differentiated from each other. Both differ from a Lesser Antillean clade, which shows evidence of a recent northward expansion from the central Lesser Antilles. The extent of phylogeographic structuring of the Caribbean *C. flaveola* suggests that individuals move infrequently between the Greater Antillean islands. However, the distribution of parasite lineage HD does not follow the same phylogeographic breaks as its primary host. This pattern suggests either that parasites are able to disperse without their hosts (i.e. through vector dispersal) or that *C. flaveola*, or another appropriate host, occasionally disperses between these islands. Strong geographical structuring among populations of the vectors of avian malaria suggests that they are confined to short-distance dispersal and that movement between islands is low (Fonseca *et al.* 2000). Therefore, occasional dispersal of hosts is a more likely explanation.

Alternatively, the distribution patterns of lineage HD may reflect slower rates of evolution in these parasite lineages than in their hosts, so that they do not demonstrate the same level of population differentiation (Ricklefs & Fallon 2002). In this case, the presence of lineage HD on Puerto Rico in 2001 could represent a resurgence of previously latent undetected infections rather than a recent colonization. Extensive sampling of *C. flaveola* from 2001 to 2002 might reveal whether a Lesser Antillean or Jamaican haplotype of this host colonized Puerto Rico, thus providing an opportunity for the introduction of lineage HD. However, even if *C. flaveola* individuals occasionally move between the Greater Antillean islands, particular parts of their genomes have a small chance of becoming established in the resident population by drift, therefore making it difficult to detect migrant haplotypes. By contrast, parasites could easily become established in a new host population. Therefore, evidence of movement between island populations may be seen in the presence of particular parasite lineages before it is seen in host genetics. That is, if lineage HD is indeed a recent introduction to Puerto Rico, then surveys of parasite lineages may be able to indicate gene flow in host populations not detected by population level studies of the hosts.

Interestingly, elevated levels of infections in *C. flaveola* were coincident with the first detection of lineage HD in January 2001. Among all the *C. flaveola* sampled that January, 75% were infected and just over 70% of these infections were lineage HD. Proportionally, lineage prevalence within this one host did not vary throughout the 2001 sampling; however, overall infections decreased significantly (figure 3). That is, lineage HD continued to represent most of the infections in *C. flaveola*, but the total number of infections declined. We cannot explain the abnormally high infection rates in *C. flaveola* during January 2001. However, elevated levels of infections would be consistent with the introduction of a novel parasite lineage against which the species was not well defended, but to which it subsequently developed some resistance. This pattern lends weight to the speculation that lineage HD is the product of a recent colonization event.

5. CONCLUSIONS

Generally, parasite communities appear to be relatively stable, notwithstanding several lineages that exhibited a marked change in prevalence over decade-long periods. Indeed, we may have witnessed the invasion of Puerto Rico by a novel parasite lineage and the local extinction of another lineage on St Lucia. If these observations reflect the typical dynamics of malaria parasites in these communities, then one might expect to observe a nearly complete turnover of the parasite fauna of a particular island in the span of approximately a century. Such rapid turnover rates, however, would probably inhibit the establishment of coevolutionary relationships, and yet historical relationships clearly exist between avian blood parasites and their hosts (Ricklefs & Fallon 2002; Bensch *et al.* 2000).

Therefore, entire parasite communities are likely to be reasonably stable, with changes being sporadic and limited to a small number of lineages. Gaps in the distributions of blood parasite lineages throughout the Lesser Antilles suggest that either island populations experience occasional local extinctions or the prevalences of certain lineages frequently fall below levels of detection (Perkins 2001; Fallon *et al.* 2003*a*). Within an archipelago, these dynamics would lead to independent patterns of infection in island populations of the same host species, as previously documented for this system (Apanius *et al.* 2000; Fallon *et al.* 2003*a*). Marked changes in prevalence again point to competition between lineages and to specific virulence–resistance relationships between hosts and their parasites.

Clearly, blood parasite communities are both spatially and temporally dynamic, and some degree of turnover probably occurs. Our data suggest that the time-scale for this turnover may be of the order of one lineage per decade for relatively isolated islands the size of St Lucia and Puerto Rico. At the same time, despite sharp changes in a few lineages, these insular malarial parasite communities remain relatively stable. Thus, seasonal and annual antigenic turnover resulting from selection and drift, as described in some populations of *P. falciparum* infecting humans (Forsyth *et al.* 1988; Babiker *et al.* 1995; Silva *et al.* 2000), does not analogously extend to community-wide variation in the lineage composition of avian malarial parasites. Instead, while the changes we observed invoke both host- and parasite-mediated selection pressure and drift, these effects probably occur on the same scale of virulence–resistance dynamics as in human malaria rather than as a rapid turnover of lineages of parasites within a community of hosts.

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