

17

ROBERT E. RICKLEFS,
SYLVIA M. FALLON, STEVEN C. LATTA,
BETHANY L. SWANSON,
AND ELDREDGE BIRMINGHAM

Migrants and Their Parasites

A Bridge between Two Worlds

MIGRATING BIRDS CARRY A VARIETY of external and internal parasites and can transmit pathogens, such as West Nile virus, between host populations in the Tropics and temperate regions. The rapid spread of diseases such as West Nile virus and mycoplasmal conjunctivitis (*Mycoplasma gallisepticum*) in eastern North America and the devastating effects of avian malaria (*Plasmodium relictum*) on the native Hawaiian avifauna illustrate the potential for emerging diseases carried by migrants to become major management and conservation problems. Recent molecular studies using PCR and DNA sequencing to identify infections and characterize lineages of avian malaria (*Plasmodium* and *Haemoproteus*) show that migrants transport these parasites between tropical and temperate regions. Some lineages of *Plasmodium* have been recovered from both tropical and temperate resident host populations, as well as migrants, indicating that migrants have been responsible for dispersal of the parasites between unrelated hosts in geographically distant locations. Analysis of parasite and host phylogenies shows that most parasite lineages have strong host phylogenetic conservatism, but switching among distantly related hosts is not infrequent. Moreover, because host switching appears to be haphazard, it is unlikely that cases of emerging diseases can be predicted. Nonetheless, we must acknowledge the ability of birds to spread diseases widely and undertake further empirical and experimental studies of disease distribution and transmission in wild bird populations.

INTRODUCTION

Each year billions of birds leave their wintering areas in tropical and subtropical regions of the Earth and migrate poleward to summer breeding grounds. Each migrant carries a variety of symbiotic organisms, many of them potentially pathogenic to individuals of other species. These global movements should cause concern about the potential of migrants to spread novel diseases worldwide (McClure 1974; Service 1991; Daniels 1995; Lundstrom 1999), even reaching isolated regions where native species may have reduced immunological defenses against pathogens (Van Riper et al. 1986; Massey et al. 1996). In addition to host movement, physical conditions in the external environment and the availability of suitable vectors and alternative hosts should also influence the distribution of parasitic organisms (e.g., Super and Van Riper 1995; Randolph and Rogers 2000).

The biogeography of parasites, especially microparasites, such as viruses, bacteria, and protozoa, is not well understood. Parasite populations typically are sparsely sampled compared with their hosts, and the taxonomy and systematic relationships of many groups of parasites are poorly known. With the advent of PCR and DNA sequencing, however, we can now characterize parasite lineages and describe their distributions unambiguously with respect to geography and hosts. From the distribution of naturally occurring parasites, we can begin to understand parasite dispersal and host switching as historical occurrences, and we can perhaps gain useful insights concerning diseases in host populations.

Our laboratories have recently begun to characterize avian malaria parasites in eastern North America and the Caribbean Basin using sequences of the mitochondrial cytochrome *b* gene (Bensch et al. 2000; Perkins and Schall 2002; Ricklefs and Fallon 2002; Fallon et al. 2003). Although our data are preliminary at this point, our results provide a glimpse of the complex patterns of geographic and host distributions of parasite lineages. Drawing on our own work and on the literature, we address the following questions: (1) What is the impact of disease organisms on host populations? Of particular interest is the effect of disease on the performance and survival of birds during migration. (2) What is the role of host dispersal in the movement of parasite organisms? Specifically, do migrants form a parasite bridge between resident species on the wintering and breeding grounds? It is not sufficient that migrants carry disease organisms; the parasites require suitable hosts, vectors, and physical conditions to become established in new areas. (3) Recognizing the importance of connecting wintering and breeding populations, we also ask whether parasites can serve as markers for the geographic origins of migrant individuals.

THE IMPACT OF DISEASES ON HOST POPULATIONS

We expect the consequences of pathogens for conservation, wildlife management, agriculture, and public health

to vary with time following a population's first exposure. Host and parasite populations tend to coevolve toward a point at which disease organisms maintain themselves at low levels controlled by reduced parasite virulence and increased parasite resistance (Ewald 1994; Hill 2001). Although endemic parasites can have profound consequences for host populations, as shown, for example, in the case of *Trichostrongylus tenuis* infecting Red Grouse (*Lagopus lagopus*) in Scotland (Dobson and Hudson 1995; Hudson and Dobson 1997; Hudson et al. 1998), newly introduced, or "emerging," diseases often have even more dramatic effects upon initial exposure.

The impact of the malaria parasite *Plasmodium relictum* on the Hawaiian avifauna has been documented by Van Riper et al. (1986) and in more recent experimental studies by Atkinson and his colleagues (e.g., Atkinson et al. 1995, 2000, 2001a, 2001b). *Plasmodium relictum* was most likely introduced to Hawaii in the early 1900s; a competent vector, the mosquito *Culex quinquefasciatus*, has been present in Hawaii since 1826. Although many introduced birds in Hawaii acquire *Plasmodium* infections (Shehata et al. 2001), the disease rarely develops to the clinical levels observed in many species of the native Hawaiian avifauna (summarized by Jarvi et al. 2001). Even native birds vary in their resistance to malaria. Hawaiian thrushes (the Omao [*Myadestes obscurus*]) survive experimental infection whereas various species of Hawaiian honeycreepers (Drepanididae) exhibit 50–100% mortality. The Hawaii Amakihi (*Hemignathus virens*) shows evidence of recently evolved resistance. Individuals from low elevation, where mosquito vectors spread the disease, now resist infection better than individuals from high elevations that lack the mosquito and the disease (Van Riper et al. 1986; Atkinson et al. 2000). Although isolated island faunas might be particularly vulnerable to new disease organisms, emerging diseases also have spread rapidly through continental wildlife populations, as in the case of rinderpest in African ungulates (Plowright 1982) and West Nile virus in North American birds (Marfin and Gubler 2001).

Because migration imposes a tremendous stress, birds may be especially vulnerable to the disease effects of parasites during this period of the annual cycle. Apapane (*Himatione sanguinea*) infected with *Plasmodium relictum* showed reduced locomotory activity and feeding and probably would not have been physiologically capable of long-distance migration (Yorinks and Atkinson 2000). Even when an infection is not intense enough to interfere directly with oxygen transport in the blood, malaria parasites elicit a strong immune response (Apanius et al. 2000; Atkinson et al. 2001a, 2001b), which may impair physiological function (Fair et al. 1999), including flight performance. Avian hematozoa may be a particular problem for young birds controlling their first infections, which are likely to be contracted on the breeding grounds either in the nest or shortly after fledging. Many passerine nestlings and fledglings brought to the St. Louis Wild Bird Rehabilitation Center are infected with hematozoans (Ricklefs et al., unpubl.). Although this may be a biased sample, it does highlight the po-

tential vulnerability of young birds to disease prior to their first long-distance migration.

A particularly dramatic effect of parasitic organisms on survival of migration can be inferred from the failure of individual Palm Warblers (*Dendroica palmarum*) infected with *Knemidokoptes* mites on their wintering grounds in Hispaniola to return the following winter (Latta 2003). In this study, individuals with mite infestations, which cause abnormal hypertrophy of scales on the legs, lost weight as the winter progressed, although comparisons with uninfested individuals revealed no effect on site fidelity within the wintering area. In subsequent years of the study, however, none of the mite-infested individuals returned to their wintering grounds, compared with return rates of 40–70% among noninfested birds. Although the direct cause and timing of mortality are unknown, the physiological costs of mite infestation probably resulted in mortality during migration. A path analysis, which evaluates hypotheses concerning causal relationships, showed that mortality was directly related to mite infestation rather than to the secondary effect of parasitism on condition. Clearly, further information on the stress of migration as a selective agent on disease resistance is needed (see Sheldon and Verhulst 1996; Råberg et al. 1998).

EMERGING DISEASES AND THE SPREAD OF INFECTIONS

Recently, we have witnessed in North America the rapid spread of two novel diseases over large areas of the continent. This most likely resulted from movements by infected hosts and shows the power of dispersal and, by extension, migration in the spread of infectious agents. Mycoplasmal conjunctivitis, or House Finch eye disease, first appeared in populations of House Finches (*Carpodacus mexicanus*) in the vicinity of Washington, D.C., in January 1994. The responsible organism, the bacterium *Mycoplasma gallisepticum*, is the agent of a widespread endemic disease (MG) of poultry over most of the United States. The emergence of the disease in House Finches may have resulted from their contact with contaminated food and refuse after seeking food in poultry barns. Within 2 years, the disease had spread as far as Georgia, Illinois, and Quebec, and by 1997 it was broadly distributed throughout the entire range of the House Finch in eastern North America (Dhondt et al. 1998; Roberts et al. 2001). The particular form of MG contracted by House Finches appears not to have infected other birds, except for several American Goldfinches (*Carduelis tristis*) that contracted the disease in 1995 and 1996 in the southeastern United States (J. Cook [www.members.aol.com/FinchMG/IntroBac.htm]).

West Nile virus (WNV) has received more attention because it infects a broad spectrum of birds and mammals, including humans. The virus first appeared in North America in the vicinity of New York City in 1999. By 2001, it had spread west as far as St. Louis and south to Florida and the

Gulf Coast. WNV has been isolated from more than 100 species of birds and causes substantial mortality of corvids (Anderson et al. 1999; Ebel et al. 2001; Marfin and Gubler 2001). Human cases of encephalitis caused by WNV have appeared as far south as the Cayman Islands (August 2001 [www.carec.org/data/alerts/011017.htm]), possibly having been carried by migrant birds. In Europe, epidemics of WNV are associated with high populations of *Culex* mosquitoes, the primary vector species, after heavy rains and flooding, but rarely persist more than a couple of years (Hubalek 2000), suggesting that high vector densities are required for persistence of a WNV outbreak, as in the similar case of an outbreak of Japanese encephalitis virus in northern Australia (Hanna et al. 1996).

DO MIGRANTS CARRY DISEASE ORGANISMS?

Observations of parasites in migrating birds strongly suggest that migratory birds carry disease organisms (McClure and Ratanaworabhan 1973). Migrants commonly carry ixodid ticks, many of which are infected by *Ehrlichia* pathogens and the spirochete bacterium *Borellia*, which causes Lyme disease (Smith et al. 1996; Fubito et al. 2000; Alekseev et al. 2001; Scott et al. 2001). Hemoproteids, including avian malaria, are frequently found in the peripheral blood of migrants (Rintamaki et al. 1998; Bensch et al. 2000). Outbreaks of West Nile virus and related viruses in the Middle East and southern and eastern Europe are thought to have been brought from parts of tropical Africa (where the pathogen probably is endemic) by migrants returning to their northern breeding grounds (Hubalek 2000; Miller et al. 2000; Zeller and Murgue 2001; Weissenböck et al. 2002).

Regardless of the potential of migrants to carry disease organisms between regions, the establishment of new diseases as the result of migration depends on several factors. First, the probability of a disease spreading by migrants should increase in relation to the number of arriving individuals with high parasitemias (Anderson and May 1991). High parasitemias may be more likely among migrants if the stress of migration suppresses immune function (Ots and Hörak 1996; Råberg et al. 1998; Webster et al. 2002) and results in higher intensities of infection, although data on this point are lacking. Second, suitable vectors must be present to transmit the disease and maintain the infection in the population (Shroyer 1986; Mitchell 1991). Third, resident species must be susceptible to infection. The prospects for the spread of a disease vary greatly depending on the complexity of the life cycle, the duration and requirements of free-living stages, and the degree of specialization of vectors and the disease organism itself. How well particular parasite-host systems fulfill these criteria is not well understood. Viral diseases spread by physical inoculation by mosquitoes or other biting insects might encounter few barriers to their spread other than the immune defenses of potential hosts. To the extent that this is true, pathogens such as West Nile

virus are of great concern, particularly for isolated areas with few endemic pathogens to maintain strong immune defenses in potential hosts.

MALARIA IN NORTH AMERICAN AND THE CARIBBEAN BASIN

Malaria is caused in birds by apicomplexan parasites belonging primarily to the genera *Plasmodium*, *Haemoproteus*, and *Leucocytozoon* (Atkinson and Van Riper 1991). *Plasmodium* is transmitted by various species of *Culex* mosquitoes, whereas *Haemoproteus* is transmitted by biting midges (Ceratopogonidae) or louse flies (Hippoboscidae) (Atkinson and Van Riper 1991). The related *Leucocytozoon* infects leucocytes and erythrocytes in the peripheral blood and is transmitted by blackflies of the genus *Simulium* and by *Culicoides* midges (Fallis et al. 1974).

Malaria infections in birds traditionally have been identified on thin blood smears (Godfrey et al. 1987; Fedynich et al. 1995). Typically ca. 10,000 red blood cells are scanned and infections are reported as the prevalence of a parasite in a population, that is, the proportion of individuals infected of those examined. The intensity of infection in a single individual—its parasitemia—is typically reported as the number of infected red blood cells per 10,000, or other base number, examined.

Several compilations of hematozoan prevalence are summarized in fig. 17.1 for: (1) both migrant and resident birds sampled in North America, (2) Neotropical migrants in the Tropics, and (3) endemic Neotropical species. Because blackflies are uncommon in tropical regions, *Leucocytozoon* is present only at low levels, both among endemic or resident species and among Neotropical migrants. The prevalence of *Leucocytozoon* is much higher in North America, particularly at more northern latitudes within this region (Greiner et al. 1975). *Haemoproteus* is also an abundant par-

asite in North America, but the prevalence among North American migrants on their wintering grounds is low and comparable to that of tropical residents. *Plasmodium* is less frequent than *Haemoproteus* and its overall prevalence does not vary between regions.

Variation in prevalence can be caused by variation in the rate of infection or in the control of infection by host individuals. Analysis of blood smears results in false negatives (parasites present but not detected) when the intensity of infection drops below the detection limit due to sampling of about 10^{-4} cells. Recently, a number of PCR-based tests have been developed to detect infections (Feldman et al. 1995; Li et al. 1995; Bensch et al. 2000; Jarvi et al. 2002; Ricklefs and Fallon 2002), but any one of these is not fully reliable across host species (Richard et al. 2002). We now screen blood samples with several PCR primers based on protein-coding and mitochondrial RNA-coding sequences, which can detect infections having parasitemias as low as 10^{-5} infected cells across a wide variety of species (Fallon et al. 2003). We compared infection rates based on blood smears (ca. 10,000 cells) and several primer pairs in a winter sample of 100 individuals from the Guanica Forest in Puerto Rico, including both migrants and residents. Blood smears indicated an overall malaria prevalence of 28%, whereas the corresponding value based on PCR screening was 42%. Thus, parasite prevalence may typically be higher than reported from smears (Richard et al. 2002). However, if the disease is not present in the peripheral circulation, or is present at very low levels, PCR screening may also fail to detect its presence (Jarvi et al. 2002). If many infections are maintained at low levels, this also implies that a component of the variation in prevalence among samples might reflect immune control of infections rather than presence or absence of infection (Applegate 1970; Richie 1988). Thus, migrants might carry potentially pathogenic parasites cryptically.

Malaria parasites are distinguished and named according to morphological characters visible on blood smears, in-

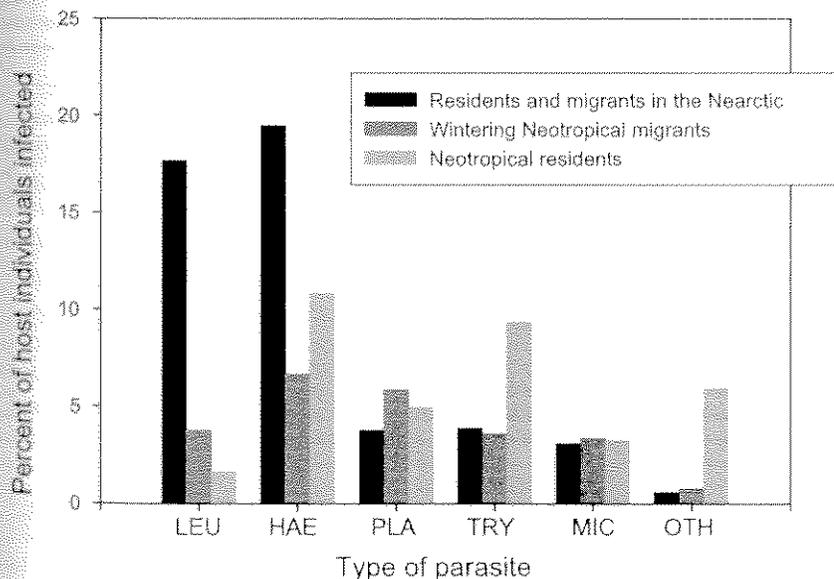


Fig. 17.1. Prevalence of several types of blood parasites, including the malaria parasites *Plasmodium* (PLA) and *Haemoproteus* (HAE), observed in blood smears of birds in the Nearctic (residents and migrants considered together) and in the Neotropics (residents and migrants separated). LEU = *Leucocytozoon*; TRY = trypanosomes; MIC = microfilariae; OTH = other. Based on summaries in Greiner et al. (1975) and White et al. (1978).

cluding the size, shape, and number of pigment granules in mature gametocyte (infective) forms of the parasites in red blood cells (Bennett et al. 1993, 1994; Peirce and Bennett 1996). Because malaria parasites are believed to be specialized (Atkinson and Van Riper 1991), host taxonomic group is also used as a diagnostic character. Analyses of phylogenetic relationships based on the mitochondrial cytochrome *b* gene are beginning to reveal substantial diversity of parasite lineages and suggest that current taxonomy does not adequately reflect either the diversity of, or relationships among, malaria parasites (Escalante et al. 1998; Bensch et al. 2000; Perkins 2000; Ricklefs and Fallon 2002; Waldenström et al. 2002). Phylogenetic analyses show that avian and mammalian malaria parasites are distinct clades and that avian *Haemoproteus* is a distinct clade nested within paraphyletic lineages of avian *Plasmodium* (Bensch et al. 2000; Perkins and Schall 2002; Ricklefs and Fallon 2002).

The most straightforward evidence for host switching in avian malaria is the presence of a single lineage in more than one host. In the analysis of Ricklefs and Fallon (2002), which was based on haphazard sampling of hosts, there were ten such cases. Two of these involved hosts in the same genus (*Sialia* and *Piranga*), seven more involved more distantly related hosts in the same family, and the tenth involved hosts in the same superfamily (in the sense of Sibley and Ahlquist 1990). Analysis of host distributions in closely related parasite lineages, including a tree-based analysis of cospeciation and host switching (Page 1995; Ronquist 1997), reinforced

the general impression that although host switching is conservative taxonomically, malaria parasites occasionally cross fairly large taxonomic distances to infect unrelated hosts (see also Bensch et al. 2000; Waldenström et al. 2002; Ricklefs et al. 2004). With the limited information currently available, these instances appear to be infrequent and unpredictable. However, like the broad host distribution of one or a limited number of strains of *Plasmodium relictum* in the Hawaiian avifauna, they emphasize the possibility of parasites switching hosts and potentially causing disease epidemics.

MIGRANTS AS BRIDGES BETWEEN NORTH AMERICA AND THE CARIBBEAN

The most straightforward evidence that migrants have transmitted parasites between birds in tropical and temperate regions would be the presence of a genetically identifiable lineage of parasite in both resident and migrant populations (Ricklefs and Fallon 2002; Waldenström et al. 2002). We are sequencing parasite cytochrome *b* from large numbers of individuals sampled in North America and the West Indies and have found several cases of shared parasite lineages (tables 17.1, 17.2). Table 17.1 includes nine malaria parasite lineages from Puerto Rico and the Lesser Antilles identified in three species of fringillid (Bananaquit [*Coereba flaveola*]; Lesser Antillean Bullfinch [*Loxigilla noctis*]; Black-

Table 17.1 The distribution of five lineages of *Haemoproteus* and four lineages of *Plasmodium* commonly found in four species of Lesser Antillean birds, among Neotropical migrants and Nearctic residents in Missouri, Alabama, and Michigan

Parasite	A	Tropical residents				Neotropical migrants	North Temperate residents
		NANA	LABU	BFCR	BWVI		
<i>Haemoproteus</i>	A	51	99	10	2	Red-eyed Vireo	
	B		1		7	Red-eyed Vireo	
	C	52	2	1			
	D	1	21	2	2	Northern Parula	
	E				10	Red-eyed Vireo (2)	
<i>Plasmodium</i>	A	24	19	28		Yellow-breasted Chat (3)	Northern Cardinal
						Black-and-White Warbler	
	B	5	2	3		Indigo Bunting (3)	Northern Cardinal (4)
						Yellow-breasted Chat	Carolina Wren
						Hooded Warbler	Eastern Towhee
						Blackpoll Warbler	Northern Mockingbird
						Rose-breasted Grosbeak	
						Scarlet Tanager	
	C	1			Gray Catbird		
	D	7			Red-eyed Vireo		

Note: Numbers in the columns for tropical residents are the numbers of individuals carrying each parasite lineage. Numbers of Nearctic birds found with each of the parasite lineages are indicated in parentheses if more than one. From Fallon et al. (2003) and Ricklefs et al. (unpubl.). NANA = Bananaquit, LABU = Lesser Antillean Bullfinch, BFCR = Black-faced Grassquit, BWVI = Black-whiskered Vireo.

Table 17.2 Additional examples of closely related parasite lineages present in small numbers of West Indian residents and also in either Nearctic residents or Neotropical migrants sampled in North America

		Tropical residents	Neotropical migrants	Temperate residents
<i>Haemoproteus</i>	F	Scaly-breasted Thrasher		Gray Catbird (Missouri)
		Pearly-eyed Thrasher (Montserrat)		
	G	Bananaquit (Trinidad)		
	H	Puerto Rican Bullfinch	Prairie Warbler (Missouri)	
			Scarlet Tanager	
			Summer Tanager	
			Kentucky Warbler	
			Red-eyed Vireo (Missouri)	
<i>Plasmodium</i>	E	Brown Trembler (Guadeloupe) Bananaquit (Jamaica)	Worm-eating Warbler	Tufted Titmouse (Missouri)
			Magnolia Warbler	
			Yellow-throated Warbler	
			Hooded Warbler	
			Black-and-White Warbler (Missouri)	
			Common Yellowthroat (Michigan)	

facéd Grassquit [*Tiaris bicolor*]; and the Black-whiskered Vireo [*Vireo altiloquus*]). Seven of the nine lineages have also been found in Neotropical migrants and two have been recovered from resident songbirds in southern Missouri and elsewhere in eastern North America. Although Lesser Antillean parasite lineages are present in migrant birds in Missouri, they are relatively uncommon. This may be due to nonoverlap of the particular migrant and tropical resident populations examined, as relatively few Neotropical migrants reach the Lesser Antilles, and migrants breeding in Missouri tend to winter in the Greater Antilles and Mesoamerica. It should also be noted that we have sampled relatively few North Temperate residents and so the sparse appearance of Lesser Antillean parasite lineages in these species is not informative. Some sharing of parasite lineages between winter migrant and resident host species has similarly been observed in Nigeria, indicating parasite transmission between migrants and residents on the wintering grounds (Waldenström et al. 2002). Table 17.2 shows additional examples of parasite connections from smaller samples among a variety of other hosts.

Clearly, migrants can form an effective bridge for parasites between tropical and temperate resident birds. These connections tend to involve hosts from the same general taxonomic groups (e.g., fringillids or mimids); however, two of the temperate residents sharing parasite lineages (Carolina Wren [*Thryothorus ludovicianus*] and Tufted Titmouse [*Baeolophus bicolor*]) are distantly related to the migrant or tropical resident co-hosts. We have not yet analyzed a large number of samples from North America to determine the extent to which parasite lineages occurring there have also been recovered from resident West Indian birds. Nor have we yet analyzed the large number of migrant individuals that we have sampled in the Greater Antilles during the northern winter. When these studies are completed, we shall be able to determine the degree to which the parasites of Neotropical migrants are unique to them as opposed to

being characteristic of the regions in which they spend the winter or summer months.

As previously mentioned, the ability of a parasite to switch to a new host depends on the presence of infective host individuals, suitable vectors, and susceptible new hosts. Whether a potential host is susceptible to a new lineage of parasite depends on antigen-immune function interactions between the parasite and the host. Parasites such as West Nile virus (Rappole et al. 2000; Marfin and Gubler 2001) and some strains of malaria parasites (this study) can infect a broad range of host species. Others appear to be more specialized (table 17.1). The outcome of the parasite-host interaction may depend on genetic factors that change rapidly in host and parasite populations, perhaps much faster than the appearance of new genetic variation in many of the gene sequences used in phylogenetic analysis (Fallon et al. 2003). This labile evolutionary interaction between parasite and host populations is indicated by host species-times-island interactions in the prevalence of malaria parasites in the Lesser Antilles (fig. 17.2). The proportion of infected individuals of four species varied independently across islands, suggesting that each island population of host responded independently to the parasite lineages present on each island (Apanius et al. 2000; Fallon et al. 2003).

As can be seen in the case of the Lesser Antillean Bullfinch, increasing intensity of infections parallels the proportion of individuals with infections detected in blood smears. Thus, it is not clear whether prevalence determined on blood smears represents the proportion of individuals infected or the ability of the immune system to control infections. Regardless, prevalence is indicative of the balance between parasite virulence and host resistance. The host-species-times-island interaction illustrates the dynamic nature of the parasite-host interaction and the importance of genetic factors in both populations to the susceptibility of individuals to infection by a particular lineage of parasite.

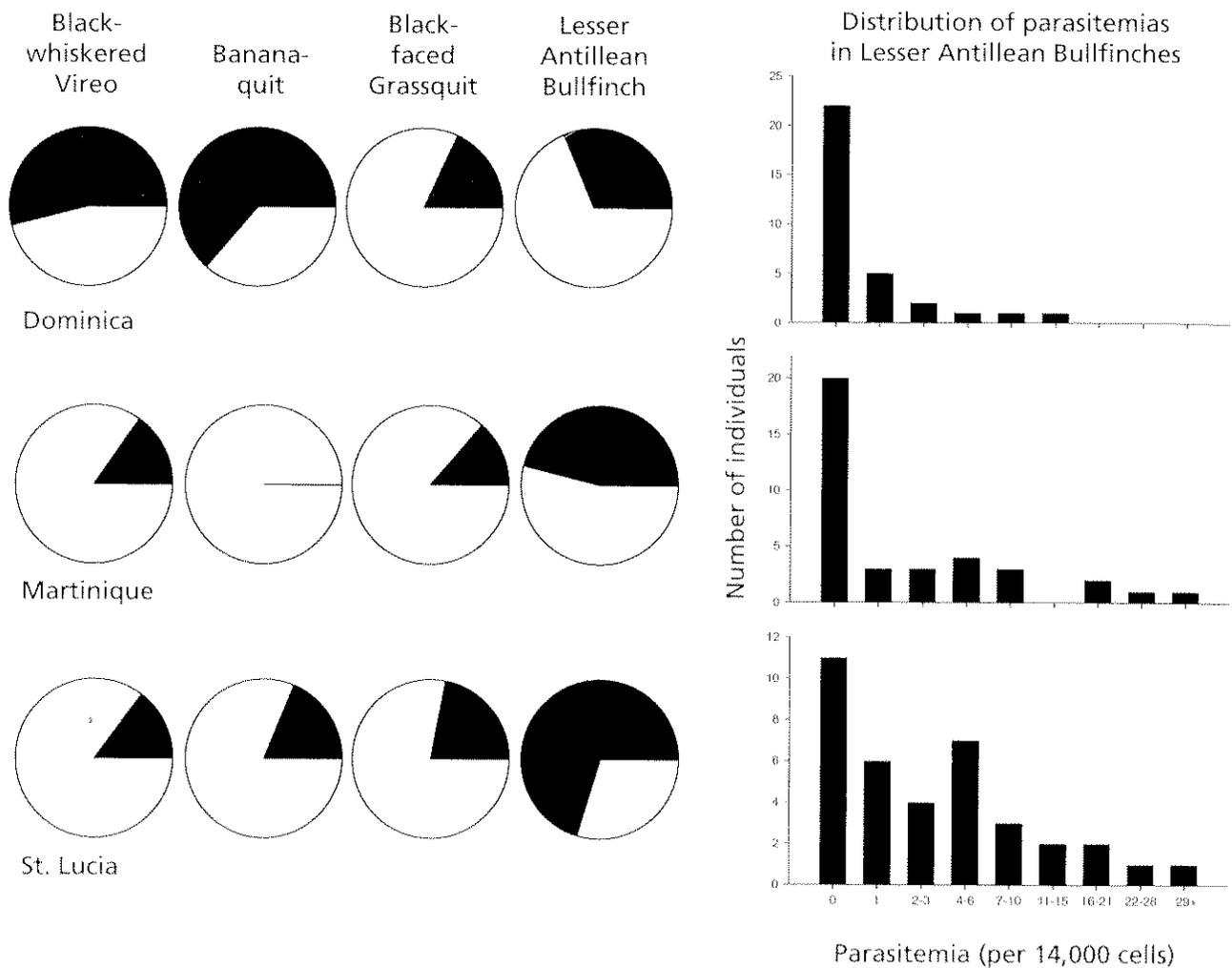


Fig. 17.2. Pie diagrams show the prevalence of malaria parasites revealed in blood smears in four species of birds on three islands in the Lesser Antilles. The host-species-times-island interaction is highly significant, indicating evolutionary independence of the host-parasite interactions (Apanius et al. 2000). The bar diagrams at right show the distribution of parasitemias for Lesser Antillean bullfinches on each of the three islands, illustrating the positive correlation between parasitemia and prevalence.

This is shown quite clearly in table 17.1 in the heterogeneous distribution of malaria parasite lineages among the four host species in the Lesser Antilles. We cannot predict whether a particular parasite lineage will infect a particular species. It is also unclear whether the single cases of *Haemoproteus* lineage C in a Black-faced Grassquit or of *Haemoproteus* D in a Bananaquit represent populations of these parasite lineages established in these hosts, spillover infections originating in the common host, or the rare manifestation of generally well-controlled infections.

PARASITE LINEAGES AS INDICATORS FOR LOCATION

To understand the causes of population trends in migrant birds, it is essential to identify the wintering and breeding areas of each population. So few birds banded in North America are recovered on the wintering grounds that the

geographical connections between summering and wintering migrants have proved elusive. Attempts to localize populations of migrants by genetic markers have been successful in some species (Haig et al. 1997; Wennerberg 2001), but mostly have been disappointing because of the general homogenization of host genotypes throughout their North American ranges (Merila et al. 1997; Mila et al. 2000; Webster et al. 2002). Recently, new applications of technologies, especially in stable isotope chemistry, have allowed some discrimination of the areas of origin of migrant birds (Chamberlain et al. 1997; Marra et al. 1998; Chamberlain et al. 2000; Hobson et al. 2001).

Several authors have suggested that parasite lineages may be sufficiently locality-specific to indicate the origins of migrant birds. For example, Rintamaki et al. (1998) found that Willow Warblers (*Phylloscopus trochilus*) passing through an autumn stopover site in southern Finland carried different blood parasites (*Leucocytozoon* vs. *Haemoproteus*) at different times during the migration period. Using morphological

evidence as well, the authors concluded that birds carrying the different parasites became infected in different areas and likely originated in different breeding populations (see also Thul et al. 1980 and Pung et al. 1997).

Parasites may be useful for distinguishing distinct populations occupying different types of habitats, but several factors argue against their general application for localizing individuals in broadly distributed populations. Such populations tend to lack genetic spatial structure, and the movement of individuals that prevents such structure from developing might be sufficient to homogenize parasite lineages, as suggested by surveys of Sehgal et al. (2001) on trypanosomes in central African birds. Rapid spread of diseases such as House Finch conjunctivitis and West Nile virus, at rates of hundreds of kilometers per year, provides little hope for local differentiation and endemism of parasite lineages. We have found only limited evidence for localization of parasite lineages on islands in the Lesser Antilles, where movement of individuals between islands is certainly less than across similar distances in North America (Fallon et al. 2003, unpubl.). Malaria parasite lineages tend to be localized to continents (Ricklefs and Fallon 2002), but this scale is not useful for localizing the origins of migratory individuals within populations.

The genetic markers used in most phylogenetic studies of parasites evolve slowly compared with the rate of dispersal of parasites through the host geographic range. Rapidly evolving genetic markers, which produce new mutations faster than they can disperse through the population, might provide worthwhile information on geographic origin. Such markers might possess transient value as spatially sensitive markers. For example, Anderson et al. (2001) surveyed a portion of the genome of West Nile virus that encodes envelope and membrane proteins and appears to be liable to rapid accumulation of nucleotide substitutions. They identified one mutation localized within 50 miles of Stamford, Connecticut. Surface proteins of malaria parasites, which mutate rapidly to avoid suppression by the host immune system, may be good candidates for geographically explicit markers.

DISCUSSION

Birds do not leave their parasites behind when they migrate from their wintering to their breeding grounds and back. Although there is some evidence that many infected individuals may not be physiologically capable of long-distance migration (Yorinks and Atkinson 2000; Latta 2003), some infected individuals do complete migration. In fact, migrant birds are flying sources of potential infection and we might wonder why they do not cause more disease epidemics than they appear to. Intensity of infection, suitable vectors, and susceptible potential hosts all appear to be important in determining whether the diseases of migrants will become established in resident populations in either the breeding or wintering areas. Parasites such as West Nile virus appear to

infect a broad spectrum of hosts, although not all express strong symptoms and parasites may be asymptomatic in many species.

In our study of malaria parasites of birds in the West Indies and North America, we are finding that parasites common in resident populations in the Lesser Antilles occasionally are recovered from migrant and resident populations in Missouri and elsewhere in eastern North America. Because of their low prevalence, it is difficult to say whether these parasites are established in these alternative host populations or merely represent spillover cases from normal hosts. However, the connections emphasize the potential for migrants to carry potentially epidemic disease organisms from one region to another. Analyses of parasites in resident populations in the Greater Antilles and Mesoamerica may be more informative, as there is more overlap of particular populations of migrating birds.

Because the vulnerability of a resident population to a novel parasite must depend on many genetic and other factors, it is pointless to try to predict the emergence of new diseases. Where knowledge of the capacity of a pathogen to infect a particular population is critical, the most reasonable course may be experimental infection in safe laboratories to determine vulnerability of potential host populations (Atkinson et al. 2000). It is possible that migrants, such as the Bobolink (*Dolichonyx oryzivorus*) will carry West Nile virus to the Galápagos Islands. Because this eventuality presents a potential management problem, a logical course would be to test various species of the endemic Galápagos avifauna in continental laboratories to determine their susceptibility, and thus the potential magnitude of the threat, before disaster strikes.

Phylogenetic analyses of parasite and host populations can provide estimates of the prevalence of host switching over the history of a parasite clade (Page and Hafner 1996; Ronquist 1997; Atkinson et al. 2000). Analyses of cospeciation between malaria parasites and their avian hosts suggest that parasites switch between unrelated hosts frequently enough to obscure deep historical relationships between lineages of hosts and their parasites (Ricklefs and Fallon 2002; Waldenström et al. 2002). This appears to be particularly true of *Plasmodium* compared with *Haemoproteus* (see tables 17.1 and 17.2). We have nonetheless found several lineages of *Plasmodium* in West Indian residents, Neotropical migrants, and temperate residents, indicating that migrants might play an important role in carrying disease organisms between distant populations, or even in maintaining diseases in resident populations in North America.

The region of origin of parasite populations shared by residents and migrants can be determined in principle when only tropical or temperate residents are infected. Several of the lineages identified in our work appear primarily in West Indian resident birds and occasionally in Neotropical migrants, especially the Red-eyed Vireo (*Vireo olivaceus*), which has been well sampled in our Missouri study area (tables 17.1 and 17.2, *Haemoproteus* lineages A, B, D, E). We assume that these lineages are primarily endemic to the West

Indies, and that they are occasionally picked up by Neotropical migrants but are infrequently transmitted to other migrants or residents in northern breeding areas. In contrast, several lineages (*Plasmodium* B and E) occur in clades in which most closely related parasites were recovered from temperate regions, in both residents and migrants. Thus, it is likely that these parasite lineages are temperate in origin and have been carried to the West Indies by migrants and transmitted to local resident populations.

Although predicting emerging disease is unlikely to become a precise science, several types of studies would help us to understand the process of host switching generally and may provide insights into effective management practices. If intense infections are required for the transmission of some parasites, then studies of the effects of environmental contaminants on immune system function might identify potential hazards. Several studies have highlighted the importance of dense populations of vectors in the transmission of vector-borne diseases, such as West Nile fever or malaria (Hanna et al. 1996; Hubalek 2000). Studies of vector transmission of disease have provided valuable information for the control of malaria in human populations (e.g., Shroyer 1986; Curtler et al. 1997; Lundstrom 1999) and presumably would yield insights for the control of particular diseases of wildlife populations.

Finally, the potential of genetic markers of parasites to localize host populations of long-distance migrants should be explored further. Suitable parasite markers should undergo nucleotide substitution rapidly, compared with their spread geographically through a host population. Although we would not expect the spatial distribution of such markers to remain unchanged for long periods, they might provide transient indicators of the origins of migrant populations.

Studies on the special relationship of parasites to migrants are just beginning. Although monitoring of emerging diseases is an important component of the migrant research program, it is also important to undertake basic studies of disease transmission, vector populations, and susceptibility of populations to understand the general conditions that promote host switching and the emergence of novel pathogens.

ACKNOWLEDGMENTS

Our research on avian malaria is generously supported by the U.S. National Science Foundation. We thank Pete Marra, Russ Greenberg, and several reviewers for helpful comments on the manuscript.

LITERATURE CITED

- Alekseev, A. N., H. V. Dubinina, A. V. Semenov, and C. V. Bolshakov. 2001. Evidence of ehrlichiosis agents found in ticks (Acari: Ixodidae) collected from migratory birds. *Journal of Medical Entomology* 38:471-474.
- Anderson, J. F., T. G. Andreadis, C. R. Vossbrinck, S. Tirrell, E. M. Wakem, R. A. French, A. E. Garmendia, and H. J. Van Kruiningen. 1999. Isolation of West Nile virus from mosquitoes, crows, and a Cooper's hawk in Connecticut. *Science* 286:2331-2333.
- Anderson, J. F., C. R. Vossbrinck, T. G. Andreadis, A. Iton, W. H. Beckwith, and D. R. Mayo. 2001. A phylogenetic approach to following West Nile virus in Connecticut. *Proceedings of the National Academy of Sciences USA* 98:12885-12889.
- Anderson, R. M., and R. M. May. 1991. *Infectious Diseases of Humans: Dynamics and Control*. Oxford University Press, Oxford.
- Apanius, V., N. Yorinks, E. Bermingham, and R. E. Ricklefs. 2000. Island and taxon effects in parasitism and resistance of Lesser Antillean birds. *Ecology* 81:1959-1969.
- Applegate, J. E. 1970. Population changes in latent avian malaria infections associated with season and corticosterone treatment. *Journal of Parasitology* 56:439-443.
- Atkinson, C. T., R. J. Dusek, and J. K. Lease. 2001a. Serological responses and immunity to superinfection with avian malaria in experimentally-infected Hawaii Amakihi. *Journal of Wildlife Diseases* 37:20-27.
- Atkinson, C. T., R. J. Dusek, K. L. Woods, and W. M. Iko. 2000. Pathogenicity of avian malaria in experimentally-infected Hawaii Amakihi. *Journal of Wildlife Diseases* 36:197-204.
- Atkinson, C. T., J. K. Lease, B. M. Drake, and N. P. Shema. 2001b. Pathogenicity, serological responses, and diagnosis of experimental and natural malarial infections in native Hawaiian thrushes. *Condor* 103:209-218.
- Atkinson, C. T., and C. Van Riper III. 1991. Pathogenicity and epizootiology of avian haematzoa: *Plasmodium*, *Leucocytozoon*, and *Haemoproteus*. Pages 19-48 in *Bird-Parasite Interactions: Ecology, Evolution, and Behavior* (J. E. Loye and M. Zuk, eds.). Oxford University Press, New York.
- Atkinson, C. T., K. L. Woods, R. J. Dusek, L. S. Sileo, and W. M. Iko. 1995. Wildlife disease and conservation in Hawaii: pathogenicity of avian malaria (*Plasmodium relictum*) in experimentally infected iwi (*Vestiaria coccinea*). *Parasitology* 111 (Supplement):S59-S69.
- Bennett, G. E., M. A. Bishop, and M. A. Peirce. 1993. Checklist of the avian species of *Plasmodium* Marchiafava and Celli, 1885 (Apicomplexa) and their distribution by avian family and Wallacean life zones. *Systematic Parasitology* 26:171-179.
- Bennett, G. E., M. A. Peirce, and R. A. Earle. 1994. An annotated checklist of the valid avian species of *Haemoproteus*, *Leucocytozoon* (Apicomplexa, Haemosporida) and *Hepatozoon* (Apicomplexa, Haemogregariniklae). *Systematic Parasitology* 29:61-73.
- Bensch, S., M. Stjernman, D. Hasselquist, O. Ostman, B. Hansson, H. Westerdahl, and R. T. Pinheiro. 2000. Host specificity in avian blood parasites: a study of *Plasmodium* and *Haemoproteus* mitochondrial DNA amplified from birds. *Proceedings of the Royal Society of London, Series B, Biological Sciences* 267:1583-1589.
- Chamberlain, C. P., S. Bensch, X. Feng, S. Akesson, and T. Andersson. 2000. Stable isotopes examined across a migratory divide in Scandinavian willow warblers (*Phylloscopus trochilus trochilus* and *Phylloscopus trochilus acredula*) reflect their African winter quarters. *Proceedings of the Royal Society of London, Series B, Biological Sciences* 267:43-48.
- Chamberlain, C. P., J. D. Blum, R. T. Holmes, X. H. Feng, T. W. Sherry, and G. R. Graves. 1997. The use of isotope tracers for

- identifying populations of migratory birds. *Oecologia* 109: 132-141.
- Daniels, P. W. 1995. Australian-Indonesian collaboration in veterinary arbovirology: a review. *Veterinary Microbiology* 46:151-174.
- Dhondt, A. A., D. L. Tessaglia, and R. L. Slothower. 1998. Epidemic mycoplasmal conjunctivitis in house finches from eastern North America. *Journal of Wildlife Diseases* 34:265-280.
- Dobson, A., and P. Hudson. 1995. The interaction between the parasites and predators of red grouse *Lagopus lagopus scoticus*. *Ibis* 137:S87-S96.
- Ebel, G. D., A. P. Dupuis, K. Ngo, D. Nicholas, E. Kauffman, S. A. Jones, D. Young, J. Maffei, P. Y. Shi, K. Bernard, and L. D. Kramer. 2001. Partial genetic characterization of West Nile Virus strains, New York State 2000. *Emerging Infectious Diseases* 7:650-653.
- Escalante, A. A., D. E. Freeland, W. E. Collins, and A. A. Lal. 1998. The evolution of primate malaria parasites based on the gene encoding cytochrome b from the linear mitochondrial genome. *Proceedings of the National Academy of Sciences USA* 95:8124-8129.
- Ewald, P. W. 1994. *Evolution of Infectious Disease*. Oxford University Press, Oxford.
- Fair, J. M., E. S. Hansen, and R. E. Ricklefs. 1999. Growth, developmental stability and immune response in juvenile Japanese quails (*Coturnix coturnix japonica*). *Proceedings of the Royal Society of London, Series B, Biological Sciences* 266:1735-1742.
- Fallis, A. M., S. S. Desser, and R. A. Khan. 1974. On species of leucocytozoon. *Advances in Parasitology* 12:1-67.
- Fallon, S. M., E. Bermingham, and R. E. Ricklefs. 2003. Island and taxon effects in parasitism revisited: avian malaria in the Lesser Antilles. *Evolution* 57: 606-615.
- Fallon, S. M., R. E. Ricklefs, B. L. Swanson, and E. Bermingham. 2003. Detecting avian malaria: an improved polymerase chain reaction diagnostic. *Journal of Parasitology* 89:1044-1047.
- Fedynich, A. M., D. B. Pence, and R. D. Godfrey. 1995. Hematozoa in thin blood smears. *Journal of Wildlife Diseases* 31:436-438.
- Feldman, R. A., L. A. Freed, and R. L. Cann. 1995. A PCR test for avian malaria in Hawaiian birds. *Molecular Ecology* 4:663-673.
- Fubito, I., T. Nobuhiro, M. Toshiyuki, and F. Takako. 2000. Prevalence of Lyme disease *Borrelia* spp. in ticks from migratory birds on the Japanese mainland. *Applied & Environmental Microbiology* 66:982-986.
- Godfrey, R. D. J., A. M. Fedynich, and D. B. Pence. 1987. Quantification of hematozoa in blood smears. *Journal of Wildlife Diseases* 23:558-565.
- Greiner, E. C., G. F. Bennett, E. M. White, and R. F. Coombs. 1975. Distribution of the avian hematozoa of North America. *Canadian Journal of Zoology* 53:1762-1787.
- Gurtler, R. E., J. E. Cohen, M. C. Cecere, and R. Chuit. 1997. Shifting host choices of the vector of chagas-disease, *Triatoma infestans*, in relation to the availability of hosts in houses in north-west Argentina. *Journal of Applied Ecology* 34:699-715.
- Haig, S. M., C. L. Grattotrevor, T. D. Mullins, and M. A. Colwell. 1997. Population identification of Western Hemisphere shorebirds throughout the annual cycle. *Molecular Ecology* 6:413-427.
- Hanna, J., S. Ritchie, D. Phillips, J. Shield, M. C. Bailey, J. S. MacKenzie, M. Poidinger, B. J. McCall, and P. J. Mills. 1996. An outbreak of Japanese encephalitis in the Torres Strait, Australia, 1995. *Medical Journal of Australia* 165:256-260.
- Hill, A. V. S. 2001. The genomics and genetics of human infectious disease susceptibility. *Annual Review of Genomics & Human Genetics* 2:373-400.
- Hobson, K. A., K. P. McFarland, L. I. Wassenaar, C. C. Rimmer, and J. E. Goetz. 2001. Linking breeding and wintering grounds of Bicknell's thrushes using stable isotope analyses of feathers. *Auk* 118:16-23.
- Hubalek, Z. 2000. European experience with the West Nile virus ecology and epidemiology: could it be relevant for the New World? *Viral Immunology* 13:415-426.
- Hudson, P. J., and A. P. Dobson. 1997. Transmission dynamics and host-parasite interactions of *Trichostrongylus tenuis* in Red Grouse (*Lagopus lagopus scoticus*). *Journal of Parasitology* 83:194-202.
- Hudson, P. J., A. P. Dobson, and D. Newborn. 1998. Prevention of population cycles by parasite removal. *Science* 282: 2256-2258.
- Jarvi, S. I., C. T. Atkinson, and R. C. Fleischer. 2001. Immunogenetics and resistance to avian malaria in Hawaiian honeycreepers (Drepanidinae). *Studies in Avian Biology* 2:254-263.
- Jarvi, S. I., J. J. Schultz, and C. T. Atkinson. 2002. PCR diagnostics underestimate the prevalence of avian malaria (*Plasmodium relictum*) in experimentally-infected passerines. *Journal of Parasitology* 88:153-188.
- Latta, S. C. 2003. The effects of scaley leg mite infestations on body condition and site fidelity of migratory warblers. *Auk* 120: 730-743.
- Li, J., R. A. Wirtz, C. A. McConkey, J. Sattabongkot, A. P. Waters, M. J. Rogers, and T. F. McCutchan. 1995. *Plasmodium*: genus-conserved primers for species identification and quantitation. *Experimental Parasitology* 81:182-190.
- Lundstrom, J. O. 1999. Mosquito-borne viruses in western Europe: a review. *Journal of Vector Ecology* 24:1-39.
- Marfin, A. A., and D. J. Gubler. 2001. West Nile encephalitis: an emerging disease in the United States. *Clinical Infectious Diseases* 33:1713-1719.
- Marra, P. P., K. A. Hobson, and R. T. Holmes. 1998. Linking winter and summer events in a migratory bird by using stable-carbon isotopes. *Science* 282:1884-1886.
- Massey, J. G., T. K. Graczyk, and M. R. Cranfield. 1996. Characteristics of naturally acquired *Plasmodium relictum capistranoae* infections in naïve Hawaiian crows (*Corvus hawaiiensis*) in Hawaii. *Journal of Parasitology* 82:182-185.
- McClure, H. E. 1974. *Migration and Survival of the Birds of Asia*. U.S. Army Component, SEATO Medical Project, Bangkok.
- McClure, H. E., and N. Ratanaworabhan. 1973. *Some Ectoparasites of the Birds of Asia*. U.S. Army Medical Component, SEATO Medical Project, Bangkok.
- Merila, J., M. Bjorklund, and A. J. Baker. 1997. Historical demography and present day population structure of the greenfinch, *Carduelis chloris*: an analysis of mtDNA control-region sequences. *Evolution* 51:946-956.
- Mila, B., D. J. Girman, M. Kimura, and T. B. Smith. 2000. Genetic evidence for the effect of a postglacial population expansion on the phylogeography of a North American songbird. *Proceedings of the Royal Society of London, Series B, Biological Sciences* 267:1033-1040.

- Miller, B. R., R. S. Nasci, M. S. Godsey, H. M. Savage, J. J. Lutwama, R. S. Lanciotti, and C. J. Peters. 2000. First field evidence for natural vertical transmission of West Nile virus in *Culex univittatus* complex mosquitoes from Rift Valley Province, Kenya. *American Journal of Tropical Medicine & Hygiene* 62:240–246.
- Mitchell, C. J. 1991. Vector competence of North and South American strains of *Aedes albopictus* for certain arboviruses: a review. *Journal of the American Mosquito Control Association* 7:446–451.
- Ots, I., and P. Hórák. 1996. Great tits *Parus major* trade health for reproduction. *Proceedings of the Royal Society of London, Series B, Biological Sciences* 263:1443–1447.
- Page, R. D. M. 1995. Parallel phylogenies: reconstructing the history of host-parasite assemblages. *Cladistics* 10:155–173.
- Page, R. D. M., and M. S. Hafner. 1996. Molecular phylogenies and host-parasite cospeciation: gophers and lice as a model system. Pages 255–270 in *New Uses for New Phylogenies* (P. H. Harvey, A. J. L. Brown, J. Maynard Smith, and S. Nee, eds.). Oxford University Press, Oxford.
- Peirce, M. A., and C. F. Bennett. 1996. A revised key to the avian subgenera of *Plasmodium* Marchiafava and Celli, 1885 (Apicomplexa). *Systematic Parasitology* 33:31–32.
- Perkins, S. L. 2000. Species concepts and malaria parasites: detecting a cryptic species of *Plasmodium*. *Proceedings of the Royal Society of London, Series B, Biological Sciences* 267:2345–2350.
- Perkins, S. L., and J. J. Schall. 2002. A molecular phylogeny of malarial parasites recovered from cytochrome b gene sequences. *Journal of Parasitology* 88:972–978.
- Plowright, W. 1982. The effects of rinderpest and rinderpest control on wildlife in Africa. *Symposium of the Zoological Society of London* 50:1–28.
- Pung, O. J., N. E. Maxwell, E. C. Greiner, J. R. Robinette, and J. E. Thul. 1997. *Haemoproteus greineri* in wood ducks from the Atlantic flyway. *Journal of Wildlife Diseases* 33:355–358.
- Råberg, L., M. Grahn, D. Hasselquist, and E. Svensson. 1998. On the adaptive significance of stress-induced immunosuppression. *Proceedings of the Royal Society of London, Series B, Biological Sciences* 265:1637–1641.
- Randolph, S. E., and D. J. Rogers. 2000. Fragile transmission cycles of tick-borne encephalitis virus may be disrupted by predicted climate change. *Proceedings of the Royal Society of London, Series B, Biological Sciences* 267:1741–1744.
- Rappole, J. H., S. R. Derrickson, and Z. Hubalek. 2000. Migratory birds and spread of West Nile virus in the Western Hemisphere. *Emerging Infectious Diseases* 6:319–328.
- Richard, F. A., R. N. M. Sehgal, H. I. Jones, and T. B. Smith. 2002. A comparative analysis of PCR-based detection methods for avian malaria. *Journal of Parasitology* 88:819–822.
- Richie, T. L. 1988. Interactions between malaria parasites infecting the same vertebrate host. *Parasitology* 96:607–639.
- Ricklefs, R. E., and S. M. Fallon. 2002. Diversification and host switching in avian malaria parasites. *Proceedings of the Royal Society of London, Series B, Biological Sciences* 269:885–892.
- Ricklefs, R. E., S. M. Fallon, and E. Bermingham. 2004. Evolutionary relationships, cospeciation, and host switching in avian malaria parasites. *Systematic Biology* 53:111–119.
- Rintamaki, P. T., W. Ojanen, H. Pakkala, and M. Tynjala. 1998. Blood parasites of migrating willow warblers (*Phylloscopus trochilus*) at a stopover site. *Canadian Journal of Zoology* 76:984–988.
- Roberts, S. R., P. M. Nolan, L. H. Lauerman, L. Q. Li, and G. E. Hill. 2001. Characterization of the mycoplasmal conjunctivitis epizootic in a house finch population in the southeastern USA. *Journal of Wildlife Diseases* 37:82–88.
- Ronquist, F. 1997. Phylogenetic approaches in coevolution and biogeography. *Zoologica Scripta* 26:313–322.
- Scott, J. D., K. Fernando, S. N. Banerjee, L. A. Durden, S. K. Byrne, M. Banerjee, R. B. Mann, and M. G. Morshed. 2001. Birds disperse ixodid (Acari: Ixodidae) and *Borrelia burgdorferi*-infected ticks in Canada. *Journal of Medical Entomology* 38:493–500.
- Sehgal, R. N. M., H. I. Jones, and T. B. Smith. 2001. Host specificity and incidence of *Trypanosoma* in some African rainforest birds: a molecular approach. *Molecular Ecology* 10:2319–2327.
- Service, M. W. 1991. Agricultural development and arthropod-borne diseases: a review. *Revista de Saude Publica* 25:165–178.
- Shehata, C., L. Freed, and R. L. Cann. 2001. Changes in native and introduced bird populations on O'ahu: infectious diseases and species replacement. *Studies in Avian Biology* 22:264–273.
- Sheldon, B. C., and S. Verhulst. 1996. Ecological immunology: costly parasite defences and trade-offs in evolutionary ecology. *Trends in Ecology & Evolution* 11:317–321.
- Shroyer, D. A. 1986. *Aedes albopictus* and arboviruses: a concise review of the literature. *Journal of the American Mosquito Control Association* 2:424–428.
- Sibley, C. G., and J. E. Ahlquist. 1990. *Phylogeny and Classification of the Birds of the World*. Yale University Press, New Haven.
- Smith, R. P., P. W. Rand, E. H. Lacombe, S. R. Morris, D. W. Holmes, and D. A. Caporale. 1996. Role of bird migration in the long-distance dispersal of *Ixodes dammini*, the vector of Lyme disease. *Journal of Infectious Diseases* 174:221–224.
- Super, P. E., and C. Van Riper III. 1995. A comparison of avian hematozoan epizootiology in two California coastal scrub communities. *Journal of Wildlife Diseases* 31:447–461.
- Thul, J. E., D. J. Forrester, and E. C. Greiner. 1980. Hematozoa of wood ducks (*Aix sponsa*) in the Atlantic flyway. *Journal of Wildlife Diseases* 16:383–390.
- Van Riper, C., III, S. G. Van Riper, M. L. Goff, and M. Laird. 1986. The epizootiology and ecological significance of malaria in Hawaiian land birds. *Ecological Monographs* 56:327–344.
- Waldenström, J., S. Bensch, S. Kiboi, D. Hasselquist, and U. Ottosson. 2002. Cross-species infection of blood parasites between resident and migratory songbirds in Africa. *Molecular Ecology* 11:1545–1554.
- Webster, M. S., P. P. Marra, S. M. Haig, S. Bensch, and R. T. Holmes. 2002. Links between worlds: unraveling migratory connectivity. *Trends in Ecology & Evolution* 17:76–83.
- Weissenböck, H., J. Kolodziejek, A. Url, H. Lussy, B. Rebel-Bauder, and N. Nowotny. 2002. Emergence of Usutu virus, and African mosquito-borne flavivirus of the Japanese encephalitis virus group, Central Europe. *Emerging Infectious Diseases* 8:652–656.

- Wennerberg, L. 2001. Breeding origin and migration pattern of dunlin (*Calidris alpina*) revealed by mitochondrial DNA analysis. *Molecular Ecology* 10:1111–1120.
- White, E. M., E. C. Greiner, G. F. Bennett, and C. M. Herman. 1978. Distribution of the hematozoa of Neotropical birds. *Revista de Biología Tropical* 26 (Supplement):43–102.
- Yorinks, N., and C. T. Atkinson. 2000. Effects of malaria on activity budgets of experimentally infected juvenile Apapane (*Himatione sanguinea*). *Auk* 117:731–738.
- Zeller, H. G., and B. Murgue. 2001. The role of migrating birds in the West Nile virus epidemiology [French]. *Médecine et Maladies Infectieuses* 31:168S–174S.