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LIVING WITH PARASITES: PREVALENCE OF A BLOOD PARASITE AND ITS EFFECT ON SURVIVORSHIP IN THE PURPLE MARTIN

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ABSTRACT.—We studied the prevalence of the blood parasite *Haemoproteus prognei* (Haematoprotozoa) from 1986 to 1990 in a breeding colony of Purple Martins (*Progne subis*) in Maryland and in overwintering martins in three Brazilian roosts in 1990. Yearling breeders were infected at a significantly lower rate than adults, and no yearlings in wintering roosts were infected. *Haemoproteus prognei* might be more virulent in immunologically naive birds and cause high mortality in young birds during the stress of their first migration. Many birds became infected over three years and most maintained a chronic infection. Infected birds returned to the colony with the same frequency as uninfected birds. Infected adults tended to arrive at the breeding site ahead of uninfected adults. Clutch size did not differ, but uninfected females had lower breeding success than infected individuals. We discuss the evolutionary implications of high mortality coupled with superior breeding success in chronically infected birds, whose immune system has been "tested" for parasite resistance, and suggest an alternative to the predominant view that parasite resistance means avoiding them altogether. *Received 2 March 1992, accepted 27 July 1992.*

THE DEBILITATING effects of parasites underlie many recent theories in evolutionary biology. They have been implicated in regulating vertebrate host populations (Anderson and May 1978, 1979, May and Anderson 1979, 1983), in sexual selection (Hamilton and Zuk 1982, Jaenike 1988, Zuk 1991), in the evolution of sex (Hamilton 1980), and in ecological and behavioral changes in host populations (van Riper et al. 1986).

Parasites have potential effects on the survival and fitness of hosts (Ewald 1983, Atkinson and van Riper 1991). Chronic parasitic infections may cause nutritional stress and mortality by decreasing the foraging ability of an infected animal (Brooke 1945, Park 1948, Chandler 1953, Jenkins et al. 1963), and affect host mating success (Jenkins et al. 1963, Dolinsky et al. 1981, Freeland 1981, Rau 1983, Borgia 1986, Møller 1990). Infected individuals might be more susceptible to predation than healthy individuals in a population (Van Dobben 1952, Holmes and Bethel 1972, Vaughn and Coble 1975). In injured raptors, hemoprotozoal infection prolongs the rehabilitation time necessary before release (Olsen and Gaunt 1985).

In contrast, parasitologists tend to emphasize coevolution between parasite and host (Holmes 1983, Hafner and Nadler 1988, Toft 1991) and suggest that most parasitic infections in vertebrates are relatively benign (Bennett et al. 1988, Cox 1989). Furthermore, several studies have not found debilitating effects of blood parasites on their avian hosts (e.g. Weatherhead and Bennett 1991, 1992). Here, we show that both host-debilitating and host-benign views of parasitic infection are probably correct, even within the same host–parasite interaction. We emphasize that, in defining what is meant by parasite resistance, avoiding blood-parasite infection may not be as evolutionarily important to vertebrate hosts as overcoming, and living with, infection.

We initiated a study in 1986 to understand the effects of internal parasites on the survival and lifetime reproductive success in Purple Martins (*Progne subis*). Purple Martins breed colonially in North America and winter in Brazil (AOU 1983). Allen and Nice (1952) described their breeding biology, and Morton and Derrickson (1990) discussed migration arrival schedule. Here we describe the prevalence of a malarialike protozoan parasite, *Haemoproteus prognei*, in the Purple Martin. *Haemoproteus prognei* also infects the Lesser Striped Swallow (*Hirundo abyssinica*), the Red-rumped Swallow (*H. daurica*), and the Barn Swallow (*H. rustica*) in Africa and Southeast Asia, but the Purple Martin is the only host known in the Western Hemisphere (White and Bennett 1978). The vector is unknown, probably a biting midge (*Culi-*

TABLE 1. Prevalence of *Haemoproteus prognei* infection in adult and yearling Purple Martins (*Progne subis*) in early July from 1986 to 1990 in Maryland.[a]

Infection status	All birds				Returns				Immigrants			
	ASY		SY		ASY		SY		ASY		SY	
	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀
Infected	32	32	16	9	25	27	5	2	7	5	11	7
Uninfected	52	56	73	47	46	32	18	8	6	24	55	39
Total	84	88	89	56	71	59	23	10	13	29	66	46
Percent infected	38	36	18	16	35	46	22	20	54	17	17	15

[a] ASY = adult, after second year, two years or older; SY = second year (yearling breeder).

coides sp.) or a hippoboscid fly (Bennett 1960). The age and sex-specific prevalence of this parasite in martins is examined and some life-history consequences for the birds are discussed.

METHODS

A colony of Purple Martins was founded in 1976 in Severna Park, Anne Arundel County, Maryland. Knowledge of arrival date, mate choice, and breeding success of individuals provides a background to this study (Morton and Patterson 1983, Morton 1987, Morton et al. 1990, Morton and Derrickson 1990). Breeding yearlings, in their second calendar year of life (termed second year or SY), are differentiated by their subadult plumage from birds in their second breeding season or older (after second year or ASY; Niles 1972). Some SY females cannot be differentiated from older females by plumage (Morton unpubl. data).

We captured breeding adults each year between 2200 and 0300 EST in early July for banding with colored plastic leg bands and U.S. Fish and Wildlife Service metal bands to permit recognition of individuals. Nests contain young by this date. Nestlings near fledging at 25 to 28 days posthatching were also sampled. From 1986 through 1990, one or two blood smears per breeding adult were obtained from a drop of blood squeezed from a clipped toenail onto a glass slide. The slide was labelled with the band number and sex of the bird, fixed in methanol, and stained. Several staining procedures were used to screen for blood parasites and found to be adequate for identification. In 1986 and 1990, the smears were stained with Giemsa and, in 1988 and 1989, Leishman's stain was used, following the protocol of Chatterjee (1977). In 1987, a formalin Wright's staining protocol was used (Santamarina 1964).

A 100× oil-immersion lens was used to detect intraerythrocytic stages of *H. prognei*. About 50 microscopic fields were scanned before a negative was declared. For positive slides, several microscopic fields were selected at random, and the number of infected red blood cells (RBC) per 600 uninfected RBCs was recorded.

We knew when adults returned to the breeding site after migration and used these data to test the hypothesis that parasite-infected birds arrived later than uninfected birds. For analysis, we used the Statistical Analysis Systems package (PC-SAS; SAS Institute 1985) and tests in Sokal and Rohlf (1981). The general-linear-model (GLM) procedure was used to examine variation in return date among different adult age classes, between sexes, and among years with regard to parasite infection status.

RESULTS

Parasite prevalence in breeding colony.—For 317 blood samples taken over five years, the average annual prevalence of *H. prognei* was 28% (Table 1). In 1986, 22% of the population was infected, 18% in 1987, 27% in 1988, 26% in 1989, and 42% in 1990.

Age-specific prevalence.—There was a significant difference in the prevalence of infection in ASY birds versus SY birds (Table 1). Only 17% of SYs were infected (25/145), whereas 37% of ASYs were infected (64/172). Of those that were infected, there was no difference in parasitemia between the sexes of ASY or SY birds (Tables 1 and 2).

The colony had an average return rate of 55%. From 1979 to 1988, 291 of 527 breeding birds had bred in the colony in at least one previous year. Almost one-half the birds screened for parasites were returns, birds that had been banded in previous years at the breeding colony (Table 1). All SY birds listed as returns were banded as nestlings the previous year. In SY birds there was no difference in number that returned to their natal colony compared to those new to the colony in infection status for either sex (males, $X^2 = 0.324$, df = 1, ns; females, $X^2 = 0.145$, df = 1, ns). Comparing ASY returns and immigrants, ASY male immigrants and returns did not differ (males, $X^2 = 1.598$, df = 1, ns). Some female immigrants may have been SY

TABLE 2. Prevalence of *Haemoproteus prognei* infection in Purple Martins by age and sex class.[a] Only banded ASY and SY birds that could be aged are included.

Age and sex class[b]	Percent red blood cells infected						*n*	Percent infected
	0	0–1	>1–2	>2–3	>3–10	10+		
SY ♂	72	8	7	2	0	0	89	19
SY ♀	16	0	1	1	0	2	20	20
ASY ♂	52	24	6	1	0	0	83	37
ASY ♀	19	15	0	1	2	0	37	49

[a] Kolmogorov-Smirnov two-group test for differences in infection rate in martins by age and sex: ASY vs. SY, $D_{max} = 0.216$, $P < 0.004$; ASY males vs. ASY females, ns; SY males vs. SY females, ns.

[b] ASY = adult, two years or older; SY = yearling breeder.

birds misclassified as ASY, so we are unable to compare them to ASY female returns.

Birds first screened in 1986–1988 that returned for at least three subsequent years of the 1986–1990 study period did not differ in infected status ($n = 34$, $G = 0.54$, df = 1, ns; Sokal and Rohlf 1981). This suggests that a bird with a chronic infection of *Haemoproteus* does not differ from one not infected in its ability to survive to the next year. If an adult did not return to the colony for three years it had probably died (Morton and Derrickson 1990).

No nestlings were infected even though all screened were near fledging at 25 to 28 days posthatching (Table 3). The majority of the infected birds had low parasitemia (<1% of RBCs; Tables 2 and 3). Only two birds, both SY females, had more than 10% parasitemia (Table 2). Of nine birds with a higher infection rate (>2% parasitemia), three were ASY females, three SY females, two SY males, and one an ASY male. Three of these birds, all females, were not recovered in subsequent years, indicating they may have not survived. Two ASY females had low parasitemia the next year and one turned negative. In addition to *Haemoproteus*, an unidentified blood microfilariad nematode, an air-sac nematode (*Diplotraena* sp.), and an unidentified intestinal cestode were found.

Between-year changes in infected status.—Of 30 birds sampled each year from 1986–1988, 9 infected birds (positives) remained positive for all years, 12 uninfected birds (negatives) stayed negative, 7 negatives turned positive, and 1 positive turned negative. In addition, one turned from positive to negative and back to positive (possibly a false negative in the intervening year).

Date of migratory return to colony in relation to infected status.—Since we knew the date that individual martins returned to the colony in the spring (Morton and Derrickson 1990), we tested the hypothesis that infection with *Haemoproteus* hampered the ability of infected birds to return as early as uninfected individuals. There was no difference between infected and uninfected birds in their return to the colony in any year from 1986–1990 (ANOVA; $F = 1.12$,

TABLE 3. Age-specific prevalence of *Haemoproteus prognei* in Purple Martins over five years. Only birds of known age included. Each year considered an independent data point even though many individuals were sampled more than once from 1986–1990.

Age[a]	Percent red blood cells infected						*n*	Percent infected
	0	>0–1	>1–2	>2–3	>3–4	10+		
25–28 days	35	0	0	0	0	0	35	0
1	117	12	9	4	0	2	144	19
2	19	14	2	0	0	0	35	46
3	10	4	0	0	0	0	14	29
4	4	1	1	0	0	0	6	33
5	3	1	0	0	0	0	4	25
6	2	2	0	0	0	0	4	50
7	1	0	1	0	0	0	2	50
8	0	1	0	0	0	0	1	100
9	0	0	1	0	0	0	1	100
Total	191	35	14	4	0	2	246	
Percent	78	14	6	2	0	1		

[a] Years unless otherwise indicated.

TABLE 4. Effects of *Haemoproteus prognei* on brood reduction in older female Purple Martins (age ≥2 years) from 1986–1990 at a breeding colony in Maryland.

	Infected	Not infected
Mean eggs/clutch (±SD)	4.97 ± 0.65	4.96 ± 0.80
Mean young fledged (±SD)	4.13 ± 1.45	3.81 ± 1.33
Individual clutches		
With brood reduction (%)	11 (34)	30 (63)
Without brood reduction (%)	21 (66)	18 (38)
No. females	32	48

$P < 0.29$). When infected status, sex, and age were considered, a significant relationship was found with return date (ANOVA; $F = 47.71$, $P < 0.001$, $r^2 = 0.82$), but this was due almost entirely to age alone (ANOVA; $F = 43.44$, $P < 0.001$, $r^2 = 0.84$), since yearlings arrive about 30 days later than older age classes (Morton and Derrickson 1990). These data support the analysis in Tables 1, 2, and 3, showing that yearling birds were uninfected relative to older birds.

Another way to examine the relationship between infected status and date of return is to compare the average day of return for each year. From 1986–1989, infected ASY birds averaged 4.7, 5.5, 5.7, and 10 days earlier than uninfected birds in return to the colony. In 1990, infected birds returned 1.7 days later than uninfected birds. None of these differences in average return day was significant on a within-year basis. The hypothesis that chronic infection slows the birds in migration is not supported.

Breeding success of females.—We compared the clutch sizes and number of young fledged in females with or without infection with *Haemoproteus*. Yearling females were not included because they laid smaller clutches and had lower nesting success compared to older females (Allen and Nice 1952) and because this difference was due in part to lower yearling-male parental care (Morton et al. 1990) rather than to infected status. There was no difference in the average clutch sizes of infected compared to uninfected females, but uninfected females suffered more brood reduction than infected females ($X^2 = 6.747$, df = 1, $P < 0.01$). Infected females fledged significantly more young on average than uninfected females ($t = 3.13$, df = 1, $P < 0.01$; Table 4).

Data for eight females that changed infected status between years (positive to negative = 2; negative to positive = 4; positive to negative to positive = 2) showed a nonsignificant within-individual difference in reproductive success ($T = 8$, $n = 8$, $P > 0.05$; Wilcoxon matched-pairs signed-ranks test; Siegel 1956). While infected they averaged 4.84 eggs laid to 4.14 (86%) young fledged; while uninfected they averaged 4.65 eggs laid to 3.5 (75%) young fledged.

TABLE 5. Prevalence of *Haemoproteus prognei* in Purple Martins from three overwintering roosts in Brazil.[a]

	ASY		SY	
	Males	Females	Males	Females
Infected (%)	4 (24)	5 (29)	0 (0)	0 (0)
Uninfected (%)	13 (76)	12 (71)	5 (100)	8 (100)

[a] Difference in prevalence between ASY and SY birds using G-test (Sokal and Rohlf 1981). $G = 9.98$, df = 1, $P = 0.01$.

Prevalence of Haemoproteus *in overwintering martins.*—Purple Martins roost gregariously in large, mixed-species roosts in the central parks of large cities in the interior of the Brazilian state of Sao Paulo. We obtained blood smears from martins in Ribeirao Preto, Barretos, and Sao Jose do Rio Preto, each with roosts of 10,000 to 20,000 birds. The Brown-chested Martin (*Phaeoprogne tapera*) made up about 70% of birds in the roosts, followed by the Purple Martin (ca. 29%) and a few Gray-breasted Martins (*Progne chalybea*; M. Levy, J. R. Hill, E. S. Morton, B. J. Stutchbury, and L. D. Vizotto pers. observ.). The prevalence of *Haemoproteus* sp. in wintering Purple Martins in early February 1990 was compared (Table 5). The prevalence of infection in the combined age and sex classes of Purple Martins was 24%. As in the breeding colony, a significantly higher percentage of ASY than SY birds were infected (36% vs. 0%; Table 5). None of the 44 Brown-chested Martins examined was infected with any blood parasite.

DISCUSSION

Possible reasons for low infection in SY martins.—Our finding that SY Purple Martins have a low

prevalence of infection suggests either that fewer of them were exposed to infection or, more likely, that the infection is more virulent in these immunologically naive birds. High mortality due to *H. prognei* would lower the percentage of infection found in surviving SY birds. Initial infections are relatively virulent, and younger age classes are more likely to die from infections (Herman et al. 1975, Gabaldon and Ulloa 1980, Atkinson 1991, Atkinson and van Riper 1991). Initial infections may be more deadly in fledgling birds undergoing their first migration than in experienced adults.

Furthermore, it is unlikely that SY and ASY martins differ in their exposure to infection because all age and sex classes roost gregariously in large, traditional sites during migration in North and Middle America (Dickerman et al. 1980; pers. observ.). Many roosts are occupied continuously by martins for two to three months during migration, affording local populations of arthropod vectors ample time for infection and transmission.

The same is true at breeding colonies, where infected martins are available to potential vectors for about 4.5 months at our study colony in Maryland. The time it takes between vector inoculation of *H. prognei* into a martin and the parasite's appearance in blood samples is unknown. However, it is likely similar to the 17-day period between inoculation and appearance in blood found for *H. meleagridis* in domestic turkey (*Meleagris gallopavo*) poults (Atkinson et al. 1988), 20 days in young California Quail (*Callipepla californica*; Herman 1968), or 17 days for *H. nettionis* in domestic Mallard (*Anas platyrhynchos*) ducklings (Julian and Galt 1980). All 24 fledgling Common Grackles (*Quiscalus quiscala*) examined by Kirkpatrick et al. (1991) were infected with *H. quiscalus*. Martins brood nestlings at night until nestlings reach about 14 days of age. Nestlings we screened were not infected. However, if brooding offers some protection from nocturnal vectors, there would not be sufficient time for infection to show in blood smears in 24- to 28-day-old nestlings, even though they might have been infected.

Therefore, it is not likely that SY birds are less infected than ASYs because they lack exposure to the parasite. Instead, if yearlings are infected, then the low incidence of infection we found in them during the breeding season and in overwintering roosts would indicate high mortality. *Haemoproteus prognei* might be transmitted in the northern latitudes and birds might acquire the infection in fall prior to or during migration. High mortality in the yearling age class would be an important source of selection.

Possible relationship between roosting habits and parasites.—The habit of martins roosting in large cities in Brazil has been acquired in the last 30 years (L. D. Vizotto pers. comm.). In Manaus, a large roost occurs inside buildings housing an oil refinery. The martins spend the night on pipes almost too hot for a human to touch and with air polluted so badly as to be nearly unbreathable (T. Saniotti pers. comm.). Urban roosts do not necessarily offer protection from predators: house cats (*Felis catus*), American Kestrels (*Falco sparverius*), and humans kill birds that use roosts of this type (pers. observ.). However, biting midges are absent or rare at such sites (pers. observ.). It is possible that high mortality from initial infections with *Haemoproteus* has favored the new use of vector-free roosting sites in Brazil.

The relationship between chronic infection, fitness, and sexual selection.—As widely reported for other species, chronic infection appears to have no effect on the survival of martins if they survive initial infection. There is equal probability of infected versus uninfected ASY birds returning to the colony. One-third of the birds with higher parasitemias in one year returned to the colony with a lower parasitemia the next year. Indeed, chronically infected females fledged more young per clutch than females showing no infection. There is no relationship between longevity and infected status for ASY birds. The data presented here suggest that adults able to live with infection have good immune systems overall relative to those individuals that have not been "tested" for resistance to *H. prognei*.

The immune system response to infection with *Haemoproteus* offers a source of selection. The genes of the major histocompatibility complex (MHC) are the most polymorphic known (Klein 1986). Initially, most of the variation was considered neutral, but this is no longer considered tenable (Hill et al. 1991, Howard 1991). Heterozygosity is high (Hedrick et al. 1991) and overdominant selection seems a reasonable cause since, given the importance of the ability of the MHC to respond to a vast array of foreign proteins, heterozygosity should be favored (Nei and Hughes 1991). Studies of susceptibility in chickens (*Gallus gallus*) to viral-induced tumors (Marek's disease) showed that particular *B* al-

loalleles of the MHC conferred resistance (Briles et al. 1977). The MHC *B* produces resistance to the bacterium causing fowl cholera (Lamont et al. 1987). Hill et al. (1991) showed that human malaria had selected for an unusually high incidence of rare MHC genes in West African children, which provided as much protection from severe malaria as the sickle-cell haemoglobin variant.

For haemosporidians, it is likely that the MHC immune response targets the cell surface of the sporozoite, the stage of the life cycle that infects the vertebrate host (Hughes 1991). However, at least for malaria (*Plasmodium* sp.), the parasite can avoid the host immune response through antigenic variation, antigenic polymorphism, antigenic mimicry, and direct interference with the generation of the host immune response (Pereira da Silva 1990). Most avian species can host multiple infections of *Plasmodium, Leucocytozoan,* and *Haemoproteus,* indicating little immunological cross-reaction (Atkinson and van Riper 1991). Therefore, it is significant that we found only *H. prognei* infecting Purple Martins, since *Leucocytozoon* and *Plasmodium* are also known to infect them (Greiner et al. 1975). *Haemoproteus* sp. are the most host-specific of the haemosporidians, supporting the idea that birds with chronic infections of *H. prognei* may have inherited MHC genes that target this parasite only. The use of markers to identify the existence of MHC genes in martins specific to *H. prognei* might be used to test this hypothesis.

We suggest that *H. prognei* creates a window of vulnerability in the Purple Martin's life history that has affected aspects of mate choice, as well as roosting behavior. The low incidence of infected yearlings means that most have not been tested for their MHC resistance to *H. prognei.* SY females form pair bonds with SY males since most ASY males are already paired before SY birds arrive at breeding colonies (Morton and Derrickson 1990). However, SY females obtain most male genes for their offspring (ca. 71%) from ASY males (Morton et al. 1990). We suggest that SY females prefer genes from adult males because ASY birds have been tested for their MHC-induced ability to live with infections of *H. prognei.* MHC-based mating preferences are known for house mice (*Mus musculus;* Potts et al. 1991), and it is not unreasonable to propose that such preferences might be found in birds, particularly when one host and one parasite species are interacting evolutionarily.

Since Stutchbury (1991, 1992) has shown that the dark ASY plumage confers no dominance advantage, an alternative explanation for the definitive glossy-purple plumage of ASY males is that it marks them as possibly carrying genes for resistance to *H. prognei.* Females may choose genes from "tested" (i.e. older) males, reducing selection favoring adult plumage in SY males because, even though they pair, they contribute a smaller proportion of genes to the offspring they care for than do adult males (Morton et al. 1990). This presupposes that there are disadvantages to the adult male plumage that, in balance, will favor delaying it. One idea is that selection promotes individual recognition in SY male martins as they compete within their age class for breeding sites (Morton and Derrickson 1990). Accordingly, their breeding plumage is highly variable (Rohwer and Niles 1979). Stutchbury (1991) suggested that the dull SY male plumage might enhance survival in winter foraging flocks and roosts, since this plumage is similar to that of other martin species in these roosts.

Birds, unlike mammals, might be more prone to use vision to communicate age-related probabilities that individuals have been tested for their ability to live with blood parasites. We suggest that highly species-specific evolutionary relations with *Haemoproteus* might be sought in other passerines in which males breed in subadult plumage (Rohwer and Butcher 1988).

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