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SEASONAL PREVALENCE OF A HAEMATOZOAN PARASITE OF RED-BELLIED WOODPECKERS (*MELANERPES CAROLINUS*) AND ITS ASSOCIATION WITH HOST CONDITION AND OVERWINTER SURVIVAL

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Abstract.—We examined seasonal prevalence of a haematozoan parasite (*Haemoproteus velans*) of the Red-bellied Woodpecker (*Melanerpes carolinus*) in the Apalachicola National Forest, northern Florida. We also investigated how infection with *H. velans* was associated with host mass, body condition, and overwinter survival. Analysis of blood smears taken from individual woodpeckers between May 2000 and July 2001 indicated that prevalence of *H. velans* peaked in July 2000, at ~80% of individuals sampled, decreased to 0% in January and February 2001, and peaked again in July 2001, at ~50% of individuals. Infection with *H. velans* was associated with low mass and poor body condition in males. Infection showed no association with female mass. In addition, infection with *H. velans* showed no relationship with overwinter survival. Our data reemphasize the importance of considering seasonal variation in parasite prevalence during testing for haematozoa. In addition, our data suggest that, although infection with *H. velans* is associated with poorer host condition, it does not negatively affect host survival. *Received 15 March 2002, accepted 22 October 2002.*

Resumen.—Evaluamos la prevalencia estacional de un parásito hematozoo (*Haemoproteus velans*) del pájaro carpintero *Melanerpes carolinus* en Apalachicola National Forest, norte de Florida. También investigamos cómo se asociaba la infección con *H. velans* con el peso, condición corporal y supervivencia a través del invierno del hospedero. Análisis de muestras de sangre tomadas de carpinteros individuales entre mayo de 2000 y julio de 2001 indicaron un pico en la prevalencia de *H. velans* en julio de 2000, cuando se presentó en ~80% de los individuos muestreados. Luego hubo una disminución a 0% en enero y febrero de 2001 y se presentó un nuevo pico en julio de 2001, con ~50% de los individuos. La infección con *H. velans* estuvo asociada con bajo peso corporal y condición física pobre en los machos, pero no mostró asociación con el peso de las hembras. Adicionalmente, la infección con *H. velans* no presentó relación alguna con la supervivencia a través del invierno. Nuestros datos enfatizan nuevamente la importancia de considerar la variación estacional en la prevalencia parasitaria al realizar pruebas para hematozoos. Nuestros datos también sugieren que aunque la infección con *H. velans* está asociada con una peor condición corporal, no afecta negativamente la supervivencia del hospedero.

Interest in the potential role of parasites in the evolution and ecology of their hosts has grown substantially since Hamilton and Zuk (1982) proposed that mate choice based on parasite resistance is an important mechanism in sexual selection. Hamilton and Zuk's (1982) hypothesis proposed that females choose mates on the basis of condition-dependent secondary sexual characters that advertise parasite-resistant genotypes. That hypothesis was based on data suggesting a positive correlation between plumage brightness and the prevalence of protozoan blood parasites (haematozoa) in passerine birds. Much debate has followed about the evidence for Hamilton and Zuk's hypothesis (Hamilton and Zuk 1989, Read and Harvey 1989, Zuk 1989) and the practical issues involved in testing it (Cox 1989; Weatherhead and Bennett 1991, 1992). Part of that debate concerns the potential for haematozoan parasites to affect host fitness and the ability of researchers to detect chronic haematozoan infections (Cox 1989, Weatherhead and Bennett 1991). More recently, a trade-off between reproductive effort and resistance to haematozoan parasites has

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been proposed as a physiological mechanism potentially involved in mediating the cost of reproduction in birds (Norris et al. 1994, Richner et al. 1995, Oppliger et al. 1997, Nordling et al. 1998). That trade-off is thought to arise when limited resources must be partitioned between reproductive effort (i.e. number of offspring) and disease resistance. Growing evidence for a parasite-mediated cost of reproduction in birds has caused ecologists to reconsider the role of disease in population dynamics and life-history evolution (Price 1991).

Although the role of haematozoan parasites in avian evolutionary ecology has engendered much interest, the biology of haematozoa in the wild has not been well studied. Much of what is known about avian haematozoa has been learned from laboratory studies of domestic animals or from studies of the effects of haematozoa on novel hosts (van Riper et al. 1986, Atkinson and van Riper 1991). Poor understanding of haematozoan biology in wild populations of natural host species has raised doubts about the role of those parasites in the ecology and evolution of their hosts. Specifically, seasonal variation in parasite prevalence and the paucity of evidence for fitness effects have caused some authors to question the utility of using haematozoa to test for parasite-mediated sexual selection and life-history trade-offs (Cox 1989; Weatherhead and Bennett 1991, 1992; Sheldon and Verhulst 1996).

Several studies have demonstrated the tendency of haematozoan infections to increase in frequency during the breeding season (for example, Weatherhead and Bennett 1991, Hatchwell et al. 2000). That trend is often referred to as the spring relapse and is thought to occur when hormone activity or the physiological stress associated with breeding causes relapse of chronic infections (Atkinson and van Riper 1991). Weatherhead and Bennett (1992) have suggested that spring relapse hinders the ability of researchers to determine accurately, from a blood smear, whether an individual is infected. Blood smears can only be used to detect circulating haematozoa, but noncirculating chronic infections may persist in the livers of infected hosts (Jarvi et al. 2001). Noncirculating chronic infections may not be detected from blood smears if the individual is sampled before or after the peak of the relapse. Therefore, we must understand how parasite prevalence varies seasonally to maximize our ability to detect infections from blood smears.

The existence of parasite-mediated sexual selection or a parasite-mediated cost of reproduction requires that the parasites involved negatively affect host fitness (Hamilton and Zuk 1982, Sheldon and Verhulst 1996). Laboratory studies on domestic species have shown haematozoa to have significant pathological effects on their hosts (Atkinson and van Riper 1991). In addition, *Plasmodium*, the haematozoan parasite that causes avian malaria, has been shown to limit the geographic distribution and abundance of Hawaiian land birds (van Riper et al. 1986). Few studies have assessed the effect of haematozoa on host fitness in the wild, however, and evidence for negative associations between haematozoan infection and measures of host condition, which are often thought to reflect fitness, is mixed. Some studies have found no effect of haematozoa on survival rates or condition (reviewed by Bennett et al. 1988, 1993; Davidar and Morton 1993), whereas other studies have shown haematozoan parasites to affect negatively the immune and condition indices and the reproductive success of their hosts (Ots and Horak 1998, Merino et al. 2000).

As part of a larger study examining the importance of a parasite-mediated cost of reproduction in the Red-bellied Woodpecker (*Melanerpes carolinus*), we examined the ecology of a haematozoan parasite (*Haemoproteus velans*) in a population of Red-bellied Woodpeckers in the Apalachicola National Forest, northern Florida. In particular, we investigated (1) how the prevalence of *H. velans* varies seasonally in that population, (2) how infection with that parasite is associated with host condition, and (3) whether infection with *H. velans* affects host survival. We must understand those aspects of the biology of *H. velans* to assess the importance of a parasite-mediated cost of reproduction in the Red-bellied Woodpecker.

The Red-bellied Woodpecker is one of the most abundant woodpeckers in eastern North America. It is distributed across the eastern United States, occurring west to the Great Plains and north to southern Ontario (Shackelford et al. 2000). Red-bellied Woodpeckers generally nest in dead trees, but in the Apalachicola National Forest many use cavities excavated by Red-cockaded Woodpeckers (*Picoides borealis*) in living pines. *Haemoproteus velans* has been described in a number of woodpecker species, and it may be distributed throughout the worldwide range of the Picidae (Greiner et al. 1977). The lifecycle of *Haemoproteus* is similar to that of other haematozoa (Atkinson and van Riper 1991). Asexual reproduction occurs in noncirculating cells of the host and vector. Gametocytes are found in circulating blood cells, which are infective to bloodsucking vectors. Gametogenesis and sexual reproduction usually take place in the midgut of the vector. Species of *Haemoproteus* are the most common and probably the least pathogenic of the avian haematozoa, although little is known of the biology of *Haemoproteus* in the wild (Atkinson and van Riper 1991).

METHODS

Blood samples were collected by M.S.S. from 196 adult Red-bellied Woodpeckers in the Apalachicola National Forest between May 2000 and July 2001. Most of the 151 individuals sampled between May 2000 and April 2001 were removed from cavities excavated by Red-cockaded Woodpeckers by E.L.W. and M.S.S. as part of another study. In addition, 25 males and 20 females were caught at nests between May and July 2001. Each individual was banded with a unique combination of three color bands and a federal aluminum band. For each individual, ~100 µL of blood was drawn from a brachial vein into a heparinized microhematocrit tube. That sample was used to prepare blood smears on two microscope slides according to the technique described by Bennett (1970). Blood smears were air-dried and fixed in absolute methanol. Birds were weighed with a spring balance to the nearest 0.1 g, and their tarsi were measured to the nearest 0.1 mm. The tarsus length of some birds was not measured. Those birds were excluded from analyses that involved tarsus length.

Blood smears were stained with Giemsa stain within approximately one week of being taken. Slides were examined for the presence of haematozoa with a conventional light microscope at 400 and 1000× under oil emersion by M.S.S. The entire blood smear was scanned for infections so that low-intensity infections would not be missed. Although screening blood smears for haematozoa is a relatively easy way to detect patent infections, subpatent infections may be impossible to detect by microscopy (Jarvi et al. 2001). For the remainder of this article, we refer to individuals with infections detected from blood smears as infected and individuals without detectable infections as uninfected, although those individuals may have subpatent infections that we were not able to diagnose from blood smears. We estimated the condition (mass corrected for size) of each individual from the residuals of an ordinary least-squares regression of ln(mass) on ln(tarsus). That type of analysis can be used to estimate body condition while controlling for the effect of body size (Jakob et al. 1996). A separate regression was performed for each sex. Mass and body condition of infected and uninfected individuals were compared by ANOVA.

We estimated overwinter survival by recapturing or resighting adults banded at 23 sites during 2001 in spring and summer of 2002. Our estimate of overwinter survival is actually a function of survival, dispersal, and recapture probability, but here we assume that individuals not captured or seen during the 2002 breeding season were dead. We examined the association between infection with *H. velans* and overwinter survival with *G*-tests with William's correction for small sample sizes (Sokal and Rohlf 1995).

RESULTS

Approximately 25% of all individual woodpeckers sampled were infected with *H. velans.* There was no difference in percentage of males (26%, $n = 126$) and females (22%, $n = 70$) infected with *H. velans* (*G*-test with William's correction, $G_{adj} = 0.436$, df = 1, $P > 0.05$). No other species of haematozoa were found in any of the blood smears. The proportion of individuals infected with *H. velans* varied seasonally; peaks occurred in July 2000 and July 2001 (Fig. 1).

A larger proportion of males was infected during the breeding season than during the nonbreeding season (*G*-test with William's correction, $G_{adj} = 20.03$, df = 1, $P < 0.001$). Males infected with *H. velans* weighed less than uninfected males ($\bar{x}_{\text{infected}} = 71$ g, $\bar{x}_{\text{uninfected}} = 73.5$ g; ANOVA, $F = 8.71$, df = 1 and 122, $P = 0.004$). Male tarsus length and male mass were significantly positively related, although tarsus length explained little of the variation in mass ($r^2 = 0.116$, $P < 0.005$). Infected males were in poorer condition than uninfected males ($\bar{x}_{\text{infected}} = -0.023$, $\bar{x}_{\text{uninfected}} = 0.008$; ANOVA, $F = 6.72$, df = 1 and 110, $P = 0.011$). Because male mass and condition vary seasonally in the Apalachicola National Forest population (M. S. Schrader unpubl. data), and because very few males were infected outside of the breeding season, we examined the association between infection with *H. velans* and male mass and condition using only males sampled during the breeding season (May–September 2000 and April–July 2001). Males infected with *H. velans* weighed less than uninfected males in the data set limited to breeding-season males ($\bar{x}_{\text{infected}} =$

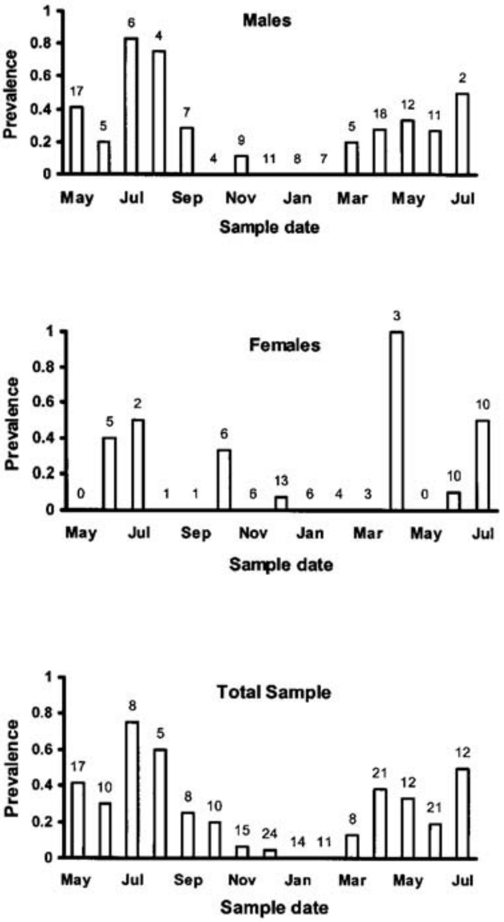

FIG. 1. Seasonal variation in prevalence (proportion of individuals with detectable infections) of *Haemoproteus velans* infections in 126 males, 70 females, and the total sample (196 individuals). Individuals were collected between May 2000 and July 2001. Parasite prevalence peaked in July 2000 and July 2001 and was at a minimum during the intervening winter. Values above bars are sample sizes.

70.9 g, $\bar{x}_{\text{uninfected}}$ = 73.4 g; ANOVA, $F = 6.92$, df = 1 and 81, $P = 0.01$). New condition values were calculated from the breeding-season data. Male mass and tarsus length were positively related during the breeding season ($r^2 = 0.14$, $P = 0.001$). Infected males were in poorer condition than uninfected males in this data set ($\bar{x}_{\text{infected}}$ = –0.018, $\bar{x}_{\text{uninfected}}$ = 0.010; ANOVA, $F = 4.25$, df = 1 and 68, $P = 0.043$).

Although parasite prevalence did not follow an obvious seasonal trend in females (Fig. 1), a higher proportion of females was infected during the breeding season than during the nonbreeding season (*G*-test with Williams correction, $G_{\text{adj}} = 9.09$, df = 1, $P = 0.01$). Females infected with *H. velans* did not differ significantly in mass from uninfected females ($\bar{x}_{\text{infected}}$ = 67.6 g, $\bar{x}_{\text{uninfected}}$ = 66.7 g; ANOVA, $F = 0.68$, df = 1 and 68, $P = 0.413$). Because female mass and tarsus length were not significantly related ($r^2 = 0.023$, $P = 0.303$), we did not calculate condition for females. We also examined association between infection with *H. velans* and female mass using only females sampled during the breeding season. In that limited data set, female mass and infection were not associated ($\bar{x}_{\text{infected}}$ = 67.04 g, $\bar{x}_{\text{uninfected}}$ = 66.2 g; ANOVA, $F = 0.33$, df = 1 and 31, $P = 0.569$). Female mass and tarsus length were not significantly related in that limited data set ($r^2 = 0.003$, $P = 0.767$), so we did not calculate condition for females.

Infection with *H. velans* had no effect on the probability that males would survive from the 2001 breeding season to the 2002 breeding season (4/8 infected [50%], 9/11 uninfected [82%], *G*-test with William's correction, $G_{\text{adj}} = 1.98$, df = 1, $P > 0.05$). Similarly, infection had no effect on the probability that females would survive from the 2001 breeding season to the 2002 breeding season (3/6 infected [50%], 6/15 uninfected [40%], *G*-test with William's correction, G_{adj} = 0.16, df = 1, $P > 0.05$).

DISCUSSION

Our results clearly demonstrate that the prevalence of *H. velans* varies seasonally in the Apalachicola National Forest Red-bellied Woodpecker population. Several studies in wild populations have demonstrated temporal differences in the prevalence of haematozoan infections (Janovy 1966; Weatherhead and Bennett 1991, 1992; Hatchwell et al. 2000; Deviche et al. 2001), but most have been conducted in migratory species on their breeding grounds, so their duration is usually limited to a few consecutive months. The study reported here revealed strong seasonal variation in parasite prevalence over a period of 15 consecutive months (Fig. 1). Parasite prevalence in that population ranged from a high of ~80% in July 2000 to a low of 0% in January and February 2001. The peak in parasite prevalence in our population occurred during the middle of the

Red-bellied Woodpecker breeding season and was consistent with spring relapse. The presence of a spring relapse has raised questions regarding some of the practical aspects of testing Hamilton and Zuk's (1982) hypothesis. For example, Weatherhead and Bennett (1991, 1992) have suggested that seasonal variation in parasite prevalence hinders the ability of researchers to detect infections from blood smears, because whether an individual tests positive may depend on when that individual was sampled. For example, individuals with chronic infections may not have circulating gametocytes, in which case they would be considered uninfected on the basis of the analysis of a blood smear. Our study further emphasizes the importance of considering seasonal variation in parasite prevalence in the attempt to determine whether an individual is infected with haematozoa. The absence of any infected birds in our January and February 2001 samples suggests that individuals had fully recovered from prior infections, that chronic infections had become undetectable in peripheral blood by that time, or that infected individuals had died. Persistent stages of malarial parasites may reside in the liver of an infected host for the duration of the host's life and may serve as a source of parasites for relapsing chronic infections (Jarvi et al. 2001). It is therefore possible that the decrease in parasite prevalence observed in winter is the result of chronic infections, becoming latent, not complete recovery from summer infections. Current efforts to develop PCR-based methods for diagnosing haematozoan infections (Jarvi et al. 2001) may increase the ability of researchers to detect haematozoa despite seasonal changes in prevalence.

An increase in the prevalence of haematozoa during the host's breeding season is often attributed to a relapse of chronic infections due to the physiological stress associated with breeding (Atkinson and van Riper 1991). That view is supported by experimental evidence that corticosterone triggers relapses of chronic *Plasmodium* infections (Applegate and Beaudoin 1970). In addition, many recent observational and experimental studies that have demonstrated a positive relationship between reproductive effort and the prevalence or intensity of haematozoa (Oppliger et al. 1997, Nordling et al. 1998). Red-bellied Woodpeckers in the Apalachicola National Forest usually attempt to raise two to three broods during each breeding season. First clutches are laid in mid- to late April and second clutches in early July. The peak in *H. velans* prevalence we observed corresponds with the approximate fledge date of most second-brood chicks (late July and early August). A trade-off may therefore occur between raising a second brood and resistance to *H. velans*. We are currently examining how manipulating reproductive effort early in the breeding season (first clutches) affects parasite resistance and reproductive effort later in the breeding season (second and third clutches). That work may increase our understanding of the relationship between reproductive effort and infection with *H. velans* in the Red-bellied Woodpecker.

Although good evidence suggests that infection with haematozoa is associated with physiological stress and reproductive effort, a number of additional factors may cause parasite prevalence to vary in the manner we observed. For example, infected birds may suffer higher overwinter mortality than uninfected birds. In our study, infection with *H. velans* did not affect overwinter survival of males or females, but uninfected males showed a nonsignificant tendency toward higher overwinter survival than infected males. Our results are similar to those of Davidar and Morton (1993) and Dawson and Bortolotti (2000), who found no association between infection with *Haemoproteus* and return rates in Purple Martins (*Progne subis*) and American Kestrels (*Falco sparverius*), respectively. Although they found no association between infection and return rates, Dawson and Bortolotti (2000) found a significant negative relationship between the intensity of *Haemoproteus* infections and the probability that female American Kestrels would return to the study area. They found a similar relationship between return rates and infection intensity in males, but that relationship was not statistically significant. A similar relationship between infection intensity and overwinter survival may exist in our population, but we did not have intensity data on enough individuals to test that possibility.

In addition, the seasonal trend in parasite prevalence that we observed may be the result of new infections associated with the emergence of insect vectors. The vector for *H. velans* in our study area is probably a biting midge (genus *Culicoides*). Our area harbors a number of ornithophilic midges that may be potential vectors. The seasonal abundance of potential vectors in our study area varies by species, but a number of them are most abundant from April to October (Blanton and Wirth 1979). If the abundance of insect vectors increases over the same period as the increase in parasite prevalence, the increase is probably due in part to new infections. Relapses coincident with the emergence of insect vectors may also reflect a life-history strategy of the parasite (Kirkpatrick and Suthers 1988). For example, a correlation between vector abundance and some physiological change in the host could be exploited by the parasite to increase its chance of transmission to a new host. Those possibilities are supported by Merila et al. (1995), who found that the prevalence of blood parasites in geographically separated Greenfinch (*Carduelis chloris*) populations was correlated with local vector abundance. A complete understanding of the association between vector abundance and prevalence of *H. velans* requires knowing the insect vector's identity, its seasonal abundance, and the prevalence of *H. velans* in the vector. We do not currently have those data.

A negative association between condition and infection with haematozoa is a necessary condition for validity of both Hamilton and Zuk's hypothesis and the suggestion of a trade-off between reproductive effort and resistance to haematozoa (Hamilton and Zuk 1982, Ots and Horak 1998), but many studies have failed to demonstrate such an association (Smith and Cox 1972, Bennett et al. 1988, Wiehn et al. 1997). In the absence of such an association, haematozoa might be a relatively unimportant force in sexual selection and life-history evolution. Our study revealed negative associations between infection with *H. velans* and male mass and condition. That result suggests that infection with *H. velans* is an important force in the evolutionary ecology of the Red-bellied Woodpecker. Current efforts to understand the relationship among reproductive effort, parasitism, and host condition may help to determine its importance. In future studies, mass alone may be a sufficient indicator of condition, because tarsus length explained little of the variation in male mass in our study.

Interestingly, mass of females was not associated with infection with *H. velans*. Dawson and Bortolotti (2000) found that the association between infection and condition in female American Kestrels was sensitive to the stage of the birds' nesting cycle. In their study, infection with *Haemoproteus* was not associated with condition in prelaying females, but was negatively associated with condition in incubating females. A similar situation may exist in Red-bellied Woodpeckers, but because we did not know the nesting-cycle stage of most females we captured, we were unable to control for its potential effect. Another possibility is that the physiological demands of reproduction have a larger effect on female condition than does infection with *H. velans*. Female Red-bellied Woodpeckers in the Apalachicola National Forest often lay three clutches during a single breeding season, and they are responsible for most of the nestling provisioning (M. S. Schrader unpublished data). It may therefore be important to control for several measures of female reproductive effort (i.e. number of eggs laid, number of clutches laid, number of nestlings, and nestling age) during assessment of the effect of infection on female condition.

In conclusion, our data reemphasize the need to consider seasonal variation in parasite prevalence when screening individuals for haematozoa. In addition, our data suggest that although infection with *H. velans* may not have a direct effect on host survival, it can have a negative effect on host condition.

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LITERATURE CITED

APPLEGATE, J. E., AND R. L. BEAUDOIN. 1970. Mechanism of spring relapse in avian malaria: Effect of gonadotropin and corticosterone. Journal of Wildlife Diseases. 6:443–447.

ATKINSON, C. T., AND C. VAN RIPER III. 1991. Pathogenicity and epizootiology of avian haematozoa: *Plasmodium*, *Leucytozoon*, and *Haemoproteus*. Pages 19–47 *in* Bird-Parasite Interactions:

Ecology, Evolution, and Behaviour (J. E. Loye and M. Zuk, Eds.). Oxford University Press, Oxford.

Bennett, G. F. 1970. Simple techniques for making avian blood smears. Canadian Journal of Zoology 48:585–586.

Bennett, G. F., J. R. Caines, and M. A. Bishop. 1988. Influence of blood parasites on the body mass of passeriform birds. Journal of Wildlife Diseases 24:339–343.

Bennett, G. F., M. A. Peirce, and R. W. Ashford. 1993. Avian haematozoa: Mortality and pathogenicity. Journal of Natural History 27: 993–1001.

Blanton, F. S., and W. W. Wirth. 1979. The Sandflies (*Culicoides*) of Florida (Diptera: Ceratopogonidae). Florida Department of Agriculture and Consumer Services, Division of Plant Industry, Gainesville.

Cox, F. E. G. 1989. Parasites and sexual selection. Nature 341:289.

Davidar, P., and E. S. Morton. 1993. Living with parasites: Prevalence of a blood parasite and its effect on survivorship in the Purple Martin. Auk 110:109–116.

Dawson, R. D., and G. R. Bortolotti. 2000. Effects of haematozoan parasites on condition and return rates of American Kestrels. Auk 117: 373–380.

Deviche, P., E. C. Greiner, and X. Manteca. 2001. Seasonal and age-related changes in blood parasite prevalence in Dark-eyed Juncos (*Junco hyemalis*, Aves, Passeriformes). Journal of Experimental Biology 289:456–466.

Greiner, E. C., A. K. Mandal, and N. C. Nandi. 1977. *Haemoproteus benneti* sp. nov. and a review of the haemoproteids from Picidae (woodpeckers). Journal of Parasitology 63:651–656.

Hamilton, W. D., and M. Zuk. 1982. Heritable true fitness and bright birds: A role for parasites. Science 218:384–387.

Hamilton, W. D., and M. Zuk. 1989. Parasites and sexual selection. Nature 341:289–290.

Hatchwell, B. J., M. J. Wood, M. Anwar, and C. M. Perrins. 2000. The prevalence and ecology of the haematozoan parasites of European Blackbirds, *Turdus merula*. Canadian Journal of Zoology 78:684–687.

Jakob, E. M., S. D. Marshall, and G. W. Uetz. 1996. Estimating fitness: A comparison of body condition indices. Oikos 77:61–67.

Janovy, J., Jr. 1966. Epidemiology of *Plasmodium hexamerium* Huff 1935 in meadowlarks and starlings of the Cheyenne Bottoms, Barton County, Kansas. Journal of Parasitology 52:573–578.

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 22:254–263.

Kirkpatrick, C. E., and H. B. Suthers. 1988. Epizootiology of blood parasite infections in passerine birds from central New Jersey. Canadian Journal of Zoology 66:2374–2382.

Merila, J., M. Bjorklund, and G. F. Bennett. 1995. Geographic and individual variation in haematozoan infections in the Greenfinch, *Carduelis chloris*. Canadian Journal of Zoology 73: 1798–1804.

Merino, S., J. Moreno, J. J. Sanz, and E. Arriero. 2000. Are avian blood parasites pathogenic in the wild? A medication experiment in Blue Tits (*Parus caeruleus*). Proceedings of the Royal Society of London, Series B 267:2507–2510.

Nordling, D., M. Andersson, S. Zohari, and L. Gustafsson. 1998. Reproductive effort reduces specific immune response and parasite resistance. Proceedings of the Royal Society of London, Series B 265:1291–1298.

Norris, K., M. Anwar, and A. F. Read. 1994. Reproductive effort influences the prevalence of haematozoan parasites in Great Tits. Journal of Animal Ecology 63:601–610.

Oppliger, A., P. Christe, and H. Richner. 1997. Clutch size and malarial parasites in female Great Tits. Behavioral Ecology 8:148–152.

Ots, I., and P. Horak. 1998. Health impact of blood parasites in breeding Great Tits. Oecologia 116: 441–448.

Price, P. W. 1991. Foreword. Pages v–vii *in* Bird-Parasite Interactions: Ecology Evolution, and Behaviour (J. E. Loye and M. Zuk, Eds.). Oxford University Press, Oxford.

Read, A. F., and P. H. Harvey. 1989. Reassessment of comparative evidence for Hamilton and Zuk theory on the evolution of secondary sexual characters. Nature 339:618–620.

Richner, H., P. Christe, and A. Oppliger. 1995. Parental investment affects prevalence of malaria. Proceedings of the National Academy of Sciences USA 92:1192–1194.

Shackelford, C. E., R. E. Brown, and R. N. Conner. 2000. Red-bellied Woodpecker (*Melanerpes carolinus*). *In* Birds of North America, no. 500 (A. Poole and F. Gill, eds.). Birds of North America, Inc., Philadelphia.

Sheldon, B. C., and S. Verhulst. 1996. Ecological immunology: Costly parasite defenses and trade-offs in evolutionary ecology. Trends in Ecology and Evolution 11: 317–321.

Smith, V. W., and F. E. G. Cox. 1972. Blood parasites and weights of Palaearctic migrants in central Nigeria. Ibis 114:105–106.

Sokal, R. R., and F. J. Rohlf. 1995. Biometry, 3rd ed. W. H. Freeman, New York.

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.

Weatherhead, P. J., and G. F. Bennett. 1991. Ecology of Red-winged Blackbird parasitism by haematozoa. Canadian Journal of Zoology 69: 2352–2359.

Weatherhead, P. J., and G. F. Bennett. 1992. Ecology of parasitism of Brown-headed Cowbirds by hematozoa. Canadian Journal of Zoology 70:1–7.

Wiehn, J., E. Korpimäki, K. L. Bildstein, and J. Sorjonen. 1997. Mate choice and reproductive success in the American Kestrel: A role for blood parasites? Ethology 103:304–317.

Zuk, M. 1989. Validity of sexual selection in birds. Nature 340:104–105.

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