

EFFECTS OF HEMATOZOAN PARASITES ON CONDITION AND RETURN RATES OF AMERICAN KESTRELS

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ABSTRACT.—We evaluated the relationship between blood parasites and body condition of American Kestrels (*Falco sparverius*) during the breeding season. Females that were infected with at least one species of parasite were in poorer condition than those without parasites during incubation but not prior to egg laying. We suggest that the relationship between parasitism and condition was masked before laying because of large increases in body mass of females during egg formation. Reduced condition of males during incubation also was associated with higher intensity of infections by *Haemoproteus* in one of two years. The negative association between condition and intensity of infection suggests that blood parasites impose costs on kestrels owing to competition for nutrients or allocation of energy by hosts to immune function or tissue repair. Alternatively, kestrels in poor condition may be more likely to have relapses of chronic infections, or they may be less able to control new infections because of resource limitations. In contrast to results during incubation, during the prelaying period the prevalence of parasites tended to be higher, and in one year infections were more intense, among males in good condition. One possible explanation for these results is that body condition of males during courtship is an important determinant of the quality of mate they are able to obtain, and males may be accumulating body reserves at the expense of decreased immune function. Return rates of female kestrels to the study area declined as the intensity of their *Haemoproteus* infections increased, suggesting that blood parasitism is associated with reduced survival or increased dispersal probability. Received 30 September 1998, accepted 1 October 1999.

THEORY SUGGESTS that host-parasite interactions are responsible for substantial genetic variation within host populations (Anderson and May 1982), the evolution and maintenance of sexual reproduction and certain sexually selected traits (Hamilton 1980, Hamilton and Zuk 1982), and the complexity of parasite-resistance mechanisms (Anderson and May 1982, Behnke et al. 1992). Although such arguments are based on the premise that parasites reduce the fitness of their hosts (Combes 1997, Goater and Holmes 1997), the effects of parasites on hosts remain controversial. Diseased or moribund animals rarely are observed in nature, and finding parasites with commensal or amensal associations with hosts also is rare (Price 1980, Anderson and May 1982). Without knowledge of the effects of a specific parasite on a host species, it is impossible to evaluate

the importance of the parasite as an evolutionary force (Price 1980).

Hematozoa are protozoan blood parasites whose life cycles have both sexual and asexual stages in an arthropod vector, and asexual stages in a vertebrate host (Desser and Bennett 1993). Detrimental effects on hosts can occur at several stages of the parasite's life cycle. Tissue damage to the host's liver, kidney, spleen, lungs, or other organs can occur when the vector injects sporozoites that enter host cells to develop into meronts (Desser and Bennett 1993, Peirce et al. 1997). The reinvasion of host cells by asexually produced merozoites also can cause damage, as can meronts that produce gametocytes. Gametocytes entering circulating erythrocytes may impair oxygen-carrying capacity or cause cells to rupture, resulting in host anemia (Kocan and Clark 1966, Maley and Desser 1977). We tested whether the presence (prevalence) and number (intensity) of circulating gametocytes were associated with body condition of breeding American Kestrels (*Falco sparverius*). We also tested whether prevalence and intensity of hematozoan infections were associated with return rates of kestrels to the study area from one year to the next.

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STUDY AREA AND METHODS

We quantified hematozoa in a wild population of American Kestrels that bred in nest boxes near Bernard Lake (55°N, 106°W) in north-central Saskatchewan, Canada, during 1994 and 1995. Kestrels arrived on our study area in mid- to late April and began laying eggs in mid-May (Bortolotti 1994). Adult kestrels were first captured during the prelaying period using bal-chatri traps or nest-box traps. We captured adults again during incubation in nest boxes.

Blood collected from the brachial vein or the jugular vein was used to make blood smears (Bennett 1970). We air-dried and fixed smears in 100% ethanol immediately after obtaining each sample. Smears were stained with Giemsa and sent to the International Reference Centre for Avian Haematozoa at Memorial University, St. John's, Newfoundland, where G. F. Bennett quantified prevalence and intensity of hematozoa by counting the number of parasites in 100 microscope fields under oil, using a 100× objective for *Haemoproteus* and *Plasmodium* and a 40× objective for *Leucocytozoon* and *Hepatozoon*.

We banded each bird, weighed it to the nearest gram, and measured length of the unflattened wing chord, 10th primary, outer rectrix, central rectrix, and culmen, and width of the tarsus. We obtained an index of body size using the first component of a principal components analysis (PCA). We used the six linear measurements for the PCA, performing separate analyses for females ($n = 454$) and males ($n = 336$; see Bortolotti and Iko 1992). Body mass is partly a function of an animal's structural size, so to obtain an index of condition we removed the effect of body size by regressing mass against PC1 and used the residuals as our measure of condition.

Body condition of kestrels varies throughout the breeding season (Dawson and Bortolotti 1997a). The condition of females in our study rapidly increased as egg laying approached (cubic regression of condition on number of days before egg laying [hereafter "breeding chronology"], $R^2 = 0.72$, $n = 69$, $P < 0.0001$). Condition also declined with sampling date among incubating females ($r = -0.18$, $n = 181$, $P < 0.02$) and prelaying males ($r = -0.20$, $n = 102$, $P = 0.04$; see also Rehder et al. 1988, Dawson and Bortolotti 1997a). Variation in condition with breeding chronology or sampling date probably represented normal seasonal patterns, as opposed to being the result of differential effects of parasitism at different stages of the breeding season, because captive kestrels that were free of blood parasites (Bortolotti et al. 1996) also showed similar patterns of variation in condition (Rehder et al. 1986, 1988). We controlled for variation in condition by using residuals from linear regressions between sampling date and condition for prelaying males and incubating females (separate analyses for each sex), and by using a cubic regression between breeding chronology and con-

dition for prelaying females. Below, the term "condition" refers to these residuals from regressions between condition and breeding chronology or sampling date. In addition, we analyzed data from each sex and each stage of the breeding season (prelaying and incubation) separately because the effects of breeding chronology or sampling date on condition were sex- and season-specific.

We examined the relationship between condition and blood parasite loads in two ways. First, we examined all parasite species together and compared infected and uninfected birds. Second, 85% of the kestrels were infected with *Haemoproteus* spp., so we also tested whether the intensity of *Haemoproteus* infection alone was related to condition. Birds that exhibit no infection may be immune to parasites or susceptible but not yet exposed. Negative smears of immune birds are biologically meaningful, but if birds that test negative have not been exposed to parasites, the added variation that results from including them in analyses is biologically meaningless (Shutler et al. 1996). Because we cannot be certain of the underlying cause of negative smears (Shutler et al. 1996), we analyzed intensity data both including and excluding *Haemoproteus*-infected kestrels.

To examine the relationship between parasite prevalence and adult condition, we entered infection status (infected vs. uninfected) as the explanatory class variable and year as a class variable into analysis of variance. To test for effects of parasite intensity on condition, we used analysis of covariance (ANCOVA) with condition as the response variable, year as a class variable, and *Haemoproteus* infection intensity as the explanatory variable. To conform to assumptions of normality, intensity data were log transformed before analyses. For all analyses, interactions and year effects were removed if they were not significant and analyses were repeated. For ANCOVA, if neither year nor the interaction were significant, we used correlation analysis to test for associations between infection intensity and body condition of kestrels.

We estimated between-year return rates by recapturing banded adults from 1995 to 1997. Return rates are a function of survival, dispersal, and recapture probability, but here we assumed that birds that failed to return were dead. We compared return rates between parasitized and unparasitized birds using Fisher's exact tests. We tested for effects of *Haemoproteus* infection intensity on return rates using logistic regression, with return (yes or no) as the binary dependent variable and infection intensity as the explanatory variable. Birds that were captured twice during the breeding season (i.e. prelaying and incubation) were considered to have been parasitized for analyses of return rates if hematozoa were detected in either sample. Similarly, we used average intensity of *Haemoproteus* infection from both samples to test for effects of parasite intensity on return

TABLE 1. Condition indices ($\bar{x} \pm \text{SE}$, with n in parentheses) of American Kestrels according to infection by blood parasites. Condition was calculated using residuals of the regression between body mass and size, where size was measured using the first component of a principal components analysis. Condition indices were corrected for variation attributable to breeding chronology (prelaying females) and sampling date (prelaying males and incubating females). Data for males during incubation are presented separately for 1994 and 1995 owing to a significant year effect.

Sex	Unparasitized	Parasitized	<i>F</i>	df	<i>P</i>
Prelaying females	-0.26 ± 0.24 (14)	0.07 ± 0.14 (55)	1.16	1, 67	0.14
Incubating females	0.41 ± 0.34 (18)	-0.06 ± 0.10 (163)	3.41	1, 179	0.03
Prelaying males	-0.26 ± 0.17 (24)	0.08 ± 0.12 (78)	2.24	1, 100	0.07
Incubating males 1994	0.17 ± 0.40 (4)	0.54 ± 0.11 (63)	0.85	1, 134	0.18
Incubating males 1995	0.01 ± 0.29 (11)	0.17 ± 0.12 (59)			

rates. Excluding birds with two samples from a single breeding season did not change the results appreciably.

We performed statistical tests using SPSS and SAS software. Because we predicted *a priori* that parasites should be associated with reduced condition or return rates, we used one-tailed tests and considered results significant when $P < 0.05$.

RESULTS

We sampled 442 individual kestrels for parasites a total of 561 times in 1994 and 1995. Ten species of hematozoa from five genera were documented. The most commonly occurring species were *Haemoproteus tinnunculi* and *H. brachiatus* (overall prevalence = 84.3%; mean intensity among infected birds = $93.9 \pm \text{SE}$ of 8.9 parasites per 100 fields, range 1 to 2,000). *Plasmodium* and *Trypanosoma* occurred at low frequencies, and *Leucocytozoon toddi* and *Hepatozoon* spp. were detected in one male and two female kestrels, respectively. For additional details on patterns of prevalence and intensity, see Dawson and Bortolotti (1999).

During the prelaying period, the condition of female kestrels with at least one species of hematozoan was similar to that of parasite-free

females (Table 1). During incubation, however, parasitized females were in poorer condition than unparasitized females (Table 1). In contrast, prelaying males harboring at least one species of hematozoan tended to be in better condition than parasite-free males (Table 1). The condition of incubating males was significantly better in 1994 than in 1995 ($F = 5.29$, $df = 1$ and 134, $P = 0.02$), but condition did not vary with parasite status (Table 1).

We did not detect any significant relationships between the intensity of *Haemoproteus* infection and female condition during the prelaying or incubation periods (Table 2). Among prelaying males, condition increased with the intensity of *Haemoproteus* infection when negative smears were included in the analysis (Table 2). Because male condition during incubation was better in 1994 than 1995, we analyzed each year separately. In 1994, there was no relationship between male condition and *Haemoproteus* intensity (Table 2). In contrast, male condition during incubation in 1995 declined as the intensity of *Haemoproteus* increased when negative smears were excluded from the analysis (Table 2).

We pooled data on return rates from both

TABLE 2. Relationship between intensity of infection by *Haemoproteus* and condition indices of American Kestrels. Data were analyzed both including and excluding smears where no parasites were detected (negatives). Data for males during incubation are presented separately for 1994 and 1995 owing to a significant year effect.

Sex	Including negatives			Excluding negatives		
	<i>r</i>	<i>n</i>	<i>P</i>	<i>r</i>	<i>n</i>	<i>P</i>
Prelaying females	0.14	66	0.13	0.01	49	0.49
Incubating females	-0.10	181	0.10	-0.01	162	0.43
Prelaying males	0.18	93	0.04	0.17	66	0.09
Incubating males 1994	-0.01	67	0.45	-0.10	63	0.23
Incubating males 1995	-0.02	70	0.41	-0.25	58	0.03

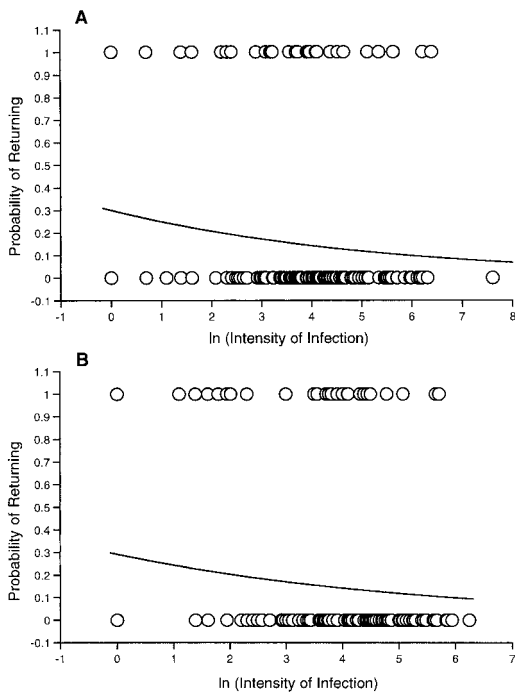


FIG. 1. Probability of (A) female, and (B) male American Kestrels returning to the study area according to intensity of infection (no. of parasites per 100 microscope fields) by *Haemoproteus*.

years because of small sample sizes. Infection status had no effect on the probability that females (3 of 31 [10.7%] uninfected vs. 36 of 234 [18.2%] infected; Fisher's exact test, $P = 0.30$) or males (4 of 26 [18.2%] uninfected vs. 27 of 173 [18.4%] infected; Fisher's exact test, $P = 0.62$) returned to the study area. When negative smears were included in the analyses, the intensity of *Haemoproteus* infection had no effect on return rates for females ($\chi^2 = 0.36$, $df = 1$, $P = 0.28$) or males ($\chi^2 = 0.98$, $df = 1$, $P = 0.16$). When negative smears were excluded from the analyses, females with lower-intensity infections returned at a higher rate than those with higher-intensity infections ($\chi^2 = 3.36$, $df = 1$, $P = 0.03$; Fig. 1A). A similar but nonsignificant trend was detected for males ($\chi^2 = 1.89$, $df = 1$, $P = 0.08$; Fig. 1B).

DISCUSSION

Detrimental effects of blood parasites occasionally have been documented in wild birds (Peirce et al. 1997) and are better known in do-

mestic birds (Atkinson et al. 1988, Bennett et al. 1993, Nakamura et al. 1997). Many studies have been unable to demonstrate that blood parasites affect the survivorship or condition of hosts (e.g. Smith and Cox 1972, Bennett et al. 1988, Weatherhead and Bennett 1992, Davidar and Morton 1993, Korpimäki et al. 1995, Shuttler et al. 1996). We found no effect of parasite status (infected vs. uninfected) or infection intensity on the condition of female kestrels prior to egg laying (Tables 1 and 2). Owing to growth of the oviduct and formation of the eggs, as well as accumulation of reserves for incubation, body mass of female kestrels increases dramatically as they approach egg laying (Newton 1979, Rehder et al. 1986, Dawson and Bortolotti 1997a). If parasites affected condition at this time, the effects may have been masked by variation in mass resulting from egg production. Therefore, it may not be surprising that we found no significant relationship between blood parasites and condition of female kestrels during the prelaying period.

During incubation, parasitized female kestrels were in poorer condition than unparasitized females (Table 1), and the condition of males in one of two years was negatively associated with the intensity of infection by *Haemoproteus* (Table 2). These results suggest that blood parasites exacted a cost on kestrels, presumably because they competed with hosts for nutrients, or forced hosts to allocate energy to immune function. Although a preliminary study by Apanius and Kirkpatrick (1988) found that body mass was negatively correlated with the intensity of *Haemoproteus* infection in female American Kestrels in Pennsylvania, subsequent analyses that controlled for stage of the nesting cycle failed to detect such an effect (Apanius 1993). Wiehn et al. (1997), working in the same study area as Apanius (1993), also did not detect a significant relationship between *Haemoproteus* infection and mass of American Kestrels during brood rearing. That the results of our study and those of Apanius (1993) and Wiehn et al. (1997) differ may be due to differences in both the prevalence and the intensity of infection between study areas. We found that 85% of the birds harbored hematozoa, and the intensity of infection averaged 93.9 parasites per 100 microscope fields (Dawson and Bortolotti 1999). Prevalence and intensity of Wiehn et al.'s (1997) birds were 61% and 28.5 parasites

per 100 microscope fields, respectively. In addition, negative effects of parasites are dependent on a wide variety of factors, including the ecological context in which parasitism occurs, the condition of hosts, and pathogenicity of the parasite in different geographic areas (see Goater and Holmes 1997). Furthermore, variation in these factors may explain why some of our results were not more general across the breeding season, or between years and the sexes. For example, condition of incubating males declined with *Haemoproteus* intensity only in 1995 (Table 2), a year when males were in relatively poor condition.

It is equally plausible that kestrels in relatively poor physical condition were more susceptible to parasites. Birds in poor condition may be resource limited and thus more likely to have relapses of chronic infections, or they may be less able to control newly acquired infections. Similarly, birds may be expending energy during reproduction at the expense of their body condition and immune system. Indeed, many recent studies have shown that parasite loads increase with the degree of effort expended in reproduction (e.g. Norris et al. 1994, Richner et al. 1995, Ots and Hörak 1996, Siikamäki et al. 1997, Wiehn and Korpimäki 1998).

Return rates of female kestrels were negatively associated with intensity of infection by *Haemoproteus* (Fig. 1A). The same pattern appeared in males, although the results were not significant (Fig. 1B). Estimates of return rates are a function of survival, dispersal, and recapture probability, and we are unable to ascertain which components were responsible for higher return rates of kestrels whose infections were less intense. Although hematozoan-induced mortality occurs occasionally in domestic birds, most studies of wild birds have failed to document such effects (e.g. Weatherhead and Bennett 1992, Bennett et al. 1993, Shutler et al. 1996; but see Herman et al. 1975, Richner et al. 1995, Sorci and Møller 1997). Given the lack of evidence for pathogenic effects of hematozoa, it seems unlikely that survival is affected by levels of infection per se. Instead, parasitism may be an indication of poor immune function or body condition, which in turn may lower survivorship from other causes. Regardless, even if our results are the product of differences in dispersal, dispersal may still represent a cost

because dispersing birds may have difficulty securing a breeding site and mate, or they may breed less successfully at a new site (Boulinier et al. 1997, Bensch et al. 1998). Similarly, birds with high intensities of parasites may have returned to the study area at rates similar to birds with low-level infections, but simply were not recaptured. Because we obtained a large proportion of our data on return rates by capturing kestrels during incubation, this might indicate that kestrels with low intensities of parasites were more likely to breed in subsequent years than were highly parasitized birds. Hence, the future fecundity of kestrels may be associated with elevated levels of blood parasites.

The condition of parasitized males during the prelaying period tended to be better than that of unparasitized males (Table 1). Similarly, male condition increased significantly as *Haemoproteus* infections became more intense during prelaying (Table 2). These results were surprising because they were in the opposite direction than we predicted. One possible explanation is that parasites may be able to better exploit hosts in good condition as opposed to those in poor condition (see Dawson and Borolotti 1997b, Møller 1997). However, this hypothesis cannot provide a general explanation for the relationship between condition and parasite load in kestrels because positive associations occurred only in males. Additionally, some negative relationships between parasites and condition were found during incubation in both sexes (Tables 1 and 2).

One plausible explanation for the positive association between parasitism and condition of prelaying males invokes the interaction of hormones and behavioral displays. Ros et al. (1997) found that nonbreeding Black-headed Gulls (*Larus ridibundus*) whose testosterone levels were experimentally increased within physiological limits increased their rates of behavioral displays. These displays apparently were costly, because rates of mass loss were highest among gulls with the highest display rates (Ros et al. 1997). Interestingly, gulls with the highest mass loss also were the most immunocompetent when challenged by injection with sheep red blood cells. These results suggest that individual quality of males is advertised by display rate, which in turn signals immunocompetence (Ros et al. 1997). A corollary is that the most immunocompetent birds in such a system

may also be the lightest in mass, owing to their high rates of display.

If the interpretations of Ros et al. (1997) are correct, they may explain the positive relationship between condition and parasite load of male kestrels that we observed. Testosterone levels of male kestrels are elevated only prior to egg laying (Rehder et al. 1988, G. R. Bortolotti unpubl. data). Male kestrels are behaviorally active during prelaying, not only provisioning their mates, but also establishing territories, attracting mates, and defending territories against intruding males (Balgooyen 1976, Bortolotti and Iko 1992). However, it seems unlikely that the highest-quality male kestrels are the lightest in mass. In our population, kestrels mate assortatively with respect to condition (Bortolotti and Iko 1992). Because a female's quality probably is dependent on her condition (Newton 1979, Village 1990), the highest-quality females would be paired with the poorest-quality males. Moreover, testosterone generally causes immunosuppression (Grossman 1985), and the results of Ros et al. (1997) therefore seem anomalous. Other studies that experimentally increased testosterone levels found increases in parasite loads (Saino et al. 1995, Salvador et al. 1996). A more plausible explanation may be that males seeking high-quality mates (i.e. in good condition) must themselves be in good physical condition (Bortolotti and Iko 1992). Males may accumulate body reserves at the expense of immune function. The value of body condition to males during the period of mate choice may be greater than the cost of allowing parasites to proliferate (Møller 1997).

In conclusion, our results suggest that blood parasites are associated with reduced condition and return rates of American Kestrels. We concur with Bennett et al. (1988), who found no effects of hematozoan parasitism on body mass of passerines and suggested that the influences of blood parasites on birds are subtle and difficult to detect. Because our study was observational, experimental manipulation of blood parasite loads is needed to unequivocally test for detrimental effects on hosts. The difficulty of experimentally transmitting most hematozoan parasites to hosts (Atkinson and van Riper 1991) represents a considerable challenge for future researchers.

ACKNOWLEDGMENTS

This paper would not have been possible without the assistance of the late Gordon F. Bennett, who graciously examined blood smears for hematozoa. We also thank the many people who helped us capture birds, especially J. Ball, C. Fehr, M. Hart, M. Miller, and S. Tomassi. Dave Shutler kindly provided unpublished manuscripts as well as many insightful comments on our work. We especially thank Victor Apanius and an anonymous referee for providing critical reviews that greatly improved a previous draft of the manuscript. Saskatchewan Environment and Resource Management provided permits. Funding for this project was provided by the Natural Sciences and Engineering Research Council through a research grant to GRB and a postgraduate scholarship to RDD. The Canadian Wildlife Federation, Northern Scientific Training Program, and the University of Saskatchewan provided additional funding to RDD.

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Associate Editor: K. L. Bildstein