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Sex-specific associations between reproductive output and hematozoan parasites of American kestrels

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Abstract Parasites have the potential to decrease reproductive output of hosts by competing for nutrients or forcing hosts to invest in immune function. Conversely, reproductive output may affect parasite loads if hosts allocate resources to reproduction such that allocation to immune function is compromised. Both hypotheses implicitly have a temporal component, so we sampled parasites both before and after egg laying to examine the relationship between reproductive output (indexed using a combined measure of clutch size, egg volume, and initiation date) and blood parasite loads of American kestrels (*Falco sparverius*). Parasite loads measured prior to egg laying had no adverse effects on subsequent reproductive output. Females that previously had large reproductive outputs subsequently had lower parasite intensities than those whose outputs were smaller, suggesting that females were capable of allocating energy to both forming clutches and reducing parasite loads. Because male kestrels provide most of their mate's energetic needs before, during, and after egg laying, mate choice by females may have consequences for their parasite loads. Females choosing high-quality mates may not only have increased reproductive output, but may also obtain sufficient resources from their mates to enable them to reduce their parasite burdens. Males whose mates had large reproductive outputs were more likely to subsequently be parasitized and have more intense infections. For individual males sampled both before and after egg laying, those whose mates had larger reproductive outputs were also more likely to become parasitized, or remain parasitized, between sampling periods. Increased parasite loads of males may be one mechanism by which the costs of reproduction are paid.

Keywords Egg laying · *Falco sparverius* · Hematozoa · *Haemoproteus* spp. · Reproductive output

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Introduction

Animals have limited resources and must prudently allocate them among life-history traits such as somatic growth, reproduction, and self maintenance (Williams 1966; Drent and Daan 1980). However, when two or more life-history traits compete for resources, a trade-off arises because individuals cannot allocate resources to one trait without a concomitant reduction in allocation to a competing trait (Stearns 1989). Allocation strategies have been the focus of life-history theory, and many factors can affect such decisions (Stearns 1992). Currently, parasites are being recognized as a potential factor influencing life-history evolution (Møller 1997). Nearly all organisms are potential parasite hosts (Minchella 1985), and parasites, by definition, are costly because they extract nutrients from hosts (Price 1980) and so have the ability to shape host evolution.

Parasite loads can correlate negatively, positively, or not at all, with reproductive output in birds (review in Møller 1997). Reductions in reproductive output of hosts can result through direct and indirect effects of parasites on hosts. Parasites may directly sequester host resources that normally would be invested in reproduction, or parasites may cause hosts to increase energy allocation to immune function or tissue repair (e.g., Møller et al. 1990). Indirect effects of parasites on reproduction can arise if parasites cause reductions in host body condition that in turn cause decreased reproductive output (Møller 1997). Alternatively, reductions in breeding effort by infected hosts may be a host strategy to reduce the detrimental effects of parasitism (Forbes 1993). Regardless of the mechanism, parasites are considered costly in terms of reproductive output, and a negative relationship between

parasite load and reproduction is expected (Møller 1997). Positive relationships between parasitism and reproductive output may arise if parasites influence a trade-off between reproduction and immunocompetence. If large investments in reproduction cause hosts to reduce energy allocation to immune function, reproduction may be costly for hosts because of the increased risk of parasitism (Ots and Hõrak 1996). Finally, parasite loads and reproductive output may not be correlated if the particular parasites are relatively harmless and have little effect on hosts (e.g., Weatherhead et al. 1993).

Many investigators examining relationships between parasitism and avian reproduction have sampled parasites during only a single time period of the birds' life cycle. However, a temporal component is implicit in both the parasite cost and reproductive cost hypotheses. For example, if parasites are costly, they will affect an individual's ability to reproduce in the future; parasite loads need to be measured prior to the onset of reproduction. Similarly, if reproduction is costly then parasite loads must be quantified at the conclusion of the reproductive event of interest.

In this paper, we test whether reproductive output, described as a combination of egg size, clutch size, and initiation date, is related to hematozoan parasite loads of American kestrels (*Falco sparverius*). We hypothesized that more energy would be invested in large clutches of large eggs (Wiebe and Bortolotti 1995) and birds breeding early in the season would have less available prey compared to birds breeding later in the breeding season (see Wiebe and Bortolotti 1992). We overcame the problem of a temporal component in hypotheses by sampling kestrels before and after egg laying, therefore providing a test of each hypothesis within the same host-parasite system. We tested the parasite cost hypothesis using parasite data collected prior to egg laying, and tested the reproductive cost hypothesis using parasite data collected at the conclusion of laying. In addition, we also tracked variation in parasite loads within individuals before and after egg laying. If parasite loads are a consequence of reproductive output, then a positive relationship between reproduction and change in parasite load is expected.

American kestrels, like most birds of prey, have distinct sex roles during reproduction. Males provide nearly all the food for females from several weeks prior to egg laying until the young are about 10 days old, during which time females remain largely sedentary (Balgooyen 1976; Newton 1979). Because of these differences in behavior during reproduction, we tested for relationships between parasitism and reproductive output for each sex. We predicted that because reproductive output of females is largely dependent on the quality of her mate (Wink et al. 1985), a female's reproductive output would be more affected by the parasite burden of her mate than her own. Similarly, reproductive output of female kestrels is predicted to have larger subsequent impacts on parasite load of her mate than it would on her own parasite load, given that it is the male that is providing most of the energy to form clutches.

Methods

We studied American kestrels breeding in nest boxes near Besnard Lake, Saskatchewan (55°N, 106°W) in 1994 and 1995. We captured kestrels during the pre-laying period using bal-chatri traps (Berger and Mueller 1959), or with nest-box traps while birds were inspecting boxes. Birds were again captured by hand in nest boxes after laying was completed.

We collected blood from either the brachial or jugular vein of each bird and made blood smears (Bennett 1970; further details in Dawson and Bortolotti 1997a, 1997b). Smears were air-dried and fixed immediately in 100% ethanol. G.F. Bennett, International Reference Centre for Avian Haematozoa, Memorial University, St. John's, Newfoundland, Canada, determined prevalence and intensity of hematozoa. Hematozoa were quantified by counting the number of parasites in 100 microscope fields under oil, using a 100× objective lens for *Haemoproteus* and *Plasmodium*, and 40× objective for *Leucocytozoon* and *Hepatozoon*. Because blood smears are inadequate to survey trypanosomes (Bennett 1962), the true prevalence of *Trypanosoma* in our population of kestrels remains unknown.

We visited each nest box every 3–5 days from mid-May to mid-June, or until egg laying had commenced. We returned when laying was complete to capture adults, and ascertain clutch size. Length and width of each egg was also measured with dial calipers (nearest 0.1 mm) and egg volume was calculated using the equation of Hoyt (1979) (volume=length×width^2×0.51). We measured reproductive output using clutch size, mean egg volume per clutch, and clutch initiation date. Because clutch size, egg size, and clutch initiation date are correlated in American kestrels (Wiebe and Bortolotti 1995), we produced a single variable (PC1) from principal components analysis that represented the relationship among these three variables (n=181 nests). Large PC1 values represent large clutch sizes (r=0.73, P<0.001) of large eggs (r=0.47, P<0.001) laid early in the season (r=–0.71, P<0.001).

We examined the relationships between reproductive output of kestrels and parasites in two ways. First, we examined all hematozoan species together and compared reproductive output between infected and uninfected birds (i.e., parasite status). Second, because the prevalence of *Haemoproteus* was high (84%, Dawson and Bortolotti 1999), we also tested whether intensity of infection by *Haemoproteus* alone was related to reproduction. Smears where no parasites are detected can arise because either the bird is immune to blood parasites, or it is susceptible but has yet to acquire parasites. Negative smears of immune birds are biologically meaningful, but if negative birds have not been exposed, the added variation due to including negative smears in analyses is biologically meaningless (Shutler et al. 1996). Because we can not be certain about the underlying cause of negative smears (Shutler et al. 1996), intensity data were analyzed both including and excluding samples where no *Haemoproteus* were detected.

To test whether parasite loads affected reproduction (parasite cost hypothesis), we used analysis of variance with reproductive output (i.e., PC1) as the response variable, parasite status (uninfected/infected) during pre-laying as the explanatory variable of interest, and year as a class variable. We used analysis of covariance (ANCOVA) to test for effects of parasite intensity on reproduction, with intensity as the explanatory variable and year as a class variable. To test whether reproductive output affected parasite loads (reproductive cost hypothesis), parasite status during incubation was used as the binary dependent variable in a logistic regression model with reproductive output as the explanatory variable and year as a class variable. We used ANCOVA to test for relationships between reproductive output and subsequent parasite intensity, with reproductive output as the explanatory variable and year as a class variable. For all analyses, we sequentially removed interactions, as well as the year effect, when not significant and analyses were then repeated. When year was not significant in ANCOVA, Pearson correlation analyses were used. To meet assumptions of normality, intensity data were ln-transformed before analyses.

By sampling birds during both pre-laying and incubation, we were able to track variation in parasite loads within individuals. Although hematozoan parasite loads of birds sampled twice within

the same year are generally similar, some birds acquire new infections, and others can lose their infections (e.g., Bennett and Bishop 1990; Weatherhead and Bennett 1991, 1992; Dawson and Bortolotti 1999). These data can be used to classify birds according to their ability to either reduce their infection levels, or remain parasite-free. If reproductive output affects parasite loads, these data provide a powerful means of detecting such effects.

Birds were classified into two groups according to whether and how parasite status changed between sampling periods: those that were either parasitized or unparasitized during pre-laying, and were unparasitized during incubation, were classified as "remaining/becoming uninfected", whereas birds that became infected or maintained their infection between pre-laying and incubation were classified as "maintaining/acquiring infections". We tested whether reproductive output was related to changes in parasite status between pre-laying and incubation using logistic regression with change in parasite status (remaining/becoming uninfected and maintaining/acquiring infections) as the binary dependent variable, and reproductive output and year as explanatory variables. We also calculated the difference in *Haemoproteus* intensity between pre-laying and incubation, and tested for associations between reproductive output and changes in intensity using ANCOVA. As with other analyses, interaction and year effects were removed from models when not significant.

Means are presented ±1 SE. Two-tailed statistical tests were performed using SPSS (Noruš 1993) and SAS (SAS Institute 1990). Results were considered significant at the 0.05 level.

Results

Parasites

We sampled 442 kestrels 561 times for parasites in 1994 and 1995. Details on patterns of prevalence and intensity are presented in Dawson and Bortolotti (1999). We documented ten species of hematozoa from five genera. *Haemoproteus tinnunculi* and *H. brachiatus* occurred most often (overall prevalence of both species combined=84.3%, intensity among infected birds=93.9±8.9 parasites per 100 fields, range 1–2000; Dawson and Bortolotti 1999). In addition, we detected three species each of *Plasmodium* and *Trypanosoma*. *Leucocytozoon toddi* and *Hepatozoon* spp. occurred in a single male and two females, respectively (Dawson and Bortolotti 1999). Our subsequent analyses use a subset of these birds for which reproductive data were available.

Parasitism and subsequent reproductive output

Parasite status, measured prior to egg laying, was not related to subsequent reproductive output of kestrels. Using PC1, we did not detect any difference in reproductive output between parasitized and unparasitized female or male kestrels (Table 1). Similarly, there was no relationship between *Haemoproteus* intensity and subsequent reproductive output of kestrels, either when negative smears were included or excluded from analyses (Table 2).

Reproductive output and subsequent parasitism

Reproductive output of female kestrels was not related to their probability of subsequently being parasitized (logistic regression, χ^2=2.52, *df*=1, *P*=0.11; Fig. 1a). However, reproductive output of females was negatively related to future *Haemoproteus* intensity both when negative smears were included (*r*=–0.23, *n*=135, *P*<0.01) and excluded in analyses (*r*=–0.20, *n*=124, *P*=0.03; Fig. 2a).

Among males, year and reproductive output significantly interacted in the logistic regression (χ^2=8.10, *df*=1, *P*=0.004), so we analyzed years separately. In 1994, reproductive output of a male's mate was not associated with the male's probability of subsequent parasitism (χ^2=2.07, *df*=1, *P*=0.15; Fig. 1b) whereas there was a positive association in 1995 (χ^2=8.29, *df*=1, *P*<0.01; Fig. 1c). Because *Haemoproteus* intensity data, including negative smears, interacted significantly with year ($F_{1,108}$=4.27, *P*=0.04), we analyzed these data by year. In 1994, no relationship was found between reproductive output of females and subsequent intensity of infection of their mates (*r*<0.01, *n*=48, *P*=0.98), but we detected a positive relationship in 1995 (*r*=0.35, *n*=64, *P*=0.005). When we excluded negative smears from the analysis, no significant year-by-intensity interaction or year effect was found, and parasite intensity of males was positively related to previous reproductive output of their mates (*r*=0.20, *n*=98, *P*=0.04; Fig. 2b).

Change in parasite loads and reproductive output

Reproductive output of females did not differ between birds maintaining or acquiring infections between pre-

Table 1 Mean (±SE) reproductive output of American kestrels that were either unparasitized or parasitized by hematozoa prior to egg laying; data pooled for 1994 and 1995. Reproductive output is the first component of a principal components analysis that used clutch size, egg volume, and clutch initiation date as input variables

Sex	Reproductive output		F	*df*	P
	Unparasitized	Parasitized			
Females	0.03 (0.22) *n*=14	–0.24 (0.20) *n*=45	0.49	1,57	0.49
Males	–0.13 (0.32) *n*=10	–0.03 (0.20) *n*=27	0.78	1,35	0.78

Table 2 Correlation between *Haemoproteus* spp. infection intensities of American kestrels measured prior to egg laying, and their subsequent reproductive output. Data were analyzed both including and excluding negative blood smears. Reproductive output is the first component of a principal components analysis that used clutch size, egg volume, and clutch initiation date as input variables

Sex	Including negatives			Excluding negatives		
	r	*n*	P	*r*	*n*	P
Females	–0.17	56	0.21	–0.22	40	0.16
Males	–0.11	34	0.55	–0.25	23	0.25

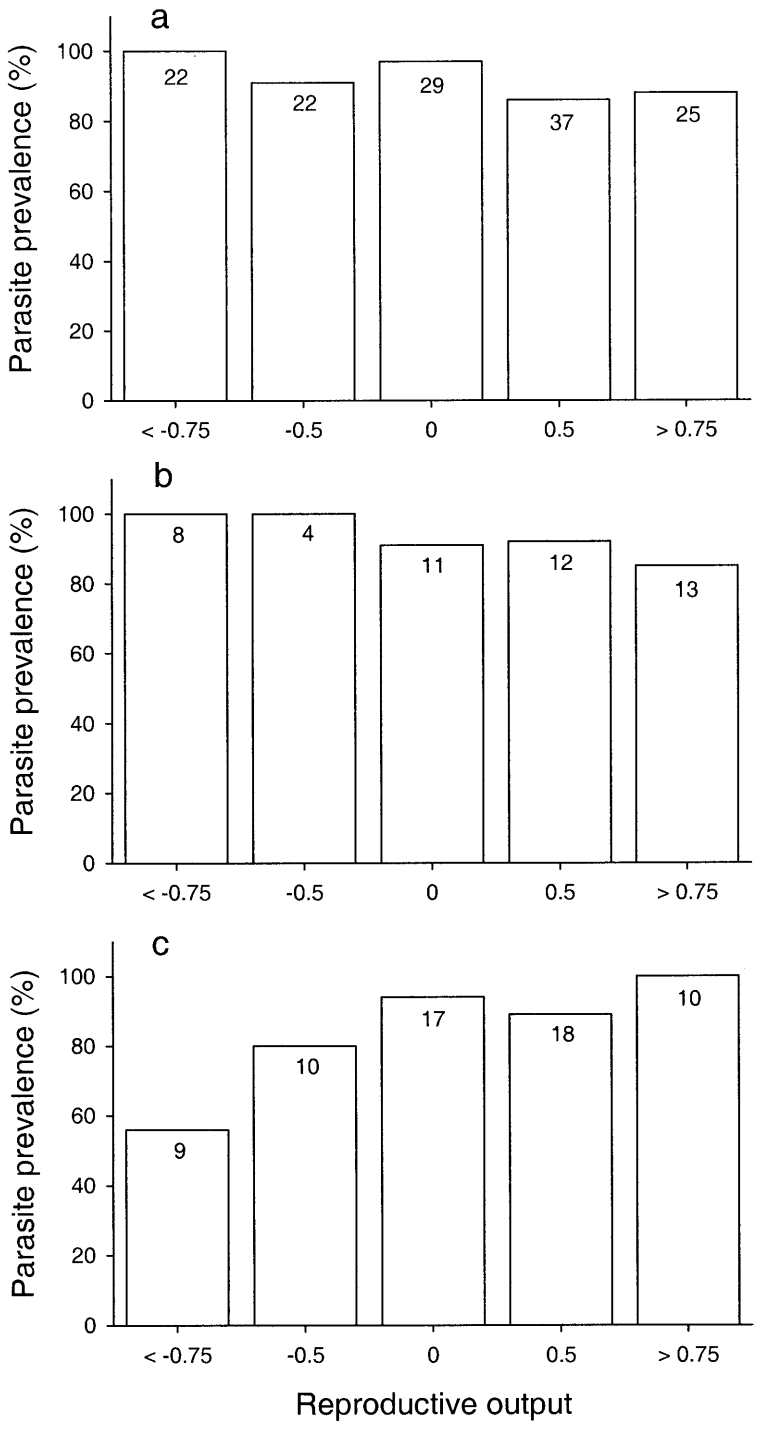

Fig. 1a–c Percentage of American kestrels infected with hematozoa during incubation according to their previous reproductive output. Reproductive output is the first component of a principal components analysis that used clutch size, egg volume, and clutch initiation date as input variables, and large values represent large clutches of large eggs, laid early in the breeding season. **a** Females, data from 1994 and 1995 pooled (logistic regression, P=0.11); **b** males, 1994 (P=0.15); **c** males, 1995 (P<0.01). *Sample sizes* are indicated within *bars*

laying and incubation and those that maintained their status as parasite free or became uninfected (χ^2=0.01, df=1, P=0.92). Similarly, reproductive output of females was not related to changes in *Haemoproteus* intensity between sampling periods (including negatives: r=–0.02, n=41, P=0.88; excluding negatives: r=0.05, n=27, P=0.79).

Males whose mates had large reproductive outputs were more likely to maintain or acquire infections between pre-laying and incubation, whereas males whose mates had smaller outputs were more likely to lose their infections or remain parasite-free (χ^2=3.89, df=1, P=0.04; Fig. 3). Overall change in *Haemoproteus* intensity of males did not significantly correlate with their mate's reproductive output when negative smears are included in analyses (r=0.30, n=28, P=0.12). When negatives were excluded, the relationship was stronger but not significant, perhaps due to small sample size (r=0.41, n=17, P=0.10).

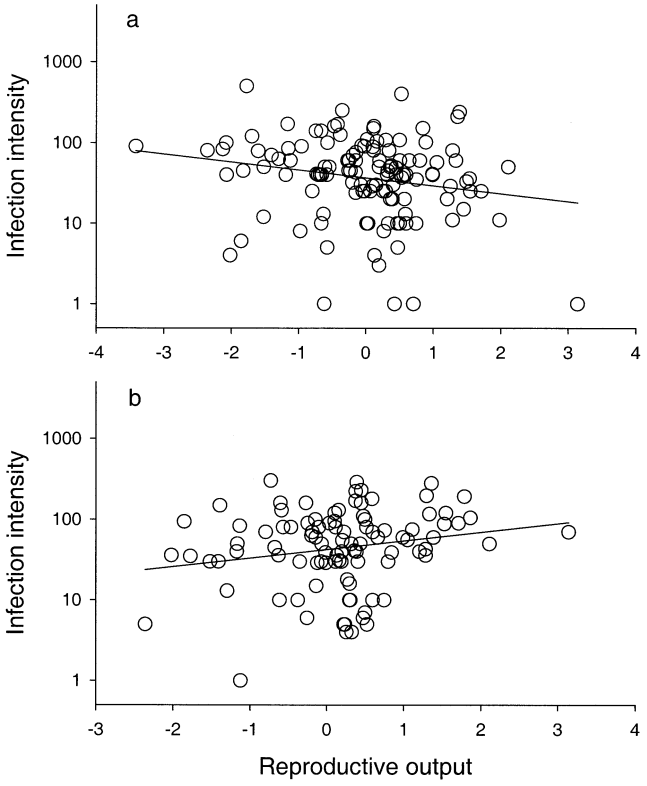

Fig. 2 Intensity of infection by *Haemoproteus* of **a** female, and **b** male American kestrels during incubation, 1994 and 1995, in relation to their previous reproductive output during egg laying (females: P=0.03, males: P=0.04). Reproductive output is the first component of a principal components analysis that used clutch size, egg volume, and clutch initiation date as input variables, and large values represent large outputs

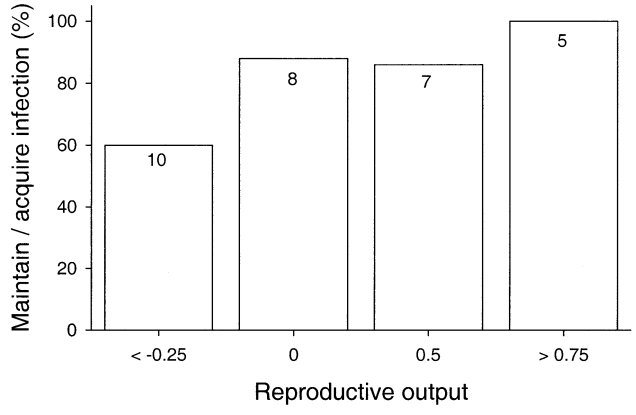

Fig. 3 Proportion of male American kestrels in 1994 and 1995 maintaining or acquiring hematozoa infections between pre-laying and incubation according to the reproductive output of their mates (logistic regression, P=0.04). Reproductive output is the first component of a principal components analysis that used clutch size, egg volume, and clutch initiation date as input variables, and large values represent large outputs. *Sample sizes* are indicated within *bars*

Discussion

Parasitism and subsequent reproductive output

Parasites, by definition, draw nutrients from their hosts (Price 1980). In addition, hosts may expend energy repairing damaged tissue or mounting an immune response to parasites (Keymer and Read 1991; Read and Skorping 1995; but see Ots and Hõrak 1996). Consequently, parasites have the potential to shift the trade-off in energy allocation away from host reproduction and towards self-maintenance. The result can be a negative correlation between parasite loads and reproductive performance. Indeed, hematozoan parasites of birds have been implicated previously, both directly and indirectly, as causing reduced reproductive output of birds (Korpimäki et al. 1993; Rätti et al. 1993; Allander and Bennett 1995; Dale et al. 1996; Dufva 1996). Our results for American kestrels suggest that blood parasites, measured just prior to egg laying, did not have any detectable relationships with attributes of the clutch (laying date, clutch and egg size; Tables 1, 2). Congruent with our results, several other studies have been unable to detect effects of blood parasites on reproduction of birds (e.g., Weatherhead 1990; Wagner et al. 1997; Shutler et al. 1999), and other studies yielding negative results may not have been published.

Our results, and those reported in several other studies (e.g., Wagner et al. 1997), suggest that hematozoa are relatively benign and have little effect on reproductive output. However, seemingly benign parasites may not always be harmless. Møller (1997) suggested that impacts of parasitism may be dependent on the context within which parasitism occurs; habitat, food supply and timing of exposure may all affect the degree to which hosts show negative effects of parasites. Although we detected no significant relationships between parasitism of individual kestrels and food abundance on their territories (Dawson and Bortolotti 1999), if food abundance is above some threshold level then parents may be able to acquire sufficient food to compensate for the removal of host resources by parasites (Møller 1997).

Reproductive output and subsequent parasitism

Parasite prevalence, measured after egg laying, was higher in one of two years among male kestrels whose mates had large reproductive outputs (Fig. 1c), and in both years *Haemoproteus* intensity of males measured after egg laying was positively related to reproductive output of their mates (Fig. 2b). Moreover, in both years males with mates having large reproductive outputs were also more likely to become or remain parasitized between pre-laying and incubation, as opposed to remaining parasite-free or losing their infection (Fig. 3). Changes in infection intensity among individual males between sampling periods were not significantly related to reproductive output of their mates, but the directions of the re-

lationships also suggest parasitism of males was associated with reproduction.

Large reproductive outputs associated with greater levels of parasitism in males could potentially be explained by age-related differences in parasitism and reproduction. In many species, older birds (or mates of older birds) tend to have larger clutches and initiate laying earlier than young birds (Martin 1995). At the same time, prevalence of blood parasites is thought to increase with age (e.g., Weatherhead and Bennett 1991; Davidar and Morton 1993; Norris et al. 1994). Older birds may therefore have larger reproductive output while maintaining higher parasite loads, but the relationship between reproduction and parasitism is not one of cause and effect. However, we have shown no age effects on either parasite prevalence or intensity during incubation among American kestrels that we were confidently able to age (Dawson and Bortolotti 1999). In fact, prevalence prior to egg laying was higher among yearling kestrels than after-second-year birds (Dawson and Bortolotti 1999). We therefore reject the idea that our results for male kestrels are simply the product of covariation of parasitism and reproduction with age.

Our results for males are similar to those of Wiehn et al. (1997), who found higher blood parasite prevalence among male American kestrels raising larger broods compared to those raising smaller broods. However, results of Wiehn et al. (1997) were specific to males they classified as yearlings. We did not control for age in our analyses because returns of banded known-age kestrels to our study area indicate that the aging criteria of Smallwood (1989), which were used by Wiehn et al. (1997), are unreliable (G.R. Bortolotti and R.D. Dawson, unpublished work). We generally acquire data on male age through recaptures of previously banded birds, and so our sample sizes are inadequate to include age as a factor in these analyses. Regardless, when we analyzed data excluding males that we were able to confidently classify as yearlings, results were the same as when the entire data set was used (results not shown). Our study suggests increased parasite loads as a consequence of large reproductive output is a general phenomenon among males, and not limited to certain age cohorts as Wiehn et al. (1997) suggested.

Increased parasite loads of male kestrels associated with large reproductive output of their mates may be the result of increased exposure to new infections. Males may spend more time in areas of high vector abundance, or may have less time for performing anti-vector behaviors (Norris et al. 1994). However, new infections may not adequately explain our results, or those from other studies where parasite loads have increased with increasing reproductive effort (Norris et al. 1994; Richner et al. 1995; Oppliger et al. 1996). The prepatent period of blood parasites ranges from approximately 10 to 18 days for *Plasmodium* and about 14 days for *Haemoproteus* (Garnham 1966), and insufficient time may have elapsed for new infections to become patent (Allander 1997). We sampled male kestrels in this study on average 10.8±0.5 days (n=155) after the completion of egg laying. For many birds, the interval between egg laying and parasite sampling may have been insufficient for new infections to become patent. Moreover, because vectors may not have become active until after kestrels completed egg laying (Bennett 1960; Bennett and Fallis 1960), we suggest that increased recrudescence of chronic infections among males whose mates had large reproductive outputs is more plausible than acquisition of new infections.

In contrast to males, reproductive output of females was not related to their subsequent parasite status. Most females captured during incubation were parasitized (Fig. 1a). Our prevalence data may have been inadequate to test the reproductive cost hypothesis because of lack variation in parasite status. However, the larger reproductive outputs associated with lower subsequent intensity of *Haemoproteus* infections among females (Fig. 2a) suggest that female kestrels that were capable of large reproductive outputs were also capable of mounting an immune response to control their levels of infection.

Sex-specific associations between reproduction and parasitism

The most studied trade-off in life-history theory is the cost of reproduction (Stearns 1992), where organisms must allocate resources prudently between current reproduction and future reproduction or survival (Williams 1966). Parasitism may be one way that costs of reproductive investment are paid. For example, blood parasite loads in great tits (*Parus major*; Norris et al. 1994; Richner et al. 1995; Oppliger et al. 1996, 1997) and Eurasian kestrels (*Falco tinnunculus*; Wiehn and Korpimäki 1998; Wiehn et al. 1999) were positively correlated with reproductive effort. Additionally, the effects of reproduction on parasitism in these studies were sex-specific and explained by the fact that higher levels of parasitism became apparent in the sex that was forced to work harder as a result of experimental manipulations.

Relationships between reproductive output and parasitism of American kestrels were also sex-specific and opposite in nature; males whose mate's had large reproductive outputs in terms of clutches had higher parasite loads, while females had lower parasite loads (Fig. 2). Female kestrels rely on food provided by their mates from about 2 weeks prior to egg laying up until nestlings are about 10 days of age (Balgooyen 1976). As a result, condition and reproductive output of females is largely dependent on the quality of their mates and the food their mates provide. Our measure of reproductive output is therefore a predominantly male-based measurement (see also Newton 1979; Wink et al. 1985). As such, we predicted a priori that elevated parasite loads associated with reproduction would be manifested in male as opposed to female kestrels. The results of our non-manipulative study are noteworthy because they demonstrate that parasites may be a potential cost of reproduction even at normal levels of reproductive effort.

Decreases in parasite loads among female American kestrels with large reproductive outputs may be related to the quality of her mate. The probability of a female Eurasian kestrel's being infected with blood parasites declined when her mate provided food at high rates during brood rearing (Wiehn and Korpimäki 1998). Such relationships might have consequences for mate choice because females choosing "good parents" as mates (e.g., Palokangas et al. 1994) may be accruing benefits not only through offspring quality, but also through enhanced well-being of the female because of reduced parasite loads (Wiehn and Korpimäki 1998). High-quality male American kestrels may also be capable of providing enough food so that their mates are able to have large reproductive outputs while reducing their parasite loads; sex-specific relationships between reproductive output and parasitism may be the rule rather than the exception in birds of prey such as kestrels. This hypothesis would predict that changes in parasite loads of males before and after clutch formation would be negatively correlated with changes in parasite loads of females. We currently do not have adequate data to rigorously test this prediction.

Conclusions

Our study found that parasite loads of male American kestrels were positively associated with reproductive output of their mates. Given that survival probabilities of kestrels in our population declines with increasing parasite intensities (Dawson and Bortolotti 2000a), infection by hematozoa may be a potential mechanism for paying the cost of reproduction. Relationships between parasite loads of males and reproductive output of their mates were more pronounced during 1995 than 1994, and it is possible that environmental factors may also have played a role in mediating the relationship between reproduction and parasitism, as suggested by Wiehn and Korpimäki (1998) and Wiehn et al. (1999). Voles, the main prey of kestrels on our study area, were more abundant in 1994 than in 1995 (Dawson and Bortolotti 2000b). Although parasite loads do not appear to be related to food abundance (Dawson and Bortolotti 1999), reproduction by kestrels is highly dependent on food supply (e.g., Bortolotti et al. 1991; Wiebe and Bortolotti 1992, 1994, 1995) and kestrels may have been under more stress during egg laying in 1995. Nonetheless, our results may be conservative because we tested for relationships between parasitism and only one component of reproduction, namely egg laying. Provisioning offspring is energetically demanding (Drent and Daan 1980), and because reproductive costs are expected to accumulate throughout the breeding season (Moreno 1993), the effects of reproduction on parasite loads that we detected may be underestimated. Regardless, the fitness payoff for males presumably exceeded the potential costs associated with elevated levels of blood parasitism.

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