

Prevalence and intensity of hematozoan infections in a population of American kestrels

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Abstract: Interest in hematozoan parasites has been considerable in recent years, mostly as a result of Hamilton and Zuk's idea that parasites influence the expression of sexually selected traits. However, little is known about the basic patterns of parasitism and the dynamics of host-parasite relationships. We describe the patterns of blood parasitism in American kestrels (*Falco sparverius*) sampled throughout the breeding seasons of 1994 and 1995, and investigate the influence of several variables on parasite load. Parasite prevalences in kestrels were high, ranging from 75 to 94% depending on the sex of the birds and stage of the breeding season. Prevalence increased with date, indicating either active parasite transmission or relapses of chronic infections. Parasite intensity also increased with date, but these effects were sex- and year-specific. We detected no sex differences in either prevalence or intensity, but prevalences were higher in young (second calendar year) birds than in older birds. Because this effect was evident early in the breeding season, we suggest that it was due to differential recrudescence of chronic infections between age-classes. Food supply had no effect on parasite load. Among birds that were sampled twice in the same year, parasite status remained relatively constant, although some birds acquired infections while others lost them. Change in intensity between samples was dependent on year; nonetheless, intensities remained relatively stable throughout the breeding season. Kestrels sampled in both years had consistent parasite loads. Parasites were detected in only 3% of kestrel nestlings.

Résumé : L'intérêt pour les hématozoaires parasites a été considérable au cours des dernières années, surtout à la suite de la publication de la théorie d'Hamilton et Zuk selon laquelle les parasites influencent l'expression de certaines caractéristiques reliées à la sélection sexuelle. Cependant, les patterns de base du parasitisme et la dynamique des relations hôte-parasite sont encore mal connus. On trouvera ici la description du parasitisme des hématozoaires chez des Crécerelles d'Amérique (*Falco sparverius*) échantillonnées durant toute la saison de reproduction en 1994 et en 1995 ainsi qu'un examen de l'influence de plusieurs variables sur le fardeau de parasites. La fréquence du parasitisme était élevée chez les crécerelles, atteignant 75 à 94% des oiseaux selon leur sexe et le stade de la reproduction qu'ils avaient atteint. La fréquence augmentait en fonction de la date, augmentation due à une transmission active des parasites ou à de nouvelles manifestations d'infections chroniques. La gravité des infections a également augmenté en fonction de la date, mais ces effets variaient selon le sexe des oiseaux ou selon l'année. Nous n'avons pas constaté de différences dans la fréquence ou dans la gravité des infections en fonction du sexe, mais la fréquence des infections était plus élevée chez les jeunes oiseaux (2^e année du calendrier) que chez les oiseaux plus âgés. Comme cet effet s'est manifesté tôt au cours de la saison de la reproduction, nous croyons qu'il a résulté d'une recrudescence des infections chroniques affectant différemment les diverses classes d'âge. La disponibilité de la nourriture n'avait pas d'effet sur le fardeau de parasites. Chez les oiseaux échantillonnés deux fois au cours de la même année, le parasitisme est resté relativement constant, mais certains oiseaux ont contracté des infections, alors que chez d'autres les infections sont disparues. Les fluctuations de la gravité des infections d'un échantillon à l'autre étaient fonction de l'année, mais la gravité était néanmoins relativement stable pendant toute la saison de reproduction. Les crécerelles échantillonnées les 2 années avaient également des fardeaux de parasites stables. Seulement 3% des crécerelles au nid étaient parasitées.

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Introduction

Interest in hematozoan parasites has increased dramatically in recent years, probably as a result of their possible relationship with host sexual selection. Hamilton and Zuk (1982) proposed that extravagant traits evolved as honest

signals of heritable parasite resistance (reviews in Read 1988, 1990; Møller 1990; Clayton 1991). Hamilton and Zuk argued that such traits were revealing handicaps (Zahavi 1975); when animals are under attack from parasites, only resistant individuals will be able to fully express extravagant traits. Females that choose highly ornamented mates will have offspring that are resistant to the parasites currently infecting the population.

While many studies have examined relationships between blood parasites and attributes of birds such as condition or sexually selected traits (references in Bennett et al. 1988; Hamilton and Poulin 1997), we are still largely ignorant of many aspects of the biology of hematozoans and their interactions with hosts (Desser and Bennett 1993). For example,

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many investigators sampled individual birds only once, and the sample of birds encompassed only a narrow portion of the birds' life cycle. Few investigators have examined patterns of parasitism throughout different stages of the breeding season within populations or individuals, and fewer still have sampled the same individual birds in different years. Notable exceptions include Bennett and Cameron (1974), Bennett and Bishop (1990), Weatherhead and Bennett (1991, 1992), Korpimäki et al. (1995), and Wiehn et al. (1999).

We sampled blood parasites of American kestrels (*Falco sparverius*) throughout the breeding season over 2 years. In addition, we sampled nestlings just prior to fledging. Our purpose was to describe the species composition, prevalence, and intensity of blood parasites between years, stages of the breeding season, and age-classes. We tested whether the food supply on kestrel territories affected parasite loads and we examined whether parasite loads changed within and between years within individual kestrels. We put our results into perspective by comparing them with those of several other parasitological investigations of American kestrels.

Materials and methods

We studied a wild population of American kestrels breeding in nest boxes near Besnard Lake in north-central Saskatchewan, Canada (55°N, 106°W), during 1994 and 1995. Kestrels arrived on our study area in mid to late April and egg laying commenced in mid-May (Bortolotti 1994). During the prelaying period we captured adult kestrels with bal-chatri traps (Berger and Mueller 1959), or with nest-box traps while birds were inspecting boxes. We captured adult birds again by hand in nest boxes during incubation.

Upon capturing birds, we attempted to identify unbanded kestrels as yearlings (second calendar year (SY)) by means of fault bars on feathers (Smallwood 1989); however, relatively few birds can be aged reliably in this manner. Age in years was known for some birds banded as nestlings that subsequently returned to the study area, and minimum age was known for some kestrels banded as breeding birds in previous years that returned to the study area (after second year (ASY)). We restricted our analyses of age effects to individuals classified reliably or known from banding to be SY or ASY.

We made smears (Bennett 1970) with blood collected from either the brachial or the jugular vein. Smears were air-dried and fixed immediately in 100% ethanol. Prevalence and intensity of hematozoa were determined at the International Reference Centre for Avian Haematology at Memorial University, St. John's, Newfoundland, by G.F. Bennett. Hematozoa were quantified by counting the parasites in 100 microscope fields under oil, using a 100× objective for *Haemoproteus* spp. and *Plasmodium* spp. and a 40× for *Leucocytozoon* spp. and *Hepatozoon* spp.

We examined patterns of blood parasitism in two ways. First, we examined all parasite species together and compared infected with uninfected birds (i.e., prevalence). Second, as the prevalence of *Haemoproteus* spp. was extremely high (see Results), we also investigated patterns of infection intensity by *Haemoproteus* spp. alone. In some smears no parasites are detected, either because the bird from which the smear was taken is immune to blood parasites or because it is susceptible but has yet to be exposed to parasites. As Shutler et al. (1996) pointed out, negative smears from immune birds are biologically meaningful, but if birds with negative smears have not been exposed, the added variation due to the inclusion of birds with negative smears in analyses is biologically meaningless. Because we cannot be certain about the underlying cause of negative smears (Shutler et al. 1996), we analyzed intensity data in two

ways: including and excluding samples in which no *Haemoproteus* spp. were detected (cf. Shutler et al. 1996).

We obtained blood samples from adult kestrels during all phases of the breeding season: prelaying, egg laying, incubation, soon after the eggs hatched, and just before the young left the nest. To reduce disturbance to birds during laying, we did not actively sample birds at this time; therefore most of our analyses were restricted to the prelaying and incubation periods, when our sample sizes were large. We treated each of these periods separately in many analyses because of marked differences in the behaviour and physiology of kestrels between the prelaying and incubation periods (Balgooyen 1976; Dawson and Bortolotti 1997a).

By sampling birds during both the prelaying and incubation periods, we were able to track variation in parasite load within individuals. In addition, a number of birds were sampled in successive years. Both data sets allow insight into the relapse phenomenon of chronic infections, transmission of parasites, and hosts' ability to eliminate parasites immunologically from their system.

We assessed the importance of prey abundance on host parasitism by censusing the main prey of kestrels, small mammals (Bortolotti et al. 1991; Iko 1991). During early July, trap lines consisting of 10 stations, spaced 30 m apart and situated parallel to and 10 m from a road, were set on some kestrel territories. At each station two snap traps were baited with peanut butter. Each line operated for 3 days and traps were checked each morning. We examined the effect of prey abundance on parasitism for incubating birds only. During the prelaying period, both sexes can be fairly transient and often switch between one or more territories before egg laying begins (Bortolotti and Iko 1992). By testing for prey-abundance effects only during incubation, we were more confident that birds had been present on territories for a sufficient length of time for potential food-mediated alterations of parasitism, if they existed, to become apparent. Although this index of prey abundance would seem to be a coarse measure of food availability, it is meaningful, as it correlates well with kestrel reproduction (e.g., Bortolotti et al. 1991; Wiebe and Bortolotti 1992, 1994, 1995). For our analyses, we considered only voles (mostly *Clethrionomys gapperi*, measured as the number of individuals/100 trap-nights), as they constitute the majority of the mammalian prey items in the diet of breeding kestrels (R.D. Dawson, unpublished data).

In addition to surveying adult kestrels for parasites, we took blood smears from nestling kestrels at 22–29 days of age (just prior to their leaving the nest) in 1994. Details on these methods are available elsewhere (Dawson and Bortolotti 1997b).

Intensity data were transformed ($\ln + 1$) before analysis. Unless stated otherwise, we used year as a factor in analyses; we followed Alisauskas and Ankney (1994) and if interactions were not significant, they were removed from the models and the analyses were rerun. We used logistic regressions to examine patterns of parasite prevalence attributable to sex of birds and stage of the breeding season, and *G* or Fisher's exact tests for age effects. Analysis of variance was used to examine effects of age, year, sex, and stage of the breeding season on intensity of *Haemoproteus* spp. infection. Logistic regressions were used to test for effects of prey abundance on prevalence, while correlation analyses were used for intensity data. Means are presented ± 1 SE. Two-tailed statistical tests were performed using SPSS (Norusis 1993), and we considered results significant at the 0.05 level.

Results

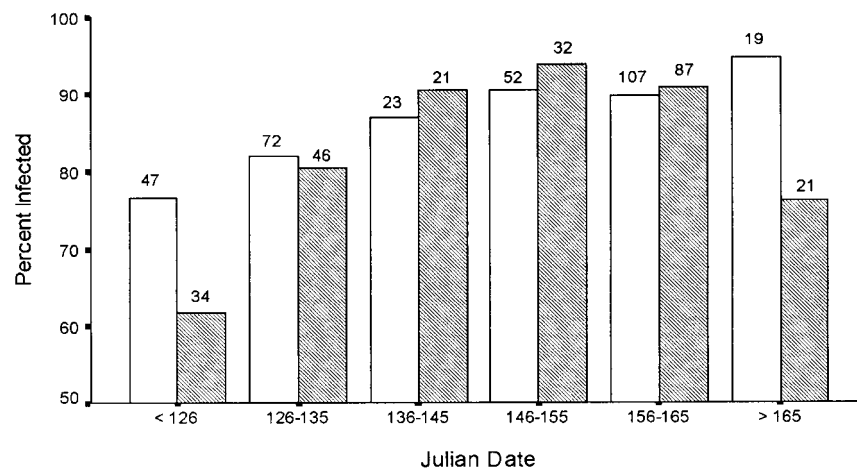
Patterns of parasitism

We sampled 442 individual kestrels for parasites a total of 561 times in 1994 and 1995. A single sample was obtained from 328 birds, two samples from 109 birds (84 within the same year, 25 in different years), and three samples from 5 birds (two samples in one year, a single sample in the other

Table 1. Prevalence of blood parasites in American kestrels sampled in north-central Saskatchewan during the prelaying and incubation periods in 1994 and 1995.

Parasite species	Females				Males			
	1994		1995		1994		1995	
	Prelaying (n = 52)	Incubation (n = 97)	Prelaying (n = 84)	Incubation (n = 87)	Prelaying (n = 27)	Incubation (n = 67)	Prelaying (n = 75)	Incubation (n = 72)
<i>Haemoproteus tinnunculi</i>	18 (34.6)	65 (67.0)	24 (28.6)	64 (73.6)	14 (51.9)	50 (74.6)	22 (29.3)	49 (68.1)
<i>H. brachiatus</i>	29 (55.8)	35 (36.1)	41 (48.8)	12 (13.8)	9 (33.3)	20 (29.9)	32 (42.7)	11 (15.3)
<i>Plasmodium circumflexum</i>	1 (1.9)	2 (2.1)	0 (0)	0 (0)	0 (0)	0 (0)	2 (2.7)	0 (0)
<i>P. vaughni</i>	2 (3.8)	1 (1.0)	5 (6.0)	0 (0)	0 (0)	0 (0)	2 (2.7)	0 (0)
<i>P. polare</i>	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	1 (1.3)	0 (0)
<i>Trypanosoma avium</i>	0 (0)	3 (3.1)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	1 (1.4)
<i>T. bennetti</i>	0 (0)	1 (1.0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
<i>T. ontarioensis</i>	3 (5.8)	0 (0)	1 (1.2)	0 (0)	0 (0)	1 (1.5)	1 (1.3)	1 (1.4)
<i>Leucocytozoon toddi</i>	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	1 (1.5)	0 (0)	0 (0)
<i>Hepatozoon</i> sp.	0 (0)	0 (0)	2 (2.4)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
All species	45 (86.5)	90 (92.8)	65 (77.4)	76 (87.4)	22 (81.5)	63 (94.0)	56 (74.7)	61 (84.7)

Note: Some birds had more than one species of parasite. Numbers in parentheses are percentages.

Fig. 1. Increase in prevalence of hematozoa in female (open bars) and male (shaded bars) American kestrels with sampling date (logistic regression, date: $\chi^2 = 12.87$, df = 1, $P = 0.0003$; year: $\chi^2 = 6.50$, df = 1, $P = 0.01$). Numbers above the bars are sample sizes.

year). We documented a total of 10 species of hematozoa from five genera, *Haemoproteus tinnunculi* and *Haemoproteus brachiatus* being the most common species (Table 1). *Plasmodium* spp. occurred at low frequencies, but sample sizes were too small for further analyses. *Leucocytozoon* sp., *Hepatozoon* sp., and *Trypanosoma* spp. infections were reasonably rare (Table 1). Among samples that contained parasites, there was a trend for females to have more mixed infections of two hematozoan species (32/276) than males (14/202; $G = 3.01$, df = 1, $P = 0.08$), but there were no differences in the proportions of SY (11/83) and ASY (8/99) birds with mixed infections ($G = 1.29$, df = 1, $P = 0.26$).

There were no sex differences in overall parasite prevalence (logistic regression, $n = 561$, $\chi^2 = 0.36$, df = 1, $P = 0.55$), but parasite prevalence was higher in 1994 than 1995 ($\chi^2 = 6.19$, df = 1, $P = 0.01$). Prevalence generally increased with sampling date (Fig. 1), resulting in higher prevalence during the incubation than the prelaying period ($\chi^2 = 9.15$, df = 1, $P = 0.003$). After year and season (i.e., between prelaying and incubation) differences were controlled for,

parasite prevalence did not depend on the time of day when the sample was taken ($\chi^2 = 0.02$, df = 1, $P = 0.90$).

For analyses of host-age effects on parasite prevalence, we pooled data from the 2 years to obtain adequate sample sizes. During the prelaying period, more SY females had parasites (31/38) than ASY females (5/11; $G = 5.23$, df = 1, $P = 0.02$). This pattern had disappeared by the incubation period, when similar proportions of ASY females (44/46) and SY females (39/46) were parasitized (Fisher's exact test, $P = 0.16$). Among males there was a trend for more SY birds to be parasitized (8/8) than ASY birds (10/16) in the prelaying period (Fisher's exact test, $P = 0.066$), but we detected no difference in parasite prevalence between ages during incubation (5/7 for SY; 41/45 for ASY; Fisher's exact test, $P = 0.18$).

Considering infection only by *Haemoproteus* spp., the pattern of infection intensity was similar to that for overall prevalence (Table 2). When negative smears were included in the analysis, birds had more intense infections during incubation than during the prelaying period ($F_{[1,544]} = 7.84$, $P = 0.005$), and intensities tended to be higher during 1994

Table 2. Intensity of parasite (*Haemoproteus* spp.) infection in American kestrels sampled in north-central Saskatchewan during the prelaying and incubation periods in 1994 and 1995.

	Females				Males			
	1994		1995		1994		1995	
	Prelaying	Incubation	Prelaying	Incubation	Prelaying	Incubation	Prelaying	Incubation
Including unparasitized birds	46.8 ±10.6 (n = 52)	63.3 ±7.4 (n = 97)	179.8 ±44.3 (n = 80)	54.9 ±7.2 (n = 87)	63.1 ±16.9 (n = 27)	77.9 ±10.3 (n = 67)	66.7 ±18.1 (n = 66)	50.8 ±6.2 (n = 72)
Excluding unparasitized birds	57.8 ±12.5 (n = 42)	69.0 ±7.7 (n = 89)	243.8 ±57.9 (n = 59)	62.8 ±7.8 (n = 76)	77.5 ±19.5 (n = 22)	82.8 ±10.6 (n = 63)	100.1 ±25.8 (n = 44)	60.6 ±6.8 (n = 60)

Note: Intensity was measured as the number of parasites detected in 100 microscope fields using a 100× objective lens. Values are given as the mean ± SE.

Table 3. Correlation coefficients for the relationship between the intensity of *Haemoproteus* spp. infection in American kestrels and the date on which they were sampled.

Sex, season, and year	Including negative smears			Excluding negative smears		
	r	n	P	r	n	P
Prelaying females						
1994	0.38	52	0.005	0.19	42	0.22
1995	-0.10	80	0.35	-0.18	59	0.16
Incubating females						
1994	-0.01	97	0.96	-0.12	89	0.25
1995	0.13	87	0.23	0.27	76	0.02
Prelaying males						
(1994 and 1995)	0.33	93	0.001	0.23	66	0.06
Incubating males						
(1994 and 1995)	-0.09	139	0.29	-0.03	123	0.76

Note: Data were analyzed both with and without negative smears (see Methods).

than 1995, although not quite significantly so ($F_{[1,544]} = 3.61$, $P = 0.058$). No sex differences in intensity were detected ($F_{[1,544]} = 0.44$, $P = 0.51$). However, these apparent differences were due to higher prevalence in 1994, and also to the seasonal increase in prevalence (Fig. 1), rather than to any differences in intensity per se because when only birds infected with *Haemoproteus* spp. were considered (i.e., intensity ≥ 1), there was no effect of year ($F_{[1,451]} = 0.06$, $P = 0.87$), season ($F_{[1,451]} = 0.01$, $P = 0.95$), or sex ($F_{[1,451]} = 0.27$, $P = 0.72$) on *Haemoproteus* spp. intensity (see also “Repeated measures of parasitism” below). Parasite intensity was not related to the time of day when the sample was taken, either for the entire data set ($r = -0.04$, $n = 548$, $P = 0.40$) or for only the birds parasitized with *Haemoproteus* spp. ($r = -0.01$, $n = 455$, $P = 0.83$).

The effects of date on *Haemoproteus* spp. intensity were analyzed using analysis of covariance with year as a main effect and sampling date as covariate. For females in both the prelaying and incubation period, the year-by-date interaction was significant in all cases but one, regardless of which data set was used. We therefore analyzed each year separately. *Haemoproteus* spp. intensity increased with date for females during the prelaying period in 1994 (with negative smears) and also during incubation in 1995 (when negative smears were excluded; Table 3). For males, *Haemoproteus* spp. intensities increased with sampling date during the prelaying period, but were unrelated to date during incubation (Table 3).

Among prelaying females, infection intensities were higher in SY than in ASY birds when negative smears were included ($F_{[1,46]} = 6.70$, $P = 0.01$). As with seasonal effects on intensity, these results are probably an artefact of the larger number of SY than ASY females parasitized during the prelaying period, because when only positive smears were considered, no age effects were apparent ($F_{[1,29]} = 2.03$, $P = 0.17$). No age effects on female parasite intensity were detected during incubation (with negative smears: $F_{[1,90]} = 0.91$, $P = 0.34$; excluding negative smears: $F_{[1,81]} = 0.46$, $P = 0.50$). Among males, there were no differences in intensity of *Haemoproteus* spp. infection during either the prelaying period (with negative smears: $F_{[1,22]} = 0.99$, $P = 0.33$; excluding negative smears: $F_{[1,15]} = 2.82$, $P = 0.11$) or incubation (with negative smears: $F_{[1,50]} = 0.12$, $P = 0.73$; excluding negative smears: $F_{[1,43]} = 1.48$, $P = 0.23$).

Repeated measures of parasitism

We sampled 89 adult kestrels twice within the same year (once during the prelaying period and once during incubation). Generally, parasite prevalence was consistent among samples, although some birds acquired infections while some lost them (Table 4). The proportions of birds changing infection status did not differ between the sexes ($G = 1.60$, $df = 1$, $P = 0.21$). Changes in *Haemoproteus* spp. intensity between the prelaying and incubation periods did not differ between the sexes ($F_{[1,81]} = 0.56$, $P = 0.46$), but changes in intensity seemed to depend on year ($F_{[1,81]} = 3.80$, $P =$

Table 4. Changes in parasite prevalence among American kestrels sampled during both the prelaying and incubation periods within the same year (1994 or 1995).

	+/+	-/-	-/+	+/-
Females	40 (75.5)	3 (5.7)	8 (15.1)	2 (3.8)
Males	23 (63.9)	2 (5.6)	6 (16.7)	5 (13.9)

Note: +/+, status unchanged, parasitized; -/-, status unchanged, unparasitized; -/+, acquired infection between prelaying and incubation periods; +/-, lost infection between prelaying and incubation periods. Values are the number of birds sampled, with the percentage in parentheses.

0.055; Fig. 2). Paired comparison within individuals, analyzed by sex and year, showed that changes in *Haemoproteus* spp. intensity did not differ from 0 (Fig. 2; paired *t* tests, females in 1994: $t_{23} = 1.25$, $P = 0.23$; females in 1995: $t_{26} = -1.13$, $P = 0.27$; males in 1994: $t_9 = 0.66$, $P = 0.53$; males in 1995: $t_{22} = -1.57$, $P = 0.13$), suggesting that intensities remained relatively stable throughout the breeding season. When the sexes were pooled for analysis, the results were similar for 1994 (paired *t* tests, $t_{33} = 1.42$, $P = 0.17$); however, there was a trend for parasite intensities in kestrels to decrease between the prelaying and incubation periods in 1995 (paired *t* tests, $t_{49} = -1.94$, $P = 0.058$).

Twenty-five kestrels were sampled in both years of the study (Table 5). In most kestrels, parasite status was consistent between years. The proportion of birds changing infection status between years did not differ between the sexes (Fisher's exact test, $P = 0.65$), although sample sizes were small. In addition to these 25 birds, an additional 5 females were sampled twice in one year and once in the other. Of these, two were parasitized in all three samples, two were parasitized during incubation 1994 and 1995 but not in the prelaying period in 1995, and one was unparasitized when first sampled in the prelaying period in 1994 but was infected during incubation in both years.

Effects of food supply on parasitism

During incubation there were no differences in the number of voles (per 100 trap-nights) on territories between 1994 (12.1 ± 1.0 , $n = 89$ territories) and 1995 (10.9 ± 0.9 , $n = 87$ territories; $t_{174} = 0.96$, $P = 0.34$). There was no effect of vole abundance on the probability of an adult kestrel being parasitized for either females ($n = 117$, logistic regression, $\chi^2 = 0.10$, $df = 1$, $P = 0.75$) or males ($n = 85$, $\chi^2 = 0.20$, $df = 1$, $P = 0.65$). Similarly, there was no correlation between vole number and intensity of *Haemoproteus* spp. infection for either females (including negative smears: $r = 0.08$, $n = 117$, $P = 0.40$; excluding negative smears: $r = 0.09$, $n = 106$, $P = 0.38$) or males (including negative smears: $r = 0.05$, $n = 85$, $P = 0.64$; excluding negative smears: $r = 0.03$, $n = 74$, $P = 0.80$).

Parasitism of nestlings

In 1994, we sampled 251 nestlings between 22 and 29 days of age (fledging age). *Haemoproteus tinnunculi* was detected in only eight nestlings (five females, three males) from seven nests. Infection intensities were high (mean = 1445 ± 334 , range = 61–3000 parasite/100 fields, $n = 8$). Although sample sizes were too small for further analysis, all

infected nestlings fledged normally and did not appear unusual in terms of mass or size compared with unparasitized nestlings.

Discussion

Patterns of prevalence

American kestrels breeding in Pennsylvania (Apanius and Kirkpatrick 1988; Wiehn et al. 1997) and those sampled during fall migration in the eastern U.S.A. (Kirkpatrick and Lauer 1985) had lower prevalences of blood parasites than kestrels from north-central Saskatchewan (Table 1). A small sample of kestrels (5) sampled in southern Quebec were all infected with hematozoa (Maloney et al. 1984). Intensities of *Haemoproteus* spp. infection in our population (Table 2) were approximately 3 times higher than in Pennsylvania (Wiehn et al. 1997) and nearly twice those reported in the Quebec study (Maloney et al. 1984). In addition, the diversity of parasite species in our study area appears high: we recorded 10 species (Table 1), whereas only 2 species, *H. tinnunculi* and *Trypanosoma* sp., have been found in Pennsylvania (Apanius and Kirkpatrick 1988; Wiehn et al. 1997) and a single species, *H. tinnunculi*, in Quebec (Maloney et al. 1984).

Our study area was in the boreal forest (for details see Gerrard et al. 1996), whereas in Pennsylvania, birds were sampled in habitats dominated by farmland (Wiehn et al. 1997). With few exceptions, all known vectors of *Haemoproteus* spp. are ceratopogonids of the genus *Culicoides* (Desser and Bennett 1993). Vectors are probably more common in the boreal environment (Bennett 1993), so the differences in prevalence, intensity, and diversity of parasite species between our study area and Pennsylvania probably result from differences in vector abundance and composition between the two areas. Similarly, Bennett et al. (1995) also implicated variation in vector populations as an explanation for differences in hematozoa infection in three populations of Fenno-Scandian pied flycatchers (*Ficedula hypoleuca*).

Using blood smears as a method of surveying trypanosomes is inadequate (Bennett 1962). In studies using centrifuge or culture techniques, much higher prevalences have been found than when blood smears were examined (Kirkpatrick and Lauer 1985; E. Korpimäki, personal communication). For example, Apanius and Kirkpatrick (1988) detected trypanosomes in 43% of kestrels in their study area in Pennsylvania, although they did not state whether trypanosomes were also detected in the smears from these birds. In contrast, Wiehn et al. (1997), working in the same study area but in different years, found no evidence of trypanosome infections in blood smears. Therefore, the true prevalence of *Trypanosoma* spp. in our population of kestrels remains unknown. In addition, the taxonomy of trypanosomes is uncertain (see Baker 1976; Apanius 1991), and although we identified three species, they should probably all be considered part of the *T. avium* complex.

Parasite prevalence increased as the breeding season progressed (Fig. 1), which could have been due either to the birds acquiring new infections or to spring relapses of chronic infections acquired in previous years (see Bennett and Cameron 1974). We suspect the latter, because parasite prevalence in our kestrels increased most rapidly in early

Fig. 2. Change (mean \pm SE) in intensity of infection with *Haemoproteus* spp. within individual American kestrels between the prelaying and incubation periods. Change in intensity was calculated by subtracting incubation intensity from prelaying intensity and is expressed as the number of parasites in 100 microscope fields (see Methods). ■, 1994; ●, 1995. Numbers above the error bars are sample sizes.

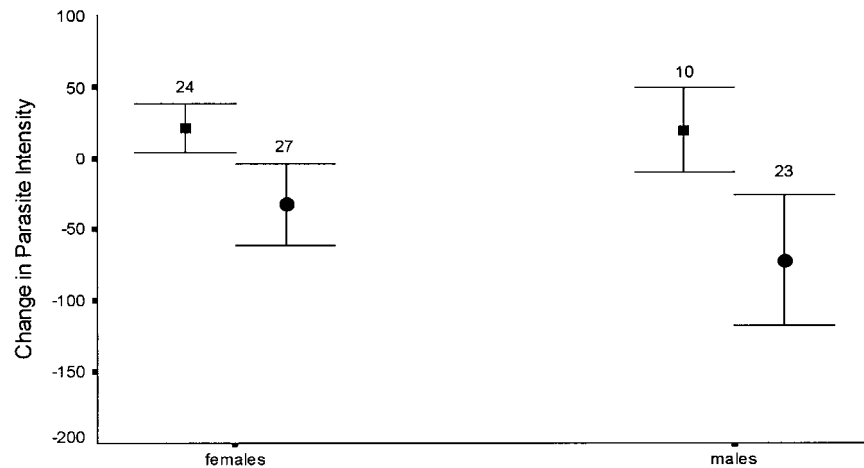


Table 5. Changes in parasite prevalence among American kestrels sampled in both 1994 and 1995.

	+/+	-/-	-/+	+/-
Females	11 (73.3)	1 (6.7)	2 (13.3)	1 (6.7)
Males	7 (70)	0 (0)	1 (10)	2 (20)

Note: +/+, status unchanged, parasitized; -/-, status unchanged, unparasitized; -/+, acquired infection between 1994 and 1995; +/-, lost infection between years. Data are number of birds sampled, with the percentage in parentheses. See the text for additional information on five females from which three samples were taken.

May (Fig. 1) and vectors probably do not become active until after that time (Bennett 1960; Bennett and Fallis 1960). Intensity of *Haemoproteus* spp. infections also increased with date, but these effects were not consistently significant (Table 3). We suspect that date effects in the prelaying period (females in 1994 and males in both years) may also be the result of a relapse of chronic infections, because in neither case were the relationships significant when negative smears were excluded from analyses (Table 3). This suggests that the observed increases with date were related to changes in prevalence rather than in intensity per se. Moreover, we did not detect differences among infected birds in intensity of *Haemoproteus* spp. infections between the prelaying and incubation periods (Table 2).

We were unable to detect any differences in either parasite prevalence or intensity that were attributable to gender (Tables 1 and 2). Similarly, other studies have failed to show sex biases in hematozoa parasitism (e.g., Allander and Bennett 1994; Dale et al. 1996; Wiehn et al. 1997). These results are perhaps surprising, given that androgens are known to have immunosuppressant effects, while estrogen may stimulate the immune system (see review in Nelson and Demas 1996). Using meta-analysis, several recent studies have shown male biases in parasitism of both birds and mammals (Poulin 1996; Schalk and Forbes 1997). However, McCurdy et al. (1998) used meta-analysis to examine sex-biased parasitism of birds by hematozoa, and showed that, contrary to expectation, females were more heavily parasit-

ized than males. Our finding that female kestrels tended to have more mixed infections of blood parasites is consistent with the results of McCurdy et al. (1998).

Bennett and Bishop (1990) hypothesized that because parasitic infections accumulate in hosts over time, older birds are more likely to be parasitized but their infection intensities are expected to be lower. Indeed, the results from numerous studies support these ideas (Weatherhead and Bennett 1991, 1992; Davidar and Morton 1993; Allander and Bennett 1994; Korpimäki et al. 1995; but see Dale et al. 1996). In contrast, we found that more SY than ASY birds had parasites, although intensity did not appear to be affected by age. We suspect that because these effects had disappeared by the incubation period, presumably before vectors were active (see above), the differences in prevalence between age-classes that we documented were the result of differential recrudescence of chronic infections acquired in the previous summer and fall. Although spring relapses are thought to be mediated by hormones (Chernin 1950; Atkinson and van Riper 1991), if age-classes differ in quality or condition, young birds may experience spring relapses sooner than older birds.

Korpimäki and co-workers suggested that variation in food supply may be responsible for detrimental effects of blood parasites on reproduction in Tengmalm's owls (*Aegolius funereus*; Korpimäki et al. 1993) and may explain among-year differences in parasite loads of Eurasian kestrels (*Falco tinnunculus*, Korpimäki et al. 1995; Wiehn and Korpimäki 1998; Wiehn et al. 1999). Indeed, a supplemental-feeding experiment confirmed that food supply mediated blood parasite loads of female Eurasian kestrels (Wiehn and Korpimäki 1998). The effects of supplemental feeding were sex-specific because only females responded by reducing the amount of time spent in flight-hunting (Wiehn and Korpimäki 1998). We are unable to invoke a similar explanation for the higher parasite prevalence in American kestrels in 1994 than in 1995 (Table 1). Vole abundances were the same in the two years, and we did not detect a relationship between parasitism of individual kestrels and the number of voles available on their territories. It is important to

note, however, that the variation in vole numbers was much lower in our study area than in that of Korpimäki et al. (1993, 1995). Korpimäki et al. (1995) also suggested that yearly differences in parasite load may be due in part to differential exposure of birds during previous years, and such a scenario may explain our results. American kestrels have high rates of natal and breeding dispersal (unpublished data), and many birds that we sampled would have been raised and (or) bred in other areas that presumably varied in vector composition and population density (Bennett and Cameron 1974; Bennett et al. 1995).

Parasitism of nestlings

Only a small proportion of nestling kestrels showed signs of hematozoa infection. These results are consistent with those of other studies in which nestling American kestrels (Maloney et al. 1984; Apanius and Kirkpatrick 1988) and other species were found to be relatively free of parasites (Weatherhead and Bennett 1991; Davidar and Morton 1993; Korpimäki et al. 1993, 1995; but see Merino and Potti 1995; Merino et al. 1996). A lack of hematozoan gametocytes in circulating blood cells may indicate that nestlings have not yet been exposed to infection, or that insufficient time has elapsed since exposure for an infection to become patent (Fallis and Bennett 1961). The prepatent period for *Haemoproteus* spp. is about 14 days (Desser and Bennett 1993), so presumably the time between hatching and sampling was sufficient for the infection to become patent. In the eight nestling kestrels infected with *H. tinnunculi*, intensities were extremely high. These results are not surprising, given that young birds are immunologically naive to many pathogens and parasites (e.g., Merino and Potti 1995; Ros et al. 1997).

Repeated measures of parasitism in adults

Relatively few studies have presented data on repeated sampling of individual birds within a year (but see Bennett and Bishop 1990). We found that when individuals were sampled twice in the same year or in successive years, repeatability was high, with approximately 75% of birds having the same parasitic infection status (Tables 4 and 5). Among birds whose status changed, most acquired infections, although others apparently were able to get rid of their infections (Table 4). Our results are similar to those reported by Bennett and Bishop (1990), Weatherhead and Bennett (1991, 1992), and Korpimäki et al. (1995). Weatherhead and Bennett (1991, 1992) interpreted changes in parasite status as being detrimental for testing Hamilton and Zuk's (1982) hypothesis, mainly because one can then never be certain whether a negative smear is a reflection of resistance or of lack of exposure (see also Shutler et al. 1996). While we concur that birds which change parasite status may pose a problem for testing Hamilton and Zuk's (1982) hypothesis, we emphasize that these are valuable data. First, many traits of interest to researchers are variable or plastic (e.g., condition, coloration of exposed integument), and by comparing such traits in birds when they are parasite-free and when they are infected, it is possible to gain insight into the true effects of parasites. Second, if birds are losing infections between samples, either completely or partially, this may rep-

resent a positive immune response. It may therefore be possible to assess the quality of an animal by its ability to rid itself of, or otherwise control, infections between sampling periods.

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