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Prevalence of blood parasites in European passeriform birds

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Variation in the prevalence of blood parasites among species of birds has been used to test hypotheses about the effects of sexual selection and parental investment on disease resistance, and how vector abundance influences infection. However, the factors causing this variation are still poorly understood. We assessed the statistical effects of biogeographic, plumage-related and life-history traits on the prevalence of the blood parasites *Plasmodium*, *Haemoproteus*, *Leucocytozoon* and *Trypanosoma* in European passerine birds. Most of the variation in parasite prevalence occurred at low taxonomic levels. Brighter male plumage and greater host body mass were associated with higher prevalence, explaining 32% of the total variation. Male plumage brightness remained a significant factor when we controlled for phylogenetic effects. These relationships were driven primarily by simuliid-transmitted parasites (*Leucocytozoon*, *Trypanosoma*), which were more frequent in species with northern distributions. Host species with greater maximum longevity and shorter nestling periods had higher prevalences of *Plasmodium*; however, the effect was not stable after controlling for phylogeny using pairwise contrasts. Coevolution between hosts and parasites appears to create temporal and spatial variation that disconnects haematozoan prevalence from evolutionarily conservative life-history traits while creating some positive associations with traits that are phylogenetically labile. Clearly, ecologists should be cautious in relating patterns of variation in haematozoan prevalence to particular host traits.

Keywords: Passeriformes; prevalence; haematozoa; avian malaria; *Plasmodium*; *Leucocytozoon*

1. INTRODUCTION

Understanding the epizootiology of haematozoa in birds has been a focus of research for at least a century, not least because of the implications of this model system for human malaria (Atkinson & van Riper 1991; Modiano *et al.* 1996; Miller *et al.* 2002; Patz *et al.* 2002). One of the prominent features of avian malaria is a wide variation among host species in parasite prevalence. Prevalence is measured as the proportion of individuals infected with haematozoa, traditionally recorded by microscopic inspection of blood smears (Peirce 1981), where haematozoa can be observed during the patent period following infection (van Riper *et al.* 1994). After this period, the parasites may be cleared from the host or enter a latent stage, remaining in host tissues, if not the blood, for an extended period and, potentially, for life (Manwell 1934; Sergent & Sergent 1952; Jarvi *et al.* 2002, 2003). Chronic infections can render a host resistant to further infection (Ahmed & Mohammed 1978). *Plasmodium* species in particular tend to decrease to chronic levels and remain dormant in the hosts' organs, leaving the blood circulation (van Riper *et al.* 1994). However, it has been shown that these dormant parasites can resume their haemoparasitic stage when hosts are stressed, as is often the case at the onset of the breeding season (Applegate 1970; Beaudoin *et al.* 1971). Prevalence of haematozoa is a function of the transmission rate of the parasites by arthropod vectors and consequently of the abundance, host specificity and ecological requirements of the vectors (van Riper *et al.* 1986). Moreover, prevalence is affected by the immunological capacity of the host to either prevent parasite infection or to clear established infections (Atkinson & van Riper 1991). The immunological mechanisms involved in resistance are poorly understood, even in humans (Congdon *et al.* 1969; Davies 1999; Buckling & Read 2001), and the information and tools to forecast human malaria outbreaks or reduce the disease's virulence are far from sufficient (Miller *et al.* 2002).

Many evolutionary biologists have argued that haematozoan prevalence primarily reflects immunocompetence. Focusing on variation in immune system competence within populations, Hamilton & Zuk (1982) associated parasite prevalence with plumage brightness in a set of North American passerine species. They proposed that higher parasite loads in a host population increased the variance in fitness among individuals according to the quality of their immune responses. Parasite loads of individuals, hence relative fitness, can be visualized by their effects on elaborated secondary sexual traits, which are therefore favoured by females as an aid to choosing males of overall high genetic quality (sexual selection). Using the compilation of Peirce (1981) of the haematozoa of European birds, Read (1987) was able to confirm the predicted positive relationship between plumage brightness of bird species and parasite prevalence. However, another study using a different scoring system for plumage brightness failed to detect such an effect (Read & Harvey 1989).

Ricklefs (1992) suggested that variation in immunocompetence might account for variation in haematozoan prevalence among higher taxa. In a family-level analysis of prevalence data compiled for 40 000 non-raptorial neotropical and nearctic land birds, he found that the prevalence of blood parasites was inversely correlated with length of the embryonic development period, and he

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concluded that prolonged embryonic development might result in a more diversified antibody response, enabling individuals to cope better with haematozoa. Tella *et al.* (1999) were able to confirm the correlation between prolonged incubation period and low haematozoan prevalence in a sample of 1264 individuals of 20 species of diurnal raptors in Spain. In addition, they detected higher parasite prevalence in raptors that lived in forest habitats, as well as in species with larger geographical ranges.

At small geographical and ecological scales (height strata within forests, altitude gradients, island versus mainland), parasite prevalence varies predictably with the abundance of vectors, regardless of host species (Bennett *et al.* 1974; Super & van Riper 1995; Garvin & Remsen 1997; Apanius *et al.* 2000; Sol *et al.* 2000). A classic example is the restriction of *Plasmodium relictum* in Hawaiian birds to elevations within the range of the introduced vector of this parasite, *Culex quinquefasciatus* (van Riper *et al.* 1986). However, the relationships of haematozoan prevalence to patterns of host distribution at large scales have not been explored in detail.

We analyse a comprehensive dataset on prevalence of haematozoa in European passerine birds (Peirce 1981), the same used by Read (1987). We extended this dataset to 14 812 individual birds by including studies published between 1981 and 2003. We ask whether variation in prevalence is related to factors that affect exposure of host species to vectors, such as geographical distribution and habitat. We also examine whether prevalence is influenced by host traits that might indicate how effectively they resist or clear parasite infections. Specifically, we tested whether a species' position on the continuum of slow/fast life histories (see Ricklefs & Wikelski 2002) determines parasite prevalence ('slow' species having smaller clutch size, slower developmental period and longer lifespan should have higher resistance against infections) or whether prevalence can be better explained by traits that are less phylogenetically conserved, such as plumage characteristics.

2. MATERIAL AND METHODS

Peirce (1981) summarized the prevalence of four genera of haemoparasites (*Haemoproteus*, *Plasmodium*, *Leucocytozoon* and *Trypanosoma*) in blood smears of adult birds of undetermined sex that breed in Europe. Data were compiled from more than 120 studies conducted over the whole of Europe between 1890 and 1978. These data were supplemented by more recent studies (Bennett *et al.* 1982; Haberkorn 1984; Valkiunas 1987; Allander & Bennett 1994; Sundberg 1995; Dale *et al.* 1996; Allander 1997; Blanco *et al.* 1997; Merino *et al.* 1997, 2000; Siikamaki *et al.* 1997; Merilä & Andersson 1999; Rintamaki *et al.* 1999; Valkiunas *et al.* 1999; Hatchwell *et al.* 2000; Shurulinkov & Golemansky 2002; Valera *et al.* 2003) and are presented in electronic Appendix A. We restricted our analysis to passerine bird species that had at least 20 individuals sampled. In total, our data included 14 812 individuals (74 species in 39 genera in 17 families), 4232 (28.6%) of which were infected with one or more haematozoan parasites.

Independent variables used in this analysis are summarized in electronic Appendix B. European breeding range and estimates of population size within Europe were taken from Hagemeijer & Blair (1997). We feel justified in combining data on parasite prevalence with atlas data because the parasite data were collected at many localities throughout Europe. We characterized the geographical distribution of each species by an ordination of a species × country-matrix of population abundance. For each species, we ranked the top 10 countries according to population estimates (bar graphs in Hagemeijer & Blair 1997), with the country holding most of the population of a particular species assigned a score of 10. Some localized species, occurring in less than 10 countries, were assigned values descending from 10. Countries with fewer than seven entries (Andorra, Iceland, Lithuania, Madeira, Malta, Moldova and Slovenia) were not included in the analysis. This procedure yielded a matrix with ranked abundance scores of 74 species in 37 European countries. A de-trended correspondence analysis (DCA; ter Braak (1995); using PCORD for Windows; McCune & Mefford (1997)) identified a single prominent axis (eigenvalue = 0.443, second eigenvalue = 0.210) representing a north–south gradient. The European population sizes of host species were obtained from Hagemeijer & Blair (1997) and $\log_{10}$-transformed.

We recorded presence/absence of species in 14 habitat types following Ehrlich *et al.* (1994). This yielded a matrix of habitat occurrence for 74 species of birds. Again, we used DCA to extract a single prominent axis (eigenvalue = 0.597, second eigenvalue = 0.355), which represented a gradient of habitat types from forests through woodlands and gardens to moorlands, rivers and reed beds.

Several life-history variables (body mass, clutch size, incubation period, fledging period, migratory status, oldest reported individual and adult annual survival rate) were obtained from Cramp & Simmons (1977–1994). The age of the oldest reported individual is highly correlated with adult survival from Cramp & Simmons (1977–1994; $r = 0.45$, $p = 0.003$, $n = 41$) and with adult survival reported by Dobson (1990; $r = 0.60$, $p < 0.0001$, $n = 38$). We used oldest reported age because it is available for more species in our sample than are survival rates. Specifically, our dataset contains 63 observations for oldest age, 43 for adult survival from Cramp & Simmons (1977–1994) and 39 for adult survival from Dobson (1990).

An egg-mass-corrected index for incubation period was calculated after Ricklefs (1992). Finally, we tabulated the degree of plumage dimorphism (overall, plus carotenoid-based colours, melanin-based colours and structural colours) after Owens & Hartley (1998) and plumage brightness after Møller & Birkhead (1994).

Prevalence (P) was arcsine-transformed [$\arcsin\sqrt{P}$]. All other data were log-transformed, except for the DCA scores and dimorphism and brightness values. The complete dataset is presented in electronic Appendix B.

We estimated general linear models (SAS Institute 1999–2000) with prevalence as the dependent variable, and geography, habitat and life-history traits as independent variables. To control for variation in sample size (ranging from 20 to 1523 individuals sampled per species), species were weighted by the square root of the number of individuals examined. Variables that did not contribute significantly to the model were removed in a stepwise fashion. Individual contributions of variables were summarized as F-values and associated probabilities (type III sums of squares); regression coefficients indicated the strength and the direction of the relationship between the dependent and independent variables. As we used 19 explanatory variables for each of the five response variables (overall prevalence; prevalences of *Haemoproteus*, *Leucozytozoon*, *Plasmodium* and

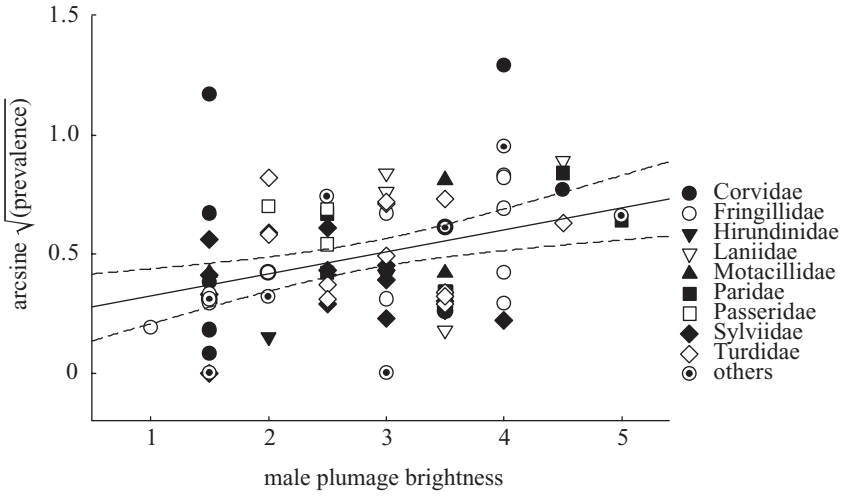

Figure 1. Male plumage brightness (after Møller & Birkhead 1994) and prevalence of haematozoa in European passeriform birds ($F_{1,73} = 21.9$; $p < 0.0001$).

Trypanosoma), we reduced the acceptable level of significance to $p = 0.0026$ (= 0.05/19) (= Bonferroni correction).

To determine the distribution of variance according to taxonomic level we used a nested random effects analysis (species within genus within families; taxonomy according to Sibley & Ahlquist (1990); Proc NESTED in SAS). Moreover, we tested the robustness of our results by employing phylogenetically matched pairwise comparisons (Martins 2000; taxonomy according to Summers-Smith 1988; Sibley & Ahlquist 1990; Winkler & Sheldon 1993; Fehrer 1996; Slikas *et al.* 1996; Price *et al.* 1997; Cibois & Pasquet 1999; Wink *et al.* 2002). The degrees of freedom for species-pairs analyses were half the number used in species analysis, reducing statistical power.

3. RESULTS

The average prevalence of haematozoa in the sample of 14 812 European passerines was 26%. A weighted general linear model relating prevalence to all independent variables showed that only male plumage brightness (positive; $F_{1,73} = 21.9$; $p < 0.0001$; see figure 1) and log(body mass) (positive; $F_{1,73} = 11.6$; $p = 0.0011$) were important effects in a model that explained 32% of the total variation ($R^2 = 0.32$). Both of these effects entered positive. By contrast, female plumage brightness did not enter into the model, although it was correlated with male plumage brightness (Pearson's $r = 0.71$; $p < 0.0001$). Because Corvidae are atypical passerine birds with respect to body mass and plumage brightness, we excluded them from a second analysis. However, male plumage brightness (positive; $F_{1,66} = 19.44$; $p < 0.0001$) and log(body mass) (positive; $F_{1,66} = 15.72$; $p = 0.0002$; see figure 2) were still important predictors of overall parasite prevalence ($R^2 = 0.35$). This suggests that the relationship between the two explanatory variables and prevalences was not driven by differences between families. An analysis using phylogenetically matched species-pairs confirmed the effect of male plumage brightness on prevalence (positive; $F_{1,35} = 2.42$; $p = 0.021$), but was unable to confirm the effect of log(body mass) on prevalence. However, in the species-pairs analysis, the first DCA axis for geography became a significant predictor of prevalence, with higher prevalences in birds with more northerly distributions ($F_{1,35} = 2.35$; $p = 0.025$).

(a) *Individual parasite lineages*

Overall prevalence of blood parasites in bird species was highly correlated with the prevalence of *Haemoproteus* (Pearson's $r = 0.55$; $p < 0.0001$), *Leucocytozoon* ($r = 0.66$; $p < 0.0001$) and *Trypanosoma* ($r = 0.42$; $p = 0.0002$) and less so with *Plasmodium* ($r = 0.25$; $p = 0.04$). There were no significant correlations between prevalences of different parasite genera, except for a positive correlation between *Leucocytozoon* and *Trypanosoma* ($r = 0.51$; $p < 0.0001$).

Prevalence of *Haemoproteus* was barely explained by male plumage brightness score (positive; $F_{1,73} = 9.55$; $p = 0.0029$; $R^2 = 0.13$), and we regard this correlation as weak if even relevant, as it is close to our Bonferroni-corrected level of $p < 0.0026$. By contrast, *Leucocytozoon* prevalence was significantly related to log(body mass) (positive; $F_{1,73} = 13.4$; $p = 0.0005$; see figure 3) and marginally related to the first DCA axis ($F_{1,73} = 9.04$; $p = 0.0037$), corresponding to a north–south gradient of species ranges. This model explained 22% of the variation in *Leucocytozoon* prevalence ($R^2 = 0.22$). Family and the family × log(body mass) interaction were not significant, which shows that the body mass effect on *Leucocytozoon* prevalence resides within and does not differ between families (family: $F_{1,73} = 0.29$; $p = 0.98$; family × log(body mass) $F_{1,73} = 0.30$; $p = 0.98$). Prevalence of *Plasmodium* was related to oldest host individual reported in Cramp & Simmons (1977–1994) (positive; $F_{1,73} = 16.6$; $p = 0.0001$; see figure 4) and the length of the period between hatching and fledging (negative; $F_{1,73} = 12.2$; $p = 0.0009$). Both variables together explained 25% of the variation in *Plasmodium* prevalence ($R^2 = 0.25$). Again, family ($F_{1,73} = 0.29$; $p = 0.28$) and the family × oldest interaction ($F_{1,73} = 1.91$; $p = 0.072$) were not significant. Prevalence of *Trypanosoma* was exclusively related to the first axis of the DCA, which corresponded to a north–south gradient ($F_{1,73} = 18.8$; $p < 0.0001$; see figure 5). Both family ($F_{1,73} = 1.05$; $p = 0.43$) and the family × first DCA axis interaction ($F_{1,73} = 0.70$; $p = 0.73$) were not significant.

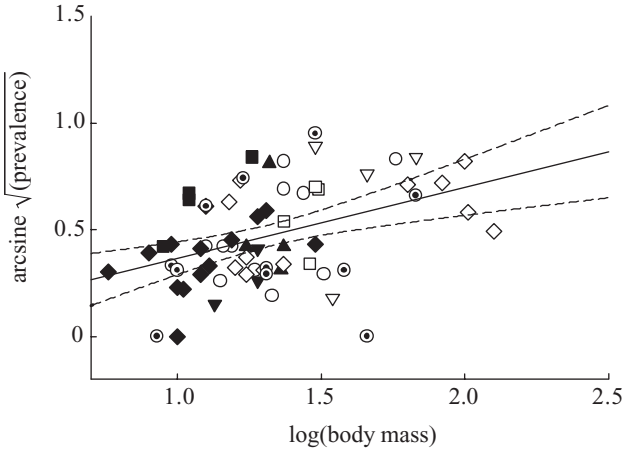

Figure 2. Log_{10} body mass and prevalence of haematozoa in European passeriform birds, excluding Corvidae ($F_{1,66} = 15.7$; $p = 0.0002$). For symbols see figure 1.

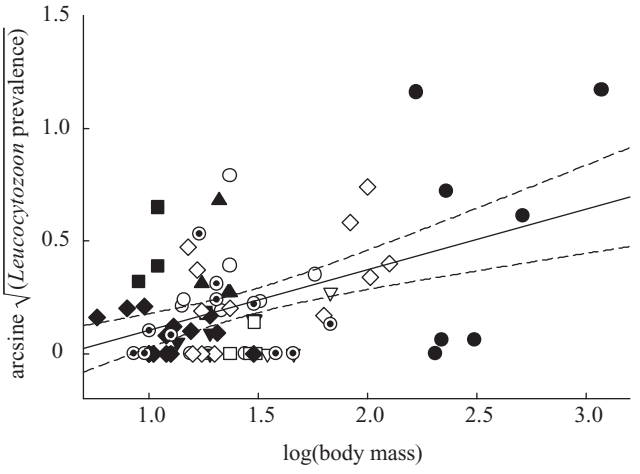

Figure 3. Log_{10} body mass and prevalence of *Leucocytozoon* in European passeriform birds ($F_{1,73} = 13.4$; $p = 0.0005$). For symbols see figure 1.

None of the phylogenetically matched comparisons based on individual types of parasites was significant.

(b) *Phylogeny*

A nested random-effects analysis showed that most of the variation in prevalence is expressed among species within genera (see table 1) with little variation present among genera or families of passerine birds. Likewise, male plumage brightness, population size and geographical distribution show high variance among species within genera. Population size, however, is not related to variation in prevalence, as shown above. By contrast, mass and related life-history variables (incubation index, fledging period and clutch size) exhibit most variation at the higher taxonomic levels of genera within families and among families. Consequently, the marginally significant association between mass and parasite prevalence must reside on the family level. We did not detect a significant effect of habitat gradient on prevalence in our data; most of the variation in the habitat gradient resides at the taxonomic level of genus within family where there is no statistically significant variation in prevalence. Brightness of males, however, is variable at both the species and genus levels, where most of its association with overall prevalence must reside. In general, prevalence as well as population size and geographical distribution appear not to be phylogenetically conserved, in contrast to habitat and life-history traits. Consequently, the explanatory variables that we were able to identify in our analysis bear out high variation on the genus and species levels.

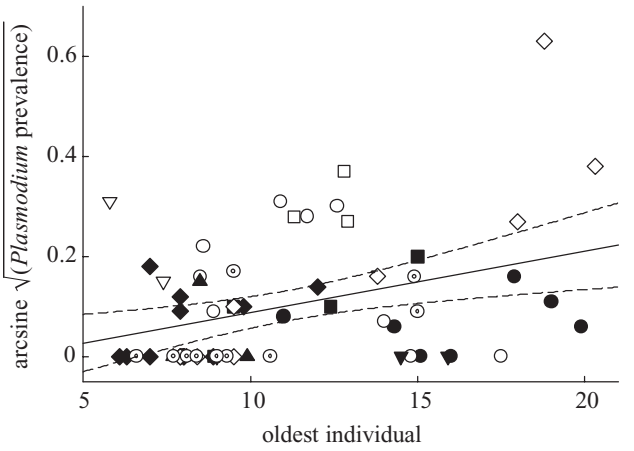

Figure 4. Oldest recorded individual reported by Cramp & Simmons (1977–1994) and prevalence of *Plasmodium* in European passeriform birds ($F_{1,73} = 16.6$; $p = 0.0001$). For symbols see figure 1.

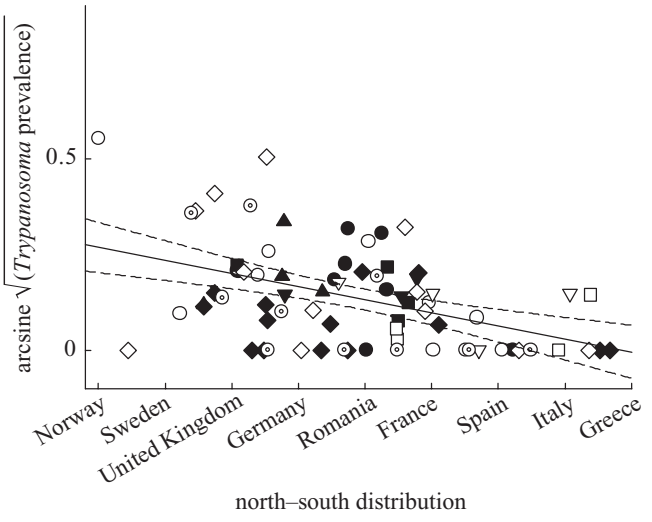

Figure 5. The first axis of the DCA corresponding to a north–south gradient and prevalence of *Trypanosoma* in European passeriform birds ($F_{1,73} = 18.8$; $p < 0.0001$). The approximate positions of several European countries on the axis are indicated. For symbols see figure 1.

4. DISCUSSION

The most robust result of our analyses is that haematozoan prevalence is positively related to male plumage brightness. Body mass was a reasonable predictor of prevalence without taking phylogeny into account, whereas geographical distribution had a significant effect in the paired-species analysis. In addition, prevalences of simuliid-transmitted parasites *Leucocytozoon* and *Trypanosoma* showed some association with geography of the host species, although the signal disappeared when we

Table 1. Results from a nested random effects analysis: proportion of variance (%) on three taxonomic levels.

variable	family	genus	species
$\sin^{-1}$ sqrt (haematozoa prevalence)	6	16	78
brightness of males scored 1–5	5	40	55
$\log_{10}$(European population size per species)	0	28	72
1st DCA axis of geographical distribution (see text)	0	53	47
$\log_{10}$(body mass) (g)	73	19	8
incubation index: residuals of regression of incubation period on egg mass	32	50	18
fledging period	73	10	17
median clutch size	52	25	23
1st DCA axis of habitat (see text)	21	52	27

used pairwise comparisons. *Haemoproteus* prevalence was not strongly related to any parameter, although *Haemoproteus* was the most common parasite in the sample. In general, parasite prevalence was conspicuously unrelated to development and lifespan, except that *Plasmodium* prevalence was higher in species with longer lifespans and lower in species having long fledging periods.

A frequently raised issue with analyses of haematozoan prevalence is the repeatability of the measure between locations and between time-points (Weatherhead *et al.* 1991; Yezerinac & Weatherhead 1995). Ricklefs *et al.* (2005) analysed 33 haematozoan prevalence values for 11 species collected in 10 studies and representing 1395 individual North American passerine birds. An ANOVA on arcsine-transformed prevalence weighted by the square root of sample size for individual species in each study revealed both a significant study effect ($F_{9,13} = 4.0$, $p = 0.012$) and a significant species effect ($F_{10,13} = 3.49$, $p = 0.019$) (overall $F_{19,13} = 4.68$, $p = 0.0034$, $R^2 = 0.87$). Thus, study and host species together explained 87% of the variation in prevalence. However, host species alone accounted for only 48% of the variation, which was marginally significant ($F_{10,22} = 2.01$, $p = 0.083$). The residual variance among studies within species was the square of 0.075 arcsine units, which is similar to the variance of prevalence on the species level in our nested random effects analysis (= 0.067). Thus, when prevalence for a species is estimated from one or a few studies, a large component of the variance might reflect variation among localities and sampling period within species, rather than species traits. We suspect that a considerable amount of variation at the species level in the European dataset is due to sampling time and sampling location (Bennett & Cameron 1974; Weatherhead *et al.* 1991; Merilä *et al.* 1995). Consequently, the most stable relationship between host traits and prevalences of haematozoa should reside at deeper phylogenetic levels than variation among species.

(a) *Taxonomic hierarchy of pattern*

Most of the variation in haematozoan prevalence occurs at the taxonomic level of species within genera, as do variation in population size, geographical distribution and plumage brightness. This result for prevalence is robust: even if half the variance at the species level resulted from differences between studies, as demonstrated in a sample of North American birds (R. E. Ricklefs, unpublished data), variation in prevalence at the species level still would exceed variation at deeper phylogenetic levels. Gaston (1996) found that the range size of birds is similarly labile. All of these parameters show idiosyncratic temporal dynamics that are poorly understood. Thus, the relatively low repeatability of prevalence in comparative studies (Yezerinac & Weatherhead 1995) might not reflect sampling error, but rather originate from the labile character of this trait (Fallon *et al.* 2003). It seems plausible that prevalence of haematozoa varies on short time-scales that characterize, for example, coevolution between parasites and their hosts (Richner 1998), competition between haematozoan species (Paul *et al.* 2002), or runaway sexual selection (Hamilton & Zuk 1982). Accordingly, the variation in parasite prevalence at a low host taxonomic level would reflect changes in host–parasite relationships that occur over shorter intervals than the diversification of species within a clade, as indicated by analyses of coevolution of haematozoa and their hosts (see Bensch *et al.* 2000; Ricklefs & Fallon 2002; Fallon *et al.* 2003). This is further corroborated by the phylogenetically matched pairwise comparison, where parallel evolution at low phylogenetic levels yields significant parameter effects for male plumage brightness and geographical distribution.

(b) *Continental and local scales*

On a continent-wide level, our study confirms patterns discerned by Greiner *et al.* (1975), who studied the distribution of haematozoa in 57 026 passeriform and non-passeriform birds in North America. They found a generally homogeneous distribution of *Haemoproteus* and *Plasmodium* prevalence among large regions within northern America but detected fewer haematozoa in the Arctic barrens. However, Greiner *et al.* (1975) also reported a higher prevalence of *Leucocytozoon*, a simuliid-transmitted parasite, in montane regions and boreal biomes, independent of host identity. Similarly, in the European data analysed here, parasites transmitted by simuliid vectors (*Leucocytozoon*, *Trypanosoma*) showed a higher prevalence in northern Europe. Thus, at a continental scale, biome ecology strongly influences vector availability, which in turn affects the prevalence of parasites. Absence of suitable vectors has also been cited as a factor contributing to the lower prevalence of blood parasites on islands close to continental land masses (Bennett *et al.* 1974; Bennett & Coombs 1975; Super & van Riper 1995). Interestingly, the positive association of haematozoan prevalence with male plumage brightness was driven by simuliid-transmitted parasites.

On a local scale, Greiner *et al.* (1975) reported a positive correlation between nest height of host species (a

surrogate for within-habitat niche position) and the prevalence of haematozoa. Nest height was confirmed as an important factor in a study by Garvin & Remsen (1997) on haematozoan prevalence in a coastal area of the southern United States. Similarly, Tella *et al.* (1999) found that the prevalence of haematozoa in their sample of Spanish birds of prey was affected by habitat, among other factors: forest species had higher prevalence than open-country species. This result corresponds to the finding of Merilä *et al.* (1995) of higher haematozoan prevalence in greenfinches (*Carduelis chloris*) in regions with natural forests, compared with regions with greater human disturbance. However, in our study of European passerines, we were unable to detect any effect of habitat. Most of the variation in prevalence resides at the taxonomic level of species within genus, whereas habitat preference varied at the genus-within-family level. As a consequence, processes affecting prevalence on a local level, including variation among habitats, tend to be obscured on larger spatial scales. For example, some bird species have been documented to shift habitats on a continental scale: dunnocks (*Prunella modularis*; Davies 1992), water pipit (*Anthus spinoletta*), fieldfare (*Turdus pilaris*), robin (*Erithacus rubecula*), bluethroat (*Luscinia svecica*) and willow warbler (*Phylloscopus trochilus*) (Cramp & Simmons 1977–1994). Thus, average parasite prevalence for these species does not represent a particular habitat.

(c) *Host traits affecting prevalence*

Presumably, prevalence of haematozoa in birds reflects a balance between exposure and resistance to infection (van Riper *et al.* 1994). In this study, we found that overall prevalence was independent of several indices of exposure, including habitat and regional population size. The clearest connection between exposure and prevalence involved *Leucocytozoon* and *Trypanosoma*, which are transmitted by vectors (simuliids) that are more common in northern environments.

Among life-history traits of individuals, only body mass and fledging period were strongly correlated with prevalence. Body mass might affect infection rate because larger birds produce greater quantities of carbon dioxide, a signal used by dipteran vectors to locate hosts (Sutcliffe 1986; Wernsdorfer & McGregor 1988). Moreover, larger birds present more surface area for parasite attacks (Atkinson & van Riper 1991). In our study, body mass alone explained over 20% of the variation in prevalence among host species. Other life-history traits associated with body mass, including nestling growth rate and incubation period, did not exhibit significant unique effects on overall prevalence. Only *Plasmodium* prevalence was influenced by the length of the period between hatching and fledging, which had a negative coefficient, and by maximum lifespan. The positive relationship between *Plasmodium* prevalence and maximum lifespan is not surprising, as some *Plasmodium* species evade clearance, enter latent stages of chronic infection, and accumulate in the host over time (Applegate & Beaudoin 1970; Weatherhead & Bennett 1991; Dale *et al.* 1996; Sol *et al.* 2000; Waldenstrom *et al.* 2002).

A negative relationship between parasite prevalence and developmental parameters was previously recorded by Ricklefs (1992) and Tella *et al.* (1999). Ricklefs' analysis was based on average prevalence within families of both passerine and non-passerine birds and included tropical families with longer incubation periods than those observed within temperate and boreal passerines. The negative incubation period–prevalence relationship was not apparent within the temperate sample alone. Likewise, our analysis did not include raptorial birds and so it is not directly comparable with that of Tella *et al.* (1999). Both authors argue that longer incubation periods promote higher immunocompetence through the development of greater potential antibody diversity. We suggest that immunocompetence should be under increased selection in host species that are exposed to more virulent parasites. Consequently, host species with higher exposure to *Plasmodium*, a parasite that evades clearance and can enter chronic stages in the host, should develop higher immunocompetence, which might involve extended developmental periods.

5. CONCLUSIONS

We found that the prevalence of haematozoa in European passerines can be explained in part by male plumage brightness and body mass. Male plumage brightness remained a robust predictor of prevalence in a phylogenetically controlled comparison. This relationship was predominantly driven by prevalences of simuliid-transmitted parasites, *Leucocytozoon* and *Trypanosoma*, and was virtually non-existent in ceratopogonid- and culicid-transmitted parasites, *Haemoproteus* and *Plasmodium*. Thus, these correlations implicate exposure to infection rather than species-specific traits, such as immune function, in determining most of the pattern in variation in prevalence of haematozoa. We found little evidence for effects of life-history traits or ecological attributes of host species. Most of the variation in prevalence resides at the taxonomic level of species within genera, or even lower, as indicated by the high species-specific variance in prevalence. We suggest that rapid coevolution between parasite infection and host resistance might create temporally and spatially labile patterns of variation in parasite prevalence, which may or may not bear some relationship to evolutionarily conservative host life-history traits, such as body size and development time. Consequently, sexually selected characters and other evolutionarily dynamic traits are more likely to exhibit strong correlations with haematozoan prevalence.

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