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# Genetic variation and prevalence of blood parasites do not correlate among bird species

Robert Poulin*, Leigh J. Marshall and Hamish G. Spencer

Department of Zoology, University of Otago, P.O. Box 56, Dunedin, New Zealand

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**Abstract**

Models of host–parasite co-evolution suggest that parasites can exert frequency-dependent selection on their hosts, favouring rare alleles that confer resistance against widespread parasites and thus contributing to the maintenance of genetic variation, at some loci at least. If parasites are important in maintaining variation at many loci, then host species incurring a high prevalence of parasite infections should exhibit greater levels of genetic variation than host species incurring a lower prevalence. Using data from electrophoretic studies and from field surveys of haematozoan infections, we constructed a dataset including 103 species of North American and European birds to test this prediction. After controlling for sampling effort and phylogenetic influences, we found no relationship between parasite prevalence and either heterozygosity or polymorphism. These results do not support a role for parasites in the overall maintenance of genetic variation via frequency-dependent selection.

**Key words**: comparative analysis, frequency-dependent selection, genetic variation, haematozoa, heterozygosity

## INTRODUCTION

The ubiquity of genetic variability in natural populations of plants and animals (reviewed in Nevo, 1978; Nei & Graur, 1984; Evans, 1987) has been described by Lewontin (1974) as 'the paradox of variation'. To date, we have no good theoretical explanation for how this variation is maintained (Lewontin, 1974; Kimura, 1983; Nei, 1987; Maynard Smith, 1989; Spencer & Marks, 1993; Kreitman & Akashi, 1995). Neither the (strictly) neutral theory nor balancing selection adequately account for the patterns of genetic variation observed, either at the allozyme or DNA level (Kreitman & Akashi, 1995). One possibility is that frequency-dependent selection favouring rare alleles and selecting against common ones actively preserves genetic variation (Clarke & Partridge, 1988). This type of selection pressure could be exerted by parasites, since rare genotypes of the host are likely to confer greater resistance against widespread parasites (Hamilton, 1982). Hence, the ubiquity of parasites should provide an omnipresent force favouring the maintenance of genetic variation. The co-evolutionary arms race between a host and a parasite results in any genetically-determined mechanism of resistance rapidly becoming obsolete and inefficient, which in turns leads to pressure on hosts to continually change gene combinations. Indeed, sexual reproduction itself is viewed as an adaptation against parasites by allowing gene recombination and promoting genetic variation (Hamilton, 1980; Lively, 1987; Hamilton, Axelrod & Tanese, 1990; but see Dybdahl & Lively, 1995; Ronsheim, 1996).

Although frequency-dependent selection should maintain high levels of genetic variation indefinitely, firm empirical evidence for this phenomenon is scanty (Chaboudez & Burdon, 1995; Clay & Kover, 1996; Apanius *et al.*, 1997). It seems that parasitic protozoans and other micro-organisms can maintain polymorphism at particular loci in humans (see review in Hamilton *et al.*, 1990), and that parasitic nematodes maintain variation at the major histocompatibility complex in sheep (Paterson, Wilson & Pemberton, 1998). But our question is whether parasites maintain overall genetic variation. Within species, heterozygous fish harbour fewer parasites than homozygous conspecifics (Lively, Craddock & Vrijenhoek, 1990), and bumblebee colonies that are genetically heterogeneous experience lower levels of parasitism than homogeneous colonies (Liersch & Schmid-Hempel, 1998). Thus, parasites appear to respond to overall levels of genetic variation and exert strong selective pressures on their hosts. We set out to test one further prediction of this hypothesis.

*All correspondence to: Robert Poulin.
E-mail: robert.poulin@stonebow.otago.ac.nz

If parasites are important in maintaining overall levels of genetic variation (rather than just variation at specific loci known to be involved in parasite resistance), a simple and testable prediction is that host species exhibiting higher levels of genetic variation do so in response to higher prevalences of parasite infections. In other words, the greater the parasite-mediated selection pressure, the greater the resultant level of genetic variation. Thus, whereas genetically more variable individuals should incur lower levels of parasitism, genetically variable species should incur higher levels. These opposite expected correlations parallel the within-species and among-species predictions of Hamilton & Zuk's (1982) hypothesis of parasite-mediated sexual selection. They argued that although heavily parasitized individuals should display a duller sexual coloration than their uninfected conspecifics, species incurring high levels of parasitism are under more intense selection pressure to evolve and maintain bright colours as sexual signals of mate quality than species incurring lower levels of parasitism.

Here we test this prediction using data gathered from protein electrophoretic studies on birds and from field surveys of prevalence of avian blood parasites. The pathogenic effects of protozoans parasitic in avian blood, or haematozoans, have been well documented (reviewed in Atkinson & van Riper, 1991; John, 1997). Across bird species, haematozoan prevalence even correlates positively with annual mortality rates (Sorci & Møller, 1997). The selective pressure that haematozoans exert on bird hosts is illustrated by their influence on the evolution of sexually selected characters, mating systems and life-history traits (Hamilton & Zuk, 1982; Read, 1991; Ricklefs, 1992; John, 1995). These influences, combined with the short generation times of protozoans relative to their avian hosts, should make them good agents of frequency-dependent selection promoting high levels of genetic variation.

Our specific objectives were to relate prevalence of haematozoan infections to genetic variation among species of North American and European birds. We investigated whether bird species incurring a higher prevalence of infection by haematozoans (and which are presumably subject to stronger frequency-dependent selection pressures) exhibit greater levels of allozyme variation than related bird species incurring lower prevalence (and subject to weaker pressures). Our analysis controls for sampling effort and phylogenetic influences, two potentially confounding factors. We used two measures of genetic variation, heterozygosity (the average proportion of heterozygous loci per individual) and polymorphism (the proportion of polymorphic loci among the total number of loci surveyed). Note that we specifically did not set out to examine loci known to be involved in resistance to blood parasites; our question is about how we can explain overall levels of genetic variation. Even if the loci commonly tested in electrophoretic studies do not provide a precise measure of genetic variation in the entire gene pool of a species, they are still useful for comparative purposes. For instance, Petrie, Doums & Møller (1998) recently showed that variation at the same allozyme loci we used correlates with the degree of extra-pair paternity among bird species. The allozymic variation we investigated may only be the tip of the iceberg with respect to overall genetic variation, but it is its best documented component and one used previously in comparative analyses. To our knowledge, our study is the first attempt to link parasitism with genetic variation across a large number of bird species.

## METHODS

Data on heterozygosity, $H$, and polymorphism, $P$, were obtained from published electrophoretic studies, many of which were found through existing literature surveys (Mani, 1983; Barrowclough, Johnson & Zink 1985; Baker & Strauch, 1986; Evans, 1987). We used 2 estimates of polymorphism, based on 2 different criteria for polymorphism: *P99*, for which the frequency of the most common allele had to be $\leq 0.99$, and *P95*, for which the frequency of the most common allele had to be $\leq 0.95$. Because of the high variance in single-locus polymorphism among loci, estimates of genetic variation are far more severely influenced by the number of loci sampled than by the number of individuals examined (Nei & Roychoudhury, 1974; Nei, 1978; Gorman & Renzi, 1979). Thus, following Nei's (1978) suggestions, estimates were only used if at least 20 loci had been investigated from at least 5 individual birds, or if at least 2 individuals had been used and scored at at least 25 loci. When 2 or more studies per bird species were available, we averaged estimates of heterozygosity and polymorphism, weighting by the number of loci, to obtain single values of $H$, *P95* and *P99* per species.

Parasite data were obtained from surveys of field studies (Greiner *et al*., 1975; Peirce, 1981). Prevalence was calculated as the percentage of individuals for which examination of blood smears revealed infections by 1 or more haematozoan taxa. Since estimates of prevalence based on small numbers of sampled individuals are imprecise (Gregory & Blackburn, 1991), bird species with $< 10$ sampled individuals were discarded. Overall prevalence was used since this value tends to correlate well with prevalence of the main haematozoan genera: *Leucocytozoon* ($r = 0.854$, $n = 76$, $P = 0.001$), *Haemoproteus* ($r = 0.647$, $n = 76$, $P = 0.001$), *Plasmodium* ($r = 0.104$, $n = 61$, $P = 0.404$), and *Trypanosoma* ($r = 0.446$, $n = 64$, $P = 0.002$). In all bird species, both the parasite and the genetic data included in the analysis came from the same geographical region, either eastern North America, western North America, or Europe. Finally, data on body size for each bird species were obtained from Dunning (1993) since this variable may be associated with parasite prevalence and mask any relationship with genetic variation (John, 1995).

We used Felsenstein's (1985) phylogenetically independent contrasts method for the comparative

analysis. Contrasts were computed on log-transformed data using the CAIC 2.0 statistical package (Purvis & Rambaut, 1994). The method consists of deriving statistically independent contrasts between sister taxa from a phylogeny, and using these contrasts to test for relationships between variables. We used the avian phylogeny of Sibley & Ahlquist (1991), supplemented with some additional taxonomic or phylogenetic information (American Ornithologists' Union, 1983; Martin & Clobert, 1996; Westneat & Sherman, 1997), to obtain contrasts. Contrasts need to be standardized for the time since divergence of sister taxa, or branch lengths in the phylogeny (Garland, Harvey & Ives, 1992). We used Sibley & Ahlquist's (1991) $\Delta T_{50}H$ measures of divergence, based on DNA–DNA hybridization, as estimates of branch lengths to standardize contrasts. Relationships among contrasts were assessed using correlations forced through the origin (Garland *et al.*, 1992). To control for the confounding effect of sampling effort (either number of individuals or number of loci examined), residuals of a regression of the variable of interest on the confounding variable were used instead of uncorrected contrasts (Garland *et al.*, 1992). This approach, using the same parasite data, avian phylogeny and comparative method, has been used previously to investigate the relationship between blood parasites and several ecological characteristics of birds (e.g. Read, 1991; John, 1995; Sorci & Møller, 1997).

## RESULTS

Overall, the data set included 103 bird species, from 56 genera and 20 families (Table 1). There is much variation among bird families in levels of parasitism and genetic variation, suggesting that these data are subject to some sort of phylogenetic constraints and that using phylogenetically independent contrasts is necessary.

Bird body mass did not correlate with any of the variables investigated and was thus ignored in further analyses. The number of individual birds examined for parasites correlated positively with the number of individuals examined to determine genetic variation ($r = 0.341$, $n = 64$ sets of contrasts, $P < 0.01$). However, the number of birds examined for parasites did not correlate with haematozoan prevalence ($r = 0.124$, $n = 64$ sets of contrasts, $P = 0.327$), and there was no need to correct prevalence for sampling effort. All three measures of genetic variation correlated positively with one another (*H* vs. *P99*: $r = 0.686$, $n = 52$ sets of contrasts, $P < 0.001$; *H* vs. *P95*: $r = 0.745$, $n = 58$, $P < 0.001$; *P99* vs. *P95*: $r = 0.874$, $n = 52$, $P < 0.001$), but were still analysed separately. Heterozygosity correlated positively with the number of loci investigated ($r = 0.391$, $n = 64$ sets of contrasts, $P < 0.01$), whereas both measures of polymorphism correlated positively with the number of individual birds examined (*P99*: $r = 0.447$, $n = 52$ sets of contrasts, $P < 0.001$; *P95*: $r = 0.239$, $n = 58$, $P < 0.10$), suggesting that correcting for sampling effort may be necessary.

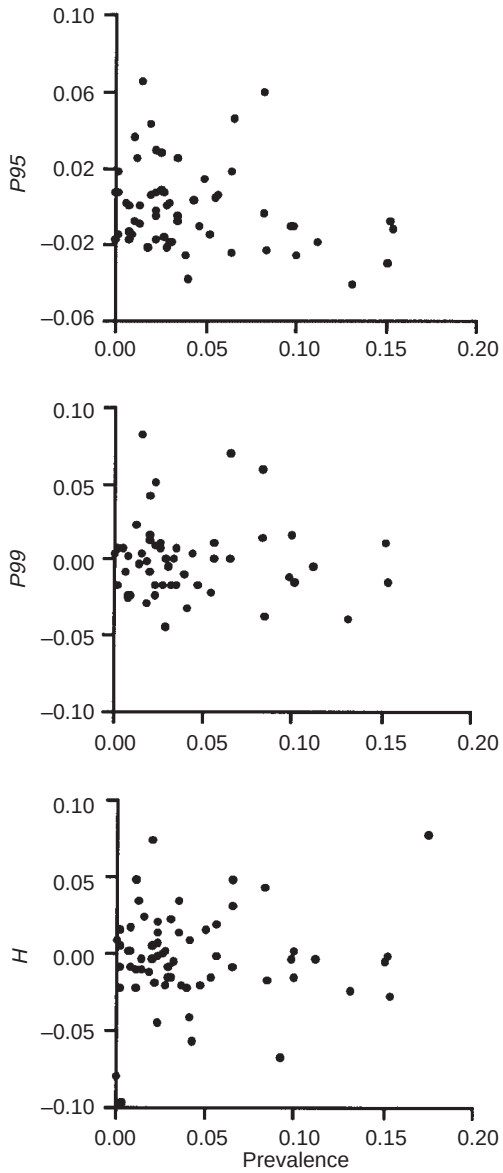


**Fig. 1**. Relationship between prevalence of avian blood parasites and three measures of genetic variation in birds, computed using phylogenetically independent contrasts standardized for branch lengths. Heterozygosity (*H*) is corrected for the number of loci investigated, whereas *P99* and *P95* are corrected for the number of individuals examined. Correlations are forced through the origin; all are statistically non-significant (*H*: $r = -0.055$, $n = 64$ sets of contrasts, $P = 0.688$; *P99*: $r = -0.045$, $n = 52$, $P = 0.765$; *P95*: $r = -0.206$, $n = 58$, $P = 0.118$).

Haematozoan prevalence did not correlate significantly with either of the three measures of genetic variation (even when each parasite genus was analysed separately), independently of whether or not these measures were corrected for sampling effort using residuals (Fig. 1). In other words, avian taxa incurring higher prevalence of haematozoans than their sister taxa do not also display consistently higher levels of genetic variation.

**Table 1**. Haematozoan prevalence data and genetic variation data for the 103 bird species included in the analysis

| Family<br>species | Prevalence, %<br>(no. birds examined) | No. loci<br>(no. birds examined) | $H^a$ | $P99^a$ | $P95^a$ | Sources[b] |
|---|---|---|---|---|---|---|
| Alaudidae | | | | | | |
| *Eremophila alpestris* | 13.0 (23) | 35 (17) | 0.102 | – | – | 1 |
| Alcidae | | | | | | |
| *Fratercula artica* | 0.0 (207) | 32 (180) | 0.043 | 0.137 | 0.119 | 2 |
| Anatidae | | | | | | |
| *Anas americana* | 20.1 (279) | 25 (1247) | 0.082 | 0.560 | 0.280 | 3,4 |
| *Anas platyrynchos* | 27.0 (1901) | 30 (752) | 0.073 | 0.218 | 0.176 | 5–7 |
| *Anas rubripes* | 54.4 (2658) | 29 (131) | 0.053 | 0.260 | 0.183 | 5 |
| *Branta canadensis* | 6.4 (1148) | 35 (249) | 0.051 | 0.322 | 0.210 | 8 |
| *Chen caerulescens* | 3.5 (577) | 41 (550) | 0.029 | 0.146 | 0.146 | 9 |
| Ardeidae | | | | | | |
| *Ardea herodias* | 35.1 (37) | 28 (46) | 0.007 | 0.107 | 0.036 | 10 |
| Charadriidae | | | | | | |
| *Charadrius vociferus* | 0.0 (61) | 40 (12) | 0.004 | – | 0.025 | 11 |
| Emberizidae | | | | | | |
| *Agelaius phoeniceus* | 14.9 (1486) | 28 (292) | 0.037 | 0.276 | 0.124 | 12 |
| *Ammodramus savannarum* | 3.0 (33) | 20 (10) | 0.045 | 0.200 | 0.200 | 13 |
| *Dendroica caerulescens* | 17.6 (17) | 26 (6) | 0.040 | – | – | 14 |
| *Dendroica castanea* | 27.7 (47) | 26 (12) | 0.024 | – | – | 14 |
| *Dendroica coronata* | 54.6 (423) | 32 (140) | 0.035 | 0.333 | 0.222 | 15,16 |
| *Dendroica discolor* | 3.2 (31) | 26 (8) | 0.012 | – | – | 14 |
| *Dendroica fusca* | 43.8 (32) | 26 (8) | 0.036 | – | – | 14 |
| *Dendroica magnolia* | 44.2 (147) | 26 (12) | 0.004 | – | – | 14 |
| *Dendroica palmarum* | 20.0 (15) | 29 (45) | 0.040 | 0.323 | 0.323 | 16 |
| *Dendroica pensylvanica* | 49.4 (83) | 26 (10) | 0.014 | – | – | 14 |
| *Dendroica petechia* | 52.0 (296) | 26 (2) | 0.068 | – | – | 14 |
| *Dendroica pinus* | 23.0 (135) | 26 (2) | 0.050 | – | – | 14 |
| *Dendroica tigrina* | 56.1 (41) | 26 (6) | 0.033 | – | – | 14 |
| *Dendroica virens* | 27.0 (63) | 26 (2) | 0.024 | – | – | 14 |
| *Geothlypsis trichas* | 25.5 (196) | 26 (32) | 0.037 | – | – | 14 |
| *Icteria virens* | 80.0 (10) | 26 (5) | 0.020 | – | – | 14 |
| *Junco hyemalis* | 28.3 (360) | 34 (112) | 0.038 | 0.165 | 0.136 | 13,17,18 |
| *Melospiza georgiana* | 60.9 (304) | 30 (26) | 0.045 | 0.217 | 0.183 | 13,18 |
| *Melospiza lincolnii* | 59.0 (78) | 39 (8) | 0.054 | 0.179 | 0.179 | 18 |
| *Melospiza melodia* | 34.9 (1518) | 39 (14) | 0.042 | 0.179 | 0.154 | 18 |
| *Mniotilta varia* | 35.0 (123) | 26 (6) | 0.026 | – | – | 14 |
| *Parula americana* | 33.3 (57) | 26 (6) | 0.030 | – | – | 14 |
| *Passerculus sandwichensis* | 30.6 (173) | 20 (10) | 0.049 | 0.250 | 0.250 | 13 |
| *Passerella iliaca* | 76.7 (477) | 39 (57) | 0.036 | 0.308 | 0.128 | 18 |
| *Pipilo erythrophthalmus* | 33.1 (169) | 39 (17) | 0.146 | – | – | 19 |
| *Pipilo fuscus* | 34.6 (78) | 21 (32) | 0.033 | 0.110 | 0.110 | 13,20 |
| *Seiurus aurocapillus* | 23.6 (127) | 26 (29) | 0.061 | – | – | 16 |
| *Seiurus noveboracensis* | 70.0 (423) | 29 (31) | 0.088 | 0.452 | 0.419 | 16 |
| *Setophaga ruticilla* | 29.0 (283) | 26 (16) | 0.031 | – | – | 14 |
| *Spizella passerina* | 34.2 (465) | 21 (11) | 0.065 | 0.143 | 0.143 | 13 |
| *Spizella pusilla* | 21.9 (151) | 21 (6) | 0.083 | 0.143 | 0.143 | 13 |
| *Vermivora celata* | 18.8 (32) | 26 (12) | 0.040 | – | – | 14 |
| *Vermivora peregrina* | 26.5 (253) | 28 (37) | 0.055 | 0.333 | 0.333 | 14,16 |
| *Zonotrichia albicollis* | 55.0 (1075) | 39 (22) | 0.048 | 0.267 | 0.200 | 13,18 |
| *Zonotrichia atricapilla* | 27.8 (212) | 39 (15) | 0.039 | 0.154 | 0.154 | 18 |
| *Zonotrichia leucophrys* | 47.1 (425) | 46 (352) | 0.055 | 0.331 | 0.235 | 17,18,21–23 |
| *Zonotrichia querula* | 21.1 (19) | 39 (32) | 0.023 | 0.123 | 0.111 | 17,18 |
| Fringillidae | | | | | | |
| *Carduelis flammea* | 66.7 (21) | 33 (5) | 0.021 | 0.034 | 0.034 | 24 |
| *Carduelis pinus* | 40.5 (37) | 33 (6) | 0.035 | 0.138 | 0.138 | 24 |
| *Carduelis psaltria* | 15.4 (13) | 33 (4) | 0.017 | 0.034 | 0.034 | 24 |
| *Carduelis tristis* | 24.7 (77) | 33 (7) | 0.016 | 0.034 | 0.034 | 24 |
| *Carpodacus cassinii* | 72.7 (11) | 33 (12) | 0.026 | 0.212 | 0.138 | 24 |
| *Carpodacus mexicanus* | 14.8 (714) | 33 (19) | 0.040 | 0.333 | 0.207 | 24 |
| *Carpodacus purpureus* | 67.7 (226) | 33 (15) | 0.034 | 0.138 | 0.138 | 24 |
| *Coccothraustes vespertinus* | 56.8 (37) | 33 (4) | 0.044 | 0.069 | 0.069 | 24 |
| *Loxia leucoptera* | 53.8 (13) | 33 (3) | 0.103 | 0.206 | 0.206 | 24 |
| *Pinicola enucleator* | 88.7 (62) | 33 (7) | 0.052 | 0.155 | 0.155 | 24 |
| Laridae | | | | | | |
| *Larus ridibundus* | 4.5 (22) | 29 (15) | 0.041 | 0.207 | 0.172 | 25 |
| *Sterna hirundo* | 0.0 (587) | 34 (82) | 0.044 | 0.199 | 0.147 | 26 |
| Mimidae | | | | | | |
| *Dumetella carolinensis* | 20.0 (235) | 24 (32) | 0.026 | 0.213 | 0.128 | 27,28 |
| *Mimus polyglottos* | 29.2 (212) | 23 (8) | 0.010 | 0.053 | 0.053 | 28 |
| *Toxostoma rufum* | 45.4 (401) | 23 (7) | 0.048 | 0.130 | 0.130 | 28 |

**Table 1**. (*cont*.)

| Family species | Prevalence, % (no. birds examined) | No. loci (no. birds examined) | $H^a$ | *P99*[a] | *P95*[a] | Sources[b] |
|---|---|---|---|---|---|---|
| Muscicapidae | | | | | | |
| *Catharus fuscescens* | 41.8 (91) | 27 (5) | 0.056 | 0.185 | 0.185 | 27 |
| *Catharus guttatus* | 40.9 (88) | 27 (13) | 0.047 | 0.259 | 0.148 | 27 |
| *Catharus minimus* | 76.2 (193) | 26 (2) | 0.021 | 0.077 | 0.077 | 27 |
| *Catharus ustulatus* | 58.7 (446) | 27 (22) | 0.048 | 0.300 | 0.148 | 14,27 |
| *Hylocichla mustelina* | 32.1 (28) | 27 (5) | 0.104 | 0.148 | 0.148 | 27 |
| *Sialia sialis* | 16.1 (56) | 27 (7) | 0.063 | 0.222 | 0.222 | 27 |
| *Turdus migratorius* | 73.5 (1323) | 26 (5) | 0.042 | 0.077 | 0.077 | 27 |
| Odontophoridae | | | | | | |
| *Callipepla californica* | 23.6 (89) | 32 (134) | 0.030 | 0.169 | 0.123 | 29,30 |
| *Callipepla gambelii* | 89.7 (907) | 27 (22) | 0.025 | 0.185 | 0.111 | 29 |
| *Callipepla squamata* | 23.1 (134) | 27 (29) | 0.032 | 0.130 | 0.130 | 29 |
| *Colinus virginianus* | 1.8 (947) | 27 (15) | 0.027 | 0.148 | 0.148 | 29 |
| *Oreortyx pictus* | 31.8 (22) | 27 (16) | 0.021 | 0.111 | 0.074 | 29 |
| Paridae | | | | | | |
| *Parus atricapillus* | 54.4 (228) | 35 (20) | 0.046 | 0.229 | 0.114 | 31 |
| *Parus bicolor* | 0.0 (14) | 36 (24) | 0.060 | 0.306 | 0.181 | 32 |
| *Parus carolinensis* | 6.8 (44) | 35 (20) | 0.042 | 0.314 | 0.200 | 31 |
| *Parus gambeli* | 35.7 (14) | 33 (15) | 0.020 | 0.152 | 0.121 | 31 |
| Passeridae | | | | | | |
| *Passer domesticus* | 18.2 (5202) | 33 (581) | 0.100 | 0.478 | 0.296 | 33–35 |
| *Passer montanus* | 29.3 (280) | 39 (93) | 0.078 | 0.445 | 0.220 | 36 |
| Phasianidae | | | | | | |
| *Alectoris chukar* | 0.0 (24) | 33 (20) | 0.085 | 0.394 | 0.333 | 37 |
| *Lagopus lagopus* | 28.4 (222) | 23 (269) | 0.082 | 0.260 | 0.260 | 38 |
| *Phasianus colchicus* | 0.0 (441) | 27 (98) | 0.056 | 0.312 | 0.226 | 29,39 |
| *Tympanuchus phasianellus* | 82.8 (134) | 30 (22) | 0.046 | 0.200 | 0.133 | 40 |
| Picidae | | | | | | |
| *Colaptes auratus* | 52.0 (177) | 31 (356) | 0.075 | 0.299 | 0.259 | 41,42 |
| *Sphyrapicus varius* | 33.3 (39) | 39 (7) | 0.022 | 0.128 | 0.103 | 43 |
| Regulidae | | | | | | |
| *Regulus calendula* | 7.8 (77) | 23 (10) | 0.048 | 0.174 | 0.174 | 27 |
| Scolopacidae | | | | | | |
| *Calidris alpina* | 0.0 (18) | 29 (25) | 0.009 | – | 0.138 | 11 |
| *Calidris fuscicolis* | 0.0 (13) | 40 (56) | 0.023 | – | 0.205 | 11 |
| *Calidris maritima* | 0.0 (13) | 40 (35) | 0.006 | – | 0.069 | 11 |
| *Calidris minutilla* | 0.0 (43) | 39 (25) | 0.030 | – | 0.205 | 11 |
| *Calidris pusilla* | 0.0 (67) | 40 (25) | 0.038 | – | 0.175 | 11 |
| *Limnodromus griseus* | 30.0 (10) | 39 (28) | 0.018 | 0.054 | 0.062 | 11,44 |
| Sturnidae | | | | | | |
| *Sturnus vulgaris* | 2.8 (604) | 24 (298) | 0.031 | – | 0.210 | 45 |
| Tyrannidae | | | | | | |
| *Contopus sordidulus* | 35.7 (14) | 38 (5) | 0.067 | – | 0.231 | 46 |
| *Contopus virens* | 37.0 (27) | 38 (7) | 0.084 | – | 0.308 | 46 |
| *Empidonax difficilis* | 60.0 (10) | 40 (388) | 0.053 | 0.261 | 0.158 | 46,47 |
| *Empidonax flaviventris* | 5.6 (36) | 38 (19) | 0.050 | – | 0.282 | 46 |
| *Empidonax minimus* | 7.7 (13) | 38 (18) | 0.050 | – | 0.154 | 46 |
| *Empidonax traillii* | 12.0 (83) | 37 (49) | 0.072 | 0.321 | 0.268 | 46,48 |
| Vireonidae | | | | | | |
| *Vireo flavifrons* | 30.0 (10) | 23 (4) | 0.043 | 0.174 | 0.174 | 28 |
| *Vireo griseus* | 40.6 (32) | 23 (10) | 0.036 | 0.087 | 0.081 | 28,49 |
| *Vireo olivaceus* | 59.4 (187) | 31 (109) | 0.057 | 0.411 | 0.233 | 14,19,28,49 |
| *Vireo solitarius* | 40.7 (27) | 23 (21) | 0.053 | 0.211 | 0.154 | 28,49 |

[a] $H$, heterozygosity; *P99*, polymorphism with criterion that the frequency of the most common allele is $\leq 0.99$; *P95*, polymorphism with criterion that the frequency of the most common allele is $\leq 0.95$.

[b] Sources of the genetic data: 1, Evans (1987); 2, Moen (1991); 3, Rhodes, Smith & Chesser (1993); 4, Rhodes & Smith (1993); 5, Ankney *et al*. (1986); 6, Rhodes, Smith & Chesser (1995); 7, Rhodes, Smith & Smith (1996); 8, van Wagner & Baker (1986); 9, Cooke, Parkin & Rockwell (1988); 10, Guttman, Grau & Karlin (1980); 11, Baker & Strauch (1986); 12, Gavin, Howard & May (1991); 13, Avise, Patton & Aquadro (1980*c*); 14, Avise, Patton & Aquadro (1980*a*); 15, Barrowclough (1980); 16, Barrowclough & Corbin (1978); 17, Zink & Watt (1987); 18, Zink (1982); 19, Barrowclough *et al*. (1985); 20, Zink (1988); 21, Corbin & Wilkie (1988); 22, Baker (1976); 23, Corbin (1981); 24, Marten & Johnson (1986); 25, Randi & Spina (1987); 26, Burson (1990); 27, Avise, Patton & Aquadro (1980*b*); 28, Avise, Aquadro & Patton (1982); 29, Gutierrez, Zink & Yang (1983); 30, Zink, Lott & Anderson (1987); 31, Braun & Robbins (1986); 32, Braun, Kitto & Braun (1984); 33, Stangel, Rodgers & Bryan (1990); 34, Bates & Zink (1992); 35, Parkin & Cole (1984); 36, St Louis & Barlow (1988); 37, Randi *et al*. (1992); 38, Gyllensten, Reuterall & Ryman (1979); 39, Scribner, Dowell & Warren (1989); 40, Ellsworth *et al*. (1994); 41, Grudzien *et al*. (1987); 42, Grudzien & Moore (1986); 43, Johnson & Zink (1983); 44, Avise & Zink (1988); 45, Ross (1983); 46, Zink & Johnson (1984); 47, Johnson & Marten (1988); 48, Seutin & Simon (1988); 49, Johnson, Zink & Marten (1988).

## DISCUSSION

If the short generation times and high evolutionary rates of parasites select for rare alleles and maintain variation among hosts, then host species exposed to higher prevalence of parasites should also display substantial genetic variation. Here we examined whether parasitism is associated with genome-wide diversity across host species. We failed to find any correlation between prevalence of blood parasites and levels of genetic variation among a large number of bird species. The same result was obtained both after removing the confounding effects of avian phylogeny and sampling effort, and when we used raw species values as independent observations. There are three possible explanations for this outcome: poor quality data, parasite prevalence being an inadequate measure of selection pressure, and electrophoretic loci being unaffected by parasite-mediated selection.

A common explanation for a failure to find a significant relationship where one is expected is that the data are of poor quality and/or contain too much noise. We tried to eliminate species for which the accuracy of the data was questionable. However, neither relaxing the criteria for inclusion of species in the data set, nor using tougher criteria, had any effect on the outcome of the analysis. Our survey found that levels of genetic variation differ slightly between populations of the same bird species. Examining different loci or even the same loci under different laboratory conditions can produce different estimates of genetic variation. It is noteworthy, however, that similar data have been used previously to uncover correlations between life-history characters and levels of genetic variation in fish and birds (Mitton & Lewis, 1989; Petrie *et al*., 1998). Data on haematozoan prevalence also often show spatial and temporal variation within host species, and this variability could perhaps mask existing relationships (Yezerinac & Weatherhead, 1995; John, 1997). Nevertheless, the same parasite prevalence data used here have been shown in other studies to reveal significant patterns between parasitism and bird ecology even when fewer bird species were included (Read, 1991; Ricklefs, 1992; John, 1995; Sorci & Møller, 1997). The power of our tests to detect statistically significant non-zero correlations was also high. For instance, if the true correlation was 0.4 (explaining just 16% of the observed data scatter), even our smallest data set ($n = 52$ contrasts) had a $>84\%$ chance of giving a statistically significant ($P < 0.05$) result. If the true correlation was 0.5, our power rises to *c*. 97%. Hence, we do not think that the quality or noisiness of either data set has masked any real relationship.

A second reason for the apparent absence of relationships between parasitism and genetic variation may be that variation within particular parasite species in infective ability, and hence selection pressure, is of little consequence to host resistance. There is consequently no evolutionary arms race and so overall parasite prevalence need not correlate with genetic variation. Given the results of within-species comparisons (e.g. Lively *et al*., 1990; Liersch & Schmid-Hempel, 1998; Paterson *et al*., 1998), however, we think this explanation is unlikely. Nevertheless, richness of parasite species might still correlate positively with genetic variation since it would reflect a more varied source of selection pressure. Data on richness of parasite species in birds, unfortunately, are not as readily available.

Finally, the data on heterozygosity and polymorphism have been obtained for loci that are not known to be directly involved in resistance against parasites. The problem with this explanation is that it leaves unanswered the very question we set out to investigate: why are such loci variable? Moreover, this explanation implies that frequency-dependent selection by parasites only affects some fraction of loci. If parasites are responsible for the evolution of such a fundamental phenomenon as sex, one might have thought their effect would be more pervasive.

Although not significant, all correlation coefficients between parasite prevalence and genetic variation are negative (Fig. 1), suggesting a weak tendency for avian taxa with high parasite prevalence to be less variable than their sister taxa with lower prevalence. If another, unidentified factor is maintaining genetic variation in birds (e.g. frequency-dependent selection mediated by viral diseases), we might expect that the more variable species are more likely than less variable species to possess the rare alleles conferring resistance against widespread parasites. This idea, only very weakly supported by our analysis, would relegate parasites from agents promoting genetic variation to incidental victims of the variation maintained by other processes.

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