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## Sex-biased parasitism of avian hosts: relations to blood parasite taxon and mating system

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Immunosuppressive effects of testosterone lead to a prediction of male-biased parasitism. To test this prediction, prevalences of blood parasites were compared between male and female birds using statistically correct vote counts of data from 33 studies. We found no overall difference in prevalence between males and females, in either breeding or non-breeding birds. However, infections by *Haemoproteus* (the most common genus of blood parasite found) were significantly more common among breeding females than breeding males. Restricting the analysis to breeding birds of polygynous species, females again were more likely than males to be infected by blood parasites; this result held for an intra-family comparison that controlled for phylogenetic effects. In comparison, measures of sexual size dimorphism did not relate to sex biases in parasitism as predicted, after controlling for phylogeny using independent comparisons. Because testosterone is often implicated in suppressing the immune system, female biases in parasitism are unexpected. Female biases in parasitism by blood parasites could result from differential exposure of the sexes to vectors, or from oestrogen-based effects on immunity.

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Host-parasite relationships are attracting increasing attention due to recent reports of unexpected ways that parasites may intervene in host life cycles. For example, parasites can alter host sex during development (Hurst et al. 1993), and may influence evolution of sexually selected traits (Hamilton and Zuk 1982). More intuitive aspects of host-parasite relationships have long been recognised; parasitism can be influenced by host age, genetic similarity, and environmental conditions (Hauschka 1947, Atkinson and van Riper 1991, Desser and Bennett 1993). However, effects of host sex on parasitism have just recently attracted attention (Goble and Konopka 1973, Alexander and Stimson 1988, Bundy 1988), in part because evidence has been accumulating which indicates that some sex hormones

(particularly androgens) can be immunosuppressive (Grossman 1985, Schuurs and Verheul 1990). Herein, we test for differences in prevalence of blood parasites between male and female birds.

Relations between hormones and immunity have provided an impetus for investigations into sex-biased parasitism. Androgens, primarily testosterone, can suppress cell-mediated (T cell-regulated) and humoral (B cell- and antibody-regulated) immunity in males (Schuurs and Verheul 1990). In contrast, oestrogen can suppress cell-mediated immunity, while at the same time boosting humoral immunity (Grossman 1985). In birds, for example, weight of the thymus (where T cells mature) increases following removal of the testes, whereas removal of the ovaries has no such effect (Alexander

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and Stimson 1988). Testosterone, in concentrations greater than normal physiological levels, also appears to negatively affect humoral immunity by decreasing the size of the Bursa of Fabricius (an avian organ in which immunocompetent B cells are stored; Norton and Wira 1977) and by inhibiting production of some antibodies (Hirota et al. 1980). Both cell-mediated and humoral immunity are implicated in fighting parasites (Seed and Manwell 1977, Grossman 1985, Schuurs and Verheul 1990).

A second important cause of sex-biased parasitism may be sex differences in exposure to parasites (reviewed in Zuk and McKean 1996). For example, differential exposure could arise from unequal time spent at the nest (e.g., Chernin 1952b, Peirce and Marquiss 1983, Korpimäki et al. 1993, Norris et al. 1994). Hormones also may influence behaviours that relate to exposure, independent of any effects on immune systems. Thus, links between hormones and sex-biased parasitism could arise via many different mechanisms.

A third possible cause of sex-biased parasitism is variation in stress levels imposed by mating systems (Zuk 1990, Zuk and McKean 1996). Zuk and McKean (1996) hypothesised that species with increasing degrees of polygyny would have greater sex-biased parasitism due to greater relative differences in immunocompetence of males and females. Specifically, Zuk (1990) and Hillgarth et al. (1997) suggested that greater male-biased parasitism would occur in polygynous species because males would be under more physiological stress (due to investment in sexually selected morphological traits and displays). We are not aware of any studies on birds that compare sex biases in parasitism among mating systems. However, Read (1991) found that prevalence of avian blood parasites was generally lower in polygynous than in monogamous sister taxa; he attributed this result to either sexual selection for resistant males, or to lower exposure to parasites in polygynous species. In either case, mating system may influence sex biases in parasitism.

A fourth potential cause of sex biases in parasitism is sexual size dimorphism (which is not independent of the former influences, such as mating system). Greater sexual size dimorphism is associated with higher mortality of the larger sex (Teather and Weatherhead 1989, Promislow et al. 1992), although the mechanisms are not known. If mechanisms causing mortality are related to immunocompetence, species with greater sexual size dimorphism also may have more pronounced sex-biased parasitism. Alternatively, larger bodies may provide more surface area or attractants to parasite vectors, or may provide more niches to parasites. Thus, dimorphism should be considered in tests for sex biases in parasitism.

As we have indicated, potential influences on sex-biased parasitism are interrelated. In addition, recent evidence suggests that there are tradeoffs among invest-

ment in immunity, hormones, and other physiological and ecological aspects of reproduction (Festa-Bianchet 1989, Folstad and Karter 1992, Forbes 1993, Ots and Hōrak 1996). Assuming that there are costs to maintaining active immunity (Behnke et al. 1992, Sheldon and Verhulst 1996, Yan et al. 1997), immune responses should be optimised, not maximised (Forbes 1993, John 1995). Because of sex-specific life histories, these tradeoffs may act differently on each sex, resulting in sex biases in parasitism (Forbes 1996, Sheldon and Verhulst 1996). Thus far, male-biased parasitism in birds and mammals by nematodes (Poulin 1996), and in mammals by arthropods and protozoans (Schalk and Forbes 1997) has been reported. Clearly, further testing is needed to confirm the generality and direction of any pattern(s).

There are several advantages to using blood parasites of birds to test for sex biases in parasitism. First, the avian immune system has been well studied, and there is information on life-history tradeoffs between reproductive effort and parasites in birds (Sheldon and Verhulst 1996). Second, there are published data for birds on mating systems and sexual dimorphism based on body mass, allowing these variables to be considered in analysing sex-biased parasitism. Finally, avian blood parasites often have been used to test hypotheses in ecology and evolution; as such a large number of studies were available for our investigation. These published data allow us to test whether sex differences in hormones or differential exposure might cause sex-biased parasitism, although they do not allow us to distinguish between these explanations (Zuk and McKean 1996). These data do allow us to test directly whether mating system or sexual size dimorphism are important influences on sex-biased parasitism.

## Methods

### Literature search

We collected primary studies (published in English) by searching keywords in the Biological Abstracts CD-ROM database (1990–1995) and by examining the reprint libraries of DS and MRF (1980–1996). We also searched citations of all of those studies. Finally, we requested unpublished data by posting advertisements in either electronic or printed newsletters (Animal Behaviour, Ecology, Evolution, and the Ornithological Newsletter of the Ornithological Societies of North America). Unpublished data should help lessen the “file drawer” problem, i.e., a publication bias in favour of studies reporting significant results (Gurevitch et al. 1992, Begg 1994, Csada et al. 1996). This problem, however, was expected to be minimal because almost all of the primary studies we included did not focus on sex biases in infection by blood parasites.

To be included in our investigation, studies (Appendix A) had to meet several criteria. Authors had to report prevalence of avian blood parasites and sample sizes for both sexes of at least one host species (or provide enough information for us to extract these data). They had to include information that allowed us to determine whether prevalence was assessed during the breeding season (if data were collected during both breeding and non-breeding periods, they were separated where possible or otherwise excluded). In cases where reproductive status was not provided, we consulted field guides to determine whether birds were sampled on breeding grounds (National Geographic Society 1983, Jonsson 1992). Infections should be expected to differ between the sexes most when differences in stresses, hormones, and immune function also are expected, so data on breeding and non-breeding birds were kept separate. Although peak stress occurs at different times for different species, we expected that stress would be high for all species during the breeding season. Hence, we expected high prevalence of parasites during the breeding season, but not necessarily outside the breeding season. Authors also had to state whether data were collected from juvenile or adult birds, in order to omit those that used juveniles only (e.g., Shutler et al. 1996) or that did not separate the two age groups. Studies were included if data were collected from either natural populations or semi-natural/experimental infections (e.g., one study using pen-raised birds; Chernin 1952a; and one where birds were inoculated with parasites; Applegate 1971), but the two data sets were kept separate. In studies where prevalence was considered as a response variable in a controlled experiment (such as in studies using brood manipulation and parasite load; e.g., Norris et al. 1994, Richner et al. 1995), only control groups were included, i.e., those that were observed, but not manipulated. This was done because experimental manipulation of reproduction could disproportionately affect susceptibility to parasitism in one sex.

We did include several studies that used techniques that can lead to poor estimates of prevalence and intensity. For example, we included studies where trypanosome prevalence was assessed using blood smears, despite the fact that this results in underestimates (Apanius 1991). We also included data from hunter-killed birds that can result in underestimates of prevalence (Herman 1968). Our rationale was that any errors should not be biased towards one sex.

We indexed mating system of species using data for European passerines from Møller (1986) and for North American birds using Ehrlich et al. (1988). There are three related considerations in using these classifications. First, references used social behaviours to classify birds as monogamous (each male only mates with one female) or polygynous (males may mate with more than one female in a breeding season). For simplicity, we

classified two lekking species as polygynous. Second, we considered a species polygynous when any level of polygyny was recorded (following Read 1991). However, incidence of polygyny was assessed differently for North American versus European birds, because Ehrlich et al. (1988) only mentioned polygyny if it applied to >15% of males, whereas Møller (1986) considered species polygynous when  $\geq 2\%$  of males mated multiply. Third, these criteria describe social rather than genetic mating systems, and there is increasing evidence that extra-pair copulations occur in species previously thought to be socially monogamous (Birkhead and Møller 1992, Andersson 1994); however, we did not have data to determine genetic mating systems for most of the populations included in this study. Ehrlich et al. (1988) also assumed that birds were monogamous when the mating system was not known, whereas Møller (1986) only listed species as monogamous if this had been at least tentatively confirmed. For these reasons, data were analysed in two ways: first, with mating systems separated by continent and, second, with the "continent effect" ignored. For New World warblers (Parulidae), both monogamy and polygyny occur; hence, sex biases in parasitism could be compared between sister taxa that differed in mating system. Thus, this approach controlled for possible phylogenetic effects (cf. Read 1991), but only for New World warblers.

To estimate sexual size dimorphism, we used male and female adult body masses from Dunning (1994). In cases where Dunning did not report masses for the sexes separately, data from Palmer (1976), Lehikoinen (1987), Bennett et al. (1988), Summers-Smith (1988), Amundsen and Slagsvold (1991), and Barber (unpubl.) were used. An index of sexual-size dimorphism was then calculated for each species by regressing male body mass against that for females and using the residuals from major axis regression (Sokal and Rohlf 1981, Ranta et al. 1994). This method is preferred over methods using body size ratios, as the relationship between male and female size is usually not isometric (Ranta et al. 1994). We then controlled for non-independence of taxa by using Felsenstein's method of pairwise independent comparisons (Felsenstein 1985, Harvey and Pagel 1991). We used Sibley and Ahlquist (1990) to resolve the phylogeny of host species. Passerines in the same genus are not resolved by Sibley and Ahlquist (1990), so in these cases we used Martin and Clobert (1996). Promislow's (1991) method was used to resolve a single case of multifurcation in the genus *Falco*.

### Statistical analysis

We used statistically correct vote counting methods (SCVC, Hedges and Olkin 1985, Poulin 1996, Schalk

and Forbes 1997) to compare prevalence between male and female hosts. Unlike traditional vote counts, this procedure controls for Type II errors, making it especially useful when sample sizes from individual studies are low and/or magnitude of the measured effect is weak (Arnqvist and Wooster 1995). This procedure is particularly important as it has been suggested that sex differences in parasitism often may be missed with small samples (Zuk and McKean 1996). A “true” meta-analysis would require a measure of variability for each comparison; however, prevalence has no comparable measure of variability associated with it. Using intensities of infection would provide a measure of variability, but most papers did not report such data. Moreover, intensities of blood parasites can vary temporally and spatially (Weatherhead and Bennett 1991, Shutler et al. 1996) and are thought to be associated with far more error than is prevalence (Weatherhead and Bennett 1991). For these reasons, we decided against using intensity measures.

Before calculating mean differences in prevalence (i.e. effect sizes), results from each study were separated by location and year, where possible. We then removed all comparisons with samples <10 males or females to avoid the problem of the positive relationship between prevalence and sample size, which is often strong for small sample sizes (Gregory and Blackburn 1991). Remaining comparisons were then combined across years, locations, and studies so that each host-parasite combination represented one datum (resulting prevalence values are hereafter defined as overall values). Overall values were assessed to minimise a potential lack of independence at lower levels, which is a difficulty in meta-analysis and SCVC (Gurevitch et al. 1992). For example, there were 12 comparisons for *Haemoproteus* sp. in pied flycatchers (*Ficedula hypoleuca*) when broken down by study, location, and date, whereas there was only one comparison at this level for studies of most other host species. While analyses could be justified at other levels, combining results to produce a single datum per host species allowed us to make use of many studies (many studies could not be separated by location or year).

To calculate an effect size for each comparison, male prevalence was subtracted from female prevalence, such that negative and positive values indicate greater prevalence in males and females, respectively. Each effect size was then corrected so that comparisons with smaller sample sizes were given less weight than those with larger samples (Poulin 1996). We chose Poulin’s method to calculate effect sizes over a similar procedure used by Schalk and Forbes (1997) because Poulin’s provided a relative value for differences in prevalence; this could be analysed against any continuous variables, such as sexual size dimorphism (Poulin’s study) or residuals from regression (this study).

Once standardised effect sizes were calculated for each comparison, they were combined by averaging them. To determine if overall prevalence between host sexes was random (normally distributed around a mean of zero), two-tailed *t*-tests were used. We also used *t*-tests to compare sex differences in overall prevalence for separate parasite taxa. When comparing sex biases in parasitism in relation to mating system, we used a nested analysis of variance (mating systems nested within continent) to account for different criteria used in assessing mating systems in North American and European studies (see above). In all cases, we used Shapiro-Wilk’s and Levene’s tests to verify that assumptions of parametric tests were met. For relations between sexual size dimorphism and sex differences in prevalence, Spearman’s rank correlations were used separately for breeding and non-breeding birds, as the residuals were not homogeneous.

## Results

Thirty-three studies met our criteria and these provided 35 comparisons for adults of different host species (28 breeding, 5 non-breeding, 2 both breeding and non-breeding) from 16 different families. Of 60 cases where blood parasites were identified, *Haemoproteus* was the most common genus (24 comparisons), followed by *Leucocytozoon* (21), *Trypanosoma* (7), *Plasmodium* (7), and *Microfilaria* (1). Monogamy was the most common mating system in the studies we used (63%).

No sex bias in overall parasitism was found for either breeding or non-breeding adults (Table 1). Prevalence did not differ between breeding versus non-breeding studies ( $t = 0.1$ ,  $n = 36$ ,  $p = 0.93$ ). When broken down by parasite taxon, a significant sex bias in prevalence was found in only one of four comparisons in which a test could be conducted, i.e. for breeding birds parasitised by *Haemoproteus* (females > males, Table 1).

For the two experimental studies, mean effect sizes in hosts were female-biased (mean = 19.8,  $n = 2$ ,  $t = 11.6$ ,  $0.05 < p < 0.10$ ). One comparison was mallards parasitised by *Leucocytozoon simondi*, with overall female biases in prevalence (Chernin 1952a, effect size = 18.1,  $n = 68$ ). The other comparison, a study on effects of *Plasmodium relictum* on house sparrows (Applegate 1971), was the only study where exposure of both sexes to the parasite was controlled. This comparison had the strongest female bias in parasitism of any comparison in the analysis (effect size = 21.5,  $n = 87$ ).

Sex biases in overall prevalence differed between monogamous and polygynous birds, but only for those sampled during the breeding season (breeding:  $t = 2.1$ ,  $n = 29$ ,  $p = 0.04$ ; non-breeding:  $t = 0.1$ ,  $n = 7$ ,  $p = 0.93$ ). Sex biases in overall prevalence for monogamous breeding birds were not significantly different from

Table 1. Differences in prevalence of avian blood parasites between male and female hosts. Each comparison represents one host species-parasite species combination (results combined across years, locations, and studies). Overall values treat each host species and all of its parasites as a single datum.

Parasite taxa	No. host-parasite comparisons (no. studies)	Mean of female minus male prevalence (SD)	
<b>Breeding adults:</b>			
<i>Haemoproteus</i>	15 (18)	3.43 (5.84)	2.27*
<i>Leucocytozoon</i>	11 (13)	0.38 (13.10)	0.10
<i>Plasmodium</i>	5 (6)	-8.61 (15.68)	-1.23
<i>Trypanosoma</i>	7 (9)	2.22 (5.16)	1.14
Microfilaria	1 (1)	-6.68 (1)	†
Overall	29 (24)	1.05 (9.52)	0.59
<b>Non-breeding adults:</b>			
<i>Haemoproteus</i>	4 (7)	3.78 (9.57)	0.79
<i>Leucocytozoon</i>	5 (6)	0.63 (4.70)	0.30
<i>Trypanosoma</i>	1 (1)	2.31 (1)	†
Overall	7 (9)	1.40 (5.07)	0.73

\*  $P < 0.05$ .

† No value was calculable.

zero, whereas those for polygynous birds were female-biased (Fig. 1a). When broken down by parasite taxon, however, mean effect sizes for prevalence by mating system were not significantly different from zero or from each other (all  $p$ s  $> 0.05$ ). For breeding birds, it

was possible to compare monogamous and polygynous birds within the family Parulidae to correct for a possible lack of independence in phylogeny. Within this family, mean effect sizes for monogamous and polygynous birds were significantly different from each other ( $t = 4.3$ ,  $n = 10$ ,  $p = 0.003$ ) and prevalence for polygynous birds also was significantly female-biased (Fig. 1b). When mating systems were nested within continents (North America or Europe), the same trend (female  $>$  male) as found for Parulidae (female  $>$  male) was still evident ( $F = 2.7$ ,  $df = 2, 25$ ,  $p = 0.09$ ).

Sex biases in prevalence were not influenced by relative sexual size dimorphism for either breeding ( $r = -0.31$ ,  $n = 25$ ,  $p = 0.13$ ) or non-breeding birds ( $r = 0.03$ ,  $n = 6$ ,  $p = 0.96$ ).

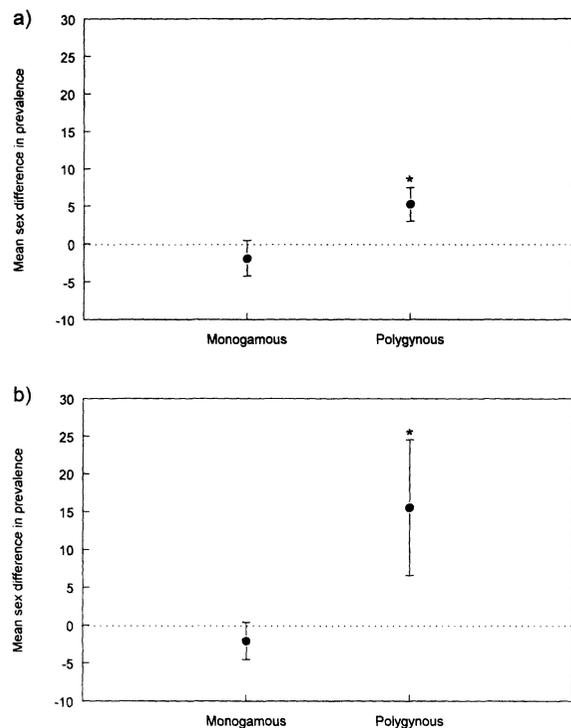


Fig. 1. Mean difference in overall prevalence (female minus male  $\pm$  standard error) of avian blood parasites for monogamous and polygynous birds during the breeding season for a) all bird species, b) the family Parulidae only. In both sets of comparisons, each host species was treated as an independent datum. Means for mating system were significantly different from each other (\* indicates means significantly different from zero).

## Discussion

### Parasite taxon

In contrast to previous research showing that males were more frequently and more heavily infected (Poulin 1996, Schalk and Forbes 1997), we found that females were more frequently infected. This trend was particularly compelling for *Haemoproteus*. It is unlikely that this is explained by *Haemoproteus* causing differential mortality of males because *Haemoproteus* is considered relatively benign (Atkinson and van Riper 1991, Desser and Bennett 1993). For example, anaemia and other symptoms of disease commonly observed with haematozoan infections (Atkinson and van Riper 1991, Desser and Bennett 1993) have not been observed with *Haemoproteus* even when parasites occupied more than half of the erythrocytes in blood smears (Atkinson et al. 1988, Atkinson and van Riper 1991; but see Markus 1972). Hence, hormones, differential exposure, mating system, or size dimorphism are more likely explanations

than is differential mortality for host-sex biases in *Haemoproteus* infections.

Researchers have speculated that hormonally mediated differences in immune systems between the sexes may explain sex-biased parasitism (Grossman 1985, Schuurs and Verheul 1990). Although both humoral and cell-mediated immunity are involved in resisting infections and relapses of *Plasmodium* (a close relative of *Haemoproteus*), cell-mediated immunity is thought to be more important in resisting malarial infections (Seed and Manwell 1977). This is important because, although humoral immunity is reduced in the presence of testosterone, both testosterone and oestrogen can reduce cell-mediated immunity to parasites (Grossman 1985, Schuurs and Verheul 1990). Furthermore, Benison and Coatney (1948) found that female chicks inoculated with *Plasmodium gallinaceum* developed higher intensities of infection than did males. Hence, sex-biased parasitism need not be attributed solely to testosterone, and thus need not be male-biased.

Another explanation for sex-biased parasitism may be differential exposure to vectors, something that other researchers have emphasised in host-parasite relationships (Yezerinac and Weatherhead 1995, Zuk and McKean 1996). For example, even if males were more susceptible to parasitism due to immunosuppression, sex differences in exposure to vectors may obscure this relationship. Differences in exposure are commonly used to explain female biases in parasitism because female birds may have increased exposure while nesting (e.g., Chernin 1952b, Peirce and Marquiss 1983, Korpi-mäki et al. 1993, Norris et al. 1994). It is possible that vectors of *Haemoproteus* may be found near nests of hosts more frequently than vectors of other haemosporidians. In addition, *Haemoproteus* may not be as costly to hosts as are other haemosporidians (Atkinson and van Riper 1991, Desser and Bennett 1993). Hence, female birds may not spend as much energy on vector-avoiding behaviour, such as occurs in native Hawaiian birds exposed to vectors carrying relatively pathogenic plasmodia (van Riper et al. 1986).

Finally, we acknowledge that we cannot exclude chance as an explanation for sex-biased parasitism by *Haemoproteus* because of the number of tests that we ran (Rice 1989). Nonetheless, there are both ecological and immunological reasons that could promote the female bias in *Haemoproteus* infections: this particular parasite-host association is worthy of more direct study.

### Mating system

Prevalence of blood parasites in polygynous birds was significantly female-biased, in contrast to monogamous species, where there was no bias. Once again, ecological and immunological differences between the two mating

systems may be important. As discussed above, differences in exposure of the sexes to parasite vectors may lead to increased susceptibility for females. In polygynous species, the difference between maternal and parental care is greater than in monogamous species (Searcy and Yasukawa 1995). As a result, females of polygynous species may spend a relatively greater proportion of their time than males of polygynous species, or than males and females of monogamous species, in caring for young which may increase female exposure to parasites, or decrease their energy available to combat parasites. Hence, increased maternal care could be costly for females of polygynous species. However, in this study, polygynous species did not have higher prevalence of blood parasites relative to monogamous species. In contrast, Read (1991) found that prevalence of blood parasites was lower in polygynous than in monogamous species.

Hillgarth et al. (1997) predicted that males of polygynous species would be more susceptible to parasites due to increased stress associated with courtship displays. However, polygyny may have no costs for either males or females (Lightbody and Weatherhead 1987, Searcy and Yasukawa 1995). Searcy and Yasukawa (1995) found that as the percentage of polygyny in territorial males increased, male contribution to feeding decreased proportionally. Thus, any increase in energy expenditure due to stresses associated with breeding may be offset by a decrease in parental care. Another possibility raised by Hillgarth et al. (1997) is that immune systems of polygynous males may be more tolerant to increased levels of testosterone. Further studies on immunological differences in avian immune systems and studies of other mating systems, such as polyandrous birds, may help to explain the influence of mating system on sex biases in parasitism.

### Sexual size dimorphism

Sexual size dimorphism was not related to prevalence of blood parasites in either breeding or non-breeding birds. Poulin (1996) also found no significant correlation between sexual size dimorphism and prevalence or intensity of parasitism in mammals (although breeding versus non-breeding species could not be separated). Thus, there is so far no evidence that the larger sex pays an additional cost in terms of increased susceptibility to some parasites.

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## References

- Alexander, J. and Stimson, W. H. 1988. Sex hormones and the course of parasitic infection. – *Parasitol. Today* 4: 189–193.
- Amundsen, T. and Slagsvold, T. 1991. Asynchronous hatching in pied flycatchers: an experiment. – *Ecology* 72: 797–804.
- Andersson, M. 1994. Sexual selection. – Princeton Univ. Press, Princeton, NJ.
- Apanius, V. 1991. Avian trypanosomes as models of hemoflagellate evolution. – *Parasitol. Today* 7: 87–90.
- and Kirkpatrick, C. E. 1988. Preliminary report of *Haemoproteus tinnunculi* infection in a breeding population of American Kestrels. – *J. Wildl. Dis.* 24: 150–153.
- Applegate, J. E. 1971. Spring relapse of *Plasmodium relictum* infections in an experimental field population of English Sparrows (*Passer domesticus*). – *J. Wildl. Dis.* 7: 37–42.
- Arnqvist, G. and Wooster, D. 1995. Meta-analysis: synthesizing research findings in ecology and evolution. – *Trends Ecol. Evol.* 12: 236–240.
- Ashford, R. W., Wyllie, I. and Newton, I. 1990. *Leucocytozoon toddi* in British sparrowhawks *Accipiter nisus*: observations on the dynamics of infection. – *J. Nat. Hist.* 24: 1101–1107.
- Atkinson, C. T. and van Riper III, C. 1991. Pathogenicity and epizootiology of hematozoa. – In: Loye, J. E. and Zuk, M. (eds), *Bird-parasite interactions*. Oxford Univ. Press, New York, pp. 19–48.
- , Forrester, D. J. and Greiner, E. C. 1988. Pathogenicity of *Haemoproteus meleagridis* (Haemosporina: Haemoproteidae) in experimentally infected domestic turkeys. – *J. Parasitol.* 74: 228–239.
- Begg, C. B. 1994. Publication bias. – In: Cooper, H. and Hedges, L. V. (eds), *The handbook of research synthesis*. Russell Sage Foundation, New York, pp. 399–409.
- Behnke, J. M., Barnard, C. J. and Wakelin, D. 1992. Understanding chronic nematode infections: evolutionary considerations, current hypotheses and the way forward. – *Int. J. Parasitol.* 22: 861–907.
- Bennett, G. F., Caines, J. R. and Bishop, M. A. 1988. Influence of blood parasites on the body mass of passeriform birds. – *J. Wildl. Dis.* 24: 339–343.
- , Squires-Parsons, D., Siikamäki, P., Huhta, E., Allander, K. and Hillström, L. 1995. A comparison of the blood parasites of three Fenno-Scandian populations of the Pied Flycatcher *Ficedula hypoleuca*. – *J. Avian. Biol.* 26: 33–38.
- Bennison, B. E. and Coatney, G. R. 1948. The sex of the host as a factor in *Plasmodium gallinaceum* infections in young chicks. – *Science* 107: 147–148.
- Birkhead, T. and Møller, A. P. 1992. Sperm competition in birds: evolutionary causes and consequences. – Academic Press, Orlando, FL.
- Bundy, D. A. P. 1988. Sexual effects of parasite infection. – *Parasitol. Today* 4: 186, 198, 193.
- Chermin, E. 1952a. The epizootiology of *Leucocytozoon simondi* infections of domestic ducks in northern Michigan. – *Am. J. Hyg.* 56: 39–57.
- 1952b. The relapse phenomenon in the *Leucocytozoon simondi* infection in the domestic duck. – *Am. J. Hyg.* 56: 101–118.
- Cook, R. S., Trainer, D. O. and Glazener, W. C. 1966. *Haemoproteus* in wild turkeys from the coastal bend of south Texas. – *J. Protozool.* 13: 588–590.
- Csada, R. D., James, P. C. and Espie, R. H. M. 1996. The “file drawer problem” of non-significant results: does it apply to biological research? – *Oikos* 76: 591–593.
- Dale, S., Kruszewicz, A. and Slagsvold, T. 1996. Effects of blood parasites on sexual and natural selection in the pied flycatcher. – *J. Zool.* 238: 373–393.
- Davidar, P. and Morton, E. S. 1993. Living with parasites: prevalence of a blood parasite and its effect on survivorship in the Purple Martin. – *Auk* 110: 109–116.
- Deibert, P. 1995. Effects of parasites on sage grouse (*Centrocercus urophasianus*) mate selection. – Ph.D. Thesis, Univ. of Wyoming, Laramie, WY.
- Desser, S. S. and Bennett, G. F. 1993. The genera *Leucocytozoon*, *Haemoproteus*, and *Hepatocystis*. – In: Krier, J. P. (ed.), *Parasitic protozoa*, vol. 4. Academic Press, New York, pp. 273–305.
- Dunning, J. B., Jr. 1994. CRC handbook of avian body mass. – CRC Press, Boca Raton, FL.
- Ehrlich, P. R., Dobkin, D. S. and Wheye, D. 1988. The birder's handbook: a field guide to the natural history of North American birds. – Simon and Schuster, New York.
- Felsenstein, J. 1985. Phylogenies and the comparative method. – *Am. Nat.* 125: 1–15.
- Festa-Bianchet, M. 1989. Individual differences, parasites, and the costs of reproduction for bighorn ewes (*Ovis canadensis*). – *J. Anim. Ecol.* 58: 785–795.
- Folstad, I. and Karter, A. J. 1992. Parasites, bright males, and the immunocompetence handicap. – *Am. Nat.* 139: 603–622.
- Forbes, M. R. L. 1993. Parasitism and host reproductive effort. – *Oikos* 67: 444–450.
- 1996. More on parasitism and host reproductive effort. – *Oikos* 75: 321–322.
- Forrester, D. J., Hon, L. T., Williams, Jr., L. E. and Austin, D. H. 1974. Blood protozoa of wild turkeys in Florida. – *J. Protozool.* 21: 494–497.
- Goble, F. C. and Konopka, E. A. 1973. Sex as a factor in infectious disease. – *Trans. N.Y. Acad. Sci.* 35: 325–346.
- Godfrey, Jr., R. D., Pence, D. B. and Fedynich, A. M. 1990. Effects of host and spatial factors on a haemoproteid community in Mourning Doves from western Texas. – *J. Wildl. Dis.* 26: 435–441.
- Gregory, R. D. and Blackburn, T. M. 1991. Parasite prevalence and host sample size. – *Parasitol. Today* 7: 316–318.
- Grossman, C. J. 1985. Interactions between the gonadal steroids and the immune system. – *Science* 227: 257–261.
- Gurevitch, J., Morrow, L. L., Wallace, A. and Walsh, J. S. 1992. A meta-analysis of competition in field experiments. – *Am. Nat.* 140: 539–572.
- Hamilton, W. D. and Zuk, M. 1982. Heritable true fitness and bright birds: a role for parasites? – *Science* 218: 384–387.
- Harvey, P. H. and Pagel, M. D. 1991. *The comparative method in evolutionary biology*. – Oxford Univ. Press, Oxford.
- Hauschka, T. S. 1947. Sex of host as a factor in Chagas' disease. – *J. Parasitol.* 33: 399–404.
- Hedges, L. V. and Olkin, I. 1985. *Statistical methods for meta-analysis*. – Academic Press, San Diego, CA.
- Herman, C. M. 1968. Blood parasites of North American waterfowl. – *Trans. N. Am. Wildl. Nat. Resour. Conf.* 33: 348–358.
- Hillgarth, N., Ramenofsky, M. and Wingfield, J. 1997. Testosterone and sexual selection. – *Behav. Ecol.* 8: 108–112.
- Hirota, Y., Suzuki, T. and Bito, Y. 1980. The development of unusual B-cell functions in the testosterone-propionate-treated chicken. – *Immunology* 39: 29–36.
- Hurst, G. D. D., Hurst, L. D. and Majerus, M. E. N. 1993. Altering sex ratios: the games microbes play. – *Bioessays* 15: 695–697.
- John, J. L. 1995. Parasites and the avian spleen: helminths. – *Biol. J. Linn. Soc.* 54: 86–106.
- Jonsson, L. 1992. *Birds of Europe; with North Africa and the Middle East*. – Princeton Univ. Press, Princeton, NJ.
- Kirkpatrick, C. E., Robinson, S. K. and Kitron, U. D. 1991. Phenotypic correlates of blood parasitism in the common grackle. – In: Loye, J. E. and Zuk, M. (eds), *Bird-parasite interactions*. Oxford Univ. Press, New York, pp. 344–358.
- Korpimäki, E., Hakkarainen, H. and Bennett, G. F. 1993. Blood parasites and reproductive success of Tengmalm's owls: detrimental effects on females but not on males? – *Funct. Ecol.* 7: 420–426.
- , Tolonen, P. and Bennett, G. F. 1995. Blood parasites, sexual selection and reproductive success of European Kestrels. – *Ecoscience* 2: 335–343.

- Lehikoinen, E. 1987. Seasonalities of the daily weight cycle in wintering passerines and its consequences. – *Ornis Scand.* 18: 216–226.
- Lightbody, J. P. and Weatherhead, P. J. 1987. Polygyny in the yellow-headed blackbird: female choice versus male-male competition. – *Anim. Behav.* 35: 1670–1684.
- Markus, M. B. 1972. Pathogenicity of *Haemoproteus columbae*. – *Trans. R. Soc. Trop. Med. Hyg.* 66: 186–187.
- Martin, T. E. and Clobert, J. 1996. Nest predation and avian life-history evolution in Europe versus North America: a possible role of humans? – *Am. Nat.* 147: 1028–1046.
- Merilä, J., Björklund, M. and Bennett, G. F. 1995. Geographic and individual variation in hematozoan infections in the greenfinch, *Carduelis chloris*. – *Can. J. Zool.* 73: 1798–1804.
- Merino, S. and Potti, J. 1995. High prevalence of hematozoa in nestlings of a passerine species, the Pied Flycatcher (*Ficedula hypoleuca*). – *Auk* 112: 1041–1043.
- Møller, A. P. 1986. Mating systems among European passerines: a review. – *Ibis* 128: 234–250.
- National Geographic Society. 1983. Field guide to the birds of North America. – National Geographic Society, Washington, DC.
- Norris, K., Anwar, M. and Read, A. F. 1994. Reproductive effort influences the prevalence of hematozoan parasites in great tits. – *J. Anim. Ecol.* 63: 601–610.
- Norton, J. M. and Wira, C. R. 1977. Dose-related effects of the sex hormones and cortisol on the growth of the Bursa of Fabricius in chick embryos. – *J. Steroid Biochem.* 8: 985–987.
- O'Dell, J. P. and Robbins, L. W. 1994. Hematozoa of wood ducks (*Aix sponsa*) in Missouri. – *J. Wildl. Dis.* 30: 36–39.
- Ots, I. and Hörak, P. 1996. Great tits *Parus major* trade health for reproduction. – *Proc. R. Soc. Lond. B* 263: 1443–1447.
- Palmer, R. S. 1976. Handbook of North American birds, vol. 2. – Yale Univ. Press, New Haven, CT.
- Peirce, M. A. and Marquiss, M. 1983. Hematozoa of British birds. VII. Hematozoa of raptors in Scotland with a description of *Haemoproteus nisi* sp. nov. from the sparrowhawk (*Accipiter nisus*). – *J. Nat. Hist.* 17: 813–821.
- Poulin, R. 1996. Sexual inequalities in helminth infections: a cost of being male? – *Am. Nat.* 147: 287–295.
- Promislow, D. E. L. 1991. The evolution of mammalian blood parameters: patterns and their interpretation. – *Physiol. Zool.* 63: 393–431.
- , Montgomerie, R. and Martin, T. E. 1992. Mortality and the costs of sexual dimorphism in birds. – *Proc. R. Soc. Lond. B* 250: 143–150.
- Ranta, E., Laurila, A. and Elmberg, J. 1994. Reinventing the wheel: analysis of sexual dimorphism in body size. – *Oikos* 70: 313–321.
- Read, A. F. 1991. Passerine polygyny: a role for parasites? – *Am. Nat.* 138: 434–459.
- Rice, W. R. 1989. Analyzing tables of statistical tests. – *Evolution* 43: 223–225.
- Richner, H., Christe, P. and Oppliger, A. 1995. Parental investment affects prevalence of malaria. – *Proc. Natl. Acad. Sci. USA.* 92: 1192–1194.
- Schalk, G. and Forbes, M. R. 1997. Male biases in parasitism of mammals: effects of study type, host age, and parasite taxon. – *Oikos* 78: 67–74.
- Schuurs, A. H. W. M. and Verheul, H. A. M. 1990. Effects of gender and sex steroids on the immune response. – *J. Steroid Biochem.* 35: 157–172.
- Searcy, W. A. and Yasukawa, K. 1995. Polygyny and sexual selection in red-winged blackbirds. – Princeton Univ. Press, Princeton, NJ.
- Seed, T. M. and Manwell, R. D. 1977. Plasmodia of birds. – In: Kreier, J. P. (ed.), Parasitic protozoa vol. III. Academic Press, New York, pp. 311–357.
- Seutin, G. 1994. Plumage redness in redpoll finches does not reflect hemoparasitic infection. – *Oikos* 70: 280–286.
- Sheldon, B. C. and Verhulst, S. 1996. Ecological immunity: costly parasite defenses and trade-offs in evolutionary ecology. – *Trends Ecol. Evol.* 11: 317–321.
- Shutler, D., Ankney, C. D. and Dennis, D. G. 1996. Could the blood parasite *Leucocytozoon* deter mallard range expansion? – *J. Wildl. Manage.* 60: 569–580.
- Sibley, C. G. and Ahlquist, J. E. 1990. Phylogeny and classification of birds: a study in molecular evolution. – Yale Univ. Press, London.
- Sokal, R. R. and Rohlf, F. J. 1981. Biometry. 2nd ed. – Freeman, San Francisco.
- Stabler, R. M., Braun, C. E. and Beck, T. D. I. 1977. Hematozoa in Sage Grouse from Colorado. – *J. Wildl. Dis.* 13: 414–417.
- Stacey, L. M., Couvillion, C. E., Siefker, C. and Hurst, G. A. 1990. Occurrence and seasonal transmission of hematozoa in wild turkeys. – *J. Wildl. Dis.* 26: 442–446.
- Summers-Smith, J. D. 1988. The sparrows: a study of the genus *Passer*. – Poyser, Calton.
- Super, P. E. and van Riper III, C. 1995. A comparison of avian hematozoa in two California coastal shrub communities. – *J. Wildl. Dis.* 31: 447–461.
- Taft, S. J., Rosenfield, R. N. and Bielefeldt, J. 1994. Avian hematozoa of adult and nestling Cooper's Hawks (*Accipiter cooperii*) in Wisconsin. – *J. Helminthol. Soc. Wash.* 61: 146–148.
- Teather, K. L. and Weatherhead, P. J. 1989. Sex-specific mortality in nestling great-tailed grackles. – *Ecology* 70: 1485–1493.
- Tella, J. L., Forero, M. G., Gajón, A., Hiraldo, F. and Donazar, J. A. 1996. Absence of blood-parasitization effects on Lesser Kestrel fitness. – *Auk* 113: 253–256.
- Trainer, D. O., Schildt, C. S., Hunt, R. A. and Jahn, L. R. 1962. Prevalence of *Leucocytozoon simondi* among some Wisconsin waterfowl. – *J. Wildl. Manage.* 26: 137–143.
- van Riper III, C., van Riper, S. G., Goff, M. L. and Laird, M. 1986. The epizootiology and ecological significance of malaria in Hawaiian land birds. – *Ecol. Monogr.* 56: 327–344.
- Weatherhead, P. J. and Bennett, G. F. 1991. Ecology of Red-Winged Blackbird parasitism by hematozoa. – *Can. J. Zool.* 69: 2352–2359.
- and Bennett, G. F. 1992. Ecology of parasitism of Brown-headed Cowbirds by hematozoa. – *Can. J. Zool.* 70: 1–7.
- , Bennett, G. F. and Shutler, D. 1991. Sexual selection and parasites in wood-warblers. – *Auk* 108: 147–152.
- Williams, N. A., Mahrt, J. L. and Zwickel, F. C. 1980. The ecology of blood parasites in blue grouse from Vancouver Island, British Columbia. – *Can. J. Zool.* 58: 2175–2186.
- Yan, G., Severson, D. W. and Christensen, B. M. 1997. Costs and benefits of mosquito refractoriness to malaria parasites: implications of genetic variability of mosquitoes and genetic control of malaria. – *Evolution* 51: 441–450.
- Yezerinac, S. M. and Weatherhead, P. J. 1995. Plumage coloration, differential attraction of vectors and hematozoa in birds. – *J. Anim. Ecol.* 64: 528–537.
- Zuk, M. 1990. Reproductive strategies and disease susceptibility: an evolutionary viewpoint. – *Parasitol. Today* 6: 231–233.
- and McKean, K. A. 1996. Sex differences in parasite infections: patterns and processes. – *Int. J. Parasitol.* 26: 1009–1024.

Appendix A. Hosts, parasites, reproductive phenology and mating system, body mass, and sample sizes for each comparison used in statistically correct vote counts. More detailed information can be obtained from the primary studies (species names and order follow Dunning 1994, sources provided below). Generic abbreviations for haemosporidian parasites are as follows: *Haemoproteus* (H), *Leucocytozoon* (L), *Plasmodium* (P), *Trypanosoma* (T) and ? for unknown. Microfilaria of unidentified nematodes are abbreviated as MF. For host phenology and mating system, the first letter indicates whether the host was breeding (B) or not breeding (N) when prevalence was assessed. The second letter indicates whether the host breeds monogamously (M) or polygynously (P, which also includes lekking species). Studies in parentheses are those from non-natural populations or where experimental infections were conducted. Where body mass could not be determined separately for both sexes, a “?” is shown. Sample sizes given are those for each host-parasite comparison (locations and years within studies combined) where  $n \geq 10$  for each sex of adult at each location and in each year.

Sources: 1) O'Dell and Robbins 1994. 2) Chernin 1952a. 3) Trainer et al. 1962. 4) Ashford et al. 1990. 5) Peirce and Marquiss 1983. 6) Taft et al. 1994. 7) Tella et al. 1996. 8) Korpimäki et al. 1995. 9) Apanius and Kirkpatrick 1988. 10) Dawson and Bortolotti unpubl. 11) Williams et al. 1980. 12) Deibert 1995. 13) Stabler et al. 1977. 14) Cook et al. 1966. 15) Forrester et al. 1974. 16) Stacey et al. 1990. 17) Godfrey et al. 1990. 18) Korpimäki et al. 1993. 19) Bennett et al. 1988. 20) Bennett et al. 1995. 21) Dale et al. 1996. 22) Merino and Potti 1995. 23) Davidar and Morton 1993. 24) Super and van Riper 1995, Super unpubl. 25) Norris et al. 1994. 26) Richner et al. 1995. 27) Applegate 1971. 28) Merilä et al. 1995. 29) Seutin 1994. 30) Weatherhead et al. 1991. 31) Weatherhead and Bennett 1991. 32) Kirkpatrick et al. 1991. 33) Weatherhead and Bennett 1992.

Source	Host	Parasite	Phenology of host and mating system	Body mass (g) (female, male)	Sample size (female, male)
1	<i>Aix sponsa</i>	<i>H. nettionis</i> <i>L. simondi</i>	N, M	635, 681	44, 70 29, 35
2	<i>Anas platyrhynchos</i>	<i>L. simondi</i>	(N, M)	(1080, 1240)	39, 29
3		<i>L. simondi</i>	N, M	1080, 1240	24, 24
4	<i>Accipiter</i>	<i>L. toddi</i>	B, M	325, 150	45, 45
5		<i>H. sp.</i> <i>L. sp.</i> <i>T. sp.</i>			39, 22 39, 22 39, 22
6	<i>Accipiter cooperii</i>	<i>H. sp.</i> <i>L. toddi</i>	B, M	529, 349	17, 11 17, 11
7	<i>Falco naumanni</i>	<i>H. tinnunculi</i>	B, M	164, 141	210, 168
8	<i>Falco tinnunculus</i>	<i>H. brachiatatus</i> <i>H. tinnunculi</i> <i>L. toddi</i> <i>P. circumflexum</i> <i>T. avium</i>	B, M	217, 186	113, 94 113, 94 113, 94 113, 94 113, 94
9	<i>Falco sparverius</i>	<i>H. tinnunculi</i> <i>H. tinnunculi</i> & <i>H. brachiatatus</i>	B, M	120, 111	23, 14 137, 105
10		<i>H. mansoni</i>	B, P	891, 1188	578, 395
11	<i>Dendragapus obscurus</i>	<i>L. bonasae</i> MF <i>T. avium</i>			629, 395 629, 395 629, 395
12	<i>Centrocercus urophasianus</i>	<i>P. peidioceti</i>	B, P	1745, 3190	49, 152
13		<i>H. sp.</i> <i>L. sp.</i> <i>T. sp.</i> <i>H. sp.</i> <i>L. sp.</i> <i>T. sp.</i>	N, L		115, 173 115, 173 115, 173 29, 25 29, 25 29, 25
14	<i>Meleagris gallopavo</i>	<i>H. meleagridis</i>	N, P	4222, 7400	50, 20 299, 185
15		<i>H. sp.</i> <i>L. sp.</i>			299, 185 61, 33
16		<i>H. sp.</i> <i>L. sp.</i>			61, 33 61, 33
17	<i>Zenaida macroura</i>	<i>H. columbae</i>	N, M	115, 123	21, 14
18	<i>Aegolius funereus</i>	<i>H. noctuae</i> <i>H. syrnii</i> <i>L. ziemanni</i>	B, P	167, 101	97, 85 97, 85 97, 85
19	<i>Catharus guttatus</i>	<i>H. sp.</i> & <i>L. sp.</i>	B, M	32.0, 29.4	24, 19
20	<i>Ficedula hypoleuca</i>	<i>H. balmorali</i> <i>H. pallidus</i> <i>L. sp.</i> <i>P. vaughani</i> & <i>P. everetti</i> <i>T. sp.</i> <i>H. sp.</i> <i>T. sp.</i>	B, P	12.7, 12.3	462, 307 412, 270 412, 270 462, 307 462, 307 130, 141 130, 141
21		<i>H. sp.</i> <i>T. sp.</i>			111, 99 111, 99
22		<i>H. sp.</i> <i>L. sp.</i> <i>T. sp.</i>			111, 99 111, 99 111, 99
23	<i>Progne subis</i>	<i>H. prognei</i> <i>H. prognei</i>	B, P N, P	48.8, 53.5	144, 173 25, 22

## Appendix A. (continued)

Source	Host	Parasite	Phenology of host and mating system	Body mass (g) (female male)	Sample size (female, male)
24	<i>Regulus calendula</i>	<i>L. sp.</i>	N, M	6.4, 6.9	22, 19
25	<i>Parus major</i>	<i>L. sp.</i>	B, P	17.6, 18.9	60, 53
26		<i>P. sp.</i>			18, 19
27	<i>Passer montanus</i>	<i>P. relictum</i>	(B, M)	(?)	34, 53
28	<i>Carduelis chloris</i>	<i>H. chloris</i>	B, M	26.1, 27.9	84, 95
29	<i>Carduelis flammea</i>	<i>H. sp.</i>	B, M	12.7, 12.6	19, 76
		<i>L. sp.</i>			19, 76
		<i>T. sp.</i>			19, 76
30	<i>Vermivora peregrina</i>	?	B, M	9.8, 10.2	78, 89
19	<i>Dendroica magnolia</i>	<i>H. sp. &amp; L. sp.</i>	B, M	8.5, 8.9	46, 72
30	<i>Dendroica coronata</i>	?	B, M	12.2, 12.9	97, 175
19	<i>Dendroica striata</i>	<i>H. sp. &amp; L. sp.</i>	B, P	11.3, 13.9	77, 116
30		?			120, 182
19	<i>Mniotilta varia</i>	<i>H. sp. &amp; L. sp.</i>	B, M	10.6, 11.0	47, 77
30		?			88, 112
30	<i>Setophaga ruticilla</i>	?	B, M	8.1, 8.5	310, 381
30	<i>Seiurus aurocapillus</i>	?	B, P	22.1, 22.9	11, 25
30	<i>Seiurus noveboracensis</i>	?	B, M	20.8, 21.9	211, 271
30	<i>Oporornis philadelphia</i>	?	B, M	12, 13	19, 61
30	<i>Wilsonia pusilla</i>	?	B, P	7.5, 7.9	30, 173
19	<i>Passerella iliaca</i>	<i>H. sp. &amp; L. sp.</i>	B, M	36.2, 37.4	43, 57
19	<i>Melospiza lincolni</i>	<i>H. sp. &amp; L. sp.</i>	B, M	17.7, 18.1	32, 58
31	<i>Agelaius phoeniceus</i>	<i>H. quisqualis</i>	B, P	41.5, 63.6	212, 226
		<i>L. fringillinarum</i>			212, 226
		<i>P. cathemerium,</i>			
		<i>P. vaughani &amp; P. polare</i>			212, 226
32	<i>Quiscalus quiscula</i>	<i>H. quisqualis</i>	B, P	100, 127	294, 300
33	<i>Molothrus ater</i>	<i>H. quisqualis &amp;</i>	B, P	38.8, 49.0	311, 517
		<i>L. fringillinarum</i>			311, 517
		<i>P. cathemerium,</i>			
		<i>P. vaughani &amp; P. polare</i>			311, 517
		<i>T. sp.</i>			311, 517