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PARASITISM, HOST IMMUNE FUNCTION, AND SEXUAL SELECTION

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ABSTRACT

Parasite-mediated sexual selection may arise as a consequence of 1) females avoiding mates with directly transmitted parasites, 2) females choosing less-parasitized males that provide parental care of superior quality, or 3) females choosing males with few parasites in order to obtain genes for parasite resistance in their offspring. Studies of specific host-parasite systems and comparative analyses have revealed both supportive and conflicting evidence for these hypotheses. A meta-analysis of the available evidence revealed a negative relationship between parasite load and the expression of male secondary sexual characters. Experimental studies yielded more strongly negative relationships than observations did, and the relationships were more strongly negative for ectoparasites than for endoparasites. There was no significant difference in the magnitude of the negative effect for species with and without male parental care, or between behavioral and morphological secondary sexual characters. There was a significant difference between studies based on host immune function and those based on parasite loads, with stronger effects for measures of immune function, suggesting that the many negative results from previous analyses of parasite-mediated sexual selection may be explained because relatively benign parasites were studied. The multivariate analyses demonstrating strong effect sizes of immune function in relation to the expression of secondary sexual characters, and for species with male parental care as compared to those without, suggest that parasite resistance may be a general determinant of parasite-mediated sexual selection.

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INTRODUCTION

PARASITE-MEDIATED sexual selection has had a prominent position among studies of sexual selection ever since Hamilton and Zuk (1982) proposed their hypothesis that only males in prime condition, and therefore likely to carry genes for parasite resistance, should be able to express the most extreme secondary sexual characters. A large number of studies have subsequently addressed the role of parasites in sexual selection.

The Hamilton and Zuk hypothesis is not the only one that seeks to explain why parasites may play an important role in host sexual selection. The parasite avoidance hypothesis and the efficient parent hypothesis are two alternatives. The parasite avoidance hypothesis claims that females prefer the most extravagantly ornamented males because the probability of acquiring contagious parasites from such males is smaller than from the average male in the population (Freeland 1976; Borgia 1986; Borgia and Collis 1989). The efficient parent hypothesis posits that females prefer extravagantly ornamented males because such males are more efficient parents, simply because they can allocate more resources to paternal care than parasite-infested males (Hamilton 1990; Møller 1990c). While the Hamilton-Zuk hypothesis provides a mechanism for choosy females to accrue indirect fitness benefits, the two alternatives provide mechanisms entirely based on direct fitness benefits.

A number of different assumptions and predictions have been raised for the three hypotheses. For example, the Hamilton-Zuk hypothesis predicts 1) a genetic correlation between parasite resistance and secondary sexual characters, and 2) a microevolutionary increase in parasite resistance as a consequence of female choice (Kirkpatrick and Ryan 1991). Their obvious intractability has apparently discouraged tests of these predictions.

The exact mechanism that generates a relationship between the expression of secondary sexual characters and parasite load has been the subject of some debate. The original model proposed by Hamilton and Zuk (1982) assumed that secondary sexual characters acted as reliable indicators of male quality,

based on a revealing handicap mechanism (see Andersson 1994). Alternatively, secondary sexual characters influenced by parasites may act as true condition-dependent handicaps, with the secondary sex trait reliably reflecting the condition of individual males. Subsequent analyses have demonstrated that females may obtain resistance genes by choosing the most extravagantly ornamented males, even if their secondary sexual characters do not act as a handicap (Wedekind 1994a).

Folstad and Karter (1992) proposed that the mechanism generating reliability in the sexual signaling system depended on the immunosuppressive effects of testosterone, which is essential for full expression of male secondary sexual characters in many vertebrates. This so-called immunocompetence handicap could act as a result of the constraints imposed by androgens on the signaling system. Alternatively, males may be able to optimize their level of sexual display without compromising their immune system (Møller and Saino 1994; Wedekind and Folstad 1994). A second type of mechanism that could directly enforce reliability of signaling by an indicator mechanism is based on the assumption that testosterone affects spermatogenesis, but simultaneously influences immune function negatively (Folstad and Skarstein 1997; Hillgarth et al. 1997). Hence females will obtain fertile and immunocompetent mates with low parasite loads by choosing the most extravagantly ornamented males (Folstad and Skarstein 1997; Hillgarth et al. 1997). Immunosuppression caused by androgens or corticosteroids can only apply to vertebrates, although similar tradeoff mechanisms based on other substances may apply to the reproductive physiology of invertebrates.

The exact signals females use to determine male parasite status could be those most severely affected by parasites and, therefore, the most reliable signals. Since many organisms use multiple signals in sexual contexts, different signals may reveal infections with different kinds of parasites, as suggested by Wedekind (1992). The exact assessment mechanisms of male parasite infection status, and thus potential parasite resistance, may be important since costly signals are particularly likely to play a role in condition-dependent sexual selection. Resistance genes could be revealed di-

rectly from sexual signals such as odor in mammals, as determined by MHC-haplotype (Potts et al. 1991; Potts and Wakeland 1993; Wedekind 1994b). For example, some studies of rodents and humans have now suggested that mate preferences are directly associated with MHC-dependent odors (Potts et al. 1991; Wedekind et al. 1995), and similar properties may be revealed by visual signals in other organisms, such as birds (von Schantz et al. 1996). Finally, other olfactory properties of parasitized individuals, such as opionates, may reliably reveal the parasite status of potential partners (Hudson et al. 1992; Kavaliers and Colwell 1993).

Male ornamentation is associated with heritable parasite resistance, and parasite resistance is coupled with the expression of secondary sexual characters (reviews in Møller 1990b; Read 1990; Clayton 1991; Zuk 1992; Hamilton and Poulin 1997). A large number of studies of particular host-parasite systems have been conducted, and although some of these provide evidence consistent with parasite-mediated sexual selection, there is little agreement about the pertinence of the three alternative hypotheses (reviews in Møller 1990b; Read 1990; Clayton 1991; Zuk 1992; Able 1996; Hamilton and Poulin 1997). Some studies have demonstrated clear effects of parasites on the expression of secondary sexual characters, with females choosing the most elaborately ornamented males because they are parasite-free or have fewer parasites than the average male.

The original hypothesis explaining female preference for extravagantly ornamented males because of indirect benefits of genetically based parasite resistance was supported by comparative evidence showing a relationship between plumage brightness, in particular sexual dichromatism, and blood parasites (Hamilton and Zuk 1982). Subsequent analyses have demonstrated that this finding may have been biased because humans differ in the way they subjectively score a species as dull or bright (Read and Harvey 1989). Consequently, the six independent sets of brightness scores obtained by Read and Harvey (1989) were shown to be repeatable among scorers, and correlated significantly with those of Hamilton and Zuk (1982); but they did not correlate with the

parasite prevalences reported by Hamilton and Zuk, however, particularly when similarity due to common descent was controlled for (Read and Harvey 1989). A number of subsequent comparative studies of the relationship between sexual dichromatism, or other measures of sexual display, and parasitism provide a mix of supportive and negative evidence for parasite-mediated sexual selection in birds (Johnson 1991; Pruett-Jones et al. 1991; Read 1987, 1991; Read and Weary 1990; Scott and Clutton-Brock 1990; Sorci and Møller 1997; Weatherhead et al. 1991; Zuk 1991), fishes (Chandler and Cabana 1991; Ward 1988, 1989) or lizards (Lefcort and Blaustein 1991). Some possible reasons for the absence of supportive evidence were offered: the original hypothesis was wrong, or the sexual dichromatism database was biased, or the parasite database consisted of unrepeatable measures of prevalence, or uncontrolled confounding variables were present (Endler and Lyles 1989; Read and Harvey 1989; Møller 1990b; Read 1990; Clayton 1991; John 1995; Yezerinac and Weatherhead 1995).

The three hypotheses of parasite-mediated sexual selection apply to somewhat different kinds of organisms, and this fact so far has not been used to discriminate among the alternatives. The efficient parent hypothesis applies only to species with male parental care, and both directly and indirectly transmitted parasites may be involved. The parasite avoidance hypothesis applies to directly transmitted parasites (Able 1996), and to any mating system. Finally, the parasite resistance hypothesis applies to both directly and indirectly transmitted parasites and to hosts with any mating system.

The equivocal support for the three hypotheses of parasite-mediated sexual selection is obvious from both reviews and original papers, since a large number of studies have not provided evidence that particular parasite species affect the expression of secondary sexual characters, that females obtain parasite-free mates when choosing brightly colored males, or that male brightness or other types of sexual display are associated with measures of parasite prevalence (reviews in Møller 1990b; Read 1990; Clayton 1991; Zuk 1992; Able 1996; Hamilton and Poulin 1997). The mixed evidence raises

the possibility that the hypotheses may be of minor importance or that other mechanisms of sexual selection are more relevant. There is at least one alternative explanation, however, that has not been fully appreciated (Møller et al. 1998a). Species may be host to many kinds of parasites; several hundred species of micro-parasites and macroparasites may be associated with a given vertebrate (Marshall 1981). Certainly the number of macroparasites (ectoparasites and helminths) that affect humans and well-studied domesticated animals reaches this level. If research efforts were naively concentrated on the effects of a particular parasite species on the sexual displays of the host, the probability that this one parasite will have severely negative consequences for the fitness of the host is indeed small. Although many scientists have studied parasites believed to have important fitness consequences for their hosts, it is obvious that only a small fraction of these studies have used particularly debilitating parasites. If a parasite species is chosen at random for a study of its effects on host sexual selection, it is highly unlikely that the study will reveal a negative association between the intensity of infection and the expression of secondary sexual characters. The intensity of infection with different species of parasites generally is not strongly positively correlated (e.g., Møller 1991b; Weatherhead et al. 1993). The same caveat applies to comparative studies: even though malarial parasites or immunodeficiency viruses are particularly debilitating in one host species (e.g., humans), we cannot make strong inferences about the fitness consequences for closely related host species (Anderson and May 1991). Thus, although the expression of a secondary sexual character may be strongly negatively correlated with the intensity of infection by one kind of parasite in a particular host, this may not hold true even for closely related host-parasite associations.

A possible way to approach this problem is to use measures of host antiparasite defenses rather than parasite intensities for studies of parasite-mediated sexual selection. The immune system of invertebrates and, in particular, of vertebrates is likely to have evolved to provide efficient defense against the most debilitating parasites that have affected a particular host species during the recent past. Hence,

measures of the ability of hosts to raise immune responses towards parasites may provide much more powerful estimates of the role of parasite-mediated sexual selection than estimates of the intensity of parasite infections.

Folstad and Karter (1992) hypothesized that secondary sexual characters may reveal important information about immunocompetence, because of the tradeoffs between the positive effects of androgens (or similar substances) on secondary sex traits and the negative effects on immune function. Other mechanisms that suggest secondary sexual characters as reliable signals of immune function are conceivable, such as immune function and secondary sex traits that have condition-dependent expression (Møller and Saino 1994). A consideration of selection for immune systems able to cope with particularly debilitating parasites would enlighten comparative studies, because relative investment of a host species in immune function may reflect the importance of parasitic infection in the past. As one test of this idea, a comparative study of the relative size of two immune defense organs (bursa of Fabricius and spleen) in birds revealed a consistently larger size in sexually dichromatic species, as compared with closely related sexually monochromatic species (Møller et al. 1998a), and that males suffered more from immunosuppression as adults in bird species with intense sexual selection, as determined from the frequency of extra-pair paternity (Møller et al. 1998b).

In this study we used a meta-analytic approach to investigate the magnitude of the relationship between expression of secondary sexual characters, and both parasite abundance and host immune function. We decided to investigate these relationships because this association is central to all ideas about parasite-mediated sexual selection: males signal the level of parasitism through the quality of their secondary sexual characters. There is much evidence that females use the expression of male secondary sexual characters in the acquisition of mates (review in Andersson 1994). Meta-analysis provides a stringent way of summarizing a body of research while taking sample size into account (Hunter et al. 1982; Hedges and Olkin 1985; Rosenthal 1991; Cooper and Hedges 1994). Meta-analysis also offers a way

to calculate whether a large number of papers demonstrate an overall effect in a predicted direction, based on the calculation of average effect size. If there is heterogeneity in effect size, diverse moderator variables can be used to investigate whether they may account for some of this heterogeneity. A recent meta-analysis of parasitism and sexual selection (Hamilton and Poulin 1997) was based on a small number of intraspecific studies (18 samples versus 69 samples in the present study); they did not report on any studies of immune response in relation to sexual display. They reported a weak but significant average negative relationship between parasite load and the expression of secondary sexual characters. Furthermore, some analyses indicated a weak heterogeneity of results owing to differences among host taxa, and a slightly stronger effect for endoparasites as compared to ectoparasites.

We specifically tested the following hypotheses in this study: 1) The expression of secondary sexual characters is negatively related to both intensity of parasite infection and host immune function; 2) Experimental studies demonstrate stronger effects than observational studies; 3) Phenotypically labile behavioral traits are more strongly affected by parasites than rigid morphological signals; 4) The expression of secondary sexual characters is more greatly affected by directly transmitted ectoparasites than by indirectly transmitted endoparasites; 5) The expression of secondary sexual characters is more greatly affected by parasites in species with male parental care; 6) Measures of host immune function are more strongly negatively related to the expression of secondary sexual characters than are the intensity of parasite infections. Finally, by using analyses of variance we tested for the statistical independence of these variables and for potential interactions. Our main finding was that host immune function, rather than parasite load, demonstrated strong relationships with the expression of secondary sexual characters, and that the focus on relatively benign parasites in many previous studies of parasite-mediated sexual selection may have tended to de-emphasize the importance of parasites in host sexual selection.

DATA SETS

For this meta-analysis we obtained data from an extensive number of studies. The single criterion for inclusion of a paper was that it reported on the relationship between the expression of a male sexual display or secondary sexual character and either parasite load or measures of immune function. A few studies without statistical information that could be transformed into a standardized measure of effect size were not used. Our database contains many more studies than were employed by Hamilton and Poulin (1997), and we believe that it includes essentially all relevant studies published to date.

Meta-analyses may be problematic if null results are not published (Hunter and Schmidt 1990). Obviously, we cannot know how many unpublished studies of negative results exist, but one way of addressing this problem is to calculate the fail-safe number of publications (Rosenthal 1991:104). The fail-safe number estimates the number of unknown additional studies that would be needed to eliminate an overall effect's significance at the 5% level, when those studies showed an average null result ($z = 0.00$). The literature on parasite-mediated sexual selection includes both supportive and nonsupportive results, and we think it unlikely that the field suffers seriously from unpublished null results.

For the predictions, we used the null hypothesis of no relationship between the expression of male secondary sexual characters and parasite load and immune response; the alternative hypothesis is that the most extravagantly ornamented individuals are also the healthiest ones. Thus, we predicted negative relationships between male secondary sexual characters and parasite load across individuals, so all studies were based on the intensity of parasitic infection. Concerning immune function, we assumed that measures of immune system activity fall into two different categories: immune defense, which reflects adaptations to an evolutionary history of parasite exposure, and immune response, which reflects a physiological response to recent parasite infections.

Measures of immune defense reflect the ability of an individual to raise an immune reaction to a standardized challenge, such as in-

jections with sheep red blood cells or a similar antigen (National Research Council 1992; Lefkovits 1997). These measures are likely to reflect the maximum immune reaction for a given individual under defined conditions. For measures of immune function, we predicted a positive relationship with the expression of secondary sexual characters across individuals if the magnitude of a secondary sexual character reliably reflects immunocompetence.

Measures of immune response reflect the physiological responses, and thus the current health status, of individuals. These measures do not necessarily indicate the maximum response for an individual, because they depend on the kind of parasite and the intensity of infection (Klein 1990; Roitt et al. 1996; Coles 1997). For immune response measures, such as circulating levels of leukocytes and immunoglobulins and the size of immune defense organs (such as the bursa of Fabricius and the spleen), we expected healthy individuals to have low measures and sick individuals to have high measures. There is considerable evidence for higher leukocyte and immunoglobulin concentrations after attacks by various parasites (Arvy 1965; Hodges 1979; Fox and Solomon 1981; Rose 1981; Keymer 1982; Glick 1983, 1994; Toivanen and Toivanen 1987; John 1995). Similarly, the sizes of the bursa of Fabricius and the spleen reflect current health status—diseased individuals have larger immune defense organs (Vincent and Ash 1978; Ali and Behnke 1985; Garside et al. 1989; Watkins et al. 1991; John 1994; Giacomo et al. 1997). Therefore we predicted a negative relationship between the expression of secondary sexual characters and measures of immune response.

The assumptions about immune system relationships to secondary sexual characters were confirmed in a number of species. In the peacock *Pavo cristatus*, for example, leukocyte and immunoglobulin concentrations are negatively related to the size of the extravagant train, whereas antibodies produced in reaction to sheep red blood cells and lymphocyte proliferation in response to phytohemagglutinin are positively related to the size of the secondary sexual character (Møller and Petrie, unpublished data). In the barn swallow *Hirundo rustica*, both spleen size and leukocyte and im-

munoglobulin concentrations are negatively correlated or unrelated to male tail length (the secondary sexual character) (Saino and Møller 1994; Saino et al. 1995; Møller et al. 1998a), whereas antibody production in reaction to sheep red blood cells and lymphocyte proliferation in response to phytohemagglutinin are positively related to tail length (Saino and Møller 1996; Merino and Møller, unpublished data). In the house sparrow *Passer domesticus*, male badge size is negatively correlated with the size of the bursa of Fabricius and spleen (Møller et al. 1996, 1998a). In the lizard *Psammodromus algirus*, the concentration of leukocytes and immunoglobulins is negatively related to the intensity of male coloration, whereas lymphocyte proliferation in response to phytohemagglutinin is positively related to coloration (Sorci and Soler, unpublished data).

Given statistically significant heterogeneity among effect sizes, we investigated the effects of five moderator variables that could potentially explain some or all of this heterogeneity. Studies and samples were scored with respect to:

- 1) whether they were based on experimental manipulation of parasites and immune variables or observations of relationships; this comparison was based on the assumption that experiments control for the effects of confounding variables, which are less strictly controlled in observational studies.
- 2) whether stable morphological characters (such as color, extravagant feathers, antlers) or labile behavioral displays were involved; this comparison was based on the assumption that labile behavioral traits should more closely reflect current parasite infections than morphological traits, which develop over a longer period of time.
- 3) whether parasites were directly transmitted ectoparasites or indirectly transmitted endoparasites (all endoparasites in the present review were indirectly transmitted); this comparison was based on the assumption that, if male infection status was determined by the rate of encounters with already infected hosts, we would predict a positive association between parasite load and male attractiveness because attractive males potentially encounter more infected conspecifics.
- 4) whether males provide parental care, according to information in publications or elsewhere in the literature; this comparison was based on the assumption that effects

should be stronger for species with male parental care if females assess the quality of male care from the health status of males as determined by their sexual displays.

- 5) whether intensity of parasitism or immune function assessments were used in the sample; this comparison was based on the assumption that although not all parasites are equally detrimental, immune responses have evolved and are maintained to cope with debilitating parasites. Hence stronger effects were predicted for intensity of immune response.

META-ANALYSIS

We used Pearson's correlation coefficient as a measure of effect size for the relationship between the expression of secondary sexual characters and the intensity of parasitism or measure of immune function. If the original reports did not provide a correlation coefficient, we transformed the statistics into a correlation coefficient using the formulas for transformation given by Rosenthal (1991:19). These correlation coefficients were subsequently converted by means of Fisher's transformation to Z_r -values, on which all subsequent analyses were performed. We adjusted this measure of effect size for sample size by using $(N - 3)$ as a factor (Rosenthal 1991:27–28), based on the assumption that a larger sample size should provide more reliable estimates of the relationship between the factors being analysed. We tested for an overall effect using the mean sample-size-adjusted effect size after z -transformation to test whether it differed significantly from zero (Rosenthal 1991), using the equation

Mean weighted $Z_r = \sum (N_j - 3) Z_{rj} / \sum (N_j - 3)$, where Z_{rj} is the z -transformed effect size for analysis unit j . For the study of effect size at the level of species, we used the Z_r values directly if only one study was available, or the mean Z_r of the samples weighted by $(N - 3)$ in the case of multiple studies, because studies would have dissimilar sample sizes. The mean weighted Z_r values were tested against the null hypothesis of no effect by examining the significance of their associated r 's.

We subsequently calculated an estimate of heterogeneity in effect sizes among samples, using the formula provided by Rosenthal (1991: 73–74)

$$\chi^2 = \sum (N_j - 3) (Z_{rj} - \text{mean } Z_r)^2,$$

which has a χ^2 -distributed with $K - 1$ degrees of freedom, where K is the number of analysis units (e.g., studies or species).

Given statistically significant heterogeneity, we proceeded by testing for the effects of five different potential explanatory variables by calculating a standard normal deviate as suggested by Rosenthal (1991:79–84)

$$\sum \lambda_j Z_r / \left(\sqrt{\sum (\lambda_j^2 / w_j)} \right),$$

where λ_j is the contrast weight determined by a hypothesis of the analysis unit (samples, species), chosen so that the sum of λ_j 's equals zero. λ_j is defined as $1 / N_j$, the weighting factor, where N_j is the number of samples in each of the j categories. w_j is the inverse of the variance of the effect size for the analysis unit. The 95% confidence intervals were calculated according to Hedges and Olkin (1985:230–232). As indicated above, there is little basis for believing the data biased by lack of publication of negative findings. The fail-safe number of studies X needed to nullify an effect was calculated, after Rosenthal (1991:104), as

$$X = (\sum Z_j)^2 / 2.706 - K,$$

where $Z_j = Z_{rj} \sqrt{(N_j - 3)}$, and K is the number of analysis units. The fail-safe number estimates the number of studies with null results that are unknown to us because of being rejected by journals or remaining unpublished by scientists. The fail-safe number also provides an estimate of the number of future studies needed to change a significant effect to a nonsignificant one. We used one-tailed tests when the direction of a relationship for a moderator variable was predicted; in other cases, two-tailed tests were used.

AVERAGE EFFECT SIZES

From the literature survey, 50 studies yielded 69 separate comparisons of the relationship between the expression of secondary sexual characters and parasite load or immune response, representing 39 species (Table 1). Birds were the best represented group, with 27 species; there were 5 species of fishes, 2 species of mammals, 3 species of reptiles, and one species each of amphibians and insects (Table 1). Using the values of all effect sizes ($N = 69$),

TABLE 1

Information on effects of parasites and immune function on the expression of secondary sexual characters of their hosts

Species	Parasite type	Effect size (r)	N	Trait	Parental Care	Data	Reference
Insects							
<i>Gryllus pennsylvanicus</i>	END	−0.190	34	BEH	0	EXP	1
Fishes							
<i>Gasterosteus aculeatus</i>	END	0.480	35	SEC	♂	OBS	2
<i>Gasterosteus aculeatus</i>	END	0.197	45	SEC	♂	OBS	3
<i>Gasterosteus aculeatus</i>	END	−0.457	26	SEC	♂	EXP	4
<i>Gasterosteus aculeatus</i>	END	0.025	97	SEC	♂	OBS	5
<i>Poecilia reticulata</i>	ECT	−0.399	48	SEC	0	EXP	6
<i>Poecilia reticulata</i>	ECT	−0.939	13	BEH	0	EXP	7
<i>Rutilus rutilus</i>	END	−0.314	8	SEC	0	OBS	8
<i>Salvelinus alpinus</i>	IMM	−0.319	20	SEC	0	OBS	9
<i>Salvelinus alpinus</i>	END	−0.227	20	SEC	0	OBS	9
<i>Thalassoma bifasciatum</i>	END	0.030	63	SEC	0	OBS	10
Amphibians							
<i>Hyla versicolor</i>	END	0.026	127	BEH	0	OBS	11
Reptiles							
<i>Cnemidophorus arubensis</i>	BLD	0.272	174	SEC	0	OBS	12
<i>Sceloporus occidentalis</i>	BLD	−0.770	16	BEH	0	EXP	13
<i>Psammodromus algirus</i>	IMM	−0.284	14	SEC	0	EXP	14
Birds							
<i>Agelaius phoeniceus</i>	END	0.135	45	SEC	♂ ♀	OBS	15
<i>Agelaius phoeniceus</i>	ECT	−0.309	45	SEC	♂ ♀	OBS	15
<i>Agelaius phoeniceus</i>	BLD	0.030	45	SEC	♂ ♀	OBS	15
<i>Agelaius phoeniceus</i>	ECT	0.180	22	BEH	♂ ♀	OBS	15
<i>Agelaius phoeniceus</i>	ALL	0.450	22	BEH	♂ ♀	OBS	15
<i>Carduelis cannabina</i>	IMM	−0.900	5	SEC	♂ ♀	OBS	16
<i>Carduelis flammea</i>	BLD	0.005	76	SEC	♂ ♀	OBS	17
<i>Carpodacus mexicanus</i>	ECT	−0.298	75	SEC	♂ ♀	OBS	18
<i>Carpodacus mexicanus</i>	END	−0.259	75	SEC	♂ ♀	OBS	18
<i>Centrocercus urophasianus</i>	BLD	−0.066	110	BEH	♀	OBS	19
<i>Centrocercus urophasianus</i>	BLD	0.125	21	BEH	♀	OBS	20
<i>Columba livia</i>	ECT	−0.327	26	BEH	♂ ♀	EXP	21
<i>Dendrocopus major</i>	IMM	−0.450	15	SEC	♂ ♀	OBS	16
<i>Falco sparverius</i>	END	−0.358	20	BEH	♂ ♀	EXP	22
<i>Falco sparverius</i>	BLD	−0.221	40	SEC	♂ ♀	OBS	23
<i>Falco tinnunculus</i>	BLD	0.224	84	SEC	♂ ♀	OBS	24
<i>Ficedula hypoleuca</i>	BLD	0.110	40	SEC	♂ ♀	OBS	25
<i>Ficedula hypoleuca</i>	BLD	0.104	141	SEC	♂ ♀	OBS	26
<i>Gallus gallus</i>	END	−0.186	63	SEC	♀	EXP	27
<i>Gallus gallus</i>	IMM	−0.390	62	SEC	♀	OBS	28
<i>Gallus gallus</i>	IMM	−0.320	81	SEC	♀	EXP	29
<i>Hirundo rustica</i>	ECT	−0.343	83	SEC	♂ ♀	OBS	30
<i>Hirundo rustica</i>	ECT	−0.425	60	SEC	♂ ♀	EXP	31
<i>Hirundo rustica</i>	ECT	−0.108	194	SEC	♂ ♀	OBS	32
<i>Hirundo rustica</i>	IMM	0.100	91	SEC	♂ ♀	OBS	32

TABLE 1 *continuation*
Information on effects of parasites and immune function on the expression of secondary sexual characters of their hosts

Species	Parasite type	Effect size (r)	N	Trait	Parental Care	Data	Reference
(Birds)							
<i>Hirundo rustica</i>	ECT	−0.091	120	SEC	♂ ♀	OBS	33
<i>Hirundo rustica</i>	ECT	−0.190	305	SEC	♂ ♀	OBS	34
<i>Hirundo rustica</i>	IMM	−0.117	42	BEH	♂ ♀	OBS	35
<i>Hirundo rustica</i>	IMM	−0.480	36	BEH	♂ ♀	OBS	35
<i>Hirundo rustica</i>	ECT	−0.628	22	BEH	♂ ♀	EXP	36
<i>Hirundo rustica</i>	IMM	−0.990	6	SEC	♂ ♀	OBS	16
<i>Meleagris gallopavo</i>	ECT	−0.008	10	SEC	♀	OBS	37
<i>Meleagris gallopavo</i>	END	−0.137	9	SEC	♀	OBS	37
<i>Meleagris gallopavo</i>	BLD	−0.047	6	SEC	♀	OBS	37
<i>Oenanthe leucura</i>	ECT	−0.289	45	SEC	♂ ♀	OBS	38
<i>Parotia lawesii</i>	BLD	−0.316	13	BEH	♂ ♀	OBS	39
<i>Parus major</i>	BLD	0.207	45	SEC	♂ ♀	OBS	40
<i>Parus major</i>	IMM	0.016	45	SEC	♂ ♀	OBS	40
<i>Passer domesticus</i>	IMM	−0.220	70	SEC	♂ ♀	OBS	41
<i>Passer domesticus</i>	IMM	−0.870	26	SEC	♂ ♀	OBS	16
<i>Pavo cristatus</i>	ECT	0.302	17	SEC	♀	OBS	42
<i>Phasianus colchicus</i>	IMM	−0.240	107	SEC	♀	OBS	43
<i>Ptilonorhynchus violaceus</i>	ECT	−0.297	39	SEC	♀	OBS	44
<i>Quiscalus quiscula</i>	ECT	0.180	156	SEC	♂ ♀	OBS	45
<i>Quiscalus quiscula</i>	BLD	0.220	178	SEC	♂ ♀	OBS	45
<i>Remiz pendulinus</i>	ECT	−0.330	47	SEC	♂ ♀	OBS	46
<i>Sylvia atricapilla</i>	IMM	−0.940	6	SEC	♂ ♀	OBS	16
<i>Sylvia communis</i>	IMM	−0.900	5	SEC	♂ ♀	OBS	16
<i>Taeniopygia guttata</i>	ECT	0.103	52	SEC	♂ ♀	OBS	47
<i>Tetrao tetrix</i>	END	−0.580	22	SEC	♀	OBS	48
<i>Tetrao tetrix</i>	BLD	0.020	49	SEC	♀	OBS	48
<i>Turdus merula</i>	IMM	−0.900	110	SEC	♂ ♀	OBS	16
Mammals							
<i>Odocoileus virginianus</i>	END	−0.093	629	SEC	♀	OBS	49
<i>Rangifer tarandus</i>	END	0.001	40	SEC	♀	OBS	50

Parasite: ECT = ectoparasite, BLD = blood parasite, END = endoparasite, IMM = immune defense (response to immune challenge test, leukocyte concentration, or size of immune defense organ), ALL = all parasite taxa.

Trait: SEC = secondary sexual trait, BEH = sexual behavior.

Parental Care: ♀ = only female, ♂ ♀ = male and female, ♂ = only male, 0 = without parental care.

Data: EXP = experiment, OBS = observation.

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the magnitude of the overall mean effect size weighted by sample size was $Z_r = -0.124$, which equals a Pearson's r of -0.123 (C.I. [confidence interval] = $-0.152, -0.095$) and a probability (one-tailed) of $P < 0.001$. This result implies that individuals with few parasites or strong immune responses, or both, generally have the most extravagant secondary sexual characters. When considering species as units of analysis ($N = 39$), the overall mean effect size (weighted by sample size) was $Z_r = -0.125$, which equals $r = -0.124$ (C.I. = $-0.162, -0.087$) with $P < 0.001$. Therefore, the unit of observation for analysis does not change the overall conclusion.

The fail-safe number of studies (Rosenthal 1991:104) needed to nullify the average effect was calculated to be 2445 for all effect sizes and 900 when species were used as units of analysis. Since both these numbers are very large, we can conclude that it is unlikely that so many unpublished null studies exist. The general conclusion that parasites affect the expression of secondary sexual characters thus seems to be robust.

There was highly significant heterogeneity in effect sizes among samples ($\chi^2 = 501.23$, $df = 68$, $P < 0.001$) as well as species ($\chi^2 = 348.58$, $df = 38$, $P < 0.001$). This suggests that one or more moderator variables are involved in mediating differences in effect size, and we tested for the effects of such moderator variables.

MODERATOR VARIABLES

1. *Experiments vs. observations.* Experimental studies differ from observational ones by controlling for confounding variables and being able to demonstrate causal relationship. Hence we predicted a stronger effect size for experimental than for observational studies, and demonstrated a highly significant difference ($z = -4.498$, $P < 0.001$). The Pearson's r of the relationship between parasites and sexual traits was significantly more negative than the r 's for the relationship in observational studies. The weighted mean Z_r for experiments was -0.416 ($r = -0.394$, $N = 12$, C.I. = $-0.474, -0.307$) and for observations $Z_r = -0.095$ ($r = -0.095$, $N = 57$, C.I. = $-0.125, -0.064$).

2. *Effects of parasites on morphological signals vs. behavioral display.* If behavioral traits are

phenotypically more flexible on a shorter time scale, they should reflect current parasite status better than more rigid morphological signals that are developed over longer periods, such as once per year. But this was not the case ($z = 1.007$, $P = 0.156$). The weighted mean Z_r for secondary sexual morphological traits was -0.112 ($r = -0.111$, $N = 54$, C.I. = $-0.143, -0.080$) and for behavioral traits $Z_r = -0.206$ ($r = -0.203$, $N = 15$, C.I. = $-0.281, -0.122$).

3. *Ectoparasites vs. endoparasites.* If secondary sexual characters reflected direct fitness benefits in terms of parasite avoidance, we would predict that effect size should be larger for directly transmitted ectoparasites than for indirectly transmitted endoparasites. The ectoparasites had a significantly stronger negative influence on the expression of secondary sexual characters than internal parasites ($z = -3.694$, $P < 0.001$). The mean Z_r weighted by sample size was -0.178 for ectoparasites, corresponding to a Pearson's $r = -0.176$ ($N = 19$, C.I. = $-0.227, -0.123$). For endoparasites $Z_r = 0.003$ with $r = 0.003$, $N = 32$, C.I. = $-0.037, 0.043$). A qualitatively similar result was reported by Able (1996).

4. *Paternal care vs. no paternal care.* If females chose males whose sexual displays reliably signaled absence of parasites and therefore male ability to provide parental care, then we should expect parasite-mediated sexual selection to be more prominent in species with male parental care than in species lacking it. The relationship between parasites and sexual traits was not more strongly negative in species without paternal care ($z = -1.518$, $P = 0.064$). For species without male care, the mean $Z_r = -0.105$ ($r = -0.105$, $N = 25$, C.I. = $-0.152, -0.058$) and for species with paternal care the mean $Z_r = -0.136$ ($r = -0.135$, $N = 44$, C.I. = $-0.172, -0.098$).

5. *Parasites vs. measures of immune function.* Since many parasites are relatively benign to their hosts, we should expect effect sizes of the relationship between intensity of parasite infections and the expression of secondary sexual characters on average to be weaker than effect sizes based on measures of immune function. This was indeed the case, and the difference between the two categories of effects was statistically highly significant ($z = 6.972$, $P < 0.001$). The mean Z_r for the relationship be-

tween expression of secondary sexual characters and immune function was -0.465 , corresponding to $r = -0.434$, C.I. = -0.492 , -0.372 ($N = 17$), while the corresponding value for studies based on estimates of the intensity of parasite loads was $Z_r = -0.060$, $r = -0.060$, $N = 52$, C.I. = -0.092 , -0.028 .

If various moderators interact in the expression of secondary sexual characters, the inter-correlation could be statistically analysed. We investigated the effects of intercorrelations and interactions among variables in analyses of variance. Since it was impossible to make a fully saturated model with all two-way interactions, when both ectoparasite vs. endoparasite and parasites vs. immune function variables were factors, we made two separate analyses. The first analysis, which included behavior vs. morphology, observation vs. experiment, parasites vs. immune function, and absence vs. presence of paternal care, was statistically highly significant ($F = 299.73$, $df = 10, 58$, $r^2 = 0.49$, $P < 0.001$). Only one interaction (between parasites vs. immune function and paternal care) was marginally significant ($F = 4.93$, $df = 1, 58$, $P = 0.03$). We deleted the remaining two-way interactions to generate the simplest model that could account for the maximum amount of variance in the data set. This model revealed significant main effects of experiment vs. observation (stronger effects for experiments), parasite vs. immune function (stronger effects for immune function) and absence vs. presence of paternal care (stronger effects for absence of paternal care), with a significant interaction between parasite vs. immune function and absence vs. presence of paternal care (Table 2). Adjusted least squares mean values for this interaction revealed less importance of the immune response variable in species with no paternal care (Table 2).

In the second multivariate analysis, which included the variables behavior vs. morphology, observation vs. experiment, ectoparasites vs. endoparasites, and absence vs. presence of paternal care, the overall model was statistically highly significant ($F = 217.07$, $df = 10, 40$, $r^2 = 0.51$, $P < 0.001$), with a significant main effect for experiment vs. observation ($F = 12.06$, $df = 1, 40$, $P < 0.001$), but none of the interactions reached statistical significance ($P >$

0.24). This conclusion was not changed by deletion of the nonsignificant two-way interactions, since the main effect of experiment vs. observation remained significant ($F = 14.31$, $df = 1, 46$, $P < 0.001$), while ectoparasite vs. endoparasite became marginally significant ($F = 4.41$, $df = 1, 46$, $P = 0.04$). Hence, the multivariate analyses revealed independent strong effects of observation vs. experiment, parasites vs. immune function, and absence vs. presence of paternal care, and a strong interaction between parasites vs. immune function and paternal care (Table 2).

MODELS OF PARASITE-MEDIATED SEXUAL SELECTION

Effects of parasites on the expression of secondary sexual characters of their hosts may be explained by the contagion avoidance hypothesis, the efficient parent hypothesis, and the parasite-resistance hypothesis (the original hypothesis suggested by Hamilton and Zuk 1982). There is no general agreement about the relative importance of these three hypotheses, and they are not necessarily mutually exclusive. A recent paper by Able (1996), however, suggested that more significant relationships between parasite intensity and expression of secondary sexual characters have been found for parasites that could be transmitted directly from male to female.

We used a meta-analytic approach to test some simple predictions arising from the three hypotheses. This approach is superior to vote-counting methods as adopted by Able (1996), because not only the direction of the effect but also its magnitude is considered. Furthermore, effect sizes can be adjusted for differences in sample size, which is reasonable given that an effect based on a very large sample is more likely to be reliable than one based on just a few observations, and independent effects of multiple variables and their interactions can be evaluated.

Ectoparasites that can be directly transmitted between adults during sexual contact had a significantly larger average effect on the expression of secondary sexual characters than did endoparasites that cannot be transmitted by contact. This finding is consistent with the conclusion of Able (1996), but contrary to that

TABLE 2
Analysis of variance for *z*-transformed effect sizes with significant two-way interactions

Variable	df	Mean square	<i>F</i>	<i>P</i>
Behavior vs. morphology	1	0.068	0.33	0.57
Observation vs. experiment	1	2.113	10.22	0.002
Parasites vs. immune function (PI)	1	2.377	11.49	0.001
Paternal care (PC)	1	1.493	7.21	0.009
PI x PC	1	2.119	10.24	0.002
Error	63	0.207		

F = 247.34, *df* = 5,63, *r*² = 0.40, *P* < 0.001

Adjusted least squares means

	Mean (SE)	<i>N</i>
Parasites–Paternal care	–0.246 (0.097)	32
Parasites–No paternal care	–0.314 (0.109)	20
Immune response–Paternal care	–1.176 (0.152)	12
Immune response–No paternal care	–0.332 (0.215)	5

of Hamilton and Poulin (1997). None of the hypotheses are excluded by this finding.

A second finding of the meta-analysis was the absence of a difference in negative effect on the expression of secondary sexual characters for species with or without male parental care. In fact, there was a tendency for effects to be stronger for species without male parental care. This conclusion changed in the multivariate analyses, however, because of intercorrelations among factors (Table 2). The multivariate analyses revealed a stronger effect for immune function in species with male parental care (Table 2). This observation supports the efficient parent hypothesis as a general explanation for parasite-mediated sexual selection, but is also consistent with the contagion avoidance and the parasite-resistance hypotheses. A final observation related to the three hypotheses is the stronger negative relationship between expression of secondary sexual characters and measures of immune function as compared to intensity of parasite infection. Since parasite resistance often is genetically variable (e.g., Wakelin and Blackwell 1988; Wakelin 1996), this observation is consistent with the parasite-resistance hypothesis. Obvi-

ously, the tests of parasite-mediated sexual selection based on the meta-analysis are only tests of general effects, which does not rule out the possibility that other mechanisms may be important in particular cases.

GENERAL DISCUSSION

Any host species is affected by numerous species of parasites during its lifetime, and it seems unreasonable to expect that the expression of sexual displays or secondary sexual characters should be related to the intensity of any one parasite species. Since the intensities of infections for different species of parasites are not generally positively correlated, we should therefore expect the relationship between secondary sexual displays and parasite intensity to be very heterogeneous across host-parasite systems. This was indeed the case.

The second prediction relating to this issue is that measures of antiparasite defense rather than parasite burden may be more directly related to the expression of secondary sex traits. Immune system defenses can be inferred to have evolved to cope with the most debilitating parasites that affect a particular host species cur-

rently and in the recent past. We should therefore expect to find more consistent relationships between measures of immune function and the exaggeration of secondary sexual character, as compared to measures of parasite intensity. The immune system of vertebrates is complex, consisting of cell-mediated immunity, humoral immunity and peripheral lymphoid tissue (Toivanen and Toivanen 1987; Wakelin 1996). The above prediction assumes that different measures of immunocompetence should be positively correlated. The only evidence available from free-living organisms to test this prediction are recent studies of immunocompetence in peacocks *Pavo cristatus*, showing a positive relationship between induced T-cell and B-cell responses to a standardized immune challenge test (Møller and Petrie, unpublished data), and a study of barn swallows *Hirundo rustica* showing a trade-off between B-cell and T-cell response (Saino and Møller, unpublished data). Our meta-analysis supported the prediction that the expression of secondary sexual characters was more strongly related to measures of immune function than to measures of parasite load by showing a highly significant negative relationship for immune function measures and a weak negative effect for parasite loads.

The finding that measures of host immune defense are more strongly negatively related to secondary sexual characters than parasite loads is consistent with the conclusions of three other comparative studies. First, a pairwise comparative study of the relative size of two immune defense organs (bursa of Fabricius and spleen, corrected for the effects of body size) and sexual dichromatism in birds was used to test the prediction that sexually dichromatic species, which are supposedly affected by particularly virulent parasites, should have evolved larger immune defense organs than closely related monochromatic species (Møller et al. 1998a). These results are consistent with models of parasite-mediated sexual selection based on female choice of resistant males as mates (Hamilton and Zuk 1982). Alternatively, sexual selection may be the driving force behind the evolution of increased virulence of parasites in host species. Sexual selection arises from a skewed mating success of males, giving rise to multiple contacts between

very attractive males and females, and therefore indirectly with the mates of these females. Hence, sexually selected species such as birds with a high frequency of extra-pair paternity (as opposed to species with little or no extra-pair paternity) should be characterized by frequent horizontal transmission of parasites and a high frequency of multiple infections with parasites of different genetic origin; these are factors supposed or known to increase parasite virulence (Bull 1994).

Once sexual selection occurs, it will increase female propensity to discriminate among males, but simultaneously fuel the need for further discrimination due to increase in virulence, which will favor stronger female discrimination and evolution of even more virulent parasites. Frequency-dependent selection on parasite virulence and host resistance genes, as envisaged by Hamilton and Zuk (1982), could play an important role in such species with intense sexual selection. The pairwise comparative study of Møller et al. (1998a) demonstrated significantly larger bursae of Fabricius and spleens in the sexually dichromatic species, even after controlling for potentially confounding variables. If intraspecific variation in organ size reflects current health status, as suggested by numerous studies, we expect the degree of plumage elaboration to be negatively related to the size of immune defense organs, particularly in sexually dichromatic species. This was also the case in the study by Møller et al. (1998a).

The second study investigated the relationship between immune function, extra-pair paternity and sexual dichromatism in birds (Møller 1997). Extra-pair paternity is a direct measure of the intensity of sexual selection because female copulations with multiple males generally result in an increase in variance in male reproductive success (Møller 1998). Sexual dichromatism is known to be related directly to current sexual selection, as demonstrated by numerous observational and experimental studies (reviewed in Andersson 1994). Sexual dichromatism and extra-pair paternity are strongly positively associated across species of birds (Møller and Birkhead 1994). However, the study by Møller (1997) demonstrated that extra-pair paternity was independently and strongly positively related to both

the degree of sexual dichromatism and the relative size of the spleen. If sexual dichromatism has evolved as a signal of immunocompetence, then we should predict that sexual dichromatism should be unrelated to measures of immune function once sexual selection has been accounted for. This was also the case.

The third study demonstrated a reduction in the size of male immune defense organs, in particular among reproductive individuals (Møller et al. 1998b). The magnitude of this reduction in the size of male immune defense organs was directly related to the intensity of sexual selection, estimated from the frequency of extra-pair paternity in different species of birds (Møller et al. 1998b). Thus intense sexual selection reduced the ability to raise an immune response, as determined from the size of male immune defense organs. Hence, the present study and those by Møller

(1997) and Møller et al. (1998a,b) have demonstrated consistent associations between sexual selection and host immune function. Parasites may thus have a general impact on host sexual selection, even though this is most readily assessed from measures of responses of the host immune system.

In conclusion, we believe that the general relationship between measures of host immune function and the expression of sexual displays suggests that overall, parasites play an important role in host sexual selection, at least in vertebrates.

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