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Reappraisal of the Interspecific Prediction of Parasite-mediated Sexual Selection: Opportunity Knocks

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Parasite-mediated sexual selection (PMSS) has been hypothesized to occur when individuals choose mates on the basis of parasite-indicative secondary sexual traits in order to acquire "good-genes" for parasite resistance, protection from parasite transmission, or healthy mates for assistance in parental care. Interspecific studies of PMSS test the prediction that parasite load and the extent of sexual selection, or "showiness", are positively correlated across host species. The assumption inherent in this prediction is that larger parasite loads cause more intense sexual selection because larger loads have a greater effect on the mean fitness of host populations. This assumption is invalid on theoretical grounds: selection is not governed by mean fitness, but by variance in relative fitness, or the "opportunity for selection". In this paper we model the potential influence of parasites on host fitness and examine the relationship of two measures of parasite load, prevalence and intensity, to the opportunity for parasite-mediated selection (I). Our results indicate that prevalence and intensity covary with I in some cases, but not others, depending on the precise effect of the parasite on host fitness and how this effect varies across host species. On the basis of these results, the interspecific prediction of PMSS is not necessarily warranted. The relationship of parasites to sexual selection, and hence the predicted relationship of parasites to showiness, depends on the nature of host–parasite interactions.

Introduction

Freeland (1976) was the first to propose that sexual selection could be influenced by parasites: "If a female can induce a state of behavioral and/or nutritional stress among group males, she has a high probability of exposing the presence of individuals with low genetic resistance to diseases, thus allowing her to mate with the more resistant males". In a more elaborate model of parasite-mediated sexual selection (PMSS hereafter), Hamilton (1982) and Hamilton & Zuk (1982) suggested that females choose genetically resistant males on the basis of male secondary sexual traits that are fully expressed only by parasite-free individuals. Examples of such traits include brightly colored skin or feathers subject to parasite-induced fading and energetically expensive display behavior that parasitized individuals cannot perform.

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According to the Hamilton–Zuk model, females assess resistance in males on the basis of elaborate traits that are costly to maintain, i.e. parasite-indicative "handicap" traits (Zahavi, 1975). Read (1990) reviewed the relationship of the Hamilton–Zuk model to other handicap models of sexual selection.

Both the Freeland and Hamilton–Zuk scenarios are "good-genes" views of sexual selection; they assume that choice of a parasite-free male benefits females indirectly through the inheritance of resistance by offspring. (We discuss PMSS in terms of female choice, but parasites may also influence sexual selection via male choice or male–male competition; Clayton, 1991.) Parasite-based mate choice could also lead to more direct fitness benefits. Females might choose parasite-free males to protect themselves and/or their offspring from parasite transmission (Freeland, 1976; Borgia & Collis, 1989, 1990; Clayton, 1990; Hamilton, 1990) or if they rely on healthy males for assistance in parental care (Hamilton, 1990; Read, 1990; Kirkpatrick & Ryan, 1991). These scenarios differ from good-genes models in that they require no assumptions about the genetics of host–parasite interactions. Like good-genes models, however, they predict that female preference will occur on the basis of parasite-indicative traits. The arguments presented in this paper are relevant to both good-genes and direct-benefit models of PMSS.

Empirical studies of PMSS have adopted two approaches: (1) experimental or correlational studies within host species, and (2) comparative studies across host species or higher taxa. These different approaches are derived from the original predictions of Hamilton & Zuk (1982): "Our hypothesis is contradicted if *within* a species preferred mates have most parasites; it is supported if *among* species those with most evident sexual selection are most subject to attack by debilitating parasites" (italics theirs). Male parasite load and mating success are predicted to covary negatively within species [Fig. 1(a)], whereas parasite load and the extent of sexual selection, or "showiness" (Pruett-Jones *et al.*, 1990, 1991), should covary positively

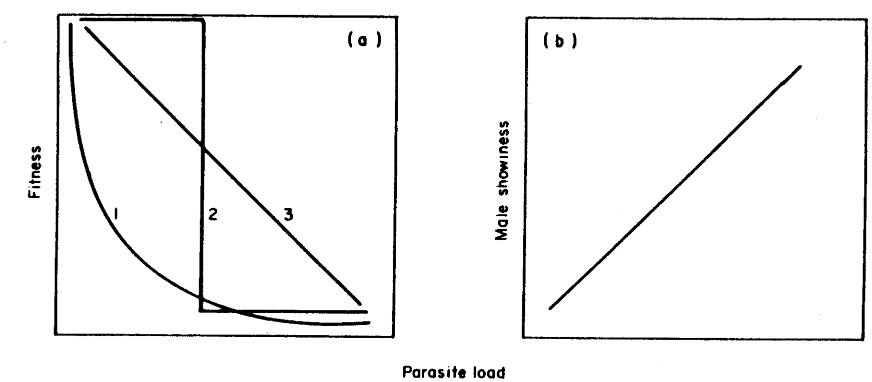

Fig. 1. Qualitative predictions of parasite-mediated sexual selection within host species (a) and across host species (b). Three conceivable relationships between parasite load and host fitness within species are shown: (1) a geometric function, (2) a step function, or (3) an arithmetic function.

across species [Fig. 1(b)]. Although Read (1988, 1990) reviewed the theoretical justification for within-species tests of PMSS, the rationale for interspecific tests has not been discussed in detail. In this paper we argue that the prediction of interspecific correlations between parasite load and showiness is poorly motivated from both an evolutionary and a parasitological perspective, and is erroneous in specific cases.

Many interspecific studies of PMSS have been conducted (Hamilton & Zuk, 1982; Read, 1987; Ward, 1988; Garvin, 1989; Read & Harvey, 1989*a*; Scott & Clutton-Brock, 1989; Ward, 1989; Read & Weary, 1990; Pruett-Jones *et al.*, 1990, 1991; Harvey *et al.*, 1991; Cabana & Chandler, 1991; Johnson, 1991; Weatherhead *et al.*, 1991; Zuk, 1991). The results of these studies are mixed (Read, 1990; Møller, 1990) and controversial (Harvey & Partridge, 1982; Cox, 1989; Hamilton & Zuk, 1989; Read & Harvey, 1989*b*; Zuk, 1989). All test the prediction that, across host taxa, showiness is correlated with parasite load. This prediction is based on the assumption that the intensity of sexual selection is correlated with parasite load. Why selection intensity should be correlated with load has not been fully addressed, however. The implicit assumption of the interspecific prediction is that larger parasite loads cause more intense sexual selection because larger loads have a greater effect on the mean fitness of host populations. In other words, the prediction can be interpreted to assume that the intensity of sexual selection is a function of relative mean fitness. This assumption is invalid; selection is not governed by mean fitness but by *variation* in fitness. Selection requires variation on which to act: "When there is no variance in fitness, there can be no selection; when there is a large variance in fitness, there is great opportunity for selection" (Arnold & Wade, 1984). Thus, the opportunity for selection in a host population comprised solely of individuals with large parasite loads may be less than that for a population comprised of individuals with both large and small loads, regardless of the fact that the former population has a larger mean load. Haskell (1991) has independently raised this point.

To illustrate, imagine an array of host species in which mean parasite load is positively correlated with host population density, but the variance in load is negatively correlated with density. This scenario is likely for parasites transmitted more efficiently among individuals in dense populations (Anderson & May, 1982). Now imagine, for the same set of species, that mean parasite load is also positively correlated across species with the elaboration of some parasite-indicative trait, but only because the cost of trait expression reduces the host's ability to resist parasites (Read, 1990). In this case, even though the interspecific prediction of PMSS (correlation of load and showiness) is supported, the greatest opportunity for PMSS will occur in species with the smallest parasite loads, not in species with the largest loads. The former have the greatest variation in load and thus the greatest variance in relative fitness (assuming that parasite load determines fitness—see Discussion), which is the opportunity for selection by definition (Crow, 1958; Arnold & Wade, 1984; Wade, 1987).

The opportunity for any form of parasite-mediated selection, not restricted to sexual selection, will be a function of a two chief parameters: (1) the average effect of an individual parasite on its host, and (2) the distribution of parasites among hosts in a population. To examine the explicit relationship of these parameters to the opportunity for parasite-mediated selection we constructed two theoretical models presented below. Before introducing these models it is necessary to review several definitions and concepts.

Parasitological Background

We adopt a broad-sense definition of parasite that includes viruses, fungi, bacteria, protozoa, helminths, and arthropods (May, 1985). We use parasite "load" as a generic phrase encompassing two explicit measures of parasite abundance: "prevalence", the proportion of individuals in a host population that is parasitized; and "intensity", the number of parasites harbored by an individual host (Margolis *et al.*, 1982). "Mean intensity" is the average number of parasites across all individual hosts sampled, including uninfected individuals. Note that parasite intensity should not be confused with the concept of selection intensity (Arnold & Wade, 1984).

Parasites can be divided into two classes: microparasites and macroparasites (Anderson & May, 1979, 1982; Rollinson & Anderson, 1985). Microparasites (e.g. viruses and bacteria) are small and have short generation times compared to macroparasites (e.g. helminths and arthropods). Microparasites undergo rapid reproduction and induce acquired immunity in hosts, which may last a lifetime. Because intensities of microparasites increase rapidly upon infection, hosts generally fall into three discrete classes: uninfected but susceptible, infected, and recovered with immunity. Microparasite prevalence is potentially a useful index of host fitness because it distinguishes infected from uninfected hosts. (But note that susceptible vs. recovered-immune hosts cannot be distinguished using parasite load data alone, a problem that will be addressed later in the paper.)

In contrast, macroparasites tend to be larger and often require indirect reproduction in an intermediate host, or at least undergo more gradual direct reproduction. Macroparasites generally have a chronic effect on the host that is a function of parasite intensity; however, the precise relationship between parasite intensity and host fitness can be complicated. Parasites may feed on host tissue without reducing host survival or reproductive success (McLennan & Brooks, 1991). In cases where parasites do reduce host fitness, the relationship can be arithmetic, geometric, or a step function [Fig. 1(a)]. In the arithmetic and geometric cases, macroparasite intensity is potentially a useful index of host fitness. In the case of a step function, however, hosts supporting below-threshold intensities will have the same relative fitness as non-parasitized individuals, making intensity a less reliable index of host fitness.

The labels microparasite and macroparasite are broad generalizations that reflect the ends of a continuum rather than well-defined classes. Some parasites fall in the middle of this continuum. Many protozoa, for example, show demographic properties of microparasites, yet have effects on host fitness that depend on rates of infection (May, 1984). In such cases parasite prevalence may approximate host fitness, but data on parasite intensity may predict relative host fitness more accurately.

Models

OPPORTUNITY FOR MICROPARASITE-MEDIATED SELECTION

The influence of microparasites on the opportunity for host selection can be modeled assuming a simple binomial distribution; *viz* hosts are either parasitized (infected class) or non-parasitized (susceptible and recovered-immune classes). Let W_0 be the fitness of non-parasitized individuals, W_1 the fitness of parasitized individuals, p the probability of being parasitized (parasite prevalence), and $1-p$ the probability of not being parasitized.

Mean fitness of the population is given by

$$\bar{W} = W_0(1-p) + W_1(p) = W_0 - p(W_0 - W_1) \quad (1)$$

and variance in fitness by

$$\sigma_w^2 = (W_0 - W_1)^2 p(1-p). \quad (2)$$

If we let the proportional reduction in the fitness of parasitized hosts be

$$s = 1 - \frac{W_1}{W_0}, \quad (3)$$

then the opportunity for microparasite-mediated selection is given by

$$I = \frac{\sigma_w^2}{(\bar{W})^2} = \frac{s^2 p(1-p)}{(1-sp)^2}. \quad (4)$$

By differentiating I with respect to p, and setting the derivative equal to 0, it is possible to determine the prevalence at which the opportunity for selection is maximized:

$$p = \frac{1}{2-s}. \quad (5)$$

OPPORTUNITY FOR MACROPARASITE-MEDIATED SELECTION

The influence of macroparasites on the opportunity for host selection is more difficult to model. Because host fitness is presumed to be a continuous function of macroparasite intensity, the mean and variance in fitness will depend on the relative distribution of parasites among hosts. A general feature of macroparasites is their

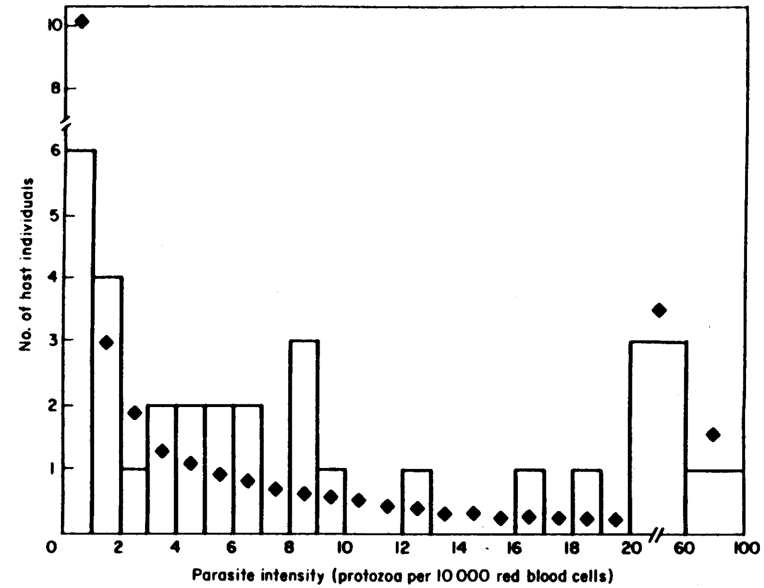

FIG. 2. Typical distribution of macroparasites among host individuals. Shown here is the combined distribution of *Haemoproteus* and *Plasmodium* blood protozoa (open bars) among 30 individuals of the Superb Bird of Paradise (*Lophornia superba*) (Pruett-Jones *et al.*, unpublished), compared with expected values for the negative binomial distribution (solid diamonds). The observed values do not differ significantly from the expected distribution ($\chi^2 = 6 \cdot 56$, df. $= 4$, $P > 0 \cdot 10$).

clumped or aggregated distribution among hosts, concordant with a negative binomial distribution (Fig. 2; Price, 1980; Anderson & Gordon, 1982). The following model therefore assumes a negative binomial distribution of parasites among hosts.

Let the fitness of an individual host be defined by

$$W(x) = (1 - s_1)^x \quad (6)$$

where s_1 is a constant representing the reduction in fitness attributable to a single parasite, and x is the number of parasites harbored by the host (Fig. 3). Note that for small values of s_1, $W(x)$ is approximated closely by $e^{-s_1 x}$.

Mean fitness is given by

$$\bar{W} = \sum_{x=0}^{\infty} f(x) W(x) \quad (7)$$

and the variance in fitness by

$$\sigma_w^2 = \sum_{x=0}^{\infty} f(x)[W(x)]^2 - (\bar{W})^2. \quad (8)$$

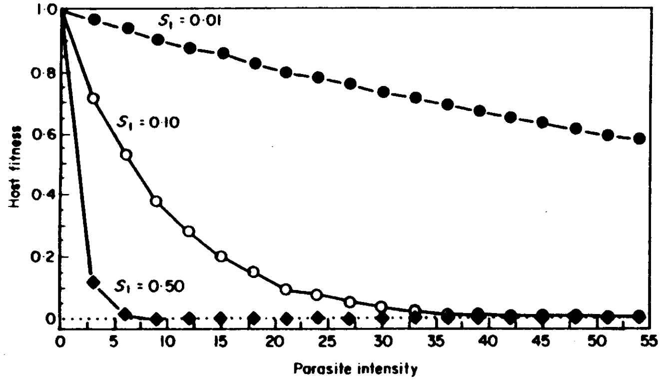

FIG. 3. Relationship of host fitness to parasite intensity. The curves depict the function $W(x) = (1 - s_1)^x$, where s_1 is the reduction in fitness attributable to a single parasite, and x is the number of parasites harbored by the host.

The negative binomial distribution (Johnson & Kotz, 1969) is described by the two parameters N and P, such that the frequency distribution of x is given by

$$f(x) = \binom{N+x-1}{N-1}\left(\frac{P}{Q}\right)^x Q^{-N} \tag{9}$$

where $Q = 1 + P$. Incorporating this distribution, mean host fitness in our model is given by

$$\bar{W} = \sum_{x=0}^{\infty} \binom{N+x-1}{N-1}\left(\frac{P(1-s_1)}{Q}\right)^x Q^{-N}. \tag{10}$$

If we define the substitution

$$\frac{P'}{Q'} = \frac{(1-s_1)P}{Q}, \tag{11}$$

where $Q' = 1 + P'$, then

$$P' = \frac{P(1-s_1)}{1+Ps_1}, \tag{12}$$

and the equation for mean host fitness becomes

$$\bar{W} = \left(\frac{Q'}{Q}\right)^N \sum_{x=0}^{\infty} \binom{N+x-1}{N-1}\left(\frac{P'}{Q'}\right)^x (Q')^{-N}$$
$$= \left(\frac{Q'}{Q}\right)^N = (1+Ps_1)^{-N}. \tag{13}$$

Similarly, if we let

$$\frac{P''}{Q''} = \frac{(1-s_1)^2 P}{Q} \tag{14}$$

then

$$P'' = \frac{(1-s_1)^2 P}{1+s_1(2-s_1)P}, \tag{15}$$

and variance in host fitness is given by

$$\sigma_w^2 = \left(\frac{Q''}{Q}\right)^N \sum_{x=0}^{\infty} \binom{N+x-1}{N-1}\left(\frac{P''}{Q''}\right)^x (Q'')^{-N} - (\bar{W})^2$$
$$= \left(\frac{Q''}{Q}\right)^N - \left(\frac{Q'}{Q}\right)^{2N} = [1+s_1(2-s_1)P]^{-N} - (1+s_1P)^{-2N}. \tag{16}$$

Finally, the opportunity for macroparasite-mediated selection is given by

$$I = \frac{\sigma_w^2}{(\bar{W})^2} = \left(\frac{(1+s_1P)^2}{1+s_1(2-s_1)P}\right)^N - 1. \tag{17}$$

Results

OPPORTUNITY FOR MICROPARASITE-MEDIATED SELECTION

The opportunity for microparasite-mediated selection [I; eqn (4)] is governed by parasite prevalence (p) and the relative effect of the parasite on host fitness (s). The relationship of prevalence to I is not linear (Fig. 4). I increases with prevalence to a maximum, then decreases at higher prevalence values. The effect of prevalence on I is most pronounced for virulent parasites that reduce host fitness by more than 50% ($s > 0{\cdot}5$). As virulence increases, the prevalence at which I reaches a maximum also increases [eqn (5), Fig. 4]. In the hypothetical case where parasitized hosts have zero fitness ($s = 1{\cdot}0$), I approaches infinity with increasing prevalence. At a given prevalence, I always increases with virulence, most notably at higher prevalences.

OPPORTUNITY FOR MACROPARASITE-MEDIATED SELECTION

The opportunity for macroparasite-mediated selection [I; eqn (17)] is governed by the constant s_1, and the parameters of the negative binomial distribution N and P. The mean of the negative binomial distribution is given by the product NP and the variance is given by the product $NP(1+P)$ (Johnson & Kotz, 1969). Although I increases with increasing values of s_1, N, or P, simple generalizations about how I will change with changes in the mean or variance in parasite intensity cannot be made.

To examine the relationship of I to mean parasite intensity in a specific case, we calculated I for each of 11 species of passeriform birds sampled for blood protozoa

TABLE 1

Abundance and distribution of Haemoproteus *and* Plasmodium *protozoa (combined data) from 11 species of passeriform birds in Papua New Guinea. Data summarized from Pruett-Jones* et al. (1991)

Host species	Host individuals†	Parasite prevalence	Mean intensity‡	Parameters§ N	P
Poecilodryas albispecularis	11	0·82	23·7	0·420	56·47
Melanocharis versteri	13	1·00	67·2	0·631	106·53
Melanocharis striativentris	26	1·00	61·7	1·144	53·95
Toxorhamphus poliopterus	56	0·25	8·0	0·621	128·87
Ptiloprora guisea	11	0·73	5·0	0·992	5·04
Melipotes fumigatus	12	0·83	21·5	0·338	63·52
Erythrura papuana	20	0·50	16·2	0·223	72·53
Amblyornis macgregoriae	110	0·82	20·7	0·260	79·65
Cnemophilus loriae	16	0·06	0·1	0·120	1·0
Epimachus meyeri	10	0·90	25·1	0·233	107·7
Lophorina superba	30	0·80	10·6	0·298	35·63

† Number of individuals sampled.
‡ Protozoa per 10 000 red blood cells.
§ Parameters of the negative binomial distribution.

selection is governed by variation in fitness, not mean fitness, taxa with more parasites will not necessarily experience greater selection. The opportunity for PMSS will be greatest in taxa with the most variable parasite loads, other things being equal. The extent to which PMSS actually covaries with variation in parasite load will depend

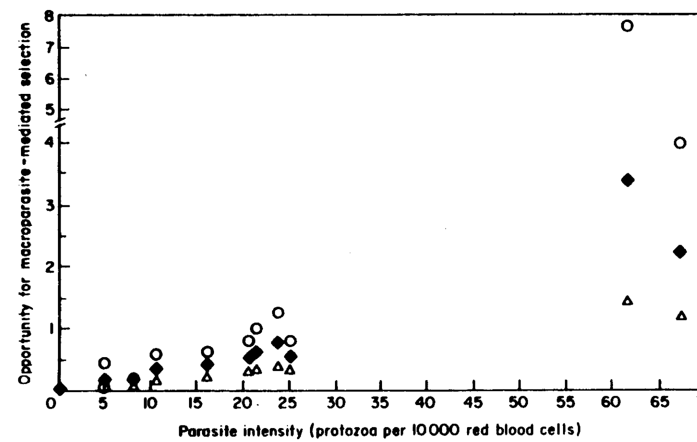

FIG. 5. Opportunity for macroparasite-mediated selection as a function of mean parasite intensity and the reduction in fitness attributable to a single parasite (s_1). For a given value of s_1, each point represents one of 11 species of passeriform birds sampled for blood protozoa (*Haemoproteus* and *Plasmodium*) in Papua New Guinea (Table 1). (○), $s_1=0·20$; (◆), $s_1=0·10$; (△), $s_1=0·05$.

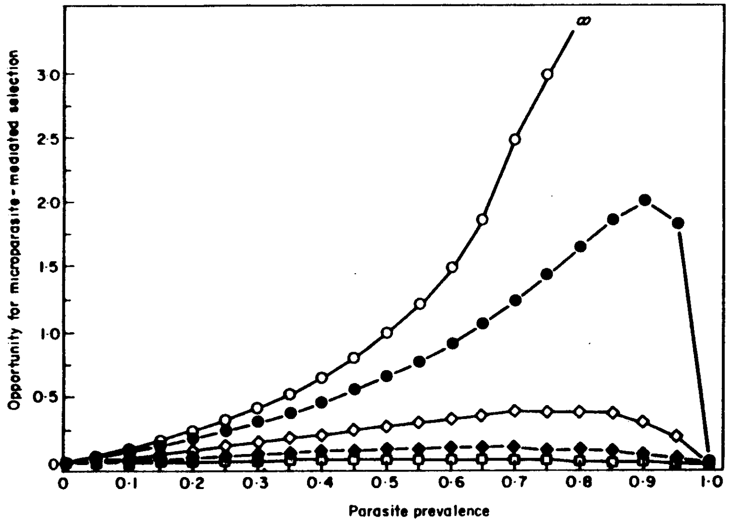

FIG. 4. Opportunity for microparasite-mediated selection as a function of parasite prevalence and the relative effect of the parasite on host fitness (s). (○), $s=1·0$; (●), $s=0·9$; (◇), $s=0·7$; (◆), $s=0·5$; (□), $s=0·3$.

(*Haemoproteus* and *Plasmodium*) in Papua New Guinea, and for which the distribution of parasites across individuals of each species was shown to fit a negative binomial distribution (Pruett-Jones *et al.*, 1991; Table 1). Values of N and P (Table 1) were calculated using the method of Williamson & Bretherton (1963) and then substituted into eqn (17) assuming different values of s_1. At all three values of s_1, I was highly correlated with mean intensity (Fig. 5, $r_s=0·96$ and $P<0·003$ for each value of s_1). For the set of 11 species, mean intensity was also highly correlated with parasite prevalence (Fig. 6, $r_s=0·936$, $P<0·005$), and prevalence was correlated with I at each value of s_1 ($P<0·003$ in each case).

We also examined the relationship of I to mean intensity when s_1 is not a constant, but varies inversely with mean intensity, as might be the case when avirulent strains of host-specific parasites achieve higher intensities, on average, than virulent strains. To approximate such a case, we recalculated s_1 for each species according to the function: $s_1=1/(\text{mean intensity}+1)$. For the set of 11 species there was a non-significant trend between I and mean intensity (Fig. 7, $r_s=0·527$, $P<0·10$).

Discussion

Interspecific studies of PMSS have generally tested the prediction that host taxa with more parasites show greater elaboration of secondary sexual traits. Because

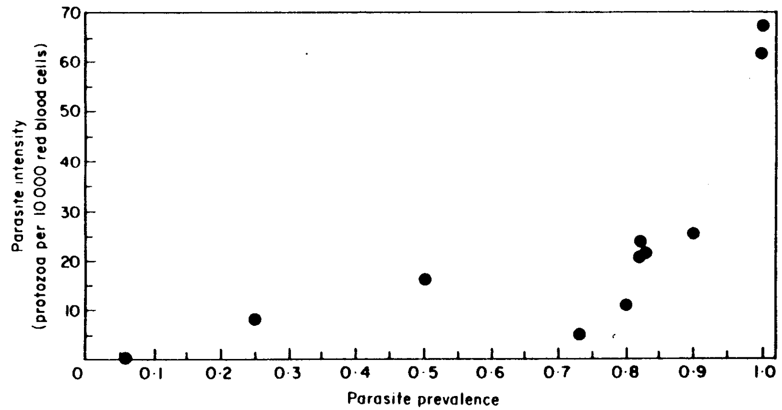

FIG. 6. Relationship of parasite prevalence to mean parasite intensity for species in Fig. 5.

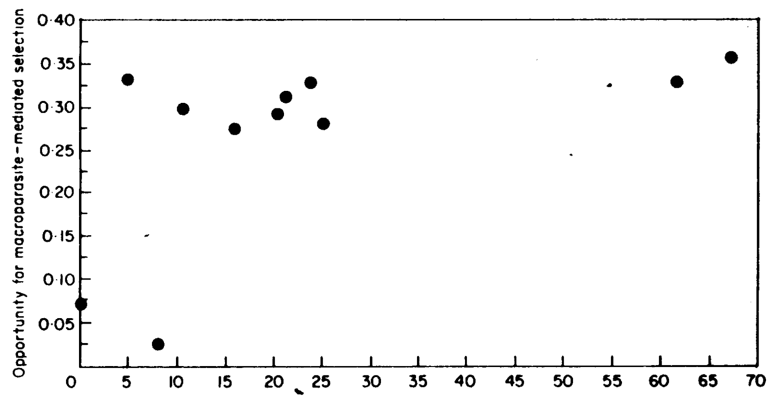

FIG. 7. Opportunity for macroparasite-mediated selection as a function of mean parasite intensity and the reduction in fitness attributable to a single parasite (s_1), where $s_1 = 1/(\text{mean intensity} + 1)$. Data as for Fig. 5; note change in scale of Y-axis.

on a number of interacting parameters, including the direct effect of parasites on male indicator traits and the relationship between trait expression and female preference. Few studies have measured these parameters in natural populations of hosts and parasites. For this reason, we modeled the opportunity for parasite-mediated selection in general, rather than the opportunity for PMSS in particular, thus minimizing the number of assumptions required for the models.

The opportunity for selection is not a measure of selection per se, but an index of the maximum intensity of selection that can occur in a particular population at a

given time (Crow, 1958; Arnold & Wade, 1984; Wade, 1987). If one assumes that populations with large opportunities are likely to experience greater selection than populations with small opportunities, the index can be used to predict the relative intensity of selection among populations. If one further assumes that evolutionary response is proportional to the opportunity for selection, the latter can be used to predict the relative elaboration of sexually selected traits, e.g. showiness. Arnold & Wade (1984) discuss the conditions necessary for these assumptions to be met.

Our results suggest that the relationship of parasites to sexual selection, and thus the predicted relationship of parasites to showiness, will depend on the exact nature of the host–parasite interaction. The relationship of prevalence to the opportunity for parasite-mediated selection (I) is not linear, but is a curvilinear function with a maximum at intermediate prevalence, except in cases where parasitized hosts have zero fitness (Fig. 4). Because most parasites do not reduce host fitness to zero, the prediction of a linear relationship between prevalence and showiness is not warranted for data sets with high prevalence values (e.g. Table 1). The linear prediction is more realistic for data sets lacking high prevalence values (e.g. Johnson, 1991; Weatherhead *et al.*, 1991).

In contrast, the relationship of intensity to I was highly correlated for the sample of New Guinea birds and their hematozoa, assuming the reduction in host fitness attributable to a single parasite (s_1) is constant among host species. This suggests that the interspecific prediction is justified for macroparasites with relatively constant virulence. When virulence was presumed to vary inversely with parasite intensity among species in our study, the relationship between intensity and I weakened. Thus the relationship of macroparasite intensity to host showiness may be influenced by how virulence varies among host species, which can be assessed by comparing the susceptibility of different hosts to experimental infections with the same parasite (e.g. van Riper *et al.*, 1986). More explicit predictions of the relationship between macroparasite intensity and I will require analyzing a larger data set than we have done here, with appropriate control for the fact that related species are not statistically independent points (Harvey & Pagel, 1991).

Although our approach suggests a firmer theoretical foundation for comparative analyses of PMSS, it does not address a major shortcoming of good genes models of PMSS: the assumption that parasite load is an accurate predictor of host resistance. Having a low parasite load does not necessarily mean that an individual is resistant to parasites; it may mean that the individual has never been exposed. Conversely, having a high load does not necessarily mean that an individual has low resistance; it may mean that the individual is resistant pending the activation of acquired immunity. Hence, exposure and immunity complicate the relationship of parasite load to resistance. What is needed for good genes tests is a measure of exposure that controls for evasive action on the part of the host (e.g. behavioral or immunological responses). Parasite load is an indirect measure at best, particularly in the case of microparasites (Read, 1990; Clayton, 1991).

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