Reviews

The Influence of Parasites on Host Sexual Selection

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In 1982 Hamilton and Zuk[1] proposed a provocative solution for the unexplained fact that the males of many species exhibit 'showy' traits such as brightly coloured plumage or vigorous courtship displays. They suggested that showy traits are fully expressed only by males who are resistant to parasites and that females examine such traits in order to choose resistant males as mates. Hamilton and Zuk's proposal has been the topic of extensive research and vigorous debate for nearly a decade. This article reviews the research, relevant criticisms and unanswered questions pertaining to the influence of parasites on sexual selection.

Parasitologists have long been interested in animal behaviour[2], whereas behaviourists have traditionally shown less interest in parasitology. Over the past decade this has changed. The influence of parasites on host behaviour is now a topic of widespread interest among behaviourists and parasitologists alike, as evidenced by the recent publication of two books devoted largely to the subject[3,4]. The most active area of research concerns the potential influence of parasites on host sexual selection.

Darwin[5] proposed the concept of sexual selection (Box 1) to explain the evolution of traits that are enigmatic in the light of natural selection. Secondary sexual traits such as the elaborate plumes, complex songs and vigorous displays of birds should be selected against, other things being equal, because showy traits are presumed to be energetically costly and conspicuous to predators. However, if such traits improve the ability of males to attract females, showiness can evolve by sexual selection regardless of its natural selection cost[6]. But why should females be attracted to showy males?

A novel explanation for female choice was proposed nearly a decade ago by Hamilton and Zuk[1], who suggested that females prefer showy males because showiness reveals genetic resistance to parasites and diseases. According to the Hamilton–Zuk hypothesis, females choose resistant males on the basis of secondary sexual traits whose full expression depends on health and vigour, such as brightly coloured plumage subject to fading, or vigorous courtship displays that parasitized individuals cannot perform (Fig. 1). Hamilton and Zuk argued that, over time, female choice leads to the elaboration of those traits that improve the ability of females to identify resistant males.

The Hamilton–Zuk idea is a 'good genes' model of sexual selection, which assumes that choice of resistant males benefits females indirectly through the inheritance of resistance by offspring. Parasite-based mate choice could also lead to more direct fitness benefits. Females might choose unparasitized males to protect themselves and/or offspring from parasite transmission (transmission avoidance model)[7–11], or to provide resources such as parental care (resource provisioning model)[11–13]. These models differ from the good genes model in that they require no assumptions about the genetics of host–parasite interactions. Like the good genes model, they predict that female choice will occur on the basis of parasite-indicative traits. All three models of parasite-mediated sexual selection (PMSS) predict that showy males benefit through enhanced mating success, but the models differ with respect to the postulated benefits acquired by choosy females.

PMSS has been addressed by several theoretical studies[6,11,13–19], including mathematical simulations of the good genes model. Neither the transmission avoidance model nor the resource provisioning model has been modelled quantitatively. Read[12] has reviewed some of the theoretical studies.

Empirical studies of PMSS have adopted one of two analytical approaches: (1) correlational or experimental analyses within host species or (2) comparative analyses across host species. These approaches follow from the original predictions of Hamilton and Zuk[1]: '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 therefore predicted to covary negatively within species, whereas parasite load and showiness are predicted to covary positively across species.

Although the focus of this review will be within-species tests of PMSS, across-species tests are important because they examine the scope of evolution by comparing the endpoints of numerous within-species evolutionary trajectories. The prediction that showiness and parasite load covary

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Box 1. Sexual Selection

Sexual selection occurs when the members of one sex mate disproportionately with members of the opposite sex on the basis of secondary sexual traits, ie. behaviours or structures other than the reproductive organs or gametes. Sexual selection can occur in two ways: as the result of *competition* among the members of one sex, usually males, for mates, or as the result of active *choice* by the members of one sex, usually females, for particular individuals of the opposite sex. These two modes of assortative mating may sometimes operate simultaneously, as when females actively choose to mate with a male victorious in combat against other males. The distinction between male competition and female choice is of heuristic importance because it emphasizes that patterns of assortative mating can be determined by the actions of males, females or both sexes. Males can also choose among females or both sexes can simultaneously exert choice. Few cases have been documented of direct competition between females for males.

The literature on parasites and sexual selection deals primarily with the influence of parasites on female choice, which is thus the focus of this review. However, it is important to note that parasites may also influence the outcome of male competition, as proposed by Freeland[8]: 'If a female can induce a state of behavioural 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.' Several studies have documented a negative relationship between parasites and male competitive ability[51], suggesting that parasites may influence sexual selection via male competition as well as female choice.

across species has been the subject of considerable controversy. Early tests largely supported the prediction[1,20–23], whereas later tests produced mixed results[24–31]. Comparative studies of PMSS have also been questioned recently on theoretical grounds[12,19].

PMSS is based on three general assumptions and three assumptions specific to the different models of PMSS. These assumptions and the data relevant to each are discussed below, followed by a brief discussion of criticisms and unanswered questions.

General assumptions of PMSS

Parasites reduce host fitness. Some parasites feed on host tissue without reducing either host survival or reproductive success. Yet only parasites that reduce host fitness will affect host evolution. Thus it is necessary to test the assumption of an effect on host fitness to assess the adaptive potential of parasite-mediated mate choice. Four correlational and five experimental studies report data relevant to this assumption. Although the results of the correlational studies are inconsistent, the results of four of the experimental studies support the assumption and the fifth study[32] provides conflicting results (Table 1). Thus, parasites have the potential to reduce host fitness, but this cannot be assumed to be the case.

Parasites alter male showiness. Parasites can conceivably alter two kinds of showy traits: indirect indicators and direct indicators[12]. Indirect indicators are traits correlated with a male's general condition, such as revealing handicaps[6] that can be fully expressed only by males in good condition (eg. elaborate plumes or displays). Because parasites are one of the factors influencing condition, females may use revealing handicaps as cues for the identification of unparasitized males. Direct indicators are traits that invariably reveal the presence of parasites. An example of this is the air sacs of male Sage Grouse, which highlight lesions made by chewing lice (Box 2).

For showy traits to be favoured by PMSS, they must improve the ability of females to identify parasitized males. If females can observe parasites directly, or if parasites affect a male's general appearance or behaviour (eg. if they cause general malaise), showiness is not apt to be favoured[9,12]. Furthermore, traits relevant to PMSS are likely to be those affected by parasites more than by other factors, at least during the host's mating season.

Eleven correlational and seven experimental studies report data relevant to the assumption that parasites alter male showiness. The results of the correlational studies are inconsistent or conflicting, whereas the experimental studies provide support for the assumption (Table 1). In each case the effect of the parasite was to reduce showiness (despite the fact that some correlational studies found a positive association between parasite load and showiness[33,34]). For example, Zuk *et al.*[35] showed a disproportionately negative effect of *Ascaridia* nematodes on the ornamental traits of Red Jungle Fowl (*Gallus gallus*) compared to non-sexually selected traits such as bill size: 'At sexual maturity infected roosters had duller combs and eyes, shorter combs and tail feathers, and paler hackle feathers than control roosters.'

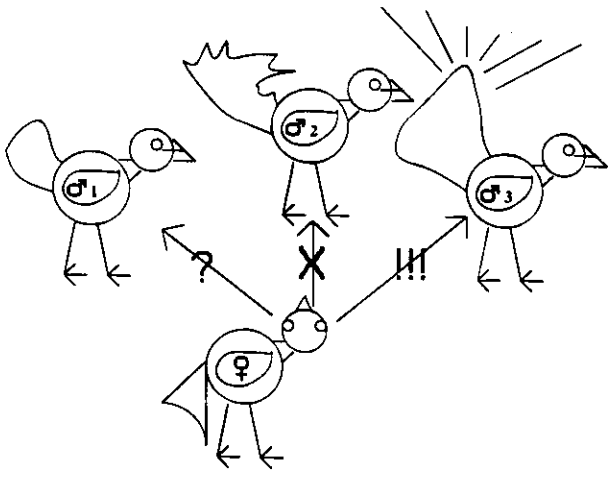

Fig. 1. Parasite-mediated mate choice. Given a choice of three males, the female rejects Male 1, whose tail is too inconspicuous to reveal his parasite load. The female also rejects Male 2 because the poor condition of his conspicuous tail reveals a large parasite load. The female chooses to mate with Male 3 because the good condition of his conspicuous tail reveals freedom from parasites.

Females choose less parasitized males. All models of PMSS predict that females will choose relatively unparasitized males as mates (or at least males able to cope with parasites). This assumption has been tested in nine correlational and five experimental studies. The correlational studies looked for an inverse relationship between parasite load and mating success in males. The results of eight of these studies are inconsistent; the ninth study reported conflicting results (Table 1).

Five experimental studies determined the mating preferences of females allowed to choose between parasitized and relatively unparasitized males. In all five cases the assumption was supported. The five studies also identified one or more showy traits that were altered by parasites in a way that could serve as mate choice cues for females. Two studies[36,37] performed manipulations showing that particular cues were actually used by females in mate choice. For example, Milinski and Bakker[36] showed that female stickleback fish discriminate against males formerly parasitized by *Ichthyophthirius* ciliates and that females recognize such males by the lower intensity of their breeding coloration. Thus, females can and do choose less parasitized males, at least under experimental conditions.

Table 1. Within-species tests of the major assumptions of parasite-mediated sexual selection

Host	Parasite	General assumptions			Specific assumptions			Refs
		Parasites reduce host fitness[a]	Parasites alter male showiness	Females choose less-parasitized male	Heritable resistance[b]	Direct transmission[c]	Diminished resource[d]	
Field cricket	Gregarines	–	Y[e,f]	Y[e,f]	–	–	–	52
	Gregarines	Y/N[g]	**N**[e]	**Y/N**[e]	–	–	–	32, 53–55
Fruit fly	Nematodes	Y[h]	Y[e,i]	Y[e,i]	–	–	–	56
Guppy	Nematodes	Y[h]	Y[g]	Y[e]	–	–	–	57,58
	Monogenea	Y[h]	Y[g]	–	y[h]	y[k]	–	57
Stickleback	Ciliates	Y[h]	Y[g]	Y[g]	y[h]	y[k]	y[l]	36
Tree frog	Nematodes	–	**N**[e]	**Y/N**[e]	–	–	–	43
	Monogenea	–	**N**[e]	**N**[e]	–	–	–	43
	Digenea	–	**N**[e]	**N**[e]	–	–	–	43
Spadefoot toad	Monogenea	**Y**[e]	–	**N**[e]	–	–	–	59
Whiptail lizard	Haematozoa	**N**[e]	**Y**[e]	–	–	–	–	33
Fence lizard	Haematozoa	Y[h]	**Y**[e]	–	–	–	–	60
Sage grouse	Haematozoa	–	N[e]	N[e]	–	–	–	45
	Protozoa/Bacteria?[m]	Y[e]	Y[e,g]	Y[e,g]	–	–	–	61,62
	Lice	–	Y[e]	Y[e,g]/N[g]	–	y[k]	–	61,62
Pheasant	Coccidia/ nematodes[n]	Y[e,g]	Y[e,g]	Y[e,g]	Y[g]	–	–	63[o]
	Lice	–	Y[e]	Y[e]	–	y[k]	–	63[o]
Jungle fowl	Nematodes	Y[g]	Y[g]	Y[g]	–	–	–	35
Rock dove	Lice	**Y**[g]	Y[g]	Y[g]	–	y[k]	–	7[p]
Barn swallow	Mites	**Y**[g]	**Y**[g]	**Y**[e]	**Y**[g]	**Y**[e]	–	37,39,42,49,50
	Lice	–	**Y**[e]	**Y/N**[e]	–[q]	y[k]	–	37,39,42,49,50
Blackbird	Haematozoa	**N**[e]	**Y**[e]	**N**[e]	–	–	–	64
Grackle	Haematozoa	–	**Y/N**[e]	–	–	–	–	65
	Lice	–	**Y/N**[e]	–	–	y[k]	–	65
Zebra finch	Mites	–	**N**[e]	–	–	y[k]	–	34
	Lice	–	**Y/N**[e]	–	–	y[k]	–	34
Bowerbird	Lice	–	**N**[e]	**Y**[e]	–[q]	y[k]	–	9,10,40
Bird of paradise	Haematozoa	–	**Y**[e]	**N**[e]	–	–	–	26

General: Y, assumption supported; N, assumption rejected; y, circumstantial support; –, untested/unlikely; bold, wild populations.

[a] Effect on components of host survival and/or postmating reproductive success.

[b] Good genes model: assumes genetic resistance to parasites by male.

[c] Transmission avoidance model: assumes direct transmission of parasites from male to female and/or offspring.

[d] Resource provisioning model: assumes diminished ability of male to provide resources for female and/or offspring.

[e] Correlational data.

[f] Females left the vicinity of parasitized males, apparently on the basis of overzealous mate-guarding behaviour by such males.

[g] Experimental data (parasite load manipulated).

[h] Citation of data published elsewhere.

[i] Parasitized males show lightly coloured, distended abdomens; this may not be a secondary sexual trait.

[j] Female choice possibly confounded by male–male competition.

[k] Ectoparasite with direct life cycle.

[l] Probable effect of parasite on male's ability to care for eggs and young after spawning.

[m] Males treated with an antibiotic strutted more and were preferred by females, but the authors[62] '... did not observe consistent differences between control and treatment birds in parasite loads, although control birds were observed with coccidia and trypanosomes whereas no parasites were observed in treated males'.

[n] Experimental birds, which were raised under conditions promoting parasitism, had significantly larger numbers of coccidia and nematodes than did controls.

[o] N. Hillgarth, PhD thesis, University of Oxford, 1990.

[p] D.H. Clayton, PhD thesis, University of Chicago, 1989.

[q] Maximum possible heritability calculated by measuring repeatability of parasite load estimates over time.

Box 2. Is PMSS Testable?

PMSS could be incidental to the evolution of showiness, which may evolve for reasons having nothing to do with parasites[12,43,45]. Even if showy males have low parasite loads it is difficult to disprove the possibility that females examine showy traits to acquire information about other variables affecting both showiness and parasite load, such as nutrition. If it could be shown that females choose males on the basis of showy traits affected *only* by parasites, it would constitute reasonably strong evidence for PMSS (given concurrent tests of the other PMSS assumptions, see text). Direct indicator traits may prove useful in this regard. For example, Spurrier and colleagues[62] demonstrated that chewing lice create haematomas on the air sacs of male Sage Grouse. They further showed that females avoid mating with experimental males that are given simulated haematomas (unfortunately the experiment was not carefully controlled). Their results suggest that air sacs are direct indicators of louse load. If air sacs serve no other signaling function (unlike revealing handicaps which indicate a male's general condition), it would strongly imply that PMSS has played a role in the evolution of air sacs.

This analytical approach could be strengthened considerably by coupling it to a historical analysis of the origin and distribution of indicator traits and the parasites they indicate. Given a reliable host phylogeny and parasite phylogeny (no mean feat), one could perhaps examine the evolutionary origin of the indicator trait in relation to the original establishment of the parasite on its host[66]. Demonstrating that air sacs originated at approximately the same time that lice became established on grouse would constitute strong evidence for the role of PMSS in the evolution of air sacs. In particular, this kind of evidence would argue against the possibility that female choice and male showiness are adaptively neutral traits subject to 'runaway' sexual selection (see Ref. 13 for a discussion of this process).

Model-specific assumptions of PMSS

Good genes model. Hamilton and Zuk[1] proposed that females who mate with unparasitized males acquire heritable resistance for their offspring. They assumed that resistance and virulence undergo continuous coevolutionary cycles that maintain heritable variation in both traits. This solves the theoretical paradox of good genes models, that selection on a fitness-enhancing trait will exhaust the heritable variation in that trait, thus neutralizing its relative fitness benefit and potential evolutionary response to further selection[6,13]. However, coevolutionary cycles are not the only way in which heritable variation can be maintained. Pomiankowski suggested that 'Variations in space and time of the direction and intensity of selection, migration and mutation (both beneficial and deleterious), for example, are all common forces that can contribute to the maintenance of heritable variation . . .' (Ref. 6).

A more basic assumption of the good genes model is that parasite resistance is heritable. This assumption has been tested directly in two studies (Table 1), both of which demonstrated heritable components to parasite resistance. These and other studies[38] clearly indicate that resistance to parasites can be heritable but they do not test for coevolutionary cycles or other mechanisms for maintaining heritable resistance. Tests of coevolutionary cycles will presumably be difficult because such cycles are likely to occur with a periodicity of the order of tens of generations (W.D. Hamilton, pers. commun.).

Transmission avoidance model. This model assumes that, by choosing to mate with relatively unparasitized males, females avoid the direct transmission of parasites to themselves or offspring. The model is largely applicable to ectoparasites and sexually transmitted diseases but could apply to any situation in which the probability of transmission increases with proximity to infected individuals. For example, Møller[39] showed a significant correlation between the number of mites on swallows arriving from their wintering grounds and the number of mites in their nests after the fledging of offspring. The potential importance of transmission avoidance is highlighted by the fact that over one-third of the tests in Table 1 involve ectoparasites with direct life cycles. The avoidance of direct transmission is clearly a potential benefit of choosing a relatively unparasitized mate.

Resource provisioning model. This model assumes that females who mate with unparasitized males are more likely to acquire resources from the male, such as help with parental care duties. The only published evidence bearing on this assumption is the realistic suggestion by Milinski and Bakker[36] that ciliates reduce the ability of male sticklebacks to care for eggs and young after spawning (Table 1), which could be costly, given that parental care in this species is provided solely by males.

Criticisms and questions

The expertise of traditional parasitologists is essential for addressing the numerous criticisms of PMSS, the most pressing of which are briefly addressed below and in Box 2.

Identification of parasites. Few authors have cited sources for the identification of parasites. In one study[40] lice were erroneously assigned to a genus of Ischnocera, a suborder of feather-feeding lice that are presumably unsusceptible to specific immunity[7]. In subsequent studies[9,10], the same lice were correctly assigned to a genus of Amblycera, a suborder of blood-feeding lice that are susceptible to specific immune responses and which have a greater effect on host fitness[41]. Considering the difficulty of identifying most parasite taxa, one is left wondering how many misidentifications persist in the literature.

Choice of parasites. PMSS is hard to falsify for several reasons[12]. One reason is that it is difficult to account for the many parasite taxa potentially infesting a given host. One can always argue that results are inconsistent with PMSS because an inappropriate parasite was studied. Perhaps the only way to circumvent this problem is to study a host's entire parasite community, which will be

difficult, given that relevant parasites could include viruses, fungi, bacteria, protozoa, helminths and/or arthropods. The need for a community approach is emphasized by the presumed importance of synergistic effects: although a single parasite might not affect fitness, showiness or mate choice, the combination of multiple parasites could have important consequences. The narrow taxonomic range of parasites in Table 1 demonstrates that parasite diversity has been relatively ignored in studies of PMSS, particularly with regard to viral and bacterial parasites.

Measurement of parasite load. Parasites are notoriously difficult to quantify. Parasite load is a generic phrase encompassing both prevalence and intensity, as used here. Because parasite load is the independent variable in tests of PMSS, it is critical to employ an estimate that predicts total load with a specified degree of accuracy[41] or for which repeatability has been estimated[42]. The measure of parasite load used should be appropriate to the kind of parasite studied. Other things being equal, prevalence should be used to measure microparasites and intensity to measure macroparasites[19] (but see the next section regarding exposure). A recent study of macroparasites reported conflicting results depending on whether prevalence or intensity was analysed (Table 1 and Ref. 43). One could argue that only the results based on intensity are relevant in this case. At the very least, relative intensity (the product of prevalence and intensity) should perhaps be used rather than uncoupled measures of parasite load.

Differential exposure to parasites. 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. Similarly, having a high load does not necessarily mean that an individual has low resistance; it may mean that the parasite is of low virulence or that the host is resistant pending the activation of acquired immunity. Thus, exposure may complicate the relationship of parasite load to resistance, thereby complicating the ability of females to identify and choose resistant males as mates (not to mention the ability of researchers to measure the relevant variable in tests of PMSS). One could argue that females should avoid parasite-free males in favour of individuals with small loads, to assure that potential mates have been exposed and possess resistance against the build-up of large parasite loads.

What is needed for empirical studies is some measure of the extent of exposure that controls for evasive action on the part of the host, such as behavioural or immunological responses. 'Serological surveys probably come closest to giving estimates of the proportion of a population who have been exposed to infection; these generally show that there are individuals in a population who have not been exposed . . .' (Ref. 12). In short, parasite load is at best an indirect measure of resistance, particularly in the case of microparasites.

Measurement of host showiness. Showiness should be measured in a way relevant to the sensory mode(s) of the host, not of the investigator. Absolute showiness is probably less important than the contrast of a male with his background[30]. It is preferable to show exactly how the parasite affects showiness and whether females use the trait in question during mate choice. Human intuition can be misleading. For example, Clayton[7] suggested that damage to the iridescent plumage of pigeons by feather-feeding lice might serve as a cue enabling females to detect and avoid parasitized males. However, a test of this hypothesis revealed that lice have no effect on iridescence, which is restricted to the tips of feathers, because lice feed only on the basal regions of feathers.

Demographic factors. Host population density often determines the threshold below which parasite populations cannot become established. For example, Freeland[44] showed a correlation between primate group size and the diversity of their intestinal protozoan communities. If host populations undergo strong temporal fluctuations, parasites relevant to PMSS may periodically be scarce or locally extinct. Because traits attributable to PMSS will persist in the host population during such periods, it is possible for short-term field studies to miss relevant parasites[16,17,45]. This problem emphasizes the need for long-term field studies and further exacerbates the difficulty of testing PMSS.

Host manipulation. Most studies of PMSS have ignored the coevolutionary nature of host–parasite interactions. Parasites may be under strong selection to counteract PMSS by manipulating host sexual behaviour, akin to the manipulation of other forms of host behaviour[46]. In the case of parasites that depend on host contact for transmission, it might be in the parasite's best interest to neutralize the indicativeness of showy traits or even to increase the attractiveness of males if possible[47]. Although parasites are known to have a negative impact on host reproductive behaviour (eg. reduced copulation rates of nematode-infected mice[48]), the possibility that parasites might enhance reproduction has not been tested.

Concluding comments

The results of experimental studies show consistent support for the general assumptions of PMSS. With the exception of Møller's work[37,39,42,49,50], all of the experimental studies were performed with captive populations. In contrast, the results of correlational studies, most of which employed natural populations, are inconsistent, suggesting that extraneous variables may complicate the influence of parasites on sexual selection in the real world. What the captive studies gain in analytical power they may lose in applicability to natural

situations. The experimental work bears repeating in natural populations, preferably in the context of long-term field studies. It is critical that negative as well as positive results of such studies be published.

Discriminating tests of the model-specific assumptions of PMSS have not been performed. It is therefore impossible to evaluate the relative benefits accrued by choosy females. Although parasite resistance can clearly be heritable, a more complete test of the good genes model will require documentation of the maintenance of heritable variation over multiple generations by coevolutionary cycles, mutation pressure or other mechanisms. Furthermore, experimental tests of the transmission avoidance and resource provisioning models, which are more parsimonious than the good genes model, have not been performed despite the fact that both are quite plausible.

The feasibility of the three models of PMSS could be determined by testing each model using a host–parasite system that reduces the potential role of the other two models. For example, the good genes model might be tested with a host species in which males provide no resources and a parasite species that is not transmitted through host proximity. The transmission avoidance model might be tested with a nonprovisioning host species and a directly transmitted parasite less subject to heritable resistance (assuming such a parasite exists; Ischnoceran lice[7] could be one possibility). The resource provisioning model might be tested using a host species in which males provide resources and a parasite species that is independently transmitted and less subject to heritable resistance. Once the feasibility of each model is shown, their relative importance could be determined by testing them with a host–parasite system potentially subject to all three processes. Needless to say, parasitologists must play a central role in the design and execution of such studies.

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