Evolution, 53(3), 1999, pp. 957–961

SEXUALLY TRANSMITTED DISEASE AND PARASITE-MEDIATED SEXUAL SELECTION

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Abstract.—Few studies have investigated the consequences of parasite-mediated sexual selection on the parasites involved. In some cases parasite-mediated sexual selection could lead to increased virulence, but I develop a simple model that shows that, if a parasite is sexually transmitted (i.e., is a sexually transmitted disease, or STD) and if mating success of the host is adversely affected by the parasite, then less virulent STDs will be selected for because transmission of the STD depends on the mating success of the host. This selection for reduced virulence could have important consequences for the role of STDs in sexual selection.

Key words.—parasite, parasite-mediated sexual selection, sexual selection, sexually transmitted disease, STD, virulence.

Received August 5, 1998. Accepted December 22, 1998.

Hamilton and Zuk (1982) first suggested that if brightly colored or otherwise extravagant male ornaments act as a signal of parasite resistance, then females could gain from mating with males with such ornaments by obtaining parasite-resistance genes for their offspring. The role of parasitism in sexual selection has received a considerable amount of scrutiny since then, and there have been a large number of studies relating parasitism to mating success or to the size of extravagant male ornaments (Clayton 1991; Hamilton and Poulin 1997). In most of these studies of parasite-mediated sexual selection, there has been a focus on the evolutionary implications for the host and no consideration of the consequences for the parasite (Møller 1996), beyond a recognition of the fact that a coevolutionary arms race between parasite and host can provide the variability in male fitness needed for "good genes" models to work (Hamilton and Zuk 1982; Kirkpatrick 1986; Atkinson 1991). There are many other aspects of the parasite's relationship with its host that may also be important in these systems, one of which is the virulence of the parasite.

Møller (1996) suggested that parasite virulence should increase under parasite-mediated sexual selection because the types of parasites that are usually implicated in sexual selection are horizontally transmitted, which can lead to selection for increased virulence, whereas vertical transmission can select for decreased virulence (Herre 1993; Bull 1994; Ewald 1994; Agnew and Koella 1997). This may be true for some host-parasite systems, but I argue here that in at least one group of horizontally transmitted parasites, namely sexually transmitted diseases (STDs), there should be selection for reduced virulence if there is a link between parasitism and mating success, which may have important consequences for the evolution of parasite-mediated sexual selection in host-STD systems.

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Parasite fitness is traditionally approximated by calculating the "basic reproductive rate," or R_0 of the species or genotype in question. This provides a measure of the number of new infections (in the case of microparasites) or the number of new adult parasites (for macroparasites) that a single infected individual or adult parasite will produce over its lifetime in an otherwise uninfected host population (Anderson and May 1979; May and Anderson 1990). This is a very simple approach to parasite fitness, but it can still provide useful insights (e.g., see Thrall et al. 1998).

R_0 is calculated by dividing the rate at which new infections are produced by the time period during which the infected individual remains infectious (Anderson and May 1982; Knell et al. 1998). R_0 for a basic host-parasite model, with direct nonsexual transmission is given by

$$R_0 = \frac{\beta X}{\alpha + b + \gamma} \quad (1)$$

(Anderson and May 1982), where β is the probability of transmission per contact between infectious and susceptible hosts, X is the population of susceptible hosts, b is the death rate of healthy hosts, γ is the rate of recovery of infected hosts, and α is the death rate due to the disease, which is traditionally interpreted as the virulence of the parasite (May and Anderson 1990; Bull 1994). There are a number of other definitions of virulence, such as the reduction in the lifetime reproductive success of the host caused by the parasite (Herre 1993), but in the absence of a universally agreed definition (Bull 1994), I shall use the simplest.

The rate of production of new infected individuals in equation (1) is determined by the rate of contact between the infectious and susceptible individuals. If a parasite is sexually transmitted, however, production of new infections will depend on the mating frequency of the infectious individual. Therefore, for a sexually transmitted microparasite or a parasite that is transmitted to mates by close association, but rarely otherwise, a simple expression for R_0 is

$$R_0 = \frac{\beta c}{\alpha + b + \gamma}, \quad (2)$$

where c represents the number of sexual contacts; β is the probability of transmission per sexual contact; b, γ, and α are as in equation (1).

In equation (2) transmission of the parasite is independent of α, and so R_0 will be maximized by the parasite having minimal virulence. If transmission is linked to virulence in some way (transmission probability = $\beta f[\alpha]$ where f describes the relationship between transmission and virulence), then minimum virulence will no longer be selected for. Maximum virulence will be favored if β increases linearly or more than linearly with increases in α. If β is related to α in some other way, then R_0 will be maximized by an intermediate level of virulence, the value of this being dependent on the details of the relationship between the two variables. This is a well-known consequence of models of parasite virulence: Damage to the host is often linked to parasite transmission, so that parasites will trade one off against the other and evolve to intermediate levels of both (Anderson and May 1982; May and Anderson 1990; Bull 1994; Lipsitch et al. 1995; for a similar result based on a more ecological model, see also Lenski and May 1994).

However, if female hosts choose mates on the basis of some characteristic that is an indicator of the degree of infection the implications for parasite virulence change considerably. It is likely that the degree of reduction in brightness or character size that the male experiences will be related in some way to the virulence of the parasite. As the damage that the parasite causes increases, so will the cost to the host of repairing that damage, and the amount of resources the male is able to devote to its sexually selected character(s) will be reduced. Our expression for R_0 in this case has now changed:

$$R_0 = \frac{\beta f(\alpha) c f_1(\alpha)}{\alpha + b + \gamma}, \tag{3}$$

with f_1 being a function that describes how overall mating probability changes with parasite virulence (because STDs are also transmitted from females to males, if males are not choosing their mates [the simplest scenario], then the overall reduction in mating success associated with virulence will be one-half of the reduction in male mating success). The rate of new sexual encounters is therefore also dependent upon the virulence of the parasite. Substituting in some fairly general expressions for f and f_1, $f = \alpha^y$ and $f_1 = 1/(1 + \alpha)^z$, allows us to explore the effect of linking virulence to mating success:

$$R_0 = \frac{\beta \alpha^y \left[\frac{c}{(1 + \alpha)^z}\right]}{\alpha + b + \gamma}. \tag{4}$$

If $y > 1 + z$ there will be no maximum value for R_0, and once again maximum virulence will be selected for. This is unlikely biologically, because it requires transmission to increase exponentially with virulence. If $z = 0$ there is no relationship between mating success and virulence, but if $z > 0$, maximum R_0 decreases with increases in z. In other words, because mating frequency decreases with α, R_0 will be maximized by a lower virulence than would be found in a system in which mating success was not dependent upon parasitism (Fig. 1).

The parasites considered so far have been assumed to rely exclusively on transmission during mating. However, many parasites can be transmitted either during mating or by other routes (Lockhardt et al. 1996; Thrall et al. 1998). For such parasites, our expression for R_0 will be more complex, for example:

$$R_0 = \frac{\beta f(\alpha) f_1(\alpha) c + \beta_1 f_2(\alpha) Y}{(\alpha + b + \gamma)}. \tag{5}$$

Y is the population density. β_1 is the likelihood of transmission per contact with a susceptible host that is not mated with (the nonsexual transmission coefficient), and f_2 determines the relationship between nonsexual transmission and virulence. Equation (5) assumes the rate of sexual encounters to be independent of the population density because STD transmission is thought to be dependent on the frequency of infectious hosts rather than on population density (Getz and Pickering 1983).

The value of R_0 is now also dependent upon the population density, the value of β_1, and the form of the relationship between f_2 and α. In the simplest scenario, if we assume that the relationship between the transmission rate and virulence is the same for nonsexual transmission as it is for sexual transmission, then the addition of this form of transmission simply acts as a scaling factor and the optimum value of α remains low (Fig. 2). Our conclusion from equation (4), namely that low virulence will be selected for, is unchanged. Although the intensity of selection for low virulence will decline as the contribution to R_0 of transmission to individuals other than mates increases, the gains to females of selecting unparasitized mates will also decline as the probability of their acquiring the parasites in question from other individuals in the population increases.

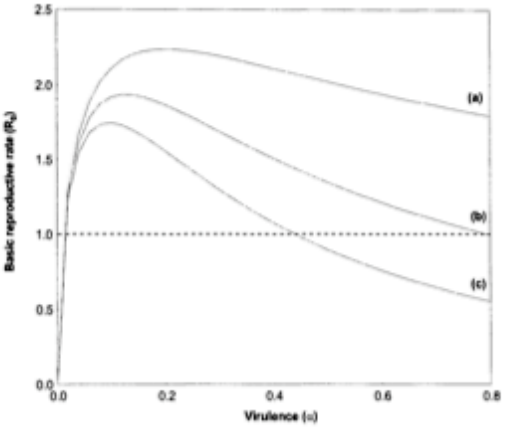

FIG. 1. Changes in R_0 with varying levels of virulence and values of z from equation (4). (a) $z = 0$, no sexual selection: a sexually transmitted parasite where the rate of sexual encounters is not linked to virulence, although transmission is. (b) $z = 1$, parasite-mediated sexual selection via transmission avoidance: the rate of sexual encounters declines linearly with virulence. (c) $z = 2$, as (b), but the rate of sexual encounters now decreases exponentially with virulence. The dotted line gives the threshold value of $R_0 = 1$, below which the parasite will be unable to sustain itself and will become extinct. Other parameter values: $y = 0.5$, $\beta = 1$, $c = 2$, $\gamma = 0.1$, $b = 0.1$.

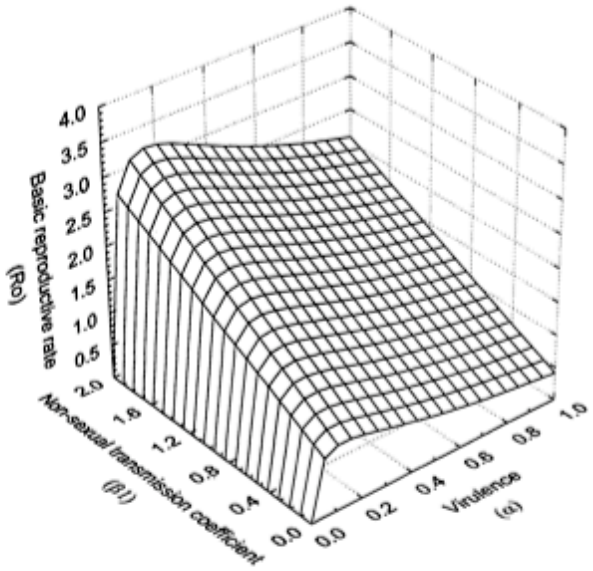

FIG. 2. Changes in R_0 with varying levels of virulence and varying amounts of transmission to hosts other than mates. R_0 increases generally as nonsexual transmission increases, but the optimum virulence is still low. The nonsexual transmission coefficient is β_1 of equation (3), the virulence is α. The equation used here is $R_0 = [\beta\alpha^{0.5}c/(1 + \alpha) + \beta_1\alpha^{0.5}Y]/(\alpha + b + \gamma)$. Other parameter values used were $\beta = 1$, $c = 1$, $\gamma = 0.1$, $b = 0.1$, $Y = 1$.

DISCUSSION

This model demonstrates that if individuals choose mates on the basis of a characteristic that indicates the degree of parasitism suffered by the potential mate, then less virulent STDs will be selected. These less virulent parasites will have hosts that will acquire mates more frequently, giving more opportunities for transmission. Because the relationship between the size or brightness of sexually selected characters and the number of matings is often skewed, so that those few individuals with the brightest or largest characters get most of the matings (Andersson 1994), the advantages for the less virulent parasite may be very great.

What are the limitations of this model? The model presented here, like many others, ignores many features of the host-parasite interaction for the sake of simplicity. One important aspect of host parasite systems that is beyond the scope of the present paper is the effect of competition between parasite strains. This competition has been shown to lead to selection for increased virulence when virulence is linked to competitiveness and can lead to the coexistence of multiple parasite strains with variable virulences (Bremermann and Thieme 1989; May and Nowak 1994; Nowak and May 1994). Therefore, competition between different STD strains could lead to more virulent STD strains being maintained in a population. The strength of selection for increased virulence would still be reduced from the normal situation, however, as a consequence of the increased costs that virulent strains would have to pay from the reduced mating success of their hosts. This would affect the trade-off between competitive ability and reproductive rate that maintains the more virulent strains in the population (Nowak and May 1994).

One other aspect of the host-parasite relationship that is not considered here is the effect of host density and particularly the interaction between host density and virulence. Lenski and May (1994) demonstrated that for conventionally transmitted parasites, lower virulence than might otherwise be expected could evolve as a consequence of the reductions in host density brought about by the parasites. This suggests that care must be taken when using R_0 as an indicator of the evolutionarily stable virulence of the parasite. It is not clear how Lenski and May's conclusions apply to the current study, however, because their conclusion relied on transmission being a strictly increasing function of the density of susceptible hosts. This is likely to be the case for conventionally transmitted parasites, but not for STDs, which have frequency-dependent rather than density-dependent transmission dynamics (Getz and Pickering 1983). This is an area that deserves further study.

The use of host mortality rate as an index of virulence makes the assumption that the costs to the male host that result in a reduction in brightness or size of an extravagant character will be related to the mortality rate. I consider this assumption to be reasonable, because these costs will arise from physiological damage to the host or increased metabolic costs from resisting the STD, both of which should be related in some way to the death rate from the parasite. Even if the form of this relationship is not particularly simple, the qualitative conclusions of the model are unlikely to be affected.

One effect on the host that is not necessarily related to mortality is the effect of the parasite on fecundity. STDs often cause sterility rather than mortality (Lockhardt et al. 1996), but effects of an STD on host fertility have no impact on R_0 (Thrall et al. 1998), because R_0 is calculated assuming a single infected host in an uninfected population. This may say more about the limitations of using the basic reproductive rate to make predictions about parasite evolution than it does about the relevance of effects on fecundity to parasite evolution. A full model of the coevolution of hosts and STDs under parasite-mediated sexual selection would need to consider this.

What are the implications of this model for parasite-mediated sexual selection? A number of authors have speculated that STDs could be important in the evolution of mating systems (Hamilton 1990; Sheldon 1993; Hurst et al. 1995; Loehle 1995; Able 1996), and the possible impact of STDs on the evolution of promiscuity has been theoretically investigated by Thrall et al. (1997). All of these authors have essentially considered the consequences for the host without considering the consequences for the parasite. If the parasite evolves to become less virulent, its role in sexual selection at the level of female choice will become less important for two reasons. First, the parasite will not be as detectable as it would be if it exhibited high virulence. Second, the selective advantage to the female of detecting it will be reduced, but not necessarily eliminated: Although the optimal virulence is reduced by the addition of female choice into the model (Fig. 1), it is still greater than zero. In general this would lead to STDs being less likely than other parasites to

be important in the evolution of extravagant sexually selected characters.

One theoretical study that has considered the evolution of STDs should be mentioned here. Thrall et al. (1998) considered the evolution of parasite transmission mode in relation to the number of sexual and nonsexual contacts that the host makes and modeled the degree of conventional versus sexual transmission that would be selected for under various combinations of these. The optimum strategy for a parasite was found to be related to the ratio of sexual to nonsexual contacts, with conventional transmission being favored when nonsexual contacts were more common than sexual ones, with the inclusion of mortality and fecundity effects of the parasite giving more complex predictions, including mixed strategies. The outcomes of such a model would probably be affected considerably by the inclusion of female choice for uninfected mates because this could favor allocation to conventional transmission even when there are more sexual than nonsexual contacts.

What predictions does this model make, and are they compatible with empirical data? There are two predictions that can be made here. First, if STDs are unlikely to be important in the evolution of exaggerated characters, then within a species the inverse relationship between parasitism and ornament size predicted by Hamilton and Zuk (1982) should not be found. Instead either no relationship or a positive relationship between ornament size and parasitism is predicted, the latter because those animals with larger ornaments achieve more matings and may therefore acquire more parasites. Second between species, the virulence of STDs should be negatively related to the amount of mate choice exhibited by a particular species.

Although there is a considerable body of evidence both for and against parasite-mediated sexual selection in general (e.g., Clayton 1991; Hamilton and Poulin 1997), there are no published studies of that the author is aware which have investigated the role of STDs in sexually selected systems. This is surprising considering the ubiquity of STDs (Lockhart et al. 1996), and it is probably a consequence of biases toward certain easily sampled parasite groups by behavioral ecologists. A recent review of the characteristics of STDs (Lockhardt et al. 1996) found that STDs generally cause less mortality than do conventionally transmitted parasites. This is compatible with the predictions from this model, although Lockhardt et al. (1996) discuss a number of other good reasons for expecting them to evolve toward reduced virulence in terms of host mortality.

It should be pointed out that the conclusions from this model only apply to cases where the mating probability of an animal is directly affected by a parasite that is itself transmitted either sexually or by close contact during mating. STDs in the many systems where there is no female choice for male characters obviously do not fall into this category. The results presented here are therefore not universal, but they do demonstrate two important points: STDs are likely to play a different role than conventionally transmitted parasites in parasite-mediated sexual selection and considering the evolution of sexually selected systems in terms of host-parasite interactions without thinking about the evolution of the parasite will only ever give half of the story.

Acknowledgments

I would like to thank M. Möller for helpful advice during the preparation of the manuscript, and two anonymous reviewers for insightful comments on a previous version of the manuscript.

Literature Cited

Able, D. G. 1996. The contagion indicator hypothesis for parasite-mediated sexual selection. Proc. Natl. Acad. Sci. USA 93:2229–2233.

Agnew, P., and J. C. Koella. 1997. Virulence, parasite mode of transmission, and host fluctuating asymmetry. Proc. R. Soc. Lond. B Biol. Sci. 264:9–15.

Anderson, R. M., and R. M. May. 1979. Population biology of infectious disease. Part 1. Nature 280:361–367.

———. 1982. Coevolution of hosts and parasites. Parasitology 85: 411–426.

Andersson, M. 1994. Sexual selection. Princeton Univ. Press, Princeton, NJ.

Atkinson, D. 1991. Sexual showiness and parasite load: correlations without parasite coevolutionary cycles. J. Theoret. Biol. 150:251–260.

Bremermann, H. J., and H. R. Thieme. 1989. A competitive-exclusion principle for pathogen virulence. J. Math. Biol. 27: 179–190.

Bull, J. J. 1994. Perspective: virulence. Evolution 48:1423–1437.

Clayton, D. H. 1991. The influence of parasites on host sexual selection. Parasitol. Today 7:329–334.

Ewald, P. 1994. Evolution of infectious disease. Oxford Univ. Press, Oxford.

Getz, W. M., and J. Pickering. 1983. Epidemic models: thresholds and population regulation. Am. Nat. 121:892–898.

Hamilton, W. D. 1990. Mate choice near or far. Am. Zool. 30: 341–352.

Hamilton, W. D., and R. Poulin. 1997. The Hamilton and Zuk hypothesis revisited: a meta-analytical approach. Behaviour 134: 299–320.

Hamilton, W. D., and M. Zuk. 1982. Heritable true fitness and bright birds: a role for parasites. Science 218:382–387.

Herre, E. A. 1993. Population structure and the evolution of virulence in nematode parasites of fig wasps. Science 259:1442–1445.

Hurst, G. D., R. G. Sharpe, A. H. Broomfield, L. E. Walker, T. M. O. Majerus, I. A. Zakharov, and M. E. N. Majerus. 1995. Sexually transmitted disease in a promiscuous insect, *Adalia bipunctata*. Ecol. Entomol. 20:230–236.

Kirkpatrick, M. 1986. Sexual selection and cycling parasites: a simulation study of Hamilton's hypothesis. J. Theoret. Biol. 119: 263–271.

Knell, R. J., M. Begon, and D. J. Thompson. 1998. Host-pathogen population dynamics, basic reproductive rates and threshold densities. Oikos 81:299–308.

Lenski, R. E., and R. M. May. 1994. The evolution of virulence in parasites and pathogens: reconciliation between two competing hypotheses. J. Theoret. Biol. 169:253–265.

Lipsitch, M., E. A. Herre, and M. A. Nowak. 1995. Host population structure and the evolution of virulence: a "law of diminishing returns." Evolution 49:743–748.

Lockhart, A. B., P. H. Thrall, and J. Antonovics. 1996. Sexually transmitted diseases in animals: ecological and evolutionary implications. Biol. Rev. 71:415–471.

Loehle, C. 1995. Social barriers to pathogen transmission in wild animal populations. Ecology 76:326–335.

May, R. M., and R. M. Anderson. 1990. Parasite-host coevolution. Parasitology 100:S89–S101.

May, R. M., and M. A. Nowak. 1994. Superinfection, metapopulation dynamics and the evolution of diversity. J. Theoret. Biol. 170:95–114.

Møller, A. P. 1996. Effects of host sexual selection on the population biology of parasites. Oikos 75:340–344.

NOWAK, M. A., AND R. M. MAY. 1994. Superinfection and the evolution of parasite virulence. Proc. R. Soc. Lond. B Biol. Sci. 255:81–89.

SHELDON, B. C. 1993. Sexually transmitted disease in birds: occurrence and evolutionary significance. Philos. Trans. R. Soc. Lond. B Biol. Sci. 339:491–497.

THRALL, P. H., J. ANTONOVICS, AND J. D. BEVER. 1997. Sexual transmission of disease and host mating systems: within season reproductive success. Am. Nat. 149:485–506.

THRALL, P. H., J. ANTONOVICS, AND W. G. WILSON. 1998. Allocation to sexual versus nonsexual disease transmission. Am. Nat. 151:29–45.

Corresponding Editor: B. Sullivan