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SEXUALLY TRANSMITTED DISEASE AND THE EVOLUTION OF MATING SYSTEMS

HANNA KOKKO,[1,2] ESA RANTA,[3] GRAEME RUXTON,[1] AND PER LUNDBERG[4]

[1]*Division of Environmental and Evolutionary Biology, Institute of Biomedical and Life Sciences, University of Glasgow, Glasgow G12 8QQ, United Kingdom*

[3]*Integrative Ecology Unit, Division of Population Biology, University of Helsinki, P.O. Box 65 (Viikinkaari 1), FIN-00014 Helsinki, Finland*

[4]*Department of Theoretical Ecology, Lund University, 223 62 Lund, Sweden*

Abstract.—Sexually transmitted diseases (STDs) have been shown to increase the costs of multiple mating and therefore favor relatively monogamous mating strategies. We examine another way in which STDs can influence mating systems in species in which female choice is important. Because more popular males are more likely to become infected, STDs can counteract any selective pressure that generates strong mating skews. We build two models to investigate female mate choice when the sexual behavior of females determines the prevalence of infection in the population. The first model has no explicit social structure. The second model considers the spatial distribution of matings under social monogamy, when females mated to unattractive males seek extrapair fertilizations from attractive males. In both cases, the STD has the potential to drastically reduce the mating skew. However, this reduction does not always happen. If the per contact transmission probability is low, the disease dies out and is of no consequence. In contrast, if the transmission probability is very high, males are likely to be infected regardless of their attractiveness, and mating with the most attractive males imposes again no extra cost for the female. We also show that optimal female responses to the risk of STDs can buffer the prevalence of infection to remain constant, or even decrease, with increasing per contact transmission probabilities. In all cases considered, the feedback between mate choice strategies and STD prevalence creates frequency-dependent fitness benefits for the two alternative female phenotypes considered (choosy vs. randomly mating females or faithful vs. unfaithful females). This maintains mixed evolutionarily stable strategies or polymorphisms in female behavior. In this way, a sexually transmitted disease can stabilize the populationwide proportion of females that mate with the most attractive males or that seek extrapair copulations.

Key words.—Disease dynamics, extrapair paternity, female choice, monogamy, sexual selection.

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Susceptibility to disease has featured prominently in research of sexual selection ever since Hamilton and Zuk (1982) proposed their hypothesis on female choice for healthy, resistant males. If males vary in their resistance to pathogens or parasites, females should be able to use sexual signals as indicators of genetic quality. The Hamilton-Zuk hypothesis proposes that host-parasite coevolution maintains variation in heritable quality. More recently, much attention has been focused on the related immunocompetence-handicap hypothesis, which states that the immune system is costly to maintain and therefore trade-offs between resistance to disease and other aspects of an individual's life history are expectable (for recent reviews, see Siva-Jothy and Skarstein 1998; Norris and Ewans 2000).

With nonsexually transmitted disease, males with best resistance genes can be the ideal mates for a large number of females. This could explain the evolution of female choice that leads to strong mating skews—unless other factors counteract, such as choice for complementary resistance genes (Penn and Potts 1999; Tregenza and Wedell 2000). Given the interest in disease as a factor that shapes animal mating systems, sexually transmitted diseases (STDs) have received relatively little attention (although see Freeland 1976; Hamilton 1990; Sheldon 1993; Møller 1994; Loehle 1995, 1997; Lockhart et al. 1996; Lombardo 1998). It appears that STD can have the opposite effect on mating systems than other forms of infection (Thrall et al. 1997). With STDs, the prevalence of disease is directly determined by sexual contact. A popular male will therefore be much more likely to carry the disease than a male whose mating success is meager. If the disease has direct negative fitness consequences on the female, this means a considerable cost of female behavior that leads to strong mating skews. The idea that mating with multiple males may increase a female's risk from STDs has consequently been suggested as a possible selection pressure for monogamy (Sheldon 1993; Loehle 1995; Poiani and Wilks 2000a). STD avoidance has even been proposed as the reason behind human monogamy (Immerman 1986; Immerman and Mackey 1997).

Thrall et al. (1997) presented one of the first formal models of optimal mating behavior in the presence of STD (see Discussion for other models). They assumed that females benefit from multiple matings through reducing the risk of infertility, but that females also risk catching the disease when mating. As a result, STDs could reduce multiple mating, although strict monogamy was not generally expected to evolve either (Thrall et al. 1997).

Here, we consider another angle of STD: Matings with some males will be riskier than matings with others. Specifically, males that have had previous matings with a larger number of females are more likely to be infected than males that have had fewer matings (Anderson 1991; Thrall et al. 2000). Thus, STDs may not just influence how many males a female may be prepared to mate with, but may also influence which males she chooses. This extension has rarely been considered (but see Poiani and Wilks 2000a) but has the potential to considerably alter our understanding of mate choice. The costs of STD transmission will counteract any selection pressures driving females to mate with a limited subset of males in a population. This counterselection is of special interest because the costs of STDs

[2] Present address: Department of Biological and Environmental Science, P.O. Box 35, University of Jyväskylä, Finland; E-mail: hanna.kokko@cc.jyu.fi.

are frequency dependent. The prevalence of a sexually transmitted disease will depend on mating behavior in the population as a whole (Anderson 1991). Thus, the risks a female takes by mating with specific males will depend on the behavior of other females.

Modeling Sexually Transmitted Disease and Mate Choice

We present two models that explore the effects of a sexually transmitted disease on mating skews. In both models, we consider males that vary in some aspect of their quality, which is of interest to the female. Males also differ in that they may or may not be infected. For simplicity, we assume that the quality of the male does not directly influence the transmission of disease. In other words, we assume that females are interested in high-quality males for reasons other than superior resistance to the STD; for example, high-quality males may resist other disease or parasites better than low-quality males. We assume there is no latent period to the disease and no recovery.

Model 1: No Explicit Social Structure

In this model, we examine the effect of an STD in the simplest setting that captures the costs and benefits of female choice for the same high-quality males. We consider a population in which mating and breeding occurs continuously over time. Mortality likewise occurs at a constant rate μ per unit time. There are two types of males: Type *A* is of especially high quality and consequently especially attractive to females, whereas type *B* is of lower quality and therefore relatively unattractive. Likewise, there are two types of females: Type *C* is choosy and mates with *A* males only, whereas type *R* mates randomly and therefore accepts both *A* and *B* males as mates. The fecundity of choosy females is denoted f_C and that of randomly mating females f_R. We assume that mating with an attractive male is intrinsically advantageous for the female, either in the form of direct or indirect benefits. She therefore produces surviving offspring at a higher rate if she is choosy: $f_C > f_R$. We assume that sufficiently many attractive males exist and they court females actively enough, so that choosy females do not have to spend additional time finding their preferred mate. This means that both choosy and randomly mating females mate at the same rate (once per unit time). These assumptions mean the female population should evolve to become completely choosy, were it not for the risk of the STD. We keep the model simple by assuming that a fraction α of males are born attractive regardless of their parent's phenotypes, but that the female strategy is maternally inherited (see Discussion).

Every individual is born uninfected and can become infected through mating with infected individuals. The infection is transmitted with probability δ in each mating (sensu Thrall et al. 1997). Infected individuals become immediately infectious (there is no latent period). In this model, we assume that the infection has no effect on fecundity or attractiveness of the male, but that the mortality μ of infected individuals increases. Thus, if attractive males are infected more often than the average of the whole population of males, being choosy increases the fecundity but potentially decreases the life span of a female.

These assumptions translate to the following dynamics for males,

$$\frac{dN_{AU}}{dt} = -\mu_{AU}N_{AU} - \delta(M_{AU,CI} + M_{AU,RI}) + f_C\alpha(N_{CU} + N_{CI}) + f_R\alpha(N_{RU} + N_{RI}), \quad (1a)$$

$$\frac{dN_{AI}}{dt} = -\mu_{AI}N_{AI} + \delta(M_{AU,CI} + M_{AU,RI}), \quad (1b)$$

$$\frac{dN_{BU}}{dt} = -\mu_{BU}N_{BU} - \delta(M_{BU,CI} + M_{BU,RI}) + f_C(1 - \alpha)(N_{CU} + N_{CI}) + f_R(1 - \alpha)(N_{RU} + N_{RI}), \quad \text{and} \quad (1c)$$

$$\frac{dN_{BI}}{dt} = -\mu_{BI}N_{BI} + \delta(M_{BI,CI} + M_{BI,RI}) \quad (1d)$$

and for females,

$$\frac{dN_{CU}}{dt} = -\mu_{CU}N_{CU} - \delta(M_{AI,CU} + M_{BI,CU}) + f_C(N_{CU} + N_{CI}), \quad (2a)$$

$$\frac{dN_{CI}}{dt} = -\mu_{CI}N_{CI} + \delta(M_{AI,CU} + M_{BI,CU}), \quad (2b)$$

$$\frac{dN_{RU}}{dt} = -\mu_{RU}N_{RU} - \delta(M_{AI,RU} + M_{BI,RU}) + f_R(N_{RU} + N_{RI}), \quad \text{and} \quad (2c)$$

$$\frac{dN_{RI}}{dt} = -\mu_{RI}N_{RI} + \delta(M_{AI,RU} + M_{BI,RU}), \quad (2d)$$

where N denotes population sizes of males or females, with subscripts AU for type A males, uninfected; AI for type *A* males, infected; BU and BI similarly for type B males; CU and CI for choosy females; and RU and RI for randomly mating females. M denotes the number of matings per time unit between specific male and female types, in which one of the partners is uninfected and the other infected (these are the only types of mating in which the disease can spread, and thus the only ones for which we need expressions). Because choosy females never mate with type *B* males, we have $M_{BI,CU} = M_{BU,CI} = 0$. The other types of mating rates are

$$M_{AU,CI} = \frac{N_{AU}N_{CI}}{N_{AU} + N_{AI}}, \quad (3a)$$

$$M_{AI,CU} = \frac{N_{AI}N_{CU}}{N_{AU} + N_{AI}}, \quad (3b)$$

$$M_{AU,RI} = \frac{N_{AU}N_{RI}}{N_{AU} + N_{AI} + N_{BU} + N_{BI}}, \quad (3c)$$

$$M_{BU,RI} = \frac{N_{BU}N_{RI}}{N_{AU} + N_{AI} + N_{BU} + N_{BI}}, \quad (3d)$$

$$M_{AI,RU} = \frac{N_{AI}N_{RU}}{N_{AU} + N_{AI} + N_{BU} + N_{BI}}, \quad \text{and} \quad (3e)$$

$$M_{BI,RU} = \frac{N_{BI}N_{RU}}{N_{AU} + N_{AI} + N_{BU} + N_{BI}}. \quad (3f)$$

These rates are derived assuming that females cannot distinguish between uninfected and infected males, that is, they choose their mate randomly from the pool of acceptable males (all males for randomly mating females, attractive males only for choosy females) regardless of their disease status.

The proportion of choosy females, x, is calculated as

$$x = \frac{N_{CU} + N_{CI}}{N_{CU} + N_{CI} + N_{RU} + N_{RI}}. \quad (4)$$

The prevalence of infection is calculated similarly, for example, the prevalence of infection in type A males is

$$p_A = \frac{N_{AI}}{N_{AI} + N_{AU}}. \quad (5)$$

Model 2: Social Monogamy with Extrapair Copulations

Model 1 does not have density-dependent mortality, ignores stochasticity, assumes that an attractive infected male can transmit the disease to females residing in any part of the population (i.e., that there is no spatial or social structure in the population), does not take into account that many organisms reproduce seasonally, and assumes that the cost of infection lies in mortality rather than, for example, fecundity. We now develop a model that contrasts with model 1 in all these respects. This allows us to examine if an STD has very different implications for the evolution of mating strategies depending on the details of ecological situation in which the process is embedded.

We consider an individually based simulation of a population of socially monogamous individuals. A fraction $0 < \alpha < 1$ of males are particularly attractive as mates. A fraction $x \leq 1$ of females seek extrapair copulations with an attractive male if they are mated to an unattractive male; the remaining $1 - x$ only mate with their social partner.

The details of the simulation are as follows. Each female is characterized by her health (infected or not), mate choice strategy (faithful or unfaithful), and spatial location (see below). Each male is characterized by his health (infected or not), attractiveness (one or zero), and spatial location. Initially, each female is assigned the unfaithful strategy with probability x independently of others, and each male obtains the attractiveness value one with probability α and value zero with probability $1 - \alpha$.

At the start of the simulation, there are N males and N females, and 50% of them are infected. Because reproductive interactions are often expected to be local rather than global (Webster et al. 2001), we also specify the spatial location of each individual by assigning it to a territory, with territories numbered 1 to N. Males and females inhabiting the same territory form a pair. At the start of the mating season, pairs mate with each other an unspecified number of times. These matings are summarized with a single parameter, γ_1: If only one of the pair members was infected before the start of the mating season, the other one becomes infected with probability γ_1.

After pair matings, if their own partner is unattractive, those females (fraction x) who use the unfaithful strategy mate in random order with a neighboring male. These females mate with the nearest attractive male. In a mating between uninfected and infected individuals, the uninfected individual becomes infected with probability γ_2. Pair members also mate with each other after extrapair copulations have taken place, and in these matings the transmission rate is γ_3. We may have $\gamma_1 \neq \gamma_2$ and $\gamma_1 \neq \gamma_3$ because of the different number of matings involved; for example, a female might have just one extrapair copulation but copulate with her social partner several times. Infected individuals become immediately infectious to others.

After the mating season, pairs produce young. The expected number of surviving offspring, F, increases with the attractiveness of the female's sexual partner or partners, $A \in \{0, 0.5, 1\}$, and decreases with the number of (social) parents infected, $P_I \in \{0, 1, 2\}$:

$$F = F_0 + aA - bP_I. \quad (6)$$

The constants F_0, a, and b are selected so that F is always nonnegative. We assume that the attractiveness benefit is genetic—and is therefore present when the father was an extrapair mate—but the cost of infection only applies if the members of the pair caring for the young are infected. Thus, if the pair father is unattractive but the mother has sought an attractive extrapair father, we set $A = 0.5$, which assumes that half of the offspring are sired by the extrapair male, but leave P_I unchanged. Note that if the effect of the sexually transmitted disease is to reduce the ability of the parents to care for the brood, the infection status of the extrapair male, who does not provide care for the focal brood, has no direct effect on the number of young. There still can be an indirect effect if he infected the female and/or both members of the primary pair.

The actual number of surviving offspring is drawn from a Poisson distribution with mean F for each pair independently. Offspring are born uninfected. Sex is determined stochastically for each offspring, each sex having a 50% likelihood. Each male offspring is assigned an attractiveness value of one with probability α, and a fraction x of female offspring are likewise assigned to use the unfaithful strategy. Attractiveness and mate choice strategy remain unchanged for the individual's lifetime.

Although it is easy to let the population evolve in such a model, we made the additional assumption that proportions of each strategy remain constant over generations (i.e., we do not specify any inheritance of the strategy). This was done to be able to quantify strength of selection at a specific gene frequency x. By keeping the proportion of females who use the unfaithful strategy x constant and evaluating the fitness of mothers (measured as number of surviving offspring), we are able to obtain large samples of the difference in fitness between these strategies. This enables us to quantify the strength of selection that operates at a specific gene frequency x and make predictions on the direction of evolutionary change. Selection will disappear, that is, faithful and unfaithful females will be equally fit, at a value of x that characterizes an evolutionarily stable strategy (ESS).

After the offspring have fledged, they join the pool of adults. N males and N females are drawn randomly to form the survivors that form the breeding population in the next year. This introduces density dependence—with a fixed number of breeding positions, the probability of surviving to

breed decreases with increasing population size. Infected individuals remain infected for the rest of their lives. The population is followed for 150 years. We present numbers of cuckolding and noncuckolding females, numbers of attractive and unattractive males, prevalences of infection in different female and male groups, and fledged offspring produced by cuckolding and noncuckolding females during the final 50 years. By this time the disease prevalence has settled to an equilibrium, around which it fluctuates stochastically. Each run was replicated 20 times with the same parameter values.

Results

Model 1

The STD does not limit the population, even though average mortality rates depend on the prevalence of infection. Although the population grows indeterminately (assuming that average mortality rates fall below fecundities), the prevalence of infection and the proportion of females using the choosy strategy quickly reach a stable equilibrium (Fig. 1), to which it returns if perturbed. In principle, these equilibria can be sought analytically, but they obey unwieldy expressions that offer little illumination. We therefore present only numerical results here.

Figure 1B shows the time series of the prevalence of infection among males. The heavy line derives from the same simulation as Figure 1A and represents the dynamically evolving model described. For comparison, we show the much higher prevalence that would occur if all females were always choosy and the much lower prevalence that would occur if all females mated randomly. This strong difference arises because the less attractive type *B* males are generally free from infection. For the parameters shown, less than 2% of type *B* males are infectious at equilibrium, whereas over 60% of type *A* males are. Additional simulations, not shown, suggest that this result holds qualitatively for other choices of parameter values, although type *B* males sometimes attain higher levels of infection too (but generally a clear difference to type *A* males remains).

The tendency of females to revert to the same strategy if perturbed indicates that being choosy versus nonchoosy forms a mixed ESS or polymorphism maintained by frequency-dependent selection. If no females are choosy, disease prevalence will be the same between both male types. Under these conditions, choosiness will always be favored because of the fecundity advantage provided by *A* males. This is why, even when per contact transmission probability δ is very high, some female choosiness remains (although it can be very low, e.g., $x \approx 10^{-8}$ at $\delta = 1$, Fig. 2A) and why disease prevalence is always greater among *A* males than *B* males. When the proportion of choosy females increases, the disease spreads primarily through type *A* males, and this makes it increasingly risky to mate with them. Eventually, the risk of disease offsets the inherent fecundity advantage of mating with type *A* males.

We now turn to exploring the model's predictions as we vary the disease transmission probability (δ). As we would expect, if δ is very low, the disease dies out. The risk of infection therefore disappears, and so at equilibrium all females are choosy. This remains unchanged as δ is increased, until a threshold values is exceeded. The disease now becomes established, and *A* males are the main carrier of the disease (left arrow, Fig. 2B). Females respond to this by switching away from the choosy strategy. Between the two arrows in Figure 2B, something very interesting happens. The prevalence of the disease decreases with increasing per encounter disease transmission probability δ. This occurs because the rapid switching of females away from choosiness

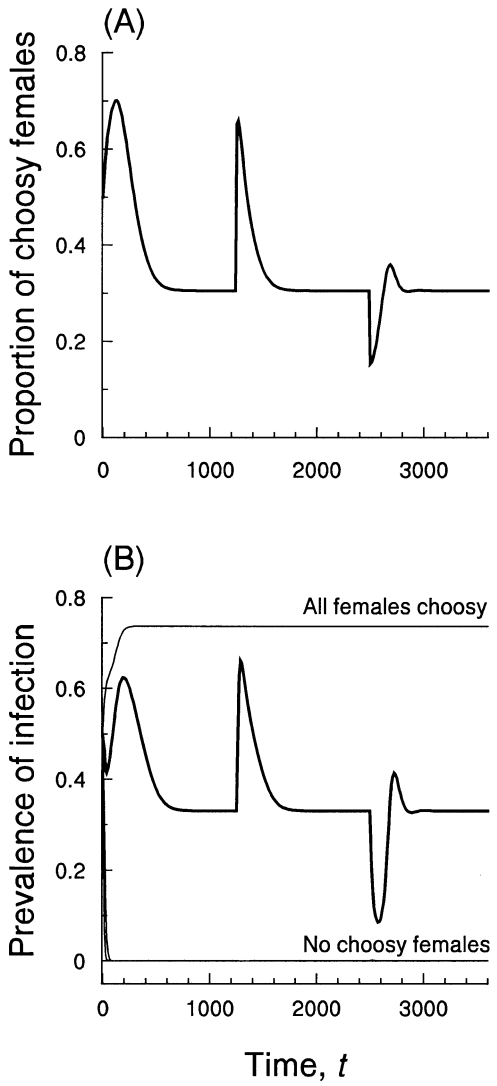

FIG. 1. The evolution of female choosiness (model 1), with parameter values $\delta = 0.1$, $f_R = 0.08$, $f_C = 0.1$, $\mu_{AU} = \mu_{BU} = \mu_{CU} = \mu_{RU} = 0.05$, $\mu_{AI} = \mu_{BI} = \mu_{CI} = \mu_{RI} = 0.12$, and $\alpha = 0.15$. Twenty units of time correspond to average life span of an uninfected individual. (A) An initial proportion 0.5 of choosy females settles to the value $x = 0.304$, to which it returns if it is artificially perturbed either upward (at $t = 1125$) or downward (at $t = 2250$). (B) Prevalence of infection in type *A* males either in the case where female choosiness evolves as in (A; thick solid line) or where only choosy females or randomly mating females are present in the population (thin lines). The prevalence of infection always drops to less than 0.015 in less-attractive males, regardless of the female strategy. A high proportion of choosy females amplifies the difference in infection prevalence between attractive and less-attractive males. This selects for lower choosiness in females, implying negative frequency dependence for the female strategy.

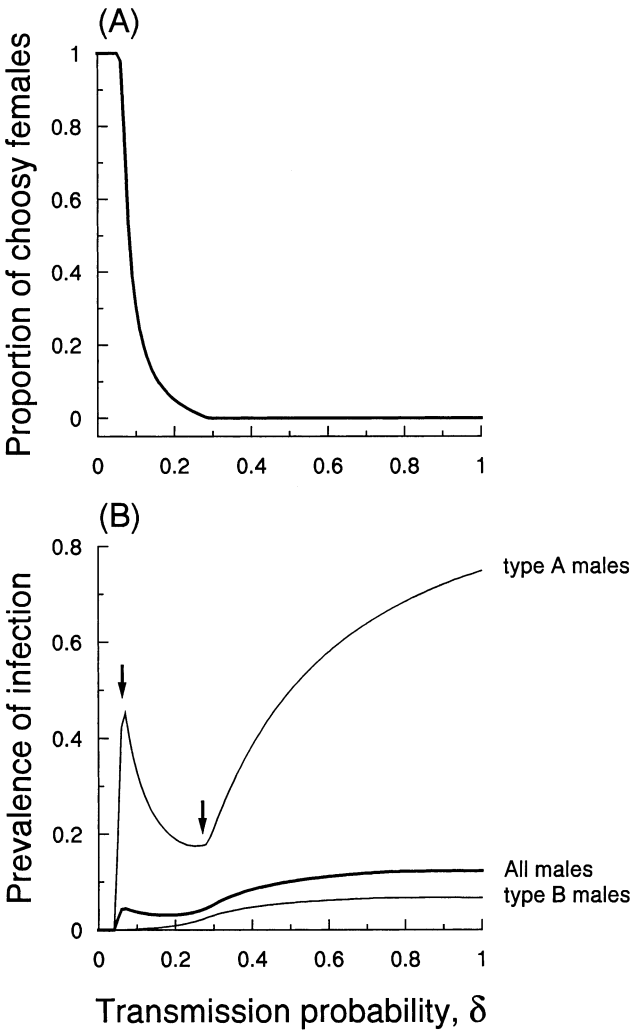

FIG. 2. Stable proportion x of choosy females (A) and the prevalence of infection in males at this level of female choice (B) for various values of the transmission probability, γ. Other parameter values are as in Figure 1. Between the values $\delta = 0.05$ and $\gamma = 0.25$ (marked with arrows), the proportion of females using the choosy strategy decreases with increasing δ. In this region, the evolutionary response of females means that increasing transmission probability leads to a reduction in the prevalence of infection in attractive males. In males overall, infection levels remain roughly constant over this region, indicating a buffering effect of female behavior. The proportion x never drops to zero, although it becomes very small at high or moderately high values of δ ($x < 10^{-5}$ for any $\delta > 0.3$).

greatly reduces the number of matings that type *A* males get and so reduce the number of new infections produced by an infectious *A* male. Although *B* males now mate more often, the absolute number of matings per *B* male is still low, so the prevalence of the disease in *B* males remains low (although increasing). This trend of decreasing female choosiness and reduced prevalence of the infection in *A* males and across the whole male population continues with increasing δ, until we reach another critical point, at the rightmost arrow in Figure 2. By now, the female population has almost completely shifted so that virtually no females are choosy. After this, the disease prevalence in *A* males increases with increasing δ, because the number of matings by an *A* male no longer drops (due to female strategy change) with increasing δ. The number of matings that a *B* individual obtains no longer increases, but because δ is increasing disease prevalence increases in this type too. Notice that female choosiness, x, never falls to exactly zero.

Model 2

Figure 3A illustrates the relative advantage of unfaithful females over faithful ones in terms of offspring produced as a function of the proportion x of the female population that seeks extrapair copulations if mated to an unattractive male. When few females are unfaithful (low x), the disease is rare. The benefits to females paired with lower quality males of seeking extrapair copulations then outweigh disease concerns, and selection favors unfaithful females. Thus, we would expect x to increase over evolutionary time (leftmost arrow pointing to the right in Fig. 3A). However, as x increases, the disease becomes more prevalent (Fig. 3B), so that the net advantage of extrapair behavior declines. After x has reached about 15%, any further increase leads to unfaithful females doing worse, which introduces selection pressure for x to decline (arrow pointing to the left, Fig. 3A). Thus, there is a locally stable equilibrium with approximately 15% of females being unfaithful. However, notice that if the proportion of unfaithful females, x, starts from a very high value, there is again a net advantage to unfaithfulness and the population evolves toward an equilibrium where all females paired to unattractive males are unfaithful ($x = 1$). The system has therefore two alternative equilibria, and the one that is reached depends on the initial proportion x of unfaithful females. If the population initially uses a high x, the relatively promiscuous mating system will be associated with very high disease prevalence in all sections of the population, including the unattractive males (Fig. 3B). In this situation, uninfected females mated to unattractive males are very likely to contract the disease from their social partner, and so they have little to lose by being unfaithful. This fatalistic selection pressure increases disease prevalence and so is reinforcing, eventually leading to $x = 1$.

Our results with model 2 suggest that fitness benefits of female mate choice are frequency-dependent in a manner similar to model 1 and that multiple equilibria are possible. We now turn to explore a wide range of rates of disease transmission (γ) on the expected equilibria, to see if the pattern produced in Figure 3 is typical or if other alternatives are possible. The model produces a variety of possible shapes of frequency dependence (Fig. 4 shows examples for which $\gamma_1 = \gamma_2 = \gamma_3$ are all equal to a single transmission parameter γ, and Fig. 5 displays the associated disease prevalence values). At low disease transmission rate, the disease dies out regardless of female behavior, and so all females evolve to be unfaithful (Figs. 4A, 5A). However, if γ is somewhat higher, then a very high proportion of unfaithfulness x leads to such high disease prevalence among attractive males (but not unattractive ones) that fidelity is selected for. Conversely, at low levels of populationwide unfaithfulness, the risk of disease is sufficiently low to favor extrapair copulations. Thus, we find a single equilibrium at an intermediate level

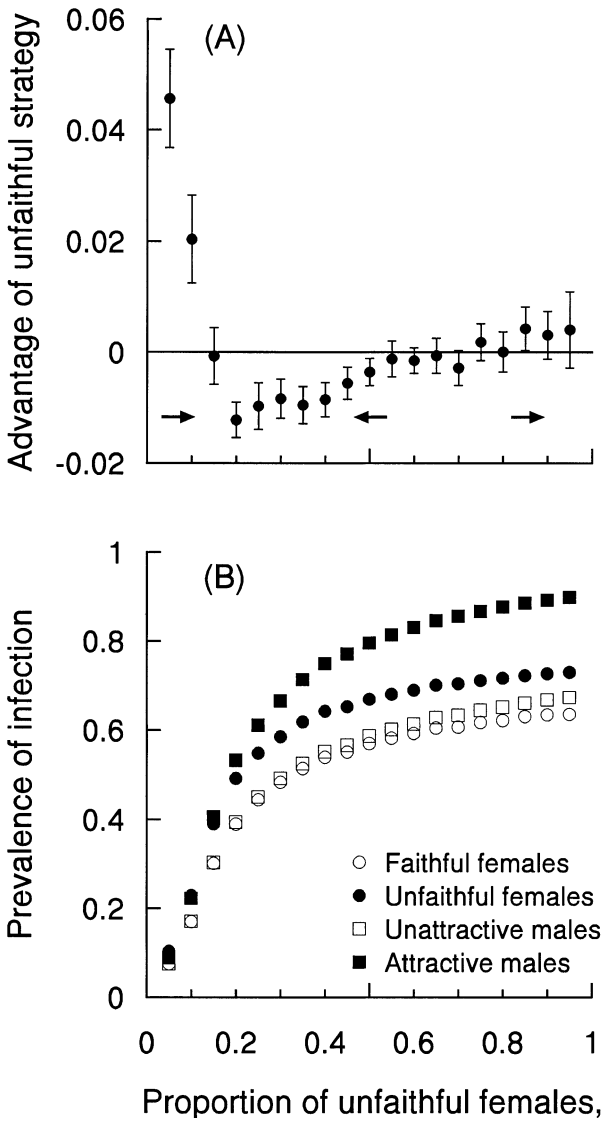

FIG. 3. (A) The benefit of cuckoldry expressed as the difference between the mean number of surviving offspring per breeding season of females using the cuckolding and noncuckolding strategy. (B) The mean prevalence of infection in different female and male groups in the cuckoldry model (model 2). Parameters used: $\gamma_1 = 0.5$, $\gamma_2 = 0.2$, $\gamma_3 = 0.4$, $F_0 = 1.5$, $a = 0.15$, $b = 0.5$, $y = 0.1$, $N = 1000$. In (A), arrows indicate the direction in which the frequency of cuckoldry, x, is expected to evolve. In (B), error bars are small (always < 0.02) and are not drawn for clarity. Open and filled circles and open and filled squares give prevalence of infection in noncuckolding and cuckolding females and unattractive and attractive males, respectively, measured after the mating season before breeding commences.

of fidelity (Figs. 4B, 5B). As γ increases, the costs of extrapair matings rise and so the equilibrium shifts toward fidelity (Figs. 4C–D, 5C–D). For even higher γ-values, disease costs become strong enough to predict complete absence of extrapair copulations (Figs. 4E, F; 5E, F). Eventually, for very high values of γ, we find dynamics with two alternative equilibria: Either no females or all females pursue extrapair copulations if mated to an unattractive male ($x = 0$ or $x = 1$; Figs. 4G–I, 5G–I).

Figure 5 shows the prevalence of infection for the cases considered in Figure 4. Clearly, the evolution of female mating behavior is not directly determined by the prevalence of the disease: Both $x = 0$ and $x = 1$ are possible outcomes when disease is widespread. The essential determinant of female behavior is the difference between the prevalence in type *A* or *B* males. At relatively low per contact transmission rates, increasing female choice allows the disease to spread mainly in *A* males, which selects against choosiness and produces intermediate equilibria through negative frequency dependence. At high transmission rates, most males are infected, regardless of their type. If most females are faithful, the probability of remaining healthy can still be high enough for a female to benefit from avoiding extrapair copulations (even if prevalences among types *A* and *B* are the same, an extrapair copulation still increases the risk of infection, because it means mating with two males instead of just the social mate). But, if most females are unfaithful, avoiding infection is so unlikely that females are selected to not care about the disease. Thus, high transmission probabilities produce positive frequency dependence and multiple equilibria. Finally, unequal transmission probabilities in pair versus extrapair matings can produce fitness curves with both negatively and positively frequency-dependent sections (Fig. 3).

The prevalence of infection at the evolutionary equilibria is shown with arrows in Figure 5. Comparing prevalences across the different transmission probabilities, γ, indicates that the evolution of female choice has a similar buffering effect as that shown in model 1. For example, the prevalences with $\gamma = 0.2$ (Fig. 5B) are lower than with $\gamma = 0.3$ (Fig. 5C) for any fixed value of female behavior x. However, as the transmission probability increases, the evolutionarily stable proportion of unfaithful females evolves from $x \approx 0.825$ (Fig. 5B) to $x \approx 0.275$ (Fig. 5C). This brings about smaller prevalences at the evolutionary equilibrium at the higher value $\gamma = 0.3$ (Figs. 5B, C).

DISCUSSION

Our models explore potential effects of STDs on female choice as a determinant of mating systems. We used two models, both of which shared the property that the optimal strategy for females in the absence of the disease is simple and easily understood. In model 1, females should only mate with attractive males; in model 2, females with low-quality partners should always be unfaithful. Both models show that STDs can select for a change in mating strategies, generally leading to a reduction in mating skew (increased matings by less-attractive males in model 1; fewer extrapair matings in model 2). This confirms the predictions by Sheldon (1993) and Loehle (1995) that disease dynamics can lead to selection for monogamy. However, the models display several interesting features that would have been more difficult to predict a priori.

First, both models show that the STD need not always have the general effect of influencing female mate choice strategies. It has been hypothesized, for example, that costs of extrapair copulations are higher in socially polygynous than monogamous species because of higher prevalence of STD in the former (Hasselquist and Sherman 2001) or that species

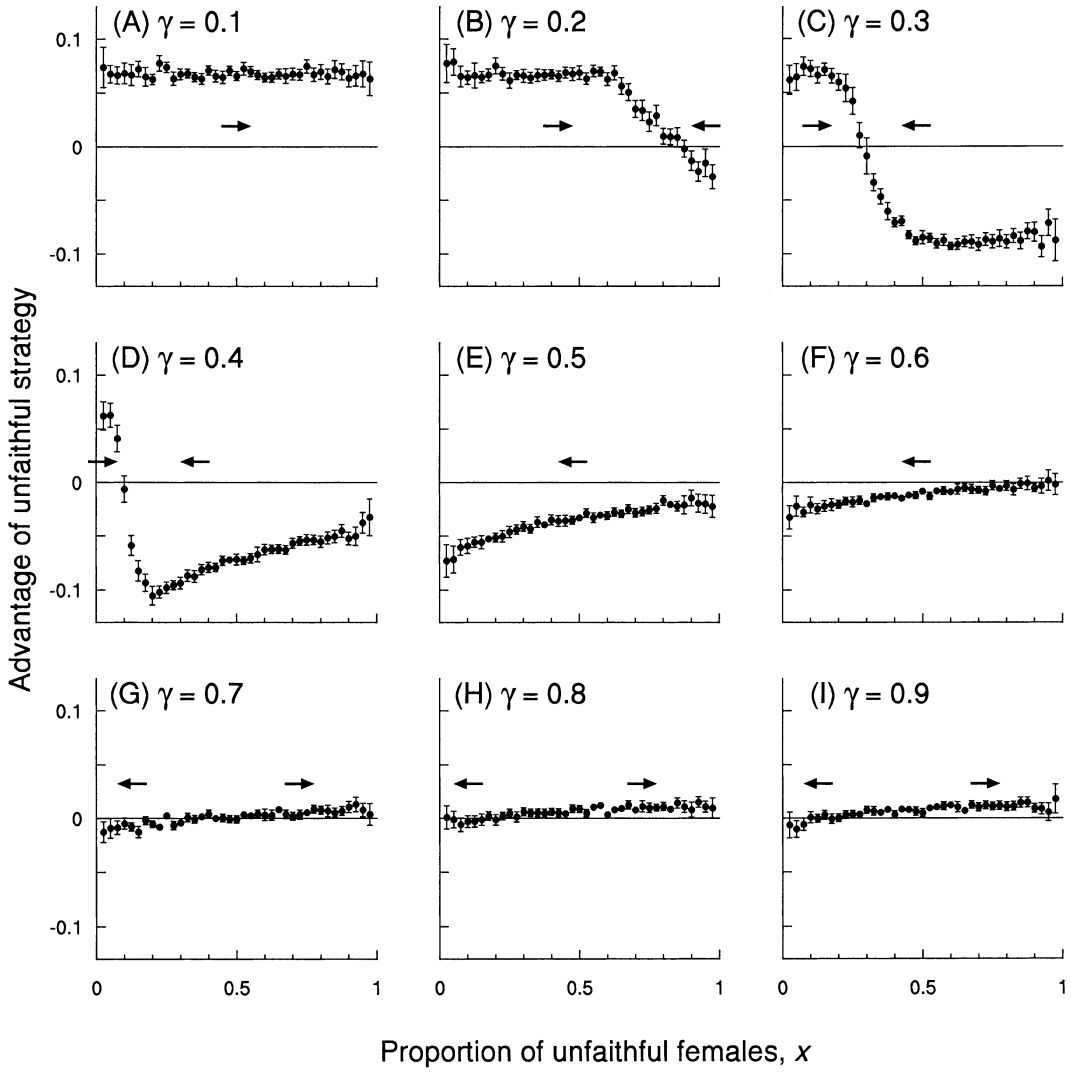

FIG. 4. The benefit of cuckoldry (the difference in fitness of faithful and unfaithful females), calculated as in Figure 3, for various values of the transmission probability, γ. Parameters used as in Figure 3, except $\gamma_1 = \gamma_2 = \gamma_3 = \gamma$ as given in each panel. Arrows indicate the direction in which the frequency of unfaithful females is expected to evolve.

with high prevalence of STDs are less likely to mate multiply compared with species with low levels of STDs (Sheldon 1993). However, if mating behavior is promiscuous enough, then prevalences may be so high within all sections of the male population that a female is almost certain to become infected regardless of her strategy. Under such circumstances, the disease has little effect on choice, and females make the same decisions that they would make if there was no disease (see also Thrall et al. 1997). Such insensitivity to the risk of disease will only arise if the disease is highly prevalent across all sections of the male population. If heterogeneities in disease prevalence are maintained despite high transmission rates, these can lead to female choice behavior very different from that of the system without disease (consider $\delta = 1$ in Fig. 2).

Another surprising result of both of our models is that the prevalence of a disease in the population can decrease when the likelihood of disease transmission in risky contacts increases. This occurs because females are expected to respond to such a change by changing their mating strategy to reduce their exposure to risky contacts. For the disease the net effect of this can be greater than the effect of enhanced transmission success in each encounter, and so the overall prevalence of the disease declines until fidelity cannot practically evolve any stronger. The mating system therefore evolves in a way that buffers against increases in disease transmission.

Both of our models produce clear examples of frequency dependence of female behavior. In many bird populations, a fraction of broods are found to contain extrapair young, and the proportion of extrapair young varies greatly among species (from 0% to 76%; Petrie et al. 1998). In addition to asking why extrapair young are more common in some populations than others (see below), it is useful to ask why the intermediate percentages occur in the first place. Why don't all females seek extrapair mates when this is beneficial enough for some females? It is, of course, possible that some females (i.e., those mated to the best males) do not benefit from extrapair copulations and thus refrain from such be-

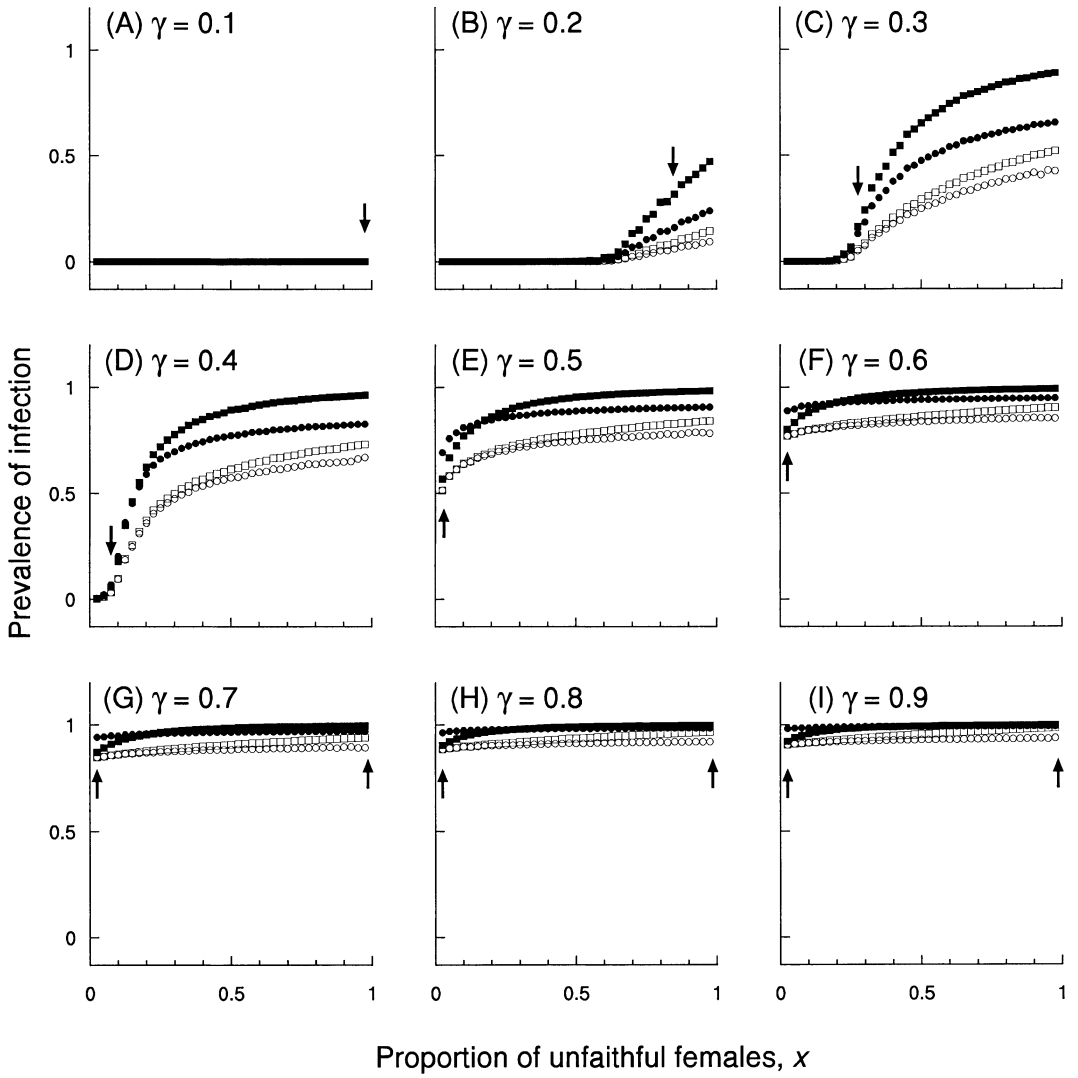

FIG. 5. The prevalences of infection for the model of Figure 4. Open and filled circles and open and filled squares give prevalence of infection in noncuckolding and cuckolding females and unattractive and attractive males, respectively, measured after the mating season before breeding commences. Arrows indicate the prevalence of infection at the evolutionarily stable equilibrium value of female cuckoldry, *x*.

havior (Kempenaers et al. 1992). Our model 2 incorporates this effect, but it also shows the possibility of true frequency dependence of the female strategy due to a feedback between female choice and disease dynamics. This can lead to intermediate equilibria where due to costs of STDs, a fraction of females do not seek extrapair fertilizations even if their own male is unattractive. STDs, however, are not the only processes leading to such intermediates. Another example is male care of young. Kokko (1999) discussed a process in which male parental care evolves according to the risk of being cuckolded. Because this risk depends on the proportion of unfaithful females, *x,* male responses to suspected cuckoldry will grow stronger as *x* increases. This, too, can stabilize an intermediate equilibrium of extrapair behavior. Shellman-Reeve and Reeve (2000) discuss a similar idea, although their model does not explicitly address frequency dependence of the female strategy.

Our model makes predictions on interspecific variation in extrapair paternity. This variation has been linked to various factors (for reviews, see Petrie and Kempenaers 1998; Hasselquist and Sherman 2001). These include genetic diversity (Petrie et al. 1998; Kokko 1999; Shellman-Reeve and Reeve 2000), the importance of male care (Kokko 1999; Møller 2000; Shellman-Reeve and Reeve 2000), the ability of males to mate-guard and to detect cuckoldry (Kokko 1999; Hasselquist and Sherman 2001), and the availability of extrapair mates as a function of breeding density and synchrony (Møller and Birkhead 1993; Stutchbury and Morton 1995). Our study adds the prevalence of sexual transmission to this list. If STDs are a strong factor influencing extrapair paternity, we would predict that extrapair fertilizations should be rarest in species with intermediate disease prevalence. At low prevalences the disease does not matter, and at highest prevalences it again matters little because almost every individual is infected.

Any modeling exercise necessitates some simplification,

and our choices leave room for further development. First, we assumed a scenario in which females who mate with attractive males produce more surviving young, but we did not include the prospect that sons inherit their father's attractiveness. If choice had this additional advantage, selection would presumably favor female choosiness more strongly. Solutions would thus be likely to simply shift toward stronger mating skews, without much qualitative difference. Second, we have assumed only horizontal transmission of the pathogen. If vertical transmission is possible, consequences of mating with an infected male become more serious, as the cost is transmitted to the next generation. We would predict lower mating skews in this case, unless the disease becomes so common through the addition of vertical transmission that the disease again does not matter. Third, we ignored the prospect that individuals may recover from the disease, and their susceptibility to disease might vary according to their immunocompetence. Such factors could lead to variability in mating behavior according to the immune status of the individual, with resistant individuals being less restrained in their mate choice. Finally, our models assume that the STD influences either mortality or fecundity but not both, and we have also ignored possible latency periods of the disease.

To our knowledge, the only previous explicit theoretical treatments of the consequences of an STD on mate choice strategies are those of Loehle (1997), Thrall et al. (1997), and Thrall et al. (2000). Below, we highlight the differences between their assumptions and predictions and ours. We have studied a very simple situation in which individuals cannot detect the infection status of others, nor can they modify their behavior in the light of their own infection status. Loehle (1997) modeled a different situation, in which females are assumed able to detect the infection status of males. He showed that a STD could then cause coevolution of male showiness and female preference for such traits. However, in his model, choosy females are a priori assumed to choose only males who are both showy and healthy and reject all others. Showiness as such does not bring about any advantage to the female but causes higher predation risk in the showy offspring. Therefore, it is difficult to see why females should not instead mate with the healthy and nonshowy males, given the assumption that they can detect the health status of a male without resorting to showiness as a signal of health. Despite the shortcomings of this particular model, we believe that handicap signaling can, in principle, evolve to reveal STDs in a similar manner to any other aspect of condition (Loehle 1995). However, the interactions between male attractiveness, mating success, and the likelihood of infection might complicate handicap signaling of health in the context of STDs in particular. The solution will clearly depend on the latency period of the disease. If males become infectious immediately after transmission but deteriorate in condition only gradually, showy (and thus popular) males could, in fact, turn out to be more infectious than others, at least if females prefer them sufficiently strongly. This, once again, suggests frequency-dependent benefits of female choice. A further complication is that sexually transmitted parasites will experience strong selection to prevent hosts from developing indicators of infection (Sheldon 1993; Knell 1999). Some STDs can even increase the attractiveness of the host, probably as a form of host manipulation by the parasite (Lockhart et al. 1996; Thrall et al. 1997).

Our model is similar to the one by Thrall et al. (1997) in that females lack means to detect the health status of a potential mate. We have assumed that the cost of the STD can be either a reduction in fecundity or mortality. Thrall et al. (1997) measure both costs and benefits in terms of fertility (the STD is assumed to cause infertility, and the benefit of mating multiply likewise lies in avoiding infertility). Also, in their model, females mate randomly with any mate and only differ in the number of mates they have, and the feedback between individual behavior and disease prevalence is not explicit. We have included an intrinsic advantage to mating with specific (high-quality) males and solved for dynamic equilibria where female behavior is optimal given the disease prevalence that the behavior itself causes. Despite these differences, the main conclusions of Thrall et al. (1997) show some similarity with ours. For example, they showed that an STD would increase selection for monogamy, but that strict monogamy is not always expected to evolve. Moreover, they also mention a situation in which a female should not attempt to avoid infection if she is already likely to be infected. These predictions were confirmed by our model.

Modeling by Thrall et al. (2000) quantified the impact of STDs on host reproductive success. They concluded, like us, that mating with males that are more popular is more risky. The Thrall et al. (2000) model was not primarily aimed at finding the causes of particular mating systems, and consequently they did not solve for optimal female behavior. Because they did not incorporate any advantage of mating with the more popular males, their model is better suited to systems in which female choice is constrained and mating groups are determined by male ownership of harems (Thrall et al. 2000). Nevertheless, their result that female fitness declines with the mating propensity of the male strengthens our conclusion of frequency-dependent female behavior. Had Thrall et al. (2000) included some intrinsic advantage to mating with the more popular males, frequency-dependent equilibria would have been likely to arise in their model too. Thus, recent theoretical work appears consistent with our results. Recently, empirical support has been accumulating for STD occurrence in captive and natural populations (Sheldon 1993; Lockhart et al. 1996; Poiani and Wilks 2000b; Westneat and Rambo 2000), although their evolutionary significance may depend on the life history of the species (Lombardo 1998). Overall, we have good reason to believe that STDs have the potential to substantially shape the evolution of mating systems, by frequency-dependent counteraction of any selective force that leads to strong mating skews.

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