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SEXUAL TRANSMISSION OF DISEASE AND HOST MATING SYSTEMS: WITHIN-SEASON REPRODUCTIVE SUCCESS

PETER H. THRALL,* JANIS ANTONOVICS,† AND JAMES D. BEVER‡

Department of Botany, Duke University, Durham, North Carolina 27708

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Abstract.—While there is an extensive literature on the evolution of mating systems in animals, little consideration has been given to the possible evolutionary interactions between sexually transmitted pathogens and the mating systems of their hosts. We use individual-based models that incorporate measurable per-contact probabilities of infection and fertilization to show that not only can the presence of sexually transmitted (and sterilizing) pathogen have significant effects on optimal within-season mating strategies for both males and females. We show that, contrary to expectations, monogamy is not always predicted to be the optimal strategy. The optimal strategy may also often differ between the two sexes, and the optimal strategy will not always be the one that minimizes disease transmission. Similarly, we show that the optimal level of virulence for a sexually transmitted pathogen is a function of the degree of promiscuity of its host. Overall, these results suggest that sexually transmitted diseases can impact host mating behavior, often in nonintuitive directions.

The enormous variation in animal mating behavior, both within and among species, has long attracted the interest of evolutionary biologists (Darwin 1871). Discussions of the evolution of mating behavior have posited a variety of ecological factors to explain this diversity of mating systems in adaptive terms (Wittenberger 1979; Wittenberger and Tilson 1980; Flinn and Low 1986). More recently, interest has grown in the evolutionary interaction between sexually transmitted diseases and host mating behavior. This increased interest is primarily for two reasons. First, there has been increasing realization that far from being a small and curious subset of diseases (Rosebury 1971; Hunter et al. 1993), sexually transmitted diseases (STDs) are common in natural populations of both plants (Mink 1993; Thrall et al. 1993, 1995) and animals (Oriel and Hayward 1974; Smith and Dobson 1992; Lockhart et al. 1996). Second, it has been speculated that mating structure itself might evolve as a consequence of disease presence, either in a quite general sense (Hamilton and Zuk 1982) or with regard to STDs in particular (Freeland 1976; Hamilton 1990; Sheldon 1993; Loehle 1995). It has even been suggested that human monogamy is an outcome of venereal disease transmission (Immerman 1986).

* E-mail: dukesmut@acpub.duke.edu.
† E-mail: danton@acpub.duke.edu.
‡ E-mail: bever@acpub.duke.edu.

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In a recent review, Loehle (1995) presented evidence of the ways in which various types of social behaviors might provide mechanisms for preventing disease spread in natural populations of animals. In particular, Loehle raised a number of interesting questions with respect to STDs and how they might influence the evolution of mating behaviors that would reduce disease transmission (e.g., through mate choice or monogamy). He hyopothesized that one of the primary reasons for the occurrence of lifetime monogamy in long-lived species of birds might be to avoid infection by sexually transmitted pathogens. Indeed, disease transmission during mating is being suggested more and more frequently as a factor that could influence mating behavior (Birkhead and Møller 1993; Hunter et al. 1993; Hardy 1994; Able 1996).

In spite of the seemingly obvious and intimate connections among host sexual activity, reproductive success, and disease risk, no rigorous theoretical studies of the role of STDs in shaping host mating behavior have been done. Likewise, the rate of transmission of a sexually transmitted pathogen (and therefore evolution of SDT virulence) will be critically dependent on the number of sexual contacts between infected and uninfected hosts, which in turn will be determined by the mating structure of the host population. For example, it has been suggested that the evolution of virulence of HIV may be guided by changes in human behavior and opportunities for transmission (Ewald 1994). In the present article, we focus on a theoretical scheme based on the idea that associated with each copulation event are a probability of disease transmission and a probability of fertilization. We incorporate different aspects of disease expression (e.g., effects on host fertility vs. mortality) and host mating systems (monogamy vs. promiscuity). We then use this theoretical framework to examine how mating system and disease severity interact to determine the reproductive success of a host and its sexually transmitted pathogen.

THE MODELS

To examine the relationship between transmission in a sexually transmitted pathogen and host reproductive success, we assume that sexual contacts can result in fertilization, infection, or both. We describe these processes on the basis of single contact or copulation events. This approach differs from that used in many disease transmission models in which a contact is defined as a sexual partner rather than a single sexual encounter and in which the transmission parameter β is defined as the probability that an infected individual will infect a susceptible partner over the course of the partnership (e.g., May and Anderson 1987; May et al. 1988; Anderson et al. 1989; Anderson 1991). Although the importance of per-contact transmission rates has been recognized in the theoretical literature (e.g., Heesterbeek and Metz 1993), the use of the per-partnership transmission rate has been largely a pragmatic decision in the modeling of human STDs (Hethcote and Yorke 1984; Garnett et al. 1992; Swinton et al. 1992), because information on per–sex act probabilities of transmission is limited. For example, it has been pointed out that, with respect to the spread of HIV-1 in Africa, data on

per–sex act probabilities of transmission are likely to be important for understanding the epidemiology of the disease (Anderson et al. 1991).

Initially, we develop expressions for within–breeding season pathogen and host reproductive success. We make several simplifying assumptions. First, we assume that disease frequency does not change during the course of a mating season (i.e., within-season disease spread is slow). Typically, because of the requirement for partner exchange (which is usually limited), sexually transmitted pathogens would be expected to have lower rates of spread than many other types of pathogens (e.g., respiratory tract infections). Lockhart et al. (1996) showed that STDs were distinguishable from other infectious diseases by having characteristics more typical of endemic diseases (e.g., less population cycling). Second, we assume that mate availability is not limiting to reproductive success of either males or females. Moreover, because sexually transmitted diseases are often sterilizing and have relatively low effects on host mortality (Lockhart et al. 1996), we assume that the negative effects of the disease are entirely in terms of effects on fertility and that there is no within-season mortality. Finally, we assume that there is no latent period for the disease; that is, individuals are infectious with respect to all mates acquired subsequent to the disease transmission event, infected males have reduced probability of fertilizing any subsequent mates, infected females (if fertilized) have reduced probability of successful reproduction (in that breeding season), and all contacts are heterosexual (as is generally the case in most animals). We ask how host and pathogen reproductive success are influenced by the number of mates, the number of copulations per mate, disease prevalence, and the effect of disease on fertility. For reference, a complete list of model parameters and definitions is given in the appendix.

Disease Transmission and Female Reproductive Success

We begin by considering the situation in which females can mate repeatedly but can have only one successful reproductive episode per breeding season. Within a single partnership (i.e., pair of individuals that have repeated sexual contact), the probabilities of a healthy unfertilized female ending up as either healthy and unfertilized ($P_{hu \Rightarrow hu}$), healthy and fertilized ($P_{hu \Rightarrow hf}$), infected and unfertilized ($P_{hu \Rightarrow iu}$), or infected and fertilized ($P_{hu \Rightarrow if}$) are as follows (see bifurcation diagram, fig. 1):

$$P_{hu \Rightarrow hu} = (1 - y)(1 - \rho)^c + y[(1 - \delta)(1 - \rho')]^c, \tag{1}$$

$$P_{hu \Rightarrow hf} = (1 - y)[1 - (1 - \rho)^c] + y(1 - \delta)^c[1 - (1 - \rho')^c], \tag{2}$$

$$P_{hu \Rightarrow iu} = y(1 - \rho')^c[1 - (1 - \delta)^c], \tag{3}$$

and

$$P_{hu \Rightarrow if} = y[1 - (1 - \rho')^c][1 - (1 - \delta)^c], \tag{4}$$

where we assume that there are c copulations within a single partnership, the per-copulation probability of infection is δ, and y is the population disease frequency. The parameter ρ is the per-contact probability that a female will be fertilized by a healthy male, and ρ' is the discounted probability of fertilization

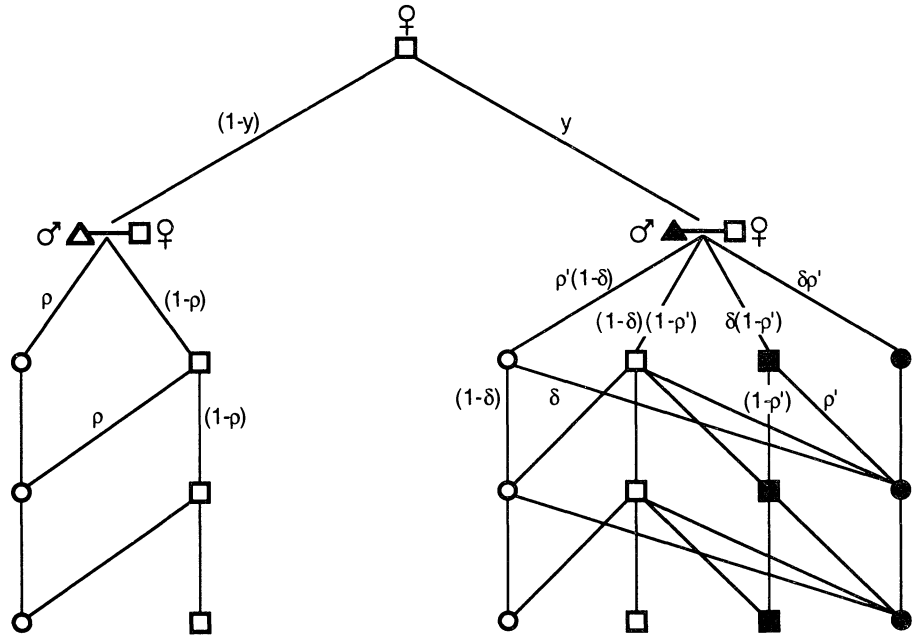

FIG. 1.—Bifurcation diagram for female reproductive success showing the possible outcomes following c contacts between an initially healthy female and either a healthy (*open triangle*) or infected (*filled triangle*) male under the assumption of a constant population disease frequency (y). In the diagram, males are represented by triangles and females by circles and squares; for both sexes, infected individuals are represented by filled symbols. For females, squares and circles represent unfertilized and fertilized individuals, respectively. The parameter δ is the per-copulation probability of infection, ρ is the per-contact probability that a female will be fertilized by a healthy male (ρ), and ρ' is the discounted probability of fertilizaton by an infected male (see the appendix for a further explanation of symbols).

by an infected male, where $\rho' = (1 - \gamma_m)\rho$ and γ_m is the degree of sterility caused by the infection in males.

With subsequent mates, other transitions are also possible because females can be in any of the above states after the first mate. Thus, the other possible transitions for females are given by

$$P_{hf \Rightarrow hf} = 1 - y[1 - (1 - \delta)^c], \tag{5}$$

$$P_{hf \Rightarrow if} = y[1 - (1 - \delta)^c], \tag{6}$$

$$P_{iu \Rightarrow iu} = (1 - y)(1 - \rho)^c + y(1 - \rho')^c, \tag{7}$$

$$P_{iu \Rightarrow if} = 1 - (1 - \rho)^c(1 - y) - y(1 - \rho')^c, \tag{8}$$

and

$$P_{if \Rightarrow if} = 1. \tag{9}$$

Note that this formulation assumes that there is no recovery from infection (i.e., the transition probability for $P_{if \Rightarrow if} = 1$).

The expression for female reproductive success can be translated into matrix algebra notation using equations (1)–(9). The complete matrix for the among-state transition probabilities (**C**) is given by

$$\mathbf{C} = (1 - y)\mathbf{H}^c + y\mathbf{D}^c,$$

where the matrices **C, H,** and **D** are as shown in figure 2.

We can extend this is to consider the entire breeding season as follows. Let m be the number of mates encountered (i.e., number of partnerships). If we multiply the matrix **C** by a vector $\mathbf{v}_0$ representing the initial distribution of females among the four possible states (healthy and unfertilized; healthy and fertilized; infected and unfertilized; infected and fertilized), we obtain

$$\boldsymbol{v}_m = \mathbf{c}^m \boldsymbol{v}_0,$$

where $\boldsymbol{v}_m$ is a vector of the probabilities of being in one of the four states at the end of the breeding season after m sexual partners. Assuming the probability of successful reproduction of a healthy female is θ and of an infected female is θ' ($= [1 - \gamma_f]\theta$ where γ_f is the degree of sterility caused by the disease in females), the seasonal fertility of a female (i.e., probability that she successfully bears offspring) is then

$$F_f = \boldsymbol{v}_m^T \boldsymbol{v}_w,$$

where $\boldsymbol{v}_w$ is the vector of weights used to calculate female fitness (fig. 2). Note from $\boldsymbol{v}_w$ in figure 2 that the reproductive success of infected females is discounted at the end of the breeding season rather than for each sexual contact, as it is for males (see next section).

Disease Transmission and Male Reproductive Success

We can calculate the reproductive success of males within a breeding season by an analogous procedure. For male reproductive success, there are only two initial states (healthy or infected). We assume unlimited sperm supplies such that whether a male successfully fertilized the previous female is irrelevant to the outcome of the current mating and that there is no displacement of sperm from the female's last sexual encounter. Although in nature females are likely to become limited as the breeding season progresses and the number of successfully mated females increases, here we assume that no such limit exists (i.e., regardless of how many mates a male has, the probability of choosing a healthy and unfertilized female does not change).

We begin by calculating the overall probability of a male remaining healthy ($P_{h \Rightarrow h}$) following c contacts with a female. This probability is straightforwardly calculated from the bifurcation diagram shown in figure 3, where

$$P_{h \Rightarrow h} = 1 - y + y(1 - \delta)^c, \tag{10}$$

and the probability of becoming infected ($P_{h \Rightarrow i}$) is simply $1 - P_{h \Rightarrow h}$. We assume that infection is not instantaneous in the sense that males that become infected have the same probability of fertilization as healthy males (ρ) during all subse-

A. Multiple Contacts with a Single Partner

$$H = \begin{bmatrix} 1-\rho & 0 & 0 & 0 \\ \rho & 1 & 0 & 0 \\ 0 & 0 & 1-\rho & 0 \\ 0 & 0 & \rho & 1 \end{bmatrix}$$

$$D = \begin{bmatrix} (1-\rho')(1-\delta) & 0 & 0 & 0 \\ \rho'(1-\delta) & 1-\delta & 0 & 0 \\ (1-\rho')\delta & 0 & 1-\rho' & 0 \\ \rho'\delta & \delta & \rho' & 1 \end{bmatrix}$$

B. Mate States

$$C = \begin{bmatrix} P_{hu\to hu} & 0 & 0 & 0 \\ P_{hu\to hf} & P_{hf\to hf} & 0 & 0 \\ P_{hu\to iu} & 0 & P_{iu\to iu} & 0 \\ P_{hu\to if} & P_{hf\to if} & P_{iu\to if} & P_{if\to if} \end{bmatrix}$$

C. Total Fitness within a Breeding Season

$$V_m = \begin{bmatrix} P_{\to HU} \\ P_{\to HF} \\ P_{\to IU} \\ P_{\to IF} \end{bmatrix}, \qquad V_0 = \begin{bmatrix} 1 \\ 0 \\ 0 \\ 0 \end{bmatrix}, \qquad V_w = \begin{bmatrix} 0 \\ \theta \\ 0 \\ \theta' \end{bmatrix}$$

FIG. 2.—Description of matrices and vectors used to derive within-season female fitness functions. *A*, Contacts with a single partner: **H** and **D** are per-contact transition matrices for contacts with a healthy or infected male, respectively. *B*, Mate states: the matrix **C** gives among-state transition probabilities, following c contacts with a single partner (*hu*, healthy, unfertilized; *hf*, healthy, fertilized; *iu*, infected, unfertilized; *if*, infected, fertilized); for example, $P_{hu\Rightarrow if}$ is the probability of a healthy but unfertilized female becoming both infected and fertilized. *C*, Total within–breeding season fitness: the vectors v_m, v_w, and v_0 represent, respectively, the total probabilities of being in one of the four possible states (hu, hf, iu, if) at the end of the breeding season, weights used to calculate female fitness, and the initial distribution of females (see the appendix for all parameter definitions).

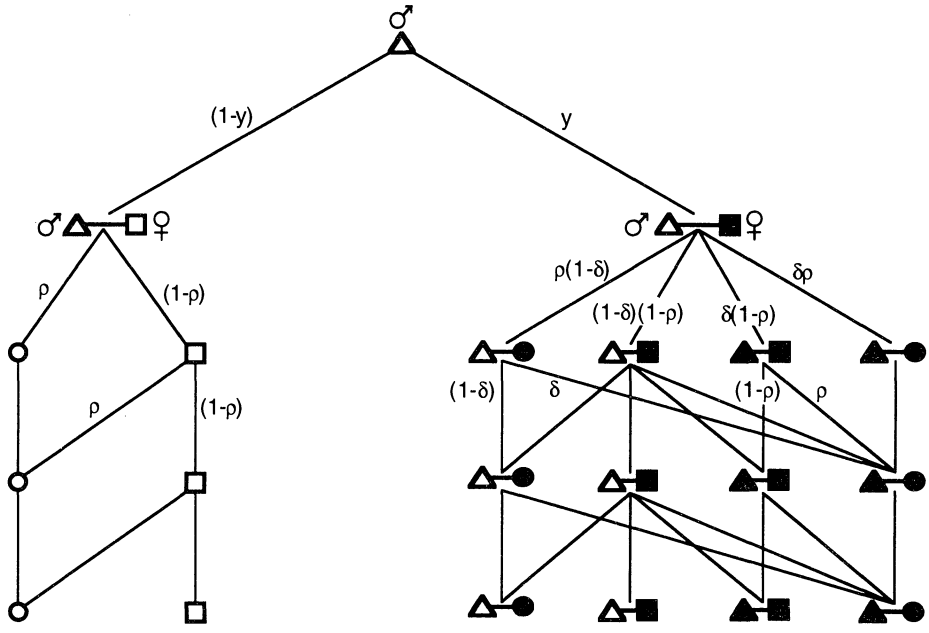

FIG. 3.—Bifurcation diagram for male reproductive success showing the possible outcomes following c contacts between an initially healthy male and either a healthy or infected female. All symbols and parameters are the same as those defined for figure 1.

quent contacts with that female. Once infected, however, contacts with all subsequent females result in fertilization with probability ρ' rather than ρ.

Using the bifurcation diagram shown in figure 3 and equation (10), we can also calculate the joint probabilities associated with fertilization of females and infection of males. These joint probabilities can then be divided by the total probability of a male remaining healthy ($P_{h\Rightarrow h}$) or becoming infected ($P_{h\Rightarrow i}$) to derive the conditional probabilities associated with each mate (note that the probability of remaining infected if already in that condition is one since there is no recovery from infection):

$$P(\text{hf/h} \Rightarrow \text{h}) = (1 - y)[1 - (1 - \rho)^c]/P_{h\Rightarrow h},$$

$$P(\text{if/h} \Rightarrow \text{h}) = (1 - \delta)^c[1 - y(1 - \rho)^c]/P_{h\Rightarrow h}, \tag{11}$$

$$P(\text{if/h} \Rightarrow \text{i}) = y[1 - (1 - \rho)^c][1 - (1 - \delta)^c]/P_{h\Rightarrow i}, \tag{12}$$

$$P(\text{hf/i} \Rightarrow \text{i}) = (1 - y)(1 - \delta)^c[1 - (1 - \rho')^c],$$

and

$$P(\text{if/i} \Rightarrow \text{i}) = [1 - (1 - \rho')^c][1 - (1 - y)(1 - \delta)^c]. \tag{13}$$

For example, P(hf/h $\Rightarrow$ h) is the probability that a male that remained healthy fertilized a healthy female; P(if/h $\Rightarrow$ i) is the probability that a male that became infected fertilized an infected female.

Because females that have been fertilized have the possibility of subsequently mating with other males, the reproductive success of males must be discounted by the probability that a fertilized (but healthy) female will go on to become infected by one of her subsequent mates. At the upper limit, we assume that females encounter a total of m mates, and in this case the discounting function (D_H) is given by

$$D_H = \theta P_{hf\Rightarrow hf}^{m-1} + \theta'(1 - P_{hf\Rightarrow hf}^{m-1}), \tag{14}$$

where $P_{hf\Rightarrow hf}$ is the probability that a healthy fertilized female stays healthy after c contacts with a mate (eq. [5]). Note that the discounting term given in equation (14) equals θ if either $P_{hf\Rightarrow hf} = 1$ (e.g., if females do not continue to mate once they have been successfully fertilized) or the disease does not cause sterility ($\theta' = \theta$). However, in general, the reproductive success of males must also take into account the discounted fertility of fertilized but infected females. Therefore, in the summary expression for male reproductive success (see below), the probabilities given by equations (11), (12), and (13) must be multiplied by θ'.

We develop the summary recursion for male reproductive success by first defining the conditional expectations for the fitness of initially healthy males that remain healthy after c contacts with a female, initially healthy males that become infected, and males that are infected at the start of the breeding season, where

$$E(rs/h \Rightarrow h) = P(hf/h \Rightarrow h)\, D_H + P(if/h \Rightarrow h)\theta',$$

$$E(rs/h \Rightarrow i) = P(if/h \Rightarrow i)\theta',$$

and

$$E(rs/i \Rightarrow i) = P(hf/i \Rightarrow i)\, D_H + P(if/i \Rightarrow i)\theta'.$$

As an example, E(rs/h $\Rightarrow$ i) is the expected fitness of a male that becomes infected following sexual contact with an infected female; the probability that she will produce his offspring is the product of the probability that he successfully fertilized her (P[if/h $\Rightarrow$ i]) and her own discounted probability of bearing offspring (θ'). The overall reproductive success (RS) of a male that is initially healthy at the beginning of the breeding season is then calculated as follows: given that a male is healthy at the beginning of the breeding season, after c encounters with a single female, male reproductive success is given by

$$RS = P_{h\Rightarrow h}E(rs/h \Rightarrow h) + P_{h\Rightarrow i}E(rs/h \Rightarrow i); \tag{15}$$

after two mates, reproductive success is

$$\begin{aligned} RS = {} & 2P_{h\Rightarrow h}^2 E(rs/h \Rightarrow h) + P_{h\Rightarrow h}P_{h\Rightarrow i}[E(rs/h \Rightarrow h) + E(rs/h \Rightarrow i)] \\ & + P_{h\Rightarrow i}[E(rs/h \Rightarrow i) + E(rs/i \Rightarrow i)]; \end{aligned} \tag{16}$$

and after three mates, reproductive success is

$$\begin{aligned} RS = {} & 3P_{h\Rightarrow h}^{3}E(rs/h\Rightarrow h) + P_{h\Rightarrow h}^{2}P_{h\Rightarrow i}[2E(rs/h\Rightarrow h) + E(rs/h\Rightarrow i)] \\ & + P_{h\Rightarrow h}P_{h\Rightarrow i}[E(rs/h\Rightarrow h) + [E(rs/h\Rightarrow i) + E(rs/i\Rightarrow i)] \\ & + P_{h\Rightarrow i}[E(rs/h\Rightarrow i) + 2E(rs/i\Rightarrow i)]. \end{aligned} \tag{17}$$

As the number of mates increases, so does the number of possible pathways along which male reproductive success can be calculated. Simple inspection of the pattern developed in equations (15)–(17) allows derivation of the summation giving total within-season reproductive success for males that are healthy at the beginning of the breeding season (F_{hm}), and with m mates:

$$\begin{aligned} F_{hm} = {} & mP_{h\Rightarrow h}^{m}E(rs/h\Rightarrow h) + P_{h\Rightarrow i}\sum_{j=1}^{m} P_{h\Rightarrow h}^{j-1}[(j-1)E(rs/h\Rightarrow h) \\ & + E(rs/h\Rightarrow i) + (m-j)E(rs/i\Rightarrow i)]. \end{aligned} \tag{18}$$

Calculation of reproductive success for males that are infected at the beginning of the breeding season (F_{im}) is relatively simple because there are only two paths by which infected males may fertilize females:

$$F_{im} = mE(rs/i\Rightarrow i).$$

In general, the derivations are more complicated for males than for females because of the assumption that males can fertilize multiple females during a breeding season. This means that calculation of reproductive success requires knowledge of intermediate states and the final state of a male at the end of the breeding season; this situation is not easily translated into matrix notation.

RESULTS

Female Mating Behavior

Within-season reproductive success.—If there are no costs associated with having additional contacts or mates, then, as expected, in the absence of disease (or if the disease has negligible sterilizing effects on infected hosts, i.e., $\gamma \approx 0$), reproductive success increases with both number of copulations per mate and number of mates to an asymptote given by θ. However, when the disease has a large effect on host fertility, there are likely to be multiple optima for reproductive success. Figure 4 shows within-season fitness surfaces, where the left-hand rear face of each surface represents situations in which females have a single contact with many mates (= promiscuity), while the right-hand front face represents situations in which females have many contacts with a single mate (= monogamy). For small values of γ, asymptotes at each of these faces are approximated by $\theta(1-\gamma)$ and $\theta(1-\gamma y)$ respectively. The asymptote for monogamy is greater than the asymptote for promiscuity whenever the disease is present and there is a negative effect of the disease on fertility ($0 < y < 1$ and $\gamma > 0$). However, for larger values of γ there are intermediate optima, such that neither asymptote

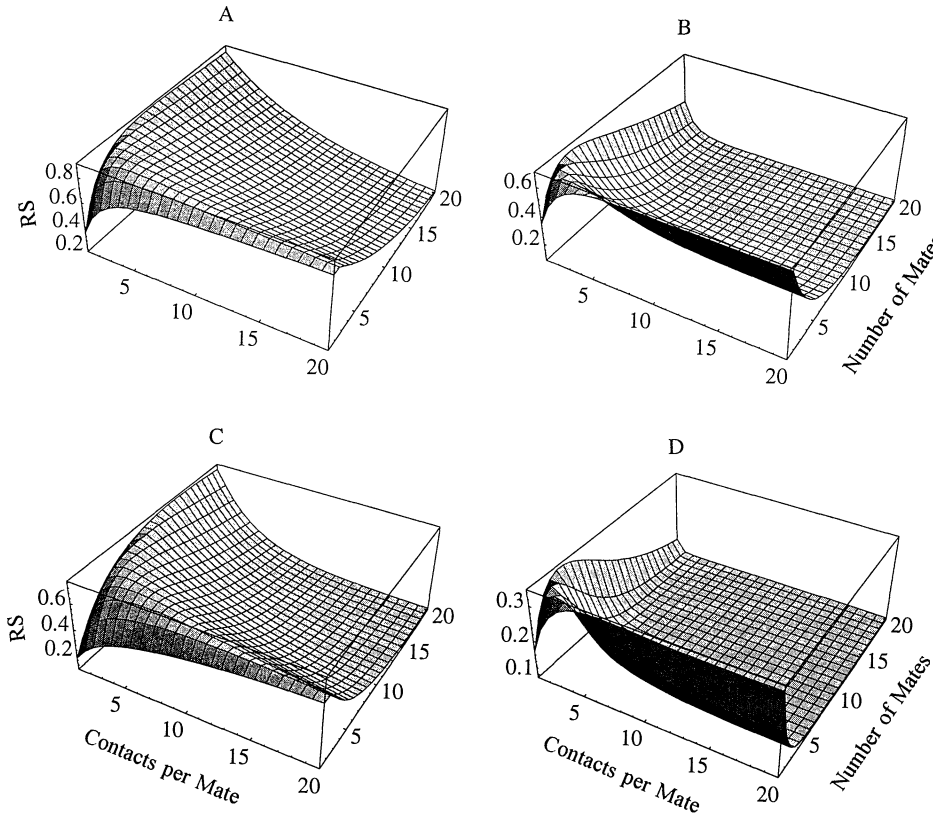

FIG. 4.—Within-season fitness surfaces for female reproductive success for different values of the per-contact probability of infection (δ) and population disease prevalence (y). *A*, $\delta = 0.02$, $y = 0.3$; *B*, $\delta = 0.2$, $y = 0.3$; *C*, $\delta = 0.02$, $y = 0.7$; *D*, $\delta = 0.2$, $y = 0.7$. In all of these figures, we assume that there is no cost to additional contacts or mates but that there are large negative effects of infection ($\gamma = 0.9$ for all surfaces). We further assume that the per-contact probability of fertilization by males ($\rho = 0.5$) and the probability that a female will successfully produce an offspring ($\theta = 0.95$) are held constant.

necessarily represents the maximum within-season reproductive success (fig. 4*B*, *D*). As the probability of infection increases, having either multiple contacts or multiple mates becomes less advantageous; this effect is more pronounced as the degree of sterility caused by infection increases (not shown). When both the population decrease prevalence and the per-contact probability of infection are high (fig. 4*D*), reproductive success is maximized by having multiple contacts with a single mate.

It is extremely unlikely in nature that either the number of sexual contacts per mate or the number of mates is unlimited. For example, in many cases, there are likely to be costs associated with an increased number of contacts (e.g., decreased foraging time, limited sperm supplies, energy expended on the mating act itself, risk of predation; Daly 1978; Rowe et al. 1994) or with switching mates (e.g.,

costs associated with finding new mates, risk of disease; Daly 1978; Hunter et al. 1993). Alternatively, positive effects may be associated with having additional mates (e.g., increased skill at finding new mates through learned behavior, enhanced ability to detect infection in potential mates, previous success increasing success with subsequent mates). We can easily encapsulate these possibilities by assuming that the total number of contacts per season, k, is given by

$$k = mc - m\alpha, \tag{19}$$

where α is the cost (or benefit, for $\alpha < 0$) associated with acquiring a new mate. This assumes the simplest case in which mate acquisition costs are constant throughout the season.

If α is unity or greater (fig. 5*A*) and the per-contact probability of infection (δ) is small, then female reproductive success is maximized at an intermediate number of mates. When δ is larger, there are two local maxima for female reproductive success, which suggests the possibility of individuals becoming "stuck" at suboptimal strategies. If α is less than unity or negative (fig. 5*B*), then for small values of δ, there is still an intermediate optimum number of mates, but for larger values of δ monogamy is always favored. Similarly, if δ is fixed and the degree of sterility caused by infection (γ) is allowed to vary, females have highest reproductive success with multiple matings if γ is small, but monogamy is optimal when γ is large.

The preceding results assume that females are healthy at the beginning of the breeding season. It is also of interest to ask whether the optimal reproductive strategy of an initially infected female would be different. Given that there is no recovery from infection and that there are no costs to additional contacts or mates, then the reproductive success of females infected at the beginning of the season will clearly be maximized when both c and m are large, regardless of the per-contact probability of infection or the degree of sterility caused by infection (i.e., they have nothing to lose). If we assume a trade-off between contacts and mates of the form given by equation (19) with $\alpha = 1$ (therefore a cost to additional mates), then there is no clear optimum but a broad range for the optimal number of mates (with $m > 1$).

Probability of infection.—In general, under the assumption that per-contact probabilities of infection are the same from males to females as for females to males, the probability of infection for females (following c copulations with a single mate) is the same as that for males. The overall probability of becoming infected for a female who has m mates during a breeding season is simply

$$1 - (1 - P_{h \Rightarrow i})^m, \tag{20}$$

where $P_{h \Rightarrow i}$ is the per-mate probability of infection given earlier (see eq. [10]).

Male Mating Behavior

Within-season reproductive success.—For males, reproductive success is a function of the total number of females with whom they mate and who then successfully produce offspring. Similar to the results for females, under the assumption of no costs to multiple contacts or mates and negligible disease effects,

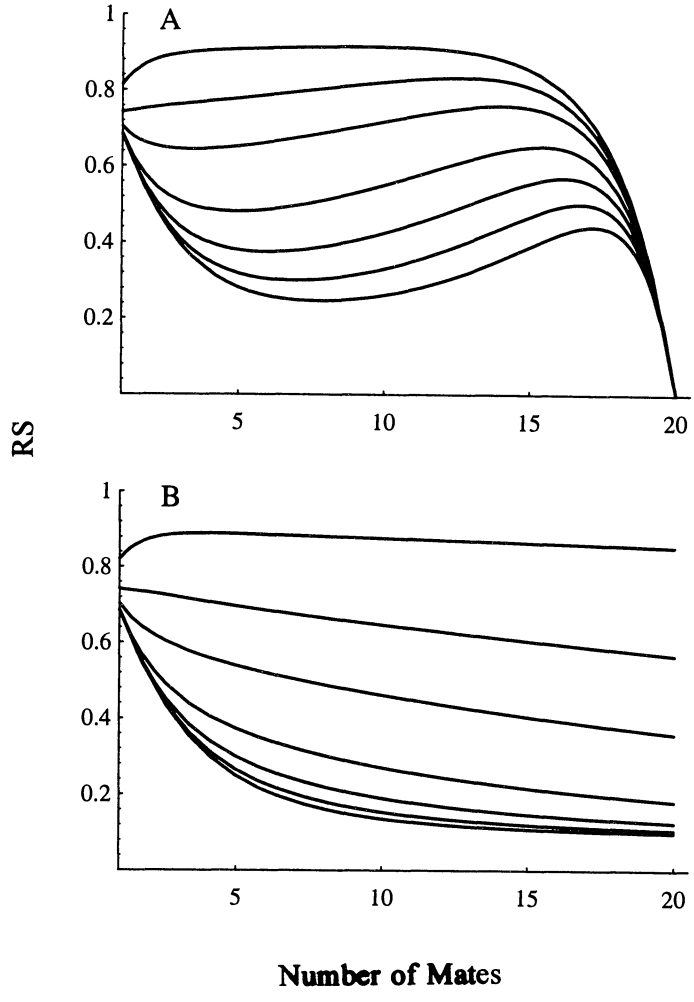

FIG. 5.—Within-season reproductive success as a function of per-contact probability of infection and number of mates for initially healthy females, assuming a trade-off between number of contacts per mate and number of mates (i.e., $c = k/m - \alpha$). *A*, $\alpha =$ (i.e., there is a cost to acquiring additional mates); *B*, $\alpha = -1$ (i.e., there is a benefit to acquiring additional mates). In both figures, lines represent fixed values of δ (from top to bottom, $\delta = 0.01$, 0.05, 0.1, 0.2, 0.3, 0.4, and 0.5, respectively), and all other parameters are fixed ($y = 0.3$, $\gamma = 0.9$, $\rho = 0.5$, and $\theta = 0.95$).

males have maximum reproductive success when both the number of contacts per mate and the number of mates are high. When infection has severe effects on fertility (large values of γ) but δ is small (fig. 6*A*, *C*), then male reproductive success is maximized with few contacts per mate and many mates regardless of disease prevalence. For larger values of δ but with low disease prevalence (fig. 6*B*), there is more than one local optimum; a male should have many mates but can achieve relatively high reproductive success by either having few or many

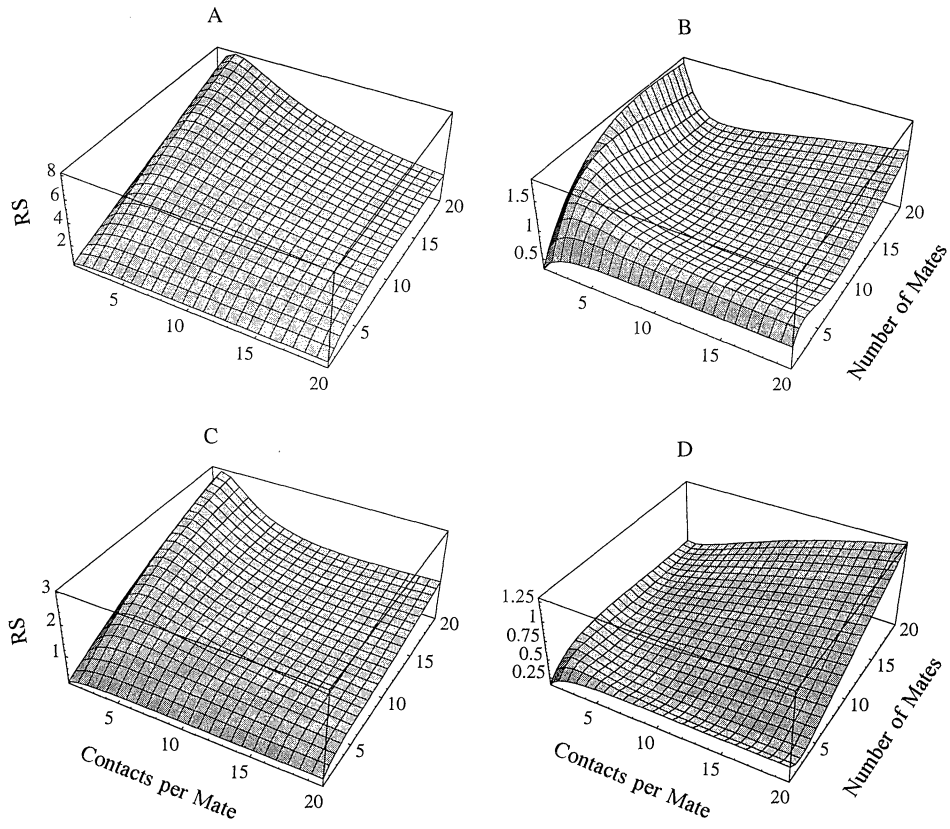

FIG. 6.—Within-season reproductive success for initially healthy males, assuming no trade-off between number of contacts and number of mates but large negative effects of infection. For all surfaces shown, $\gamma = 0.9$, $\rho = 0.5$, and $\theta = 0.95$. *A*, $\delta = 0.02$, $y = 0.3$; *B*, $\delta = 0.2$, $y = 0.3$; *C*, $\delta = 0.02$, $y = 0.7$; *D*, $\delta = 0.2$, $y = 0.7$.

contacts per mate. If both the per-contact infection rate and disease prevalence are high (fig. 6*D*), then initially healthy males are likely to become diseased early, and (as with initially infected males) they should mate as much as possible.

In contrast, if we assume a cost to having additional mates and use the trade-off function given by equation (19), then for all parameter combinations of γ and δ that we examined, the reproductive success of initially healthy males is maximized for intermediate values of m (fig. 7*A*). Therefore, the expectation that males should always maximize the number of mates is not borne out in the more realistic situation (i.e., numbers of contacts or mates are not unlimited). When there are benefits to having additional mates, then for most values of δ, males should mate with as many females as possible; at very high values of δ, male reproductive success will be optimized for values close to unity (fig. 7*B*).

In the results presented earlier, for purposes of calculating the discounting expression D_H in equation (14), we assume that females are promiscuous (i.e.,

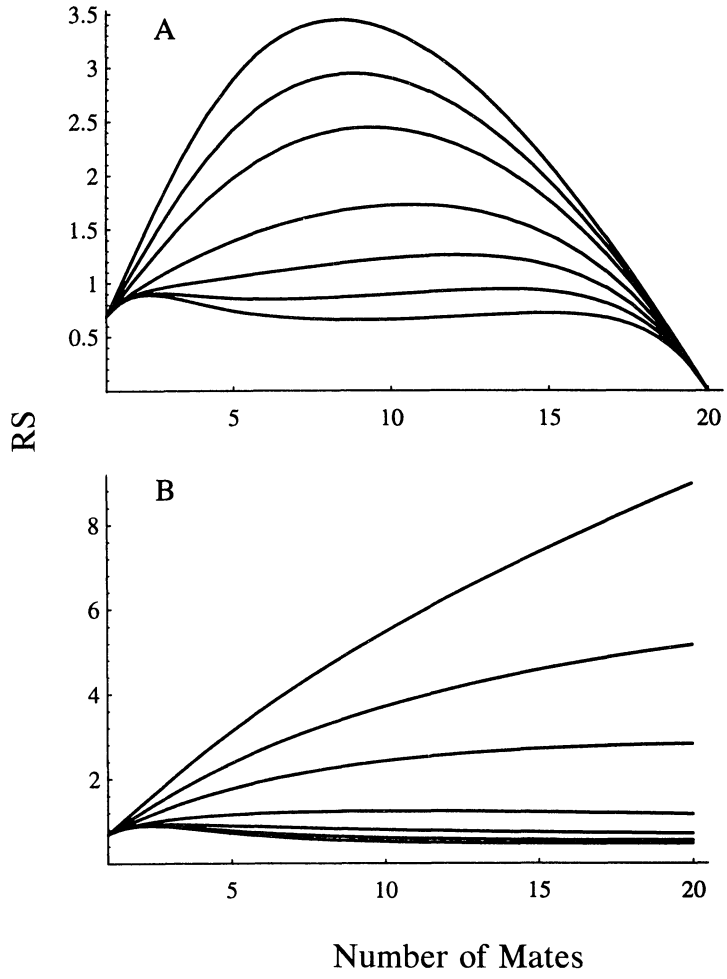

FIG. 7.—Within-season reproductive success as a function of per-contact probability of infection and number of mates for initially healthy males, assuming that $c = k/m - \alpha$. *A*, $\alpha = 1$ (i.e., there is a cost to acquiring additional mates); *B*, $\alpha = -1$ (i.e., there is a benefit to acquiring additional mates). In both figures, lines represent fixed values of δ (from top to bottom, $\delta = 0.01$, 0.05, 0.1, 0.2, 0.3, 0.4, and 0.5, respectively), and all other parameters are fixed ($y = 0.3$, $\gamma = 0.9$, $\rho = 0.5$, and $\theta = 0.95$). All parameter values are the same as those for figure 5.

always have m mates). While incorporating the number of mates (for females) into the model as a variable would complicate the formulations considerably, we can easily explore the contrasting situation in which females are completely monogamous. In this case, the discounting term (D_H) reduces to θ. When there are no costs to additional mates, so that both c and m can potentially be quite large, the reproductive success of initially healthy males increases with increasing m and c; when costs are assumed, male reproductive success is maximized in a

manner qualitatively similar to that in the situation in which females are assumed to be promiscuous (see fig. 7*A*). It is interesting that in the case in which females are assumed to be promiscuous, for very high values of δ, male reproductive success is maximized for values of m close to unity; this does not occur when females are assumed to be monogamous (and, instead, intermediate numbers of mates are always optimal for males).

Probability of infection.—If the probability of infection following sexual contact with a single mate is the same for males as for females (see above), then the overall probability of infection within a breeding season is the same as that for females (see eq. [20]).

DISCUSSION

The issue of how sexually transmitted diseases should interact with the mating systems of their hosts has been, until recently (Sheldon 1993; Loehle 1995), largely ignored in the literature on mating system evolution. This study provides an initial step toward developing a quantitative framework for examining mating system–STD interactions that can take into account a range of behaviors between the extremes of promiscuity and monogamy. We do this using biologically meaningful (and measurable) per-contact probabilities of fertilization and infection as a basis for exploring the problem. The present study confirms that STDs can potentially have major impacts on host mating system evolution and that, conversely, host mating systems may greatly influence the evolution of pathogen characteristics. However, we also show that the outcomes are often not intuitively obvious. For example, although we confirm that it is reasonable to expect that promiscuity will lead to higher rates of transmission and disease prevalence than will monogamy, the corresponding expectation that monogamy will always evolve in the presence of STDs (Freeland 1976; Immerman 1986) is not borne out. We have shown that the situation is more complex, with a possibility of multiple optima such that similar high levels of reproductive success can result from different mating behaviors. For example, in females, when per-contact infection rates are high, having multiple mates results in a high probability of infection. Therefore, a female can either minimize her probability of becoming diseased by being monogamous or be promiscuous and maximize her probability of being fertilized regardless of the high probability that she will become diseased (see fig. 5*A*). In other words, if the first mate is likely to be infected (because population disease levels are high), then the female may be better off maximizing her reproductive success by having many mates. This leads to the expectation that there may be more variability in degree of female promiscuity at high levels of disease.

The simple assumptions of our models lead to the prediction that both males and females should be highly promiscuous in the absence of disease. Therefore, our result that the optimal mating system in the presence of disease may differ between males and females indicates that disease alone (rather than, say, costs of rearing offspring) can influence the evolution of differences between the sexes in mating behavior. For example, if we assume females have at most a single successful reproductive episode per breeding season, in the presence of a steriliz-

ing STD, females should generally be monogamous, and males should be completely promiscuous. However, because these optima have been calculated independently for males and females, the actual evolutionary outcome is difficult to predict; this prediction would require other approaches that incorporate the joint optimization of male and female reproductive success.

An interesting biological difference between the infection/fertilization process in males and females is that the reproductive success of a female does not depend on whether the male with whom she mates becomes subsequently infected (either by herself if she is diseased or by another diseased female). On the other hand, male reproductive success does depend on whether the female with whom he copulated becomes infected during that mating or during subsequent matings in that breeding season. This asymmetry occurs because the female usually harbors the zygote for a period after fertilization (in our case, until the end of the breeding season). Selection for different mating behaviors in males and females may therefore be at least in part disease driven and not simply a consequence of different physiological demands or ecological risks associated with rearing offspring.

One area of evolutionary biology in which mating system and disease have been explicitly considered is the evolution of secondary sexual characteristics. Hamilton (1990) and Hamilton and Zuk (1982) suggest that secondary sexual characteristics may have evolved as a signal of good health. However, given that the disease in question is sexually transmitted, the evolutionary expectations may be quite different. For example, because attractive males are likely to be doing the majority of the mating, they may be more likely to carry an STD. Therefore, it is conceivable that females that avoid mating with such males may actually have a greater reproductive success. Correspondingly, males with conspicuous secondary sexual features may be healthier (now) because they have been avoided (in the past) by females. Furthermore, one might expect sexually transmitted pathogens themselves to increase the attractiveness of their hosts since this would clearly increase their transmission. Certainly, there are numerous records of parasites altering either host behavior and/or appearance to enhance their own transmission (Moore 1984; Dobson 1988), and some STDs are known to increase the attractiveness or sexual activity of infected hosts (Lockhart et al. 1996). For example, dourine, a sexually transmitted trypanosome in horses, causes infected stallions to increase their level of sexual activity relative to healthy males. A bacterial STD in cattle, bovine genital campylobacteriosis, results in abnormally long estrous cycles in infected cows, and, in herd situations, bulls will mate repeatedly and for longer periods than would occur in healthy herds, presumably because infected females do not conceive. Maintenance of genetic variation in secondary sexual characteristics in males may therefore be driven in part by the interactions of such traits with sexually versus nonsexually transmitted pathogens (Able 1996).

Another obvious but rather interesting result to emerge from our analyses is that the optimal mating system may not be the one that reduces disease transmission the most. In all cases that we examined, monogamy resulted in the lowest disease transmission, yet monogamy was clearly not always the favored mating strategy. Therefore, hosts may evolve mating strategies that are optimal with

regard to their own fitness, yet these self-same strategies may permit substantial disease spread and persistence. Furthermore, because mean contact number is always the same between the two sexes (and assuming male-to-female transmission equals female-to-male transmission), disease incidence will also be independent of sex even though the two sexes may have quite different mating behaviors. It should be noted that few, if any, rigorous studies have been done of sex-specific per-contact probabilities of infection for STDs in animal populations. In humans, it has been suggested that single-exposure probabilities of infection may often be higher from males to females than from females to males because of ejaculate retention in the vagina (e.g., gonorrhea: Hook and Handsfield 1990; HIV and HTLV: Ewald 1994).

Even though our analyses only concerned within-season reproductive success, the latter may be proportional to lifetime reproductive success under certain conditions. If we assume that fecundity and survival of adults remain constant after they reach reproductive age (e.g., a situation approximated in many species of birds, at least during middle age; Forslund and Pärt 1995), then we can represent the growth rate of a population of hosts (X) as

$$\frac{dX}{dt} = S_O FX - \mu X,$$

where S_O, F, and μ are the survival of newborns to reproductive age, per capita reproductive output, and adult death rate, respectively. In this situation, lifetime reproductive success (LRS) is given by

$$\text{LRS} = \frac{S_O F}{\mu}.$$

Usually F and μ are considered "normal" birth and death rates, but they can also be functions that include disease-induced sterility or mortality. If we also assume that disease frequency remains constant throughout the host's life span (i.e., the disease frequency is at equilibrium or changing very slowly relative to the lifetime of the host), then the disease would reduce the fecundity of the host in a proportional manner every breeding season. Under these circumstances, the LRS of the host is directly proportional to the within-season reproductive success.

As mentioned earlier, our current formulations are simplified in that we calculate male and female reproductive success independently of each other. Although this assumption precludes us from making general predictions as to how mating systems will evolve, it may not be unreasonable in several specific cases. This assumption implies that either access to mates is not limited (e.g., mating occurs with only a small fraction of the alternative mates) or that mates are sampled with replacement (and, as assumed throughout, disease frequency does not change throughout the season). In most real-world situations, these assumptions are probably only reasonable for females. However, it should be noted that the mean RS of the focal sex (calculated on the assumption that the other sex is not limiting) will by definition equal the mean RS of the other sex, but the form of the distribution of individual mating behaviors and how they sum to give that mean RS in the

other sex will be undefined. For example, consider the situation in which males are not limiting and we determine reproductive success in females. If females sample males with replacement, then we would expect number of mates per male to follow a Poisson distribution, with most males having few mates but a few males having many mates. The consequences of variance in the number of mates for disease transmission therefore also needs to be evaluated. However, in the context of the models developed here (i.e., assuming that disease levels within a season remain unchanged), it can be seen from equation (18) that disease transmission is independent of the distribution of m.

However, if there is substantial within-season disease transmission (or transmission is essentially continuous and aseasonal, as in humans), reproductive success is likely to be strongly influenced by variance in the number of contacts. Those individuals with a large number of mates (or partners) may contribute disproportionately to disease transmission, and transmission becomes a function of both the mean and variance of the number of partners (May and Anderson 1987; Anderson et al. 1989). A similar argument would apply to lifetime reproductive success, if there were among-season covariance among individuals in their number of mates. Differences in variance are often used as a basis for mating system classification. For example, polygyny (one male mating with several females) implies that the variance in mating success of males is much greater than that of females (given a 50:50 sex ratio); under these situations, within-season disease transmission may be underestimated by only taking into account the average number of mates.

Although we do not present formal analyses, it can be readily shown that pathogen reproductive success will always increase with the number of mates and number of contacts per mate, but there will be a decreasing benefit per sexual contact per partnership as c increases. If various trade-offs are present (e.g., between virulence and the per-contact probability of transmission) and if the total number of copulations per season is constant (see eq. [19]) then, as expected, there is some intermediate level of virulence that maximizes pathogen fitness. The precise level of virulence will depend on both the form of the cost function and the mating system. It has been generally argued that pathogen virulence (defined as a negative effect of the pathogen on host fitness) will increase with transmission opportunity as long as the transmission mode remains the same (Ewald 1988, 1990, 1994); our studies confirm that STDs are no exception.

Although it is easy to speculate on the potential coevolutionary interactions between STDs and animal mating systems, almost no explicit tests of the relationship between STDs and host mating systems have been performed. It has been shown (Thrall et al. 1993) that pollinator-transmitted anther smuts were more common in plant species that were outcrossing and had larger flowers. Smith and Dobson (1992) have suggested that the variation among mammalian host species in mating behavior may correlate with the degree of sexual transmission of the bacterial pathogen *Brucella* spp. (Nielsen and Duncan 1990). Moreover, Sheldon (1993) has postulated that mating behaviors in birds (e.g., cloacal inspection, the likelihood of extrapair copulations) might be related to the risk of infection through sexual transmission. One clear example from "cultural evolution" is the

recent shift in human mating behavior due to the widespread fear of AIDS (Ostrow 1990; Schwartz and Gillmore 1990).

Application of our models to real-world situations minimally requires detailed knowledge of not only the mating system but also the per-contact transmission and fertilization rates. We know of no system (other than humans) in which these aspects have been investigated, let alone characterized quantitatively. Tests of model predictions regarding the impact of STDs on mating system evolution may require the development of experimental systems. Recently, Hurst et al. (1995) demonstrated that the mite *Coccipolipus hippodamiae* is transmitted among adult two-spotted ladybugs (*Adalia bipunctata*) during mating in laboratory populations; moreover, behaviors observed in *Adalia* during mating may well be the result of evolution to avoid mite transmission or to avoid copulating with already infected mates. Whisler (1968) studied sexual transmission and effects on host longevity of a fungal disease (*Stigmatomyces baeri,* Laboulbeniales) found on the fly *Fannia canicularis*. Such sexually transmitted fungal diseases appear to be commonplace in insects (Thaxter 1896; Strandberg and Tucker 1974; Whitney 1982) and may provide some of the best experimental systems in which to study the numerical and evolutionary dynamics of STDs.

While it has frequently been argued that mating and social structure are determined by the distribution of resources and avoidance of predators, pathogens may have an equally important influence (Dobson 1988; Dobson and Lyles 1989). Our study provides a theoretical framework for studying the interactions between sexually transmitted pathogens and their hosts. Our results indicate that STDs can, at least in theory, impact on host mating behavior; however, it remains to be seen how these results relate to the diversity of mating systems actually observed in nature.

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APPENDIX

TABLE A1

DEFINITIONS OF MODEL PARAMETERS AND SYMBOLS

Symbol	Definition
Within-season reproductive success:	
c	Number of sexual contacts per mate
m	Number of mates encountered
y	Population-level disease prevalence
k	Total number of sexual contacts available (assumed to be fixed)
α	Cost/benefit of added mates when a trade-off between c and m is assumed (see eq. [19]); $\alpha > 0$ = cost, $\alpha < 0$ = benefit
δ	Per-contact probability of infection
γ	Fractional reduction in the probability of fertilization for infected males; reduction in the probability of successfully bearing offspring for infected females
ρ	Per-contact probability of fertilization for healthy males
ρ'	Per-contact probability of fertilization for infected males ($= \rho[1 - \gamma]$)
θ	Probability that a healthy fertilized female successfully produces offspring
θ'	Probability that an infected (and fertilized) female produces offspring ($= \theta[1 - \gamma]$)
Lifetime reproductive success:	
X	Number of host individuals
S_0	Survival of newborns to reproductive age
F	Per-capita reproductive output (= within–breeding season reproductive success)
μ	Adult death rate
μ'	Death rate of infected hosts
β	Disease transmission coefficient

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