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Sexually transmitted diseases in polygynous mating systems: prevalence and impact on reproductive success

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Studies of disease in relation to animal mating systems have focused on sexual selection and the evolution of sexual reproduction. Relatively little work has examined other aspects of ecological and evolutionary relationships between host social and sexual behaviour, and dynamics and prevalence of infectious diseases; this is particularly evident with respect to sexually transmitted diseases (STDs). Here, we use a simulation approach to investigate rates of STD spread in host mating systems ranging from permanent monogamy to serial polygyny or polyandry and complete promiscuity. The model assumes that one sex (female) is differentially attracted to the other, such that groups of varying size are formed within which mating and disease transmission occur. The results show that equilibrium disease levels are generally higher in females than males and are a function of variance in male mating success and the likelihood of a female switching groups between mating seasons. Moreover, initial rates of disease spread (determining whether an STD establishes in a population) depend on patterns of host movement between groups, variance in male mating success and host life history (e.g. mortality rates). Male reproductive success can be reduced substantially by a sterilizing STD and this reduction is greater in males that are more 'attractive' to females. In contrast, females that associate with more attractive males have lower absolute fitness than females associating with less attractive males. Thus, the potential for STDs to act as a constraint on directional selection processes leading to polygyny (or polyandry) is likely to depend on the details of mate choice and group dynamics.

Keywords: social and sexual behaviour; disease dynamics; mating system evolution

1. INTRODUCTION

The extensive debate about evolutionary impacts of disease on animal and plant reproductive systems has centred almost entirely on the evolution of sex (Hamilton *et al*. 1990; Howard & Lively 1994) or on sexual selection (Hamilton & Zuk 1982; Borgia & Collis 1990; Loehle 1997), particularly the role of disease in sustaining genetic variance in male quality. With few exceptions (Freeland 1976; Loehle 1995; Thrall *et al*. 1997), the literature has been silent regarding other ecological and evolutionary interactions between animal mating systems and disease. This is surprising because variation in mating systems results in changing contacts between individuals of different sexual and social status, and thus has important consequences for infectious-disease spread (Anderson *et al*. 1989; Anderson 1991; Thrall & Antonovics 1997; Thrall *et al*. 1998). Conversely, the presence of a sexually transmitted disease (STD) can influence male and female reproductive success, and thus may be important in determining mating-behaviour evolution (Guldbrandtsen 1997; Thrall *et al*. 1997). In the context of STDs in human populations, it is well understood that disease spread (Hethcote 1976; Anderson *et al*. 1989), and perhaps the evolution of virulence (Ewald 1990, 1994; Van Baalen & Sabelis 1995), is influenced by shifts in human sexual behaviour. It has even been speculated that human monogamy is an outcome of STD transmission (Immerman 1986; Immerman & Mackey 1997).

It is probable that diseases have failed to be considered as an important factor in the evolution of animal mating systems not because of direct evidence for their lack of effect on host fitness (Grenfell & Dobson 1995; Clayton & Moore 1997) but because of the effort and interdisciplinary commitment required to obtain evidence for pathogen incidence in the context of behavioural studies of mating systems. Moreover, it is only recently appreciated that STDs are much more abundant in animal populations than previously thought (Lockhart *et al*. 1996) and that such diseases may be an important component of population regulation. For example, some koala populations are endangered because of the high prevalence of chlamydial infections (Canfield *et al*. 1991) and in humans the AIDS epidemic in Africa has the potential to cause population growth rates to become negative over the next few decades (May *et al*. 1988; Anderson *et al*. 1991).

We have previously shown that STDs can differentially affect the reproductive success of males and females with contrasting mating systems (Thrall *et al*. 1997). However, in that study we considered only within-season reproductive success, contact number was deterministic and we assumed that mate availability did not limit either male or female reproductive success. These assumptions permitted analytical solutions but were unrealistic in that the theory could only be applied to special cases. More

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commonly, disease spread will occur over many seasons, contacts between individuals will be stochastic with variable contact numbers among individuals and the number of contacts may be limited by mate availability.

Here we use a computer simulation to examine the relationships between mating systems, rates of spread of STDs and the impact of STDs on individual reproductive success. This approach allows us to consider disease spread over multiple seasons, includes stochastic effects associated with group formation and host mortality, and mate limitation (by assuming an equal sex ratio). We focus on a simple model of a polygynous (or polyandrous) mating system, which we represent by allowing the variance in contact number to be greater in one sex (designated as males for the remainder of the paper) than in the other. Mating groups are reformed each season, and different degrees of 'serial polygyny' are represented by allowing intergroup migration of a fraction of the females between breeding seasons. Disassociation and reassociation of groups, or changes in group membership, can also occur as a result of demographic turnover. We investigate how the degree of polygyny influences the spread of an STD, its equilibrium prevalence in the two sexes and how the disease impacts on the fitness of individuals with different mating frequencies. In particular, we ask whether the presence of an STD can act as a 'brake' on the evolution of extreme polygyny.

2. THE MODEL

We model a polygynous system where males vary in their propensity to acquire females (the C code is available from P.T. on request). Females have only one male partner per season but may change groups in successive breeding seasons. Such situations are exemplified by the mating systems of red deer (Clutton-Brock *et al*. 1982), elephant seals (LeBoeuf 1974), impala (Jarman 1974) and wild horses (Rubenstein & Wrangham 1986). Group formation in polygynous systems may occur through multiple behavioural pathways (Altman *et al*. 1977); here we indicate how our assumptions fit into Altman *et al*.'s (1977) classification. We assume that groups are formed by variation in male quality (Altman *et al*.'s assumption I) and that there is no female competition (although we later assume that adding more females to a group decreases the reproductive success of those females; assumption III). We also assume that a female associates with only one male per season (assumption V). To minimize the number of parameters, and generate a simple model of polygyny, we are neutral to Altman *et al*.'s assumptions II, IV and VI. Thus we assume that male quality and the per-capita reproductive success of the female are uncorrelated, that there are no advantages to females choosing previously mated high-quality males and that the order in which females choose males does not impact on their fitness. A simple biological translation of our model would be that it represents males that differ in their abilities to occupy territories of different sizes, and that females associate with males in direct proportion to territory size.

With regard to the disease, we assume that the latent period extends beyond the mating season (i.e. no secondary transmission among members of a group within a season), that the disease affects fertility but not mortality and that there is no recovery. Although the impacts of STDs on their hosts can be very variable, these assumptions reflect in a general way many of the characteristics of such diseases in animals (Lockhart *et al*. 1996).

(a) *Simulation protocols*

(i) *Formation of mating groups*

Mating propensities (relative ability to attract females) are assigned randomly to males according to a log-normal distribution, which allows us to easily produce a range of mating structures (monogamy to harem polygyny). The mean of this distribution is always one, but we adjust the variance to represent different degrees of polygyny. At the beginning of each season, mating groups are created by choosing females to be associated with males in direct proportion to the male mating propensities using the following procedure. Each male is given a segment on a linear scale between 0 and 1 the length of which is directly proportional to his mating propensity. For each female, a random number is generated between 0 and 1, and she is assigned to the male within whose linear segment the random number falls. This procedure continues until all females are assigned to males. We note, however, that some males may end up with no females. Unless otherwise noted, we generally assume that there are a total of 250 males and 250 females in the population.

It is important to note that this procedure of selecting mating groups has the effect that even when the variance in male mating propensity is zero, the number of females per male follows a Poisson distribution, i.e. a propensity variance of zero does not represent 'pure' monogamy but a polygynous system where, in each mating group, the mean and variance of number of females per male is 1. Such distributions have been reported in the literature as representing the random association of females with males (Hartley & Shepherd 1995).

(ii) *Disease transmission*

In spite of considerable exploration of the disease-transmission process in human sexual interactions (Anderson *et al*. 1989; Anderson 1991), theoretical studies have largely taken a phenomenological approach, compressing the potential complexity of contact events into the single, notoriously simplistic, transmission parameter, β (e.g. Anderson & May 1981; Getz & Pickering 1983; Thrall *et al*. 1993). Here we use a 'per mating' measure of transmission, δ, defined as the probability that a male acquires disease when mating with a diseased female, and vice versa (we assume equal female-to-male and male-to-female transmission rates). For simplicity, we further assume that a male mates with all females in his group, such that the probability that a male becomes diseased is given by $1-(1-\delta)^{Y_i}$ where Y_i is the number of diseased females in the ith group. The probability that a female becomes diseased if the male in that group is diseased is simply δ, reflecting the assumption that the latent period of the disease is longer than the mating season. This is biologically reasonable, given the relatively short mating season for most animals. Note that δ either represents a per-contact transmission rate assuming one contact per mating pair, or, if there are several contacts per pair, then δ is a function of the number of contacts per pair and the per-contact probability of infection (cf. Thrall *et al*. 1995, 1997; Antonovics *et al*. 1995).

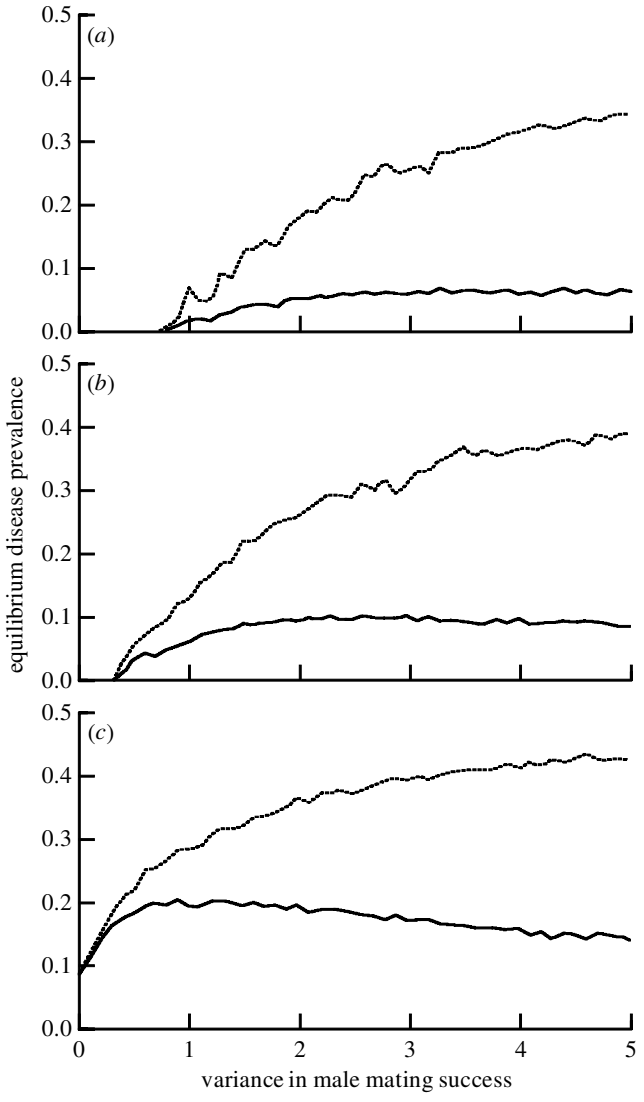

Figure 1. Equilibrium disease prevalence as a function of variance in male mating success, for different degrees of intergroup female movement, with the per-season transmission rate (δ) = 0.125; (*a*) 0% female movement, (*b*) 10% movement (25 individuals per breeding season), (*c*) 100% movement (250 individuals per breeding season). Dotted lines represent prevalence in females and solid lines show prevalence in males. We assume that the host mortality rate = 0.1 and the total population size (n) = 500.

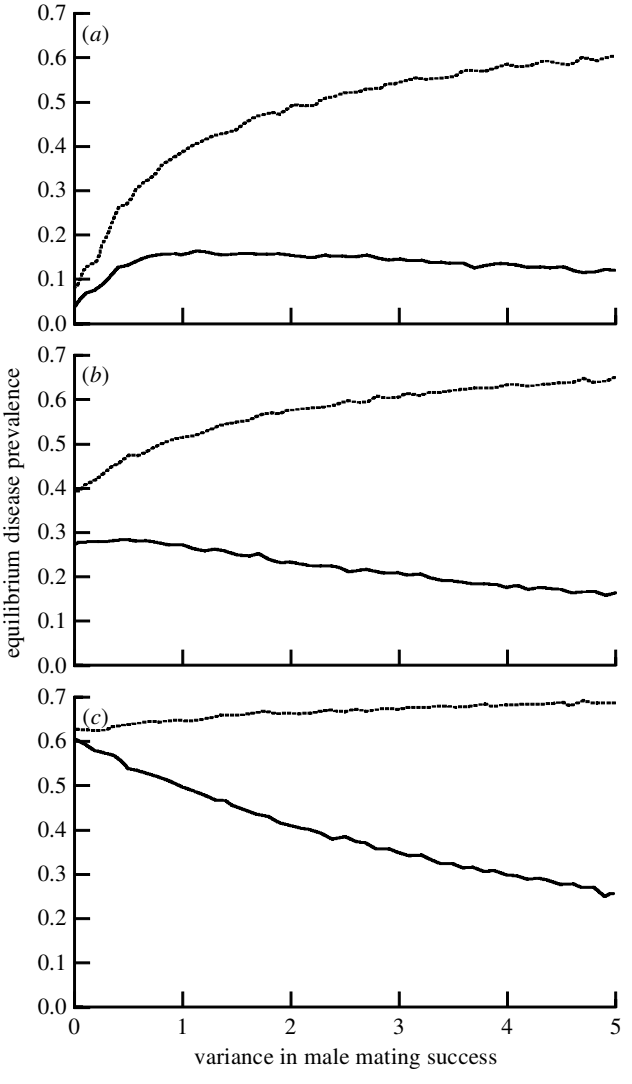

Figure 2. Equilibrium disease prevalence as a function of variance in male mating success, for different degrees of intergroup female movement, with the per-season transmission rate (δ) = 0.3; (*a*) 0% female movement, (*b*) 10% movement, (*c*) 100% movement. Dotted lines represent prevalence in females and solid lines show prevalence in males. We assume that the host mortality rate = 0.1 and the total population size (n) = 500.

(iii) *Reproductive success and fitness*

We assume infection is sterilizing and that there is no host recovery or disease-induced effects on host mortality. We explore two scenarios: in the first case, we assume that per-capita reproductive success is independent of group size or composition. We therefore consider that matings between healthy individuals result in unit reproductive success of the male and female, while contacts that involve at least one diseased individual result in zero reproductive success. Female reproductive success is therefore equal to the number of healthy males that she mates with during her lifetime. For both males and females, reproductive success is therefore measured as the number of healthy individuals mated with before acquiring disease.

In the second case, we explore a situation in which we make the more realistic assumption that individual fitness is directly dependent on group size (Kempenaers 1995; Smith *et al*. 1994), such that within each breeding episode, per-capita reproductive success for females is given by $\rho e^{-\alpha(Ni-1)}$, where ρ, α and N_i respectively represent the per-mating probability of fertilization (= 1 in the results presented here), the strength of group-size effects on reproduction and the total number of females in the ith group. Similarly, per-capita male reproductive success is given by $X_i \rho e^{-\alpha(Ni-1)}$ (X_i is the number of healthy females).

In each simulation run, after 250 generations, when disease prevalence has stabilized, we identify each newly recruited male or female (see § 2(a)(v)) and follow its mating history with other healthy individuals, until it dies or becomes diseased. The cumulative count of matings with other healthy individuals then represents a measure of the reproductive success of that individual. We repeat this for the next 250 generations for all newly recruited

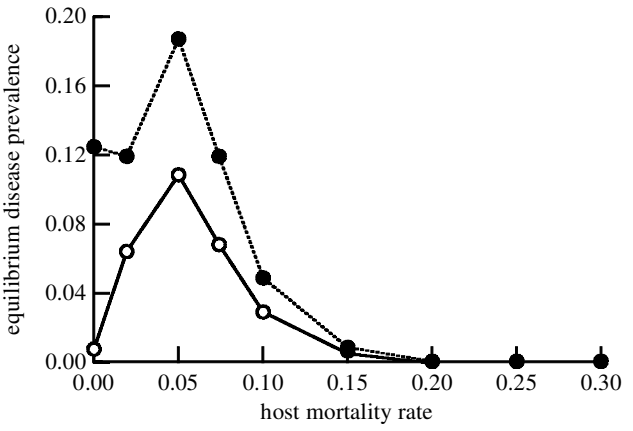

Figure 3. Effect of mortality on disease prevalence in males (solid line) and females (dotted line). We assume a variance in male mating success of 1.0, 10% (25 individuals) female movement, per-season transmission rate = 0.3 and a total population size of 500 individuals.

males and females and calculate the mean and variance in reproductive success.

(iv) *Movement among groups*

After each mating season, a certain fraction of the females are chosen from the groups at random and assigned to a pool of females. These are then reassigned to new groups at the start of the next season according to the propensity of the males in those groups to acquire females. At one extreme, 0% movement represents lifetime monogamy or permanent 'harem' polygyny (depending on the variance in male mating success), while at the other extreme, 100% movement represents complete promiscuity or 'serial' polygyny. Thus, by varying both the degree to which females move between groups and variance in male mating success, we are able to easily generate a wide range of realistic mating situations.

(v) *Host birth and death*

Demographic turnover is imposed by assuming that a fraction of the males and females die each year after the mating season and are replaced by an equal number of healthy males and females recruited into mating groups at the start of the following season. Both population size and sex ratio are therefore held constant. Females that die are replaced in a manner identical to females that migrate between groups; female recruits are put into a pool of females that are then assigned to males as described in §2(a)(i). Females from groups where the male has died are also placed in the pool and then reassigned to males. Males that die are replaced by healthy males with new mating propensities; the mating propensities of all males are then relativized on a linear scale from 0 to 1 and unattached females are allocated according to these values.

For each simulation, a single diseased male and female are introduced into the population, and the simulation run for 500 generations, by which time disease prevalence has generally stabilized apart from stochastic fluctuations. Unless otherwise stated, the following 'canonical' values were used: variance in male mating propensity = 2; per-mating disease transmission rate, $\delta = 0.3$; and group turnover due to mortality = 0.1. Although lower values of δ resulted in qualitatively similar outcomes, disease was much less likely to establish when the variance in male mating success was also low. Similarly, high turnover rates meant that the disease was less likely to spread from the initially infected hosts. The canonical values were therefore chosen to allow a reasonable chance of disease establishment and persistence.

We focus on the consequences of the following variables for disease spread: variance in male mating propensity (increasing variance indicates an increase in the degree of polygyny); per-mating disease-transmission rate; fraction of females migrating between groups at the end of each season; and group turnover due to host mortality.

3. RESULTS

(a) *Disease prevalence*

Equilibrium disease prevalence was nearly always considerably higher in females than in males (figures 1 and 2). With a high variance in male mating success there were always males that did not mate with any (or with only a few) females and these males were unlikely to become infected. Therefore the low prevalence in males was the result of a large fraction of unmated males in this group. At very low variances, transmission was only from a few females for any given male. Thus, in many cases, prevalence in males was greatest for intermediate levels of variance in male mating success; this was particularly evident for lower values of the per-mating-season transmission rate ($\delta = 0.125$; figure 1*b*,*c*). In females, there was an asymptotic nonlinear relationship between disease prevalence and variance in male mating success (figures 1 and 2); above a certain variance in mating success, prevalence remained uniformly high.

Prevalence in both males and females generally increased with greater female migration among groups, especially when variance in male mating success was low (figures 1 and 2). At this point the system approximated monogamy, and increased female migration represented increased promiscuity. When the transmission parameter, δ, was fixed at 0.3, prevalence in males was often highest when the variance in mating success was zero (figure 2*b*,*c*). We note, however, that simulations with $\delta = 0.125$ often resulted in failure of the disease to spread at low levels of female movement (figure 1*a*,*b*).

Disease prevalence in both males and females was highest at intermediate levels of mortality (figure 3). When mortality was generally low, an increase in mortality had the effect of increasing group turnover, with the result that diseased females were then assigned to other groups; this increased overall transmission. When mortality became very high, however, the duration of the infectious period decreased, and it was more difficult for the disease to persist (Thrall *et al*. 1993), and therefore overall prevalence again fell.

(b) *Disease transmission*

To estimate the effect of mating system on rates of disease spread, the disease was introduced into the population as either one diseased female or as one diseased male, and the number of new infections generated after a

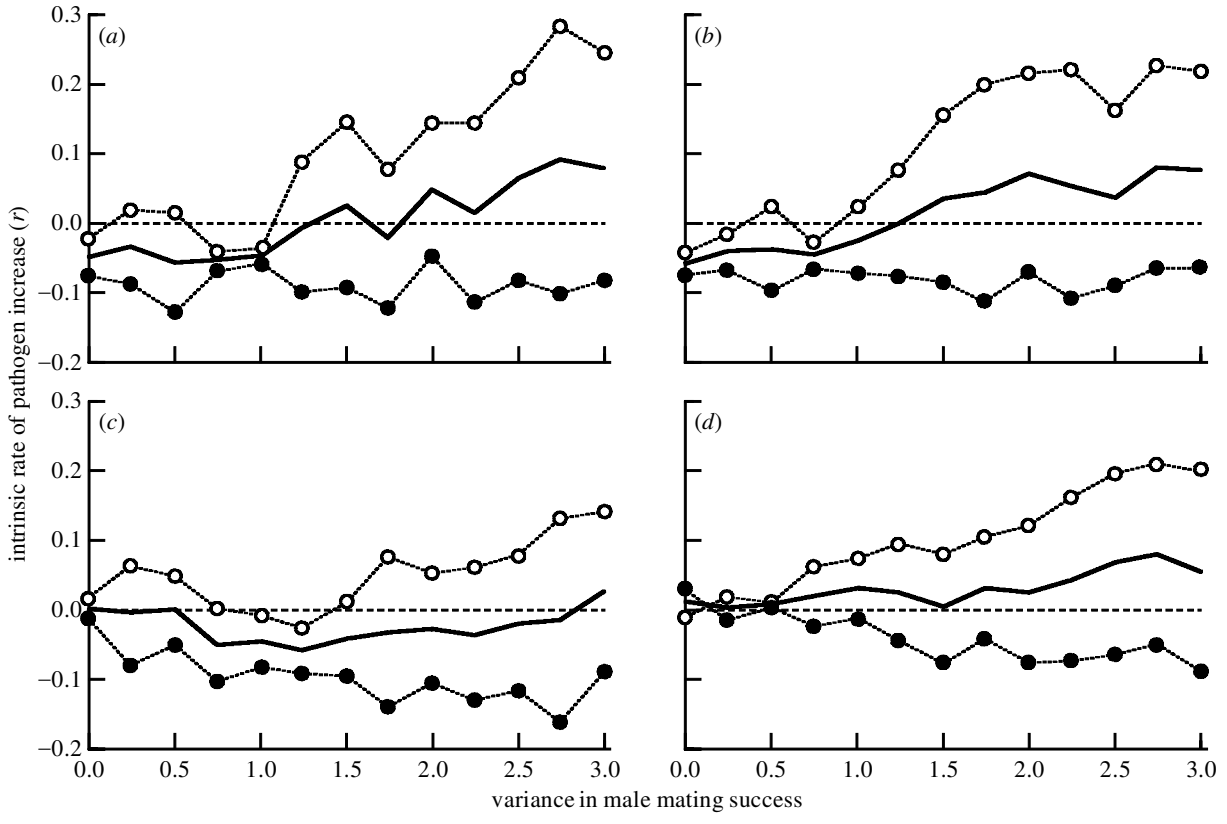

Figure 4. The effect of variance in male mating success and different degrees of female movement on rates of disease spread when the disease is introduced as one diseased female (open circles, dotted line) or one diseased male (solid circles, dotted line). The solid line is the average of these values. Disease spread is calculated as the intrinsic rate of increase of the pathogen (number of new infections generated in the next generation by one initial infection) and each point represents an average of 1000 simulations. (*a*) No female movement between breeding seasons, (*b*) 0.4% female movement (one individual), (*c*) 10% female movement (25 individuals), (*d*) 100% female movement. We assume a per-season transmission rate = 0.3, mortality rate = 0.1 and $n = 500$.

small number of generations by this one initial infection was estimated for 1000 replicate runs. The average rate of initial disease increase for these runs was calculated as follows: given the initial number of diseased individuals ($Y_0 = 1$) and the number of diseased individuals after t mating cycles (Y_t), then the intrinsic rate of initial disease increase (r) is given by

$$r = \sqrt[t]{Y_t} - 1, \qquad (1)$$

from the well-known relationships, $Y_t = Y_0\lambda^t$ and $\lambda = 1 + r$. For the results presented here, $t = 3$ mating cycles; this was the minimum number of cycles required for disease to spread from an initially infected female to a male and back into the female population (or vice versa for simulations where disease was introduced in a male).

When introduced in males, the disease failed to spread in the population, whereas when introduced in females, the disease always had a positive rate of increase (except for very low variances in male mating success; figure 4). Rates of disease spread when introduced in males generally decreased with increasing variance in male mating success, because at high variances, disease was more likely to initially be in a male that failed to acquire any mates. When introduced in females, disease spread increased with increasing variance in male mating success, because such females were more likely to find themselves in a group with a male and other females, where transmission opportunities were large. Indeed, the effect of variance in male mating success was greater when female movement between groups was smaller, presumably because high migration evened out effective group sizes. Thus, the average rate of pathogen increase increased with variance in male mating success when intergroup migration was low (figure 4), whereas it was relatively unaffected by variation in male mating success when migration between groups was high.

(c) *Male and female reproductive success*

The presence of an STD sharply curtailed male reproductive success and the effect was larger for more attractive males, as well as for higher levels of female movement (figure 5). However, under all the conditions we investigated where fitness was assumed to be independent of group size, there was still a positive relationship between mating propensity and male fitness (figure 5*a*). Therefore, the effect of disease was to reduce the selection gradient acting on mating propensity, rather than curtail it completely or favour an intermediate optimum. However, under the biologically more realistic assumption that the per-mating-season fitness of females was a decreasing function of group size, then, as expected, overall male fitness was reduced and an intermediate optimum was favoured (figure 5*b*). For higher values of α,

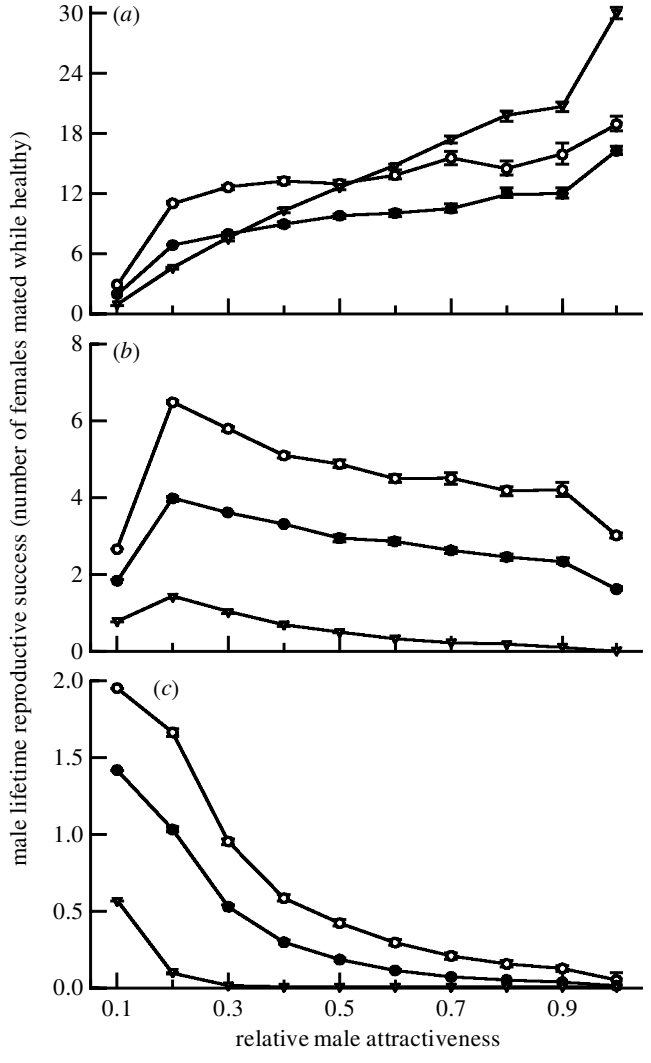

Figure 5. The relationship between male reproductive success (average lifetime number of offspring sired) and male attractiveness in the presence of a sexually transmitted disease; (*a*) assuming per-capita female reproductive success does not vary with group size, (*b*) assuming that per-capita female reproductive success declines exponentially with group size, with $\alpha = 0.1$, and (*c*) assuming that $\alpha = 0.5$. Open circles represent 1% female movement, filled circles represent 10% female movement and open triangles represent 100% female movement. We assume per-season transmission rate = 0.3, mortality rate = 0.1 and a total population size of 1000.

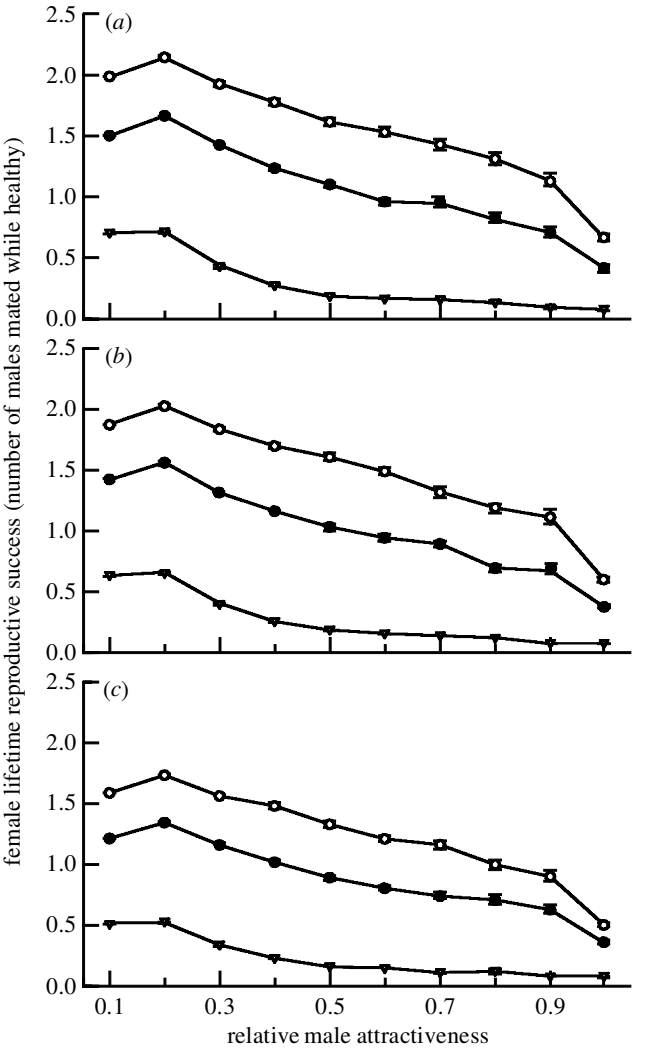

Figure 6. The relationship between female reproductive success (average lifetime number of offspring) and the average attractiveness of the males with which those females associated over their lifetimes, in the presence of an STD; (*a*) assuming per-capita female reproductive success does not vary with group size, (*b*) assuming that per-capita female reproductive success declines linearly with group size, with $\alpha = 0.1$, and (*c*) $\alpha = 0.5$. Within each graph, open circles represent 1% female movement, filled circles represent 10% female movement and open triangles represent 100% female movement. We assume per-season transmission rate = 0.3, mortality rate = 0.1 and $n = 1000$.

the relationship between lifetime male fitness and attractiveness became negative, and males with low levels of attractiveness had the highest lifetime fitness (figure 5*c*).

In contrast to the situation seen in males, the presence of a sterilizing STD always resulted in a negative relationship between the average attractiveness of males that a female associated with and her lifetime fitness (figure 6). This was true even in the case where we assumed that reproductive success was independent of group size (figure 6*a*). When reproductive success was assumed to be a function of group size, then the difference in lifetime fitness between females associated with low- and high-attractiveness males was reduced, but the relationship was still negative (figure 6*b*,*c*). As with male lifetime fitness, increased levels of female movement led to greater overall reductions in fitness.

4. DISCUSSION

We have used a simple model to investigate factors affecting disease spread in polygynous mating systems. Our primary interest is not in the behavioural causes of particular mating systems, but in the consequences of polygyny for STD transmission and, conversely, in the impact of such diseases on reproductive success. We ignore complicating factors such as age- or experience-dependent behaviours (Oring *et al.* 1992), relatedness of group members and other forms of individual heterogeneity, and only consider two mating-system parameters

Table 1. *Dependence of the reproductive rate, R_0, of an individual pathogen on both mating structure and social contacts*

(R_0 is the product of partners per lifetime, copulations per partner (times per-copulation probability of transmission) and number of non-sexual encounters (times per-encounter probability of transmission). g_0, the average size of mating groups; c, number of copulations per partner; μ, the instantaneous mortality rate; N, total number of individuals summed over the mating groups within a larger social group; δ_o, the transmission parameter for an ordinary infectious disease when contacts are between individuals from different mating groups; and δ_w, the transmission parameter for non-sexual contacts between members of the same mating group. Note that group size refers to number of individuals with which a focal individual associates.)

mating structure	group size	partners per lifetime	copulations per partner	non-sexual encounters
monogamy				
permanent	1	1	c	$\delta_o(N-1)+\delta_w$
serial	1	$1/\mu$	c	$\delta_o(N-1)+\delta_w$
polygyny				
permanent	g_0	g_0	c/g_0	$\delta_o(N-g_0)+\delta_w g_0$
serial	g_0	g_0/μ	c/g_0	$\delta_o(N-g_0)+\delta_w g_0$
promiscuity	N	N/μ	c/N	$\delta_o N$

that are likely to influence disease transmission: variation in male mating success, and variation in female fidelity to males (these are readily measurable by field studies). We consider only one demographic parameter, mortality rate (and assume, because population size is constant, it equals the recruitment rate), but show that this is crucial to understanding disease spread and prevalence; mortality results in decreased disease transmission opportunities, increases recruitment rates of individuals into groups, and (especially where it acts on males) results in group dissolution and females dispersing to other groups in the following season.

As variance in male mating success (i.e. the degree of polygyny) increases, there is a greater rate of disease spread from females (relative to that from males) as a source of infection. This reflects the high likelihood that a female will be in a group with other females, but the low likelihood that a random male will be in a group with a large number of females. At high variances in male mating success, many males remain unmated (or 'poorly' mated), thus equilibrium disease prevalence in males as a whole is less than that in females. Females are therefore more likely to be a route of disease spread in polygynous systems than males; correspondingly we might predict that STDs have evolved mechanisms to transmit more efficiently via females than males in polygynous systems. We know of no empirical study that has tested the differential prevalence of STDs in males and females in polygynous systems. Further empirical and theoretical studies are needed to determine the consequences of differential prevalence, and contribution of the sexes to disease spread, on models of mate choice based on traits that directly or indirectly signal disease condition (Loehle 1997).

Models of STDs in groups where there is heterogenity in sexual behaviour show that disease transmission increases with increasing variance in partner exchange rates such that the net transmission coefficient of an STD is a function of both the mean number of partners and the variance in number of partners per unit time (Anderson & May 1991). It is tempting to apply this generalization to polygynous systems, with the expectation that increasing polygyny (variance in male mating success) would increase disease transmission. While this expectation is borne out in some situations, there are important exceptions and caveats. Thus increasing variance in male mating success does increase rates of disease transmission but only when there is very little intergroup migration of females (harem polygyny); when there is substantial intergroup migration, variance in male mating success has little effect on disease spread. This somewhat surprising result is explained by the fact that at low rates of intergroup migration, overall disease spread is largely a function of group size. Thus in groups where males monopolize a large number of females disease spread is rapid, whereas when the groups are small and relatively isolated, disease spread in the population as a whole is restricted. When there is substantial female migration between groups, the different group sizes (caused by high variance in male mating success) have little effect and are homogenized by high rates of 'pathogen flow' (cf. gene flow).

Males that are effective at attracting females are more likely to become diseased than unmated males. Not surprisingly, our results confirm the intuitive expectation that STDs (and other diseases transmitted during mating aggregation) may have debilitating effects on dominant males (Graves & Duvall 1995) and hasten their replacement by subordinates. However, unless females suffered reproductively by joining multi-female groups, there was no evidence that STDs would limit the evolution of male traits that increased polygyny. Even in the presence of an STD, the most attractive males still had the highest reproductive success when fitness was independent of group size. Nevertheless, the selection gradient on polygyny is much lower in the presence of an STD, suggesting that while there may be no limit, evolutionary rates may be slowed (and less resilient to correlated effects of other characters).

Our initial simulations made the simplifying assumption that individual reproductive success was independent of group size or composition. This is almost certainly not true in many real-world systems, and indeed variation in individual reproductive success as a function of group composition has been considered to be the major source of selection for individual behaviours that favour or disfavour association into particular group structures. The polygyny threshold model (Orians 1969) argues that females will join groups until the costs of group membership exceed the

benefits of association with a dominant male. There is empirical evidence for reduced reproductive success or survival of females that join groups where females are already present (Kempenaers 1995; Smith *et al*. 1994) and under the polygyny threshold model one might expect that per-capita reproductive success declines as group size increases. When we did simulations assuming that per-capita reproductive success of females declines as group size increases, then highly attractive males showed disproportionately reduced fitness in the presence of a disease. Regardless of whether or not we assumed that reproductive success was a function of group size, females that associated with highly attractive males always had lower lifetime fitness than those associating with less attractive males.

Our studies show that it is possible to use simple models to evaluate the usefulness of different descriptors of mating-system traits when the goal is specifically to assess the potential for disease spread. For example, we have shown that a measure of variance in male mating success is important when intergroup migration is small, but relatively unimportant when females show low fidelity to particular males across seasons. Mating systems, just as contact processes in humans, can be studied at a variety of levels ranging from identification of individual–individual contacts to quite general statistical measures such as mean and variance of the number of mates per group. It would be very desirable to develop descriptors of animal mating systems (and life-history features) that can lead to predictions with regard to rates of spread of different types of diseases.

The study of contact patterns has been a central focus of human infectious-disease epidemiology. A common approach is to group individuals into classes (e.g. age, social status, degree of sexual activity) and describe contacts among individuals in terms of a 'mixing matrix' where cell entries describe the frequency distribution of contacts per unit time between members of the different classes (Anderson *et al*. 1989; Blower & McLean 1991). Such mixing matrices are not readily applicable to animal mating systems, as they assume sequential rather than concurrent partnerships, i.e. they consider only transient relationships rather than those where several partners may cohabit simultaneously in mating groups. Only recently has the importance of concurrency of multiple partnerships been recognized in human epidemiology (Watts & May 1992; Kretzschmar *et al*. 1994), but the theory has proved analytically difficult and has not yet been extended to encompass different mating systems; evaluation of the consequences of group structure beyond simple pair-formation (Hesterbeek & Metz 1993) requires computer simulation (de Jong *et al*. 1994).

Developing a rigorous theoretical framework for the population and genetic dynamics of STDs is likely to be important for understanding mating-system evolution (Lockhart *et al*. 1996; Loehle 1995; Thrall *et al*. 1997). There has been little investigation of the relationship between social and mating structure and rates of disease spread or disease evolution, and it is difficult to know how to relate different classes of mating (e.g. polygyny, serial monogamy) or social structure to disease spread in animals. While we can posit quite general qualitative expectations regarding disease spread (R_0) for different mating systems (Table 1), the present study shows that these expectations will be influenced by heterogeneity in group size, demography and intergroup movement. Mating and social structures are therefore not only important as fascinating examples of behavioural interactions that require explanation but they are also almost certainly crucial components in the understanding of the emergence and control of new diseases in wildlife. Many such diseases, including STDs with alternate transmission modes (e.g. brucellosis), can also be transmitted to humans.

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REFERENCES

Altman, S. A., Wagner, S. S. & Lenington, S. 1977 Two models for the evolution of polygyny. *Behav. Ecol. Sociobiol.* **2**, 397–410.

Anderson, R. M. 1991 The transmission dynamics of sexually transmitted diseases: the behavioral component. In *Research issues in human behavior and sexually transmitted diseases in the AIDS era* (ed. J. N. Wasserheit, S. O. Aral & K. K. Holmes), pp. 38–60. Washington, DC: American Society for Microbiology.

Anderson, R. M. & May, R. M. 1981 The population dynamics of microparasites and their invertebrate hosts. *Phil. Trans. R. Soc. Lond.* B **291**, 451–524.

Anderson, R. M. & May, R. M. 1991 *Infectious diseases of humans: dynamics and control*. Oxford University Press.

Anderson, R. M., Blythe, S. P., Gupta, S. & Konings, E. 1989 The transmission dynamics of the human immunodeficiency virus type 1 in the male homosexual community in the United Kingdom: the influence of changes in sexual behaviour. *Phil. Trans. R. Soc. Lond.* B **325**, 45–98.

Anderson, R. M., May, R. M., Boily, M. C., Garnett, G. P. & Rowley, J. T. 1991 The spread of HIV-1 in Africa: sexual contact patterns and the predicted demographic impact of AIDS. *Nature* **352**, 581–589.

Antonovics, J., Iwasa, Y. & Hassell, M. P. 1995 A generalized model of parasitoid, venereal, and vector-based transmission processes. *Am. Nat.* **145**, 661–675.

Blower, S. M. & McLean, A. R. 1991 Mixing ecology and epidemiology. *Proc. R. Soc. Lond.* B **245**, 187–192.

Borgia, G. & Collis, K. 1990 Female choice for parasite-free male satin bowerbirds and the evolution of bright male plumage. *Behav. Ecol. Sociobiol.* **25**, 445–454.

Canfield, P. J., Love, D. N., Mearns, G. & Farram, E. 1991 Chlamydial infection in a colony of captive koalas. *Aust. Vet. J.* **68**, 167–169.

Clayton, D. H. & Moore, J. 1997 *Host parasite evolution: general principles and avian models*. Oxford University Press.

Clutton-Brock, T. H., Guinness, F. E. & Albon, S. D. 1982 *Red deer: ecology and behavior of two sexes*. University of Chicago Press.

de Jong, M. C. M., Diekmann, O. & Heesterbeek, J. A. P. 1994 The computation of R_0 for discrete-time epidemic models with dynamic heterogeneity. *Math. Biosci.* **119**, 97–114.

Ewald, P. W. 1990 Transmission modes and the evolution of virulence (with special reference to cholera, influenza, and AIDS). *Hum. Nat.* **2**, 1–30.

Ewald, P. W. 1994 *Evolution of infectious disease*. Oxford University Press.

Freeland, W. J. 1976 Pathogens and the evolution of primate sociality. *Biotropica* **8**, 12–24.

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

Graves, B. M. & Duvall, D. 1995 Effects of sexually transmitted diseases on heritable variation in sexually selected systems. *Anim. Behav.* **50**, 1129–1131.

Grenfell, B. T. & Dobson, A. P. (eds) 1995 *Ecology of infectious diseases in natural populations.* Cambridge University Press.

Guldbrandtsen, B. 1997 Maintenance of variation in mating success in hermaphrodites with a sexually transmitted disease. *Am. Nat.* **149**, 693–705.

Hamilton, W. D. & Zuk, M. 1982 Heritable fitness and bright birds: a role for parasites? *Science* **218**, 384–387.

Hamilton, W. D., Axelrod, R. & Tanese, R. 1990 Sexual reproduction as an adaptation to resist parasites (a review). *Proc. Natl Acad. Sci. USA* **87**, 3566–3573.

Hartley, I. R. & Shepherd, M. 1995 A random female settlement model can explain polygyny in the corn bunting. *Anim. Behav.* **49**, 1111–1118.

Hesterbeek, J. A. P. & Metz, J. A. 1993 The saturating contact rate in marriage and epidemic models. *J. Math. Biol.* **31**, 529–539.

Hethcote, H. W. 1976 Qualitative analyses of communicable disease models. *Math. Biosci.* **28**, 335–356.

Howard, R. S. & Lively, C. M. 1994 Parasitism, mutation accumulation and the maintenance of sex. *Nature* **367**, 554–557.

Immerman, R. S. 1986 Sexually transmitted disease and human evolution: survival of the ugliest? *Hum. Ethol. Newslett.* **4**, 6–7.

Immerman, R. S. & Mackey, W. C. 1997 Establishing a link between cultural evolution and sexually transmitted diseases. *Genet. Soc. Gen. Psychol. Monogr.* **123**, 441–459.

Jarman, P. J. 1974 The social organization of antelope in relation to their ecology. *Behaviour* **48**, 215–266.

Kempenaers, B. 1995 Polygyny in the blue tit: intra- and intersexual conflicts. *Anim. Behav.* **49**, 1047–1064.

Kretzschmar, M., Jager, J. C., Reinking, D. P., Van Zessen, G. & Brouwers, H. 1994 The basic reproduction ratio R_0 for a sexually transmitted disease in a pair formation model with two types of pairs. *Math. Biosci.* **124**, 181–205.

LeBoeuf, B. J. 1974 Male–male competition and reproductive success in elephant seals. *Am. Zool.* **14**, 160–176.

Lockhart, A., Thrall, P. H. & Antonovics, J. 1996 Sexually-transmitted diseases in animals: ecological and evolutionary implications. *Biol. Rev. Camb. Phil. Soc.* **71**, 415–471.

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

Loehle, C. 1997 The pathogen transmission avoidance theory of sexual selection. *Ecol. Model.* **103**, 231–250.

May, R. M., Anderson, R. M. & McLean, A. R. 1988 Possible demographic consequences of HIV/AIDS epidemics. I. Assuming HIV infection always leads to AIDS. *Math. Biosci.* **90**, 475–505.

Orians, G. H. 1969 On the evolution of mating systems in birds and mammals. *Am. Nat.* **103**, 589–603.

Oring, L. W., Reed, J. M. & Alberico, J. A. R. 1992 Mate acquisition tactics in polyandrous spotted sandpipers (*Aetitis macularia*): the role of age and experience. *Behav. Ecol.* **5**, 9–16.

Rubenstein, D. I. & Wrangham, R. W. (eds) 1986 *Ecological aspects of social evolution.* Princeton University Press.

Smith, H. G., Ottosson, U. & Sandell, M. 1994 Intrasexual competition among polygynously mated female starlings (*Sturnus vulgaris*). *Behav. Ecol.* **5**, 57–63.

Thrall, P. H. & Antonovics, J. 1997 Polymorphism in sexual versus non-sexual disease transmission. *Proc. R. Soc. Lond.* B **264**, 581–587.

Thrall, P. H., Antonovics, J. & Hall, D. W. 1993 Host and pathogen coexistence in vector-borne and venereal diseases characterized by frequency-dependent disease transmission. *Am. Nat.* **142**, 543–552.

Thrall, P. H., Biere, A. & Uyenoyama, M. K. 1995 Frequency-dependent disease transmission and the dynamics of the *Silene–Ustilago* host–pathogen system. *Am. Nat.* **145**, 43–62.

Thrall, P. H., Antonovics, J. & Bever, J. D. 1997 Sexual transmission of disease: implications for disease heterogeneity and host mating system evolution. *Am. Nat.* **149**, 485–506.

Thrall, P. H., Antonovics, J. & Wilson, W. G. 1998 Allocation to sexual vs. nonsexual disease transmission. *Am. Nat.* **151**, 29–45.

Van Baalen, M. & Sabelis, M. W. 1995 The scope for virulence management: a comment on Ewald's view on the evolution of virulence. *Trends Microbiol.* **3**, 414–416.

Watts, C. H. & May, R. M. 1992 The influence of concurrent partnerships on the dynamics of HIV/AIDS. *Math. Biosci.* **108**, 89–104.

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