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The evolution of risky behaviour in the presence of a sexually transmitted disease

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Sexually transmitted diseases (STDs) are widespread in nature, often sterilizing their hosts or causing other pathogenic effects. Despite this, there is a widespread occurrence of behaviours that are likely to increase the risk to an individual of contracting an STD. Here, we examine the evolution of behaviours such as promiscuity or mate choice that increase the risk of contracting an STD, but also provide a fitness benefit. As might be expected, the balance between risk and fitness benefit defines the optimal strategy, but this relationship is not straightforward. In particular, we often predict the coexistence of highly risky and highly risk-averse individuals. Surprisingly, very safe strategists that only suffer a small cost will tend to coexist with highly risky strategists rather than outcompete them as might have been expected. Rather than selecting for monogamy or for reduced mate choice, therefore, the presence of an STD may often lead to variability in either promiscuity or mate choice.

Keywords: sexually transmitted disease; promiscuity; monogamy; mate choice; polymorphism

1. INTRODUCTION

Considerable attention has recently been paid to the role of parasites in the evolution of mating systems, especially in the context of the adaptive advantages of female mate choice and the evolution of showy sexual characters in males (Hamilton & Zuk 1982; Clayton 1991; Hamilton & Poulin 1997). One often overlooked group of parasites that probably have important effects on mating systems are those that cause sexually transmitted diseases (STDs). By definition, STDs spread as a consequence of mating and therefore, given their pervasiveness in nature (Sheldon 1993; Lockhart *et al.* 1996), they are bound to be important in the evolution of mating behaviour in many species. Clearly, each time an animal mates with a new partner it is exposing itself to infection, meaning that less-promiscuous individuals will be less likely to become infected. This has led to the suggestion that STDs might cause selection for monogamy both in humans (e.g. Immerman 1986; Immerman & Mackey 1997) and in other animals (Hamilton 1990; Loehle 1995; Lockhart *et al.* 1996; Lombardo 1998).

Thrall *et al.* (1997, 2000) analysed two detailed models of mating systems and STDs in frameworks most representative of mammalian systems. The first was an individual-based model of the costs and benefits of mating frequency and the numbers of different partners in the presence of an STD. The mating behaviour resulting in the highest reproductive success for an individual was shown to vary according to sex, the prevalence of the STD and the likelihood of transmission per contact. When the STD was common and transmission was probable, the highest reproductive success was enjoyed by females who mated with only one male (monogamy), although under the same circumstances male fitness generally increased with the number of mates. Interestingly, when disease prevalence was low, or the probability of infection per sexual contact was low, then the model produced multiple optima. This shows that in some cases the increased probability of fertilization associated with multiple mating could compensate for the increased risk of contracting an STD. The second model (Thrall *et al.* 2000) was a simulation of a harem polygyny system with an STD present. Prevalence of the STD increased and female lifetime reproductive success was sharply reduced with increasing female movement between harems, which is equivalent to increasing female promiscuity. These two models therefore seem to support the idea that a high risk from STDs should select for monogamous individuals.

Despite these results, and increasing evidence that STDs are more common than previously thought (Sheldon 1993; Lockhart *et al.* 1996), selection for monogamy as a consequence of STDs does not appear to have been a particularly important force in nature. There is now considerable evidence that monogamy is relatively rare in animal systems, and many species previously thought to be monogamous are now known to exhibit high rates of extra-pair copulation (Jennions & Petrie 2000). Furthermore, some highly promiscuous species have been shown to have STDs. One example of a very promiscuous animal that is infected with a common and highly virulent STD is the two-spot ladybird *Adalia bipunctata*, populations of which in central and eastern Europe are infected with the sexually transmitted mite parasite *Coccipolipus hippodamiae* (Hurst *et al.* 1995; Webberley *et al.* 2002). High prevalences of the mite are normal, with rates of 90% having been recorded (Webberley *et al.* 2002). Because the mite effectively sterilizes females (Hurst *et al.* 1995) and increases male overwintering mortality (G. Hurst, unpublished data), genes for reduced promiscuity should be strongly selected for in this species, whereas in fact it remains exceedingly promiscuous.

What then is responsible for the maintenance of multiple mating in species with STDs? The most probable explanation is that the 'risky' mating strategy persists

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despite the presence of a virulent STD because it also brings with it a fitness benefit. In the case of promiscuity, there is now evidence from many species that promiscuous females can gain a fitness benefit beyond the simple increased likelihood of fertilization. These benefits include direct benefits, such as nuptial gifts, genetic benefits, and also increases in oviposition rates from the effects of male accessory gland secretions, which can lead to substantial increases in the fitness of promiscuous females (Yasui 1998; Arnqvist & Nilsson 2000; Jennions & Petrie 2000; Zeh & Zeh 2001).

Female mate choice is another behaviour that can increase exposure to STDs. At first sight, it might seem that the most obvious interaction between mate choice and an STD is that there may be an adaptive advantage to the female's choosiness if it enables females to avoid males that are infected with STDs (Sheldon 1993; Hurst *et al.* 1995; Loehle 1995, 1997; Able 1996). Knell (1999), however, pointed out that STDs that cause conspicuous damage to their hosts, allowing females to distinguish infected from uninfected males, will be selected against and unlikely to persist. STDs in systems with strong female choice acting on them will therefore be selected to be undetectable by females, and their selective influence on the host is likely to be subtler than simply affecting mate choice. It is noteworthy here that females of *A. bipunctata* appear to be unable to discriminate between uninfected males and those that are infected with *C. hippodamiae* (Webberley *et al.* 2002).

Graves & Duvall (1995) discussed the effects of a cryptic STD in a system with female mate choice. They argued that males with high mating success would probably become infected, and females that chose to mate with those males would themselves be likely to become infected, a point also made independently by Lombardo (1998). In the light of this, we might expect the presence of a virulent STD to lead to selection against females with strong preferences for particular males. However, when females exercise mate choice they can once again gain direct or indirect genetic benefits from mating with a particular male (Andersson 1994). This can also increase fitness and, thus, mate choice can be regarded as a 'risky' mating strategy that also brings fitness benefits in much the same way as promiscuity can.

The question that we address here, therefore, is under what circumstances can risky behaviour such as promiscuity or mate choice persist in the presence of a virulent STD? In particular, we ask when we would expect the evolution of risky behaviour that also carries a fitness benefit to occur in the presence of a virulent STD. By using a general mathematical model we demonstrate the possibility of mixed strategies within populations of highly risky and risk-averse individuals.

2. METHODS

Using an approach based on models that are focused on the evolution of costly resistance (Antonovics & Thrall 1994; Bowers *et al.* 1994; Boots & Bowers 1999), we present a model of a host–STD system with two strains of host; one (X_R) that shows risky mating behaviour, whereas the second (X_S) follows a safer mating strategy. The strain that indulges in risky behaviour gains a general fitness benefit such that its reproductive rate (a_R) is higher than that (a_S) of the other strain ($a_R > a_S$). We assume that there is also a sexually transmitted microparasite that can infect both host strains. As we described above, the 'risky' strain is at a higher risk of infection, such that the transmission rate of the risky behaviour strain (β_R) is greater than the safe strain (β_S). The total host population has density-dependent regulation with a linear dependence of birth rate (a) on total population density H, such that $a = a_0 - hH$, where h is a constant representing the strength of density dependence. With this assumption there is the possibility of negative death rates, however this only occurs when $H > a/h$ and, as the carrying capacity is $(a - b)/h$, it only occurs at densities well above the carrying capacity and therefore outside the region in which we are interested. We assume that the disease is sterilizing, shows no other pathogenicity (does not affect mortality) and that there is no recovery. These are characteristics of STDs and have been shown to allow the coexistence of hosts and STDs most easily (Sheldon 1993; Lockhart *et al.* 1996; Thrall & Antonovics 1997). We assume a haploid system and can represent the system as

$$\mathrm{d}X_R/\mathrm{d}t = r_R X_R - hHX_R - \beta_R X_R Y/H, \tag{2.1}$$

$$\mathrm{d}X_S/\mathrm{d}t = r_S X_S - hHX_S - \beta_S X_S Y/H, \tag{2.2}$$

$$\mathrm{d}Y/\mathrm{d}t = \beta_R X_R Y/H + \beta_S X_S Y/H - bY, \tag{2.3}$$

where b is the natural death rate, r is the intrinsic rate of increase $(a - b)$ and Y is the density of infectious hosts. H is the total population, $X_R + X_S + Y$. As there is no recovery or reproduction from the infected class, all the infected individuals can be grouped together. A diagrammatic representation of the model is shown in figure 1.

A standard stability analysis of equations (2.1)–(2.3) shows that if the natural death rate is greater than the transmission rate of the risky strain, $b > \beta_R$, the infection is lost from the population and we find, as $r_R > r_S$, only the risky strain at its carrying capacity ($X_R^* = r_R/h$, $X_S^* = 0$, $Y^* = 0$). This is the standard criterion for persistence of an STD. Otherwise, we can have either the risky or the susceptible strain in endemic equilibrium with the disease or coexistence of both strains with the pathogen. The risky strain is able to invade the population when

$$b(\beta_S - \beta_R) + \beta_R(a_S - a_R) - \beta_R(\beta_S - \beta_R) > 0, \tag{2.4}$$

whereas the safe strain can invade when

$$\beta_S(\beta_S - \beta_R) - b(\beta_S - \beta_R) - \beta_S(a_S - a_R) > 0. \tag{2.5}$$

Coexistence occurs when both equations (2.4) and (2.5) are fulfilled (this can be confirmed by simulation). From the invasion criteria of equations (2.4) and (2.5), we determine the conditions for when the evolutionarily stable strategy (ESS) is risky, safe or a mixed strategy of the two (figure 2).

3. RESULTS

Figure 2 shows how the balance between higher reproduction on the one hand and an increased risk of transmission on the other hand affects the ESS. If there is a great advantage in reproduction and only a small increased risk of infection from the risky strategy ($a_R \gg a_S$ and $\beta_R \approx \beta_S$), we find as expected that the risky strategy is favoured. Importantly, there is a wide area in parameter space where there is a protected polymorphism between the two strategies. As might be expected, there needs to

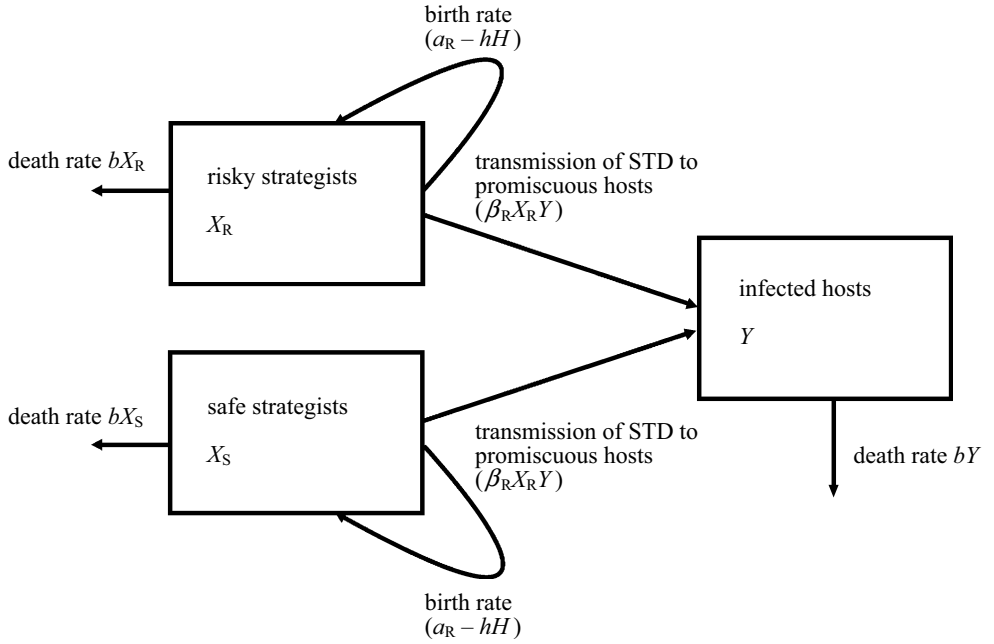

Figure 1. A diagrammatic representation of the model, showing how birth, death and infection contribute to the size of the populations of risky and safe strategists and infected individuals. STD, sexually transmitted disease.

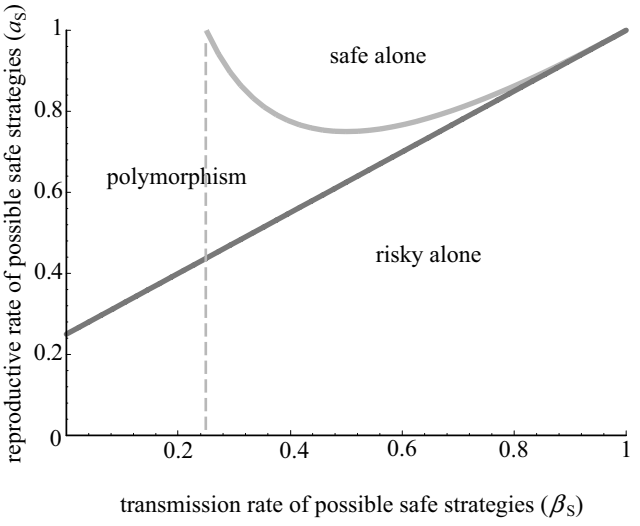

Figure 2. Outcomes in the parameter space of several possible safe strategies. The risky strain is fixed at the top right of the graph ($\beta_R = 1.0$, $a_R = 1.0$) and, therefore, the parameter space includes safer strategies ($\beta_S < 1.0$) that pay a cost in terms of reduced birth rates ($a_S < 1.0$). The polymorphic region is defined by the two criteria equations (2.4) and (2.5). The safe strain is only able to support the disease by itself to the right of the vertical dotted line (the transmission rate must be greater than the natural death rate ($\beta_S > b$).

be a balance between the costs and benefits of risky behaviour for a mixed strategy to be maintained, but this balance is far from linear. We are much more likely to get coexistence of risky and safer behaviours if the risky behaviour leads to a substantial increase in the chance of contracting the STD, because there is then a significantly higher incidence of the disease, facilitating the invasion of a much safer strategy. Similarly a very safe strategy reduces the incidence of the STD, allowing a risky strain to invade. However, if there is little difference between the strains, there is little effect on disease incidence and, therefore, it is much more difficult for coexistence to occur.

Crucially, it should be noted that the polymorphism is maintained even when there is little fitness benefit in terms of higher reproduction for the more risky strategy. This apparently paradoxical result arises in part because a 'very safe' strategist, such as a completely monogamous strain, will not maintain the STD on its own, and so it is unable to exclude the risky strain even when there is a negligible benefit to risky behaviour. Even a minimal benefit to risky mating behaviour will allow the risky strategist to outcompete the safely mating strain in the absence of the parasite. Therefore, minimal benefits may allow risky behaviour to coexist with safely mating strains when there is a very high risk of infection. It should be emphasized that only when the increased likelihood of infection from risky behaviour is minimal and the benefits are equivalently small ($a_S \approx a_R$ and $\beta_S \approx \beta_R$) will we get the exclusion of the risky strain.

The polymorphic equilibrium can be shown to be stable when relevant and, therefore, we do not get periodic strain changes. However, simulations reveal (figure 3) that we may see damped oscillations as we head towards the asymptotically stable coexistence equilibrium. These imply that under the stochastic buffeting probable in nature, there may be constant oscillation in the frequency of multiple mating or mate choice strains.

4. DISCUSSION

We have demonstrated that the presence of a virulent STD will not necessarily cause monogamy or random mating to be selected in the host. Instead, as long as it carries any fitness benefit, a highly risky strategy is liable to persist in the host population and will often coexist with really safe strategies, even when these pay almost no cost

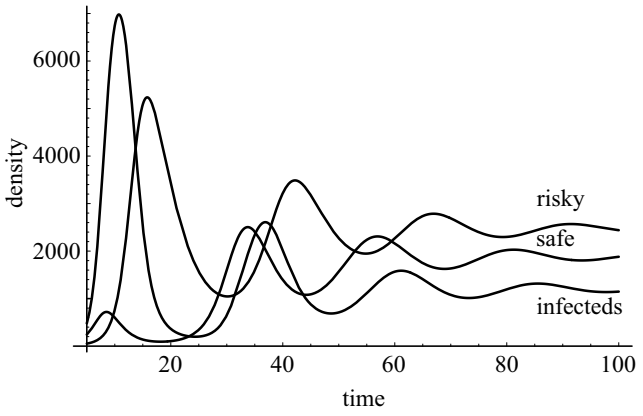

Figure 3. A simulation of the strain densities through time, showing the damped oscillations on the way to the asymptotically stable polymorphic equilibrium. The parameters are $a_S = 0.6$, $a_R = 1.0$, $\beta_S = 0.1$, $\beta_R = 1.0$, $h = 0.0001$, $b = 0.25$. Density is given in individuals, time in arbitrary units.

for their safer behaviour. This happens because these safe strategies fail to support significant numbers of infected individuals, giving the risky strategy an advantage. This non-intuitive result demonstrates how host–STD interactions can lead to important and unpredictable selective effects on host-mating behaviour.

Fitness benefits from multiple mating can be surprisingly high. Arnqvist & Nilsson (2000) analysed the results of 122 experimental studies of insects and concluded that the mean increase in lifetime reproductive success associated with multiple mating is between 30 and 70%. The benefits from mate choice are not as well quantified, but in many systems, such as those in which the female receives a substantial nuptial gift, they may be of a similar order. This translates into values of a_S of between *ca*. 0.6 and 0.75, which suggests that real host–STD systems will fall into the region of parameter space where the ESS is either mixed or risky, as in figure 2. Rather than selecting for monogamy (Hamilton 1990; Loehle 1995; Lockhart *et al.* 1996; Thrall *et al.* 1997, 2000), as often thought, or against mate choice (Graves & Duvall 1995), the presence of a virulent STD will often either have a minimal effect overall if the benefits to the host from the risky strategy are great enough, or will lead to greater variability in the degree of such risky behaviour.

This very general model does not differentiate between the different fitness benefits that males and females gain from, say, promiscuity. The fitness benefits to each sex from alternative mating strategies will obviously be different, and so it might be expected that the ESS for males and females would occupy different regions of parameter space, as in figure 1, leading to potential conflicts between the sexes. Encouragingly, more complex formalizations, in which the sexes are considered separately, reveal consistent results to ours (H. Kokko, personal communication). Other aspects of the model that might affect the results are the assumptions about the STD: we have assumed a completely sterilizing, horizontally transmitted STD. Many STDs cause considerable reductions in host fertility: Lockhart *et al.* (1996) concluded that STDs of mammals often reduce the fertility of the host. Also, a recent review of insect STDs (R. J. Knell, unpublished data) found that 10 out of 15 studies that reported any pathological effects, reported reductions in fertility. However, in reality, most STDs do not cause 100% sterilization and some are also vertically transmitted (Lockhart *et al.* 1996). On the one hand, changing the extent to which the STD sterilizes the host would probably have little effect on the general conclusions of the model as it would effectively only change the degree of risk associated with each strategy. Introducing vertical transmission, on the other hand, could lead to a reduction in the area of parameter space, where polymorphism is the ESS, as the STD population would now persist even with very safe (i.e. monogamous) hosts. This would be complicated by selection on the STD: vertically transmitted parasites are thought to be selected for reduced virulence (Bull *et al.* 1991; Herre 1993). So, as the amount of vertical transmission changed with the hosts mating behaviour, the virulence of the parasite might also change, which would then feed back into the evolution of host behaviour in unpredictable ways.

Variance in the degree of choice that females exhibit will affect the strength and possibly the direction of sexual selection. Such variability in female choice can have substantial evolutionary effects (Jennions & Petrie 1997). For example, female choice has been linked to speciation, so increased variance in mate choice could lead to a reduction in the rate of speciation in some systems (Panhuis *et al.* 2001). Although we have discussed them as two separate processes, it is clear that mate choice and promiscuity may be linked. Thus, a high variance in the number of matings may lead to differential pay-offs for males, and may lead to other polymorphisms in mating behaviour in both males and females.

Graves & Duvall (1995) argued that host–STD systems might display cycles of more- and less-choosy females, with more-choosy females invading when STDs are rare and then being outcompeted when the risk of contracting an STD is high. Our model does not display stable cycles, but we do find damped cycling that can occur in the proportions of the two strains. The system thus has a propensity to fluctuations in the proportions of safe and risky strains. Under the influence of unpredictable environmental effects, we might expect to see repeated oscillations, as the proportions of the two strains are repeatedly pushed away from equilibrium and then cycle back towards it. Cycles in female choice have previously been found in models of both the Fisher process (Iwasa & Pomiankowski 1995) and the handicap process (Iwasa & Pomiankowski 1999). It has been suggested that such oscillations could lead to rapid divergence of allopatric populations and possibly to the evolution of multiple sexual ornaments. Our results also indicate that the presence of an STD may contribute to such processes.

Empirical data that would allow a test of the conclusions set out in this paper are not yet available. Although several recent studies have quantified variation in paternity (i.e. Dunn & Cockburn 1999) within species, we are not aware of any that have looked at variation in promiscuity in any detail, and certainly not in relation to STD infection. Similarly, although there is an increasing realization that female preferences can vary within a species (Brooks & Endler 2001), there are no data available relating this to STD infection.

In summary, as expected, a fitness benefit may allow the existence of risky behaviour in the presence of an STD. Less intuitively, we have shown that the STD tends to maintain the coexistence of highly risky and very safe individuals. This result demands empirical tests that examine variation in risky behaviours within populations that maintain STDs.

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