Why have parasites promoting mating success been observed so rarely?

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Abstract

Host manipulation by sexually transmitted parasites which increases host mating rate and thus parasite transmission rate has long been viewed as a plausible parasite adaptation. However, empirical evidence for it is rare. Here, using an adaptive dynamics approach to evolution, we explore conditions under which such disease-induced mating enhancement is (or is not) likely to occur. We find that increased mating success is less likely to evolve if the host reproduction rate, or the baseline disease transmission rate, are reduced, and the parasite affects just one sex, compared to when it affects both. We also find that it is less likely to evolve if the virulence-transmission trade-off curve is stronger, since we assume that enhanced disease transmission can only be achieved at the cost of increased virulence and as this trade-off is concave. In addition, we demonstrate that if disease-induced mating enhancement is equally acting in both sexes the mating system has no effect

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on evolutionary outcomes. On the contrary, if disease-induced mating enhancement is acting in just one sex, the potential for its evolution is the higher the more polygynous the host population is. To study the examined phenomenon in greater detail we encourage further empirical research on this apparently less explored impact of sexually transmitted parasites on host fitness.
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1. Introduction

Parasites, like any other organism, evolve to maximize their fitness. Parasite fitness is maximized by maximizing transmission and minimizing virulence, and it is widely accepted that these two parasite characteristics are constrained by a concave virulence-transmission trade-off (Dieckmann, 2002; Alizon et al., 2009). This trade-off states that parasites can increase their transmission only at the cost of elevated virulence, which causes parasite evolution to tend to intermediate degrees of both transmission and virulence (Dieckmann, 2002; Alizon et al., 2009).

Various parasites use various transmission routes to reach susceptible hosts. Sexually transmitted parasites, in particular, can increase their transmission by infecting susceptible hosts more effectively upon any sexual contact or by enhancing the rate at which infected individuals succeed to mate. Although disease-induced mating enhancement has been expected by many to be a natural adaptation for sexually transmitted parasites to increase their transmission (Knell and Webberley, 2004), it has only been observed rarely. Whereas McLachlan (1999) found that infestation by the mite *Unionicola ypsilophora* enhanced the mating success of males of the midge *Paratrichocladius rufiventris*, males of the milkweed leaf

beetle *Labidomera clivicollis* infected with the mite *Chrysomelobia labidomera* were shown to displace rival males from mating pairs more often than uninfected males (Abbot and Dill, 2001). Furthermore, Raina et al. (2000) found that females of the corn earworm *Helicoverpa zea* infected by a gonad-specific virus produced more sex pheromone than uninfected females, thus enhancing their ability to attract males; however, due to accompanying changes of internal reproductive organs, the infected females forcefully avoided copulation.

Whether these observed instances of disease-induced mating enhancement are indeed adaptations by the parasite or rather some by-products of the parasite's pathology, it is not clear why this phenomenon has been observed so rarely. Does this apparent rareness reflect underexploration of this phenomenon or is the accompanying increase in parasite virulence countering the potential increase in mating enhancement? In this paper, we aim to address this question by means of modeling dynamics of sexually transmitted infections, considering the degree of disease-induced mating enhancement as an evolving trait. The diversity and extent of eventual evolutionary endpoints might suggest the expected frequency with which we may anticipate occurrence of this phenomenon in nature, and thus provide working hypotheses to be considered in future empirical research.

2. Methods

2.1. Models

The disease-induced mating enhancement, the phenomenon we study in this paper, involves sexually transmitted diseases (STDs) and may, in principle, affect one or both sexes. Therefore, we start by formulating a general two-sex model of

host population dynamics affected by an STD:

$$\begin{aligned}
\frac{dS_f}{dt} &= b\gamma_f\left(\mathcal{M}(S_f,S_m)+\delta_m\mathcal{M}(S_f,I_m)+\delta_f\mathcal{M}(I_f,S_m)+\delta_{fm}\mathcal{M}(I_f,I_m)\right)\\
&\quad -\bar{\mu}_f S_f-\xi_m\delta_m\mathcal{M}(S_f,I_m)\\
\frac{dS_m}{dt} &= b\gamma_m\left(\mathcal{M}(S_f,S_m)+\delta_m\mathcal{M}(S_f,I_m)+\delta_f\mathcal{M}(I_f,S_m)+\delta_{fm}\mathcal{M}(I_f,I_m)\right)\\
&\quad -\bar{\mu}_m S_m-\xi_f\delta_f\mathcal{M}(I_f,S_m)\\
\frac{dI_f}{dt} &= \xi_m\delta_m\mathcal{M}(S_f,I_m)-\bar{\mu}_f I_f-\alpha_f I_f\\
\frac{dI_m}{dt} &= \xi_f\delta_f\mathcal{M}(I_f,S_m)-\bar{\mu}_m I_m-\alpha_m I_m
\end{aligned} \tag{1}$$

This model describes dynamics of the density of susceptible females (S_f), susceptible males (S_m), infected females (I_f), and infected males (I_m), assuming that infected individuals cannot recover. The two-sex modeling framework we use was introduced by Kendall (1949) and Goodman (1953) and is now considered a standard for modeling two-sex population dynamics (Kot, 2001). In animals, mating and giving birth are often intertwined, and even tightly coupled. Just consider a system where males guard their mates until they cannot be taken by others. To account for structural consistency between the reproduction and disease transmission processes, mediated by mating, a generic mating function $\mathcal{M}(X,Y)$ in which X represents susceptible or infected females and Y represents susceptible or infected males occurs in both the reproduction term and the disease transmission term (Berec and Maxin, 2013). Moreover, we assume the background (i.e. in the absence of infection) host mortality to be negatively density-dependent: $\bar{\mu}_f=\mu_f+wP$ and $\bar{\mu}_m=\mu_m+wP$, where $P=S_f+S_m+I_f+I_m$ is the total population density. This ensures that in the absence of infection the host popula-

tion grows logistically, and is a common assumption in epidemiological models with populations of varying size (e.g. Pugliese, 1990; Altizer and Augustine, 1997).

The parameters that we mostly focus on in this paper are δ_f, δ_m, and δ_{fm}. They represent the mating enhancement factors, due to infected females, infected males, and both infected females and males, respectively. Therefore, we assume that δ_f and/or δ_m are greater than 1; moreover, $\delta_{fm} \geq \max\{\delta_f, \delta_m\}$ since there may or need not be a synergistic effect of δ_f and δ_m if both exceed 1. All model parameters are explained in Table 1.

Given a mating function $\mathcal{M}(S_f + I_f, S_m + I_m)$ describing the mating rate among females and males of any type, we assume random encounters between individuals (a common feature of many epidemiological models). Therefore, the mating rate $\mathcal{M}(S_f, S_m)$ between susceptible females and susceptible males can be expressed as

$$\mathcal{M}(S_f, S_m) = \mathcal{M}(S_f + I_f, S_m + I_m) \frac{S_f}{S_f + I_f} \frac{S_m}{S_m + I_m} \tag{2}$$

and similarly for the other three cases.

Most published epidemiological models do not distinguish between females and males. This entails an implicit assumption that female and male life histories are identical and hence that model parameters can be assumed sex-independent. Setting $\mu_f = \mu_m = \mu$, $\delta_f = \delta_m = \delta > 1$, $\xi_f = \xi_m = \xi$, and $\gamma_f = \gamma_m = 1/2$, it follows that $S_m = S_f = S/2$ where $S = S_m + S_f$ and $I_m = I_f = I/2$ where $I = I_m + I_f$ (provided this also holds for the respective initial conditions), and

the two-sex model (1) reduces to the following asexual model:

$$\begin{aligned}\frac{dS}{dt} &= b\mathcal{M}\left(\frac{P}{2},\frac{P}{2}\right)\left(\frac{S^2}{P^2}+2\delta\frac{SI}{P^2}+\delta_{fm}\frac{I^2}{P^2}\right)-\bar{\mu}S-2\xi\delta\mathcal{M}\left(\frac{P}{2},\frac{P}{2}\right)\frac{SI}{P^2}\\ \frac{dI}{dt} &= 2\xi\delta\mathcal{M}\left(\frac{P}{2},\frac{P}{2}\right)\frac{SI}{P^2}-\bar{\mu}I-\alpha I\end{aligned} \tag{3}$$

where $P = S + I$ and $\bar{\mu} = \mu + wP$.

To close the models (1) and (3), we need a specific mating function $\mathcal{M}(X, Y)$. A variety of mating functions have been proposed, most of which originate in the demographic literature where they are commonly referred to as marriage functions (Iannelli et al., 2005). Of these, most two-sex population models adopt mating functions that are degree-one homogeneous: $\mathcal{M}(ax, ay) = a\mathcal{M}(x, y)$ for any positive x, y, and a (Caswell and Weeks, 1986; Hadeler et al., 1988; Castillo-Chavez and Huang, 1995; Lindström and Kokko, 1998; Iannelli et al., 2005; Rankin and Kokko, 2007; Miller et al., 2007; Miller and Inouye, 2011, 2013). This assumption implies that if the female and male populations change by the same factor, the mating rate also changes by this factor. Here we use a degree-one homogeneous mating function. The model (3) becomes

$$\begin{aligned}\frac{dS}{dt} &= \frac{b}{2}\mathcal{M}(1,1)\left(\frac{S^2}{P}+2\delta\frac{SI}{P}+\delta_{fm}\frac{I^2}{P}\right)-\bar{\mu}S-\xi\delta\mathcal{M}(1,1)\frac{SI}{P}\\ \frac{dI}{dt} &= \xi\delta\mathcal{M}(1,1)\frac{SI}{P}-\bar{\mu}I-\alpha I\end{aligned} \tag{4}$$

Denoting $\beta \equiv b\mathcal{M}(1, 1)/2$ and $\lambda \equiv \xi\mathcal{M}(1, 1)$, we eventually get the asexual population model

$$\begin{aligned}\frac{dS}{dt} &= \beta\left(\frac{S^2}{P}+2\delta\frac{SI}{P}+\delta_{fm}\frac{I^2}{P}\right)-\bar{\mu}S-\lambda\delta\frac{SI}{P}\\ \frac{dI}{dt} &= \lambda\delta\frac{SI}{P}-\bar{\mu}I-\alpha I\end{aligned} \tag{5}$$

where $P = S + I$ and $\bar{\mu} = \mu + wP$. Note that we need not provide any specific form of the degree-one homogeneous mating function here, since the model (5) covers any of these.

2.2. Evolutionary analysis

To study evolution of disease-induced mating enhancement, we assume $\delta_{fm} = \delta^2$ in the model (5) (i.e. multiplicative effect of infected females and males) and consider the parameter δ as an evolving trait. We use the techniques of adaptive dynamics (Dieckmann, 2002; Diekmann, 2004), assuming that a 'resident' parasite strain with δ is established in the host population and a rare 'mutant' strain with $\hat{\delta}$ invades an endemic equilibrium set by the resident. Let the evolution proceed in small steps, i.e. let the mutant's $\hat{\delta}$ be close to the resident's δ. Then, under rather mild conditions which our models satisfy, successful invasion implies extinction of the resident population and establishment of the mutant population (Dercole, 2002, page 46). Now, the formerly mutant population becomes resident and is challenged by a new mutant strain. Ecological time is thus assumed to run much faster than evolutionary time (Dieckmann, 2002; Diekmann, 2004).

In order to determine whether a rare mutant takes over an established resident, we calculate the mutant's invasion fitness as the initial growth rate of the mutant when the resident is at its endemic equilibrium $(S^*(\delta), I^*(\delta))$. When this invasion fitness

$$f(\hat{\delta}, \delta) \equiv f(\hat{\delta}, \delta; S^*(\delta), I^*(\delta)) \tag{6}$$

is positive, the mutant invades the resident, if it is negative the mutant dies out and the resident stays at its endemic equilibrium. We go on by calculating the selection gradient as the slope of the invasion fitness when the mutant trait is equal to the

resident trait,

$$g(\delta) = \left. \frac{\partial f(\hat{\delta}, \delta)}{\partial \hat{\delta}} \right|_{\hat{\delta}=\delta} \tag{7}$$

The value of δ at which the selection gradient is equal to zero is referred to as the evolutionary singular point δ^*. If $f(\hat{\delta}, \delta)$ as a function of $\hat{\delta}$ is maximized at δ^*, or more formally

$$E = \left. \frac{\partial^2 f(\hat{\delta}, \delta)}{\partial \hat{\delta}^2} \right|_{\hat{\delta}=\delta=\delta^*} < 0 \tag{8}$$

then this evolutionary singular point is evolutionary stable. That is, populations with trait values near δ^* cannot invade the population with the trait value δ^*. If it is minimized ($E > 0$) then δ^* is evolutionary unstable. In addition, if the selection gradient is positive in a left neighborhood of δ^* and negative in a right neighborhood of δ^*, the evolutionary singular point δ^* is convergence stable. That is, populations with trait values δ closer to δ^* replace those with more distant δ values. If the opposite inequalities hold, δ^* is convergence unstable. If an evolutionary singular point is both evolutionary and convergence stable, it is an evolutionary attractor. If it is convergence stable but evolutionary unstable, it is an evolutionary branching point at which a dimorphic parasite population arises (Dieckmann, 2002; Diekmann, 2004).

2.3. Host and parasite trade-offs

Because of several trade-offs, the mating enhancement factor δ is likely to both directly and indirectly affect host mortality. The direct way is due to an interaction with the host's immune system (Alizon and Van Baalen, 2005). The indirect way is due to the fact that enhanced mating and reproduction may bring about a survival cost (Berec and Maxin, 2012, and references therein). This 'cost of reproduction' occurs because by increasing the energy outlay on reproduction

individuals with enhanced reproduction live for a shorter amount of time. Alternatively, fertile individuals or individuals with higher mating success are more susceptible to predators or parasitoids if the latter can capitalize on mating signals of the former (Zuk and Kolluru, 1998; Pavlova et al., 2010).

In the absence of infection host mortality is $\bar{\mu}$. In its presence, mortality of infected individuals raises to $\bar{\mu} + \alpha$. So α in the asexual model (5) (and similarly in the two-sex model (1)) is a compound parameter accounting for the disease-induced mortality and representing virulence. In addition, $\lambda\delta$ in the asexual model (and similarly in the two-sex model) represents disease transmission. Since the widely accepted virulence-transmission trade-off prescribes that transmission can increase only at the cost of enhanced virulence (Alizon et al., 2009), we should expect α to increase with δ. Moreover, the virulence-transmission trade-off is predicted to be concave (Alizon and Van Baalen, 2005). The function

$$\alpha(\delta) = b\delta^z,\ z > 1 \tag{9}$$

is consistent with the expected concavity. Indeed, denoting $\Lambda(\delta) = \lambda\delta$ we get the concave function of α,

$$\Lambda(\alpha) = \lambda \left(\frac{\alpha}{b}\right)^{1/z},\ z > 1 \tag{10}$$

Moreover, the closer z is to 1, the less curved the trade-off function (10) is (Fig. 1). The formula (9) is a simple phenomenological description that aims to cover both direct and indirect effects of parasites on host mortality.

3. The asexual model: females and males equally affected by infection

We first analyze the asexual model (5), since it is simpler, allows for a complete analysis, and also for an easier comparison with existing, mostly asexual epidemiological models.

3.1. Population dynamics

Analysis of population dynamics provides the necessary first step for the study of evolution via adaptive dynamics. Therefore, we first present results on population dynamics described by the asexual model (5). In the absence of infection, $(P_{DFE}, 0)$ with $P_{DFE} = (\beta - \mu)/w$ is the unique non-zero equilibrium of the model (5); we refer to it as the disease-free equilibrium (DFE). To allow for population persistence in the absence of infection we assume $\beta > \mu$.

The basic reproduction number of the infection described by the model (5) is

$$R_0 = \frac{\lambda\delta}{\mu + wP_{DFE} + \alpha} = \frac{\lambda\delta}{\beta + \alpha} \tag{11}$$

It is equal to the average number of adequate contacts, $\lambda\delta$, of an infected individual during its mean infectious period, $1/(\mu + wP_{DFE} + \alpha)$, when the host population is at the DFE. Note that R_0 does not depend on μ, the intrinsic host mortality rate, w, the strength of negative density dependence in the background mortality rate, and δ_{fm}, the mating enhancement factor between infected females and infected males.

In the rest of this subsection, we summarize the main results of our analysis; detailed analysis is carried out in Appendix A. If $R_0 > 1$ the infection is able to invade the host population and the DFE $(P_{DFE}, 0)$, which always exists, is unstable. The invasion can be successful in which case the system attains a unique, globally stable endemic equilibrium, or unsuccessful in which case the infection is too harmful, causing host extinction. The latter possibility is a direct consequence of the standard-incidence-like transmission term in the model (5). Denoting

$$\Delta = [2\beta(\delta - \delta_{fm}) - \lambda\delta + \alpha]^2 - 4\beta^2(1 - 2\delta + \delta_{fm})\delta_{fm}$$

the invasion is successful if $K^* > \mu$ where

$$K^* = \beta \left[(s^*)^2 + 2\delta s^*(1-s^*) + \delta_{fm}(1-s^*)^2\right] - \alpha(1-s^*) \tag{12}$$

and

$$s^* = \frac{2\beta(\delta - \delta_{fm}) - \lambda\delta + \alpha + \sqrt{\Delta}}{2\beta(2\delta - 1 - \delta_{fm})} \tag{13}$$

is the unique equilibrium proportion of the susceptible hosts. The resulting endemic equilibrium is

$$S^* = \frac{s^*(K^* - \mu)}{w} \quad \text{and} \quad I^* = \frac{(1-s^*)(K^* - \mu)}{w} \tag{14}$$

On the other hand, if $K^* < \mu$ the extinction equilibrium $(0, 0)$ is globally stable and the invasion is unsuccessful.

If $R_0 < 1$ we need to distinguish two cases. When $\delta \leq \delta_{fm} < 2\delta - 1$, the infection cannot invade the host population and the DFE is globally stable. This is also the case in a part of the parameter space when $\delta_{fm} > 2\delta - 1$. Otherwise, if

$$[2\beta(\delta - \delta_{fm}) - \lambda\delta + \alpha]^2 - 4\beta^2(1 - 2\delta + \delta_{fm})\delta_{fm} > 0$$

and

$$-2\beta(1 - 2\delta + \delta_{fm}) < 2\beta(\delta - \delta_{fm}) - \lambda\delta + \alpha < 0$$

the DFE is only locally stable and there are two equilibrium proportions of the susceptible hosts. The lower of these, denoted s^-, is locally stable and the higher is unstable. In terms of S and I, if $K^- > \mu$ where

$$K^- = \beta \left[(s^-)^2 + 2\delta s^-(1-s^-) + \delta_{fm}(1-s^-)^2\right] - \alpha(1-s^-)$$

and

$$s^- = \frac{-\left[2\beta(\delta - \delta_{fm}) - \lambda\delta + \alpha\right] - \sqrt{\Delta}}{2\beta(1 - 2\delta + \delta_{fm})}$$

the following, locally stable endemic equilibrium exists:

$$S^- = \frac{s^-(K^- - \mu)}{w} \quad \text{and} \quad I^- = \frac{(1-s^-)(K^- - \mu)}{w}$$

If $K^- < \mu$, on the other hand, the extinction equilibrium $(0, 0)$ is locally stable.

Hence, even if $R_0 < 1$ the disease can persist in the host population or even drive it extinct whenever the initial proportion of infected individuals is sufficiently large. In biological terms, this happens because if the infected population reaches a certain proportion, the regenerative effect of δ_{fm} can keep transmission of the parasite going, and even boost it such that the host population goes extinct. As a consequence, we may have bistability between the disease-free population state and either host population extinction or disease persistence at an endemic equilibrium. This bistability regime is a direct consequence of our assumption of structural consistency between the processes of host reproduction and disease transmission, mediated by mating (Sect. 2.1 and Berec and Maxin, 2013).

3.2. Evolution

To study evolution of disease-induced mating enhancement, we assume $\delta_{fm} = \delta^2$ (i.e. multiplicative effect of infected females and males) and consider evolution of the single parameter δ. Then, if $R_0 > 1$ the unique equilibrium proportion of the susceptible hosts is (recall that we have set $\alpha(\delta) = b\delta^z$, $z > 1$)

$$s^* = \frac{2\beta\delta(\delta-1) + \lambda\delta - b\delta^z - \sqrt{(\lambda\delta - b\delta^z)[4\beta\delta(\delta-1) + \lambda\delta - b\delta^z]}}{2\beta(\delta-1)^2} \tag{15}$$

and

$$K^* = \beta\left(s^* + \delta(1-s^*)\right)^2 - b\delta^z(1-s^*) \tag{16}$$

Moreover, even though $\delta_{fm} = \delta^2 > 2\delta - 1$ for any $\delta > 1$, no bistability regime can occur in this case and the DFE is thus always globally stable when $R_0 < 1$.

Let the model (5) with a resident parasite strain with mating enhancement δ be at the endemic equilibrium $(S^*(\delta), I^*(\delta))$. The invasion fitness of a mutant strain with mating enhancement $\hat{\delta}$ is

$$f(\hat{\delta}, \delta) = \lambda\hat{\delta}\frac{S^*(\delta)}{S^*(\delta) + I^*(\delta)} - \mu - w(S^*(\delta) + I^*(\delta)) - b\hat{\delta}^z$$

Since $f(\delta, \delta) = 0$ we have

$$\mu + w(S^*(\delta) + I^*(\delta)) + b\delta^z = \lambda\delta\frac{S^*(\delta)}{S^*(\delta) + I^*(\delta)}$$

and hence

$$f(\hat{\delta}, \delta) = \lambda(\hat{\delta} - \delta)s^*(\delta) - b(\hat{\delta}^z - \delta^z)$$

For $z > 1$, this fitness function implies

$$\frac{\partial^2 f(\hat{\delta}, \delta)}{\partial\hat{\delta}^2} = -bz(z-1)\hat{\delta}^{z-2} < 0 \tag{17}$$

Therefore, if an evolutionary singular point exists it is always evolutionary stable. This excludes the possibility of evolutionary branching.

Let us start with the limiting case $z = 1$ for which $\alpha(\delta) = b\delta$ and

$$f(\hat{\delta}, \delta) = (\hat{\delta} - \delta)[\lambda s^*(\delta) - b]$$

Mating enhancement will evolve to lower or higher values, depending on the sign of the selection gradient $g(\delta) = \lambda s^*(\delta) - b$, for which

$$s^*(\delta) = \frac{2\beta\delta}{2\beta(\delta-1) + \lambda - b + \sqrt{(\lambda - b)[4\beta(\delta-1) + \lambda - b]}}$$

In Appendix B we show that if the infection rate is low compared with the reproduction rate, $\lambda < 4\beta$, then the virulence-transmission trade-off is not strong enough to prevent the evolution to ever higher mating enhancement. Conversely,

if $\lambda > 4\beta$ then whether δ evolves to higher or lower values depends on how strong the trade-off factor b is and how large the current strain value of δ is. If b is relatively large and the current strain value of δ is close to 1 then the system will evolve to either a value $\delta^* > 1$ or to no disease-induced mating enhancement. For large enough current δ's, the system evolves to ever higher mating enhancement. If the trade-off factor b is too small then the system always evolves to ever higher mating enhancement.

The only case beyond $z = 1$ that we can rigorously analyze is $z = 2$. Then, the mutant's invasion fitness is

$$f(\hat{\delta}, \delta) = (\hat{\delta} - \delta)[\lambda s^*(\delta) - b(\hat{\delta} + \delta)]$$

The endemicity condition $R_0 > 1$ now implies

$$\lambda > \sqrt{4b\beta} \quad \text{and} \quad R_1 < \delta < R_2 \tag{18}$$

where we define

$$R_1 \equiv \frac{\lambda - \sqrt{\lambda^2 - 4b\beta}}{2b} \quad \text{and} \quad R_2 \equiv \frac{\lambda + \sqrt{\lambda^2 - 4b\beta}}{2b}$$

Since the resident strain with a trait value $\delta > 1$ needs to be endemic, we have either

$$1 < R_1 < R_2 \Leftrightarrow \beta + b > \lambda > 2b \tag{19}$$

($R_1 > 1$ implies $\lambda - 2b > \sqrt{\lambda^2 - 4b\beta} > 0$) or

$$R_1 < 1 < R_2 \Leftrightarrow \lambda > \beta + b \tag{20}$$

The selection gradient is now

$$g(\delta) = \lambda s^*(\delta) - 2b\delta = 2\delta h(\delta)$$

where

$$h(\delta) = \frac{\lambda\beta}{2\beta(\delta-1)+\lambda-b\delta+\sqrt{(\lambda-b\delta)[4\beta(\delta-1)+\lambda-b\delta]}} - b \qquad (21)$$

To find the evolutionary singular points δ^* and assess their convergence stability, we first need to establish the domain of the function $h(\delta)$ and its possible roots (i.e. the values of δ at which the selection gradient vanishes). The expression under the radical of $h(\delta)$ has two roots,

$$D_1 = \frac{4\beta-\lambda}{4\beta-b} \quad \text{and} \quad D_2 = \frac{\lambda}{b}$$

Since $R_0 > 1$ implies $\lambda > b\delta > b$, then $D_2 > 1$ and the domain of $h(\delta)$ is (D_1, D_2) if $b < 4\beta$, with $D_1 < 1$, or $(-\infty, D_2) \cup (D_1, \infty)$ if $b > 4\beta$, with $D_1 > 1$. Since $R_0 > 1$ implies $\delta < D_2$ and since δ cannot be negative, only the interval $[0, D_2)$ is relevant to explore in the latter case.

While technical details are provided in Appendix C, the evolutionary outcomes in the case $z = 2$ are summarized in Table 2 and visualized in Fig. 2. The evolutionary analysis reveals that evolution to disease-induced mating enhancement occurs if $R_1 < 1 < R_2$, $b < 4\beta$, $h(D_1) > 0$, $h(1) > 0$, and $h(D_2) < 0$, or if $1 < R_1 < R_2$. The latter case requires that some form of mating enhancement already exists in the host, since in this case $R_0(1) < 1$. In all of the other scenarios evolution pushes δ below one, which actually means evolution to no disease-induced mating enhancement. Within this latter group of scenarios we can further distinguish two subcases. First, we may have evolution to an evolutionary attractor $\delta^* < 1$, which we will refer to as evolution to disease-induced mating decrement. Second, we may have evolutionary suicide when δ passes through the lower endemicity boundary R_1 (the infection prevalence is positive and the selection gradient $g(\delta)$ is negative at R_1). As Fig. 2 suggests, evolution

to disease-induced mating enhancement is more likely the higher β and λ are and the lower b is; this is because the 'cusp' point at which the shaded area emerges has components $\beta = b$ and $\lambda = 2b$.

However, this is still not the whole story. The above evolutionary results were based on the assumption that $R_0 > 1$ guarantees parasite persistence at an endemic equilibrium. This assumption arose quite naturally here since invasion fitness, the quantity of fundamental importance to our evolutionary analysis, was independent of the host intrinsic mortality rate μ. However, we know from the previous subsection that the infection can drive the host population to extinction and that this happens as soon as $K^* < \mu$. The direct implication of this is that the actual endemicity range of δ is an intersection of the interval (R_1, R_2) and an interval for which $K^* > \mu$. More importantly, any δ^* found to be an evolutionary attractor by our evolutionary analysis must also satisfy $K^*(\delta^*) > \mu$. Otherwise, an evolutionary suicide is the evolutionary outcome in such a situation, since if $K^* > \mu$ changes to $K^* < \mu$ due to an evolutionary step in δ, we face sudden extinction of both the parasite and its host. For appropriate parameter values, this situation may affect both disease-induced mating decrement ($\delta^* < 1$) and disease-induced mating enhancement ($\delta^* > 1$) (Fig. 2), as also exemplified in Fig. 3.

On the way to population extinction, chance effects can play a significant role. Before or even just beyond the point of evolutionary suicide, population densities are commonly very low (Fig. 3). Due to chance effects, extinction of the host and hence of the parasite is not the only possible outcome. Alternatively, one may expect an infection fade-out whereby all infected host individuals go extinct before all susceptible host individuals do. Then, the remaining susceptible host population may escape the pitfalls of stochasticity and recover to the DFE.

For $z \neq 2$ we are not able to derive any analytical results. Therefore, we provide just a single figure showing how evolutionary outcomes may vary with increasing costs of disease-induced mating enhancement (i.e. with increasing z). Figure 4 shows that when relatively low values of z lead to relatively high values of mating enhancement, for high enough z evolution will rather tend to mating decrement (i.e. to no mating enhancement). That is, the higher cost of mating enhancement z lowers the likelihood that disease-induced mating enhancement will evolve.

4. The two-sex model: just one sex affected by infection

The empirical observations of disease-induced mating enhancement that we refer to in the introduction suggest that the corresponding parasites may be sex-specific in their mating enhancement ability. Moreover, the cost of reproduction can also be biased towards the sex that is more active in its mating behavior (Pavlova et al., 2010; Berec and Maxin, 2012). Therefore, in this section, we assume that infected individuals of only one sex demonstrate disease-induced mating enhancement.

4.1. Population dynamics

Consideration of sex-specific mating enhancement requires the two-sex model (1) and, contrary to the asexual model (5), an explicit specification for the mating function $\mathcal{M}(S_f + I_f, S_m + I_m)$. Here we choose the (modified) harmonic mean mating function, which is the most commonly used degree-one homogeneous mating function (Caswell and Weeks, 1986; Lindström and Kokko, 1998; Miller et al., 2007; Bacelar et al., 2011; Miller and Inouye, 2013) and which has

also got some empirical support (Miller and Inouye, 2011):

$$\mathcal{M}(S_f + I_f, S_m + I_m) = 2c\,\frac{(S_f + I_f)(S_m + I_m)}{(S_f + I_f)/h + S_m + I_m} \tag{22}$$

where c is a positive scaling constant. The modification of the standard harmonic mean mating function concerns the introduction of the parameter h, which allows for capturing a variety of mating systems. In particular, $h = 1$ represents monogamy and corresponds to the standard harmonic mean mating function, $h > 1$ corresponds to polygyny (a mating system involving one male and two or more females), and $h < 1$ is a model for polyandry (a mating system involving one female and two or more males) (Caswell and Weeks, 1986; Lindström and Kokko, 1998; Miller et al., 2007; Bacelar et al., 2011; Miller and Inouye, 2013). This will allow us to assess whether polygamous mating systems have different evolutionary outcomes than the monogamous one.

Since analysis of the resulting two-sex model (the model (D.2) in Appendix D) is cumbersome, we assume that, besides mating enhancement, all other model parameters are sex-independent. Setting $\delta_f = \delta_{fm} = \delta$, $\delta_m = 1$ (only females affected by the infection) and denoting $\beta \equiv 2cb$, $\lambda \equiv 2c\xi_m = 2c\xi_f$, our two-sex model becomes

$$\begin{aligned}
\frac{dS_f}{dt} &= \frac{\beta}{2}\left(\frac{S_f S_m}{T} + \frac{S_f I_m}{T} + \delta\frac{I_f S_m}{T} + \delta\frac{I_f I_m}{T}\right) - \bar{\mu} S_f - \lambda\frac{S_f I_m}{T} \\
\frac{dS_m}{dt} &= \frac{\beta}{2}\left(\frac{S_f S_m}{T} + \frac{S_f I_m}{T} + \delta\frac{I_f S_m}{T} + \delta\frac{I_f I_m}{T}\right) - \bar{\mu} S_m - \lambda\delta\frac{I_f S_m}{T} \\
\frac{dI_f}{dt} &= \lambda\frac{S_f I_m}{T} - \bar{\mu} I_f - \alpha_f I_f \\
\frac{dI_m}{dt} &= \lambda\delta\frac{I_f S_m}{T} - \bar{\mu} I_m - \alpha_m I_m
\end{aligned} \tag{23}$$

where $T = (S_f + I_f)/h + S_m + I_m$. An analogous model results when only males are affected by the infection, i.e. when $\delta_m = \delta_{fm} = \delta$, $\delta_f = 1$. In both, the basic reproduction number of the infection (D.5) given in Appendix D simplifies to

$$R_0 = \frac{2\lambda\sqrt{\delta}}{\sqrt{(\beta + 2(1/h+1)\alpha_f)(\beta + 2(1/h+1)\alpha_m)}} \tag{24}$$

In Appendix E we show that the only boundary equilibrium of the model (23) that can be stable is the DFE. In particular, the DFE is stable if $R_0 < 1$ and unstable if $R_0 > 1$. Unfortunately, endemic equilibria of the model (23) are difficult to analyze. Still, the equation for the total population size P (E.2) implies that if

$$K_2^* \equiv \frac{\beta(x^* + \delta z^*)(1 - x^* - z^*)}{1 + (1/h - 1)(x^* + z^*)} - \alpha_f z^* - \alpha_m(1 - x^* - y^* - z^*) < \mu \tag{25}$$

the host population goes extinct due to the disease. Here x^*, y^* and z^* refer to proportions of the susceptible females, susceptible males and infected females, respectively, in a stable positive equilibrium of the proportional model (E.1). If $K_2^* > \mu$, on the other hand, the host population grows when small and attains a stable endemic equilibrium. In addition, numerical simulations suggest that if $R_0 > 1$ then the proportional model (E.1) has a unique equilibrium that satisfies $0 < x^* < 1$, $0 < y^* < 1$, and $0 < z^* < 1$. If a need arises during the subsequent evolutionary analysis, we calculate it numerically. These results stay the same if males instead of females are the sex affected by the infection.

4.2. Evolution

To study evolution of disease-induced mating enhancement we assume that the resident parasite strain has $\delta \geq 1$ and the system is in a stable endemic equilibrium $(S_f^*(\delta), S_m^*(\delta), I_f^*(\delta), I_m^*(\delta))$. Also here we assume that disease virulence is traded

off with disease transmission, but this trade-off now acts only in the sex in which the parasite promotes mating enhancement. Therefore, $\alpha_f(\delta) = b\delta^z$, $z > 1$, $\alpha_m(\delta) = b$ in the model (23), and vice versa in the analogous model with mating enhancement in males.

Following Bacelar et al. (2011), we show in Appendix E that the invasion fitness of the mutant strain in the two-sex model (23) is

$$f(\hat{\delta}, \delta) = \lambda^2(\hat{\delta} - \delta)\frac{S_m^* S_f^*}{(T^*)^2} - b(\hat{\delta}^z - \delta^z)\lambda\delta\frac{S_m^* I_f^*}{T^* I_m^*} \tag{26}$$

If males instead of females are the sex affected by the infection, the invasion fitness becomes

$$f(\hat{\delta}, \delta) = \lambda^2(\hat{\delta} - \delta)\frac{S_m^* S_f^*}{(T^*)^2} - b(\hat{\delta}^z - \delta^z)\lambda\delta\frac{S_f^* I_m^*}{T^* I_f^*} \tag{27}$$

For the limiting trade-off with $z = 1$ the invasion fitness (26) simplifies to

$$f(\hat{\delta}, \delta) = \lambda\,\frac{S_m^* I_f^*}{T^* I_m^*}(\hat{\delta} - \delta)\bar{\mu}^*$$

This implies that $f(\hat{\delta}, \delta) > 0$ whenever $\hat{\delta} > \delta$. For $\alpha_f = b\delta$ and $\alpha_m = b$ the endemicity condition $R_0(\delta) > 1$ is equivalent to

$$\delta > \delta_c \equiv \frac{\beta(\beta + 2b(1/h + 1))}{4\lambda^2 - 2b(1/h + 1)(\beta + 2b(1/h + 1))}$$

Hence, evolution will always proceed to ever higher mating enhancement. The same analysis can be used to show that this is also true if males instead of females are affected by the infection.

In the case of trade-off with $z = 2$ the invasion fitness (26) simplifies to

$$f(\hat{\delta}, \delta) = \lambda\,\frac{S_m^* I_f^*}{T^* I_m^*}(\hat{\delta} - \delta)(\bar{\mu}^* - b\delta\hat{\delta})$$

The selection gradient

$$g(\theta) = \left.\frac{\partial f(\hat{\delta}, \delta)}{\partial \hat{\delta}}\right|_{\hat{\delta}=\delta} = \lambda \frac{S_m^* I_f^*}{T^* I_m^*}(\bar{\mu}^* - b\delta^2)$$

vanishes whenever

$$\bar{\mu}^* = b\delta^2$$

where $\bar{\mu}^*$ is a complicated function of δ,

$$\bar{\mu}^* = \frac{\beta(x^* + \delta z^*)(1 - x^* - z^*)}{1 + (1/h - 1)(x^* + z^*)} - b\delta^2 z^* - b(1 - x^* - y^* - z^*)$$

The same analysis can be used to show that the sign of the selection gradient coincides with the sign of the term $\bar{\mu}^* - b\delta^2$ also if males instead of females are affected by the infection.

Since we are not able to conduct any detailed analysis comparable to that of the asexual model (5), we instead demonstrate that mating enhancement can also evolve in the two-sex model (23). Before we do that, we emphasize that all types of results observed for the asexual model (evolutionary suicide, evolution to mating decrement, and evolution to mating enhancement) were also observed here. Regarding evolution of disease-induced mating enhancement, we observe that the area in the (β, λ) parameter space in which this is possible is more restricted; compare the left panel of Fig. 2 with the middle bottom panel of Fig. 5. This suggests that mating enhancement is less likely to evolve if the parasite is affecting just one sex relative to when it affects both. As in the asexual case, the area with a potential for disease-induced mating enhancement shrinks (or more precisely moves up and to the right) when b increases, but also when the mating system goes from polygyny ($h > 1$) through monogamy ($h = 1$) to polyandry ($h < 1$) (Fig. 5). Polygynous populations with low b thus have the highest potential for evolution

of disease-induced mating enhancement. These results are independent of which sex is affected by the infection.

As the area in the (β, λ) parameter space in which evolution of mating enhancement is possible shrinks, the magnitude of the evolutionary attractor δ^* also decreases. All else being equal, δ^* declines with increasing b and decreasing h (Fig. 6). These results, other than small quantitative differences, are independent of the affected sex.

5. Discussion

In this paper we were interested in the potential of parasites that are transmitted sexually in animals to evolve the means to spread more effectively by promoting mating success in the infected hosts. Such a disease-induced mating enhancement, while expected by many to be a natural adaptation of sexually transmitted parasites (Knell and Webberley, 2004), has so far been observed only rarely, and we are aware of only three studies that unequivocally demonstrated it (McLachlan, 1999; Abbot and Dill, 2001; Raina et al., 2000, details are given in the introduction). While it is possible that this phenomenon might occur more frequently in nature, we were primarily interested in identifying conditions under which evolution of disease-induced mating enhancement is not likely. In other words, we aim to provide a plausible explanation as to why disease-induced mating enhancement appears to be rare in nature. The results of this study can then be used to make empirical predictions.

Unfortunately, it is difficult to generalize the three existing studies demonstrating disease-induced mating enhancement in order to provide a foundation for our models. Therefore, we simply assumed that mating enhancement is either equal

in both females and males or present in just one sex. In the former case we also assumed that the effects on females and males are multiplicative, whereas in the latter we separately studied the cases of either females or males affected by the STD. Also, we explored the effect of mating system (i.e. polyandry, monogamy, polygyny) on evolutionary outcomes. Each of these scenarios assumed that the degree of mating enhancement traded off with virulence so that enhanced disease transmission could only be achieved at the cost of increased virulence. It was also assumed that this trade-off was concave.

Using techniques from adaptive dynamics (Dieckmann, 2002; Diekmann, 2004), we found that disease-induced mating enhancement was generally less likely to evolve with a lower host reproduction rate β or a lower baseline disease transmission rate λ. In fact, β and λ are not only functions of the progeny size and the likelihood of disease transmission upon mating, respectively, but both are also functions of a constant scaling of the actual mating rate (c and $\mathcal{M}(1,1)$ when one or both sexes are affected by the infection, respectively). Hence, the higher the mating rate, the more effective the sexual route for disease transmission appears to be and therefore a higher potential for disease-induced mating enhancement can be expected. Further, if the parasite was affecting one sex only, disease-induced mating enhancement was found to be less likely to evolve relative to when the parasite affected both sexes.

We also demonstrated that evolution to disease-induced mating enhancement was less likely with lower b (the scaling for disease virulence) and higher z (quantifies the cost of mating enhancement). Hence, increasing the strength of the virulence-transmission trade-off may limit the evolution of mating enhancement. In this context it is interesting to note that the limiting, linear trade-off (with $z = 1$)

frequently led to evolution of ever higher mating enhancement (this was always the case when just one sex was affected by the infection). This implies that if real virulence-transmission trade-offs are linear then the model is missing an important component to capture why mating enhancement is not observed. However, it appears that real virulence-transmission trade-offs are concave (Alizon and Van Baalen, 2005; Alizon et al., 2009).

When disease-induced mating enhancement was equal in both females and males, the mating system had no effect on evolutionary outcomes. In contrast, if it acted on just one sex, the mating system played a significant role. In particular, the potential for evolution to disease-induced mating enhancement was higher the more polygynous the host population was. Therefore, it might be less probable to detect mating enhancement in polyandrous and monogamous species, relative to polygynous ones. Somewhat unexpectedly, this result did not depend on the affected sex; only minor quantitative differences were detectable between the alternative cases.

What are the other hypotheses concerning why mating enhancement is so rare? This is a difficult question to answer, given that literature addressing this is rare. However, given that we are discussing a sexually transmitted parasite it is not unreasonable to consider the possibility of a host protection being inherited and coevolving with the parasite strategy. In particular, an idea has been proposed that females may choose mates based on their resistance to parasites (Kokko et al., 2002, and references therein). For males, higher resistance would thus result in an increased chance of mating, but with lowered or no mating enhancement. While we acknowledge that this idea is compelling and worthy of further research within our modeling framework, it currently receives minimal support for STDs. In par-

ticular, it was suggested that there was a strong selection on STDs to become cryptic (Knell, 1999; Knell and Webberley, 2004) and that STDs could even act as a cost of choosing mates (Kokko et al., 2002; Knell and Webberley, 2004). Nevertheless, we believe that this would be an interesting topic for a follow-up study.

Alternatively, an infection may enhance one component of mating behavior while suppressing another one. As mentioned in the introduction, corn earworm *Helicoverpa zea* females infected by a gonad-specific virus produced up to three times more sex pheromone compared to uninfected females; however, because of severe deformities in their reproductive organs these females did not mate, forcefully avoiding copulation (Raina et al., 2000). Hence, the net effect of infection in this particular case is no reproduction of infected females. Thus, looking only at the net effect would not reveal the existing mating enhancement. Still, there is a possibility that the lack of evidence for disease-induced mating enhancement is in part due to a lack of studies, rather than because it is actually rare in nature. In any case, it remains a challenge to confidently state how widespread this type of mating enhancement is.

What values of δ correspond to the known observations of disease-induced mating enhancement as referred to in the introduction? For the midge *Paratrichocladius rufiventris* infested by the mite *Unionicola ypsilophora*, males form mating swarms that females enter and males then capture, suggesting that only males undergo behavioral changes from the infection. McLachlan (1999) observed that while the proportion of infected males in swarms was $\sim 4\%$, it was $\sim 15\%$ in mated pairs. We can use these values to estimate the degree of mating enhancement in this species as $\delta = (0.15/0.85)/(0.04/0.96) \approx 4.24$, a value that

fits our observed range e.g. in Fig. 6. For the milkweed leaf beetle *Labidomera clivicollis* infected with the mite *Chrysomelobia labidomera*, males were shown to displace rival males from mating pairs twice as much than uninfected males (Abbot and Dill, 2001). This suggests that it is males that might be affected by the infection; however the authors of that study doubt this male behavior is an adaptation by the parasite. Nonetheless, δ can in this case be roughly estimated as 2, which is again in the observed range (Fig. 6). Of course, a bit of caution is needed here, since we do not know an appropriate mating function as well as whether there is any potential trade-off that can act in these species. Still, it is appealing that the degree of mating enhancement observed in these two species is not at odds with what we arrive at with our relatively simple and generic models. As we mention in the previous paragraph, the net effect of infection of females of the corn earworm *Helicoverpa zea* by a gonad-specific virus is no reproduction of infected females, so it is not sensible to quantify the degree of mating enhancement in this particular case. Still, it is interesting to observe that there are systems in which females, not males, are the sex affected by the infection.

Acknowledging that disease-induced mating enhancement makes sense only for sexually transmitted infections, the key ingredients of our modeling approach are: (i) initial model formulation that accounts for both sexes, (ii) structural consistency between the processes of reproduction and disease transmission, mediated by mating, (iii) modeling mating rate via a degree-one homogeneous function, specifically the harmonic mean mating function when mating enhancement occurs in just one sex, and (iv) considering that enhanced mating and hence increased disease transmission will likely occur at the cost of increased virulence.

The points (i), (ii) and (iv) above were broadly motivated and justified within

the methods section. Degree-one homogeneous functions as models of mating (point (iii)) are widely accepted but at the same time also controversial. While scale invariant and 'naturally generalizing' linear birth rates are commonly assumed in many asexual models (Iannelli et al., 2005), these functions keep the per female mating rate constant if the adult sex ratio stays constant, irrespectively of how low male and female densities might be. This can be questionable, for example, when hosts are challenged by an Allee effect due to difficulty in finding mates at low densities (Courchamp et al., 2008; Gascoigne et al., 2009). A way to introduce this mate-finding Allee effect is to replace the harmonic mean mating function $\mathcal{M}(N_f, N_m) = 2cN_fN_m/(N_f+N_m)$ by the function $\mathcal{M}(N_f, N_m) = 2cN_fN_m/(N_f + N_m + \theta)$, where θ is a positive constant. Indeed, for a fixed female-to-male ratio, the per female mating rate $\mathcal{M}(N_f, N_m)/N_f$ now decreases with decreasing male density. Introduction of the mate-finding Allee effect into population models commonly begets population extinction when the host density falls below a critical value and there is a discontinuous change in equilibrium population density when the parameter θ exceeds a value (Courchamp et al., 2008; Boukal and Berec, 2009). Therefore, we expect such a modification to change the qualitative behavior of the model, the issue certainly worth separate investigation.

Another potential future direction of this work would be to include finite population sizes and demographic stochasticity. When the male mating potential h is not close to 1, the harmonic mean mating function amplifies demographic stochasticity such that populations can have larger fluctuations in size and sex ratio and are consequently more likely to go extinct (Bessa-Gomes et al., 2010). Because h also affects the potential for evolution of mating enhancement by an STD, adding

the next level of demographic realism could affect the results reported in this paper.

Beyond possible model extensions and alternative formulations of certain model components, the important issue is that there is need for an effort to identify more host populations and their parasites that interact such that mating within the host is enhanced by an action of the parasite. Only further empirical evidence can allow us to further distill fundamental characteristics of that interaction and allow us to develop and study the most appropriate models.

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Parameter	Meaning
b	Birth rate
γ_m (γ_f)	Fraction of males (females) among offspring
δ_m (δ_f)	Factor enhancing mating rate of infected males (females)
δ_{fm}	Factor enhancing mating rate between infected males and infected females
μ_m (μ_f)	Male (female) intrinsic mortality rate
w	Strength of negative density dependence in background mortality rate
α_m (α_f)	Male (female) disease-induced mortality rate
ξ_m (ξ_f)	Probability of disease transmission upon mating between a susceptible female and an infected male (a susceptible male and an infected female)

Table 1: Parameters used in the model (1).

Condition	Cases	$h(D_1)$	$h(1)$	$h(D_2)$	Evolutionary outcome
$1 < R_1 < R_2$		$> 0^a$	$> 0^a$	$< 0^a$	evolution to $\delta^* > 1^b$
$R_1 < 1 < R_2$, $b < 4\beta$	1	> 0	< 0	any	evolution to $\delta^* < 1^{b,c}$ or evolutionary suicide in $\delta < 1$ (δ passing through R_1)^c
	2	< 0	> 0	any	impossible case
	3 & 4	< 0	< 0	any	evolutionary suicide in $\delta < 1$ (δ passing through R_1)^c
	5	> 0	> 0	> 0	impossible case
	6	> 0	> 0	< 0	evolution to $\delta^* > 1^b$
$R_1 < 1 < R_2$, $b > 4\beta$	1 & 2	$\times^d$	$< 0^a$	any	evolutionary suicide in $\delta < 1$ (δ passing through R_1)^c

Note: ^{a} this is not an option but an implied result; ^{b} $\delta^* = [2\beta(\lambda + 2b) - b\lambda - \sqrt{\Delta}]/(4b\beta)$ where $\Delta = b\lambda[4\beta\lambda + b(\lambda - 8\beta)]$ (see Appendix C); ^{c} this case actually means no disease-induced mating enhancement; ^{d} $h(D_1)$ is irrelevant in this case

Table 2: Possible outcomes of evolution of the mating enhancement factor δ in the asexual case described by the model (5) and $z = 2$; see Appendix C for more details.

Figure legends:

Figure 1: The virulence-transmission trade-off (10), with $b = 3$ and $\lambda = 1$.

Figure 2: The (β, λ) parameter space split according to different potential evolutionary outcomes. Parameters: $z = 2$, $b = 3$ (left panels), $b = 6$ (right panels). The shaded area to the right of each panel corresponds to where the potential evolutionary attractor is disease-induced mating enhancement with $\delta^* > 1$ (white corresponds to $\delta^* = 1$ and black to the maximum observed δ^* in this area of the parameter space and for these parameter values). The shaded area to the left of each panel corresponds to either $\beta < \mu$ (to the left of the vertical dashed line; resident cannot persist) or $K^* < \mu$ for the respective potential evolutionary outcome (to the right of the vertical dashed line; evolutionary suicide is here the actual evolutionary outcome); $\mu = 0.1$ for the top panels, $\mu = 1.5$ for the middle panels, and $\mu = 4$ for the bottom panels. In the bottom left figure, the two shaded areas overlap, so that a dark gray line is used to emphasize the border of the left shaded area.

Figure 3: Example of evolutionary suicide. The top panel is the pairwise invasibility plot (PIP) for $\beta = 4.5$, $\lambda = 10$, $b = 3$, $z = 2$, $\mu = 4$, and $w = 0.1$. These parameters correspond to the mating enhancement area in the left column of Fig. 2. Evolution of δ here proceeds towards the lower left corner of the PIP, at which $\delta = 1.59$. The middle panels follows the resident-only dynamics for $\delta = 1.61$, i.e. just above the lower left corner of the PIP. After it establishes at the endemic equilibrium, a rare mutant with $\delta = 1.57$, i.e. just below the lower left corner of the PIP, is introduced, resulting in extinction of both hosts and parasites (the bottom panel).

Figure 4: Evolutionary outcome as a function of the cost of mating enhancement z. The thin black lines delimit the interval of δ for which $R_0(\delta) > 1$, and the thick gray line connects the evolutionary attractors as they vary with z. Parameter values: $\beta = 7$, $\lambda = 15$, $b = 3$.

Figure 5: Potential for disease-induced mating enhancement in the two-sex model (23) with $z = 2$ and females affected by the infection. In the grey area the infection persists in an endemic equilibrium and the selection gradient is positive for $\delta = 1.01$. Other parameter values: $\mu = 0.2$, $w = 0.1$. Virtually identical results were obtained when males were affected by the infection.

Figure 6: Evolutionary attractor δ^* as a function of the parameter b and the mating system parameter h, for two pairs of (β, λ) values and either females (left) or males (right) affected by the infection. Solid lines: $\beta = 10$, $\lambda = 20$; dashed lines: $\beta = 20$, $\lambda = 20$; black: $h = 0.5$ (polyandry); dark gray: $h = 1$ (monogamy); light gray: $h = 2$ (polygyny). Other parameter values: $z = 2$, $\mu = 0.2$, $w = 0.1$.

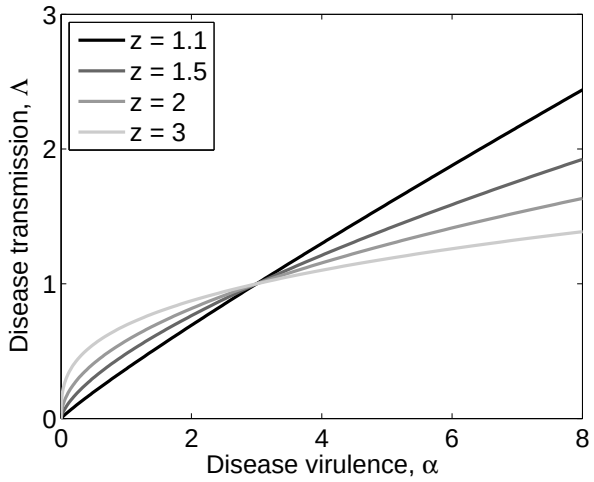
z = 1.1
z = 1.5
z = 2
z = 3
Disease transmission, Λ
Disease virulence, α
0
1
2
3
0
2
4
6
8

Figure 1:

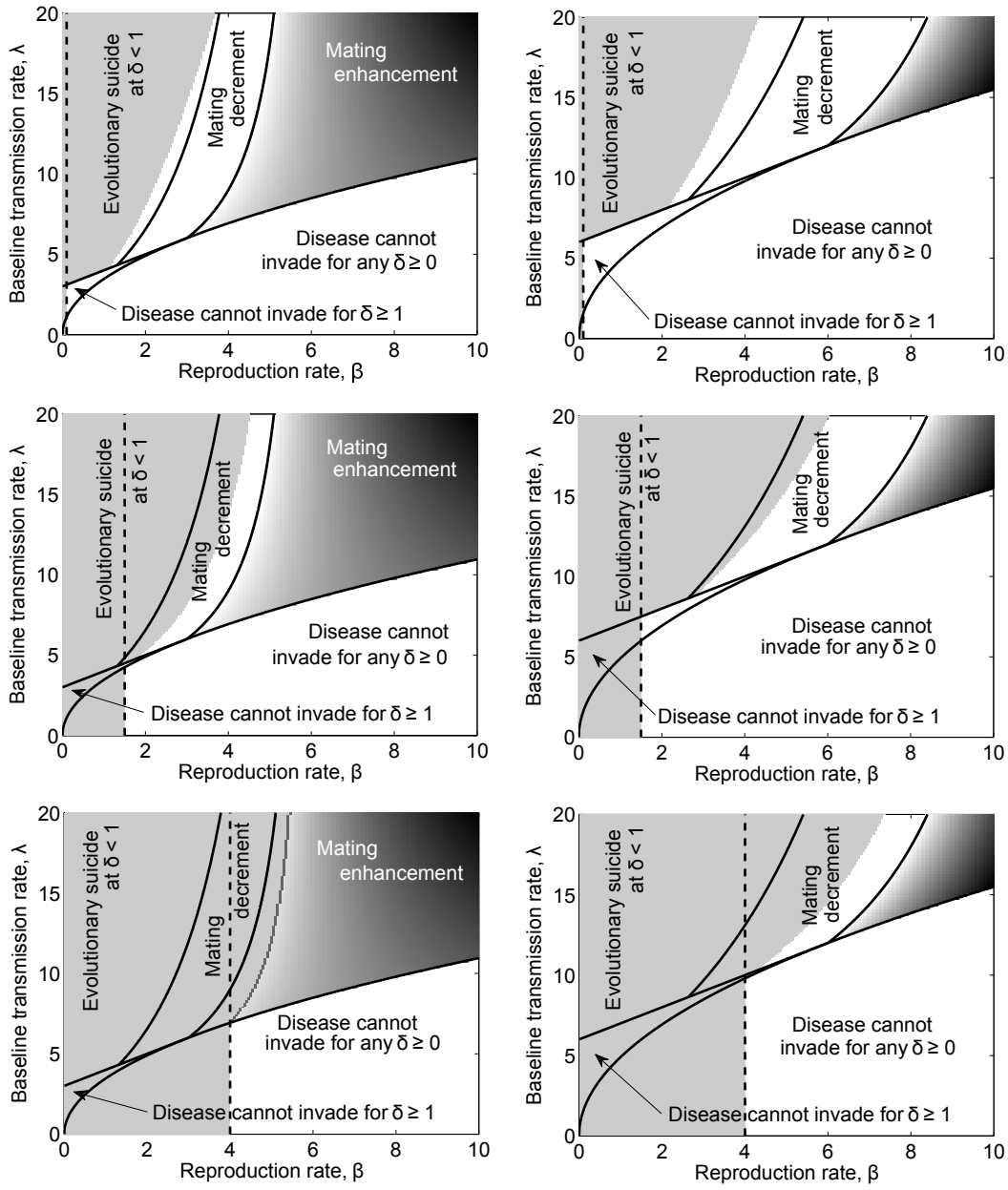
Baseline transmission rate, λ
Reproduction rate, β
Evolutionary suicide at δ < 1
Mating decrement
Mating enhancement
Disease cannot invade for any δ ≥ 0
Disease cannot invade for δ ≥ 1

Figure 2:

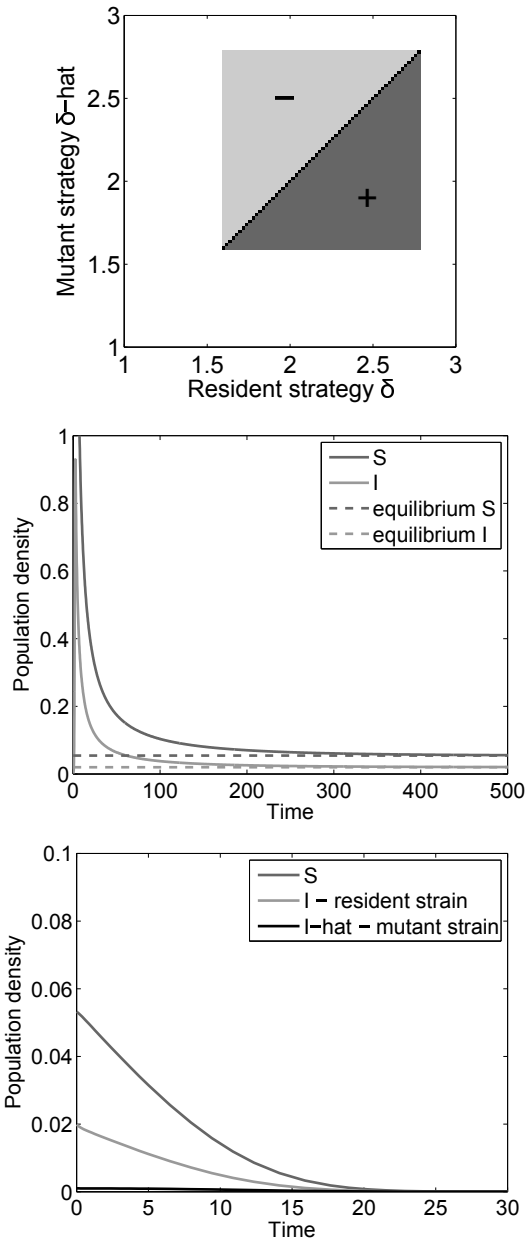
Mutant strategy δ−hat
Resident strategy δ
−
+
Population density
Time
S
I
equilibrium S
equilibrium I
Population density
Time
S
I − resident strain
I−hat − mutant strain

Figure 3:

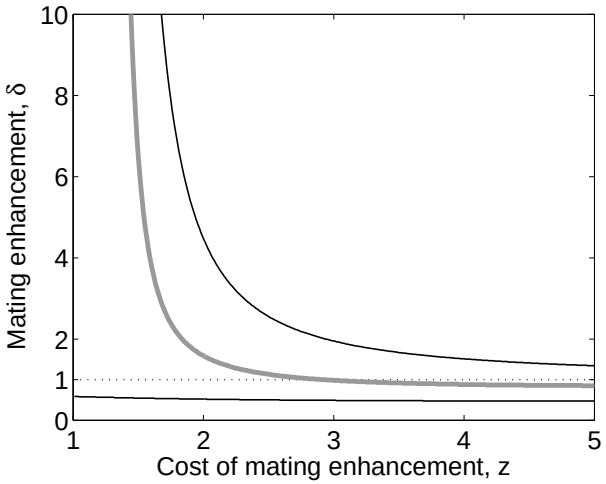
Mating enhancement, δ
Cost of mating enhancement, z
10
8
6
4
2
1
0
1
2
3
4
5

Figure 4:

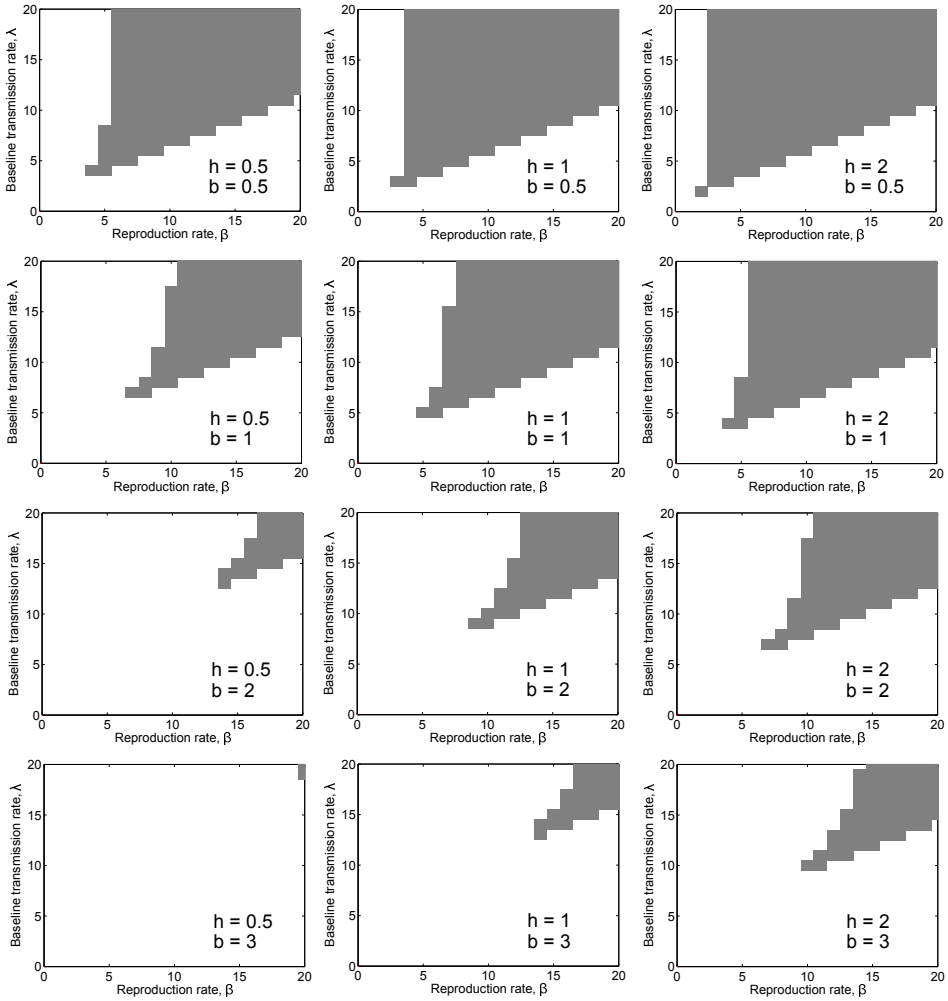
Baseline transmission rate, λ
Reproduction rate, β
h = 0.5
b = 0.5
h = 1
b = 0.5
h = 2
b = 0.5
h = 0.5
b = 1
h = 1
b = 1
h = 2
b = 1
h = 0.5
b = 2
h = 1
b = 2
h = 2
b = 2
h = 0.5
b = 3
h = 1
b = 3
h = 2
b = 3

Figure 5:

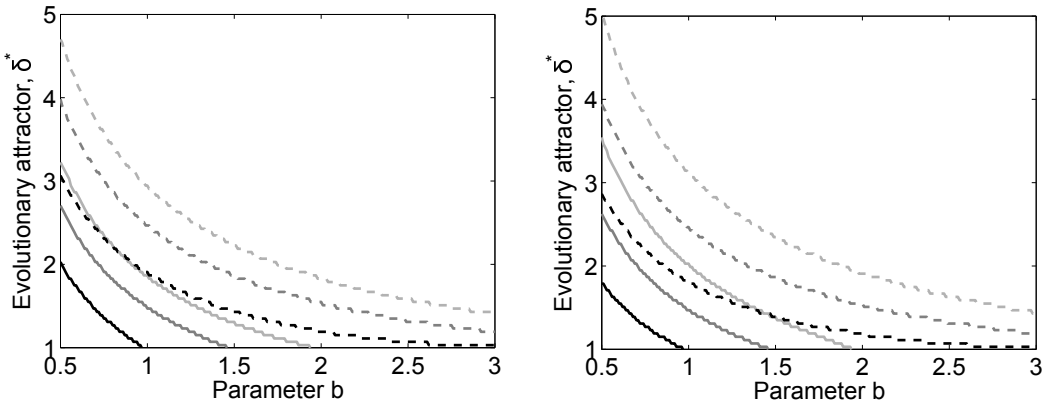
Evolutionary attractor, δ*
Parameter b
5
4
3
2
1
0.5
1
1.5
2
2.5
3
Evolutionary attractor, δ*
Parameter b

Figure 6:

Appendix A. Analysis of the asexual model (5)

We start with rewriting model (5) for the proportion of susceptible individuals $s = S/(S+I)$,

$$\frac{ds}{dt} = (1-s)\left\{\beta(1-2\delta+\delta_{fm})s^2 + [2\beta(\delta-\delta_{fm}) - \lambda\delta + \alpha]\, s + \beta\delta_{fm}\right\} \quad \text{(A.1)}$$

and the total population density $P = S + I$,

$$\frac{dP}{dt} = \left[\beta s^2 + 2\beta\delta s(1-s) + \beta\delta_{fm}(1-s)^2 - \alpha(1-s) - \mu - wP\right] P \quad \text{(A.2)}$$

Let $f(s)$ denote the right-hand side of equation (A.1). The equilibrium 1 of this equation corresponds to the disease-free equilibrium (DFE) of the original model (5) and $f'(1) = -\beta - \alpha + \lambda\delta$. Hence, the DFE is stable if and only if $R_0 < 1$. If $s \to 1$, it follows from equation (A.2) that $S \to (\beta - \mu)/w$ and $I \to 0$.

Let $g(s)$ denote the quadratic function in the curly brackets of equation (A.1), so that $ds/dt = (1-s)g(s)$. The discriminant of $g(s)$ is

$$\Delta = [2\beta(\delta - \delta_{fm}) - \lambda\delta + \alpha]^2 - 4\beta^2(1 - 2\delta + \delta_{fm})\delta_{fm}$$

and we consider two cases:

(1) $\delta \le \delta_{fm} < 2\delta - 1$. Since in this case $\Delta > 0$ and the coefficient by s^2 is negative, we have two distinct real roots of which one is positive and one is negative. The positive root is

$$s^* = \frac{2\beta(\delta - \delta_{fm}) - \lambda\delta + \alpha + \sqrt{\Delta}}{2\beta(2\delta - 1 - \delta_{fm})} \quad \text{(A.3)}$$

The negative leading coefficient of $g(s)$ also implies that $0 < s^* < 1$ if and only if $g(1) < 0$. Since $g(1) = \beta - \lambda\delta + \alpha < 0$ if and only if $R_0 > 1$ then, as expected, the

interior equilibrium for s is feasible if and only if the basic reproduction number of the infection is greater than one.

Denoting by s_1 the other (negative) real root of $g(s)$ we can write $f(s) = a(1-s)(s-s_1)(s-s^*)$, where $a = \beta(1-2\delta+\delta_{fm})$. Since $a < 0$ and $s_1 < s^* < 1$, then $f'(s^*) = a(1-s^*)(s^*-s_1) < 0$, which means that s^* is stable whenever it exists.

Returning to equation (A.2) for P and denoting

$$K^* = \beta\left[(s^*)^2 + 2\delta s^*(1-s^*) + \delta_{fm}(1-s^*)^2\right] - \alpha(1-s^*)$$

we distinguish two subcases. First, if $K^* < \mu$ then $P \to 0$ (note that K^* does not depend on μ) and the host population goes extinct. Second, if $K^* > \mu$ then the following endemic equilibrium is globally stable:

$$S \to \frac{s^*(K^*-\mu)}{w} \quad \text{and} \quad I \to \frac{(1-s^*)(K^*-\mu)}{w}$$

(2) $\delta_{fm} > 2\delta - 1$. In this case the coefficient by s^2 in $g(s)$ is positive and the product of two roots of $g(s)$ is also positive. Hence, if they are real, these roots are either both negative or both positive.

Suppose $R_0 > 1$ which is equivalent to $g(1) < 0$. Since $g(0) > 0$ and $g(\infty) > 0$ there are two positive real roots of $g(s)$ of which just the smaller one lies in the interval $(0,1)$. Moreover, this smaller root is defined by expression (A.3). A similar argument as above shows that also in this case s^* is stable.

Now suppose $R_0 < 1$ and write $g(s)$ shortly as $g(s) = a_2s^2 + a_1s + a_0$. The vertex of this quadratic function is located at $s_{\min} = -a_1/(2a_2)$ and has the value $g(s_{\min}) = a_0 - a_1^2/(4a_2)$. Since $a_2 > 0$ and $R_0 < 1$ is equivalent to $g(1) > 0$, the only case with feasible equilibria is when $s_{\min} \in (0,1)$ and $g(s_{\min}) < 0$. But this

also means that there will be two feasible equilibria in $(0, 1)$,

$$s^- = \frac{-\left[2\beta(\delta - \delta_{fm}) - \lambda\delta + \alpha\right] - \sqrt{\Delta}}{2\beta(1 - 2\delta + \delta_{fm})}$$

and

$$s^+ = \frac{-\left[2\beta(\delta - \delta_{fm}) - \lambda\delta + \alpha\right] + \sqrt{\Delta}}{2\beta(1 - 2\delta + \delta_{fm})}$$

with $s^- < s^+$. In addition, $a_2 > 0$ implies $f'(s^-) < 0$ and $f'(s^+) > 0$. Hence, the lower equilibrium s^- is stable and the higher equilibrium s^+ is unstable. As a result, we observe bistability in the dynamics of the proportion of susceptible individuals s. Whereas for $s(0) < s^+$ model solutions approach s^-, for $s(0) > s^+$ they approach 1. In terms of model parameters, the condition $g(s_{\min}) < 0$ is equivalent to $\Delta > 0$, and the condition $s_{\min} \in (0, 1)$ is equivalent to

$$-2\beta(1 - 2\delta + \delta_{fm}) < 2\beta(\delta - \delta_{fm}) - \lambda\delta + \alpha < 0 \tag{A.4}$$

For $s(0) < s^+$, system dynamics with respect to the original state variables S and I are as follows. Denoting

$$K^- = \beta\left[(s^-)^2 + 2\delta s^-(1 - s^-) + \delta_{fm}(1 - s^-)^2\right] - \alpha(1 - s^-)$$

we distinguish two subcases. First, if $K^- < \mu$ then $P \to 0$ (note that K^- does not depend on μ) and the host population goes extinct. Second, if $K^- > \mu$ then the following endemic equilibrium is (now locally) stable:

$$S \to \frac{s^-(K^- - \mu)}{w} \quad \text{and} \quad I \to \frac{(1 - s^-)(K^- - \mu)}{w}$$

Appendix B. Evolution of disease-induced mating enhancement for the asexual model (5) and $z = 1$

The assumption of endemicity of a resident strain, $R_0 > 1$ is equivalent to $\lambda > b$ and $\delta > \delta_R = \beta/(\lambda - b)$. The selection gradient $g(\delta) = \lambda s^*(\delta) - b$ where

$$s^*(\delta) = \frac{2\beta\delta}{2\beta(\delta-1) + \lambda - b + \sqrt{(\lambda-b)[4\beta(\delta-1)+\lambda-b]}}$$

is defined for any $\delta > \delta_s = 1 - (\lambda - b)/(4\beta)$ and has two roots (note that $\delta_s < 1$):

$$\delta_1 = \frac{b(\lambda - 2\beta - \sqrt{\lambda^2 - 4\beta\lambda})}{2\beta(\lambda - b)} \quad \text{and} \quad \delta_2 = \frac{b(\lambda - 2\beta + \sqrt{\lambda^2 - 4\beta\lambda})}{2\beta(\lambda - b)}.$$

Since $\delta_1\delta_2 > 0$ both roots, if real, have the same sign. Also, $\delta_R > \delta_s$ and $\delta_s < \delta_1 < \delta_2$. Moreover, $g(\delta)$ has a positive horizontal asymptote $g(\infty) = \lambda - b > 0$.

If $\lambda < 4\beta$ then $g(\delta)$ has no real root, hence $g(\delta) > 0$ for all δ in the domain of g. This implies that the system evolves to ever higher mating enhancement.

If $\lambda > 4\beta$ then both δ_1 and δ_2 are real and positive. This implies $g(\delta) < 0$ whenever $\delta_1 < \delta < \delta_2$. It remains to compare these thresholds with 1 since by definition $\delta > 1$.

(1) $1 < \delta_1 < \delta_2$ which is equivalent to

$$b > \frac{\lambda + \sqrt{\lambda^2 - 4\beta\lambda}}{2}$$

In this case $g(\delta) > 0$ for $\delta \in (1, \delta_1) \cup (\delta_2, \infty)$ and negative otherwise. Also, $\delta_R < \delta_1$. Therefore, if our current strain has $\delta < \delta_2$ then it will evolve to δ_1. If it has $\delta > \delta_2$ then it will evolve to ever higher mating enhancement.

(2) $\delta_1 < 1 < \delta_2$ which is equivalent to

$$\frac{\lambda - \sqrt{\lambda^2 - 4\beta\lambda}}{2} < b < \frac{\lambda + \sqrt{\lambda^2 - 4\beta\lambda}}{2}$$

Here $g(\delta) < 0$ if $\delta < \delta_2$ and $g(\delta) > 0$ if $\delta > \delta_2$. This also implies $\delta_R < \delta_2$. Moreover, since $\delta_1 < \delta_R$ is equivalent to $b < [\lambda - 2\beta + \sqrt{\lambda^2 - 4\beta\lambda}]/2$ this case splits into two subcases. If

$$\frac{\lambda - \sqrt{\lambda^2 - 4\beta\lambda}}{2} < b < \frac{\lambda - 2\beta + \sqrt{\lambda^2 - 4\beta\lambda}}{2}$$

then $\delta_1 < \delta_R$ so that if our current strain has $\delta < \delta_2$ we observe evolutionary suicide as the parasite aims to reach δ_1. If, on the other hand,

$$\frac{\lambda - 2\beta + \sqrt{\lambda^2 - 4\beta\lambda}}{2} < b < \frac{\lambda + \sqrt{\lambda^2 - 4\beta\lambda}}{2}$$

then $\delta_1 > \delta_R$ so that if our current strain has $\delta < \delta_2$ it will evolve to $\delta_1 < 1$ (i.e. sterilization). In both subcases, if our current strain has $\delta > \delta_2$ it will evolve to ever higher mating enhancement.

(3) $\delta_1 < \delta_2 < 1$ which is equivalent to

$$b < \frac{\lambda - \sqrt{\lambda^2 - 4\beta\lambda}}{2}$$

In this case $g(\delta) > 0$ for all $\delta > 1$. Hence, for all initial $\delta > \delta_R$ the system evolves to ever higher mating enhancement.

Appendix C. Evolution of disease-induced mating enhancement for the asexual model (5) and $z = 2$

The function $h(\delta)$ defined by (21) has at most two roots:

$$\delta_1 = \frac{2\beta(\lambda + 2b) - b\lambda - \sqrt{\Delta}}{4b\beta} \quad \text{and} \quad \delta_2 = \frac{2\beta(\lambda + 2b) - b\lambda + \sqrt{\Delta}}{4b\beta}$$

with $\Delta = b\lambda[4\beta\lambda + b(\lambda - 8\beta)]$. Since $R_0 > 1$ requires $\lambda > \sqrt{4b\beta}$ and since it is easy to show that

$$\sqrt{4b\beta} > \frac{8b\beta}{4\beta + b}$$

we have $\lambda > 8b\beta/(4\beta + b)$ and hence $\Delta > 0$. Therefore, both δ_1 and δ_2 are real.

However, one or both of these roots may be extraneous, since they are computed by squaring the radical in the equation $h(\delta) = 0$ (after isolating the radical). The true solutions of the equation $h(\delta) = 0$ must satisfy

$$\lambda(\beta - b) + 2b\beta + b\delta(b - 2\beta) = b\sqrt{(\lambda - b\delta)[4\beta(\delta - 1) + \lambda - b\delta]} > 0 \quad \text{(C.1)}$$

We also note that $h(\delta_1) < 0$ or $h(\delta_2) < 0$ whenever δ_1 or δ_2 is extraneous, respectively.

For δ_1 or δ_2 to be evolutionary singular points, they must lie in the interval $(\max\{R_1, 1\}, R_2)$. However, if δ_2 is a root of $h(\delta)$, then $\delta_2 > R_2$ and hence δ_2 cannot be an evolutionary singular point. Indeed, if δ_2 satisfies condition (C.1) then by inserting δ_2 into this condition, one obtains

$$\lambda(\beta - b) + 2b\beta + b(b - 2\beta)\frac{2\beta(\lambda + 2b) - b\lambda + \sqrt{\Delta}}{4b\beta} > 0$$

and hence

$$4b\beta[\lambda(\beta - b) + 2b\beta] + b(b - 2\beta)[2\beta(\lambda + 2b) - b\lambda] + b(b - 2\beta)\sqrt{\Delta} > 0$$

Moving the radical to the right and simplifying the left-hand side,

$$b^3(4\beta - \lambda) > b(2\beta - b)\sqrt{\Delta} \Leftrightarrow b^2(4\beta - \lambda) + b\sqrt{\Delta} > 2\beta\sqrt{\Delta}$$

We need to prove $\delta_2 - R_2 > 0$ which is equivalent to

$$b^2(4\beta - \lambda) + b\sqrt{\Delta} - 2b\beta\sqrt{\lambda^2 - 4b\beta} > 0$$

but the previous inequality implies

$$b^2(4\beta - \lambda) + b\sqrt{\Delta} - 2b\beta\sqrt{\lambda^2 - 4b\beta} > 2\beta(\sqrt{\Delta} - b\sqrt{\lambda^2 - 4b\beta})$$

Finally, the right-hand side of this inequality is positive since $\Delta - b^2(\lambda^2 - 4b\beta) = 4b\beta(\lambda - b)^2 > 0$, and we are done.

As far as location of the $h(\delta)$ domain end-points D_1 and D_2 relative to the endemicity thresholds R_1 and R_2 we have the following result. If the $h(\delta)$ domain is (D_1, D_2) then $D_1 < R_1 < R_2 < D_2$. The right side of the inequality is easy to check. To show $D_1 < R_1$ requires some computation but it is equivalent to

$$(4\beta - b)\sqrt{\lambda^2 - 4b\beta} < \frac{\Delta}{b\lambda} \Leftrightarrow 4b\beta(b + 4\beta - 2\lambda)^2 > 0$$

If the (relevant) $h(\delta)$ domain is $[0, D_2)$ it is easy to show that $R_1 < R_2 < D_2$.

We are now going to analyze all possible situations in detail, noting that

$$h(D_1) = \frac{2b^2 + \lambda(4\beta - 3b)}{2(\lambda - b)},\ h(1) = \frac{2b^2 + \lambda(\beta - 2b)}{2(\lambda - b)},\ h(D_2) = \frac{b(2b - \lambda)}{2(\lambda - b)}$$

First, assume (18) and (19), i.e. $1 < R_1 < R_2$, for which the domain of $h(\delta)$ is (D_1, D_2). Moreover, $\beta + b > \lambda > 2b$ which in turn implies $\beta > b$ and $2\beta > \lambda$. Since $h(D_1) > 0$ and $h(D_2) < 0$, $h(\delta)$ has a unique root in its domain. This root must be δ_1 since if it was δ_2 it would imply that δ_1 is extraneous and $h(\delta_1) > 0$, contrary to what we know from above. We also note that $h(1) > 0$. This is obvious if $\beta > 2b$, otherwise, it follows from $\lambda < \beta + b < 2b^2/(2b - \beta)$. A tedious computation also shows that $R_1 < \delta_1 < R_2$. Specifically,

$$\delta_1 > R_1 \Leftrightarrow [4b\beta(4\beta - \lambda)\sqrt{\lambda^2 - 4b\beta} + 4\beta(\beta - b)(\lambda^2 - 4b\beta)] > 0$$

and

$$\delta_1 < R_2 \Leftrightarrow [4\beta\sqrt{\Delta}\sqrt{\lambda^2 - 4b\beta} + 4\beta(\beta + b)(\lambda^2 - 4b\beta)] > 0$$

Hence, altogether, we have $D_1 < 1 < R_1 < \delta_1 < R_2 < D_2$ and $h(\delta) > 0$ if $\delta < \delta_1$ and $h(\delta) < 0$ if $\delta > \delta_1$. So, the mating enhancement trait $\delta^* = \delta_1 > 1$

is convergence stable. Since we already know it is evolutionary stable, it is the unique evolutionary attractor.

Second, assume (18) and (20), i.e. $R_1 < 1 < R_2$ and hence $\lambda > \beta + b > b$. We need to distinguish several subcases. We first assume $b < 4\beta$ for which the $h(\delta)$ domain is (D_1, D_2) and for which we have six subcases:

(1) $h(D_1) > 0$ and $h(1) < 0$. This implies that $h(\delta)$ has a unique root in the interval $(D_1, 1)$. If $h(D_2) > 0$ there is another root δ_2 in the interval (R_2, D_2) and the first root is δ_1. If $h(D_2) < 0$ the first root must be δ_1 since if it was δ_2 it would imply that δ_1 is extraneous and $h(\delta_1) > 0$, contrary to what we know from above. In both cases we have $h(\delta) > 0$ in the interval (D_1, δ_1) and $h(\delta) < 0$ in the interval (δ_1, R_2). The evolutionary outcome thus depends on how δ_1 compares to R_1. For $D_1 < R_1 < \delta_1 < 1 < R_2 < D_2$ evolution leads to $\delta^* = \delta_1 < 1$ (i.e. mating decrement). For $D_1 < \delta_1 < R_1 < 1 < R_2 < D_2$ evolution leads to evolutionary suicide in the domain $\delta < 1$. In any case, we have here evolution to no disease-induced mating enhancement.

(2) $h(D_1) < 0$ and $h(1) > 0$. This case is impossible. To realize this, first note that $h(D_1) < 0$ implies $\beta < 3b/4 < b$ which also implies $\beta < 2b$. Given this we see that $h(1) > 0$ implies $\lambda < 2b^2/(2b - \beta)$. However, this must be consistent with $\lambda > \beta + b$. Some computation shows that

$$\beta + b < \frac{2b^2}{2b - \beta} \Leftrightarrow b < \beta$$

which contradicts the upper bound on β established above.

(3) $h(D_1) < 0$, $h(1) < 0$ and $h(D_2) > 0$. In this case there is a unique root in the $h(\delta)$ domain which must be δ_2 for reasons similar to those given in the case (1). Since we know $\delta_2 > R_2$, we have $D_1 < R_1 < 1 < R_2 < \delta_2 < D_2$ and evolution leads to evolutionary suicide in the domain $\delta < 1$. Again, we have here

evolution to no disease-induced mating enhancement.

(4) $h(D_1) < 0$, $h(1) < 0$ and $h(D_2) < 0$. This implies there are no roots of $h(\delta)$ at all. For if there is a root, there must be two of them and, due to continuity of the derivative of $h(\delta)$ on its domain there must be a positive maximum of $h(\delta)$ between those two roots. However, this is not possible since

$$\tilde{\delta} = \frac{b^2 + 4\beta\lambda - 2b\lambda}{b(4\beta - b)}$$

is the only critical point of $h(\delta)$ and

$$h(\tilde{\delta}) = -\frac{\Delta}{8\beta\lambda(\lambda - b)} < 0$$

So $h(\delta)$ is negative on its entire domain and we again observe evolutionary suicide as in the case (3), so evolution to no disease-induced mating enhancement.

(5) $h(D_1) > 0$, $h(1) > 0$ and $h(D_2) > 0$. This case is impossible. To see this, first note that $h(D_2) > 0$ implies $b > \beta$. Then it follows from $h(1) > 0$ that $\lambda < 2b^2/(2b - \beta)$ but this leads to contradiction since $\lambda > \beta + b$ implies

$$\beta + b < \frac{2b^2}{2b - \beta} \Leftrightarrow b < \beta$$

(6) $h(D_1) > 0$, $h(1) > 0$ and $h(D_2) < 0$. This implies a unique root in the interval $(1, D_2)$ which must be δ_1 for reasons similar to those given in the case (1). Moreover, we know from the case (1) that $\delta_1 < R_2$. So we have $h(\delta) > 0$ for $\delta < \delta_1$ and $h(\delta) < 0$ for $\delta > \delta_1$. Therefore, $D_1 < R_1 < 1 < \delta_1 < R_2 < D_2$ and evolution leads to $\delta^* = \delta_1 > 1$.

Second, we assume $b > 4\beta$ for which the (relevant) $h(\delta)$ domain is $(0, D_2)$. Note first that this implies $h(1) < 0$ ($h(1) > 0$ would again imply a conflict with $\lambda > \beta + b$ as shown above). Two possible cases are thus $h(D_2) > 0$ and

$h(D_2) < 0$. For both, we observe evolutionary suicide when the declining δ hits the R_1 value, so evolution to no disease-induced mating enhancement:

(1) $h(D_2) > 0$. This implies a unique root in the interval $(1, D_2)$ which must be δ_2. Since $\delta_2 > R_2$ we have that $h(\delta) < 0$ on (R_1, R_2) and we thus get evolutionary suicide.

(2) $h(D_2) < 0$. In this case, also using the observation that $h(-\infty) = -b < 0$, we can show that there are no roots of $h(\delta)$, using a similar argument as in the case (4). So again we have $h(\delta) < 0$ on its domain and thus get evolutionary suicide.

Appendix D. The basic reproduction number R_0 of the infection described by the two-sex model (1) with the harmonic mean mating function

Let us assume the harmonic mean mating function

$$\mathcal{M}(S_f + I_f, S_m + I_m) = 2c\frac{(S_f + I_f)(S_m + I_m)}{(S_f + I_f)/h + S_m + I_m} \tag{D.1}$$

where $h < 1$ corresponds to polyandry, $h = 1$ to monogamy, and $h > 1$ to polygyny. With this function, model (1) becomes

$$\begin{aligned}
\frac{dS_f}{dt} &= 2cb\gamma_f\left(\frac{S_fS_m}{T} + \delta_m\frac{S_fI_m}{T} + \delta_f\frac{I_fS_m}{T} + \delta_{fm}\frac{I_fI_m}{T}\right) - \bar{\mu}_fS_f - 2c\xi_m\delta_m\frac{S_fI_m}{T}\\
\frac{dS_m}{dt} &= 2cb\gamma_m\left(\frac{S_fS_m}{T} + \delta_m\frac{S_fI_m}{T} + \delta_f\frac{I_fS_m}{T} + \delta_{fm}\frac{I_fI_m}{T}\right) - \bar{\mu}_mS_m - 2c\xi_f\delta_f\frac{I_fS_m}{T}\\
\frac{dI_f}{dt} &= 2c\xi_m\delta_m\frac{S_fI_m}{T} - \bar{\mu}_fI_f - \alpha_fI_f\\
\frac{dI_m}{dt} &= 2c\xi_f\delta_f\frac{I_fS_m}{T} - \bar{\mu}_mI_m - \alpha_mI_m
\end{aligned} \tag{D.2}$$

where $T = (S_f + I_f)/h + S_m + I_m$.

In the absence of disease, model (D.2) reduces to the two-sex model

$$\begin{aligned}\frac{dS_f}{dt} &= 2cb\gamma_f \frac{S_f S_m}{S_f/h + S_m} - \bar{\mu}_f S_f \\ \frac{dS_m}{dt} &= 2cb\gamma_m \frac{S_f S_m}{S_f/h + S_m} - \bar{\mu}_m S_m\end{aligned} \tag{D.3}$$

This model has been analyzed in Maxin et al. (2012) for the monogamous case ($h = 1$). Here, we obtain a similar result for the polygamous case ($h \neq 1$). From inequalities

$$\frac{S_m}{S_f/h + S_m} < 1 \quad \text{and} \quad \frac{S_f}{S_f/h + S_m} < h$$

we obtain that

$$S_f' < 2cb\gamma_f S_f - \bar{\mu}_f S_f \quad \text{and} \quad S_m' < 2cb\gamma_m h S_m - \bar{\mu}_m S_m$$

This means that a necessary condition for host population persistence is

$$\mathcal{R}_f = \frac{2cb\gamma_f}{\mu_f} > 1 \quad \text{and} \quad \mathcal{R}_m = \frac{2cb\gamma_m h}{\mu_m} > 1$$

Rewriting model (D.3) in terms of the proportion of females $x = S_f/P$ and the total population density P we obtain, noting that $\gamma_f + \gamma_m = 1$,

$$\begin{aligned}\frac{dx}{dt} &= x(1-x)\left[\frac{2cb(\gamma_f - x)}{x/h + 1 - x} + \mu_m - \mu_f\right] \\ \frac{dP}{dt} &= \left[\frac{2cbx(1-x)}{x/h + 1 - x} - \mu_f x - \mu_m(1-x) - wP\right] P\end{aligned} \tag{D.4}$$

There are three equilibria for x: 0 and 1 which both lead to population extinction, and an interior one

$$x^* = \frac{2cb\gamma_f - \mu_f + \mu_m}{2cb + (1/h - 1)(\mu_f - \mu_m)}$$

which is globally stable whenever it exists in the feasible interval $(0, 1)$.

It is easy to see that $\mathcal{R}_f > 1$ and $\mathcal{R}_m > 1$ imply $x^* \in (0,1)$. Letting $x(t) \to x^*$ in the equation for P (D.4) we obtain a limiting logistic equation in P,

$$P' = \left[\frac{2cb\gamma_f\gamma_m - \gamma_m\mu_f - \gamma_f\mu_m/h}{\gamma_f/h + \gamma_m} - wP \right] P$$

With this equation, the limit of $P(t)$ as $t \to \infty$ is as follows:

$$P(t) \to P^* = \frac{2cb\gamma_f\gamma_m}{w(\gamma_f/h + \gamma_m)} \left(1 - \frac{1}{\mathcal{R}}\right)$$

if and only if

$$\mathcal{R} = \frac{\mathcal{R}_m\mathcal{R}_f}{\mathcal{R}_m + \mathcal{R}_f} > 1$$

Otherwise, if $\mathcal{R} < 1$, $P(t) \to 0$. In addition, $S_f(t) \to S_f^* = x^*P^*$ and $S_m(t) \to S_m^* = (1 - x^*)P^*$.

Hence, altogether, if $\mathcal{R} > 1$ then the interior equilibrium (S_f^*, S_m^*) is globally stable and the extinction equilibrium $(0,0)$ is unstable. Conversely, if $\mathcal{R} < 1$, the extinction equilibrium $(0,0)$ is globally stable and the interior equilibrium (S_f^*, S_m^*) is not (biologically) feasible.

Now, let the population persist in the absence of disease, i.e. assume that $\mathcal{R} > 1$, and denote $\mu_f^* = \mu_f + w(S_f^* + S_m^*)$ and $\mu_m^* = \mu_m + w(S_f^* + S_m^*)$. Using the next generation matrix approach (van den Driessche and Watmough, 2002), the basic reproduction number R_0 of the infection corresponding to model (D.2) is given by the spectral radius of the matrix FV^{-1} where

$$F = \begin{pmatrix} 0 & \dfrac{2c\xi_m\delta_m S_f^*}{S_f^*/h + S_m^*} \\ \dfrac{2c\xi_f\delta_f S_m^*}{S_f^*/h + S_m^*} & 0 \end{pmatrix}$$

and

$$V = \begin{pmatrix} \mu_f^* + \alpha_f & 0 \\ \\ 0 & \mu_m^* + \alpha_m \end{pmatrix}$$

The characteristic polynomial of the matrix FV^{-1} is

$$\lambda^2 - \frac{2c\xi_m 2c\xi_f \delta_m \delta_f S_f^* S_m^*}{(S_f^*/h + S_m^*)^2(\mu_f^* + \alpha_f)(\mu_m^* + \alpha_m)}$$

It has one positive root and one negative root. Since both roots have the same absolute value, the basic reproduction number R_0 of the infection described by the two-sex model (1) with the harmonic mean mating function is

$$R_0 = 2c\sqrt{\frac{\xi_m \xi_f \delta_m \delta_f S_f^* S_m^*}{(S_f^*/h + S_m^*)^2(\mu_f^* + \alpha_f)(\mu_m^* + \alpha_m)}} \tag{D.5}$$

Appendix E. Population dynamics and evolution for the two-sex model (23)

We start with analyzing population dynamics of the two-sex model (23). In terms of proportions $x = S_f/P$, $y = S_m/P$ and $z = I_f/P$, the model (23) can be rewritten as:

$$\begin{aligned}
\frac{dx}{dt} &= \beta\left(\frac{1}{2} - x\right)\frac{(x+\delta z)(1-x-z)}{1+(1/h-1)(x+z)} + x[\alpha_f z + \alpha_m(1-x-y-z)] - \frac{\lambda x(1-x-y-z)}{1+(1/h-1)(x+z)} \\
\frac{dy}{dt} &= \beta\left(\frac{1}{2} - y\right)\frac{(x+\delta z)(1-x-z)}{1+(1/h-1)(x+z)} + y[\alpha_f z + \alpha_m(1-x-y-z)] - \frac{\lambda \delta yz}{1+(1/h-1)(x+z)} \\
\frac{dz}{dt} &= \frac{\lambda x(1-x-y-z) - \beta z(x+\delta z)(1-x-z)}{1+(1/h-1)(x+z)} - \alpha_f z + z[\alpha_f z + \alpha_m(1-x-y-z)]
\end{aligned} \tag{E.1}$$

with the total population density P evolving as

$$\frac{dP}{dt} = \left[\frac{\beta(x+\delta z)(1-x-z)}{1+(1/h-1)(x+z)} - \alpha_f z - \alpha_m(1-x-y-z) - \mu - wP\right]P \tag{E.2}$$

The subsystem of x, y and z has five boundary equilibria: $(1,0,0)$, $(0,1,0)$, $(0,0,1)$, $(0,0,0)$ and $(1/2,1/2,0)$. The first four equilibria are all unstable since they have positive eigenvalue $\beta h/2$, $\beta/2$, α_f, and α_m, respectively. The equilibrium $(1/2,1/2,0)$ is the DFE; its highest eigenvalue is

$$-\frac{\beta+(1/h+1)(\alpha_f+\alpha_m)-\sqrt{(1/h+1)^2(\alpha_f-\alpha_m)^2+4\delta\lambda^2}}{2(1/h+1)}$$

This eigenvalue is negative if and only if $R_0<1$; the DFE is thus stable if $R_0<1$ and unstable if $R_0>1$. We note that these results stay the same if males instead of females are affected by the infection.

To study evolution of disease-induced mating enhancement, i.e. of the mating enhancement factor δ, we assume that the resident parasite strain has $\delta\geq 1$ and that the system is in a stable endemic equilibrium $(S_f^*(\delta),S_m^*(\delta),I_f^*(\delta),I_m^*(\delta))$. To see how δ evolves in the two-sex model (23) we look at when this resident endemic equilibrium becomes unstable (Bacelar et al., 2011). Assuming no co-infection or superinfection, the full model containing both the resident parasite strain δ and the mutant strain $\hat{\delta}$ is

$$\begin{aligned}
\frac{dS_f}{dt}&=\frac{\beta}{2}\frac{(S_f+\delta I_f+\hat{\delta}J_f)(S_m+I_m+J_m)}{T}-\bar{\mu}S_f-\lambda\frac{S_fI_m}{T}-\lambda\frac{S_fJ_m}{T}\\
\frac{dS_m}{dt}&=\frac{\beta}{2}\frac{(S_f+\delta I_f+\hat{\delta}J_f)(S_m+I_m+J_m)}{T}-\bar{\mu}S_m-\lambda\delta\frac{S_mI_f}{T}-\lambda\hat{\delta}\frac{S_mJ_f}{T}\\
\frac{dI_f}{dt}&=\lambda\frac{S_fI_m}{T}-\bar{\mu}I_f-b\delta^zI_f\\
\frac{dI_m}{dt}&=\lambda\delta\frac{I_fS_m}{T}-\bar{\mu}I_m-bI_m\\
\frac{dJ_f}{dt}&=\lambda\frac{S_fJ_m}{T}-\bar{\mu}J_f-b\hat{\delta}^zJ_f\\
\frac{dJ_m}{dt}&=\lambda\hat{\delta}\frac{J_fS_m}{T}-\bar{\mu}J_m-bJ_m
\end{aligned}\tag{E.3}$$

where J_f and J_m are densities of females and males infected by the mutant, respectively, and $T = (S_f + I_f + J_f)/h + S_m + I_m + J_m$. Following Bacelar et al. (2011), we calculate the Jacobian of the (J_f, J_m) subsystem of the model (E.3) and evaluate it at the resident boundary equilibrium $E_I = (S_f^*, S_m^*, I_f^*, I_m^*, 0, 0)$. This Jacobian is

$$J_{E_I} = \begin{pmatrix} -\mu - wP^* - b\hat{\delta}^z & \lambda\frac{S_f^*}{T^*} \\ \\ \lambda\hat{\delta}\frac{S_m^*}{T^*} & -\mu - wP^* - b \end{pmatrix}$$

Since the trace of J_{E_I} is negative, E_I will be unstable and the mutant will be able to invade as soon as $\det(J_{E_I}) < 0$. Hence, we can consider the function

$$f(\hat{\delta}, \delta) = -\det J_{E_I}$$

as a proxy for the invasion fitness in the two-sex model (23); see also Bacelar et al. (2011). The equations for I_f and I_m in the model (E.3) imply that at E_I

$$\mu + wP^* + b\hat{\delta}^z = b(\hat{\delta}^z - \delta^z) + \lambda\frac{S_f^* I_m^*}{T^* I_f^*}$$

and

$$\mu + wP^* + b = \lambda\delta\frac{S_m^* I_f^*}{T^* I_m^*}$$

Using these identities, the invasion fitness of the mutant strain is

$$f(\hat{\delta}, \delta) = -\det J_{E_I} = \lambda^2(\hat{\delta} - \delta)\frac{S_m^* S_f^*}{(T^*)^2} - b(\hat{\delta}^z - \delta^z)\lambda\delta\frac{S_m^* I_f^*}{T^* I_m^*} \tag{E.4}$$

If males instead of females are the sex affected by the infection, the invasive fitness is

$$f(\hat{\delta}, \delta) = -\det J_{E_I} = \lambda^2(\hat{\delta} - \delta)\frac{S_m^* S_f^*}{(T^*)^2} - b(\hat{\delta}^z - \delta^z)\lambda\delta\frac{S_f^* I_m^*}{T^* I_f^*} \tag{E.5}$$

The evolution makes sense only if it starts at δ for which $R_0(\delta) > 1$. If we replace $\alpha_f = b\delta^2$ and $\alpha_m = b$ in the formula (24) then

$$R_0(\delta) = \frac{2\lambda\sqrt{\delta}}{\sqrt{(\beta + 2(1/h+1)b\delta^2)(\beta + 2(1/h+1)b)}} > 1$$

if and only if $\Delta \equiv 16\lambda^4 - 8b\beta(1/h+1)(\beta + 2b(1/h+1))^2 > 0$ and $\delta_1 < \delta < \delta_2$ where

$$\delta_1 = \frac{4\lambda^2 - \sqrt{\Delta}}{4b(1/h+1)(\beta + 2b(1/h+1))},\ \delta_2 = \frac{4\lambda^2 + \sqrt{\Delta}}{4b(1/h+1)(\beta + 2b(1/h+1))}$$

So if we look at Δ a bigger b will decrease the area in the (β, λ) parameter subspace where $\Delta > 0$. The same happens if h decreases, i.e. if there is more polyandry.

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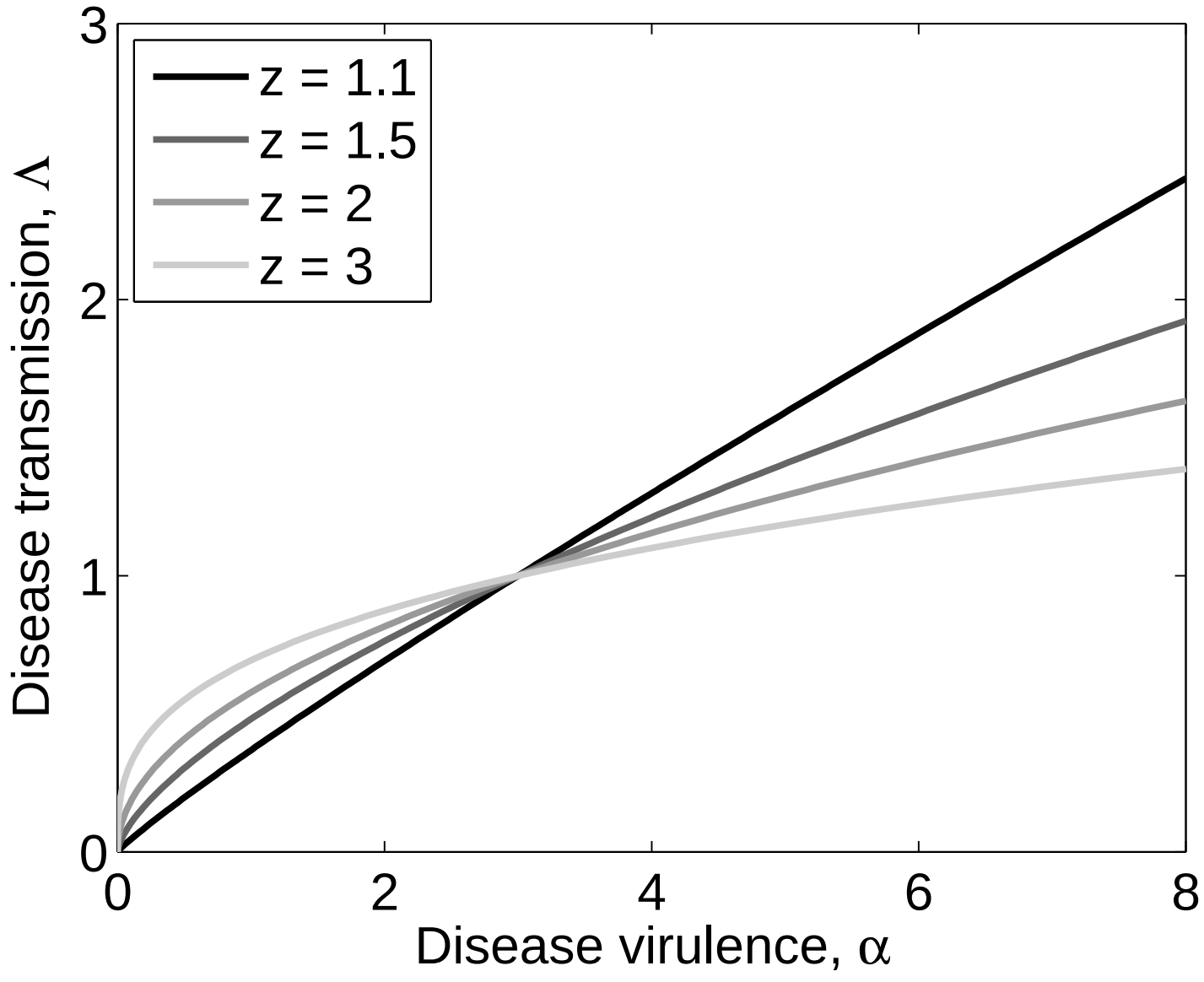

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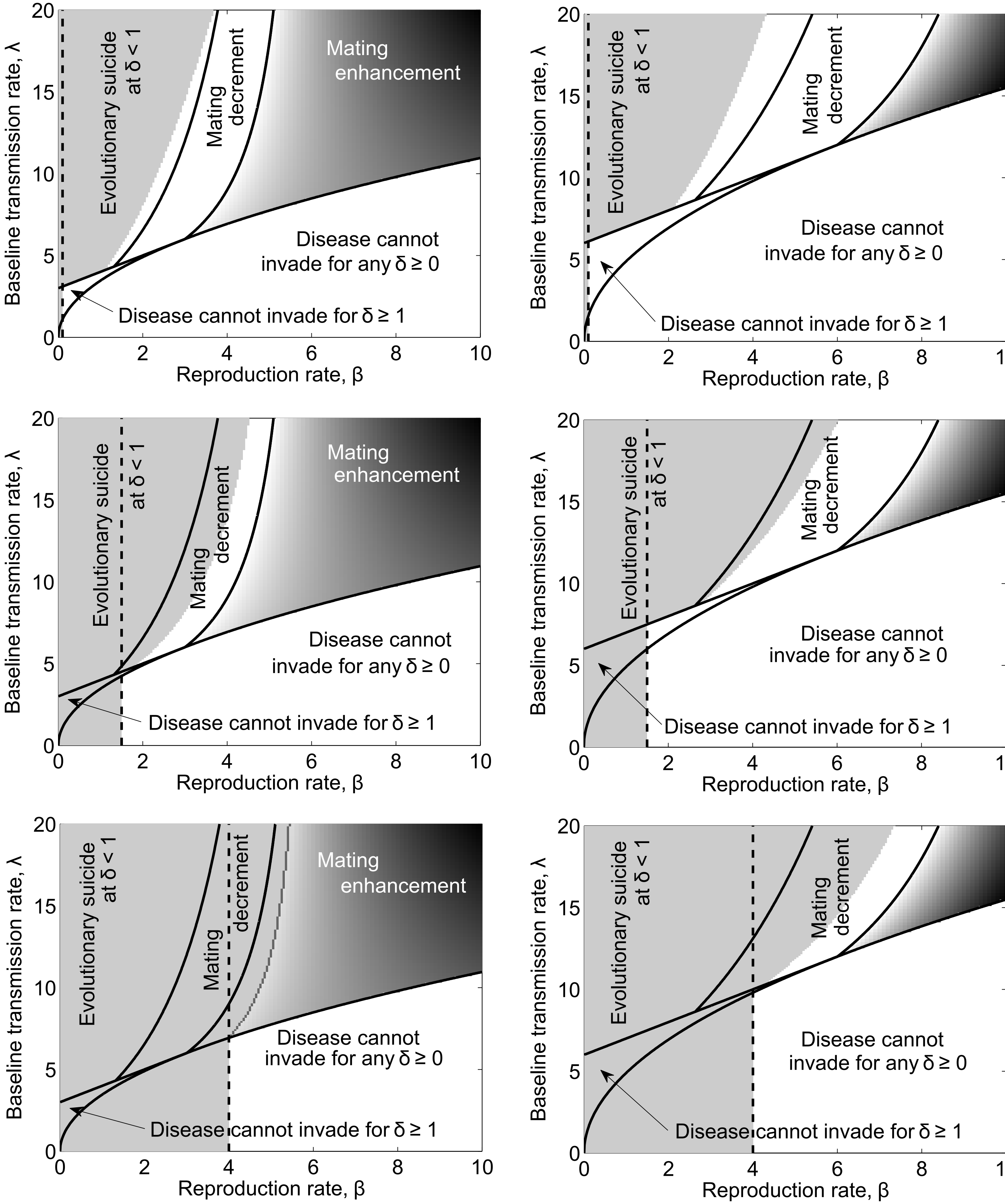

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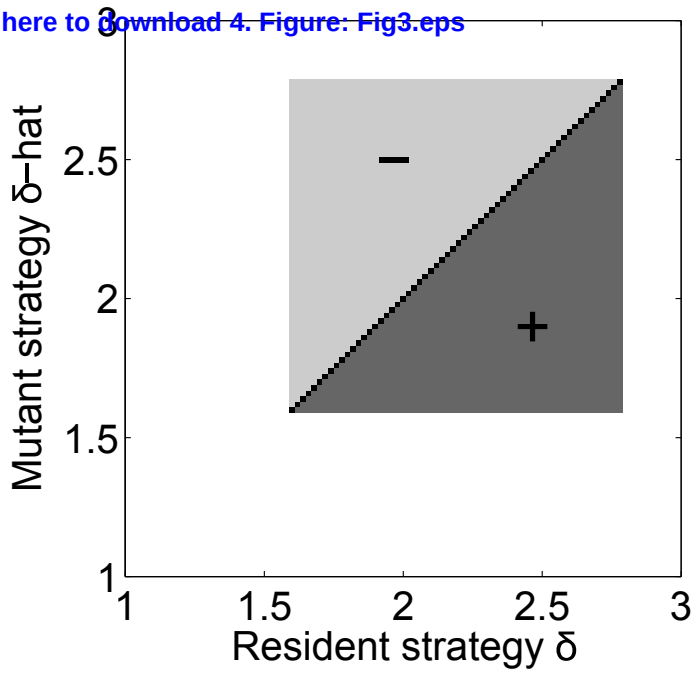

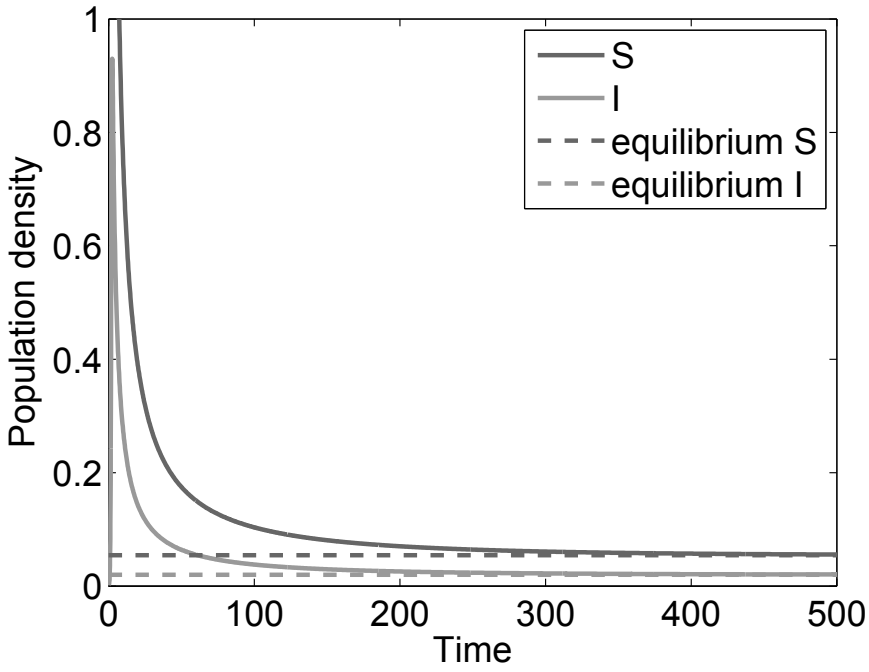

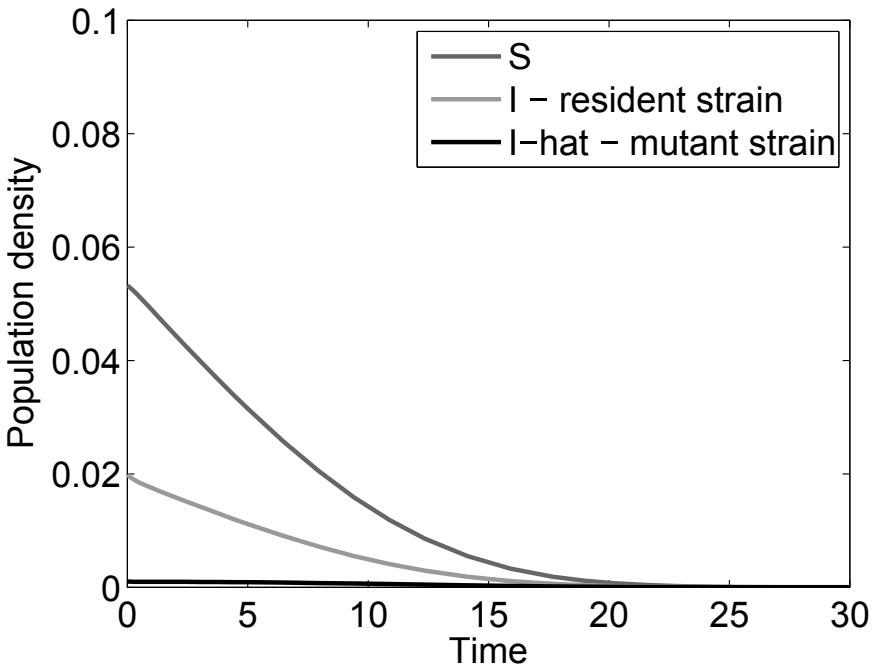

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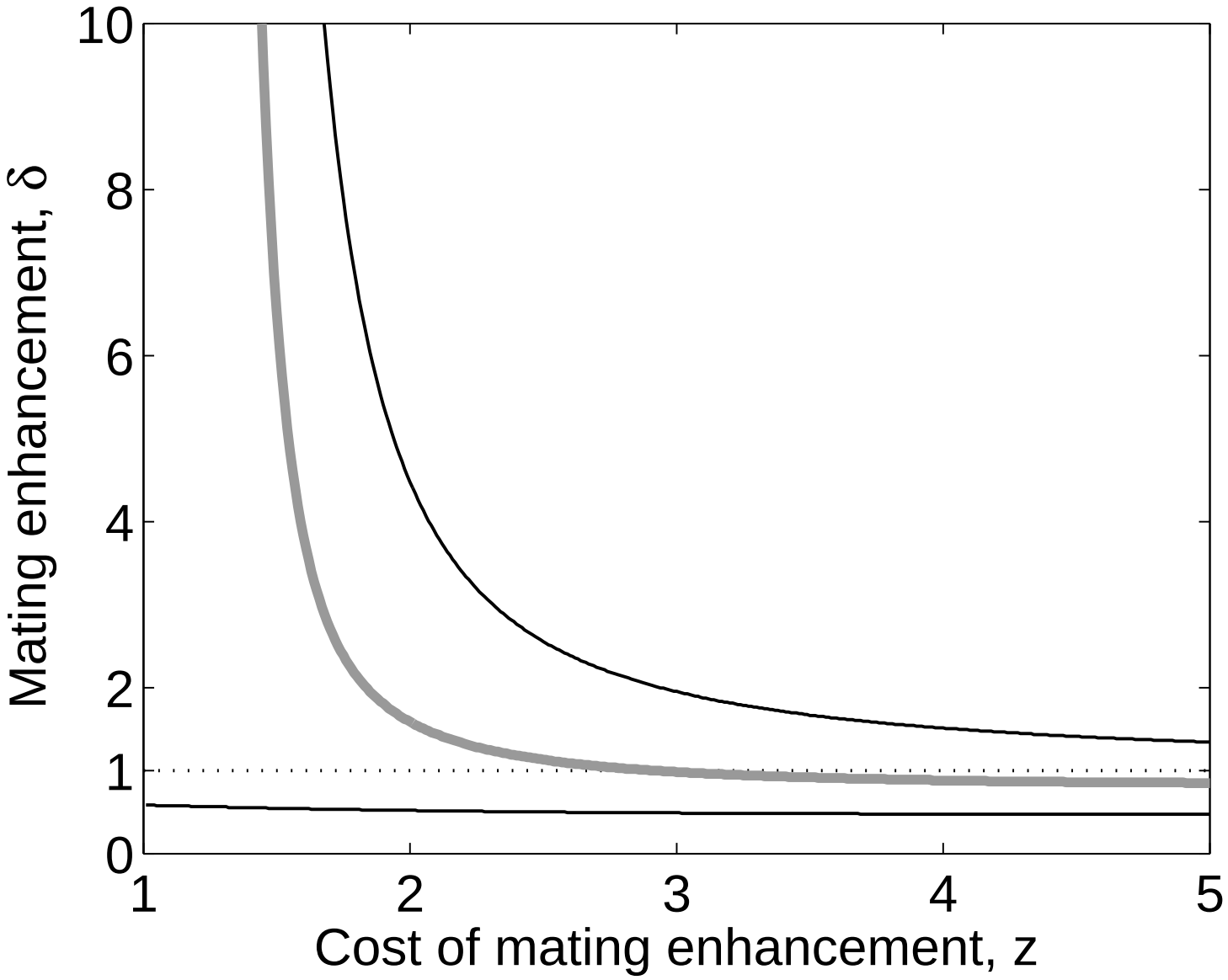

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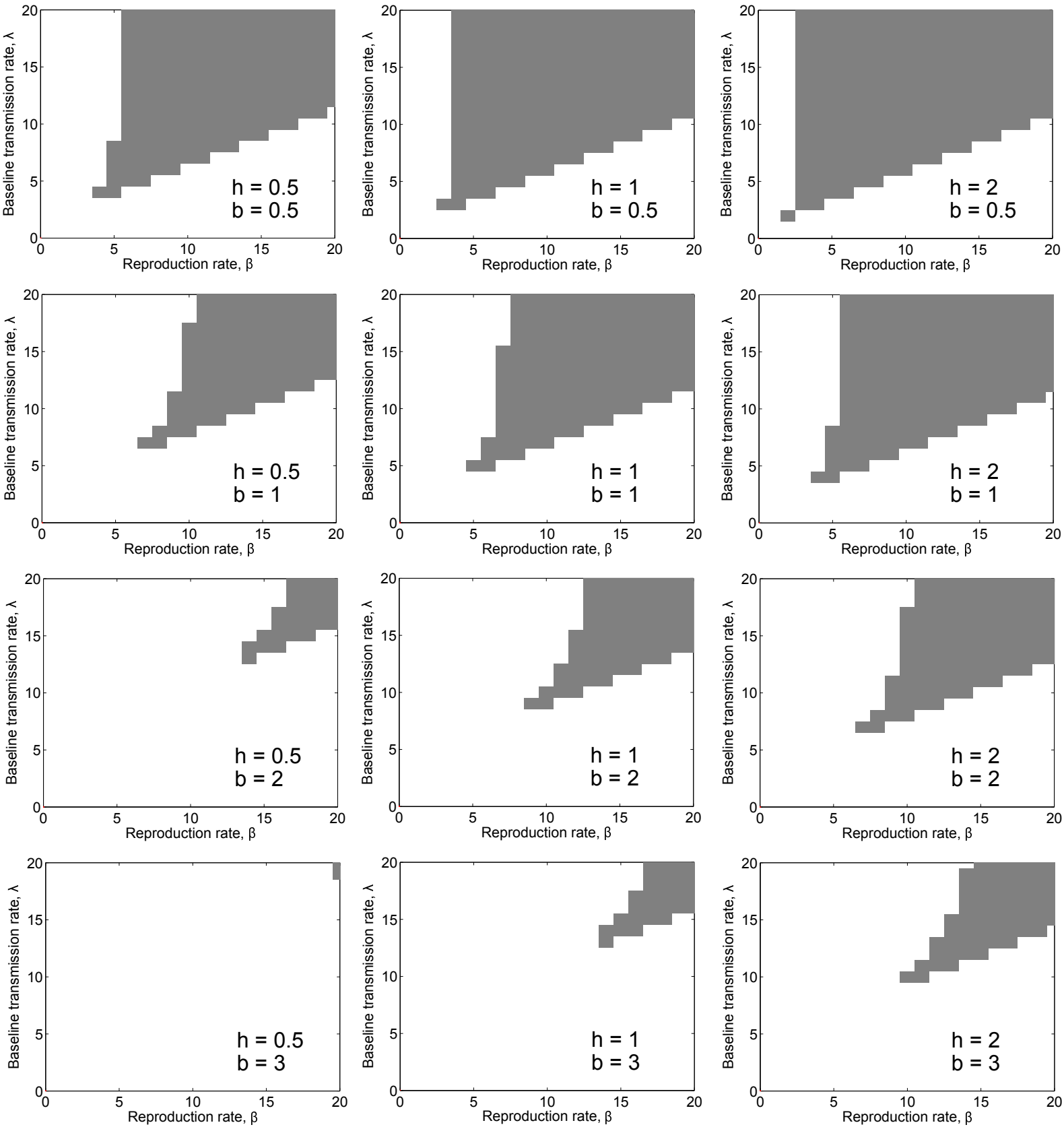

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