Journal of Theoretical Biology 342 (2014) 47–61

Contents lists available at ScienceDirect

Journal of Theoretical Biology

journal homepage: www.elsevier.com/locate/yjtbi

Why have parasites promoting mating success been observed so rarely?

L. Berec [a,b,*], D. Maxin [c]

[a] Department of Biosystematics and Ecology, Institute of Entomology, Biology Centre ASCR, Branišovská 31, 37005 České Budějovice, Czech Republic
[b] Institute of Mathematics and Biomathematics, Faculty of Science, University of South Bohemia, Branišovská 31, 37005 České Budějovice, Czech Republic
[c] Department of Mathematics and Computer Science, Valparaiso University, 1900 Chapel Drive, Valparaiso, IN 46383, USA

HIGHLIGHTS

- Although plausible, disease-induced mating enhancement has rarely been observed.
- We explore conditions under which this phenomenon is (or is not) likely to occur.
- Trade-off between parasite virulence and transmission limits its occurrence.
- Enhancement is less likely with decreasing host reproduction or parasite transmission.
- If acting in just one sex, chance for enhancement increases with host polygyny.

ARTICLE INFO

Article history:
Received 3 October 2012
Received in revised form
15 October 2013
Accepted 19 October 2013
Available online 30 October 2013

Keywords:
Population dynamics
Two-sex population model
Sexually transmitted disease
Host manipulation
Evolution

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, is 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 on evolutionary outcomes. On the contrary, if disease-induced mating enhancement is acting in just one sex, the potential for its evolution increases with the degree of polygyny in the host population. 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.

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

* Corresponding author at: Department of Biosystematics and Ecology, Institute of Entomology, Biology Centre ASCR, Branišovská 31, 37005 České Budějovice, Czech Republic. Tel.: +420 387 775 293.
E-mail addresses: berec@entu.cas.cz (L. Berec), daniel.maxin@valpo.edu (D. Maxin).
URL: http://www.entu.cas.cz/berec/ (L. Berec).

0022-5193/$ - see front matter © 2013 Elsevier Ltd. All rights reserved.
http://dx.doi.org/10.1016/j.jtbi.2013.10.012

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:

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

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: $\overline{\mu}_f=\mu_f+wP$ and $\overline{\mu}_m=\mu_m+wP$, 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 population grows logistically, and is a common assumption in epidemiological models with populations of varying size (e.g. Pugliese, 1990; Altizer and Augustine, 1997).

Table 1
Parameters used in the model (1).

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)

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. 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:

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

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

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

$$\frac{dS}{dt}=\frac{b}{2}\mathcal{M}(1,1)\left(\frac{S^2}{P}+2\delta\frac{SI}{P}+\delta_{fm}\frac{I^2}{P}\right)-\overline{\mu}S-\xi\delta\mathcal{M}(1,1)\frac{SI}{P}$$