



# Sexually transmitted infections and mate-finding Allee effects



Luděk Berec<sup>a,b,\*</sup>, Eva Janoušková<sup>c</sup>, Michal Theuer<sup>c</sup>

<sup>a</sup> Department of Ecology, Institute of Entomology, Biology Centre CAS, Branišovská 31, 37005 České Budějovice, Czech Republic

<sup>b</sup> Institute of Mathematics and Biomathematics, Faculty of Science, University of South Bohemia, Branišovská 1760, 37005 České Budějovice, Czech Republic

<sup>c</sup> Department of Mathematics and Statistics, Faculty of Science, Masaryk University, Kotlářská 2, 61137 Brno, Czech Republic

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## ABSTRACT

Infectious diseases can seriously impact dynamics of their host species. In this study, we model and analyze an interaction between a sexually transmitted infection and its animal host population affected by a mate-finding Allee effect. Since mating drives both host reproduction and infection transmission, the Allee effect shapes the transmission rate of the infection which we show takes a saturating form. Our model combining sexually transmitted infections with the mate-finding Allee effect in the host produces quite rich dynamics, including oscillations, several multistability regimes, and infection-induced host extinction. However, many of these complex patterns are restricted to a relatively narrow parameter range. We find that the host extinction occurs at intermediate levels of infection virulence, as well as for Allee effect strengths much lower than when the infection is absent. In both cases, a sequence of events comprising destabilization of an endemic equilibrium, growth of oscillation amplitude, and a heteroclinic bifurcation forms an underlying mechanism. We apply our model to the feline immunodeficiency virus (FIV) in domestic cats.

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## 1. Introduction

Infections can be important regulators of host species dynamics. Indeed, it has been repeatedly demonstrated that they can cause a significant drop in abundance of their host populations. Examples include impacts of amphibian chytridiomycosis and Tasmanian devil facial tumor disease (McCallum, 2012), the Ebola virus in gorillas, chimpanzees, and duikers (Leroy, 2004), and rabies in the African wild dog (Woodroffe and Ginsberg, 1999). In some cases, infections may even induce extinction of their host species, as for instance was documented for a land snail (de Castro and Bolker, 2005).

Significant insight into the effects of infectious diseases on host population dynamics has been gained by means of mathematical models (Anderson and May, 1991; Keeling and Rohani, 2008; Vynnycky and White, 2010). Indeed, for both exponentially and logistically growing host populations, the effects of infections

transmitted in both the density-dependent way and the frequency-dependent way are well known (Pugliese, 1990; Mena-Lorca and Hethcote, 1992; Antonovics et al., 2011). In particular, infections with density-dependent transmission require a minimum host density for successful invasion, to stabilize exponentially growing populations, and to reduce equilibrium host abundance in logistically growing populations. On the other hand, due to the density-independent host contact rate, infections with frequency-dependent transmission can invade populations of any size or density and even has the potential to make them extinct.

Mathematical models of infectious disease dynamics have also taught us a lot about a need to prevent populations of endangered species from further deterioration (McCallum and Dobson, 1995; McCallum et al., 2009), as well as about a possibility to use pathogens as potential control agents (Barlow and Kean, 1998; Deredec et al., 2008; Berec and Maxin, 2012, 2013). For example, one of the most commonly used microbial agents today, the bacterium *Bacillus thuringiensis*, has been successful in suppressing populations of the gypsy moth *Lymantria dispar* and many other non-native insect pests (Lacey et al., 2001). In Australia, myxomatosis (in the 1950s) and rabbit hemorrhagic disease (in the 1990s) were effectively used in control of the European rabbit *Oryctolagus cuniculus* (Saunders et al., 2010).

\* Corresponding author at: Department of Ecology, Institute of Entomology, Biology Centre CAS, Branišovská 31, 37005 České Budějovice, Czech Republic. Fax: +420 385310354.

E-mail addresses: [berec@entu.cas.cz](mailto:berec@entu.cas.cz) (L. Berec), [janouskova.eva@mail.muni.cz](mailto:janouskova.eva@mail.muni.cz) (E. Janoušková), [mithe@mail.muni.cz](mailto:mithe@mail.muni.cz) (M. Theuer).

In the absence of infection, host populations may demonstrate dynamics more complex than an exponential or logistic growth. In particular, they may be subject to strong Allee effects whereby low-density populations have negative growth rates and tend to go extinct (Berec et al., 2007; Courchamp et al., 2008). Saying that host populations affected by strong Allee effects are particularly threatened by lethal and/or sterilizing infections then comes as no surprise (Dereced and Courchamp, 2003, 2006). Existing models with Allee effects and density-dependent infection transmission produce surprisingly rich dynamics, including sustained oscillations and multiple (stable) endemic steady states (Hilker et al., 2009; Thieme et al., 2009; Friedman and Yakubu, 2012). These dynamics are absent in models with Allee effects and frequency-dependent infection transmission, but such an infection can induce host extinction even if the basic reproduction number  $R_0$  is less than 1 provided that its initial prevalence is sufficiently high (Hilker, 2010; Berec and Maxin, 2013).

Exploration of joint interplay of infectious diseases and strong Allee effects in the host population is not new (Dereced and Courchamp, 2006; Hilker et al., 2009; Thieme et al., 2009; Hilker, 2010; Friedman and Yakubu, 2012). Previous models generally did not specify the mechanism behind the Allee effect in detail, i.e. whether it is due to inbreeding depression, reduced foraging efficiency, or failure to attract or find mates and saturate or repel natural enemies in small or sparse populations (Berec et al., 2007; Courchamp et al., 2008; Tobin et al., 2011), nor they assumed any specific type of disease. This is fully justified if they study a pathogen transmission of which is independent of the Allee effect mechanism, such as when an airborne infection combines with a mate-finding Allee effect in the host. However, for a sexually transmitted infection spreading through a host population subject to the mate-finding Allee effect, such independence cannot be assumed and a more detailed model is required that takes mating dynamics into account. Indeed, mating here mediates both host reproduction and sexual transmission of the infection. Here we develop and study a simple epidemiological model with the mate-finding Allee effect in the host and a sexually transmitted infection, assuming a structural consistency between the processes of host reproduction and infection transmission, mediated by mating (Berec and Maxin, 2013). We find that this consistency assumption implies the infection transmission form often termed asymptotic, saturating or mixed (density-dependent at low densities and frequency-dependent at high densities; McCallum et al., 2001). We complement existing knowledge on the joint interplay of strong Allee effects and infectious diseases on host population dynamics, and apply our model to the population of domestic cats infected by the feline immunodeficiency virus (FIV), a sexually transmitted virus homologous to human HIV (Courchamp et al., 1995).

## 2. The model

We consider a sexually transmitted infection invading a host species for which mating and reproduction are tightly coupled. Mating thus mediates both host reproduction and infection transmission. Letting  $N_M$  and  $N_F$  represent male and female population density, respectively, we denote by  $\mathcal{M}(N_M, N_F)$  the rate at which males and females mate (the number of matings that occur per unit time). Assuming further a 1:1 sex ratio at birth and sex-independent vital rates,  $N_M = N_F = N/2$  where  $N$  is the total host density and  $\mathcal{M}(N_M, N_F) = \mathcal{M}(N/2, N/2)$ . Finally, denoting by  $S$  and  $I$  density of susceptible and infected hosts, respectively, we show in Appendix A that an appropriate model of the host–infection dynamics is in this case (see also Berec and

Maxin, 2013):

$$\frac{dS}{dt} = \gamma w \mathcal{M}\left(\frac{N}{2}, \frac{N}{2}\right) \frac{(S + (1 - \sigma)I)^2}{N^2} - 2\xi \mathcal{M}\left(\frac{N}{2}, \frac{N}{2}\right) \frac{SI}{N^2} - (\mu + bN)S, \quad (1)$$

$$\frac{dI}{dt} = 2\xi \mathcal{M}\left(\frac{N}{2}, \frac{N}{2}\right) \frac{SI}{N^2} - (\mu + bN)I - \alpha I.$$

In this model,  $\gamma$  is the number of newborns per reproductive event,  $w$  is the proportion of matings that result in reproduction,  $\sigma$  is the probability that an infected individual becomes sterilized,  $\xi$  is the probability of infection transmission upon mating between a susceptible and an infected individual,  $\mu$  is the background mortality rate in the absence of infection which can be enhanced by a crowding factor  $b$ , and  $\alpha$  is the infection-induced mortality rate. We discuss model parameters introduced both here and also below in more detail and primarily from the biological perspective in Appendix B.

Berec and Maxin (2013) assumed the mating function  $\mathcal{M}(N_M, N_F)$  to be degree-one homogeneous. Whereas this type of mating function is widely used (Lindström and Kokko, 1998; Iannelli et al., 2005; Rankin and Kokko, 2007; Miller and Inouye, 2011), it is not without drawbacks. In particular,  $\mathcal{M}(N/2, N/2) = (N/2)\mathcal{M}(1, 1)$  for any degree-one homogeneous mating function and there is thus a linear relationship between the mating rate and population density. As a consequence, the per female mating rate  $\mathcal{M}(N/2, N/2)/(N/2)$  stays constant anyhow low the male density  $N/2$  is. This could be an issue at low population densities where females may have an enhanced difficulty in finding mates, a phenomenon commonly referred to as the mate-finding Allee effect (Gascoigne et al., 2009; Kramer et al., 2009; Fauvergue, 2013).

To account for the mate-finding Allee effect, we use an alternative mating function

$$\mathcal{M}(N_M, N_F) = c \frac{N_F N_M}{N_M + \vartheta}, \quad (2)$$

in which  $c$  is a constant of proportionality (with units of per unit time) and  $\vartheta$  is the Allee effect strength (with units of density) (Courchamp et al., 2008). For  $N_M = N_F = N/2$ , the mating function (2) becomes

$$\mathcal{M}\left(\frac{N}{2}, \frac{N}{2}\right) = \frac{c N^2}{2(N + 2\vartheta)}. \quad (3)$$

Indeed, for the mating function (3) the per female mating rate decreases as the population density  $N$  declines. Substituting the Allee effect mating function (3) into the population model (1), we have

$$\frac{dS}{dt} = \gamma w c \frac{(S + (1 - \sigma)I)^2}{2(N + 2\vartheta)} - \xi c \frac{SI}{N + 2\vartheta} - (\mu + bN)S, \quad (4)$$

$$\frac{dI}{dt} = \xi c \frac{SI}{N + 2\vartheta} - (\mu + bN)I - \alpha I.$$

Finally, denoting  $\beta = \gamma w c / 2$ ,  $\lambda = \xi c$  and  $\theta = 2\vartheta$ , the model (4) becomes

$$\frac{dS}{dt} = \beta \frac{(S + (1 - \sigma)I)^2}{N + \theta} - \lambda \frac{SI}{N + \theta} - (\mu + bN)S, \quad (5)$$

$$\frac{dI}{dt} = \lambda \frac{SI}{N + \theta} - (\mu + bN)I - \alpha I.$$

It is the model (5) that we are going to analyze in this article. We note that the resulting infection transmission term  $\lambda SI / (N + \theta)$  represents an asymptotic transmission term used in other modeling studies on infectious disease dynamics (McCallum et al., 2001; Dereced and Courchamp, 2006; Breban et al., 2010). On the contrary, any degree-one homogeneous mating function gives rise to a frequency-dependent transmission term  $\lambda SI / N$  (Berec and Maxin, 2013).

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