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Is more better? Higher sterilization of infected hosts need not result in reduced pest population size

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Abstract We analyze the effect of sterilization in the infected hosts in several epidemiological models involving infectious diseases that can be transmitted both vertically and horizontally. Sterilizing pathogens can be used as pest control agents by intentionally inoculating the target population, with the goal of reducing or eliminating it completely. Contrary to previous models that did not include vertical transmission we found that the population size at the endemic equilibrium may actually increase with higher levels of sterility. This effect is proved to exist for low to high efficiencies of vertical transmission. On the other hand, if the disease is sexually transmitted and the host reproduction and disease transmission are both consistently mediated by mating,

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we do not observe such a counter-intuitive effect and the population size in the stable endemic equilibrium is decreasing with higher levels of sterility. We suggest that models of the pest control techniques involving the release of sterilizing pathogens have to carefully consider the routes such pathogens use for transmission.

Keywords vertical transmission · pest control · sterilization

1 Introduction

Invasions of non-native species are a global and increasing threat to the function and diversity of ecosystems (Vitousek et al, 1997; Lockwood et al, 2007). Among these, small mammals such as foxes, rabbits and mice have been repeatedly reported to be more damaging than any other vertebrate group (Ebenhard, 1988; Courchamp et al, 2003). Whereas more traditional control methods such as hunting, trapping and baiting have often been found inefficient (Deredec et al, 2008), many disseminating pathogens enhancing pest mortality have been found inhumane (Tyndale-Biscoe, 1994).

An alternative to these methods is an introduction of a sterilizing pathogen. Virus-vectored immunocontraception (VVIC) has been proposed as a viable technique for controlling small mammals (Hardy et al, 2006). VVIC assumes a genetically engineered, species-specific virus that is intended to trigger an autoimmune response whereby antibodies are produced against the species' gametes and fertilization is thus blocked (Tyndale-Biscoe, 1994; Hardy et al, 2006). As an example, the myxoma virus and the murine cytomegalovirus have been suggested as potential immunocontraceptives for rabbits and mice, respectively (Hardy et al, 2006).

The introduction of additional transmission pathways may also increase the effectiveness of the sterilizing pathogens. Altizer and Augustine (1997) showed that vertical, in addition to horizontal, transmission widened the range of parameters values for which the sterilizing pathogen could successfully control the pest population. The efficiency of vertical transmission, i.e. the ability of an infectious disease to be passed from mother to a newborn, varies depending on the infectious agent and/or the possibility of treatment. It can be as low as 1–4% in the transmission of dengue virus in mosquitoes (Adams and Boots, 2010), but as high as ∼90%, and even complete, in the transmission of *Orientia tsutsugamushi* in mites *Leptotrombidium deliense* (Frances et al, 2001).

Intuition suggests that as the degree of sterilization increases, the host population size decreases. Therefore, when the host is at the same time a pest, increasing sterility should at the same time improve our control efficiency (quantified as the proportional decrease in equilibrium host size with respect to its disease-free stable state). Many modeling studies on diseases without vertical transmission support this view. For example, in their model studying impacts of VVIC for pest reduction, Deredec et al (2008) found that an increase in sterilization efficiency invariably caused the pest population to decrease. In addition, they showed that virus choice should focus more on its sterilizing

power rather than transmission efficiency. Berec and Maxin (2012) studied the added value of increased life expectancy due to disease-induced sterilization on pest control effectiveness, finding again that an increase in sterilization efficiency brought about lowered equilibrium population size.

In this paper, we study a series of infectious disease models with vertical transmission and several different types of horizontal transmission (mass action incidence, asymptotic incidence and standard incidence). Our purpose is two-fold. First, we wish to analyze how the introduction of vertical transmission may change the pest control efficiency relative to a model in which this transmission route is absent. Second, we wish to see how these results change in the case of a sexually transmitted disease when we consider structural consistency between the processes of host reproduction and disease transmission, i.e. when we assume that both are mediated by mating and modeled by the same functional form (Berec and Maxin, 2013). Accounting for such consistency has already proved to change the results of conventional epidemiological models under some conditions (Berec and Maxin, 2013). In particular, we show that without the consistency assumption and contrary to intuition, under certain conditions, increasing the pathogen's ability to sterilize the host is not necessarily beneficial from the pest control perspective because it tends to increase the population size at equilibrium.

The paper is structured as follows. In the next section, we formulate and analyze a conventional epidemiological model with logistic host growth in the absence of infection, vertical and horizontal disease transmission, disease-induced mortality and disease-reduced reproduction. As the horizontal incidence term, we consider in sequence mass action incidence, asymptotic incidence and standard incidence. Then, we formulate and analyze a two-sex epidemiological model with both vertical and sexual transmission in which we assume that the host reproduction process and the (sexual) disease transmission process are both mediated by mating, a feature to be expected in many realistic cases. We use two types of mating function, a degree-one homogeneous mating function which results in a standard-incidence-like disease transmission term and a mating function accounting for mate-finding Allee effects which results in an asymptotic-incidence-like disease transmission term. In all models we focus on how endemic equilibria vary with the disease sterilization efficiency, and discuss the results from the perspective of using sterilizing infectious diseases as potential pest control agents.

2 The classic one-sex model

In this section we consider the following model with both horizontal and vertical transmission:

$$\begin{aligned} \tfrac{dS}{dt} &= \beta(S+\xi\sigma I) - \Phi(N)\tfrac{SI}{N} - \bar{\mu}S, \\ \tfrac{dI}{dt} &= \beta(1-\xi)\sigma I + \Phi(N)\tfrac{SI}{N} - \bar{\mu}I - \alpha I. \end{aligned} \tag{1}$$

In this model, $N = S+I$ is the total population size, $\bar{\mu} = \mu+bN$ is the density-dependent natural mortality rate and $\Phi(N)$ is a general disease transmission term. In particular, $\Phi(N)$ can be λ (standard incidence), λN (mass action incidence) or $\frac{\lambda N}{c+N}$ (asymptotic incidence). Moreover, β is the host birth rate and ξ is the probability that an offspring of an infected host is susceptible. Therefore $1-\xi$ is the efficiency of vertical transmission, i.e. the proportion of infected newborns from an infected parent. Finally, σ is the disease sterilization efficiency, i.e. the reduction in fertility of infected individuals and α is the additional mortality rate caused by the disease. Thus, $0 \leq \xi \leq 1$ and $0 \leq \sigma \leq 1$. We also assume $\beta > \mu$ which ensures that the population does not go extinct in the absence of the disease. The model (1) was analyzed by Pugliese (1990), and we summarize its main results below.

For the time being we assume that $\Phi(N)$ is a (continuously) differentiable function with respect to N. Moreover, we assume this function is increasing ($\Phi'(N) > 0$). This effectively rules out standard incidence which is analyzed in more detail at the end of this section and in Appendix A. Let us denote

$$K := \frac{\beta-\mu}{b} \text{ and } K_i := \frac{\beta\sigma-\mu-\alpha}{b}.$$

We can think of K as the carrying capacity of the host population in the absence of infection. The parameter K_i represents the carrying capacity of the infected population if there were no susceptible individuals; this is possible only in the case of full vertical transmission ($\xi = 0$). With this notation, the basic reproduction number of the infection is (noting that $\beta = \mu + bK$)

$$R_0 := \frac{\Phi(K) + \beta(1-\xi)\sigma}{\mu + bK + \alpha} = \frac{\Phi(K) + \beta(1-\xi)\sigma}{\beta + \alpha}. \tag{2}$$

The model (1) may have four equilibrium points:

$$(S^o, I^o) = (0,0), \quad (\bar{S}, \bar{I}) = (K, 0), \quad (\hat{S}, \hat{I}) = (0, K_i),$$

$$(S^*, I^*) = \left(\frac{N^*(\mu + bN^* + \alpha - \beta\sigma)}{\beta(1-\sigma)+\alpha}, \frac{N(\beta - \mu - bN^*)}{\beta(1-\sigma)+\alpha} \right),$$

with N^* satisfying

$$\Phi(N^*) = \frac{[\beta(1-\sigma)+\alpha][\beta(1-\xi)\sigma - \mu - bN^* - \alpha]}{\beta\sigma - \mu - bN^* - \alpha}. \tag{3}$$

We note that the equilibrium $(\hat{S}, \hat{I}) = (0, K_i)$ exists only in the case of full vertical transmission ($\xi = 0$), i.e. when all newborns from the infected parent are infected. For the endemic equilibrium (S^*, I^*) to be feasible we require that

$$\beta\sigma - \alpha < \mu + bN^* < \beta.$$

From Pugliese (1990) the stability results are:

1. The case of imperfect vertical transmission $0 < \xi < 1$:

(a) If $R_0 < 1$ or $\Phi(K) < \beta[1-(1-\xi)\sigma]+\alpha$ then $(\bar{S},\bar{I})$ is globally stable.
(b) If $R_0 > 1$ or $\Phi(K) > \beta[1-(1-\xi)\sigma]+\alpha$ then (S^*,I^*) is globally stable.

2. The case of full vertical transmission $\xi = 0$:
(a) If $R_0 < 1$ or $\Phi(K) < \beta(1-\sigma)+\alpha$ then $(\bar{S},\bar{I})$ is globally stable.
(b) If $R_0 > 1$ and $\Phi(K_i) < \beta(1-\sigma)+\alpha < \Phi(K)$ then (S^*,I^*) is globally stable.
(c) If $R_0 > 1$ and $\Phi(K_i) > \beta(1-\sigma)+\alpha$ then $(\hat{S},\hat{I})$ is globally stable.

The main issue we consider in this section is how the total population size at an endemic equilibrium responds to the increasing level of sterility (i.e. decreasing σ). This has practical implications for population control since one of the techniques used to control unwanted pest populations consists of releasing a sterilizing pathogen into it, with the aim to suppress host reproduction and hence its total population size.

Differentiating the equation (3) with respect to σ we obtain

$$\frac{dN^*}{d\sigma} = \frac{\beta^3\sigma^2(\xi-1)+\beta^2(\mu+bN^*+\alpha)(\xi+2\sigma(1-\xi))-\beta(\mu+bN^*+\alpha(1-\xi))(\mu+bN^*+\alpha)}{\Phi'(N^*)[\mu+bN^*+\alpha-\beta\sigma]^2+b\beta\xi\sigma(\alpha+\beta-\beta\sigma)}.$$

For the full vertical transmission ($\xi = 0$), this derivative is

$$\left(\frac{dN^*}{d\sigma}\right)_{\xi=0} = -\frac{\beta}{\Phi'(N^*)} < 0.$$

This implies that, at the endemic equilibrium, the total population size actually increases with the increasing level of sterility (i.e. decreasing σ). On the other hand, for no vertical transmission ($\xi = 1$), we have

$$\left(\frac{dN^*}{d\sigma}\right)_{\xi=1} = \frac{\beta(\mu+bN^*+\alpha)(\beta-\mu-bN^*)}{\Phi'(N^*)[\mu+bN^*+\alpha-\beta\sigma]^2+b\beta\sigma[\alpha+\beta(1-\sigma)]} > 0.$$

That is, in the absence of vertical transmission, the population at the endemic equilibrium will always decrease with the increasing level of sterility (i.e. decreasing σ).

Due to continuity, there are certain values of ξ for which $dN^*/d\sigma$ has at least one critical point. By this we mean that N^* as a function of σ will decrease until a certain threshold after which it will increase. In Fig. 1 we show a contour plot of N^* as a function of σ and ξ for the mass action incidence ($\Phi(N) = \lambda N$) and the other parameters fixed at some values. Notice that if ξ is greater than a certain threshold (i.e. not enough vertical transmission) then N^* will be increasing in σ whereas if ξ is below that threshold then N^* will first decrease and then increase. Put differently, increasing the level of sterility beyond a certain value (i.e. lowering σ) no longer reduces the population size, but rather worsens the control effectiveness, defined as $E = 1 - N^*/K$.

An analogous example for the asymptotic incidence ($\Phi(N) = \lambda N/(c+N)$) is given in Fig. 2. Here we need to distinguish two cases. First, when $\lambda > \alpha+\beta$ the endemic equilibrium (S^*,I^*) exists and is globally stable for all $0 \le \xi \le 1$ and $0 \le \sigma \le 1$ and we observe a pattern similar to that in Fig. 1: a critical

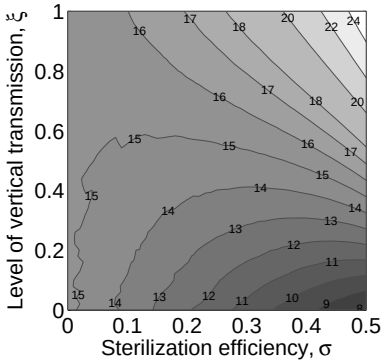

Fig. 1: The effect of changing the sterilization efficiency σ and the level of vertical transmission ξ in the model (1) on the total population size at the endemic equilibrium. Disease transmission occurs through mass action incidence. Parameter values: $\beta = 1.5$, $\mu = 1$, $b = 0.002$, $\lambda = 0.1$, $\alpha = 0.02$. In the contour plots, the lighter the color is the higher the value of N^* is.

value of ξ exists such that below it the control efficiency initially increases with the increasing level of sterility, but later on this trend chops around and the control efficiency decreases with the increasing level of sterility. Second, when $\lambda < \alpha + \beta$ the endemic equilibrium does not exist for high ξ and low σ where the disease-free equilibrium $(K, 0)$ is instead globally stable. Here the concave curve at which the endemic equilibrium ceases to exist forces endemic equilibrium isolines to also be concave. Hence, we again observe the control efficiency to decrease (or the endemic equilibrium to increase) with the increasing level of sterility.

In the case of standard incidence ($\Phi(N) = \lambda$) we can carry out the full analysis of the monotonicity of N^* with respect to σ for any intermediate value of vertical transmission efficiency ξ. Denoting

$$\bar{\sigma} := \frac{(\alpha+\beta-\lambda)(1-\xi) + \sqrt{\xi\lambda(1-\xi)(\alpha+\beta-\lambda)}}{\beta(1-\xi)} \text{ and}$$

$$\sigma_R := \frac{\alpha+\beta-\lambda}{\beta(1-\xi)},$$

we provide the main result in the theorem below while the details of its proof are given in Appendix A.

Theorem 1 *Assuming the intrinsic mortality rate μ is low enough to prevent disease-induced population extinction, the population size at an endemic equilibrium N^* varies with the sterilization efficiency σ as follows:*

1. *If $\lambda > \alpha + \beta$ then $N^*(\sigma)$ is increasing on $(0, 1)$ and the disease is endemic for all σ and ξ;*
2. *If $\lambda < \alpha+\beta$ and $\max\left\{\frac{\alpha+\beta-\lambda}{\beta\sigma}, \frac{\lambda(\alpha+\beta-\lambda)}{\alpha^2+\lambda(\beta-\alpha)}\right\} < 1-\xi$ then $N^*(\sigma)$ is decreasing on $(\sigma_R, \bar{\sigma})$ and increasing on $(\bar{\sigma}, 1)$.*

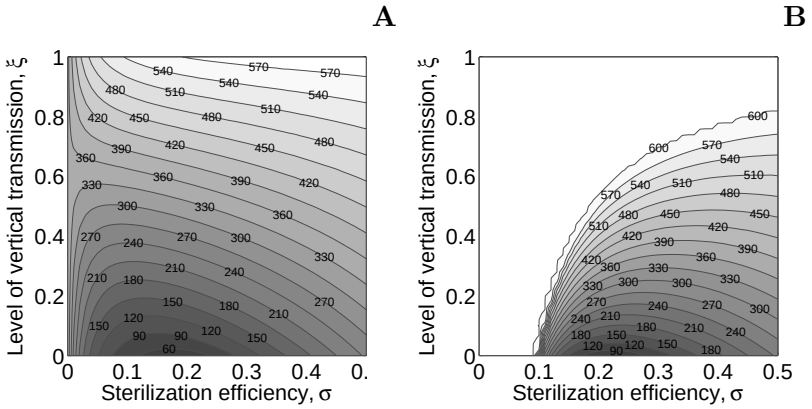

Fig. 2: The effect of changing the sterilization efficiency σ and the level of vertical transmission ξ in the model (1) on the total population size at the endemic equilibrium. Disease transmission occurs through asymptotic incidence. Parameter values: $\beta = 1.4$, $\mu = 0.2$, $b = 0.002$, $\alpha = 0.01$, $c = 10$; we set $\lambda = 1.5$ in panel A, which corresponds to $\lambda > \alpha + \beta$, and $\lambda = 1.3$ in panel B, which corresponds to $\lambda < \alpha + \beta$. In the contour plots, the lighter the color is the higher the value of N^* is. The white area in panel B corresponds to parameter combinations where the disease-free equilibrium $(K, 0)$ is globally stable.

3. *If* $\lambda < \alpha + \beta$ *and* $\frac{\alpha+\beta-\lambda}{\beta\sigma} < 1 - \xi < \frac{\lambda(\alpha+\beta-\lambda)}{\alpha^2+\lambda(\beta-\alpha)}$ *then* $N^*(\sigma)$ *is decreasing on* $(\sigma_R, 1)$.

These cases are illustrated in Fig. 3. As with asymptotic incidence, we again distinguish two situations. Now, however, when the endemic equilibrium exists and is globally stable for all $0 \leq \xi \leq 1$ and $0 \leq \sigma \leq 1$ (which again occurs when $\lambda > \alpha + \beta$), the control efficiency always increases with the increasing level of sterility. On the other hand, when $\lambda < \alpha + \beta$, we observe the same pattern as with asymptotic incidence in Fig. 2B: the control efficiency initially increases and then decreases with the increasing level of sterility.

3 The one-sex model with consistency between host reproduction and disease transmission

We now consider a sterilizing, sexually transmitted disease where the host reproduction and disease transmission are both mediated by mating. Hence, the mating function (denoted as $\mathcal{M}(F, M)$) appears in both the reproduction

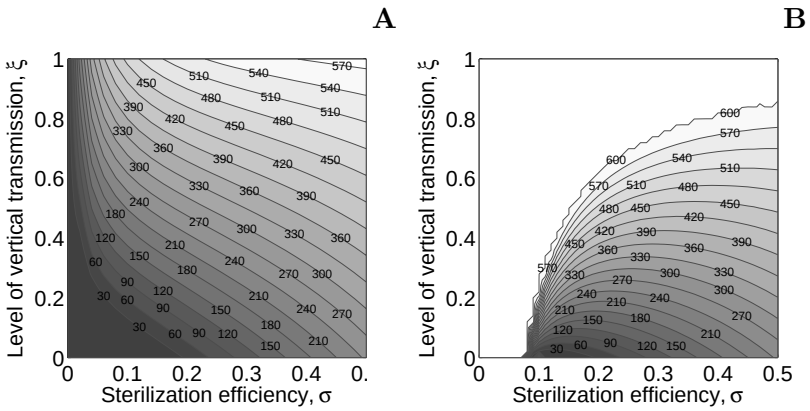

Fig. 3: The effect of changing the sterilization efficiency σ and the level of vertical transmission ξ in the model (1) on the total population size at the endemic equilibrium. Disease transmission occurs through standard incidence. Parameter values: $\beta = 1.4$, $\mu = 0.2$, $b = 0.002$, $\alpha = 0.01$; we set $\lambda = 1.5$ in panel A, which corresponds to $\lambda > \alpha + \beta$, and $\lambda = 1.3$ in panel B, which corresponds to $\lambda < \alpha + \beta$. In the contour plots, the lighter the color is the higher the value of N^* is. The white area in panel B corresponds to parameter combinations where the disease-free equilibrium $(K, 0)$ is globally stable. In both panels, the darkest grade of gray color corresponds to disease-induced population extinction.

and disease transmission terms of the epidemiological model:

$$\begin{aligned}
S_f' &= \tfrac{\beta\gamma_f \mathcal{M}(F,M)}{FM}[S_f S_m + \xi(\sigma_f I_f S_m + \sigma_m I_m S_f + \sigma_f\sigma_m I_f I_m)] - \lambda\tfrac{\mathcal{M}(F,M)}{FM} S_f I_m - \bar{\mu}_f S_f,\\
S_m' &= \tfrac{\beta\gamma_m \mathcal{M}(F,M)}{FM}[S_f S_m + \xi(\sigma_f I_f S_m + \sigma_m I_m S_f + \sigma_f\sigma_m I_f I_m)] - \lambda\tfrac{\mathcal{M}(F,M)}{FM} S_m I_f - \bar{\mu}_m S_m,\\
I_f' &= \tfrac{\beta\gamma_f \mathcal{M}(F,M)}{FM}(1-\xi)(\sigma_f I_f S_m + \sigma_m I_m S_f + \sigma_f\sigma_m I_f I_m) + \lambda\tfrac{\mathcal{M}(F,M)}{FM} S_f I_m - \bar{\mu}_f I_f - \alpha_f I_f,\\
I_m' &= \tfrac{\beta\gamma_m \mathcal{M}(F,M)}{FM}(1-\xi)(\sigma_f I_f S_m + \sigma_m I_m S_f + \sigma_f\sigma_m I_f I_m) + \lambda\tfrac{\mathcal{M}(F,M)}{FM} S_m I_f - \bar{\mu}_m I_m - \alpha_m I_m.
\end{aligned} \tag{4}$$

Here S_f, S_m are the female and male susceptible populations and I_f, I_m are their infectious counterparts. Furthermore, $F = S_f + I_f$, $M = S_m + I_m$, $N = F + M$, $\bar{\mu}_f = \mu_f + bN$ and $\bar{\mu}_m = \mu_m + bN$. In addition, ξ is the proportion of susceptible newborns from infected parents, σ_f and σ_m are the fertility reduction coefficients for infected females and males, respectively, and γ_f and γ_m are the probabilities of a newborn being a female or male ($\gamma_f + \gamma_m = 1$).

The structure of the model (4) arises as a consequence of considering rates at which different classes of females and males mate. Assuming random mating then for example the rate at which susceptible females and susceptible males mate is a product of the total rate at which females and males of any type mate, $\mathcal{M}(F, M)$, the probability that the female is susceptible, S_f/F, and the probability that the male is susceptible, S_m/M. And similarly for the three remaining possibilities.

A variety of mating functions has been proposed, most of which originating in the demographic literature where they are commonly referred to as marriage functions (Iannelli et al, 2005). Of these, most demographic and ecological two-sex models use mating functions that are degree-one homogeneous (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), assuming that

$$\mathcal{M}(\eta F, \eta M) = \eta \mathcal{M}(F, M), \text{ for any positive } F, M, \eta.$$

In our model, we use a harmonic mean mating function (a degree-one homogeneous function resulting in standard-incidence-like disease transmission) and a mating function accounting for mate-finding with Allee effects (not a degree-one homogeneous function resulting in asymptotic-incidence-like disease transmission).

This model is too complex to be analyzed in its generality. Analytical results are possible by making various simplifying assumptions and we provide these results in the subsections that follow. Also, we accompany these analytical results with numerical simulations that span the whole range of σ and ξ values. The main insight here is that for both mating functions we use the total population size at the endemic equilibrium N^* is no longer increasing with higher levels of sterility σ.

3.1 The one-sex model derived from the model (4) with a harmonic mean mating function

Consider the harmonic mean mating function

$$\mathcal{M}(F, M) = w \frac{FM}{F + M} \tag{5}$$

for a positive scaling constant w (Lindström and Kokko, 1998; Iannelli et al, 2005; Miller and Inouye, 2011, 2013). Assuming equal sex-related parameters we can reduce the model (4) to a planar system as follows. The assumptions $\mu_f = \mu_m := \mu$, $\alpha_f = \alpha_m := \alpha$ and $\gamma_f = \gamma_m = 1/2$ imply that

$$S_f = S_m := \frac{S}{2}, \; I_f = I_m := \frac{I}{2} \text{ and } F = M := \frac{N}{2}.$$

Thus, the model (4) becomes

$$\begin{aligned} S' &= \tfrac{\hat{\beta}}{N}[S^2 + \xi(2\sigma SI + \sigma^2 I^2)] - \tfrac{\hat{\lambda}}{N} SI - \bar{\mu} S, \\ I' &= \tfrac{\hat{\beta}}{N}(1 - \xi)(2\sigma SI + \sigma^2 I^2) + \tfrac{\hat{\lambda}}{N} SI - \bar{\mu} I - \alpha I. \end{aligned} \tag{6}$$

Here we also used the fact that $\mathcal{M}\left(\frac{N}{2}, \frac{N}{2}\right) = \frac{N}{2}\mathcal{M}(1, 1)$ for the harmonic mean mating function (5) and we denoted $\hat{\beta} := \frac{\beta}{2} w \mathcal{M}(1, 1)$ and $\hat{\lambda} := \lambda w \mathcal{M}(1, 1)$.

The basic reproduction number of the infection described by the model (6) can be derived from the condition $dI/dt > 0$ or

$$\hat{\lambda} > \alpha + \hat{\beta}[1 - 2\sigma(1-\xi)]. \tag{7}$$

Notice that this inequality is satisfied for any $\hat{\lambda} > 0$ provided that

$$\alpha + \hat{\beta}[1 - 2\sigma(1-\xi)] < 0 \text{ or } \sigma(1-\xi) > \frac{\alpha + \hat{\beta}}{2\hat{\beta}}.$$

Otherwise, if $\sigma(1-\xi) < (\alpha + \hat{\beta})/(2\hat{\beta})$ then

$$R_0 = \frac{\hat{\lambda}}{\alpha + \hat{\beta}[1 - 2\sigma(1-\xi)]}.$$

The model (6) rewritten into the proportion of susceptibles $x = S/N$ and the total population size N is

$$\begin{aligned} x' &= (1-x)G(x), \\ N' &= N\{\hat{\beta}[x + \sigma(1-x)]^2 - \bar{\mu} - \alpha(1-x)\} \end{aligned} \tag{8}$$

where

$$G(x) = \hat{\beta}(1-\sigma)^2 x^2 - [\hat{\beta}\sigma^2(1+\xi) - 2\sigma\hat{\beta}\xi + \hat{\lambda} - \alpha]x + \hat{\beta}\xi\sigma^2.$$

Since the first equation is closed in x we will analyze it first. In addition to the obvious equilibrium $x^* = 1$ any other equilibrium is a root of $G(x)$ in the feasible interval $(0, 1)$. The main result concerning the model (8) is given in the following theorem (the proof is given in Appendix B):

Theorem 2 *For the epidemiological model (8), the population size at an endemic equilibrium N^* decreases with the increasing level of sterility $\left(\frac{dN^*}{d\sigma} > 0\right)$ in the following particular cases:*

1. *Full vertical transmission ($\xi = 0$),*
2. *No vertical transmission ($\xi = 1$),*
3. *Any level of vertical transmission ξ assuming no disease-induced mortality ($\alpha = 0$) and equal birth and transmission rates ($\hat{\beta} = \hat{\lambda}$).*

Numerical simulations suggest that the same is true also for any $\xi > 0$ (Fig. 4). Note that although in panel B of Fig. 4 the curve delimiting the white area is also concave, compared with panels B in Figs 2 and 3 the control efficiency here never decreases with the increasing level of sterility.

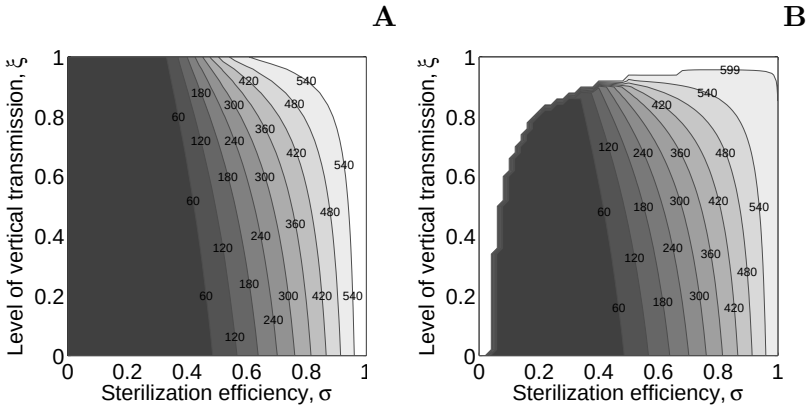

Fig. 4: The effect of changing the sterilization efficiency σ and the level of vertical transmission ξ in the model (6) on the total population size at the endemic equilibrium. Disease transmission occurs through a standard-incidence-like function. Parameter values: $\beta = 1.4$, $\mu = 0.2$, $b = 0.002$, $\alpha = 0.01$; we set $\lambda = 1.5$ in panel A, which corresponds to $\lambda > \alpha + \beta$, and $\lambda = 1.3$ in panel B, which corresponds to $\lambda < \alpha + \beta$. In the contour plots, the lighter the color is the higher the value of N^* is. The white area in panel B corresponds to parameter combinations where the disease-free equilibrium $(K, 0)$ is globally stable. In both panels, the darkest grade of gray color corresponds to disease-induced population extinction.

3.2 The one-sex model derived from the model (4) with a mating function accounting for mate-finding Allee effects

Consider now the mating function

$$\mathcal{M}(F, M) = w \frac{FM}{F + M + c}, \text{ where } w > 0,\ c > 0. \tag{9}$$

This function accounts for mate-finding Allee effects whereby individuals in low-density populations challenge reduced opportunities for finding a mate and hence for successful reproduction Boukal and Berec (2002); Courchamp et al (2008). Using the same assumptions on the parameters as in the previous subsection the one-sex reduced model is now as follows:

$$\begin{aligned} S' &= \beta \tfrac{\mathcal{M}(\frac{N}{2},\frac{N}{2})}{N^2}[S^2 + \xi(2\sigma SI + \sigma^2 I^2)] - (2\lambda)\tfrac{\mathcal{M}(\frac{N}{2},\frac{N}{2})}{N^2} SI - \bar{\mu} S, \\ I' &= \beta \tfrac{\mathcal{M}(\frac{N}{2},\frac{N}{2})}{N^2}(1-\xi)(2\sigma SI + \sigma^2 I^2) + (2\lambda)\tfrac{\mathcal{M}(\frac{N}{2},\frac{N}{2})}{N^2} SI - \bar{\mu} I - \alpha I. \end{aligned} \tag{10}$$

Also here, the model (10) can be rewritten into the proportion of susceptibles $x = S/N$ and the total population size N:

$$\begin{aligned} x' &= \beta \tfrac{m(N)}{N}[x^2 + \xi(2\sigma x(1-x) + \sigma^2(1-x)^2) - x(x + \sigma(1-x))^2] + x(1-x)\left[\alpha - \bar{\lambda}\tfrac{m(N)}{N}\right], \\ N' &= N\{\beta \tfrac{m(N)}{N}[x + \sigma(1-x)]^2 - \bar{\mu} - \alpha(1-x)\} \end{aligned} \tag{11}$$

where we denoted

$$m(N) := \mathcal{M}\left(\frac{N}{2}, \frac{N}{2}\right) = \frac{w}{4}\frac{N^2}{N+c} \tag{12}$$

and $\bar{\lambda} := 2\lambda$.

The equation for the proportion x of susceptible individuals is no longer independent of N. To simplify the analysis we assume from here on that there is full vertical transmission ($\xi = 0$) and no disease-induced mortality ($\alpha = 0$). The model becomes

$$\begin{aligned} x' &= \tfrac{m(N)}{N} x(1-x)[\beta(1-\sigma)^2 x - \beta\sigma^2 - \bar{\lambda}], \\ N' &= N\{\beta\tfrac{m(N)}{N}[x + \sigma(1-x)]^2 - \bar{\mu}\} \end{aligned} \tag{13}$$

We have the following possible equilibrium points with non-zero population size:

1. Disease-free equilibrium (DFE), $(1, \bar{N})$ where $\bar{N}$ satisfies
$$\beta\frac{m(\bar{N})}{\bar{N}} = \mu + b\bar{N},$$
2. Susceptible extinction equilibrium (SEE), $(0, \hat{N})$ where $\hat{N}$ satisfies
$$\beta\sigma^2\frac{m(\hat{N})}{\hat{N}} = \mu + b\hat{N},$$
3. Endemic equilibrium, (x^*, N^*) with
$$x^* = \frac{\beta\sigma^2 + \bar{\lambda}}{\beta(1-\sigma)^2}$$
and N^* satisfying
$$\beta\frac{m(N^*)}{N^*}[x^* + \sigma(1-x^*)]^2 = \mu + bN^*.$$

Note that in each case there is an additional possible extinction equilibrium if μ is big enough or, due to the Allee effect, if c is big enough. The main result for the model (13) is summarized in the following theorem while its proof is given in Appendix C:

Theorem 3 *Assuming full vertical transmission and no disease-induced mortality in the model (13), the only possible stable endemic equilibrium is SEE and the population size at this equilibrium decreases with the increasing level of sterility* $\left(\frac{dN^*}{d\sigma} > 0\right)$.

Numerical simulations suggest that the same is true also for $\xi > 0$ and $\alpha > 0$ (Fig. 5). Note that we get the same qualitative result as for the model with the harmonic mean mating function analyzed in the previous subsection. Hence, the endemic equilibrium can only decrease with the increasing level of sterility of the infection.

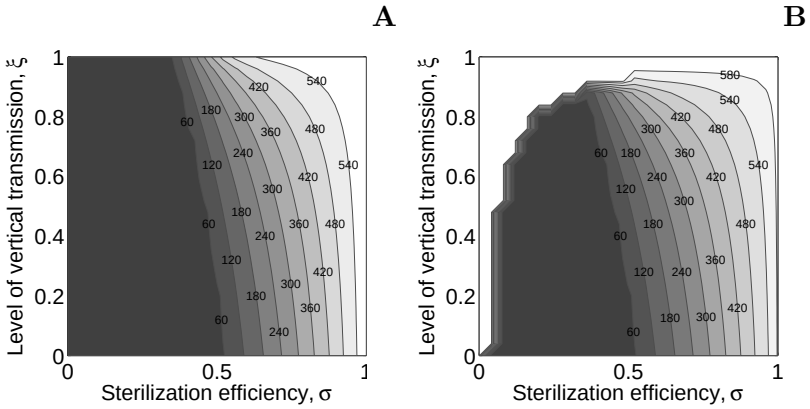

Fig. 5: The effect of changing the sterilization efficiency σ and the efficiency of vertical transmission ξ in the model (10) on the total population size at the endemic equilibrium. Disease transmission occurs through the asymptotic-incidence-like function. Parameter values: $\beta = 1.4$, $\mu = 0.2$, $b = 0.002$, $\alpha = 0.01$; we set $\lambda = 1.5$ in panel A, which corresponds to $\lambda > \alpha+\beta$, and $\lambda = 1.3$ in panel B, which corresponds to $\lambda < \alpha+\beta$. In the contour plots, the lighter the color is the higher the value of N^* is. The white area in panel B corresponds to parameter combinations where the disease-free equilibrium $(K, 0)$ is globally stable. In both panels, the darkest grade of gray color corresponds to disease-induced population extinction.

3.3 The two-sex model derived from the model (4) with a harmonic mean mating function and sex-specific sterilization rates

In this subsection we aim to study the impact of sex-specific sterilization rates (i.e. $\sigma_f \neq \sigma_m$). In order to make an analysis possible we keep all the other simplifying assumptions (i.e. equal sex-related parameters and no disease-induced host mortality). Below is the corresponding model in the total populations of females and males and the female and male susceptible proportions $x = \frac{S_f}{F}$ and $y = \frac{S_m}{M}$:

$$
\begin{aligned}
F' &= \tfrac{\beta\gamma_f \mathcal{M}(F,M)}{FM}(S_f + \sigma_f I_f)(S_m + \sigma_m I_m) - \bar{\mu}F,\\
M' &= \tfrac{\beta\gamma_m \mathcal{M}(F,M)}{FM}(S_f + \sigma_f I_f)(S_m + \sigma_m I_m) - \bar{\mu}M\\
x' &= \tfrac{\beta\gamma_f \mathcal{M}(F,M)}{F}\{xy + \xi[\sigma_f y(1-x) + \sigma_m x(1-y) + \sigma_f\sigma_m(1-x)(1-y)]\} -\\
&\quad -\lambda\tfrac{\mathcal{M}(F,M)}{F}x(1-y) - \tfrac{\mathcal{M}(F,M)}{F}x[x + \sigma_f(1-x)][y + \sigma_m(1-y)],\\
y' &= \tfrac{\beta\gamma_f \mathcal{M}(F,M)}{M}\{xy + \xi[\sigma_f y(1-x) + \sigma_m x(1-y) + \sigma_f\sigma_m(1-x)(1-y)]\} -\\
&\quad -\lambda\tfrac{\mathcal{M}(F,M)}{M}y(1-x) - \tfrac{\mathcal{M}(F,M)}{M}y[x + \sigma_f(1-x)][y + \sigma_m(1-y)].
\end{aligned}
\tag{14}
$$

Assuming a 1:1 sex ratio at birth ($\gamma_f = \gamma_m = 1/2$), we can study the following planar system in x and y:

$$\begin{aligned} x' &= \mathcal{M}(1,1)\Big\{\tfrac{\beta}{2}[xy(1-x) + (\xi - x)(\sigma_f y(1-x) + \sigma_m x(1-y) + \\ &\quad + \sigma_f\sigma_m(1-x)(1-y))] - \lambda x(1-y)\}, \\ y' &= \mathcal{M}(1,1)\Big\{\tfrac{\beta}{2}[xy(1-y) + (\xi - y)(\sigma_f y(1-x) + \sigma_m x(1-y) + \\ &\quad + \sigma_f\sigma_m(1-x)(1-y))] - \lambda y(1-x)\}. \end{aligned} \tag{15}$$

This system admits the following equilibrium points: $(1,1)$, (x_1,x_1) and (x_2,x_2) with

$$x_{1,2} = \frac{2\lambda + \beta\sigma_f\sigma_m + \beta\xi(\sigma_f\sigma_m - \sigma_f - \sigma_m) \pm \sqrt{\Delta}}{2\beta(1-\sigma_f)(1-\sigma_m)} \text{ where}$$

$$\Delta := [2\lambda + \beta\sigma_f\sigma_m + \beta\xi(\sigma_f\sigma_m - \sigma_f - \sigma_m)]^2 - 4\beta^2\xi\sigma_f\sigma_m(1-\sigma_f)(1-\sigma_m).$$

The stability of these equilibria is resolved in the following theorem (proof is given in Appendix D):

Theorem 4 *Concerning dynamics of the model (15) and denoting*

$$A(\xi) = 1 - (\sigma_f + \sigma_m) + \xi(\sigma_f + \sigma_m),$$

$$B(\xi) = 2 - 2(\sigma_f + \sigma_m) + \sigma_f\sigma_m + \xi(\sigma_f + \sigma_m - \sigma_f\sigma_m),$$

$$C(\xi) = [\xi(\sigma_f + \sigma_m - \sigma_f\sigma_m) - \sigma_f\sigma_m] + 2\sqrt{\xi\sigma_f\sigma_m(1-\sigma_f)(1-\sigma_m)},$$

we distinguish the following three cases:

1. $\frac{2\lambda}{\beta} > A(\xi)$*:* $(1,1)$ *unstable,* (x_1,x_1) *stable and* (x_2,x_2) *does not exist;*
2. $C(\xi) < \frac{2\lambda}{\beta} < \min\{A(\xi), B(\xi)\}$*:* $(1,1)$ *stable,* (x_1,x_1) *stable and* (x_2,x_2) *unstable. In this case we have bistability between the DFE and the endemic equilibrium (which becomes the SEE for full vertical transmission* $\xi = 0$*);*
3. *In all other cases* $(1,1)$ *is stable and there is no endemic equilibrium.*

From this theorem we see that the only case that guarantees some form of control effectiveness is the first one (in all the other cases the DFE is locally stable).

Interestingly, the last case when no control effectiveness is achieved can happen due to too much sterility if the infection rate is too low. For example, if $\frac{2\lambda}{\beta} < \min\{A(\xi), C(\xi)\}$ then only $(1,1)$ is stable. This is only possible if $A(\xi) > 0$ which is equivalent to

$$\sigma_f + \sigma_m < \frac{1}{1-\xi}.$$

This means that there is at least one situation where increasing the sterility (i.e. decreasing σ_f or σ_m) may prevent the disease from invading. It is also clear that this is tied up with the vertical transmission: without vertical transmission ($\xi = 1$) this case will have no implications for the values of σ_f or σ_m.

We now analyze whether the population size at the endemic equilibrium increases or decreases with the increasing level of sterility. The theorem above implies that (x_1, x_1) is the only stable endemic proportion of the susceptible females and males. From the first equation of the model (14) we see that

$$F(t) \to \frac{\frac{\beta}{2}\mathcal{M}(1,1)[x_1 + \sigma_f(1-x_1)][x_1 + \sigma_m(1-x_1)] - \mu}{2b}.$$

Notice that in the case of $\sigma_f = \sigma_m := \sigma$ the monotonicity of the total population size is the same as that of the function

$$q(\sigma) := x_1 + \sigma(1 - x_1).$$

In the case of full vertical transmission ($\xi = 0$) the endemic equilibrium becomes the SEE ($x^* = 0$) and $q(\sigma) = \sigma$. Thus, in this case, the total population size increases with σ. Below we show that the population size increases with σ in the absence of vertical transmission as well. Indeed, if $\xi = 1$ the thresholds in Theorem 4 become

$$A(1) = 1,\ B(1) = 2(1-\sigma) \text{ and } C(1) = 4\sigma(1-\sigma).$$

We thus have two cases:

1. $\frac{2\lambda}{\beta} > 1$ in which case only (x_1, x_1) is stable;
2. $4\sigma(1-\sigma) < \frac{2\lambda}{\beta} < 1$ and $\sigma < \frac{1}{2}$ in which case we have bistability between the DFE and the endemic equilibrium.

Notice also that, in this case,

$$q(\sigma) = \frac{\lambda - \sqrt{\lambda(2\beta\sigma^2 - 2\beta\sigma + \lambda)}}{\beta(1-\sigma)}$$

and

$$q'(\sigma) = \frac{\lambda[\sqrt{\lambda(2\beta\sigma^2 - 2\beta\sigma + \lambda)} - \lambda + \beta(1-\sigma)]}{\beta(1-\sigma)^2\sqrt{\lambda(2\beta\sigma^2 - 2\beta\sigma + \lambda)}}.$$

With some computation we see that $q'(\sigma) > 0$ whenever either $\lambda < \beta(1-\sigma)$ or $2\lambda > \beta$. One of these conditions is always satisfied given the conditions in Theorem 4. Therefore, in the absence of vertical transmission the total population size is also increasing with σ. Numerical simulations suggest that this is also the case for any intermediate level of vertical transmission $0 < \xi < 1$.

If the pathogen is sex-specific, i.e. it only reduces the fertility in females or males, we can see that the DFE is never stable in the case of full vertical transmission. Indeed, if $\sigma_f = 1$ and $\xi = 0$ then $A(\xi) = -\sigma_m$ and the condition $\frac{2\lambda}{\beta} > A(\xi)$ in Theorem 4 is trivially satisfied. Analogously, if $\sigma_m = 1$ and $\xi = 0$ then $A(\xi) = -\sigma_f$ and the condition $\frac{2\lambda}{\beta} > A(\xi)$ is also trivially satisfied. This means that if the sterilization is sex-specific and the vertical transmission is high enough, the pest control measure never fails.

4 Conclusions and discussion

In this paper we analyzed the effect of sterilization in the infected hosts in several epidemiological models involving infectious diseases that can be transmitted both vertically and horizontally. Our particular focus has been on how the host population size at an endemic equilibrium responds to an increasing level of sterilization. This question has been motivated by the fact that sterilizing pathogens can be used as pest control agents by intentionally inoculating the target population, with the goal of reducing or eliminating it completely. We addressed this question using two types of epidemiological models of SI (susceptible-infectious) type. The first type was a conventional model with logistic host growth in the absence of infection, vertical and horizontal disease transmission, disease-induced mortality and disease-reduced reproduction (Pugliese, 1990; Zhou and Hethcote, 1994). For the second type, we assumed that the host reproduction process and the (sexual) disease transmission process were both mediated by mating and modeled by the same structural term, a feature to be expected in many realistic cases (Berec and Maxin, 2013).

Contrary to previous models that did not include vertical transmission (e.g. Deredec et al, 2008; Berec and Maxin, 2012) we found that in the conventional model the population size at the endemic equilibrium may actually increase with higher levels of sterility. This effect appears to be most pronounced for mass action and asymptotic types of infection incidence, and occurs only to a limited extent for standard incidence. On the other hand, if the disease is sexually transmitted and the host reproduction and disease transmission are both consistently mediated by mating, we do not observe such a counter-intuitive effect and the population size at a stable endemic equilibrium is decreasing with higher levels of sterility. For the latter model type, this holds equally for both mating functions we use, a degree-one homogeneous mating function which results in a standard-incidence-like disease transmission term and a mating function accounting for mate-finding Allee effects which results in an asymptotic-incidence-like disease transmission term.

Pugliese (1990) and Zhou and Hethcote (1994) studied models of which our conventional model is just a special case. Interestingly, however, they did not observe this type of behavior that the population size at the endemic equilibrium may actually increase with higher levels of sterility. In fact, both authors focused on a quite detailed, standard analysis of their epidemiological models, consisting of looking for thresholds of disease persistence and existence and stability of model equilibria. This is because their focus was analyzing an infectious model in its generality where these thresholds are the most important features to look for. From the perspective of pest control, however, the objectives are different and somewhat contrary to other applications. Here we want disease persistence (so that the sterilizing agent can establish in the target host) and, in addition, we want a reduction of the population size at the endemic equilibrium. As we can see, simply changing the attention to a more specific case can be the way to get unexpected insight into the system under study.

The observation that the population size at the endemic equilibrium may actually increase with higher levels of sterility has important consequences for a potential of this manipulation for population control. Contrary to what one could expect, too much sterility may simply mean a decrease in pest control efficiency. Optimum values of sterility levels for any particular efficiency of vertical transmission can be deducted from contour plots we draw in the main text or any of their equivalents. When we do not limit ourselves to the endemic equilibria, we may observe this counter-intuitive effect also in the models with the host reproduction and disease transmission mediated by mating. In particular, if $\lambda < \alpha + \beta$ (panels B in all of our figures) then too much sterility causes the disease to become non-persistent, and the disease-free equilibrium becomes globally stable. On the other hand, this abrupt change in system stability when sterility levels increase is preceded by a region of disease-induced extinction, so that it plays a role just if we abruptly change the sterility level from low to too high values.

One of the techniques that exploit sterilizing pathogens is the virus-vectored immunocontraception (VVIC) (Tyndale-Biscoe, 1994; Hardy et al, 2006). This method is sex-specific, triggering an autoimmune response whereby antibodies are produced against the gametes of a sex and fertilization is thus blocked. The full two-sex model that we analyzed in the last subsection is its more appropriate description. For this model, we showed that an increase in the host population size cannot happen as the sterilization efficiency increases. In addition, we showed that when just one sex is (partially) sterilized then, when the vertical efficiency is perfect, the disease-free equilibrium cannot be stable. This implies that if our preference is to assure a degree of pest control in the first place instead of to maximize it then this is a way of how to do that. Put from another perspective, while strategies that sterilize both sexes can be more effective as regards the degree of population control, some combinations may make the parasite non-persistent and no control is then guaranteed.

Why did not Deredec et al (2008) and Berec and Maxin (2012) observe any increase in the host population size as a results of a sterility level increase? Actually, all of our models predict that once there is no vertical transmission (i.e. $\xi = 1$) then the systems behaves as expected: the host population size decreases as the sterility level increases. Hence, it is vertical transmission that is responsible for this effect. Whether we need low or high levels of vertical transmission to observe the effect depends on values of the other model parameters. Interestingly, any adverse effect of parasites, including disease-induced mortality and disease-reduced reproduction, negatively affects propensity of parasites to evolve vertical transmission (Lipsitch et al, 1995; Altizer and Augustine, 1997; Bernhauerova and Berec, 2014). It would thus be very interesting to see the extent to which such an association could naturally evolve.

Do pathogens actually considered for pest control involve vertical transmission? And if yes, can their level of vertical transmission be so large that the phenomena discussed in this paper are likely? Pathogens as agents regulating populations are commonly used against insect pests. They mostly come from baculoviruses (Cory and Myers, 2003), but other viruses such as densoviruses

from the family *Parvoviridae* can also be found useful (Hirunkanokpun et al, 2008). Of baculoviruses, vertical transmission is especially common in nucleopolyhedroviruses and cypoviruses in which it can reach levels as high as 50% (Kukan, 1999) and commonly about 10-20% (Kukan, 1999; Fuxa et al, 2002). Similarly, some mosquito densovirus strains showed 20-50% vertical transmission (Hirunkanokpun et al, 2008). Figures 1-3 show that 50% levels of vertical transmission are indeed large for the phenomena observed in this study to be likely. Playing with model parameters, we were able to observe these phenomena for levels of vertical transmission as low as 15-20% but in general, the effect is pronounced the more the higher the level of vertical transmission is. On the other side of spectrum of pathogens with both horizontal and vertical transmission that are used to control insect pests are *Wolbachia* bacteria. These bacteria reach very high levels of vertical transmission and very low levels of horizontal transmission (Haine et al, 2005). However, these bacteria also manipulate reproduction of their arthropod hosts and require a modified model of eco-epidemiological dynamics. Whether the phenomena observed in this study would also appear in that model is currently unknown.

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A Proof of Theorem 1

With $\Phi(N) = \lambda$, the expression (3) implies

$$N^* = \frac{-\sigma(1-\sigma)(1-\xi)\beta^2 + \beta\{\mu+\alpha+\sigma[\lambda-\mu+\alpha(\xi-2)]\} - (\mu+\alpha)(\lambda-\alpha)}{b[\lambda-\alpha-\beta(1-\sigma)]}.$$

Its derivative with respect to σ is

$$\frac{dN^*}{d\sigma} = \frac{\beta}{b[\lambda-\alpha-\beta(1-\sigma)]^2} h(\sigma)$$

with

$$h(\sigma) := (1-\xi)\beta^2\sigma^2 + 2\beta(1-\xi)(\lambda-\alpha-\beta)\sigma + (\lambda-\alpha-\beta)[\lambda-(1-\xi)(\alpha+\beta)].$$

This function has two roots (which are the critical values of $\frac{dN^*}{d\sigma}$):

$$\hat{\sigma} = \frac{(\alpha+\beta-\lambda)(1-\xi) - \sqrt{\xi\lambda(1-\xi)(\alpha+\beta-\lambda)}}{\beta(1-\xi)} \text{ and}$$

$$\bar{\sigma} = \frac{(\alpha+\beta-\lambda)(1-\xi) + \sqrt{\xi\lambda(1-\xi)(\alpha+\beta-\lambda)}}{\beta(1-\xi)}.$$

If these roots are complex, i.e if $\lambda > \alpha+\beta$, then $h(\sigma) > 0$ on its entire domain and N^* is increasing with σ. In what follows we assume that $\hat{\sigma}$ and $\bar{\sigma}$ are real, that is, $\lambda < \alpha+\beta$. Combined with the endemicity condition $R_0 > 1$ this becomes

$$\alpha+\beta > \lambda > \alpha+\beta[1-(1-\xi)\sigma]. \tag{16}$$

Notice also that $R_0 > 1$ provides the following lower bound for σ:

$$\sigma > \sigma_R := \frac{\alpha+\beta-\lambda}{\beta(1-\xi)}.$$

From (16) it follows that

$$h'(0) = 2\beta(1-\xi)(\lambda-\alpha-\beta) < 0 \text{ and}$$

$$h'(1) = 2\beta(1-\xi)(\lambda-\alpha) > 0.$$

Furthermore, $h'(\sigma_R) = 2\beta\xi(\alpha+\beta-\lambda) > 0$ which implies $\hat{\sigma} < \sigma_R$. This means that $\hat{\sigma}$ is always outside the endemic range of σ.

We now analyze the position of the real roots $\hat{\sigma}$ and $\bar{\sigma}$ relative to their feasible interval $(0,1)$. This can be done by analyzing the signs of

$$h(0) = (\lambda-\alpha-\beta)[\lambda-(1-\xi)(\alpha+\beta)] \text{ and}$$

$$h(1) = (1-\xi)[\alpha^2+\lambda(\beta-\alpha)] + \lambda(\lambda-\alpha-\beta).$$

First notice that $\alpha^2+\lambda(\beta-\alpha) > 0$. This follows from the fact that either $\beta > \alpha$ or, otherwise,

$$\lambda < \alpha+\beta < \frac{\alpha^2}{\alpha-\beta}.$$

We have the following cases:

1. $h(0) > 0$ and $h(1) < 0$. This is equivalent to

$$\max\left\{\frac{\lambda}{\alpha+\beta}, \frac{\alpha+\beta-\lambda}{\beta\sigma}\right\} < 1-\xi < \frac{\lambda(\alpha+\beta-\lambda)}{\alpha^2+\lambda(\beta-\alpha)}.$$

   In this case $\bar{\sigma} > 1$ and N^* increases on the interval $(0,\hat{\sigma})$ and decreases on the interval $(\hat{\sigma},1)$. Intersecting this with the endemic condition it follows that the relevant interval of σ is $(\sigma_R,1)$ and N^* is decreasing with increasing σ in this case.
2. $h(0) < 0$ and $h(1) > 0$. This is equivalent to

$$\max\left\{\frac{\lambda(\alpha+\beta-\lambda)}{\alpha^2+\lambda(\beta-\alpha)}, \frac{\alpha+\beta-\lambda}{\beta\sigma}\right\} < 1-\xi < \frac{\lambda}{\alpha+\beta}.$$

   In this case $\hat{\sigma} < 0$ and N^* is decreasing on the interval $(0,\bar{\sigma})$ and increasing on $(\bar{\sigma},1)$. To compare now $\bar{\sigma}$ with σ_R we first compute

$$h'(\bar{\sigma}) = 2\beta\sqrt{\xi\lambda(1-\xi)(\alpha+\beta-\lambda)}$$

   and see that $h'(\bar{\sigma}) > h'(\sigma_R)$ because this is equivalent to

$$1-\xi > \frac{\alpha+\beta-\lambda}{\alpha+\beta}$$

   which follows from the fact that

$$1-\xi > \frac{\alpha+\beta-\lambda}{\beta\sigma}.$$

   This proves that $\sigma_R < \bar{\sigma}$ and N^* is decreasing with σ on $(\sigma_R,\bar{\sigma})$ and increasing on $(\bar{\sigma},1)$.
3. $h(0) > 0$ and $h(1) > 0$ equivalent to

$$1-\xi > \max\left\{\frac{\lambda}{\alpha+\beta}, \frac{\lambda(\alpha+\beta-\lambda)}{\alpha^2+\lambda(\beta-\alpha)}, \frac{\alpha+\beta-\lambda}{\beta\sigma}\right\}.$$

   In this case $0 < \hat{\sigma} < \bar{\sigma} < 1$ and N^* is decreasing on $(\hat{\sigma},\bar{\sigma})$ and increasing otherwise. For the same reasons as in the previous case, $\sigma_R < \bar{\sigma}$ and N^* is decreasing with increasing σ on $(\sigma_R,\bar{\sigma})$ and increasing on $(\bar{\sigma},1)$.
4. $h(0) < 0$ and $h(1) < 0$ equivalent to

$$\frac{\alpha+\beta-\lambda}{\beta\sigma} < 1-\xi < \min\left\{\frac{\lambda}{\alpha+\beta}, \frac{\lambda(\alpha+\beta-\lambda)}{\alpha^2+\lambda(\beta-\alpha)}\right\}.$$

   In this case $\hat{\sigma} < 0$ and $\bar{\sigma} > 1$ which means that N^* is decreasing on $(0,1)$ irrespective of ξ. In this last case it is easy to see that $\sigma_R < 1$ and N^* is decreasing with increasing σ on $(\sigma_R,1)$.

B Proof of Theorem 2

Since $G(0) = \hat{\beta}\xi\sigma^2 > 0$ we have several cases:

1. $G(1) < 0$ which is equivalent to the condition (7). This means there is a unique equilibrium $x^* \in (0,1)$. Since $G(x) > 0$ for $x < x^*$ and $G(x) < 0$ for $x > x^*$ it is also globally stable. Since $x(t) \to x^*$ it follows that the equation of N is asymptotically autonomous and the limiting system is a logistic equation

$$N' = N\{[\hat{\beta}\sigma(2-\sigma)(1-\xi) + \hat{\lambda}]x^* + \hat{\beta}(1-\xi)\sigma^2 - \alpha - \bar{\mu}\} \tag{17}$$

   where we have used the equality

$$\hat{\beta}(1-\sigma)^2(x^*)^2 = [\hat{\beta}\sigma^2(1+\xi) - 2\sigma\hat{\beta}\xi + \hat{\lambda} - \alpha]x - \hat{\beta}\xi\sigma^2.$$

   Therefore, N approaches 0 or N^* with

$$N^* = \frac{[\hat{\beta}\sigma(2-\sigma)(1-\xi) + \hat{\lambda}]x^* + \hat{\beta}(1-\xi)\sigma^2 - \alpha - \mu}{b}$$

   depending on whether $[\hat{\beta}\sigma(2-\sigma)(1-\xi) + \hat{\lambda}]x^* + \hat{\beta}(1-\xi)\sigma^2 - \alpha - \mu$ is positive or not. Specifically, $N \to 0$ if and only if

$$x^* < \frac{-\hat{\beta}(1-\xi)\sigma^2 + \alpha + \mu}{\hat{\beta}\sigma(2-\sigma)(1-\xi) + \hat{\lambda}}.$$

2. $G(1) > 0$ and $G(x)$ has no roots in $(0,1)$. In this case $x^* = 1$ which corresponds to the globally stable disease-free equilibrium.
3. $G(1) > 0$ and $G(x)$ has two roots in $(0,1)$, denoted by x_1 and x_2, with $x_1 < x_2$. This case is possible if

$$G'(0) < 0,\ G'(1) > 0 \text{ and } G(x_{min}) < 0$$

   where x_{min} is the x-coordinate of the vertex of the parabola $G(x)$:

$$x_{min} = \frac{\hat{\beta}(1+\xi)\sigma^2 - 2\hat{\beta}\sigma\xi + \hat{\lambda} - \alpha}{2\hat{\beta}(1-\sigma)^2}.$$

   Whenever this case is possible we have that x_1 and 1 are the locally stable proportions. Hence we have bistability between the DFE and the endemic state or between DFE and the extinction equilibrium, depending on whether $[\hat{\beta}\sigma(2-\sigma)(1-\xi)+\hat{\lambda}]x_1+\hat{\beta}(1-\xi)\sigma^2 - \alpha - \mu$ is positive or not.

We now study the monotonicity of a possible endemic state with respect to σ, the sterilization efficiency. Solving the equation $G(x) = 0$ for x_1 we have

$$x_1 = \frac{1}{2\hat{\beta}(1-\sigma)^2}[\hat{\lambda} - \alpha - 2\hat{\beta}\sigma\xi + \hat{\beta}(1+\xi)\sigma^2 - \sqrt{\Delta}]$$

with Δ being the discriminant of $G(x)$. Let us analyze again the two extreme cases: full vertical transmission ($\xi = 0$) and no vertical transmission ($\xi = 1$). If $\xi = 0$ then $x_1 = 0$ and

$$N^* = \frac{\hat{\beta}\sigma^2 - \alpha - \mu}{b}$$

which is clearly increasing in σ.
If $\xi = 1$ first notice that (7) is equivalent to

$$\hat{\lambda} > \alpha + \hat{\beta}$$

and x_1 becomes

$$x_1 = \frac{\hat{\lambda} - \alpha - 2\hat{\beta}\sigma(1-\sigma) - \sqrt{\Delta}}{2\hat{\beta}(1-\sigma)^2}$$

with $\Delta = (\hat{\lambda}-\alpha)[\hat{\lambda}-\alpha-4\hat{\beta}\sigma(1-\sigma)]$. The total population size at the endemic equilibrium becomes

$$N^* = \frac{\hat{\lambda}(\hat{\lambda}-\alpha) - 2\hat{\beta}(1-\sigma)[\mu+\alpha+\sigma(\hat{\lambda}-\mu-\alpha)] - \hat{\lambda}\sqrt{\Delta}}{2b\hat{\beta}(1-\sigma)^2}.$$

Its derivative with respect to σ is

$$\frac{dN^*}{d\sigma} = \frac{\hat{\lambda}}{b\hat{\beta}(1-\sigma)^3\sqrt{\Delta}}[A\sqrt{\Delta} - B]$$

where we have denoted

$$A := \hat{\lambda}-\alpha-\hat{\beta}(1-\sigma) \text{ and } B := (\hat{\lambda}-\alpha)[\hat{\lambda}-\alpha+\hat{\beta}(2\sigma^2-\sigma-1)].$$

Notice that (7) implies $A > 0$ and the above derivative is positive if $B < 0$ or if $A^2\Delta - B^2 > 0$. This last inequality is equivalent to

$$4(\hat{\lambda}-\alpha)\sigma\hat{\beta}^2(1-\sigma)^3(\hat{\lambda}-\alpha-\hat{\beta}) > 0$$

which is true if (7) holds.

It remains now to analyze the bistability case when both x_1 and 1 are locally stable. This model was fully analyzed in Berec and Maxin (2013) and this case corresponds to the following conditions on the parameters:

$$4\hat{\beta}\sigma(1-\sigma) < \hat{\lambda}-\alpha < \hat{\beta} \text{ and } \sigma < \frac{1}{2}.$$

We will show that $\frac{dN^*}{d\sigma}$ is positive in this case as well. First notice that

$$\hat{\lambda}-\alpha+\hat{\beta}(2\sigma^2-\sigma-1) < 0.$$

Otherwise, it would imply

$$\hat{\beta} > \hat{\lambda}-\alpha > \hat{\beta}(1+\sigma-2\sigma^2)$$

which in turns implies $1-2\sigma < 0$, contradicting the conditions of the bistability case. Then the positivity of derivative $\frac{dN^*}{d\sigma}$ is equivalent to $B^2 - A^2\Delta > 0$, that is,

$$4(\hat{\lambda}-\alpha)\sigma\hat{\beta}^2(1-\sigma)^3(\hat{\lambda}-\alpha-\hat{\beta}) < 0$$

which is true since $\hat{\lambda}-\alpha-\hat{\beta} < 0$ and $\hat{\lambda}-\alpha > 0$.

We proceed now to show that $\frac{dN^*}{d\sigma}$ is positive for any intermediate value of vertical transmission ξ. We managed to show this only under additional simplifying assumptions: no disease induced mortality $\alpha = 0$ and $\hat{\lambda} = \hat{\beta}$. First notice that

$$N^* = \frac{\hat{\beta}[x_1+\sigma(1-x_1)]^2 - \mu}{b},$$

so it is more convenient to analyze the monotonicity of the function

$$C(\sigma) := x_1 + \sigma(1-x_1).$$

The derivative of this function is

$$\frac{dC}{d\sigma} = \frac{P\sqrt{\Delta} - Q}{(1-\sigma)^2\sqrt{\Delta}}$$

with

$$P = (1-\xi)(\sigma^2-2\sigma+2)+1,$$
$$Q = [1+(1-\xi)\sigma(2-\sigma)][(1-\xi)\sigma^2 - 2\xi(1-\sigma)+1].$$

Since $P > 0$ then the derivative $\frac{dC}{d\sigma}$ is positive if either $Q < 0$ or if $P^2\Delta - Q^2 > 0$. After some computation,

$$P^2\Delta - Q^2 = 8(1-\xi)(1-\sigma)^2 g(\xi)$$

with

$$g(\xi) := (\sigma^4 + 2\sigma^2 - 2\sigma^3)\xi^2 + (-2\sigma^2 + 2\sigma^3 - 2\sigma - 2\sigma^4)\xi + (1+\sigma^2)^2.$$

Notice that $\sigma^4 + 2\sigma^2 - 2\sigma^3 > 0$ and the discriminant of $g(\xi)$ is

$$-4\sigma^2(1-\sigma)^4 < 0$$

which means $g(\xi) > 0$ and this concludes the proof that $\frac{dN^*}{d\sigma} > 0$.

C Proof of Theorem 3

We first prove a technical lemma that will be used in analyzing the stability of the DFE and SEE of model (13):

Lemma 1 *Let $k > 0$ and E defined as*

$$E = km'(x^*) - \mu - 2bx^*$$

where x^* is a positive root of

$$k\frac{m(x)}{x} = \mu + bx.$$

Then there is at most one positive x^* such that $E < 0$ if $m(x)$ corresponds to the mating function with the mate-finding Allee effect.

Proof Suppose $m(x) = \frac{x^2}{c+2x}$. In this case x^* is a root of

$$a(x) = 2bx^2 + (2\mu + bc - k)x + \mu c = 0.$$

This quadratic can have either two positive or two negative real roots. The necessary and sufficient condition for existence of two real positive roots is

$$k - 2\mu - bc > \sqrt{8b\mu c}.$$

Replacing k with $\frac{(\mu+bx^*)(c+2x^*)}{x^*}$ we have that $E < 0$ if and only if

$$x^* > \sqrt{\frac{\mu c}{2b}}.$$

On the other hand,

$$a\left(\sqrt{\frac{\mu c}{2b}}\right) = 2\mu c + (2\mu + bc - k)\sqrt{\frac{\mu c}{2b}} < 0$$

whenever the existence condition of a positive x^* is satisfied. This shows that $\sqrt{\frac{\mu c}{2b}}$ lies between the two positive roots of $m(x)$. Hence $E < 0$ is satisfied for only one positive root whenever such root exists.

We now provide the main results concerning the stability of the equilibrium points:

1. The endemic equilibrium is unstable whenever it exists.
   Note that $x^* < 1$ if and only if

   $$\bar{\lambda} < \beta(1-2\sigma) \text{ and } \sigma < \frac{1}{2}.$$

   On the other hand, one eigenvalue of the Jacobian of (13) evaluated at (x^*, N^*) is

   $$\frac{m(N^*)}{\beta(1-\sigma)^2 N^*}[\bar{\lambda} + \beta\sigma^2][\beta(1-2\sigma) - \bar{\lambda}]$$

   which is positive.

2. There is at most one susceptible extinction equilibrium which is locally stable.
   Evaluating the Jacobian at the SEE we obtain the following eigenvalues:

$$\hat{e}_1 = -\frac{m(\hat{N})}{\hat{N}}[\bar{\lambda} + \beta\sigma^2] < 0,$$

$$\hat{e}_2 = \beta\sigma^2 m'(\hat{N}) - \mu - 2b\hat{N}.$$

   From Lemma 1 with $k = \beta\sigma^2$ we conclude that whenever an SEE exists there is only one for which $e_2 < 0$ and thus stable.
3. If the system has an endemic equilibrium then the DFE is locally stable whenever it exists.
   The Jacobian evaluated at $(1, \bar{N})$ has two eigenvalues

$$\bar{e}_1 = \frac{m(\bar{N})}{\bar{N}}[\bar{\lambda} - \beta(1 - 2\sigma)],$$

$$\bar{e}_2 = \beta m'(\bar{N}) - \mu - 2b\bar{N}.$$

   From the previous result $\bar{e}_1 < 0$ whenever x^* exists. Also, from Lemma 1 with $k = \beta$ it follows that $e_2 < 0$ whenever the DFE exists.

From these results, we see that the SEE is the only outcome for $\alpha = 0$ and $\xi = 0$ for which one can achieve some level of control effectiveness (i.e. a disease-induced reduction of the host population size). Moreover, the population level at the SEE is always an increasing function in σ:

$$\frac{d\hat{N}}{d\sigma} = -\frac{2\beta\sigma m(\hat{N})}{\beta\sigma^2 m'(\hat{N}) - \mu - 2b\hat{N}}$$

which is positive whenever the SEE is stable.

D Proof of Theorem 4

If we subtract the right-hand sides of the two equations in (15) we obtain

$$-\mathcal{M}(1,1)(x-y)\left[\frac{\beta}{2}((1-\sigma_m)y + \sigma_m)((1-\sigma_f)x + \sigma_f) + \lambda\right] = 0.$$

Hence, at any equilibrium (x^*, y^*), we have $x^* = y^*$ and x^* is a solution of

$$(1-x)\{\beta(1-\sigma_f)(1-\sigma_m)x^2 - [\beta\sigma_f\sigma_m + 2\lambda + \beta\xi(\sigma_f\sigma_m - \sigma_f - \sigma_m)]x + \beta\xi\sigma_f\sigma_m\} = 0.$$

Obviously, the equilibrium $x^* = y^* = 1$ corresponds to the disease-free equilibrium. The endemic equilibrium is a root of the function

$$f(x) := \beta(1-\sigma_f)(1-\sigma_m)x^2 - [\beta\sigma_f\sigma_m + 2\lambda + \beta\xi(\sigma_f\sigma_m - \sigma_f - \sigma_m)]x + \beta\xi\sigma_f\sigma_m.$$

Notice that $f(0) > 0$ and the product of the two roots of $f(x)$ is positive as well. So, the real roots, whenever they exists, are either both positive or both negative. Any feasible equilibrium must also belong to the interval $(0, 1)$. Hence, we distinguish two cases:

1. $f(1) < 0$ which is equivalent to

$$\frac{2\lambda}{\beta} > 1 - (1-\xi)(\sigma_f + \sigma_m).$$

   This case implies the existence of a unique steady state which is x_1.

2. $f(1) > 0$, $f'(1) > 0$, $f'(0) < 0$ and $\Delta > 0$. This case implies the existence of two steady states (x_1 and x_2) in the feasible region.
The first three conditions above are equivalent to

$$\frac{2\lambda}{\beta} < 1 - (\sigma_f + \sigma_m) + \xi(\sigma_f + \sigma_m),$$

$$\frac{2\lambda}{\beta} < 2 - 2(\sigma_f + \sigma_m) + \sigma_f\sigma_m + \xi(\sigma_f + \sigma_m - \sigma_f\sigma_m),$$

$$\frac{2\lambda}{\beta} > \xi(\sigma_f + \sigma_m - \sigma_f\sigma_m) - \sigma_f\sigma_m.$$

The last inequality above allows us to write the condition $\Delta > 0$ in a similar way as a threshold for λ and β:

$$\frac{2\lambda}{\beta} > [\xi(\sigma_f + \sigma_m - \sigma_f\sigma_m) - \sigma_f\sigma_m] + 2\sqrt{\xi\sigma_f\sigma_m(1-\sigma_f)(1-\sigma_m)}.$$

Combining these inequalities we obtain

$$C(\xi) < \frac{2\lambda}{\beta} < \min\{A(\xi), B(\xi)\}.$$

A straightforward computation also shows that the left threshold of $\frac{2\lambda}{\beta}$ is always smaller than the right threshold. Thus, the above interval always exists.

We analyze now the stability of the equilibria. We denote by $J(x, y)$ the Jacobian of the model (15). At the DFE $(1, 1)$, we have

$$Trace(J(1,1)) = -\frac{\beta}{2}\mathcal{M}(1,1)[2 - (1-\xi)(\sigma_f + \sigma_m)] < 0,$$

$$\det J(1,1) = -\frac{\beta + 2\lambda}{4}\mathcal{M}^2(1,1)[2\lambda - \beta(1 - (1-\xi)(\sigma_f + \sigma_m))] > 0$$

if and only if

$$\frac{2\lambda}{\beta} < 1 - (1-\xi)(\sigma_f + \sigma_m).$$

To analyze the stability of an endemic steady state (x^*, x^*) we first substitute β from $f(x^*) = 0$ with

$$\beta = \frac{2\lambda x^*}{(1-\sigma_f)(1-\sigma_m)(x^*)^2 + [\xi(\sigma_f + \sigma_m - \sigma_f\sigma_m) - \sigma_f\sigma_m]x^* + \xi\sigma_f\sigma_m}.$$

With this substitution we see that the trace of $J(x^*, x^*)$ is always negative, i.e.

$$Trace(J(x^*, x^*)) =$$

$$= -\frac{\lambda\mathcal{M}(1,1)\{2(1-\sigma_f)(1-\sigma_m)(x^*)^3 + [\sigma_f(1-\sigma_m) + \sigma_m(1-\sigma_f)]x^*(\xi + x^*) + 2\xi\sigma_f\sigma_m\}}{(1-\sigma_f)(1-\sigma_m)(x^*)^2 + [\xi(\sigma_f + \sigma_m - \sigma_f\sigma_m) - \sigma_f\sigma_m]x^* + \xi\sigma_f\sigma_m} < 0.$$

The determinant is

$$\det J(x^*, x^*) = -(1-x^*)\lambda^2\mathcal{M}^2(1,1)\frac{T_1[(1-\sigma_f)(1-\sigma_m)(x^*)^2 - \xi\sigma_f\sigma_m]}{T_2}$$

with

$$T_1 = (1-\sigma_f)(1-\sigma_m)(x^*)^3 + (1-\sigma_f\sigma_m)(x^*)^2 + \xi(\sigma_f + \sigma_m - \sigma_f\sigma_m)x^* + \xi\sigma_f\sigma_m > 0,$$

$$T_2 = \{(1-\sigma_f)(1-\sigma_m)(x^*)^2 + [\xi(\sigma_f + \sigma_m - \sigma_f\sigma_m) - \sigma_f\sigma_m]x^* + \xi\sigma_f\sigma_m\}^2 > 0.$$

This implies that (x^*, x^*) is stable whenever

$$x^* < \sqrt{\frac{\xi\sigma_f\sigma_m}{(1-\sigma_f)(1-\sigma_m)}}.$$

On the other hand

$$f\left(\sqrt{\frac{\xi\sigma_f\sigma_m}{(1-\sigma_f)(1-\sigma_m)}}\right) = -2\lambda\frac{[(1-\sigma_f)(1-\sigma_m)(x^*)^2 + \xi\sigma_f\sigma_m]\sqrt{\frac{\xi\sigma_f\sigma_m}{(1-\sigma_f)(1-\sigma_m)}} - 2\xi\sigma_f\sigma_m x^*}{(1-\sigma_f)(1-\sigma_m)(x^*)^2 + [\xi(\sigma_f+\sigma_m-\sigma_f\sigma_m)-\sigma_f\sigma_m]x^* + \xi\sigma_f\sigma_m}.$$

The numerator of this expression is positive since it is equivalent to

$$\frac{\xi\sigma_f\sigma_m[(1-\sigma_f)(1-\sigma_m)(x^*)^2 - \xi\sigma_f\sigma_m]^2}{(1-\sigma_f)(1-\sigma_m)} > 0.$$

This shows that

$$f\left(\sqrt{\frac{\xi\sigma_f\sigma_m}{(1-\sigma_f)(1-\sigma_m)}}\right) < 0$$

which means that, if there is only one endemic steady state, it is stable and, if there are two endemic steady states, the smaller one is stable and the larger one is unstable.