CONTROLLING PEST POPULATIONS WITH STERILIZING PATHOGENS AND VERTICAL TRANSMISSION

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Abstract. We analyze the effect of full vertical transmission in several epidemic models involving infectious diseases that cause sterilization in the infected hosts. Under certain conditions on the parameters, we found that the sterilization effect may prevent a susceptible extinction situation regardless of how large the infection rate may be. This effect is studied under several functional forms for the infection transmission term in order to assess its robustness. The implication in pest control measures is also discussed.

1. Introduction

Overpopulation and invasive species such as foxes, rabbits and mice have become critical issues in some areas. Several methods of pest control, such as hunting and increasing mortality through disease and poisoning, have proved inefficient and inhumane [7]. An alternative to these methods is the introduction of a sterilizing pathogen to the pest population. For example, researchers in Australia have studied the effects of using the sterilizing pathogens canine herpesvirus-1 in foxes, myxoma virus in rabbits, and murine cytomegalovirus and ectromelia virus in mice to control pest populations. The study found that high rates of sterility were necessary to significantly impact the pest population sizes [5].

The introduction of additional transmission pathways may also increase the effectiveness of the sterilizing pathogens. Altizer and Augustine [4] have shown that vertical, in addition to horizontal, transmission widens the range of the parameters within which the sterilizing pathogen can successfully control the pest population. For this reason, we include vertical transmission, as this property will likely increase the effectiveness of a sterilizing pathogen. In our models we assume complete vertical transmission, as this feature is ideal for persistence of the infection.

1991 *Mathematics Subject Classification.* 92D30.
Key words and phrases. sexually transmitted diseases, isolation, population models.

Vertical transmission represents the ability of an infectious disease to pass from mother to a newborn. Various infectious diseases have such ability and the probability of vertical transmission varies depending on the infectious agent and/or the possibility of treatment. For example, HIV vertical transmission can be reduced if the mother undergoes aggressive anti-retroviral treatment [2]. Although assuming complete vertical transmission may be unrealistic for many diseases, several infections display very high rates of vertical transmission. For example, hepatitis B in humans is passed from parent to offspring at a rate of 70% to 90% [8]. While studying the transmission of nucleopolyhedrovirus in insects, Kukan et al [6] found that the offspring of insects with the virus were likely to have the infection as well.

A general simple model that included vertical transmission and isolation from reproduction was studied by Maxin et. al in [3]. The most important result of that research relates to full vertical transmission when all newborn from infected individuals become infected at birth. Typically this causes the existence of an additional susceptible extinction equilibrium where the disease invades the entire population. Under some conditions on isolation rates, a susceptible extinction can be avoided with arbitrarily large infection rate. From the point of view of a human infection, this result can be seen as a positive outcome (between an endemic and a totally infected population). However, the effect of isolation from reproduction and vertical transmission can also be analyzed in a context where the goal is in fact the reduction of the total population.

L.Berec and D. Maxin studied a basic one-sex model where a pest population is deliberately infected with a sterilizing pathogen as a measure of pest control [1]. In their paper the authors focused on the possibility that sterile individuals can actually live longer and be able to further spread the sterilizing disease in the population, thus creating a double impact in their pest control ability.

In this paper our purpose is two-fold. First we want to verify the robustness of the result obtained in [3] by analyzing a pest-control model with three different types of transmission terms: standard incidence, mass-action incidence and asymptotic incidence. Thus, in all one-sex models the transmission is modeled by

$$\phi(N)\frac{SI}{N}$$

where $\phi(N)$ can be β (standard incidence), βN (mass-action incidence) or $\frac{\beta N}{c+N}$ (asymptotic incidence). We also investigate a similar two-sex model where the infection rate is replaced by a mating function, appropriate for a pathogen that is sexually transmitted. Our objective is to investigate the impact of introducing a sterilizing pathogen with vertical transmission to control pest populations.

The pest control effectiveness will be defined as in [1]

$$E = 1 - \frac{N^*}{K}$$

with N^* being the total population at a stable equilibrium and K is the carrying capacity of the pest population without any control measure. Thus

$E = 0$ will correspond to no reduction effect while $E = 1$ means that the pest population goes extinct.

2. The general one-sex model

$$(1) \quad \begin{cases} \frac{dS}{dt} &= bS - \Phi(N)\frac{S}{N}(I_f + I_s) - (d + d_1 N)S, \\ \frac{dI_f}{dt} &= (1-\sigma)bI_f + (1-\sigma)\Phi(N)\frac{S}{N}(I_f + I_s) - (d + d_1 N)I_f, \\ \frac{dI_s}{dt} &= \sigma b I_f + \sigma\Phi(N)\frac{S}{N}(I_f + I_s) - (d + d_1 N)I_s. \end{cases}$$

Where

$$N = S + I_f + I_s \text{ and } \Phi(N) \text{ is a general transmission term.}$$

We let $i_f = \frac{I_f}{N}$, $i_s = \frac{I_s}{N}$ and rewrite the model in terms of proportions and the total population:

$$(2) \quad \begin{cases} \frac{di_f}{dt} &= (i_s - \sigma)bi_f + (1-\sigma)\Phi(N)(1 - i_f - i_s)(i_f + i_s), \\ \frac{di_s}{dt} &= \sigma b i_f - (1 - i_s)bi_s + \sigma\Phi(N)(1 - i_s - i_f)(i_f + i_s), \\ \frac{dN}{dt} &= [b(1 - i_s) - (d + d_1 N)]N \end{cases}$$

We analyze the model by substituting $\Phi(N)$ with the standard incidence, mass-action incidence and asymptotic incidence transmission terms. We then return to the model in the general case.

3. The one-sex model with standard incidence transmission

The model introduced in [1] is

$$(3) \quad \begin{cases} \frac{dS}{dt} &= bS - \beta\frac{S}{N}(I_f + I_s) - (d + d_1 N)S, \\ \frac{dI_f}{dt} &= (1-\sigma)bI_f + (1-\sigma)\beta\frac{S}{N}(I_f + I_s) - (d + d_1 N)I_f, \\ \frac{dI_s}{dt} &= \sigma b I_f + \sigma\beta\frac{S}{N}(I_f + I_s) - (d + d_1 N)I_s. \end{cases}$$

Where

$$N = S + I_f + I_s.$$

The meaning of the variables and parameters is as follows

- S, I_f and I_s are the susceptible, infected fertile and sterile individuals respectively
- b: the per-capita birth rate
- β: infection rate
- σ: the probability of sterilization at the moment of infection
- $0 < \delta < 1$: the reduction factor of the natural mortality of infected sterile hosts
- $\delta d + d_1 N$: the logistic mortality rate

We let $\delta = 1$, $i_f = \frac{I_f}{N}$, $i_s = \frac{I_s}{N}$ and rewrite the model in terms of proportions:

$$\begin{cases} \frac{di_f}{dt} &= (i_s - \sigma)bi_f + (1-\sigma)\beta(1 - i_f - i_s)(i_f + i_s), \\ \frac{di_s}{dt} &= \sigma bi_f + (i_s - 1)bi_s + \sigma\beta(1 - i_s - i_f)(i_f + i_s). \end{cases} \tag{4}$$

The model (4) has three steady states:

$$(\bar{i}_f, \bar{i}_s) = (0, 0),$$

$$(\tilde{i}_f, \tilde{i}_s) = (0, 1) \quad \text{and}$$

$$(\hat{i}_f, \hat{i}_s) = (1 - \sigma, \sigma)\,.$$

Concerning this model we establish the following result:

THEOREM 3.1.

- *If $\beta < b\sigma$, then $(\bar{i}_f, \bar{i}_s)$ is locally asymptotically stable.*
- *$(\tilde{i}_f, \tilde{i}_s)$ is always unstable.*
- *If $\beta > b\sigma$ then $(\hat{i}_f, \hat{i}_s)$ is locally asymptotically stable.*

Proof. Denoting $J(i_f, i_s)$ the Jacobian of (4) we have the following results concerning its eigenvalues

- $J(0,0)$ has eigenvalues $-b < 0$ and $-\sigma b + \beta$, which is negative if $\beta < b\sigma$.
- $J(0,1)$ has eigenvalues $b(-\sigma + 1)$ and $b - \beta$. The equilibrium point will be unstable since $b(1-\sigma) > 0$.
- $J(1-\sigma, \sigma)$ has eigenvalues $-(1-\sigma)b < 0$ and $b\sigma - \beta$ which is negative if $\beta > b\sigma$.

□

Now we will compute the total population limit at each of these equilibrium points. Notice first that the equation for N can be written as

$$N' = [b(1-y) - d - d_1 N]N$$

which is an asymptotically autonomous differential equation whose limiting equation is

$$N' = [b(1-y^*) - d - d_1 N]N$$

where y^* denotes the limit of $y(t)$. Thus, according to the theorem above, if $y \to 0$ then $N \to K := \frac{b-d}{d_1}$ and the control effectiveness is $E = 0$. Otherwise, if $y \to \sigma$ then the population either goes extinct or it approaches a positive steady state which is decreasing with σ. We summarize these results below

- if $\beta < b\sigma$ then $N^* = K$
- if $\beta > b\sigma$ and $b(1-\sigma) > d$ then $N^* = \frac{b(1-\sigma)-d}{d_1}$
- if $\beta > b\sigma$ and $b(1-\sigma) < d$ then $N^* = 0$

Remark 3.1. *We note here that in the last case we have disease induced extinction. This, of course, is due to sterility of infected hosts since the disease is assumed without additional mortality.*

Below we provide a contour plot of various levels of control effectiveness that can be achieved for a range of β and σ values.

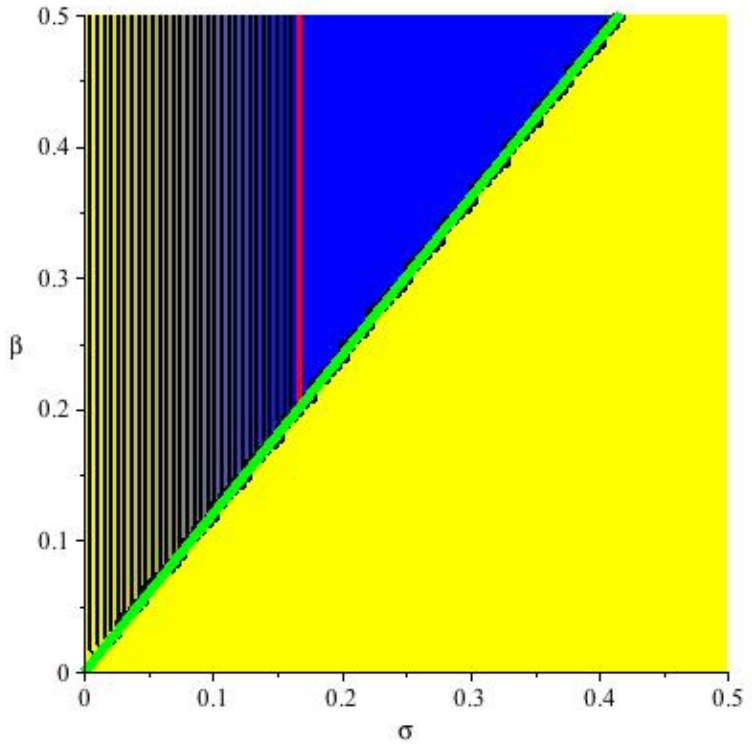

FIGURE 1. The control effectiveness E is plotted in respect to the parameters σ and β. The black contour lines represent given control effectiveness values. Blue denotes a control effectiveness closer to one, while yellow denotes a control effectiveness closer to zero. We let $b = 1.2$, $d = 1$ and $d_1 = 0.01$. The red curve marks the boundary $b(1-\sigma) = d$, which separates the susceptible extinction state and the endemic state. The green curve marks the boundary $\beta = b\sigma$, below which exists the disease free state.

4. The one-sex model with mass-action incidence

$$
(5) \qquad \begin{cases} \frac{dS}{dt} &= bs - \beta S(I_f + I_s) - (d + d_1 N)S, \\ \frac{dI_f}{dt} &= (1-\sigma)bI_f + (1-\sigma)\beta S(I_f + I_s) - (d + d_1 N)I_f, \\ \frac{dI_s}{dt} &= \sigma b I_f + \sigma\beta S(I_f + I_s) - (d + d_1 N)I_s. \end{cases}
$$

Where $N = S + I_f + I_s$.

The model has four steady states.

$$(\bar{S}, \bar{I}_f, \bar{I}_s) = (0, 0, 0),$$

$$(\tilde{S}, \tilde{I}_f, \tilde{I}_s) = (K, 0, 0)\,,$$

$$(\hat{S}, \hat{I}_f, \hat{I}_s) = \left(0, \frac{(1-\sigma)[b(1-\sigma)-d]}{d_1}, \frac{\sigma[b(1-\sigma)-d]}{d_1}\right) \quad \text{and}$$

$$(S^*, I_f^*, I_s^*) = \left(\frac{b\sigma d_1 - \beta[b(1-\sigma)-d]}{\beta^2}, \frac{(1-\sigma)[\beta(b-d)-b\sigma d_1]}{\beta^2}, \frac{\sigma[\beta(b-d)-b\sigma d_1]}{\beta^2}\right).$$

Below we provide the theorem concerning the local stability conditions of these equilibrium points.

THEOREM 4.1.

- *If $b < d$ then the extinction equilibrium is locally asymptotically stable and no intervention is necessary (i.e. the host population goes extinct due to low reproduction rate).*
- *If $\beta < \frac{\sigma b}{K}$, then the disease free equilibrium $(\tilde{S}, \tilde{I}_f, \tilde{I}_s)$ is asymptotically stable and the control effectiveness is $E = 0$.*
- *If $d < b(1-\sigma)$ and $\beta > \frac{\sigma b d_1}{b(1-\sigma)-d}$, then the equilibrium $(\hat{S}, \hat{I}_f, \hat{I}_s)$ is feasible, asymptotically stable and the control effectiveness is $E = \frac{\sigma b}{b-d}$.*
- *If either*

  $$(1-\sigma)b < d < b \text{ and } \beta > \frac{\sigma b d_1}{b-d}$$

  or

  $$(1-\sigma)b > d \text{ and } \left(\frac{\sigma b d_1}{b-d} < \beta < \frac{\sigma b d_1}{(1-\sigma)b-d}\right)$$

  then (S^*, I_F^*, I_S*) exists, is stable and has control effectiveness $E = 1 - \frac{\sigma b}{\beta K}$.

Proof.

- $J(0,0,0)$ has eigenvalues $-d$, $b-d$ and $-d+b(1-\sigma)$, which are all negative when $b < d$.
- $J\left(\tilde{S}, \tilde{I}_f, \tilde{I}_s\right)$ has eigenvalues $-b$, $d-b$ and $\frac{\beta(b-d)-\sigma b d_1}{d_1}$, which are all negative when $b > d$ and $\beta < \frac{\sigma b}{K}$.
- $J\left(\hat{S}, \hat{I}_f, \hat{I}_s\right)$ has eigenvalues $-b(1-\sigma)$, $d-(1-\sigma)b$ and $\frac{[-(1-\sigma)b+d]\beta+\sigma b d_1}{d_1}$. The equilibrium is feasible when $b(1-\sigma) > d$ and all eigenvalues are negative when

  $$d < b(1-\sigma) \text{ and } \beta > \frac{\sigma b d_1}{b(1-\sigma)-d}$$

- Finally we notice that there are two alternative feasibility conditions for the interior equilibrium (S^*, I_f^*, I_s^*): first when

  $$d > b(1-\sigma) \text{ and } \beta > \frac{\sigma b d_1}{b-d}$$

  or when

$$d < b(1-\sigma) \text{ and } \frac{\sigma b d_1}{b-d} < \beta < \frac{\sigma b d_1}{(1-\sigma)b-d}$$

We show now that the interior equilibrium is locally asymptotically stable whenever it exists in the biological feasible region defined above:
$J\left(S^*, I_f^*, I_s^*\right)$ has eigenvalues $\frac{-\beta d - \sigma b d_1}{\beta}$ and the roots of the following quadratic equation in λ

$$(-\beta^2)\lambda^2 - (\beta b \sigma d_1)\lambda + \{[(1-\sigma)b-d]\beta - \sigma b d_1\}[(b-d)\beta - \sigma b d_1]$$

It is straightforward to verify that the sum of the two roots is negative and the product is positive under the feasibility conditions which implies that all eigenvalues have negative real part.

□

Remark 4.1. *The first feasibility condition provides a region in the parameter space where the endemic equilibrium is stable with an unbounded infection rate β. Moreover, from the expression of the control effectiveness we conclude that, at the endemic equilibrium, while a higher infection rate β provides a better reduction of the host population, a larger sterilization σ is in fact detrimental since E actually decreases with σ. This is because, the endemic equilibrium is stable only when the "source" rates into the fertile infected population, $b(1-\sigma)$ and $(1-\sigma)\beta$ have an upper bound. In other words, I_f has a lower replacement rate than I_s.*

Below we provide again a contour plot with various values of the control effectiveness that illustrate this theorem.

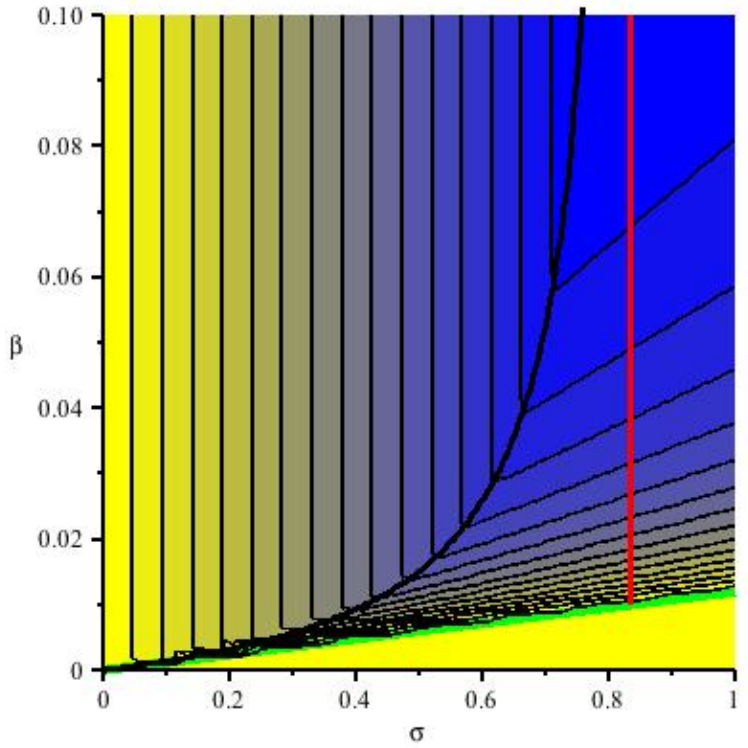

FIGURE 2. The control effectiveness E is plotted in respect to the parameters σ and β. The black contour lines represent given control effectiveness values. Blue denotes a control effectiveness closer to one, while yellow denotes a control effectiveness closer to zero. We let $b = 6$, $d = 1$ and $d_1 = 0.01$. The red curve marks the boundary $b(1-\sigma) = d$, which separates the susceptible extinction state and the endemic state. The green curve marks the boundary $\beta = \frac{b(1-\sigma)}{K}$, below which exists the disease free state.

5. The one-sex model with asymptotic incidence

$$
(6) \qquad \begin{cases} \frac{dS}{dt} &= bS - \beta\frac{S(I_f+I_s)}{c+N} - (d+d_1N)S, \\ \frac{dI_f}{dt} &= (1-\sigma)bI_f + (1-\sigma)\beta\frac{S(I_f+I_s)}{c+N} - (d+d_1N)I_f, \\ \frac{dI_s}{dt} &= \sigma bI_f + \sigma\beta\frac{S(I_f+I_s)}{c+N} - (d+d_1N)I_s. \end{cases}
$$

Where

$$N = S + I_f + I_s \text{ and } c > 0.$$

The system of equations has four steady states.

$$(\bar{S}, \bar{I}_f, \bar{I}_s) = (0, 0, 0),$$

$$(\tilde{S}, \tilde{I}_f, \tilde{I}_s) = (K, 0, 0)\,,$$

$$(\hat{S}, \hat{I}_f, \hat{I}_s) = \left(0, \frac{(1-\sigma)[(1-\sigma)b - d]}{d_1}, \frac{\sigma[(1-\sigma)b - d]}{d_1}\right)$$

and (S^*, I_f^*, I_s^*) with

$$S^* = \frac{c\{\sigma b[cd_1 - d + b(1-\sigma)] + \beta[d - b(1-\sigma)]\}}{(\sigma b - \beta)^2}$$

$$I_f^* = \frac{(1-\sigma)c[(b-d)\beta - (cd_1 + b - d)\sigma b]}{(\sigma b - \beta)^2}$$

$$I_s^* = \frac{c\sigma[(b-d)\beta - (cd_1 + b - d)\sigma b]}{(\sigma b - \beta)^2}.$$

Concerning the stability of these equilibria we establish the following theorem

THEOREM 5.1.

- *If $b < d$, then the equilibrium $(\bar{S}, \bar{I}_f, \bar{I}_s)$ is locally asymptotically stable and the host population dies out. In the following cases we assume $b > d$.*
- *If $\beta < \frac{(b-d+cd_1)\sigma b}{b-d}$, then $(\tilde{S}, \tilde{I}_f, \tilde{I}_s)$ is locally asymptotically stable and the control effectiveness is $E = 0$.*
- *If $d < (1-\sigma)b$ and $\beta > \frac{[(1-\sigma)b + cd_1 - d]\sigma b}{(1-\sigma)b - d}$, then $\left(\hat{S}, \hat{I}_f, \hat{I}_s\right)$ exists, is locally stable and the control effectiveness is $E = \frac{\sigma b}{b-d}$*
- *If either*

$$b(1-\sigma) < d \text{ and } \beta > \frac{b\sigma(b - d + cd_1)}{b - d}$$

or

$$b(1-\sigma) > d \text{ and } \frac{b\sigma(b - d + cd_1)}{b - d} < \beta < \frac{b\sigma[b(1-\sigma) - d + cd_1]}{b(1-\sigma) - d}$$

then (S^*, I_f^*, I_s^*) is feasible, locally stable and the control effectiveness is $E = 1 - \frac{cb\sigma d_1}{(-b\sigma + \beta)(b-d)}$.

Proof.

- $J(\bar{S}, \bar{I}_f, \bar{I}_s)$ has three eigenvalues: $-d$, $b - d$ and $(1-\sigma)b - d$, which are all negative when $b < d$.
- $J(\tilde{S}, \tilde{I}_f, \tilde{I}_s)$ has three eigenvalues: $-b$, $d - b$ and $\frac{\beta(b-d) - b\sigma(b-d+cd_1)}{b-d+cd_1}$, which are all negative when

$$\beta < \frac{(b - d + cd_1)\sigma b}{b - d}$$

- $J(\hat{S}, \hat{I}_f, \hat{I}_s)$ is feasible when $b(1-\sigma) > d$. $J(\hat{S}, \hat{I}_f, \hat{I}_s)$ has three eigenvalues: $-(1-\sigma)b$, $d - (1-\sigma)b$ and

$$\frac{\beta[d - b(1-\sigma)] + \sigma b[b(1-\sigma) - d + cd_1]}{b(1-\sigma) - d + cd_1}$$

which are all negative when

$$b(1-\sigma) > d \text{ and } \beta > \frac{[b(1-\sigma) - d + cd_1]\sigma b}{b(1-\sigma) - d}$$

- (S^*, I_f^*, I_s^*) exists when

$$b(1-\sigma) < d \text{ and } \beta > \frac{b\sigma(b - d + cd_1)}{b - d}$$

or

$$b(1-\sigma) > d \text{ and } \frac{b\sigma(b - d + cd_1)}{b - d} < \beta < \frac{b\sigma[cd_1 - d + b(1-\sigma)]}{b(1-\sigma) - d}$$

The characteristic equation of $J(S^*, I_f^*, I_s^*)$ has three eigenvalues: $\lambda_1 = \frac{d_1\sigma bc + d(\beta - \sigma b)}{\sigma b - \beta}$ and λ_2 and λ_3, the roots of

$$(\beta - \sigma b)\beta\lambda^2 + \sigma b c d_1 \beta\lambda + [(b - d + cd_1)\sigma b - \beta(b-d)]\{[d - b(1-\sigma) - cd_1]\sigma b + \beta[b(1-\sigma) - d]\}$$

First notice that, from the lower bound on β at the existence conditions, it follows that $\beta > b\sigma$. This ensures that λ_1 is negative. From the quadratic above, we also conclude that

$$\lambda_2 + \lambda_3 < 0 \text{ and } \lambda_2\lambda_3 > 0$$

under the existence conditions. Thus all eigenvalues have negative real parts.

□

Remark 5.1. *In this case, we again have two alternative conditions of existence for the endemic equilibrium. Under one set of conditions, the infection rate can become arbitrarily large without causing susceptible extinction. Moreover, just like in the mass-action incidence we notice that, at the endemic equilibrium, further increasing the sterilization rate σ is actually detrimental toward improving the control effectiveness.*

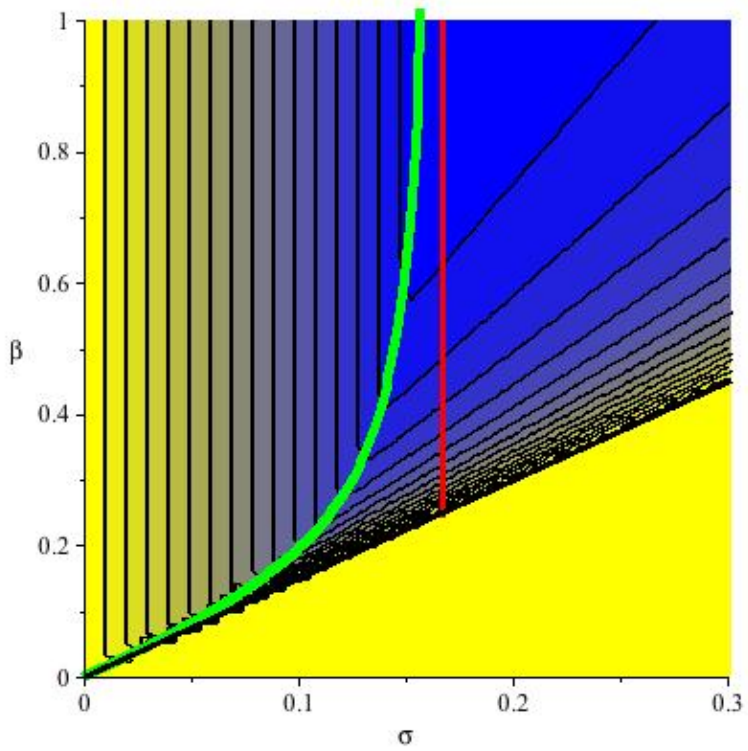

FIGURE 3. The control effectiveness E is plotted in respect to the parameters σ and β. The black contour lines represent given control effectiveness values. Blue denotes a control effectiveness closer to one, while yellow denotes a control effectiveness closer to zero. We let $b = 1.2$, $d = 1$, $d_1 = 0.1$ and $c = 5$ The green curve marks the boundaries $b(1-\sigma) = d$ and $\beta = \frac{[(1-\sigma)b+cd_1-d]\sigma b}{(1-\sigma)b-d}$, which separate the susceptible extinction state, the bounded endemic state and the unbounded endemic state. The black line marks the boundary $\beta = \frac{(b-d+cd_1)\sigma b}{b-d}$, below which exists the disease free state.

Remark 5.2. *It is due to vertical transmission that we observe the inverse correlation between the sterility rate σ and the control effectiveness E at the endemic equilibrium. In the following theorems we show that this statement is the case for both mass-action and asymptotic incidence.*

Consider the original model (1) without vertical transmission. In proportions,

$$(7) \qquad \begin{cases} \frac{di_f}{dt} &= (1-\sigma)\Phi(N)(1-i_f-i_s)(i_f+i_s), \\ \frac{di_s}{dt} &= (1-i_s)bi_s + \sigma\Phi(N)(1-i_s-i_f)(i_f+i_s), \\ \frac{dN}{dt} &= [b(1-i_s)-(d+d_1N)]N \end{cases}$$

From Berec and Maxin's Proposition 7 [1], we know that the endemic equilibrium of (7) exists and is unique if and only if the reproductive number $R_0 > 1$, where $R_0 = \frac{\Phi(K)}{b}$.

THEOREM 5.2. *Under both mass-action incidence and asymptotic incidence transmission, the total population N^* at the endemic equilibrium is a decreasing function of the sterility rate σ.*

Proof. Consider $\Phi(N) = \beta N$. At the endemic equilibrium, the total population

$$N^* = \frac{1}{2d_1\beta}\left([(1-\sigma)b - d]\beta + d_1\sigma b + \right.$$
$$\left. + \sqrt{[d - b(1-\sigma)]^2\beta^2 + 2\sigma b d_1 \beta[(1-\sigma)b + d] + {d_1}^2\sigma^2 b^2}\right).$$

After a lengthy, but straightforward computation we found that $\frac{dN^*}{d\sigma}$ is negative whenever

$$\beta > d_1 \text{ and } \beta(b-d) > bd_1.$$

These conditions follow immediately from $R_0 > 1$, which, under mass-action incidence, is $\beta(b-d) > bd_1$.

Consider $\Phi(N) = \frac{\beta N}{c+N}$. At the endemic equilibrium, the total population

$$N^* = \frac{1}{(-\beta + \sigma b)2d_1}\left([(\beta - d - cd_1)\sigma - \beta]b + d\beta + \right.$$
$$+[\{[\beta^2 - (cd_1 + d)2\beta + (cd_1 - d)^2]\sigma^2 + (-\beta + d + cd_1)2\beta\sigma + \beta^2\}b^2 +$$
$$\left. +[(\beta - d + cd_1)\sigma - \beta]2\beta d b + d^2\beta^2]^{1/2}\right).$$

Similarly, we found that $\frac{dN^*}{d\sigma}$ is negative whenever

$$\beta - cd_1 - b > 0 \text{ and } b^2 - b(\beta - cd_1 + d) + \beta d < 0.$$

These conditions follow immediately from $R_0 > 1$, which, under asymptotic incidence, is $\beta(b-d) > b(b - d + cd_1)$. □

6. The one-sex model with general transmission term $\Phi(N)$

Recall models (1) and (2). The system of equations in model (2) has an endemic state

$$(i_f^*, i_s^*, N^*)$$

with

$$i_f^* = \left(\frac{1-\sigma}{\sigma}\right)\left[1 - \left(\frac{d + d_1 N^*}{b}\right)\right]$$
$$i_s^* = 1 - \left(\frac{d + d_1 N^*}{b}\right)$$
$$\Phi(N^*) = b\sigma.$$

Concerning the stability of the endemic equilibrium we establish the following theorem

THEOREM 6.1. *If $\Phi(N^*) = b\sigma$ has a solution, $\frac{(1-i_s^*)b-d}{2d_1} < N < \frac{b-d}{d_1}$ and $\frac{d\Phi}{dt}(N^*) > 0$, then (i_f^*, i_s^*, N^*) is feasible and locally stable.*

Proof. (i_f^*, i_s^*, N^*) exists when $1 > \frac{d+d_1N}{b}$.

The characteristic equation of $J(i_f^*, i_s^*, N^*)$ has three eigenvalues: $\lambda_1 = -(1-i_s^*)b$ and λ_2 and λ_3, the roots of

$$-\sigma\lambda^2 - \sigma[d + 2d_1N^* - (1-i_s^*)b]\lambda - bN^*i_s^*\frac{df}{dt}(N^*)(\sigma + i_s^*).$$

The eigenvalues are negative when $(1-i_s^*)b < d + 2d_1N$ and $\frac{d\Phi}{dt}(N^*) > 0$. □

7. The two-sex model

$$(8)\quad \begin{cases} S_f' &= \beta\gamma_f\frac{S_fS_m}{P} - \lambda\frac{S_f(I_m+J_m)}{P} - \bar{\mu}_fS_f, \\ S_m' &= \beta\gamma_m\frac{S_fS_m}{P} - \lambda\frac{S_m(I_f+J_f)}{P} - \bar{\mu}_mS_m, \\ I_f' &= (1-\sigma)\beta\gamma_f\frac{S_fI_m+S_mI_f+I_fI_m}{P} + (1-\sigma)\lambda\frac{S_f(I_m+J_m)}{P} - \bar{\mu}_fI_f, \\ I_m' &= (1-\sigma)\beta\gamma_m\frac{S_fI_m+S_mI_f+I_fI_m}{P} + (1-\sigma)\lambda\frac{S_m(I_f+J_f)}{P} - \bar{\mu}_mI_m, \\ J_f' &= \sigma\beta\gamma_f\frac{S_fI_m+S_mI_f+I_fI_m}{P} + \sigma\lambda\frac{S_f(I_m+J_m)}{P} - \bar{\mu}_fJ_f, \\ J_m' &= \sigma\beta\gamma_m\frac{S_fI_m+S_mI_f+I_fI_m}{P} + \sigma\lambda\frac{S_m(I_f+J_f)}{P} - \bar{\mu}_mJ_m. \end{cases}$$

Where

$$P = S_f + S_m + I_f + I_m + J_f + J_m \text{ and } \bar{\mu} = \mu + bP.$$

We assume equal gender parameters: $\mu_f = \mu_m = \mu$, $\gamma_f = \gamma_m = \frac{1}{2}$, $J_f = J_m = \frac{J}{2}$, $S_f = S_m = \frac{S}{2}$, and $I_f = I_m = \frac{I}{2}$. We then reduce the system to three equations.

$$(9)\quad \begin{cases} S' &= \beta\frac{S^2}{S+I+J} - \lambda\frac{S(I+J)}{S+I+J} - \bar{\mu}S, \\ I' &= \beta(1-\sigma)\frac{I^2+2SI}{S+I+J} + \lambda(1-\sigma)\frac{S(I+J)}{P} - \bar{\mu}I, \\ J' &= \beta\sigma\frac{I^2+2SI}{S+I+J} + \lambda\sigma\frac{S(I+J)}{P} - \bar{\mu}J. \end{cases}$$

Where P=S+I+J.

We let $i = \frac{I}{P}$, $j = \frac{J}{P}$ and $1 - i - j = \frac{S}{P}$ and rewrite the model in terms of proportions.

$$\begin{cases} i' &= \beta i[(1-\sigma)(2-i-2j) - (1-j)^2] + \lambda(1-\sigma)(1-i-j)(i+j), \\ j' &= \beta[i\sigma(2-i-2j) - j(1-j)^2] + \lambda\sigma(1-i-j)(i+j). \end{cases} \tag{10}$$

The system of equations has four steady states.

$$(\bar{i}, \bar{j}) = (0, 0),$$

$$(\tilde{i}, \tilde{j}) = (0, 1)\,,$$

$$(\hat{i}, \hat{j}) = (1-\sigma, \sigma) \quad \text{and}$$

$$(i^*, j^*) = \left(\frac{[\beta(2\sigma-1) - \lambda](1-\sigma)}{\beta\sigma^2}, \frac{\beta(2\sigma-1) - \lambda}{\beta\sigma} \right).$$

THEOREM 7.1. *Stability Conditions of Equilibria*

- *If* $\lambda < \beta(2\sigma - 1)$ *and* $\sigma > \frac{1}{2}$, *then the equilibrium* $(\bar{i}, \bar{j}) = (0, 0)$ *exists, is stable and has control effectiveness* $E = 0$.
- *The equilibrium* $(\tilde{i}, \tilde{j}) = (0, 1)$ *is unstable whenever it exists.*
- *The equilibrium* $(\hat{i}, \hat{j}) = (1 - \sigma, \sigma)$ *is stable whenever it exists and has control effectiveness* $E = \frac{\sigma(2-\sigma)}{\beta - \mu}$.
- *If* $\lambda > \beta(2\sigma - 1)$, *then the equilibrium* $(i^*, j^*) = \left(\frac{[\beta(2\sigma-1)-\lambda](1-\sigma)}{\beta\sigma^2}, \frac{\beta(2\sigma-1)-\lambda}{\beta\sigma} \right)$ *exists and is unstable.*

Proof.

- $J(\bar{i}, \bar{j})$ has eigenvalues $\lambda_1 = -\beta$ and $\lambda_2 = \lambda - \beta(2\sigma - 1)$, which are negative when $\lambda < \beta(2\sigma - 1)$.
- $J(\tilde{i}, \tilde{j})$ has eigenvalues $\lambda_1 = 0$ and $\lambda_2 = -\lambda$, so the equilibrium is unstable whenever it exists.
- $J(\hat{i}, \hat{j})$ has eigenvalues $\lambda_1 = -\beta(1-\sigma)^2$ and $\lambda_2 = -\beta(1-\sigma)^2 - \lambda$, which are always negative.
- (i^*, j^*) is feasible when $(2\sigma - 1)\beta - \lambda > 0$. $J(i^*, j^*)$ has eigenvalues $\lambda_1 = -\frac{[-(1-\sigma)\beta - \lambda]^2}{\beta\sigma^2}$ and $\lambda_2 = \frac{[(2\sigma-1)\beta - \lambda][\beta(1-\sigma)^2 + \lambda]}{\beta\sigma^2}$, one of which is positive when the equilibrium is feasible.

□

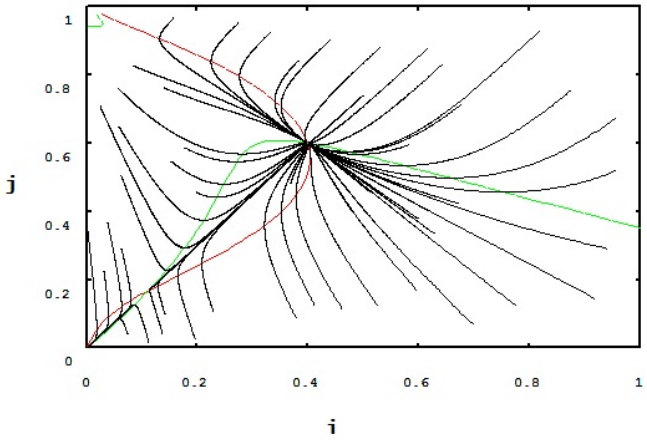

FIGURE 4. The bistability of the system in terms of i, the proportion of infected fertile individuals and j, the proportion of the infected sterile individuals. The green and red curves represent the nullclines. The intersections of the nullclines represent equilibria, two of which exhibit local stability and one which is unstable. We let $\sigma = 0.6$, $\beta = 0.4$ and $\lambda = 0.04$.

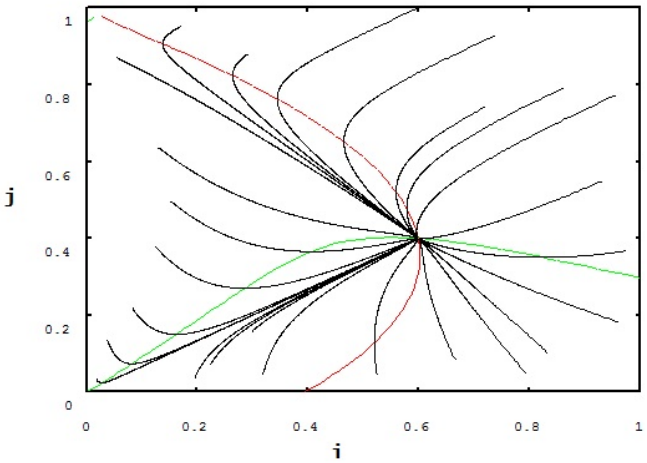

FIGURE 5. The stability of the susceptible extinction equilibria in terms of i, the proportion of infected fertile individuals and j, the proportion of the infected sterile individuals. The green and red curves represent the nullclines. We let $\sigma = 0.4$, $\beta = 0.4$ and $\lambda = 0.04$.

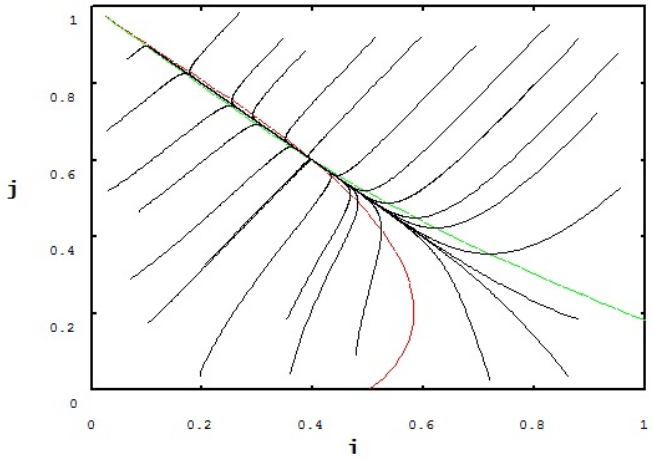

FIGURE 6. The stability of the susceptible extinction equilibria in terms of i, the proportion of infected fertile individuals and j, the proportion of the infected sterile individuals. The green and red curves represent the nullclines. We let $\sigma = 0.6$, $\beta = 0.4$ and $\lambda = 0.8$.

8. Conclusions

In the one-sex case, we noticed similar behavior between the models with mass-action and asymptotic incidence transmission terms. Both models contain stable endemic equilibria and, under certain conditions of the parameters, the infection rate can become arbitrarily large without causing susceptible extinction. However, this result did not hold under this standard incidence model, which lacked an endemic equilibrium entirely. We investigated the properties of the transmission term that lead to a stable endemic equilibrium. Our analysis indicate that a transmission term dependent on the total population will give a stable endemic equilibrium. This result supports our previous results that the models with mass-action incidence and asymptotic incidence transmission terms have endemic equilibria since both are dependent upon the total population. The model with a standard incidence transmission term, however, does not give an endemic equilibrium since the transmission term is independent of the population.

We also noticed similar behavior in control effectiveness between the models with mass-action and asymptotic incidence transmission terms. At the susceptible extinction equilibrium, the control effectiveness increases with the sterility rate and is independent of the infection rate. This result makes sense biologically as increasing the infection rate in a population with no susceptibles has no impact on the system. Therefore, the only way to reduce the population is to increase the proportion of the population that become sterile upon infection.

At the endemic equilibrium we found that the control effectiveness increases with the infection rate, but decreases with the sterility rate. We showed that the latter is a result of vertical transmission. In our model, pests can become sterile by infection through sexual transmission or when

born from an infected parent. In order to optimize the control effectiveness at the endemic equilibrium, we must consider both pathways. With a large sterility rate, a smaller portion of infected individuals will reproduce, causing a decrease in the number of infected individuals in the population. This decrease in the infected class leads to lower infection rates. An increased infection rate improves the control effectiveness by increasing the population of sterile individuals.

In the one-sex model with a standard incidence transmission term, the control effectiveness behaves similarly at the susceptible extinction equilibrium, by increasing with the sterility rate and remaining independent of the infection rate. However, the standard incidence model does not contain an endemic equilibrium. The disease free equilibrium, in each case, has a control effectiveness value of zero, since the sterilizing pathogen dies out and has no long-term effect on the host population.

In the two-sex model, the system showed bistability under certain conditions of the parameters. The possibility of bistability between the disease free equilibrium and the susceptible extinction equilibrium means that pest control measures could be ineffective if the initial proportions of infected individuals are too low. In a realistic introduction of a sterilizing pathogen into a host population it is reasonable to assume that the initial proportions of infected individuals will be low. Therefore, negating one of the conditions under which bistability occurs will prevent the system from going to a disease free state when the sterilizing pathogen is introduced.

For a more complete analysis, future research could include an investigation of a more realistic version of the two-sex model in which the gender parameters are not assumed to be equal. Another modification to explore is the effect of partial vertical transmission, which is a common characteristic of sexually transmitted diseases.

Acknowledgements

This work was partially supported by NSF Grant DMS-0851721.

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