Double impact of sterilizing pathogens: added value of increased life expectancy on pest control effectiveness

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Abstract Sterilizing pathogens are commonly assumed not to affect longevity of infected individuals, and if they do then negatively. Examples abound, however, of species in which the absence of reproduction actually increases life expectancy. This happens because by decreasing the energy outlay on reproduction individuals with lowered reproduction can live longer. Alternatively, fertile individuals are more susceptible to predators or parasitoids if the latter can capitalize on mating signals of the former. Here we develop and analyze an SI epidemiological model to explore whether and to what extent does such a life expectancy prolongation due to sterilizing pathogens affect host dynamics. In particular, we are interested in an added value of increased life expectancy on the possibility of successful pest control, that is, the effect of increased lifespan and hence increased potential of the infected individuals to spread the disease on pest control effectiveness. We show that although the parameter range in which we observe an effect of increased lifespan of the sterilized individuals is not large, the effect itself can be significant. In particular, the increase in pest control effectiveness can be very dramatic when disease transmission efficiency is close to birth rate, mortality rate of susceptibles is relatively high (i.e., the species is relatively short-lived), and sterilization efficiency is relatively high. Our results thus characterize pathogens that are promising candidates for an effective pest control and that might possibly be engineered if not occurring naturally.

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1 Introduction

International travel and trade are the major drivers of an unprecedented increase in the rate of non-native species invasions (Mack et al 2000; Lockwood et al 2007). While most such invasions turn out to eventually be unsuccessful (Willamson and Fitter 1996; Simberloff and Gibbons 2004), those few that succeed impose immense environmental, social and economic damages (Courchamp et al 2003; Tobin et al 2009). Need for control of such successful invasive species thus arises, with armies of pest managers equipped with a diversity of weapons, often specific to the focal species (Thacker 2002; Courchamp et al 2003).

Generally, control methods aim to enhance species mortality rate and/or decrease its fertility rate. A substantial intellectual effort has been invested into which of these strategies is more effective. Using an optimal control approach, Stenseth (1981) suggested that fertility reduction should be increasingly preferred the larger is the mortality of the uncontrolled population. On the contrary, if the mortality rate of the uncontrolled population is low, the optimal pest control strategy should often aim at increasing mortality as much as possible. The mating system and the way density dependence operates appear to also play a significant role (Barlow et al 1997). Reviewing fertility control by chemical or surgical means, Dell'Omo and Palmery (2002) concluded that "an ideal fertility control strategy should induce long-term or permanent sterility without secondary toxic effects such as behavioral alterations, be target-specific, and act on both sexes."

Parallel to this effort, discussions have been under way relating efficacy of the bait delivery and the cost-effectiveness of the operations (Courchamp et al 2003). Concerns have been raised that areas which are large, difficult to access, or have a low pest density are extremely difficult if not impossible to cover by hunters, traps or poisonous baits. As dissemination of control agents constitutes a major limitation in many control programs, research has focused on biological control. Biological control relies upon self-disseminating natural enemies such as viruses which may have the double advantage of economic viability and high control success. However, use of most pathogens is unethical as they inflict unnecessary suffering before killing the host (Courchamp et al 2003).

Sterilizing effects are characteristic of a variety of plant pathogens, parasitic castrators of invertebrates, and sexually transmitted diseases (Baudoin 1975; O'Keefe and Antonovics 2002; Antonovics 2009, and references therein). If no such pathogen is available to a pest species, it could possibly be engineered. Indeed, research effort has recently turned towards virus-vectored immunocontraception (VVIC), a new form of biological control that retains the advantages of self-dissemination of control agents while avoiding the unethical aspects of animal suffering. VVIC is based on a sterilization process that induces the immune system of an individual to attack its own reproductive cells – infecting an individual with a protein derived from the follicular layers activates production

of antibodies against its own gametes, thereby blocking fertilization (Tyndale-Biscoe 1994; Bradley et al 1997). VVIC agents are viruses that are genetically modified to carry a gene encoding the reproductive protein of a target species (Tyndale-Biscoe 1994). The use of modified, species-specific viruses thus allows for an efficient dissemination of a control agent through a pest population regardless of its area of distribution, accessibility and density, and combines the advantages of high specificity and optimal dissemination. This potentially powerful technique appears most appropriate for rodents and small herbivores, such as rabbits and possums (Cowan 1996; McLeod and Twigg 2006; Rodger 1997; Smith et al 1997), yet it could also be very efficient for control of small carnivores such as cats and foxes (Bradley et al 1997; Courchamp and Cornell 2000; Pech et al 1997; Verdier et al 1999).

Since the publication of seminal review articles by Anderson and May (Anderson and May 1979, 1981), very many host-parasite models with pathogens inducing reduction of host reproduction in general and sterilizing pathogens in particular have been developed. Yet, none of these models has ever accounted for a well-known aspect of life-history theory, namely the cost of reproduction or a trade-off between reproduction and survival – by decreasing the energy outlay on reproduction, individuals with lowered reproduction can live longer. This trade-off predicts that "non-reproducing females have a higher chance of surviving than reproducing females" (Neuhaus and Pelletier 2001), that there is "a negative relationship between the mean female fecundity and the mean [female] longevity" (Thomas et al 2000), or that "early reproduction may increase mortality to such an extent that delaying reproduction may increase survival and lifetime reproductive success" (Bennett and Owens 2002). These trade-offs have indeed been found to occur in birds (Bennett and Owens 2002), insect parasitoids (Ellers 1995; Ramesh and Manickavasagam 2003), mammals (Neuhaus and Pelletier 2001) and even humans (Thomas et al 2000).

Sterilizing pathogens can thus have a double impact on their hosts. Not only they depress host reproduction, but by letting the infected individuals live longer, these sterilized animals help spread the pathogen for a longer time which may further increase the control effectiveness. Mathematical models should always form a necessary first step to assess effectiveness of any control technique before it is accepted. Therefore, in this paper, we develop and analyze a mathematical model to address the question of whether and how much does effectiveness of releasing a sterilizing pathogen into a pest population increase if the cost of reproduction is accounted for.

2 Methods

We limit ourselves to an SI epidemiological model, assuming that as soon as susceptible individuals (S) get infected, i.e. sterilized, they remain so until they die. This also allows us to assess the full potential of increased longevity due to sterilizing pathogens on pest control effectiveness, since the infection will not be curbed by any latent or immunity period. To keep the model as simple as possible (and to eliminate any confounding factors) we also assume no disease-induced mortality. In fact, sterilizing pathogens may have little or no effect on host

mortality (O'Keefe and Antonovics 2002; Bonds 2006). Also, we assume no vertical transmission. Although some sexually transmitted infections can also be passed from parents to offspring (Lockhart et al 1996; Knell and Webberley 2004), we decided not to consider this additional transmission route here but rather to examine its effect separately. Finally, we assume that the infection does not always lead to sterilization so we distinguish infected individuals that become sterilized due to the disease (I_S) and the remaining infected individuals that are able to spread the virus yet also reproduce (I_F). As the sterilized individuals do not "waste" resources in reproduction, they are allowed to live longer than the fertile ones.

As a consequence, host dynamics are described by the following model:

$$\frac{dS}{dt} = b(S + I_F) - \Phi(N)\frac{S(I_F + I_S)}{N} - (d + d_1 N)S,$$

$$\frac{dI_F}{dt} = (1 - \sigma)\Phi(N)\frac{S(I_F + I_S)}{N} - (d + d_1 N)I_F,$$ (1)

$$\frac{dI_S}{dt} = \sigma\Phi(N)\frac{S(I_F + I_S)}{N} - (\delta d + d_1 N)I_S.$$

We thus assume that both fertile and sterile infectives are able to spread the disease equally. We have σ as the proportion of infected individuals that become sterilized ($0 < \sigma < 1$) and δ as the proportional reduction of the intrinsic mortality rate d in those infected individuals that become sterilized ($0 < \delta < 1$). For the sake of simpler analysis, the host population is assumed here regulated through negative density dependence in the mortality rate, with d_1 being its strength, but one might alternatively consider regulation through the birth rate, or a mixture of the two (Hassell 1975; Gao and Hethcote 1992). The function $\Phi(N)$ specifies the way disease is transmitted and covers both the contact rate between individual hosts and the probability of disease transmission upon an adequate contact between infectives and susceptibles. We start with a generic form of $\Phi(N)$, and later on explore the model (1) under the standard incidence ($\Phi(N) = \beta$), the mass action incidence ($\Phi(N) = \beta N$), and the asymptotic incidence ($\Phi(N) = \beta N/(c + N)$ for some $c > 0$; e.g. Diekmann and Kretzschmar (1991)) modes of disease transmission.

In the absence of disease, the total host population density N evolves as

$$\frac{dN}{dt} = bN - (d + d_1 N)N.$$ (2)

Hence, the host population has the intrinsic growth rate $r = b - d$ and attains the environmental carrying capacity $K = (b - d)/d_1$. We assume $b > d$ further on so that the host population is able to persist without the disease. In the presence of disease, we have

$$\frac{dN}{dt} = b(S + I_F) - (d + d_1 N)(S + I_F) - (\delta d + d_1 N)I_S.$$ (3)

The major question we address in this paper is how the ability of the disease to suppress the host population changes with δ, the factor that extends the life expectancy of sterilized hosts. This ability, or the control effectiveness, is evaluated here as $E = 1 - N^*/K$ where N^* is the host population density at a stable equilibrium of the model (1).

It can be $E = 0$ if the disease cannot invade, $0 < E < 1$ if an endemic equilibrium exists, or $E = 1$ if the disease is strong enough to drive the host to extinction.

For $\delta = 1$, i.e. no effect of sterilization on host longevity, the model (1) reduces to

$$\frac{dS}{dt} = b(S + (1 - \sigma)I) - \Phi(N)\frac{SI}{N} - (d + d_1 N)S,$$
$$\frac{dI}{dt} = \Phi(N)\frac{SI}{N} - (d + d_1 N)I. \tag{4}$$

Alternatively, for $\sigma = 1$, i.e. sterilization of any infected individual, the model (1) reduces to

$$\frac{dS}{dt} = bS - \Phi(N)\frac{SI}{N} - (d + d_1 N)S,$$
$$\frac{dI}{dt} = \Phi(N)\frac{SI}{N} - (\delta d + d_1 N)I. \tag{5}$$

See Appendix for results of an analysis of the models (4) and (5).

3 Results

3.1 Basic reproduction number

As is usual in virtually any study on infectious disease dynamics, we start with calculating the basic reproduction number R_0. Using the next generation matrix approach due to van den Driessche and Watmough (2002), one can show that (see Appendix)

$$R_0 = \Phi(K)\left(\frac{1 - \sigma}{b} + \frac{\sigma}{b - (1 - \delta)d}\right). \tag{6}$$

It is useful to rewrite this term using reciprocals of mortality rates to get its clearer biological interpretation:

$$R_0 = (1 - \sigma)\frac{\Phi(K)}{d + d_1 K} + \sigma\frac{\Phi(K)}{\delta d + d_1 K}. \tag{7}$$

Here, the first fraction represents the number of secondary infections caused by a reproductive infectious individual introduced into a fully susceptible population, while the second fraction represents the number of secondary infections caused by a sterile infectious individual, with σ the fraction of infectives that become sterilized.

The disease thus invades if $R_0 > 1$ or equivalently if

$$\Phi(K) > \frac{b[b - (1 - \delta)d]}{b - (1 - \sigma)(1 - \delta)d}. \tag{8}$$

R_0 increases with increasing σ (as $\partial R_0/\partial\sigma > 0$ if $b > d$) and, more importantly from our perspective, with decreasing δ (as $\partial R_0/\partial\delta < 0$ for $\sigma > 0$). Indeed, reduction of the intrinsic host mortality rate due to sterilization can revert the outcome of disease invasion – a disease that cannot invade the host population for a high δ might be able to do that for a low δ (Fig. 1).
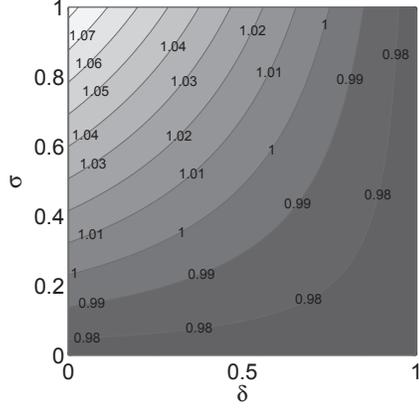
Fig. 1 Basic reproduction number R_0 of the model (1), as a function of the proportion of infected individuals that become sterilized (σ) and the proportional reduction of the intrinsic mortality rate in those infected individuals that become sterilized (δ). Other parameters values: $\Phi(K) = 1.95, b = 2, d = 0.2$

3.2 System equilibria

We start by going from densities to proportions, thus getting equations for $i_F = I_F/N$ and $i_S = I_S/N$:

$$\frac{di_F}{dt} = (1-\sigma)\Phi(N)(1-i_F-i_S)(i_F+i_S) - di_F - i_F[(b-d)(1-i_S) - \delta d i_S],$$
$$\frac{di_S}{dt} = \sigma\Phi(N)(1-i_F-i_S)(i_F+i_S) - \delta d i_S - i_S[(b-d)(1-i_S) - \delta d i_S]. \tag{9}$$

As this system contains N, it can be closed with one more equation for the total host population density

$$\frac{dN}{dt} = N[b(1-i_S) - (d+d_1N)(1-i_S) - (\delta d + d_1N)i_S]. \tag{10}$$

This system of three differential equations has several equilibrium points (i_F, i_S, N):

- $(0,0,0)$ which is an extinction equilibrium
- $(0,0,K)$ which is the disease-free equilibrium
- $(0,1,0)$ which is a disease-induced extinction equilibrium
- $(i_F^*, i_S^*, 0)$ which is another disease-induced extinction equilibrium, feasible only for transmission terms with $\Phi(0) > 0$ (only standard incidence in our case); for $\Phi(0) = 0$ and $0 < \delta < 1$, setting the right-hand sides of both equations of (9) to zero leads to two incompatible algebraic equations
- (i_F^*, i_S^*, N^*) which is an endemic equilibrium; setting the term in square brackets of the right-hand side of (10) to zero, we have

$$N^* = \frac{(b-d)(1-i_S^*) - \delta d i_S^*}{d_1} = K - i_S^* \frac{b - (1-\delta)d}{d_1} \tag{11}$$

In the last two cases, i_F^* can be expressed as a function of i_S^* by setting the right-hand sides of both equations of (9) to zero, solving both for $\Phi(N)(1 - i_F - i_S)(i_F + i_S)$ and equating the resulting expressions. This gives

$$i_F^* = i_S^* \frac{(1-\sigma)(\delta d + [(b-d)(1-i_S^*) - \delta d i_S^*])}{\sigma(d + [(b-d)(1-i_S^*) - \delta d i_S^*])} = \frac{\delta(1-\sigma)i_S^*(1-i_S^*)}{\sigma[\delta(\alpha - i_S^*) + 1 - \alpha]}, \tag{12}$$

where we have denoted

$$\alpha = \frac{1}{1 + \frac{\delta d}{b - d}} . \tag{13}$$

Note that $0 < \alpha < 1$.

We are now going to explore existence, uniqueness and stability of these equilibria for the transmission terms we consider. The underlying calculations are given as proofs of a set of propositions.

Proposition 1 *The extinction equilibrium* $(0,0,0)$ *is always unstable.*

Proof The Jacobian of the system (9) and (10) evaluated at this equilibrium is

$$\mathbf{J}(0,0,0) = \begin{pmatrix} (1-\sigma)\Phi(0) - b & (1-\sigma)\Phi(0) & 0 \\ \sigma\Phi(0) & \sigma\Phi(0) - b + (1-\delta)d & 0 \\ 0 & 0 & b - d \end{pmatrix}. \tag{14}$$

As we assume $b > d$, this matrix has at least one eigenvalue with positive real part, hence the extinction equilibrium $(0,0,0)$ is always unstable. $\square$

Proposition 2 *The disease-free equilibrium* $(0,0,K)$ *is locally stable if and only if* $R_0 < 1$ *and unstable if and only if* $R_0 > 1$.

Proof The Jacobian of the system (9) and (10) evaluated at this equilibrium is

$$\mathbf{J}(0,0,K) = \begin{pmatrix} (1-\sigma)\Phi(K) - b & (1-\sigma)\Phi(K) & 0 \\ \sigma\Phi(K) & \sigma\Phi(K) - [b - (1-\delta)d] & 0 \\ 0 & -[b-(1-\delta)d]K & -(b-d) \end{pmatrix}. \tag{15}$$

As we assume $b > d$, one eigenvalue is always negative and the other two have negative real parts if and only if

$$\Phi(K) < 2b - (1-\delta)d$$

and

$$\Phi(K) < \frac{b[b - (1-\delta)d]}{b - (1-\delta)(1-\sigma)d} .$$

Note that the second condition is equivalent to $R_0 < 1$ (8) and includes the first one, because

$$2b - (1-\delta)d - \frac{b[b-(1-\delta)d]}{b-(1-\delta)(1-\sigma)d} = \frac{[b-(1-\sigma)(1-\delta)d]^2 + \sigma(1-\sigma)d^2(1-\delta)^2}{b-(1-\delta)(1-\sigma)d} > 0.$$

Hence, the disease-free equilibrium $(0,0,K)$ is locally stable if and only if $R_0 < 1$ and unstable if and only if $R_0 > 1$.   □

Proposition 3 *The disease-induced extinction equilibrium* $(0,1,0)$ *is locally stable if and only if* $(1-\delta)d - b(1-\sigma) > 0$ *and*

$$\Phi(0) > \frac{(1-\delta)d[b-(1-\delta)d]}{(1-\delta)d - b(1-\sigma)}.$$

Proof The Jacobian of the system (9) and (10) evaluated at this equilibrium is

$$\mathbf{J}(0,1,0) = \begin{pmatrix} -\Phi(0)(1-\sigma) - (1-\delta)d & -\Phi(0)(1-\sigma) & 0 \\ -\sigma\Phi(0) & -\sigma\Phi(0) + [b-(1-\delta)d] & 0 \\ 0 & 0 & -\delta d \end{pmatrix}. \tag{16}$$

One eigenvalue is therefore always negative and the other two have negative real parts if and only if

$$\Phi(0) > b - 2(1-\delta)d,$$

$$\Phi(0) > \frac{(1-\delta)d[b-(1-\delta)d]}{(1-\delta)d - b(1-\sigma)},$$

$$(1-\delta)d - b(1-\sigma) > 0.$$

Note that the second condition includes the first one since

$$\frac{(1-\delta)d[b-(1-\delta)d]}{(1-\delta)d - b(1-\sigma)} - [b-2(1-\delta)d] = \frac{d[\delta^2(1-\sigma) + \sigma(1-\delta)^2(1-\alpha)^2]}{(1-\alpha)[\sigma(1-\alpha+\delta\alpha) - \delta]},$$

where we used the term (13) for α. The numerator is clearly positive. The denominator is also positive since

$$\sigma(1-\alpha+\delta\alpha) - \delta > 0$$

follows from

$$(1-\delta)d - b(1-\sigma) > 0.$$

So, the necessary and sufficient condition for local stability of $(0,1,0)$ is

$$(1-\delta)d - b(1-\sigma) > 0$$

and

$$\Phi(0) > \frac{(1-\delta)d[b-(1-\delta)d]}{(1-\delta)d - b(1-\sigma)}.$$

However, this is only possible if $\Phi(0) > 0$. So $(0,1,0)$, while it always exists, can be locally stable only under standard incidence (of the incidence terms we consider).   □

Corollary 1 *For both mass action and asymptotic incidences the disease-induced extinction equilibrium* $(0,1,0)$ *is always unstable, because* $\Phi(0) = 0$.

Under standard incidence, the system of two equations (9) is closed, i.e. independent of N. We are now going to explore the existence and uniqueness of its interior equilibrium (i_F^*, i_S^*).

Proposition 4 *Under standard incidence, the system* (9) *has a unique, biologically feasible endemic equilibrium,* (i_F^*, i_S^*), *if and only if one of the following two conditions is satisfied:*

$$(1-\delta)d - (1-\sigma)b > 0 \quad and \quad \frac{b[b-(1-\delta)d]}{b-(1-\delta)(1-\sigma)d} < \beta < \frac{(1-\delta)d[b-(1-\delta)d]}{(1-\delta)d - (1-\sigma)b},$$

or

$$(1-\delta)d - (1-\sigma)b < 0 \quad and \quad \frac{b[b-(1-\delta)d]}{b-(1-\delta)(1-\sigma)d} < \beta.$$

Proof Substituting i_F^* (12) into the second equation of (9) and using $\Phi(N) = \beta$ we obtain the following equation for i_S^*

$$\frac{1}{\sigma(1-\alpha)[\delta(\alpha - i_S^*) + 1 - \alpha]^2} i_S^*(i_S^* - 1)[a_2(i_S^*)^2 + a_1 i_S^* + a_0] = 0,$$

with

$$a_2 := (\delta d\sigma + \beta(-1+\alpha))\delta^2,$$

$$a_1 := 2\sigma\delta(1-\alpha+\delta\alpha)[\beta(1-\alpha) - \delta d] + \beta\delta^2(1-\alpha)(1-\sigma),$$

$$a_0 := \sigma(1-\alpha+\delta\alpha)^2(\delta d - \beta\sigma + \beta\sigma\alpha) - \beta\delta\sigma(1-\sigma)(1-\alpha)(1-\alpha+\delta\alpha).$$

We are interested in solving the quadratic equation

$$A(i_S^*) = a_2(i_S^*)^2 + a_1 i_S^* + a_0 = 0,$$

since the roots 0 and 1 correspond to the extinction and disease-free equilibria and the disease-induced extinction equilibrium, respectively. The two roots of this quadratic equation are of the form

$$m \pm n\sqrt{D},$$

with

$$D = 4\delta\beta(1-\alpha)^2(1-\sigma)^2\left(d(1-\delta)(1-\alpha+\alpha\delta)\sigma + \frac{1}{4}\beta\delta\right).$$

The existence of real roots of the quadratic equation is equivalent to $D > 0$, that is,

$$d\sigma(1-\delta)[1-\alpha(1-\delta)] + \frac{1}{4}\beta\delta > 0,$$

which is always true since $0 < \alpha < 1$ and $0 < \delta < 1$.

We first prove the uniqueness of (i_F^*, i_S^*) in the interval $(0, 1)$. Let us assume that both the roots $m \pm n\sqrt{D}$ lie in the interval $(0, 1)$. This assumption leads to two cases:

(a) $0 < m < \frac{1}{2}$ and $n^2 D < m^2$,

(b) $\frac{1}{2} < m < 1$ and $n^2 D < (1-m)^2$.

The following quantities will characterize these inequalities:

$$m = \frac{-\beta(1-\alpha)\left[\frac{\delta}{2} + \sigma\left((1-\alpha)(1-\delta) + \frac{\delta}{2}\right)\right] + \sigma\delta d(1-\alpha+\alpha\delta)}{\delta[\sigma\delta d - (1-\alpha)\beta]}, \tag{17}$$

$$\frac{1}{2} - m = -\frac{\sigma[\delta d - (1-\alpha)\beta]\left[(1-\alpha)(1-\delta) + \frac{\delta}{2}\right]}{\delta[\sigma\delta d - (1-\alpha)\beta]}, \tag{18}$$

$$1 - m = \frac{(1-\alpha)\left[\beta\left(\sigma\left((1-\alpha)(1-\delta) + \frac{\delta}{2}\right) - \frac{\delta}{2}\right) - \sigma\delta d(1-\delta)\right]}{\delta[\sigma\delta d - (1-\alpha)\beta]}, \tag{19}$$

$$m^2 - n^2 D = \frac{\sigma(1-\alpha+\delta\alpha)\left[-\beta(1-\alpha)((1-\delta)(1-\alpha)\sigma+\delta) + \delta d(1-\alpha+\delta\alpha)\right]}{\delta^2[\sigma\delta d - (1-\alpha)\beta]}, \tag{20}$$

$$(1-m)^2 - n^2 D = -\frac{\sigma(1-\delta)(1-\alpha)^2\left[\beta(\sigma(1-\alpha+\delta\alpha) - \delta) - \delta d(1-\delta)\right]}{\delta^2[\sigma\delta d - (1-\alpha)\beta]}. \tag{21}$$

First suppose $\sigma\delta d - (1-\alpha)\beta > 0$. This is equivalent to

$$\beta < \frac{\sigma\delta d}{1-\alpha},$$

and also implies that $\delta d - (1-\alpha)\beta > 0$ and hence $\frac{1}{2} - m < 0$. So the case (a) is not possible. To satisfy the case (b) we need $1 - m > 0$, which is equivalent to

$$\sigma\left[(1-\alpha)(1-\delta) + \frac{\delta}{2}\right] - \frac{\delta}{2} > 0$$

and

$$\beta > \frac{\sigma\delta d(1-\delta)}{\sigma\left[(1-\alpha)(1-\delta) + \frac{\delta}{2}\right] - \frac{\delta}{2}}.$$

Thus we have a lower and upper bound of β that need to satisfy

$$\frac{\sigma\delta d(1-\delta)}{\sigma\left[(1-\alpha)(1-\delta) + \frac{\delta}{2}\right] - \frac{\delta}{2}} < \frac{\sigma\delta d}{1-\alpha}.$$

However, this is equivalent (after simplification) to $\sigma > 1$ so the case (b) is also not possible.

Similarly, we assume now

$$\sigma\delta d - (1-\alpha)\beta < 0, \quad \text{i.e.} \quad \beta > \frac{\sigma\delta d}{1-\alpha}.$$

Now $\frac{1}{2} - m > 0$ is equivalent to

$$\delta d - (1-\alpha)\beta > 0, \quad \text{i.e.} \quad \beta < \frac{\delta d}{1-\alpha},$$

and $m^2 - n^2 D > 0$ is equivalent to

$$\beta > \frac{\delta d(1-\alpha+\delta\alpha)}{(1-\alpha)[(1-\delta)(1-\alpha)\sigma+\delta]}.$$

This again leads to the case (a) being not possible since

$$\frac{\delta d(1-\alpha+\delta\alpha)}{(1-\alpha)[(1-\delta)(1-\alpha)\sigma+\delta]} < \frac{\delta d}{1-\alpha}$$

is equivalent to $\sigma > 1$.

For the case (b) to hold, we need $1 - m > 0$ and $(1-m)^2 - n^2 D > 0$. These two inequalities are equivalent to

$$\beta\left[\sigma\left((1-\alpha)(1-\delta)+\frac{\delta}{2}\right)-\frac{\delta}{2}\right] < \sigma\delta d(1-\delta),$$

$$\beta\left[\sigma(1-\alpha+\delta\alpha)-\delta\right] > \delta d(1-\delta).$$

Notice that

$$\left[\sigma\left((1-\alpha)(1-\delta)+\frac{\delta}{2}\right)-\frac{\delta}{2}\right] > \sigma(1-\alpha+\delta\alpha)-\delta,$$

since it is equivalent to $\sigma < 1$ and

$$\sigma(1-\alpha+\delta\alpha)-\delta > 0$$

from the second inequality above. Thus we have again the following bounds for β

$$\frac{\delta d(1-\delta)}{\sigma(1-\alpha+\delta\alpha)-\delta} < \beta < \frac{\sigma\delta d(1-\delta)}{\sigma\left((1-\alpha)(1-\delta)+\frac{\delta}{2}\right)-\frac{\delta}{2}}.$$

But this is impossible since it implies

$$\sigma(1-\alpha+\alpha\delta)-\frac{\delta}{2} < 0,$$

which contradicts

$$\sigma(1-\alpha+\alpha\delta)-\delta > 0.$$

So the case (b) is also impossible.

Altogether, if an i_S^* exists, it is unique. We are now going to derive conditions for its existence. The existence of a unique biologically feasible real root is equivalent to (also taking into account $0 < i_S^* < 1$)

$$A(0)A(1) < 0 \quad \Leftrightarrow \quad a_0(a_2+a_1+a_0) < 0.$$

This is equivalent to

$$-\sigma^2(1-\delta)(1-\alpha)^2(1-\alpha+\delta\alpha)B(\beta) < 0,$$

or

$$B(\beta) > 0$$

with

$$B(x) := b_2 x^2 + b_1 x + b_0$$

and

$$b_2 := -(1-\alpha)\left[(1-\alpha+\delta\alpha)\sigma - \delta\right]\left[(1-\alpha)(1-\delta)\sigma + \delta\right],$$

$$b_1 := \left(2\left(\left(1/2 - \alpha + \alpha^2\right)\delta^2 + \left(3\alpha - 1 - 2\alpha^2\right)\delta + (\alpha-1)^2\right)\sigma - \delta^2\right)d\delta,$$

$$b_0 := -\delta^2 d^2 (1-\delta)(1-\alpha+\delta\alpha) < 0.$$

The quadratic equation $B(x) = 0$ has two real roots as follows:

$$x_1 = \frac{\delta d(1-\alpha+\alpha\delta)}{(1-\alpha)[\delta + \sigma(1-\alpha)(1-\delta)]} > 0$$

and

$$x_2 = \frac{\delta d(1-\delta)}{\sigma(1-\alpha+\delta\alpha) - \delta}.$$

Note that the signs of x_2 and b_2 are opposite to each other and they are given by $\sigma(1-\alpha+\delta\alpha) - \delta$. We have the following two sets of conditions for $B(\beta) > 0$

$$\sigma(1-\alpha+\delta\alpha) - \delta > 0 \quad \text{and} \quad \min\{x_1, x_2\} < \beta < \max\{x_1, x_2\}, \tag{22}$$

or

$$\sigma(1-\alpha+\delta\alpha) - \delta < 0 \quad \text{and} \quad x_1 < \beta. \tag{23}$$

Note that in the case of (22) both x_1 and x_2 are positive so we need to establish the min and the max of the two. We will show that $x_1 < x_2$ under the assumption in (22):

$$x_1 - x_2 = -\frac{2d\delta^2(1-\sigma)\left((\alpha-1/2)\delta - \alpha + 1\right)}{\left[(1-\alpha+\alpha\delta)\sigma - \delta\right](1-\alpha)\left[(1-\delta)(1-\alpha)\sigma + \delta\right]}.$$

The sign of this expression depends on the term

$$T := (\alpha - 1/2)\delta - \alpha + 1,$$

which, written in terms of the original parameters, is

$$T = \frac{\delta[b + d(1-\delta)]}{2(b - (1-\delta)d)} > 0.$$

Therefore, $x_1 - x_2 < 0$ and the above conditions now read:

$$(1-\delta)d - (1-\sigma)b > 0 \quad \text{and} \quad \frac{b[b - (1-\delta)d]}{b - (1-\delta)(1-\sigma)d} < \beta < \frac{(1-\delta)d[b - (1-\delta)d]}{(1-\delta)d - (1-\sigma)b}, \tag{24}$$

or

$$(1-\delta)d - (1-\sigma)b < 0 \quad \text{and} \quad \frac{b[b - (1-\delta)d]}{b - (1-\delta)(1-\sigma)d} < \beta. \tag{25}$$

$\square$

Note that negating the condition for local stability of the disease-induced extinction equilibrium $(0,1,0)$ is in agreement with conditions (24) and (25). Note also that the lower bound for β in both (24) and (25) is in fact the condition $R_0 > 1$; see (8).

Concerning the stability of (i_F^*, i_S^*), we obtained the following result:

Proposition 5 *The solutions of the system* (9) *under standard incidence are globally stable whenever they exist.*

Proof First denote

$$di_F/dt = (1-\sigma)\beta(1-i_F-i_S)(i_F+i_S) - di_F - i_F[(b-d)(1-i_S) - \delta di_S] := F(i_F, i_S),$$

$$di_S/dt = \sigma\beta(1-i_F-i_S)(i_F+i_S) - \delta di_S - i_S[(b-d)(1-i_S) - \delta di_S] := G(i_F, i_S).$$

We use the Poincaré-Bendixson Thrichotomy that says the following for a planar system: Any trajectory that remains in a closed and bounded region of the plane with finitely many fixed points has the limit set either (1) an equilibrium (2) a periodic orbit (3) a finite set of equilibria and trajectories that emerge in and converge to this finite set of equilibria.

Note that, since we have already proved the uniqueness of (i_F^*, i_S^*) in the rectangle $[0,1] \times [0,1]$, the option (3) is ruled out from the start. What remains is to show that for any initial condition in this rectangle the solution stays inside the rectangle and also to rule out periodic solutions. The original system in S, I_F and I_S is positively invariant. This ensures that the rectangle $[0,1] \times [0,1]$ is invariant for the system of proportions of infectives i_F and i_S. To rule out periodic solutions, consider the following Dulac function

$$\varphi(i_F, i_S) = \frac{1}{i_F + i_S}.$$

Then

$$\frac{\partial}{\partial i_F}[\varphi(i_F, i_S)F(i_F, i_S)] + \frac{\partial}{\partial i_S}[\varphi(i_F, i_S)G(i_F, i_S)] =$$

$$= -\frac{1}{i_F+i_S}\left\{\beta i_F + i_S[\beta - (b-(1-\delta)d)] + (b-(1-\delta)d)(1-i_S) + d(1-\delta)\frac{i_S}{i_F+i_S}\right\} < 0.$$

This follows from the existence condition on the interior equilibrium (i_F^*, i_S^*), i.e.

$$\beta > \frac{b[b-(1-\delta)d]}{b-(1-\delta)(1-\sigma)d} > b-(1-\delta)d.$$

Thus periodic solutions are ruled out and the unique interior equilibrium (i_F^*, i_S^*) is globally stable whenever it exists. □

Proposition 6 *Define*

$$B_1 := \frac{b[b-(1-\delta)d]}{b-(1-\delta)(1-\sigma)d},$$

$$B_2 := \frac{\sigma d[b-(1-\delta)d]}{[d-b(1-\sigma)][\delta+(1-\delta)\sigma]},$$

$$B_3 := \frac{(1-\delta)d[b-(1-\delta)d]}{(1-\delta)d-(1-\sigma)b}.$$

Then under standard incidence, we have that:

Case 1*:* $(1-\delta)d - b(1-\sigma) > 0$ *implies* $B_1 < B_2 < B_3$ *and*

– *if* $B_1 < \beta < B_2$ *then* (i_F^*, i_S^*, N^*) *is globally stable*
– *if* $B_2 < \beta < B_3$ *then* $(i_F^*, i_S^*, 0)$ *is globally stable*

Case 2*:* $(1-\delta)d - b(1-\sigma) < 0$ *implies* $B_1 < B_2$ *and*

– *if* $b(1-\sigma) - d < 0$ *and* $B_1 < \beta < B_2$, *or if* $b(1-\sigma) - d > 0$ *and* $\beta > B_2$ *then* (N^*, i_F^*, i_S^*) *is globally stable*
– *if* $b(1-\sigma) - d < 0$ *and* $\beta > B_2$ *then* $(0, i_F^*, i_S^*)$ *is globally stable*

Proof From the equation for N (10) we know that

– $(i_F^*, i_S^*, 0)$ is globally stable if and only if $i_S^* > \alpha$, i.e. if and only if $A(\alpha)A(1) < 0$
– (i_F^*, i_S^*, N^*) is globally stable if and only if $i_S^* < \alpha$, i.e. if and only if $A(0)A(\alpha) < 0$

where

$$A(i_S) = a_2(i_S)^2 + a_1 i_S + a_0.$$

It is

$$A(\alpha)A(1) = -\frac{d^4\delta^6\sigma(1-\delta)T_1 T_2}{[b-(1-\delta)d]^6},$$

where

$$T_1 = [b(1-\sigma) - d][\delta + (1-\delta)\sigma]\beta + \sigma d(b - [1-\delta]d),$$

$$T_2 = [(1-\delta)d - b(1-\sigma)]\beta - d(1-\delta)[b - (1-\delta)d].$$

Taking into account the existence and uniqueness conditions (24) and (25) for a steady state i_S^* in the interval $(0,1)$, we conclude that

$$T_2 < 0.$$

So $A(\alpha)A(1) < 0$ if and only if $T_1 < 0$. This is equivalent to

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

This threshold for β is greater than the lower bound for β in the conditions (24) and (25):

$$\frac{\sigma d[b-(1-\delta)d]}{[d-b(1-\sigma)][\delta+(1-\delta)\sigma]} - \frac{b[b-(1-\delta)d]}{b-(1-\delta)(1-\sigma)d} =$$

$$= \frac{[b-(1-\delta)d](1-\sigma)(b-d)[(1-\delta)(b+d)\sigma+\delta b]}{[\delta+(1-\delta)\sigma][d-b(1-\sigma)][b-d(1-\delta)(1-\sigma)]} > 0.$$

Furthermore, if the condition (24) is satisfied then this threshold is lower than the upper bound on β (24), because

$$\frac{(1-\delta)d[b-(1-\delta)d]}{(1-\delta)d-(1-\sigma)b} - \frac{\sigma d[b-(1-\delta)d]}{[d-b(1-\sigma)][\delta+(1-\delta)\sigma]} =$$

$$= \frac{\delta d(1-\sigma)[b-(1-\delta)d][(1-\delta)[d-b(1-\sigma)]+b\sigma]}{[d(1-\delta)-b(1-\sigma)][d-b(1-\sigma)][\delta+\sigma(1-\delta)]} > 0.$$

$\square$

Altogether, in terms of β, we have the following results for standard incidence:

Case 1: $(1-\delta)d - b(1-\sigma) > 0$ implies $B_1 < B_2 < B_3$ and

– if $\beta < B_1$ then $(0,0,K)$ is locally stable

– if $B_1 < \beta < B_2$ then (i_F^*, i_S^*, N^*) is globally stable

– if $B_2 < \beta < B_3$ then $(i_F^*, i_S^*, 0)$ is globally stable

– if $\beta > B_3$ then $(0,1,0)$ is locally stable

Case 2: $(1-\delta)d - b(1-\sigma) < 0$ implies $B_1 < B_2$ and

– if $\beta < B_1$ then $(0,0,K)$ is locally stable

– if $b(1-\sigma) - d < 0$ and $B_1 < \beta < B_2$ or if $b(1-\sigma) - d > 0$ and $\beta > B_2$ then (N^*, i_F^*, i_S^*) is globally stable

– if $b(1-\sigma) - d < 0$ and $\beta > B_2$ then $(0, i_F^*, i_S^*)$ is globally stable

Proposition 7 *Under both mass action incidence and asymptotic incidence, the endemic equilibrium (i_F^*, i_S^*, N^*) exists and is unique if and only if $R_0 > 1$.*

Proof We carry out the phase plane analysis to prove existence and uniqueness of the endemic equilibrium (i_F^*, i_S^*, N^*). With (12), we have

$$i_F^* + i_S^* = i_S^*\left(1 + \frac{\delta(1-\sigma)(1-i_S^*)}{\sigma[\delta(\alpha-i_S^*)+1-\alpha]}\right) =: i_S^* c(i_S^*).$$

Inserting this form into the second equation of (9) and simplifying, $i_S^* > 0$ and $N^* > 0$ will be solutions of the system

$$\sigma\Phi(N)[1 - i_S c(i_S)]c(i_S) - [b-(1-\delta)d](1-i_S) = 0,$$
$$(b-d) - [b-(1-\delta)d]i_S - d_1 N = 0, \tag{26}$$

where the second equation follows from (10). This second equation implies (recall $K = (b-d)/d_1$)

$$N = K - i_S\frac{b-(1-\delta)d}{d_1}, \tag{27}$$

so that the N-isocline is a linearly decreasing function of i_S, starting at K for $i_S = 0$ and terminating at $-\delta d/d_1$ for $i_S = 1$. The first equation of (26) implies

$$\Phi(N) = \frac{[b - (1 - \delta)d](1 - i_S)}{\sigma[1 - i_S c(i_S)]c(i_S)} > 0, \tag{28}$$

since $c(i_S) > 0$. This can be seen by noting that $\delta(\alpha - i_S) + 1 - \alpha > 0$ if and only if

$$i_S < \frac{1 - \alpha + \alpha\delta}{\delta} = \frac{b}{b - (1 - \delta)d} \tag{29}$$

if we insert the formula (13) for α. As $b/[b - (1 - \delta)d] > 1$ the inequality (29) is satisfied for any $i_S \in \langle 0, 1 \rangle$.

Let us now explore the right-hand side of (28) as a function of i_S, say $f(i_S)$,

$$f(i_S) := \frac{[b - (1 - \delta)d](1 - i_S)}{\sigma[1 - i_S c(i_S)]c(i_S)},$$

where

$$c(i_S) = \frac{\delta(1 - \sigma)(1 - i_S)}{\sigma[\delta(\alpha - i_S) + 1 - \alpha]}.$$

It is $f(1) = [b - (1 - \delta)d]/\sigma$ and $f(0) = [b - (1 - \delta)d]/[\sigma c(0)]$. In addition,

$$c(0) = 1 + \frac{\delta(1 - \sigma)}{\sigma(\delta\alpha + 1 - \alpha)} = 1 + \frac{\delta(1 - \sigma)[b - (1 - \delta)d]}{\sigma\delta b} > 1,$$

where we used the formula (13) for α. Hence, $f(0) < f(1)$. Differentiating the function $f(i_S)$ with respect to i_S, we get

$$f'(i_S) = \frac{[b - (1 - \delta)d]\delta(1 - \alpha + \alpha\delta - \delta i_S)(1 - \sigma)\sigma\{\delta[(1 - 2i_S)(1 - \alpha + \alpha\delta) + \delta i_S] + 2(1 - \alpha)(1 - \delta)\sigma(1 - \alpha + \alpha\delta)\}}{[\sigma(1 - \alpha + \alpha\delta) - \delta i_S]^2[\sigma(1 - \alpha) - \delta(1 - i_S - \sigma(1 - \alpha))]^2}.$$

Everything off the curled brackets of $f'(i_S)$ is positive, so that we are going to explore the sign of the expression in the curled brackets, which can be rewritten as

$$(1 - \alpha + \alpha\delta)[\delta + 2(1 - \alpha)(1 - \delta)\sigma] - i_S\delta[2(1 - \alpha + \alpha\delta) - \delta]. \tag{30}$$

Setting this expression to zero and solving it for i_S, we have

$$i_S^\circ = \frac{(1 - \alpha + \alpha\delta)[\delta + 2(1 - \alpha)(1 - \delta)\sigma]}{\delta[2(1 - \alpha + \alpha\delta) - \delta]}.$$

Substituting the formula (13) for α, we eventually have

$$i_S^\circ = \frac{b + (1 - \delta)d\left(\frac{2b\sigma}{b - (1 - \delta)d}\right)}{b + (1 - \delta)d}.$$

Obviously, $i_S^\circ > 0$. In addition, the expression (30) is positive for $i_S < i_S^\circ$ and negative for $i_S > i_S^\circ$. Hence, the function $f(i_S)$ is growing for $i_S < i_S^\circ$ and declining for $i_S > i_S^\circ$, and reaches a maximum at $i_S = i_S^\circ$. We note that it might be $i_S^\circ > 1$ for high enough σ, and $f(i_S)$ will then be growing for all $i_S \in \langle 0, 1 \rangle$.

Now, because N is declining in i_S (27) and both $\Phi(N) = \beta N$ (mass action incidence) and $\Phi(N) = \beta N/(c+N)$ (asymptotic incidence) are growing in N, the composite function $\Phi(N(i_S))$ will be declining in i_S. This implies that the necessary and sufficient condition for the existence of an endemic equilibrium is

$$\Phi(K) > f(0),$$

or equivalently, after insertion for $f(0)$ and trivial algebraic manipulations,

$$\Phi(K) > \frac{b[b-(1-\delta)d]}{b-(1-\sigma)(1-\delta)d}.$$

This last expression is equivalent to $R_0 > 1$, see (8). Finally, because of the shape and endpoints of the involved isoclines, the endemic equilibrium is unique provided it exists. $\qquad\square$

We were not able to rigorously prove local stability (or not) of the endemic equilibrium (i_F^*, i_S^*, N^*) under both mass action incidence and asymptotic incidence. Therefore, we resorted to large-scale numerical simulations. These consisted of randomly generating values of model parameters such that $R_0 > 1$, seeking for endemic equilibria corresponding to these parameter values, evaluating the Jacobian of the model (1) at these equilibria, and seeking for eigenvalues of the Jacobian. For both mass action incidence and asymptotic incidence, we made 10^6 such parameter values generations and eigenvalue evaluations, and all of these produced only eigenvalues with negative real parts. So, we make the following conjecture:

Conjecture 1 Under both mass action incidence and asymptotic incidence, the endemic equilibrium (i_F^*, i_S^*, N^*) is locally stable if and only if $R_0 > 1$, i.e. whenever it exists.

3.3 Control effectiveness

In this section, we run the model (1) for a variety of parameter sets and evaluate the effectiveness E with which the host population is controlled. Based on these simulations, we aim at identifying the parameter range (i.e. pathogen properties) in which a significant effect of increased lifespan of the sterilized individuals on the pest control effectiveness is observed. Once $R_0 > 1$, the results we present below do not change qualitatively with the strength of negative density dependence d_1.

3.3.1 Standard incidence

For any fixed value of δ, the control effectiveness E increases with increasing σ (Fig. 2). For relatively low β, the increase is rather unnoticed for low σ up to a point from which the control effectiveness rapidly increases to 1
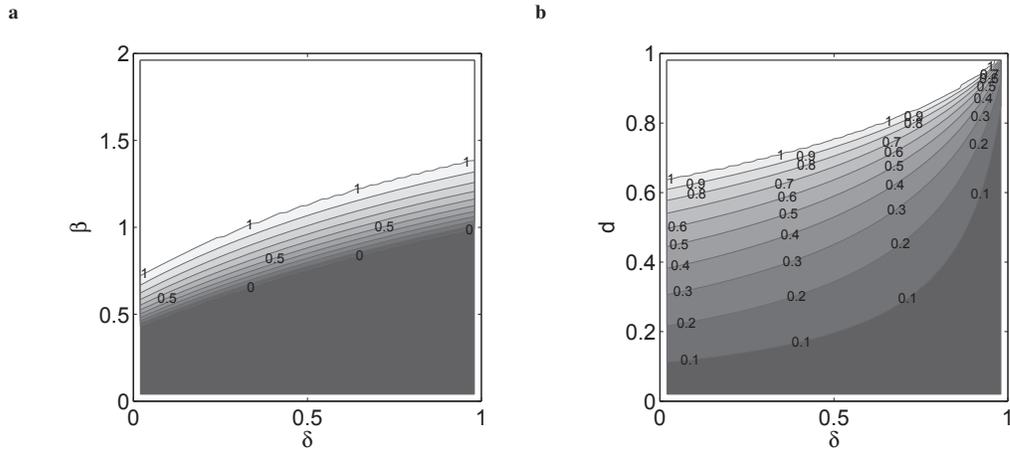
a

b

c

d
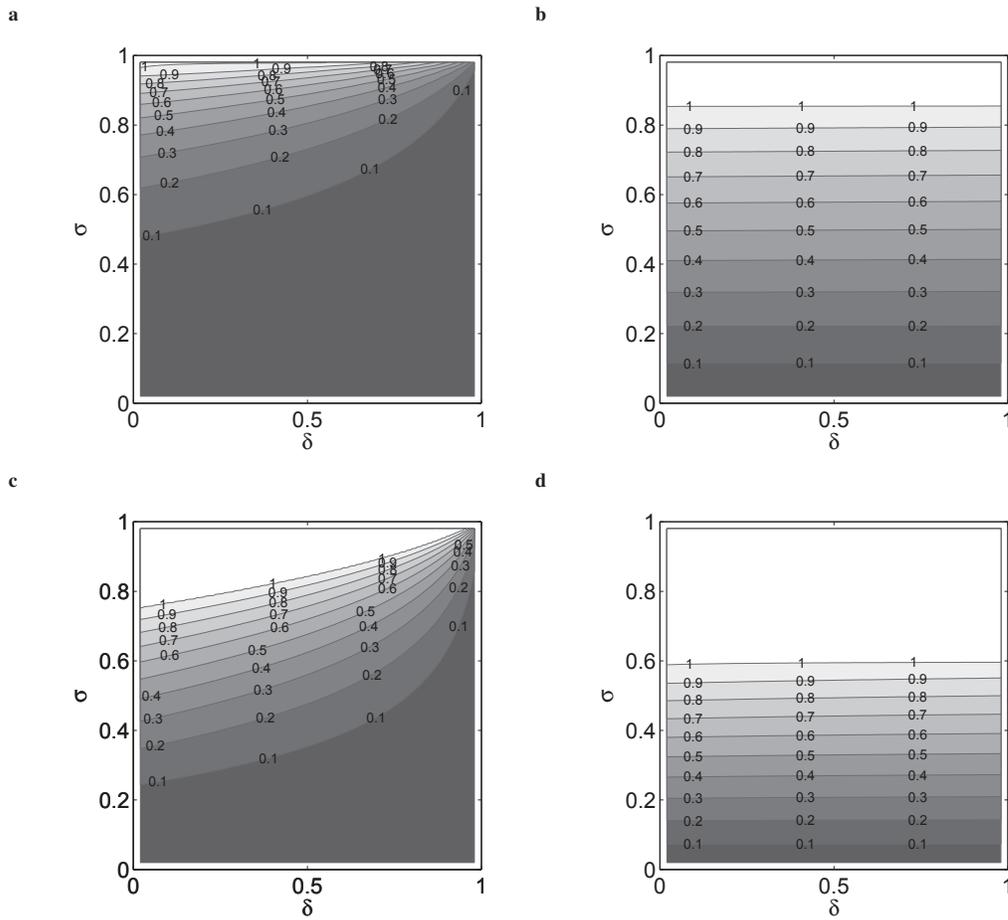
Fig. 2 Control effectiveness with standard incidence, as a function of the proportion of infected individuals that become sterilized (σ) and the proportional reduction of the intrinsic mortality rate in those infected individuals that become sterilized (δ). Other parameter values: **a** $\beta = 1$, $b = 1$, $d = 0.2$; **b** $\beta = 3$, $b = 1$, $d = 0.2$. **c** $\beta = 1$, $b = 1$, $d = 0.5$; **d** $\beta = 3$, $b = 1$, $d = 0.5$; all panels: $d_1 = 0.1$

(Fig. 2a and c). For relatively high β, however, the increase is virtually linear in σ until the maximum value $E = 1$ of the control effectiveness is reached (Fig. 2b and d).

Most importantly from our perspective, for any fixed value of σ, the control effectiveness E increases with decreasing δ. However, as Fig. 2 suggests, the factor δ that extends life expectancy of the sterilized hosts has a measurable effect only for β close to b, high enough d, and relatively high σ. Indeed, this is confirmed in Figs 3 and 4. In such cases, the increase in the control effectiveness can be very dramatic, from virtually 0 to something in between 0.5 and 1 (Fig. 2c). Still, an increase in β relative to b appears to have the most substantial effect (Figs 2d and 4).

Finally, we explore how the control effectiveness E depends on δ for $\beta = b$. The higher is d with respect to b and the higher is σ, the more rapidly E increases as δ decreases (Fig. 5). For example, for $d/b = 0.6$ and $\sigma = 0.8$, the 5% control effectiveness observed for $\delta = 1$ (no increase in longevity due to disease) increases up to about 50% control effectiveness for $\delta = 0.9$ (corresponding to the 10% increase in longevity of sterilized individuals).

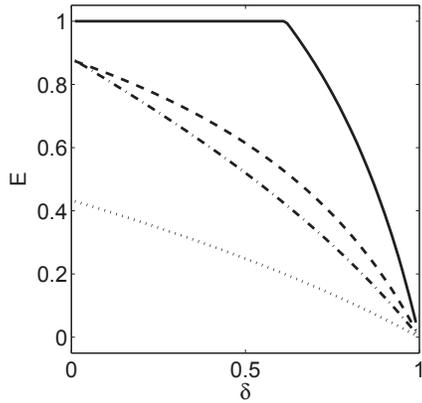**a**   **b**

Fig. 3 Control effectiveness with standard incidence, as a function of: **a** the disease transmission efficiency β and the proportional reduction of the intrinsic mortality rate in those infected individuals that become sterilized (δ); **b** the intrinsic mortality rate d and the proportional reduction of the intrinsic mortality rate in those infected individuals that become sterilized (δ). Other parameter values: **a** $b = 1$, $d = 0.7$, $\sigma = 0.6$; **b** $b = 1$, $\beta = 1$, $\sigma = 0.6$; all panels: $d_1 = 0.1$
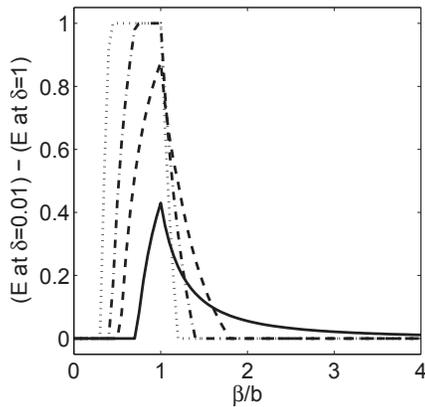

Fig. 4 Control effectiveness with standard incidence, as a function of the β/b ratio. Plotted is the difference between control effectiveness at $\delta = 0.01$ and $\delta = 1$, hence the added value of increased life expectancy in the most optimistic case. Line coding: $d = 0.4b$ (solid), $d = 0.6b$ (dashed), $d = 0.7b$ (dash-dot), $d = 0.8b$ (dotted); $\sigma = 0.6$, $b = 1$, $d_1 = 0.1$. The figure is identical for other values of b
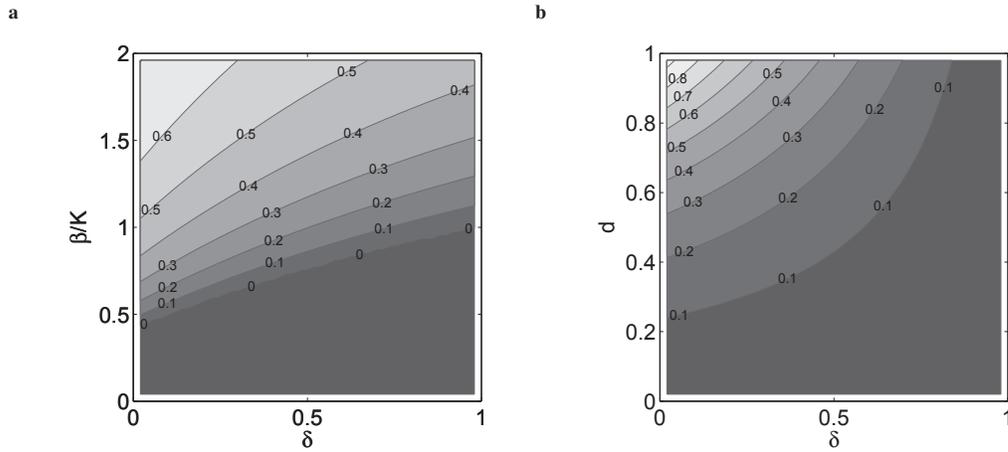
3.3.2 Mass action incidence

While the trends identified for standard incidence are maintained also here, the control effectiveness E attains much lower values (Figs 6 and 7). Note that we scale here the transmission efficiency β by the environmental carrying capacity of the host K, so as to keep the same basic reproduction numbers R_0 for all adopted disease transmission models.

Fig. 5 Control effectiveness with standard incidence, as a function of the proportional reduction of the intrinsic mortality rate in those infected individuals that become sterilized (δ). Line coding: $\sigma = 0.8$, $d = 0.6b$ (solid), $\sigma = 0.8$, $d = 0.4b$ (dashed), $\sigma = 0.6$, $d = 0.6b$ (dash-dot), $\sigma = 0.6$, $d = 0.4b$ (dotted); common parameter values: $b = \beta = 1$, $d_1 = 0.1$

a   **b**

c   **d**
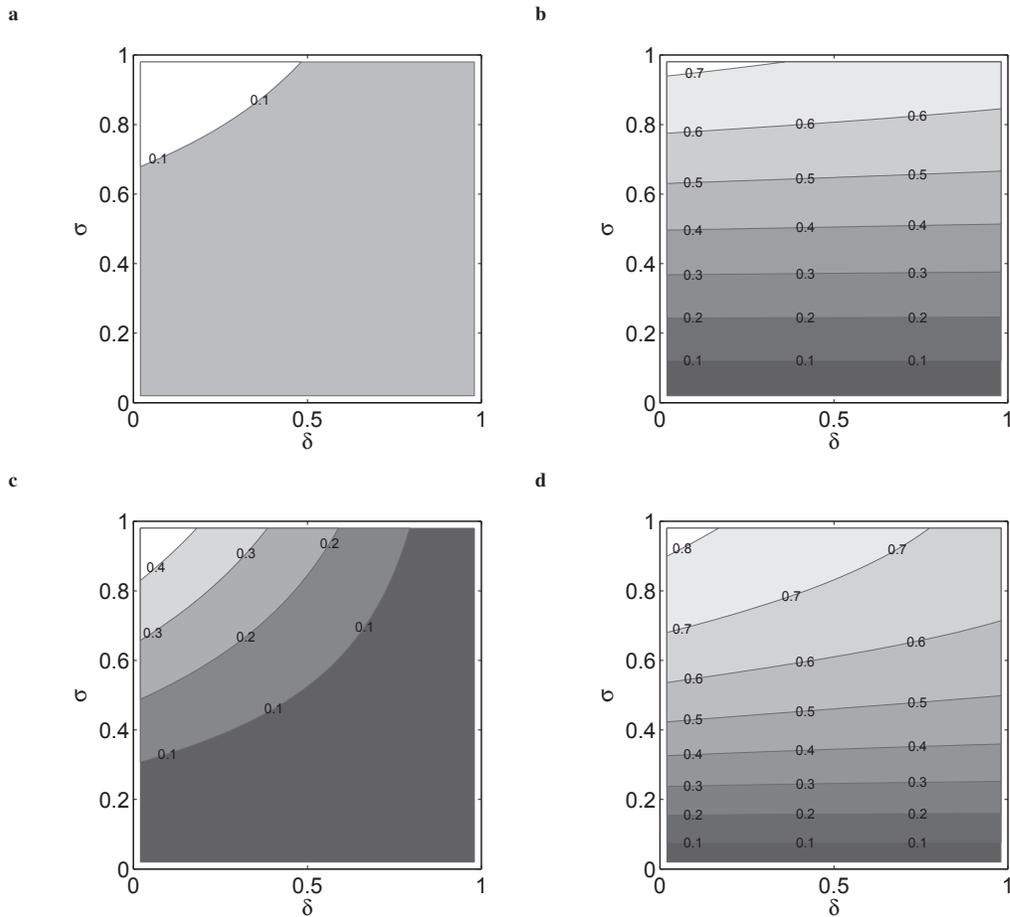
Fig. 6 Control effectiveness with mass action incidence, as a function of the proportion of infected individuals that become sterilized (σ) and the proportional reduction of the intrinsic mortality rate in those infected individuals that become sterilized (δ). Other parameter values: **a** $\beta = 1/K$, $b = 1$, $d = 0.2$; **b** $\beta = 3/K$, $b = 1$, $d = 0.2$. **c** $\beta = 1/K$, $b = 1$, $d = 0.5$; **d** $\beta = 3/K$, $b = 1$, $d = 0.5$; all panels: $d_1 = 0.1$, $K = (b-d)/d_1$

Fig. 7 Control effectiveness with mass action incidence, as a function of: **a** the (scaled) disease transmission efficiency β/K and the proportional reduction of the intrinsic mortality rate in those infected individuals that become sterilized (δ); **b** the intrinsic mortality rate d and the proportional reduction of the intrinsic mortality rate in those infected individuals that become sterilized (δ). Other parameter values: **a** $b = 1$, $d = 0.7$, $\sigma = 0.6$; **b** $b = 1$, $\beta = 1/K$, $\sigma = 0.6$; all panels: $d_1 = 0.1$, $K = (b - d)/d_1$
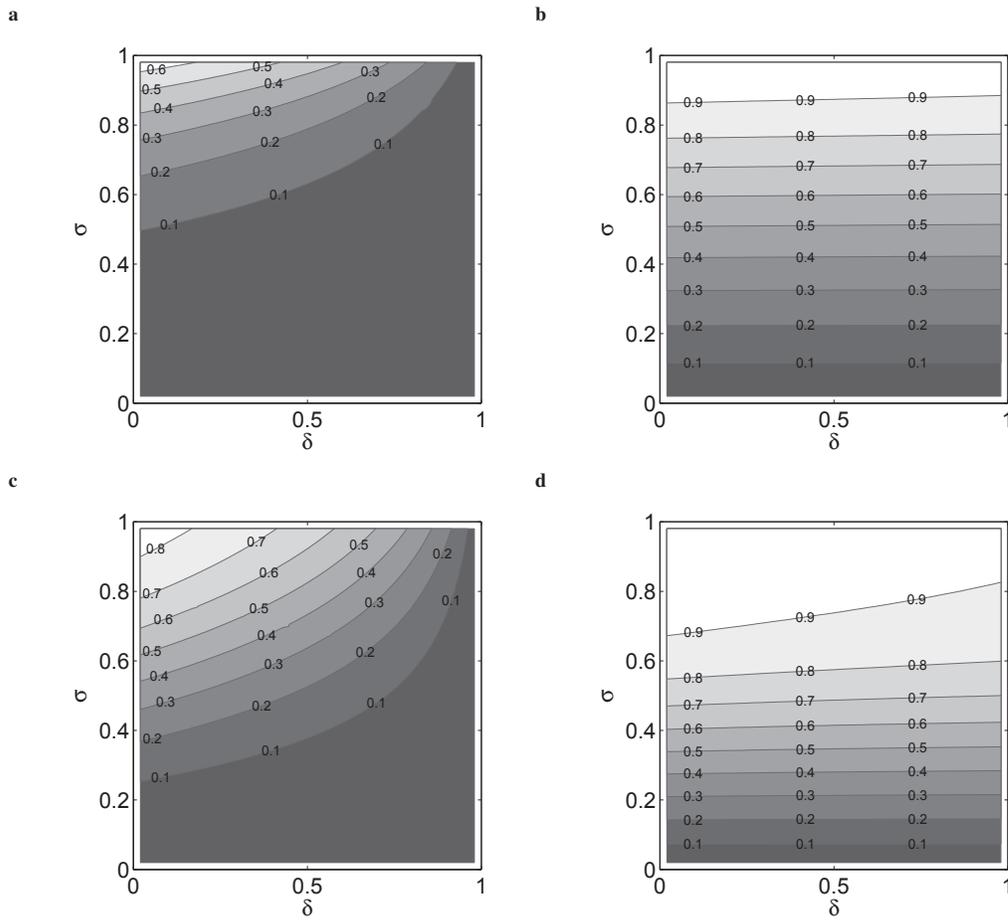
3.3.3 Asymptotic incidence

While the trends identified for the previous two incidence terms are maintained also here, the control effectiveness E attains lower values than standard incidence but higher values than mass action incidence (Figs 8 and 9). Note that we scale here the transmission efficiency β by the term $K/(K + c)$, so as to keep the same basic reproduction numbers R_0 for all adopted disease transmission models.

4 Discussion

In this paper, we explored consequences for infection spread and host population suppression of the assumption that sterilizing pathogens preventing reproduction in some infected individuals cause a redistribution of resources and hence increased longevity of sterilized population members. This in turn allows for a more efficient infection spread, as just some of the infectious individuals are those that live longer, and hence for a greater population suppression at endemic equilibrium compared with when no longevity promotion is assumed.

It is not surprising that higher efficiencies of sterilization (larger σ) and higher stretches of longevity (lower δ) promote higher effectiveness of host control by sterilizing pathogens. Much less obvious is under what circumstances these effects are 'measurable' and, in particular, of practical importance for population management. We show in this paper that the effect of increased longevity of sterilized individuals increases with (1) decreasing difference between the per capita birth rate (b) and the transmission rate between individuals at the carrying ca-

a

b

c

d

Fig. 8 Control effectiveness with asymptotic incidence, as a function of the proportion of infected individuals that become sterilized (σ) and the proportional reduction of the intrinsic mortality rate in those infected individuals that become sterilized (δ). Other parameter values: **a** $\beta/(K/(c+K)) = 1, b = 1, d = 0.2$; **b** $\beta = 3/(K/(c+K)), b = 1, d = 0.2$. **c** $\beta = 1/(K/(c+K)), b = 1, d = 0.5$; **d** $\beta = 3/(K/(c+K)), b = 1, d = 0.5$; all panels: $d_1 = 0.1, c = 1, K = (b-d)/d_1$

pacity ($\Phi(K)$), (2) decreasing the birth-to-intrinsic-death-rate ratio (i.e. $b/d \to 1$, recall we assume $b > d$), and (3) increasing sterilization efficiency of the pathogen (σ). This result makes perfect sense since it corresponds to the situation where the natural life expectancy is relatively short and the disease transmission is relatively slow. In this situation, the enhanced life expectancy of the sterilized hosts will have maximal effect in facilitating spread of the pathogen. Interestingly, with no regard to disease-induced sterilization, Stenseth (1981) concluded that "The larger the mortality of the uncontrolled population, the more likely is reproduction to be the optimal pest control."

The qualitative character of transmission is also of high importance – keeping R_0 the same across the transmission models, the largest control efficiencies are generally achieved for standard incidence, followed by asymptotic incidence and eventually mass action incidence. Standard incidence provides the most effective control mainly because this transmission dynamic allows for disease-induced population extinction, which is not the case for the other two transmission dynamics (unless $\delta = \sigma = 1$). This should not concern us much, however, as sterilizing

a **b**
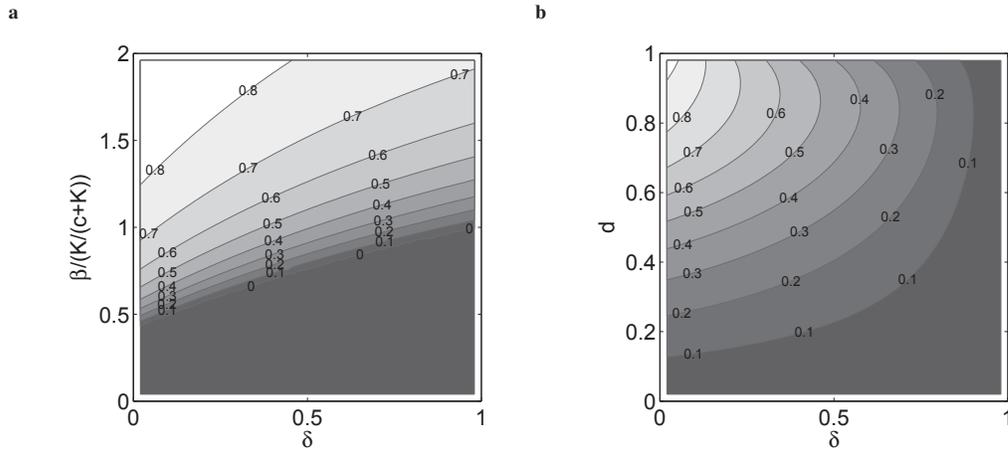
Fig. 9 Control effectiveness with asymptotic incidence, as a function of: **a** the (scaled) disease transmission efficiency $\beta/(K/(c+K))$ and the proportional reduction of the intrinsic mortality rate in those infected individuals that become sterilized (δ); **b** the intrinsic mortality rate d and the proportional reduction of the intrinsic mortality rate in those infected individuals that become sterilized (δ). Other parameter values: **a** $b = 1, d = 0.7, \sigma = 0.6$; **b** $b = 1, \beta = 1/(K/(c+K)), \sigma = 0.6$; all panels: $d_1 = 0.1, c = 1, K = (b-d)/d_1$

viruses are mostly sexually transmitted (Lockhart et al 1996) and sexually transmitted diseases most closely fit the standard incidence paradigm (McCallum et al 2001). If the sterilizing pathogens are to be engineered, on the other hand, we have to carefully consider their transmission mode. Our results have direct implications for the development of effective VVIC agents. In particular, our results indicate how effective, in terms of β and σ, the eventual control agents should be, relative to the life history parameters of a pest species (b, d and δ). Our results could also be of a value in conservation biology; we could rank the existing infections threatening endangered species according to their effect on host population suppression and try and prevent invasions of those that are most threatening.

The above results can only be applied, however, when we are able to estimate δ, the factor that extends life expectancy of the sterilized hosts. We expect this might be a problem in many species, mostly in those that are long-lived, but it might on the other hand be relatively easy in short-lived species such as insects or passerine birds or rodents. It is just in these short-lived species where δ can be significantly high, as we do not actually expect high δ in long-lived species. Studies that would allow for an estimate of δ are rare, however; some are listed in Table 1. More often, studies of fecundity-longevity or reproduction-survival trade-offs summarize their results in the form of correlations. For example, there is a highly significant negative correlation between egg production and longevity in a wing-dimorphic cricket (Tanaka and Suzuki 1998). Likewise, in *Drosophila melanogaster*, females with higher opportunities for mating had significantly lower lifespans than females with lower mating opportunities (Fowler and Partridge 1989). With regard to *Drosophila melanogaster*, males that never mated lived significantly longer than continuously mated males (Prowse and Partridge 1997) – there have been very few studies that looked at the cost of reproduction in males. Sometimes, one of the quantities in question is only indirectly measured, as in Ellers (1995) where a negative correlation between the number of eggs in the ovarioles and the fat

Table 1 Some studies of fecundity-longevity or reproduction-survival trade-offs that allow for an estimate of δ

Species	Observation	Estimate of δ	Reference
Parasitoid *Trichogramma brasiliensis*	Adult females survived shorter (10 days) when an unlimited supply of hosts was present, but longer (16 days) when no hosts were provided	$10/16 = 0.625$	Ramesh and Manickavasagam (2003)
Dung beetle *Onthophagus binodis*	Mating is not costly in terms of reduced longevity for female dung beetles; despite a longevity cost of reproduction for males, no evidence was found for differential longevity costs associated with alternative reproductive tactics – quantitative results: males in mixed sex populations = 54.8 ± 2.5 days (mean $\pm$ standard error), males in single sex populations = 62.6 ± 2.7 days, females in mixed sex populations = 55.2 ± 2.7 days, females in single sex populations = 54.7 ± 2.8 days	$54.8/62.6 \sim 0.875$	Kotiaho and Simmons (2003)
Columbian ground squirrel *Spermophilus columbianus*	Reproductive status influenced mortality in females – non-reproducing females had a higher chance of surviving (83.5%) than reproducing females (75.7%)	$16.5/24.3 \sim 0.679$	Neuhaus and Pelletier (2001)

content of females was observed – fat content served here as a proxy for longevity as it is in the examined species strongly positively correlated to longevity. Last but not least, some studies do not present intra-specific variability in fecundity vs. longevity, but rather represent mean evolutionary endpoints at higher units: Bennett and Owens (2002) thus showed that annual fecundity and clutch size were negatively correlated to adult survival rate and age at first breeding was positively correlated to adult survival rate, calculated across bird families and orders, while Thomas et al (2000) showed that in humans, a significant negative relationship exists between the mean female fecundity and the mean female longevity, calculated across countries – this result indicates that women in rich countries tend to have fewer children and live longer.

The trade-off between reproduction and survival does not need to be intrinsic to the focal species, i.e. it need not always be driven by changes in energy allocation. Many animals suffer from a conflict between mating success and survival – their mating signals are exploited by their natural enemies (Zuk and Kolluru 1998). For example, gravid females might be more conspicuous to predators and hence suffer from higher mortality such as in some copepods (Svensson 1992). As gravid females, or more generally individuals generating a mating signal, are obviously not sterile, this is yet another mechanism that may increase longevity of sterilized females. So δ might also decrease due to predators or parasitoids, and thus possibly attain relatively low values.

One of our main results is that an increase in longevity of the sterilized infected individuals further enhances their ability to spread the disease. A natural follow-up would be to extend the model to include an indigenous species that is negatively affected by the presence of an invasive species we aim to eradicate with the sterilizing virus. In that case, the lower mortality rate of the sterilized infected individuals can have both a negative and positive

effect: on the one hand, they live longer and spread the disease more efficiently, on the other hand, they have more time to cause damage to the indigenous species. It all depends on how fast the invasive species can eliminate the indigenous one. For example, in the Great Lakes, the Asian carp is a huge problem because it is extremely efficient at eradicating plankton, which in turn causes starvation and extinction in native populations (hence a competitive interaction).

We showed that the fact that some animals live longer when sterilized, due to the reproduction-survival trade-off, should be taken into consideration in some cases. If this happens, one should consider more detailed modeling to come up with more precise predictions of this phenomenon. In particular, with regards to VVIC, the virus infects both sexes but causes sterilization in just one of them (Deredec et al 2008, and references therein). Also, mating can be costly in terms of reduced longevity for one sex but not the other (Kotiaho and Simmons 2003).

If we release a pathogen that sterilizes males, we invoke a sort of "disease-induced" sterile-male-release technique (Dell'Omo and Palmery 2002; Dyck et al 2005). In particular, an increasing fraction of the male population becomes sterile, and females will loose time and opportunities in mating with sterile males. This can create a mate-finding Allee effect (Courchamp et al 2008) and accelerate the population decline, especially when females mate only once; see also Barlow et al (1997) for the importance of mating systems in effectiveness of fertility control. On the other hand, reduction of host population density makes it more difficult for pathogens to spread via mass action or asymptotic transmission, as efficiency of that transmission declines with decreasing population density. Fortunately, when an Allee effect creates a demographic extinction threshold, even diseases with mass action or asymptotic transmission can drive host populations to extinction (Hilker et al 2009). The message conveyed by the last two paragraphs is that more predictive models should be sexually structured, reflecting sex-specificity of the involved processes and possibility for a mate-finding Allee effect.

5 Appendix

5.1 Basic reproduction number R_0

Using the next generation matrix approach due to van den Driessche and Watmough (2002), we first reshuffle the state variables so that the first two represent infected classes: (I_F, I_S, S). Using the notation of van den Driessche and Watmough (2002), we have

$$\mathscr{F} = \begin{bmatrix} (1-\sigma)\Phi(N)S(I_F + I_S)/N \\ \sigma\Phi(N)S(I_F + I_S)/N \\ 0 \end{bmatrix}$$

comprising all rates of the model (1) that describe the appearance of new infections, and

$$\mathscr{V} = \begin{bmatrix} (d+d_1N)I_F \\ (\delta d+d_1N)I_S \\ -b(S+I_F)+\Phi(N)S(I_F+I_S)/N+(d+d_1N)S \end{bmatrix}$$

comprising all remaining rates (with the reverse sign). Setting $\mathbf{x} = (I_F, I_S)$ and $\mathbf{x}_0 = (0,0)$, this implies

$$\mathbf{F} = \left[\frac{\partial \mathscr{F}_i}{\partial x_j}(\mathbf{x}_0)\right]\Bigg|_{1\le i,j\le 2} = \Phi(K)\begin{bmatrix} 1-\sigma & 1-\sigma \\ \sigma & \sigma \end{bmatrix}$$

and

$$\mathbf{V} = \left[\frac{\partial \mathscr{V}_i}{\partial x_j}(\mathbf{x}_0)\right]\Bigg|_{1\le i,j\le 2} = \begin{bmatrix} b & 0 \\ 0 & b-(1-\delta)d \end{bmatrix}$$

This implies

$$\mathbf{V}^{-1} = \begin{bmatrix} 1/b & 0 \\ 0 & 1/(b-(1-\delta)d) \end{bmatrix}$$

and the next generation matrix thus becomes

$$\mathbf{FV}^{-1} = \Phi(K)\begin{bmatrix} (1-\sigma)/b & (1-\sigma)/(b-(1-\delta)d) \\ \sigma/b & \sigma/(b-(1-\delta)d) \end{bmatrix}$$

of which the dominant (in the absolute value) eigenvalue, equal to R_0, is

$$R_0 = \Phi(K)\left(\frac{1-\sigma}{b}+\frac{\sigma}{b-(1-\delta)d}\right) \tag{31}$$

5.2 Case $\delta = 1$: system (4)

For $\delta = 1$, the model (1) reduces to the model (4). This latter model is a special case of the system analyzed by Zhou and Hethcote (1994, their model (9)). Transforming the model (4) to that with state variables i and N, we get

$$\frac{di}{dt} = i\left[\Phi(N)(1-i)-b(1-\sigma i)\right]$$

$$\frac{dN}{dt} = N\left[b(1-\sigma i)-(d+d_1N)\right] \tag{32}$$

Results of the standard local stability analysis of the system (32) are summarized in Table 2. Zhou and Hethcote (1994) proved that if the equilibria are locally stable then they are also globally stable.

Table 2 Existence and local stability of equilibria of the model (4); $R_0 = \Phi(K)/b$, AI = asymptotic incidence, MI = mass action incidence, SI = standard incidence

Equilibrium	Exists	Locally stable	Unstable	E
$(0,0)$	always	–	always ($b > d$)	–
$(K,0)$	always	$R_0 < 1$	$R_0 > 1$	0
$(0,i_0^*)$	AI & MI: feasible only for $\sigma = 1$	–	exists	–
	for which $i_0^* = 1$			
	SI: $R_0 > 1$, $i_0^* = (\beta - b)/(\beta - \sigma b)$	exists & $i_0^* > 1/(\sigma b/(b-d))$	exists & $i_0^* < 1/(\sigma b/(b-d))$	1
(N^*,i^*)	AI & MI: $R_0 > 1$	exists	–	$i^* \sigma b/(b-d)$
	SI: $R_0 > 1$ & $i^* < 1/(\sigma b/(b-d))$	exists	–	$i^* \sigma b/(b-d)$

Table 3 Existence and local stability of equilibria of the model (5); $R_0 = \Phi(K)/[b - (1-\delta)d]$, AI = asymptotic incidence, MI = mass action incidence, SI = standard incidence

Equilibrium	Exists	Locally stable	Unstable	E
$(0,0)$	always	–	always ($b > d$)	–
$(K,0)$	always	$R_0 < 1$	$R_0 > 1$	0
$(0,1)$	always	MI & AI: –	MI & AI: always	–
		SI: $R_0 > 1$	SI: $R_0 < 1$	1
(N^*,i^*)	AI & MI: $R_0 > 1$	AI & MI: exists	AI & MI: –	$0 < E < 1$
	SI: –	SI: –	SI: –	

5.3 Case $\sigma = 1$: system (5)

For $\sigma = 1$, the model (1) reduces to the model (5). Transforming the model (5) to that with state variables i and N, we get

$$\frac{di}{dt} = i(1-i)\left[\Phi(N) - (b - (1-\delta)d)\right]$$

$$\frac{dN}{dt} = N\left[(b-d)(1-i) - \delta di - d_1 N\right] \tag{33}$$

Results of the standard local stability analysis of the system (33) are summarized in Table 3. Poincaré-Bendixson theory together with the Dulac criterion can be used to show that also here, if the equilibria are locally stable then they are also globally stable.

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