The impact of sexually abstaining groups on persistence of sexually transmitted infections in populations with ephemeral pair bonds

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Abstract

Individuals often stop reproducing some time before they die. In this paper we compose and analyze a logistic, two-sex population model in which individuals form pairs just to mate (i.e. pair bonds are ephemeral) and later move on to sexually abstaining groups. Using this model, we study the impact of sexually abstaining groups on persistence of a benign sexually transmitted infection (STI) in populations with such ephemeral pair bonds. We observe that the presence of sexually abstaining groups cannot prevent an STI from invasion or eliminate it when already present if the transition rates to the sexually abstaining groups are independent of the infection status of individuals (susceptible or infected). On the other hand, if they depend on that status, the presence of sexually abstaining groups can prevent an STI from invasion or eliminate it when present. Specifically, in the simple case of sex-independent vital parameters, this happens if the transition rate of the

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infected individuals to the sexually abstaining group is higher than the transition rate of the susceptible ones. These results contrast the earlier results based on assuming long-term, stable pair bonds, in which case one is capable of preventing or eliminating the disease with the same isolation rate for the susceptible and infected individuals.

1. Introduction

Individuals often stop reproducing some time before they die. In humans, non-reproductive groups are represented by those individuals who due to social and/or medical reasons or by mere choice remain childless and sometimes also sexually abstain, following a certain moment of their life. Among animals, due to often strong mating competition and/or hierarchy dictated by some mating systems, some individuals in many cases benefit at the expense of the others, and some do not mate at all [Andersson, 1994, Shuster and Wade, 2003]. Last but not least, among domesticated animals, older individuals are frequently eliminated from breeding groups and hence remain non-reproductive for the rest of life.

The presence of a class of non-reproductive individuals within a population does not only affect population dynamics relative to the absence of such a class, but may also affect the chance of a sexually transmitted infection (STI) to invade the population, and the prevalence of the infection if it succeeds to invade. Given that animal STIs are ubiquitous, both as regards a variety of host taxa and etiological agents, yet quite understudied relative to the other types of infections, both theoretical and empirical studies are needed to understand their ecological as well as evolutionary dynamics [Lockhart et al., 1996, Boots and Knell, 2002, Kokko et al., 2002]. Interestingly, STIs have been predominantly reported for humans and economically important animal species, including many domesticated animals where regular breeding is practiced [Oriel and Hayward, 1974, Lockhart et al., 1996].

Exploration of the effects of non-reproductive and/or sexually abstaining groups calls for a consideration of two-sex population models, not only because life spans can differ between males and females [Rankin and Kokko, 2007] and sex ratios at birth may be biased [Gomendio et al., 2006], but also because of potentially differential impacts of STIs on both sexes [Miller

et al., 2007]. This is why Maxin and Milner [2009] developed a logistic, two-sex population model to study the influence of sexually abstaining groups on dynamics of a (human) population affected by a benign STI. The STI was assumed to be transmitted only within heterosexual, stable pairs. The model thus excluded 'unfaithful' sexual contacts outside the established pairs and also possible births from single mothers, and was thus an approximate description of a conservative community. From an epidemiological perspective, this framework benefits from *de-facto* quarantine since as long as two individuals stay in a relationship they do not acquire the infection if they are both susceptible nor do they transmit it if they are infected. Hence, the most important parameter driving STI transmission was observed to be the pair dissolution rate. With this model, Maxin and Milner [2009] proved that, under some conditions, the presence of sexually abstaining groups can prevent an STI from invasion or eliminate it from the host population if already present.

In the present paper, we address the same question of how the presence of sexually abstaining groups impacts dynamics of an STI-host system, composing and analyzing a model more appropriate for promiscuous populations that form ephemeral rather than stable pair bonds.

The paper is organized as follows. In the next section we introduce a logistic, two-sex population model with only ephemeral pair bonds, and in Section 3 we extend this model by including the sex-specific sexually abstaining groups. We study the baseline behavior of these models and compute the thresholds that separate population extinction from a globally stable interior equilibrium. Section 4 introduces an STI to the model with the sexually abstaining groups, and the basic disease reproduction number of the infection is calculated. We also give the affirmative answer to the question as to whether the sexually abstaining groups can prevent the STI from invasion or eliminate it from the host population if already present. We finally conclude the paper with a summary of our results, compare our results to those for a logistic, two-sex population model with stable pairs, emphasizing important distinctions between the two, and suggest possible avenues of further research. For easier reading of our paper, all proofs of the formulated theorems are given in the Appendix.

2. The logistic two-sex model with ephemeral pair bonds

The basic two-sex population model with ephemeral pair bonds includes two classes of individuals: females (F) and males (M). The mating rate is taken to be the harmonic mean of F and M although there are other possibilities suggested in the literature such as the maximum between F and M [Hadeler et al., 1988]. The harmonic mean tends to be the most accepted form for the mating function in the two-sex population models [e.g. Kokko et al., 2002, Rankin and Kokko, 2007]. It is the same function that models pair formation in the two-sex models with stable pairs. So we have (throughout the paper, $'$ denotes derivative with respect to time)

$$
\begin{aligned}
F' &= \beta\gamma_f \tfrac{FM}{F+M} - (\mu_f + bP)F, \\
M' &= \beta\gamma_m \tfrac{FM}{F+M} - (\mu_m + bP)M.
\end{aligned}
\tag{1}
$$

where β is the maximum reproductive rate, γ_f and γ_m are the ratios of females and males, respectively, among the offspring ($\gamma_f + \gamma_m = 1$), μ_f and μ_m denote the intrinsic female and male mortality rates, respectively, and b measures the strength of negative density dependence in mortality – as population increases, so does competition for food and hence the per capita mortality rate; $P = F + M$ is the total population density.

The model (1) has two steady states: the extinction one $(\bar{F}, \bar{M}) = (0,0)$ and an interior one

$$
\begin{aligned}
F^* &= \frac{\gamma_f \mu_m}{b}\left(1 - \frac{1}{\mathcal{R}}\right)\left[1 + \gamma_f \mathcal{R}_m\left(1 - \frac{1}{\mathcal{R}}\right)\right], \\
M^* &= \frac{\gamma_m \mu_f}{b}\left(1 - \frac{1}{\mathcal{R}}\right)\left[1 + \gamma_m \mathcal{R}_f\left(1 - \frac{1}{\mathcal{R}}\right)\right],
\end{aligned}
\tag{2}
$$

where

$$
\mathcal{R}_f = \frac{\beta\gamma_f}{\mu_f}, \quad \mathcal{R}_m = \frac{\beta\gamma_m}{\mu_m}, \quad \text{and} \quad \mathcal{R} = \frac{\mathcal{R}_f \mathcal{R}_m}{\mathcal{R}_f + \mathcal{R}_m}
$$

are the net reproductive numbers.

Whereas $\mathcal{R}_f$ and $\mathcal{R}_m$ represent the expected number of female/male offspring from a female/male during her/his life-time, $\mathcal{R}$ represents the expected number of heterosexual pairs formed by the female and male offspring. Notice also that $\mathcal{R} > 1$ implies $\mathcal{R}_f > 1$ and $\mathcal{R}_m > 1$, the necessary conditions for a positive steady state to exist.

The following theorem describes long-term dynamics of the model (1); its proof is given in the Appendix.

THEOREM 2.1. *If $\mathcal{R} > 1$ then the interior equilibrium (F^*, M^*) is globally stable and the extinction equilibrium $(\bar{F}, \bar{M})$ is unstable. Conversely, if $\mathcal{R} < 1$, the extinction equilibrium $(\bar{F}, \bar{M})$ is globally stable and the interior equilibrium (F^*, M^*) is not feasible.*

3. Non-reproductive groups

In this section we extend the previous model to include the female and male non-reproductive groups A_f and A_m, respectively. The per capita transition rates into these groups (also referred to as isolation rates further on) are assumed to be density-independent and are denoted by ν_f and ν_m for females and males, respectively. So we have the model:

$$
\begin{aligned}
F' &= \beta\gamma_f \tfrac{FM}{F+M} - (\mu_f + bP)F - \nu_f F, \\[6pt]
M' &= \beta\gamma_m \tfrac{FM}{F+M} - (\mu_m + bP)M - \nu_m M, \\[6pt]
A'_f &= \nu_f F - (\mu_f + bP)A_f, \\[6pt]
A'_m &= \nu_m M - (\mu_m + bP)A_m.
\end{aligned}
\tag{3}
$$

where $P = F + M + A_f + A_m$. This model has two steady states: the extinction one $(\bar{F}, \bar{M}, \bar{A}_f, \bar{A}_m) = (0, 0, 0, 0)$ and an interior one $(\tilde{F}, \tilde{M}, \tilde{A}_f, \tilde{A}_m)$ where

$$
\tilde{F} = \frac{\tilde{P}}{\left(1 + \frac{\nu_f}{\tilde{\mu}_f}\right) + \left(1 + \frac{\nu_m}{\tilde{\mu}_m}\right)\frac{\mathcal{K}_m^n}{\mathcal{K}_f^n}}, \quad
\tilde{M} = \frac{\tilde{P}}{\left(1 + \frac{\nu_m}{\tilde{\mu}_m}\right) + \left(1 + \frac{\nu_f}{\tilde{\mu}_f}\right)\frac{\mathcal{K}_f^n}{\mathcal{K}_m^n}},
\tag{4}
$$

$$
\tilde{A}_f = \frac{\nu_f \tilde{F}}{\tilde{\mu}_f}, \text{ and } \tilde{A}_m = \frac{\nu_m \tilde{M}}{\tilde{\mu}_m},
$$

with

$$
\tilde{P} = \frac{\beta\gamma_f\gamma_m}{b}\left(1 - \frac{1}{\mathcal{R}^n}\right), \quad \tilde{\mu}_f = \mu_f + b\tilde{P}, \quad \tilde{\mu}_m = \mu_m + b\tilde{P},
$$

$$
\mathcal{K}_f^n = \frac{(\mu_f + \nu_f)(\mathcal{R}_f^n - 1) + \mu_m + \nu_m}{\beta}, \text{ and } \mathcal{K}_m^n = \frac{(\mu_m + \nu_m)(\mathcal{R}_m^n - 1) + \mu_f + \nu_f}{\beta}.
$$

The net reproductive numbers are in this case (note n is a superscript here, not a power)

$$\mathcal{R}_f^n = \frac{\beta\gamma_f}{\mu_f + \nu_f}, \quad \mathcal{R}_m^n = \frac{\beta\gamma_m}{\mu_m + \nu_m}, \quad \text{and} \quad \mathcal{R}^n = \frac{\mathcal{R}_f^n \mathcal{R}_m^n}{\mathcal{R}_f^n + \mathcal{R}_m^n}.$$

Whereas $\mathcal{R}_f^n$ and $\mathcal{R}_m^n$ represent the expected number of female/male offspring from a female/male during her/his expected reproductive lifetime, $\mathcal{R}^n$ represents the expected number of heterosexual pairs formed by the female and male offspring during their reproductive lifetime.

Dynamics of the model (3) are determined by the following theorem (also here, its proof is given in the Appendix).

THEOREM 3.1. *If $\mathcal{R}^n > 1$ then the interior equilibrium $(\tilde{F}, \tilde{M}, \tilde{A}_f, \tilde{A}_m)$ is globally stable and the extinction equilibrium $(\bar{F}, \bar{M}, \bar{A}_f, \bar{A}_m)$ is unstable. Conversely, if $\mathcal{R}^n < 1$, the extinction equilibrium $(\bar{F}, \bar{M}, \bar{A}_f, \bar{A}_m)$ is globally stable and the interior equilibrium $(\tilde{F}, \tilde{M}, \tilde{A}_f, \tilde{A}_m)$ is not feasible.*

4. Sexually transmitted infections

Here we extend the model (3) to include a benign STI which will affect neither fecundity nor mortality of the hosts, with infected individuals unable to recover. From an STI perspective we need to separate the non-reproductive groups into two different types: the sexually abstaining groups the members of which not only do not reproduce but also abstain (by choice or not) from sexual activity, and the rest of non-reproductive individuals who remain sexually active. Sexual abstinence can be a consequence of mating competition coupled with potential avoidance of infected individuals. Non-reproductive but sexually active individuals are likely to be more prevalent in humans, but may also occur among animals – sterile animals (whatever is the reason for their sterility) may still participate in mating, especially if the STI is cryptic. In what follows, we assume that all non-reproducing individuals also sexually abstain. The more complex case of the two distinct types of non-reproductive individuals will be treated in a separate paper.

Modeling STI transmission becomes tricky in two-sex population models with an explicit mating function. As the mating function models the way individuals meet and mate, the model of STI transmission needs in many cases to reflect this and be consistent with the selected mating function. This is why we use for the infection transmission also a kind of harmonic mean, but

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we are here not in the clash with the classical epidemiological theory, since this function essentially represents standard incidence, the term commonly used to model STI transmission [McCallum et al., 2001]. So our model is as follows:

$$
\begin{aligned}
F' &= \beta\gamma_f \frac{(F+\varphi)(M+\chi)}{F+\varphi+M+\chi} - \lambda_f \frac{F\chi}{F+M+\varphi+\chi} - (\mu_f + bP)F - \nu_f F, \\[2mm]
M' &= \beta\gamma_m \frac{(F+\varphi)(M+\chi)}{F+\varphi+M+\chi} - \lambda_m \frac{M\varphi}{F+M+\varphi+\chi} - (\mu_m + bP)M - \nu_m M, \\[2mm]
\varphi' &= \lambda_f \frac{F\chi}{F+M+\varphi+\chi} - (\mu_f + bP)\varphi - \alpha_f \varphi, \\[2mm]
\chi' &= \lambda_m \frac{M\varphi}{F+M+\varphi+\chi} - (\mu_m + bP)\chi - \alpha_m \chi, \\[2mm]
A'_f &= \nu_f F - (\mu_f + bP)A_f, \\[2mm]
A'_m &= \nu_m M - (\mu_m + bP)A_m, \\[2mm]
A'_\varphi &= \alpha_f \varphi - (\mu_f + bP)A_\varphi, \\[2mm]
A'_\chi &= \alpha_m \chi - (\mu_m + bP)A_\chi,
\end{aligned}
\tag{5}
$$

where the total population size is now

$$
P = F + M + \varphi + \chi + A_f + A_m + A_\varphi + A_\chi,
$$

and φ, χ, A_φ, and A_χ respectively denote classes of reproducing infected females, reproducing infected males, sexually abstaining infected females, and sexually abstaining infected males. Furthermore, α_f and α_m are the female and male isolation rates of infected individuals, respectively, and λ_f and λ_m scale the infection rates at which infected males infect susceptible females and vice versa, respectively.

As is common in virtually any analysis of any epidemiological model, we start with calculating the basic disease reproduction number of the infection.

THEOREM 4.1. *For the model* (5), *the basic disease reproduction number of the infection is*

$$
\mathcal{R}_0^n = \frac{\sqrt{\lambda_f \lambda_m \tilde{F}\tilde{M}}}{(\tilde{F} + \tilde{M})\sqrt{(\tilde{\mu}_f + \alpha_f)(\tilde{\mu}_m + \alpha_m)}},
$$

where $\tilde{F}$ and $\tilde{M}$ are components of the interior equilibrium of the model (3), and $\tilde{\mu}_f = \mu_f + b(\tilde{M} + \tilde{F})$ and $\tilde{\mu}_m = \mu_m + b(\tilde{M} + \tilde{F})$. If $\mathcal{R}_0^n < 1$ the disease-free equilibrium (DFE) $(\tilde{F}, \tilde{M}, 0, 0, \tilde{A}_f, \tilde{A}_m, 0, 0)$ is locally asymptotically stable; it is unstable if $\mathcal{R}_0^n > 1$.

See the Appendix for a proof of this theorem.

In order to analyze the conditions on the parameters that may lead to infection extinction or an endemic state we first consider the case when the isolation rates are independent of the infection status, a situation that might correspond to a cryptic STI:

$$\nu_f = \alpha_f \quad \text{and} \quad \nu_m = \alpha_m,$$

and further assume that the two infection transmission coefficients are equal,

$$\lambda_f = \lambda_m = \lambda.$$

In this relatively simple case, the dynamics of the total female and male populations are identical to those of the model (3). Indeed, summing up the first with the third equation, the second with the fourth, the fifth with the seventh, and the sixth with the eighth equation in the model (5), we obtain

$$
\begin{aligned}
(F + \varphi)' &= \beta \gamma_f \frac{(F+\varphi)(M+\chi)}{F+\varphi+M+\chi} - (\mu_f + bP)(F + \varphi) - \nu_f(F + \varphi), \\
(M + \chi)' &= \beta \gamma_m \frac{(F+\varphi)(M+\chi)}{F+\varphi+M+\chi} - (\mu_m + bP)(M + \chi) - \nu_m(M + \chi), \\
(A_f + A_\varphi)' &= \nu_f(F + \varphi) - (\mu_f + bP)(A_f + A_\varphi), \\
(A_m + A_\chi)' &= \nu_m(M + \chi) - (\mu_m + bP)(A_m + A_\chi),
\end{aligned}
\tag{6}
$$

which is precisely the model (3). Using Theorem 3.1 we can conclude that if $\mathcal{R}^n > 1$, then

$$\lim_{t \to \infty}[F(t) + \varphi(t)] = \tilde{F}, \quad \lim_{t \to \infty}[M(t) + \chi(t)] = \tilde{M}$$

$$\lim_{t \to \infty}[A_f(t) + A_\varphi(t)] = \tilde{A}_f, \quad \lim_{t \to \infty}[A_m(t) + A_\chi(t)] = \tilde{A}_m.$$

The model (5) has three steady states. Two boundary equilibria, the extinction equilibrium $(\bar{F}, \bar{M}, \bar{\varphi}, \bar{\chi}, \bar{A}_f, \bar{A}_m, \bar{A}_\varphi, \bar{A}_\chi) = (0, 0, 0, 0, 0, 0, 0, 0)$ and

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the DFE $(\tilde{F}, \tilde{M}, 0, 0, \tilde{A}_f, \tilde{A}_m, 0, 0)$, and an interior (endemic) equilibrium $(F^o, M^o, \varphi^o, \chi^o, A_f^o, A_m^o, A_\varphi^o, A_\chi^o)$, where

$$F^o = \tilde{F} - \varphi^o, \quad \varphi^o = \frac{\lambda^2 \tilde{F} \tilde{M} - (\tilde{F} + \tilde{M})^2 (\tilde{\mu}_f + \nu_f)(\tilde{\mu}_m + \nu_m)}{\lambda[\lambda \tilde{M} + (\tilde{F} + \tilde{M})(\tilde{\mu}_f + \nu_f)]},$$

$$M^o = \tilde{M} - \chi^o, \quad \chi^o = \frac{\lambda^2 \tilde{F} \tilde{M} - (\tilde{F} + \tilde{M})^2 (\tilde{\mu}_f + \nu_f)(\tilde{\mu}_m + \nu_m)}{\lambda[\lambda \tilde{F} + (\tilde{F} + \tilde{M})(\tilde{\mu}_m + \nu_m)]},$$

$$A_f^o = \tilde{A}_f - A_\varphi^o, \quad A_\varphi^o = \frac{\nu_f \varphi^o}{\tilde{\mu}_f},$$

$$A_m^o = \tilde{A}_m - A_\chi^o, \quad A_\chi^o = \frac{\nu_m \varphi^o}{\tilde{\mu}_m},$$

and where $\tilde{\mu}_f = \mu_f + b\tilde{P}$, $\tilde{\mu}_m = \mu_m + b\tilde{P}$ and $\tilde{P} = \tilde{F} + \tilde{M} + \tilde{A}_f + \tilde{A}_m$.

With the adopted assumptions, the basic reproduction number of the infection, in this special case denoted as $\mathcal{L}_0^n$, is

$$\mathcal{L}_0^n = \frac{\lambda \sqrt{\tilde{F} \tilde{M}}}{(\tilde{F} + \tilde{M}) \sqrt{(\tilde{\mu}_f + \nu_f)(\tilde{\mu}_m + \nu_m)}}.$$

It is easy to see (from the expressions of φ^o and χ^o) that the endemic equilibrium is feasible (positive) if and only if $\mathcal{L}_0^n > 1$.

From the first two equations of (3) we have

$$\frac{\tilde{M}}{\tilde{F} + \tilde{M}} = \frac{\tilde{\mu}_f + \nu_f}{\beta \gamma_f} \quad \text{and} \quad \frac{\tilde{F}}{\tilde{F} + \tilde{M}} = \frac{\tilde{\mu}_m + \nu_m}{\beta \gamma_m}.$$

Using these expressions, the basic reproduction number becomes

$$\mathcal{L}_0^n = \frac{\lambda}{\beta \sqrt{\gamma_f \gamma_m}}.$$

Since $\mathcal{L}_0^n$ is independent of ν_f and ν_m, the threshold between the DFE and the endemic equilibrium is in this particular case independent of the presence and intensity of sexual abstinence. Assuming $\mathcal{R}^n > 1$, the following theorem describes long-term dynamics of the model (5), depending on the value of $\mathcal{L}_0^n$ (see the Appendix for its proof).

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THEOREM 4.2. *Let $\mathcal{R}^n > 1$. If $\mathcal{L}_0^n < 1$, then the DFE $(\tilde{F}, \tilde{M}, \tilde{A}_f, \tilde{A}_m, 0, 0, 0, 0)$ is globally stable. Conversely, if $\mathcal{L}_0^n > 1$, the endemic equilibrium $(F^o, M^o, \varphi^o, \chi^o, A_f^o, A_m^o, A_\varphi^o, A_\chi^o)$ exists, is globally stable, and the DFE is unstable.*

We now summarize the conclusions related to the model with isolation rates independent of the disease:

- If $\mathcal{R}^n < 1$ then the extinction equilibrium $(\bar{F}, \bar{M}, \bar{\varphi}, \bar{\chi}, \bar{A}_f, \bar{A}_m, \bar{A}_\varphi, \bar{A}_\chi) = (0, 0, 0, 0, 0, 0, 0, 0)$ is globally stable,

- If $\mathcal{R}^n > 1$ and $\mathcal{L}_0^n < 1$ then the DFE $(\tilde{F}, \tilde{M}, 0, 0, \tilde{A}_f, \tilde{A}_m, 0, 0)$ is globally stable,

- If $\mathcal{R}^n > 1$ and $\mathcal{L}_0^n > 1$ then the endemic equilibrium of the infection $(F^o, M^o, \varphi^o, \chi^o, A_f^o, A_m^o, A_\varphi^o, A_\chi^o)$ is globally stable.

We now turn to the case when the transition rates into the sexually abstaining groups are dependent on the infection status, i.e.

$$\nu_f \neq \alpha_f \quad \text{and} \quad \nu_m \neq \alpha_m.$$

The net reproductive number and the interior equilibrium analysis are difficult to establish in this most general case. We provide, instead, an analysis in the particular case of sex-independent parameters while still maintaining infection- dependent isolation rates. Assuming

$$\mu_f = \mu_m := \mu, \ \nu_f = \nu_m := \nu, \ \alpha_f = \alpha_m := \alpha,$$

$$\gamma_f = \gamma_m = \frac{1}{2}, \text{ and } \lambda_f = \lambda_m := \lambda,$$

the model (5) becomes

$$
\begin{aligned}
F' &= \tfrac{\beta}{4}(F + \varphi) - \tfrac{\lambda}{2}\tfrac{F\varphi}{F+\varphi} - (\mu + bP)F - \nu F, \\[4pt]
\varphi' &= \tfrac{\lambda}{2}\tfrac{F\varphi}{F+\varphi} - (\mu + bP)\varphi - \alpha\varphi, \\[4pt]
A_f' &= \nu F - (\mu + bP)A_f, \\[4pt]
A_\varphi' &= \alpha\varphi - (\mu + bP)A_\varphi.
\end{aligned}
\tag{7}
$$

This model (7) has the following steady states:

- The extinction equilibrium $(0, 0, 0, 0)$,

- The disease-free equilibrium $\left(\frac{2}{b\beta} \left(\frac{\beta}{4} - \mu - \nu \right) \left(\frac{\beta}{4} - \nu \right), 0, \frac{2\nu}{b\beta} \left(\frac{\beta}{4} - \mu - \nu \right), 0 \right)$,

- An endemic equilibrium $(\hat{F}, \hat{\varphi}, \hat{A}_f, \hat{A}_\varphi)$ with

$$
\hat{F} = \frac{2\hat{x}\hat{P}(\mu + b\hat{P})}{\beta}, \ \ \hat{\varphi} = \left(\frac{1}{\hat{x}} - 1 \right) \hat{F}, \ \ \hat{A}_f = \frac{\nu\hat{F}}{\mu + b\hat{P}}, \ \ \hat{A}_\varphi = \frac{\alpha\hat{\varphi}}{\mu + b\hat{P}},
$$

$$
\hat{P} := \frac{1}{b} \left[\frac{\beta}{4\hat{x}} - \frac{\lambda}{2}(1 - \hat{x}) - \mu - \nu \right], \ \text{and} \ \hat{x} := \frac{\beta/2}{\lambda - 2(\alpha - \nu)}.
$$

The main result related to the model (7) is provided in the following theorem (see the Appendix for its proof):

THEOREM 4.3. *For the simplified model (7), if*

- $\lambda < \frac{\beta}{2} + 2(\alpha - \nu)$ *and* $\frac{\beta}{4} < \mu + \nu$*: the extinction equilibrium is globally stable,*

- $\lambda < \frac{\beta}{2} + 2(\alpha - \nu)$ *and* $\frac{\beta}{4} > \mu + \nu$*: the DFE is globally stable,*

- $\lambda > \frac{\beta}{2} + 2(\alpha - \nu)$ *and* $\frac{\beta}{2} < \frac{2(\mu + \alpha)[\lambda - 2(\alpha - \nu)]}{\lambda}$*: the extinction equilibrium is globally stable,*

- $\lambda > \frac{\beta}{2} + 2(\alpha - \nu)$ *and* $\frac{\beta}{2} > \frac{2(\mu + \alpha)[\lambda - 2(\alpha - \nu)]}{\lambda}$*: the endemic equilibrium is globally stable.*

Remark 4.1. *Note that the last pair of conditions can only be satisfied if* $\lambda > 2(\alpha + \mu)$*. Thus, the endemic situation happens if and only if*

$$
\lambda > \max \left\{ \frac{\beta}{2} + 2(\alpha - \nu), 2(\alpha + \mu) \right\}
$$

The first part of the above conditions is equivalent to $\mathcal{R}_0^n > 1$ *(*$\lambda > \frac{\beta}{2} + 2(\alpha - \nu)$*) or* $\mathcal{R}_0^n < 1$ *(*$\lambda < \frac{\beta}{2} + 2(\alpha - \nu)$*). The second part thus indicates that, in general, if the transition rates into the sexually abstaining classes are infection-dependent,* $\mathcal{R}_0^n > 1$ *may lead to either population extinction (if the isolation rate of the infected individuals is high enough) or to an endemic state (if the isolation rate of the infected individuals is below a certain threshold).*

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This result indicates that the net reproductive number has a much more complicated form in the case of infection-dependent isolation rates. This is because if $\nu \neq \alpha$ the infection acts as a transfer between groups (healthy and infected) of different reproductive power.

Remark 4.2. *These results imply that it is impossible for the abstinence to eliminate the disease if the transition rates to the sexually abstaining groups do not depend on the infection status of host individuals (susceptible or infected) since then the basic reproduction number of the infection ($\mathcal{L}^n < 1$) does not depend on these isolation rates. However, as soon as $\nu_f \neq \alpha_f$ and $\nu_m \neq \alpha_m$, this might be possible if in the absence of sexually abstaining groups the basic reproduction number is greater than one and in the presence of sexually abstaining groups it is less than one. In other words, infection-dependent isolation rates may allow for the following double inequality:*

$$\mathcal{R}_0^n < 1 < \mathcal{R}_0$$

where

$$\mathcal{R}_0 = \frac{\lambda\sqrt{F^*M^*}}{(F^* + M^*)\sqrt{\mu_f^* \mu_m^*}}$$

is the basic reproduction number of the infection in the absence of sexually abstaining groups. $\mathcal{R}_0$ has been obtained by replacing ν_f, ν_m, α_f, and α_m in $\mathcal{R}_0^n$ with zero, by replacing $\tilde{F}$ and $\tilde{M}$ with F^* and M^*, components of the interior equilibrium in the absence of non-reproductive groups (Section 2), and denoting by μ_f^* and μ_m^* the mortality rates evaluated at the equilibrium obtained in Section 2:

$$\mu_f^* = \mu_m + b(F^* + M^*) \quad and \quad \mu_m^* = \mu_m + b(F^* + M^*).$$

Figures 1–3 illustrate the above results. While an STI exemplified in Fig. 1 cannot be eliminated by the presence of the sexually abstaining groups provided that the isolation rates are independent of the infection status (Fig. 2), the disease-free equilibrium becomes stable once the isolation rate of the infected individuals is higher than the isolation rate of the susceptible ones (Fig. 3). In addition, Fig. 3 suggests that the latter result is more general and holds also when the vital parameters are sex-specific.

5. Conclusions

In this paper we introduced and analyzed a logistic, two-sex population model in which individuals form pairs just to mate (i.e. pair bonds are ephemeral) so as to study the impact of sexually abstaining groups on persistence of a benign sexually transmitted infection. Our major result states that:

- Presence of the sexually abstaining groups cannot prevent an STI from invasion or eliminate it when already present if the transition rates to the sexually abstaining groups do not depend on the infection status of individuals (susceptible or infected), because the basic reproduction number of the infection does not depend in that case on these transition rates;

- Provided that the transition rates to the sexually abstaining groups depend on the infection status of individuals, then presence of the sexually abstaining groups can prevent an STI from invasion or eliminate it when present. In the simple case of sex-independent vital parameters, this happens if the isolation rate of the infected individuals is higher than the isolation rate of the susceptible ones.

Maxin and Milner [2009] addressed the same question as we did, assuming a logistic, two-sex population model with stable, long-lasting pairs. Hence, two individuals in a relationship could not acquire the infection if both were susceptible nor could they transmit it if they were infected. Contrary to our results, with the same isolation rate for the susceptible and infected individuals, one is capable of eliminating the disease in this latter model with stable pairs. First, this shows that mating system, promiscuous in our model and monogamous in that of Maxin and Milner [2009], is a strong driver of host population dynamics in the presence of sexually abstaining groups and an STI. Second, if elimination of reproductive individuals should serve a practical way of preventing an STI invasion, our results suggest that this can only be achieved if one is able to discern the susceptible and infected individuals. This needs not always be possible, however, as many STIs might be cryptic [Knell, 1999]. On top of that, a true monogamy appears to be relatively rare in nature, as many species with the 'socially' monogamous mating system actually exhibit high rates of extra-pair copulation [Jennions and Petrie, 2000].

Put the other way round, our analysis predicts that STIs cannot be prevented from invasion or eliminated when present if the isolation rate of the infected individuals is lower than or equal to the isolation rate of the susceptible ones. Empirical data that would allow relative comparison of these rates does not seem available. Still, many STIs cause sterility in the affected individuals [Oriel and Hayward, 1974, Lockhart et al., 1996]. We have not modeled sterilizing effects of STIs in this paper, but we might speculate that at least some of these sterilized individuals will enter the sexually abstaining group. In other words, if an individual has trouble finding mates, it might have even more trouble now that she is infected. In this way, infected individuals might move on to the sexually abstaining group at a higher rate than the susceptible ones, satisfying the necessary condition for the STI prevention or elimination.

This is not that simple, however, since sterilized individuals can still participate in mating. This fact requires more complex epidemiological models than our model (5), which will most likely demonstrate more complicated dynamics [Diekmann and Kretzschmar, 1991] and produce different conditions for inability of invasion / ability of elimination of the infection. Anyway, individuals facing a cryptic STI are expected to evolve risky mating behavior, both as regards the number (increased promiscuity) and quality (lowered mate choice) of potential mates [Boots and Knell, 2002]. This will likely result in similar isolation rates of the susceptible and infected individuals. If infected individuals are aware of being infected, one can even imagine that the isolation rates can be reverted, as the infected individuals have 'nothing to lose' and hence might mate 'to the last breath'. In a follow-up paper, we will extend our current models to cover sterilizing effects of STIs and allow for non-reproductive individuals that can still participate in mating.

6. Appendix

In this section we provide the proofs of the theorems introduced in this paper.

Theorem 2.1

PROOF. It is more convenient to re-write the model (1) as an equivalent system in variables $P = F + M$ and $x = \frac{F}{P}$:

$$P' = \beta x(1-x)P - \mu_f xP - \mu_m(1-x)P - bP^2,$$

$$x' = \beta x(1-x)(\mathcal{K}_f - x),$$

(8)

where

$$\mathcal{K}_f = \frac{\mu_f(\mathcal{R}_f - 1) + \mu_m}{\beta}.$$

Notice that the equation for x is decoupled from the one for P. Standard ODE theory implies that $x(t)$ is defined for all $t \geq 0$ which, in turn, implies that $P(t)$ is defined for all $t \geq 0$. In addition, the intervals $(0,1)$ and $(0,\infty)$ are positively invariant for x and P, respectively.

Going back to the model (1) we see that either $\mathcal{R}_f < 1$ or $\mathcal{R}_m < 1$ will cause the population to go extinct. Indeed, if $\mathcal{R}_f < 1$ and taking into account that $\frac{M}{F+M} < 1$ then we can use the following differential inequality:

$$F' < (\beta\gamma_f - \mu_f)F.$$

Integrating both sides we obtain

$$F(t) < F_0 e^{(\beta\gamma_f - \mu_f)t}$$

which implies $F(t) \to 0$ as $t \to \infty$ and hence $M(t) \to 0$ from the second equation of (1). An analogous argument can be used if $\mathcal{R}_m < 1$.

Suppose now that $\mathcal{R}_f > 1$ and $\mathcal{R}_m > 1$. Notice that $\mathcal{R}_f > 1$ and $\mathcal{R}_m > 1$ imply $0 < \mathcal{K}_f < 1$. Setting

$$h(x) := \beta x(1-x)(\mathcal{K}_f - x),$$

the right-hand side of the equation for x, we have that

$$h'(0) = \beta\mathcal{K}_f > 0 \text{ and } h'(1) = \beta(1 - \mathcal{K}_f) > 0.$$

This means that the critical points $x = 0$ and $x = 1$ are both unstable. Furthermore

$$h'(\mathcal{K}_f) = -\beta\mathcal{K}_f(1 - \mathcal{K}_f) < 0$$

meaning that $\mathcal{K}_f$ is the only asymptotically stable critical point for x.

From the sign of the derivative x', if $x_0 > \mathcal{K}_f$ then $x(t)$ is decreasing in forward time, and if $x_0 < \mathcal{K}_f$ then $x(t)$ is increasing in forward time. This

implies that $x(t)$ converges to a finite limit as $t \to \infty$ which must be $\mathcal{K}_f$. Hence
$$\lim_{t \to \infty} x(t) = \mathcal{K}_f.$$
Due to symmetry and to make our notation more consistent, we also set
$$\mathcal{K}_m = 1 - \mathcal{K}_f = \frac{\mu_m(\mathcal{R}_m - 1) + \mu_f}{\beta}.$$

This result allows us to view the equation for P,
$$P' = \beta x(1 - x)P - \mu_f x P - \mu_m(1 - x)P - bP^2 := \bar{f}(t, P), \qquad (9)$$

as an asymptotically autonomous ODE, with the following limiting autonomous ODE:
$$P' = (\beta \mathcal{K}_f \mathcal{K}_m - \mu_f \mathcal{K}_f - \mu_m \mathcal{K}_m - bP)P := f(P). \qquad (10)$$
Note that $\bar{f}(t, P) \to f(P)$ locally uniformly. We are going to use the theory of asymptotically autonomous systems developed in Thieme [1992], Castillo-Chavez and Thieme [1995] in order to establish the main result of this theorem. Note that the limiting equation (10) is a logistic equation in P and
$$\beta \mathcal{K}_f \mathcal{K}_m - \mu_f \mathcal{K}_f - \mu_m \mathcal{K}_m > 0$$

is equivalent to $\mathcal{R} > 1$.

There are two equilibrium points for the limiting equation (10):
$$\bar{P} = 0 \quad \text{and} \quad P^* = \frac{\beta \mathcal{K}_f \mathcal{K}_m - \mu_f \mathcal{K}_f - \mu_m \mathcal{K}_m}{b}.$$

Equivalently,
$$P^* = \frac{\beta \gamma_f \gamma_m}{b} \left(1 - \frac{1}{\mathcal{R}}\right).$$

If $\mathcal{R} < 1$ then $\bar{P}$ is the only biologically feasible equilibrium and all solutions of the limiting equation (10) converge to it. It follows from Theorem 2.3 of Castillo-Chavez and Thieme [1995] that

$$\lim_{t \to \infty} P(t) = 0.$$

If $\mathcal{R} > 1$ then $\bar{P}$ belongs to the trivial invariant closed set $\{0\}$ and P^* lies in the open invariant set $(0, \infty)$. All solutions of (10) starting in $(0, \infty)$

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approach P^* which is also asymptotically stable. Moreover, $\bar{P}$ is a weak repeller for $(0, \infty)$ in (9). To see this, notice that, assuming there exists a solution of (9) starting with $P_0 > 0$ and approaching 0, then

$$\lim_{t \to \infty} \frac{d}{dt} \ln P(t) = \lim_{t \to \infty} [\beta x(1-x) - \mu_f x - \mu_m(1-x) - bP] =$$

$$= \beta \mathcal{K}_f \mathcal{K}_m - \mu_f \mathcal{K}_f - \mu_m \mathcal{K}_m > 0$$

which is a contradiction. Thus, from Theorems 2.4 and 2.5 of Castillo-Chavez and Thieme [1995] we have that, for $P_0 > 0$

$$\lim_{t \to \infty} P(t) = P^*.$$

From this, we obtain the global stability of (F^*, M^*) for $\mathcal{R} > 1$, since

$$\lim_{t \to \infty} F(t) = \frac{\gamma_f \mu_m}{b} \left(1 - \frac{1}{\mathcal{R}}\right) \left[1 + \gamma_f \mathcal{R}_m \left(1 - \frac{1}{\mathcal{R}}\right)\right]$$

$$\lim_{t \to \infty} M(t) = \frac{\gamma_m \mu_f}{b} \left(1 - \frac{1}{\mathcal{R}}\right) \left[1 + \gamma_m \mathcal{R}_f \left(1 - \frac{1}{\mathcal{R}}\right)\right]$$

$\square$

Theorem 3.1

PROOF. Using an analogous approach as for the model without sexual abstinence, we start with an equivalent system in

$$x = \frac{F}{F + M}, \ y = \frac{F}{F + A_f}, \ z = \frac{M}{M + A_m}, \text{ and } F:$$

$$x' = \beta x(1-x)(\mathcal{K}_f^n - x),$$

$$y' = \beta \gamma_f(1-x)y(1-y) - \nu_f y,$$

$$z' = \beta \gamma_m xz(1-z) - \nu_m z, \tag{11}$$

$$F' = \beta \gamma_f(1-x)F - \left[\mu_f + \nu_f + b\left(\frac{1}{y} - \frac{1}{z} + \frac{1}{xz}\right)F\right]F,$$

Just as in the previous theorem, the equation for x is decoupled and, using similar arguments, one can see that the solution of (11) is defined for

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all $t \geq 0$. Furthermore, the interval $(0, 1)$ is forward invariant for x, y and z and $(0, \infty)$ is forward invariant for F.

$\mathcal{R}_f^n > 1$ and $\mathcal{R}_m^n > 1$ are necessary conditions to avoid population extinction and we assume these inequalities from now on. As in the previous theorem, they imply

$$0 < \mathcal{K}_f^n < 1 \text{ and } 0 < \mathcal{K}_m^n < 1.$$

Since the equation for x has the same form as the analogous one for the model (1) without sexually abstaining groups, we conclude that

$$\lim_{t \to \infty} x(t) = \mathcal{K}_f^n.$$

Notice also that $\mathcal{R}^n > 1$ is equivalent to two inequalities needed later:

$$\beta \gamma_m \mathcal{K}_f^n > \mu_m + \nu_m \quad \text{and} \quad \beta \gamma_f \mathcal{K}_m^n > \mu_f + \nu_f. \tag{12}$$

The equation in y,

$$y' = \beta \gamma_f (1 - x) y (1 - y) - \nu_f y, \tag{13}$$

can be viewed as an asymptotically autonomous ODE in y, with the limiting equation

$$y' = (\beta \gamma_f \mathcal{K}_m^n - \nu_f - \beta \gamma_f \mathcal{K}_m^n y) \, y. \tag{14}$$

The equation (14) is a logistic equation in y and, from (12), we see that every solution of (14) starting with a positive value converges to

$$\tilde{y} = \frac{\beta \gamma_f \mathcal{K}_m^n - \nu_f}{\beta \gamma_f \mathcal{K}_m^n} \text{ if } \mathcal{R}^n > 1,$$

or approaches zero, which can only happen if $\mathcal{R}^n < 1$. In the latter case, $y \to 0$ implies $F(t) \to 0$ as $t \to \infty$ which, in turn, causes the entire population to approach the extinction equilibrium.

Assuming now that the solutions of (14) converge to $\tilde{y}$ we show that zero is a weak repeller for (13). Indeed, if a solution of (13) starting with a positive value approaches zero, then

$$\lim_{t \to \infty} \frac{d}{dt} \ln y(t) = \lim_{t \to \infty} [\beta \gamma_f (1 - x) - \nu_f - \beta \gamma_f (1 - x) y] = \beta \gamma_f \mathcal{K}_m^n - \nu_f > 0,$$

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which is a contradiction. From Theorems 2.4 and 2.5 of Castillo-Chavez and Thieme [1995] we conclude that

$$\lim_{t \to \infty} y(t) = \tilde{y}.$$

An analogous argument using the first and the third equation of (11) shows that either the male population (and then the total population) dies out or, if $\mathcal{R}^n > 1$,

$$\lim_{t \to \infty} z(t) = \tilde{z} := \frac{\beta \gamma_m \mathcal{K}_f^n - \nu_m}{\beta \gamma_m \mathcal{K}_f^n}.$$

Finally, we turn to the equation for F,

$$F' = \beta \gamma_f (1 - x) F - \left[\mu_f + \nu_f + b \left(\frac{1}{y} - \frac{1}{z} + \frac{1}{xz} \right) F \right] F, \qquad (15)$$

which can now be viewed as an asymptotically autonomous ODE, with the limiting equation

$$F' = \left[\beta \gamma_f \mathcal{K}_m^n - \mu_f - \nu_f - b \left(\frac{1}{\tilde{y}} - \frac{1}{\tilde{z}} + \frac{1}{\tilde{z} \mathcal{K}_f^n} \right) F \right] F. \qquad (16)$$

Again, the equation (16) is a logistic equation in F and, from (12), its solutions either approach zero (if $\mathcal{R}^n < 1$) or a positive equilibrium (if $\mathcal{R}^n > 1$). It is easy to see that (15) satisfies the requirements of Theorems 2.4 and 2.5 from Castillo-Chavez and Thieme [1995] which imply

$$\lim_{t \to \infty} F(t) = \frac{\beta \gamma_f \mathcal{K}_m^n - \mu_f - \nu_f}{b \left(\frac{1}{\tilde{y}} - \frac{1}{\tilde{z}} + \frac{1}{\tilde{z} \mathcal{K}_f^n} \right)}.$$

This limit (after a long but straightforward computation) can be shown to be equal to $\tilde{F}$. Thus,

$$\lim_{t \to \infty} F(t) = \tilde{F}.$$

Altogether, the interior equilibrium $(\tilde{F}, \tilde{M}, \tilde{A}_f, \tilde{A}_m)$ is globally stable if and only if

$$\mathcal{R}^n > 1.$$

Conversely, if $\mathcal{R}^n < 1$, global stability of the extinction equilibrium $(\bar{F}, \bar{M}, \bar{A}_f, \bar{A}_m)$ follows. $\qquad \square$

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Theorem 4.1

PROOF. We use the next generation matrix approach developed by van den Driessche and Watmough [2002]. Following the equations for the infected classes only, and using the notation of van den Driessche and Watmough [2002], we have the following infection and removal rates from each of the infected classes:

$$
\mathcal{G} = \begin{bmatrix} \lambda_f \frac{F\chi}{F+M+\varphi+\chi} \\ \lambda_m \frac{M\varphi}{F+M+\varphi+\chi} \\ 0 \\ 0 \end{bmatrix}, \quad \mathcal{V} = \begin{bmatrix} (\mu_f + bP)\varphi + \alpha_f\varphi \\ (\mu_m + bP)\chi + \alpha_m\chi \\ -\alpha_f\varphi + (\mu_f + bP)A_\varphi \\ -\alpha_m\chi + (\mu_m + bP)A_\chi \end{bmatrix},
$$

which implies

$$
G = \begin{bmatrix} -\frac{\lambda_f F\chi}{(F+M+\varphi+\chi)^2} & \frac{\lambda_f F}{F+M+\varphi+\chi} - \frac{\lambda_f F\chi}{(F+M+\varphi+\chi)^2} & 0 & 0 \\ \frac{\lambda_m M}{F+M+\varphi+\chi} - \frac{\lambda_m M\varphi}{(F+M+\varphi+\chi)^2} & -\frac{\lambda_m M\varphi}{(F+M+\varphi+\chi)^2} & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix},
$$

$$
V = \begin{bmatrix} \tilde{\mu}_f + b\varphi + \alpha_f & b\varphi & b\varphi & b\varphi \\ b\chi & \tilde{\mu}_m + b\chi + \alpha_m & b\chi & b\chi \\ bA_\varphi - \alpha_f & bA_\varphi & \tilde{\mu}_f + bA_\varphi & bA_\varphi \\ bA_\chi & bA_\chi - \alpha_m & bA_\chi & \tilde{\mu}_m + bA_\chi \end{bmatrix}.
$$

The matrices G and V are actually composed of partial derivatives of the components of vectors $\mathcal{G}$ and $\mathcal{V}$, respectively, with respect to the infected classes of the model; see van den Driessche and Watmough [2002] for more details. Furthermore, we have

$$
G(0,0,0,0) = \begin{bmatrix} 0 & \frac{\lambda_f \tilde{F}}{\tilde{F}+\tilde{M}} & 0 & 0 \\ \frac{\lambda_m \tilde{M}}{\tilde{F}+\tilde{M}} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix},
$$

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$$V^{-1}(0,0,0,0) = \begin{bmatrix} \frac{1}{\tilde{\mu}_f + \alpha_f} & 0 & 0 & 0 \\ 0 & \frac{1}{\tilde{\mu}_m + \alpha_m} & 0 & 0 \\ \frac{\alpha_f}{\tilde{\mu}_f(\tilde{\mu}_f + \alpha_f)} & 0 & \frac{1}{\tilde{\mu}_f} & 0 \\ 0 & \frac{\alpha_m}{\tilde{\mu}_m(\tilde{\mu}_m + \alpha_m)} & 0 & \frac{1}{\tilde{\mu}_m} \end{bmatrix},$$

$$GV^{-1}(0,0,0,0) = \begin{bmatrix} 0 & \frac{\lambda_f \tilde{F}}{\tilde{M}(\tilde{\mu}_m + \alpha_m)} & 0 & 0 \\ \frac{\lambda_m \tilde{M}}{\tilde{F}(\tilde{\mu}_f + \alpha_f)} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}.$$

As proven in van den Driessche and Watmough [2002], the basic reproduction number of the infection is the spectral radius of the latter matrix, i.e.

$$\mathcal{R}_0^n = \rho(GV^{-1}) = \frac{\sqrt{\lambda_f \lambda_m \tilde{F}\tilde{M}}}{(\tilde{F} + \tilde{M})\sqrt{(\tilde{\mu}_f + \alpha_f)(\tilde{\mu}_m + \alpha_m)}}.$$

$\square$

Theorem 4.2

PROOF. We use the theory of asymptotically autonomous planar systems established by Thieme and Castillo-Chavez in Thieme [1992] and Castillo-Chavez and Thieme [1995] to study behavior of the model (5), using a limiting planar system in $\varphi, \chi, A_\varphi,$ and A_χ. Notice that the equations for φ and χ in (5),

$$\begin{aligned} \varphi' &= \lambda \frac{F\chi}{F+M+\varphi+\chi} - (\mu_f + bP)\varphi - \nu_f \varphi := \bar{f}(t, \varphi, \chi), \\ \chi' &= \lambda \frac{M\varphi}{F+M+\varphi+\chi} - (\mu_m + bP)\chi - \nu_m \chi := \bar{g}(t, \varphi, \chi), \end{aligned} \tag{17}$$

form an asymptotically autonomous system in φ, χ, with the following limiting system:

$$\begin{aligned} \varphi' &= \frac{\lambda}{\tilde{F}+\tilde{M}}(\tilde{F} - \varphi)\chi - (\tilde{\mu}_f + \nu_f)\varphi := f(\varphi, \chi), \\ \chi' &= \frac{\lambda}{\tilde{F}+\tilde{M}}(\tilde{M} - \chi)\varphi - (\tilde{\mu}_m + \nu_m)\chi := g(\varphi, \chi). \end{aligned} \tag{18}$$

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The Poincaré-Bendixson-type trichotomoy established by Thieme and Castillo-Chaves in Thieme [1992] and Castillo-Chavez and Thieme [1995] ensures that every bounded forward solution of (17) converges to an equilibrium of the limiting system (18). All solutions of (5) and, consequently, of (18) are obviously bounded in the positive quadrant due to the assumption of negative density dependence in host mortality. It remains to establish the local stability conditions for the interior equilibria in the limiting system and to exclude the possibility of periodic solutions. Furthermore, in the endemic case, we will show that the DFE is a weak repeller for (17) which will ensure that the basins of attraction of the DFE and the endemic steady state are the same as those in the limiting system (18). From these the global stability of the DFE and the endemic steady state will follow.

In order to check the local stability of the equilibrium points, we will compute the Jacobian of (18):

$$
J(\varphi, \chi) = \begin{bmatrix} -\frac{\lambda\chi}{\tilde{F}+\tilde{M}} - (\tilde{\mu}_f + \nu_f) & \frac{\lambda(\tilde{F}-\varphi)}{\tilde{F}+\tilde{M}} \\ \frac{\lambda(\tilde{M}-\chi)}{\tilde{F}+\tilde{M}} & -\frac{\lambda\varphi}{\tilde{F}+\tilde{M}} - (\tilde{\mu}_m + \nu_m) \end{bmatrix}.
$$

From this we see that

$$
TrJ(\varphi, \chi) = -\frac{\lambda\chi}{\tilde{F}+\tilde{M}} - (\tilde{\mu}_f + \nu_f) - \frac{\lambda\varphi}{\tilde{F}+\tilde{M}} - (\tilde{\mu}_m + \nu_m) < 0
$$

for all possible positive values of φ and χ. In addition,

$$
\det J(\varphi, \chi) = \frac{\lambda\varphi}{\tilde{F}+\tilde{M}}\left(\tilde{\mu}_f + \nu_f + \lambda\frac{\tilde{M}}{\tilde{F}+\tilde{M}}\right) +
$$

$$
+\frac{\lambda\chi}{\tilde{F}+\tilde{M}}\left(\tilde{\mu}_m + \nu_m + \lambda\frac{\tilde{F}}{\tilde{F}+\tilde{M}}\right) + (\tilde{\mu}_f + \nu_f)(\tilde{\mu}_m + \nu_m) - \lambda^2\frac{\tilde{F}\tilde{M}}{(\tilde{F}+\tilde{M})^2}.
$$

From this expression, $\det(J(0,0)) > 0$ if and only if

$$
\lambda^2\frac{\tilde{F}\tilde{M}}{(\tilde{F}+\tilde{M})^2(\tilde{\mu}_f + \nu_f)(\tilde{\mu}_m + \nu_m)} < 1 \Leftrightarrow \mathcal{L}_0^n < 1,
$$

and $\det(J(\varphi^o, \chi^o)) > 0$ if and only if

$$\lambda^2 \frac{\tilde{F}\tilde{M}}{(\tilde{F} + \tilde{M})^2(\tilde{\mu}_f + \nu_f)(\tilde{\mu}_m + \nu_m)} > 1 \Leftrightarrow \mathcal{L}_0^n > 1.$$

Hence the DFE is locally asymptotically stable if $\mathcal{L}_0^n < 1$ and it is the only equilibrium in the biologically feasible region. Conversely, if $\mathcal{L}_0^n > 1$, the DFE is unstable and the endemic equilibrium is locally asymptotically stable.

The existence of periodic solutions is ruled out by the Poincaré-Bendixson Criterion:

$$\frac{\partial f}{\partial \varphi} + \frac{\partial g}{\partial \chi} = -\frac{\lambda \chi}{\tilde{F} + \tilde{M}} - (\tilde{\mu}_f + \nu_f) - \frac{\lambda \varphi}{\tilde{F} + \tilde{M}} - (\tilde{\mu}_m + \nu_m) < 0,$$

which proves the global stability of the equilibria analyzed above.

Assuming $\mathcal{L}_0^n > 1$ we now show, by contradiction, that $(0,0)$ is a weak repeller of (17). Suppose that $(\varphi, \chi) \to (0,0)$ with positive initial values. Then (17) can be written in the following way

$$\begin{array}{rl} \varphi' &= m(t)\chi - n(t)\varphi, \\ \chi' &= p(t)\varphi - q(t)\chi, \end{array} \qquad (19)$$

with

$$m(t) \to \frac{\lambda \tilde{F}}{\tilde{F} + \tilde{M}} := m_1, \quad n(t) \to \tilde{\mu}_f + \nu_f := n_1,$$

$$p(t) \to \frac{\lambda \tilde{M}}{\tilde{F} + \tilde{M}} := p_1, \quad q(t) \to \tilde{\mu}_m + \nu_m := q_1.$$

Notice that, with these notations, the condition $\mathcal{L}_0^n > 1$ is equivalent to

$$m_1 p_1 > n_1 q_1.$$

From (19) we have

$$\frac{p_1 \varphi' + n_1 \chi'}{\varphi + \chi} = [p_1 m(t) - n_1 q(t)]\frac{\chi}{\varphi + \chi} + [n_1 p(t) - p_1 n(t)]\frac{\varphi}{\varphi + \chi},$$

and

$$\frac{q_1 \varphi' + m_1 \chi'}{\varphi + \chi} = [m_1 p(t) - q_1 n(t)]\frac{\varphi}{\varphi + \chi} + [q_1 m(t) - m_1 q(t)]\frac{\chi}{\varphi + \chi}.$$

Combining these equalities leads to

$$\frac{(p_1+q_1)\varphi' + (m_1+n_1)\chi'}{\varphi+\chi} = [p_1m(t)-n_1q(t)]\frac{\chi}{\varphi+\chi}+[m_1p(t)-q_1n(t)]\frac{\varphi}{\varphi+\chi}+$$

$$+[n_1p(t)-p_1n(t)]\frac{\varphi}{\varphi+\chi} + [q_1m(t)-m_1q(t)]\frac{\chi}{\varphi+\chi}.$$

Setting $K_1 := p_1 + q_1$, $T_1 := m_1 + n_1$ and $K := \max\{K_1, T_1\}$, we obtain the following inequality

$$\frac{(K_1\varphi + T_1\chi)'}{K_1\varphi + T_1\chi} = \frac{\varphi+\chi}{K_1\varphi + T_1\chi}\left\{[p_1m(t) - n_1q(t)]\frac{\chi}{\varphi+\chi} + [m_1p(t) - q_1n(t)]\frac{\varphi}{\varphi+\chi}+\right.$$

$$\left.+[n_1p(t) - p_1n(t)]\frac{\varphi}{\varphi+\chi} + [q_1m(t) - m_1q(t)]\frac{\chi}{\varphi+\chi}\right\} >$$

$$> \frac{1}{K}\left\{[p_1m(t) - n_1q(t)]\frac{\chi}{\varphi+\chi} + [m_1p(t) - q_1n(t)]\frac{\varphi}{\varphi+\chi}+\right.$$

$$\left.+[n_1p(t) - p_1n(t)]\frac{\varphi}{\varphi+\chi} + [q_1m(t) - m_1q(t)]\frac{\chi}{\varphi+\chi}\right\}.$$

Finally, since

$$p_1m(t) - n_1q(t) \to m_1p_1 - n_1q_1, \quad m_1p(t) - q_1n(t) \to m_1p_1 - n_1q_1,$$

$$n_1p(t) - p_1n(t) \to 0, \quad \text{and} \quad q_1m(t) - m_1q(t) \to 0,$$

it follows that

$$\lim_{t\to\infty}\frac{d}{dt}\ln(K_1\varphi + T_1\chi) \geq \frac{1}{K}(m_1p_1 - n_1q_1) > 0$$

which is a contradiction.

Having established the limits for φ and χ, the limits for A_φ and A_χ follow immediately from the last two equations of the model (5). $\qquad\square$

Theorem 4.3

PROOF. We proceed as in the previous cases by re-writing the model (7) in a more convenient equivalent form in terms of

$$x := \frac{F}{F + \varphi}, \ P := 2(F + \varphi + A_f + A_\varphi), \ F, \ \text{and} \ A_f :$$

$$x' = \tfrac{1}{2}(1 - x)\left\{\tfrac{\beta}{2} - x[\lambda - 2(\alpha - \nu)]\right\},$$

$$P' = \tfrac{\beta}{2}\tfrac{F}{x} - (\mu + bP)P,$$

$$F' = \tfrac{\beta}{4}\tfrac{F}{x} - \tfrac{\lambda}{2}(1 - x)F - (\mu + \nu + bP)F,$$

$$A'_f = \nu F - (\mu + bP)A_f.$$

(20)

Standard ODE theory ensures that $x(t)$ is defined for all $t \geq 0$ which, in turn implies that the solutions of the full model (20) and hence (7) are defined for all $t \geq 0$. The biologically feasible domain for x is the interval $[0, 1]$, and $(0, 1)$ is invariant for x. Thus, the equation for x has two equilibrium points:

$$\tilde{x} := 1 \ \text{and} \ \hat{x} := \frac{\beta/2}{\lambda - 2(\alpha - \nu)}$$

where $\hat{x}$ is biologically feasible if and only if

$$\lambda > \frac{\beta}{2} + 2(\alpha - \nu)$$

Thus we have the following main result concerning the equation in x:

$$\lim_{t \to \infty} x(t) = \hat{x} \ \text{if} \ \lambda > \frac{\beta}{2} + 2(\alpha - \nu) \ \text{and} \ \lim_{t \to \infty} x(t) = \tilde{x} \ \text{if} \ \lambda < \frac{\beta}{2} + 2(\alpha - \nu).$$

Since $x(t)$ approaches a positive limit, the second and the third equations of (20) form an asymptotically autonomous system in P and F. The limiting behavior of its solutions can be analyzed using the same technique regardless of whether $x(t) \to \tilde{x}$ or $x(t) \to \hat{x}$. Thus, we denote by x^* the limit of $x(t)$. We will later replace x^* by either $\tilde{x}$ or $\hat{x}$. With this notation the limiting system in P and F becomes

$$P' = \tfrac{\beta}{2}\tfrac{F}{x^*} - (\mu + bP)P,$$

$$F' = \tfrac{\beta}{4}\tfrac{F}{x^*} - \tfrac{\lambda}{2}(1 - x^*)F - (\mu + \nu + bP)F.$$

(21)

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There are two possible equilibria: $(\bar{P}, \bar{F}) = (0, 0)$ and

$$(P^*, F^*) = \left(\frac{1}{b} \left[\frac{\beta}{4x^*} - \frac{\lambda}{2}(1 - x^*) - \mu - \nu \right], \frac{2x^* P^*(\mu + bP^*)}{\beta} \right).$$

Notice that the second one is feasible if and only if $\frac{\beta}{4x^*} - \frac{\lambda}{2}(1-x^*) - \mu - \nu > 0$. The Jacobian of (21) is

$$J(P, F) = \begin{bmatrix} -\mu - 2bP & \frac{\beta}{2x^*} \\ -bF & \frac{\beta}{4x^*} - \frac{\lambda}{2}(1 - x^*) - (\mu + \nu + bP) \end{bmatrix}.$$

Evaluated at the equilibrium points, this is

$$J(0, 0) = \begin{bmatrix} -\mu & \frac{\beta}{2x^*} \\ 0 & \frac{\beta}{4x^*} - \frac{\lambda}{2}(1 - x^*) - (\mu + \nu) \end{bmatrix}$$

and

$$J(P^*, F^*) = \begin{bmatrix} -\mu - 2bP^* & \frac{\beta}{2x^*} \\ -bF^* & 0 \end{bmatrix}.$$

From the sign of the trace and the determinant of J we have the following result:

- If $\frac{\beta}{4x^*} - \frac{\lambda}{2}(1 - x^*) < \mu + \nu$ then $(0, 0)$ is locally asymptotically stable and (P^*, F^*) is not feasible

- If $\frac{\beta}{4x^*} - \frac{\lambda}{2}(1 - x^*) > \mu + \nu$ then $(0, 0)$ is unstable and (P^*, F^*) is feasible and locally asymptotically stable.

Notice also that, in the second case, $(0, 0)$ is a weak repeller for the non-autonomous system in P and F. Indeed, if $(P, F) \to (0, 0)$ then

$$\lim_{t \to \infty} \frac{d}{dt} \ln F = \frac{\beta}{4x^*} - \frac{\lambda}{2}(1 - x^*) - \mu - \nu > 0,$$

which is a contradiction.

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Also, the limiting system (21) does not have periodic solutions as we can see from the Dulac's Criterion:

$$\frac{\partial}{\partial P}\left(\frac{1}{PF}P'\right) + \frac{\partial}{\partial F}\left(\frac{1}{PF}F'\right) = -\frac{\beta}{2x^*P^2} - \frac{b}{F} < 0.$$

It follows now from the theory of asymptotically autonomous systems [Castillo-Chavez and Thieme, 1995] that

$$(P,F) \to (0,0) \text{ if } \frac{\beta}{4x^*} - \frac{\lambda}{2}(1-x^*) < \mu + \nu$$

and

$$(P,F) \to (P^*,F^*) \text{ if } \frac{\beta}{4x^*} - \frac{\lambda}{2}(1-x^*) > \mu + \nu.$$

Replacing now x^* with either $\tilde{x}$ or $\hat{x}$ we obtain the results stated in the theorem.

Acknowledgements

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Figure legends:

Figure 1: Dynamics of the model (5) in case there are no sexually abstaining groups and $\mathcal{R}_0 > 1$. These conditions together imply that the infection invades the host population and attains the endemic equilibrium. Parameter values: $\beta = 0.2$, $\gamma_f = 0.7$, $\gamma_m = 0.3$, $\mu_f = 0.012$, $\mu_m = 0.013$, $b = 0.00002$, $\lambda_f = \lambda_m = 0.2$, $\nu_f = \nu_m = \alpha_f = \alpha_m = 0$. In this case, $\mathcal{R}_0 = 2.18 > 1$.

Figure 2: Dynamics of the model (5) in case the transition rates to the sexually abstaining groups are infection-status-independent and $\mathcal{R}_0 > 1$. These conditions together imply that the infection invades the host population and attains the endemic equilibrium. Parameter values: $\beta = 0.2$, $\gamma_f = 0.7$, $\gamma_m = 0.3$, $\mu_f = 0.012$, $\mu_m = 0.013$, $b = 0.00002$, $\lambda_f = \lambda_m = 0.2$, $\nu_f = \alpha_f = 0.03$, $\nu_m = \alpha_m = 0.01$. Notice that the basic reproduction number of the infection is here the same as in Fig.1: $\mathcal{R}_0^n = 2.18 > 1$.

Figure 3: Dynamics of the model (5) in case the transition rates to the sexually abstaining groups are infection-status-dependent, with the transition rates of the infected individuals to the sexually abstaining group higher than those of the susceptible individuals, and $\mathcal{R}_0^n < 1 < \mathcal{R}_0$. These conditions together imply that the disease-free equilibrium, unstable in the absence of the sexually abstaining groups, is stable in their presence. Parameter values: $\beta = 0.2$, $\gamma_f = 0.7$, $\gamma_m = 0.3$, $\mu_f = 0.012$, $\mu_m = 0.013$, $b = 0.00002$, $\lambda_f = \lambda_m = 0.2$, $\nu_f = 0.03$, $\alpha_f = 0.09$, $\nu_m = 0.01$, $\alpha_m = 0.08$. In this case, $\mathcal{R}_0 = 2.18 > 1$ and $\mathcal{R}_0^n = 0.88 < 1$.
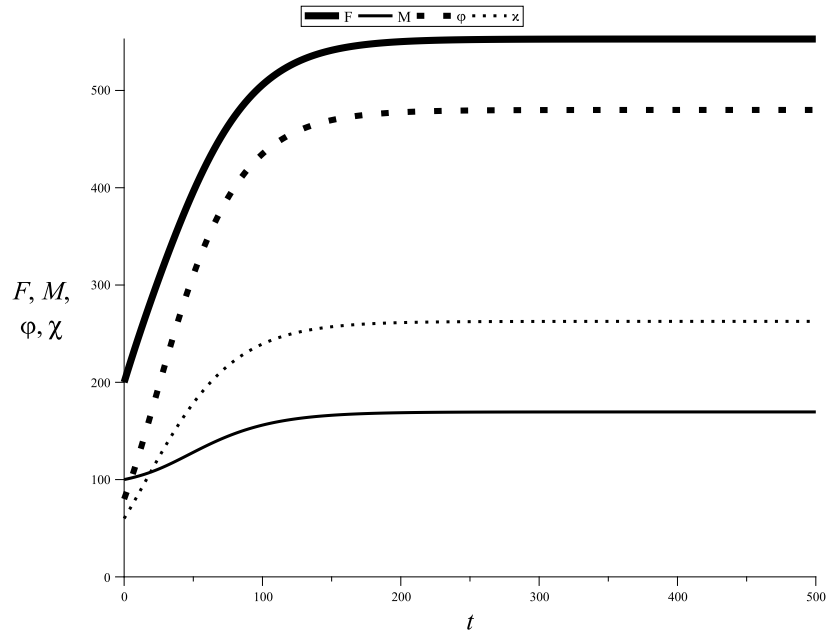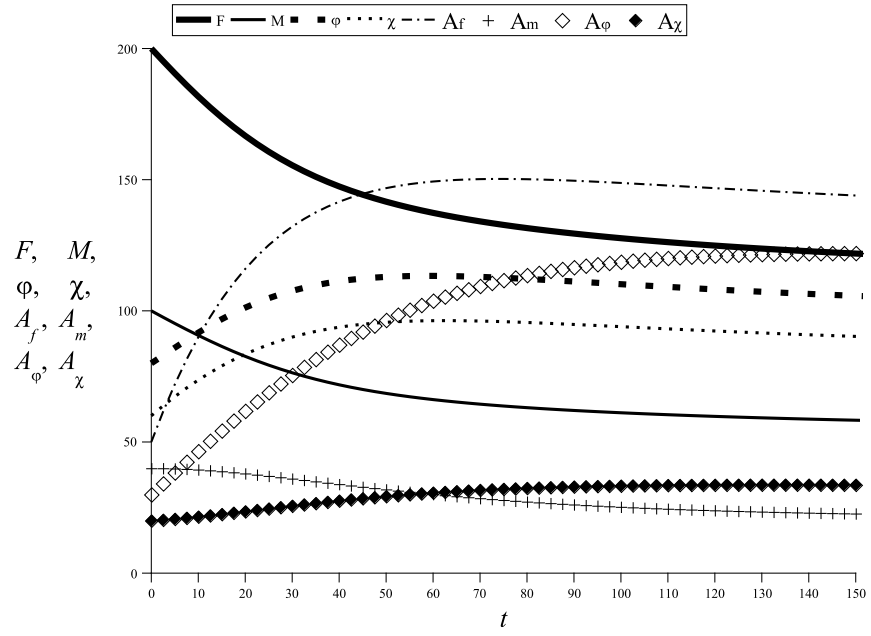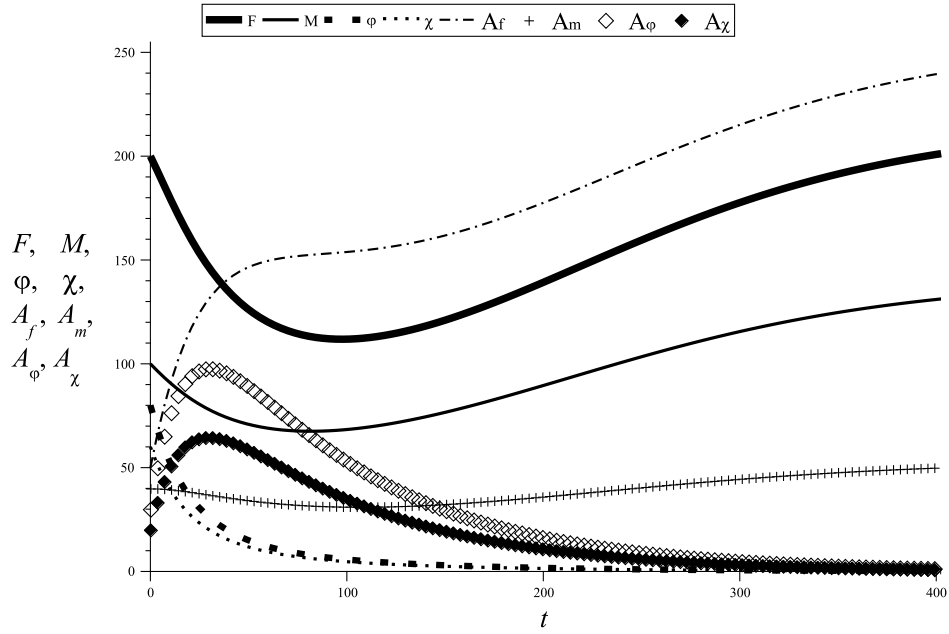
Figure 1:

Figure 2:

Figure 3: