

RESEARCH ARTICLE

The influence of sexually active non-reproductive groups on persistent sexually transmitted diseases

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We describe several population models exposed to a mild life-long sexually transmitted disease, i.e. without significant increased mortality among infected individuals and providing no immunity/recovery. We then modify these models to include non-reproductive groups consisting of those isolated from sexual contact and those who are sexually active but infertile due to choice, medical or other reasons. We analyze the potential effect on the dynamics of the population. We are interested in how the isolated class may curb the growth of the infected group while keeping the healthy population at acceptable levels. We also analyze the difference between being sexually active and abstained within the non-reproductive class and its impact on the epidemic reproductive number and the nature of the bifurcation around the disease-free equilibrium. We provide a comparison with our models introduced in a previous paper which include only the isolated from sexual contact class.

Keywords: sexually transmitted diseases; isolation; population models; non-reproductive groups

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1. Introduction

The dynamics of a population is influenced primarily by the long-term or *permanent* changes in the fertility and mortality. One factor with a long-term effect on population growth is the segregation of reproductive individuals into non-reproductive classes represented by portions of the general population who by choice, social or medical reasons remain *childless for life*.

In [4] Milner introduced several demographic exponential and logistic models, both one-sex and gender structured, that include non-reproductive groups. These models do not include the *temporary* isolation from reproduction exhibited by people who voluntarily postpone having children through birth control. They, rather, take into account individuals who, for any reason, enter the isolated groups and remain there forever. Several examples are provided by same-sex isolation groups such as prisoners, certain religious groups or life-long homosexual groups. Permanent sterility provides a medical cause for being non-reproductive while other individuals simply choose to remain without progeny for life.

A similar analysis of these models, also incorporating a sexually transmitted disease that does not increase mortality, was performed by D. Maxin and F.A.

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Milner in [3]. An example of an incurable yet mild disease is herpes simplex type 2 (HSV-2).

However, in this research we are not proposing models for herpes or any other specific STD. Our intention is to analyze how much and in what form isolation from reproduction alters the demographic trend in general and the spread of the disease in particular in a population exposed to a sexually transmitted disease. In [3] we considered the sexually abstained class only and one of the main results was that the isolation from sexual contact can induce the stability of the disease-free equilibrium in an otherwise endemic situation, keeping the total population bounded away from zero. We showed that there is a range of the isolation rates that produces enough quarantine protection while the undesirable side effect of reducing the size of the newborns is small enough to maintain a reasonable population size.

In this paper we analyze the more realistic situation that incorporates groups of individuals who are non-procreating but are sexually active in addition to those who abstain from all sexual contact. A surprising result is that, by incorporating the sexually active and non-reproductive groups, the disease-free stability range for the infection rate can be larger than the one obtained in the presence of the abstained class only. Furthermore, we study the epidemiological impact of isolation from reproduction alone, i.e. without the abstinent individuals. Abstinence acts also as quarantine and recovery from the infection, both effects being well established in the literature such as [2]. By including only sexually active groups, we can measure the potential of isolation from reproduction in curbing the disease spread.

We propose a logistic model with non-linear mortality to account for these two distinct classes of people who become permanently non-reproductive:

- *non-reproductive*, sexually active, usually represented by individuals who more or less often engage in sexual activity while they maintain their decision not to have offspring, and
- *abstained*, or sexually inactive.

The paper is structured as follows: in Section 2 we introduce the logistic model with both the abstained and the non-reproductive groups and we derive an extinction threshold. In all subsequent sections we assume that the total population does not decline to zero regardless of its initial size. In Section 3 we compute the epidemic reproductive number and derive a similar condition on the infection rates as in [3] that ensures the stability of the disease-free equilibrium (DFE). We analyze the change in this condition when adding the sexually active non-reproductive class to the model that already has the abstained one. The endemic steady state is treated in Section 4 for one particular model that includes only the sexually active non-reproductive classes and assumes equal isolation rates for susceptibles and infected individuals. We conclude in Section 5 with an analysis of the Center Manifold corresponding to the situation when the epidemic reproductive number is equal to 1 that gives an explanation of the impact of assuming different isolation rates for susceptibles and infected.

2. The model

We consider 5 classes of individuals according to their status with respect to them being infected, non-reproductive, or isolated from sexual contact:

- S_r, S_n : the susceptibles, reproductive and non-reproductive,
- I_r, I_n : the similar infectious classes,

- A : the isolated from sexual contact, healthy or infected.

The total population is, therefore:

$$P(t) = S_r(t) + S_n(t) + I_r(t) + I_n(t) + A(t),$$

and the logistic death rate will be denoted

$$\bar{\mu}(P) = \mu + bP.$$

The other parameters are:

- β , is the *per capita* birth rate, and we consider all newborn to be initially reproductive,
- λ_1 and λ_2 , the infection rates of the reproductive and sexually active non-reproductive individuals,
- α_1 and α_2 , the transition rates into the sexually active non-reproductive groups,
- ν_1 and ν_2 , the transition rates from S_r and I_r into the abstained class A and
- γ_1 and γ_2 , the transition rates from S_n and I_n into the abstained class A .

The model is as follows:

$$\begin{cases} S_r' = \beta(S_r + I_r) - \lambda_1 S_r I_r - \lambda_2 S_r I_n - (\alpha_1 + \nu_1) S_r - \bar{\mu}(P) S_r, \\ S_n' = \alpha_1 S_r - \lambda_1 S_n I_r - \lambda_2 S_n I_n - \gamma_1 S_n - \bar{\mu}(P) S_n, \\ I_r' = \lambda_1 S_r I_r + \lambda_2 S_r I_n - (\alpha_2 + \nu_2) I_r - \bar{\mu}(P) I_r, \\ I_n' = \alpha_2 I_r + \lambda_1 S_n I_r + \lambda_2 S_n I_n - \gamma_2 I_n - \bar{\mu}(P) I_n, \\ A' = \nu_1 S_r + \nu_2 I_r + \gamma_1 S_n + \gamma_2 I_n - \bar{\mu}(P) A. \end{cases} \quad (1)$$

The system is well-posed and the positive invariance of \mathbf{R}_+^5 can be established using standard methods. Moreover, due to the presence of the logistic death rate $\bar{\mu}(P)$ the solution is bounded for all time t .

We consider only one abstained class A to keep the model simple and also because any permanently infected individual in it will no longer spread the disease. Another pertinent remark is that once a steady state is reached with $I_r = 0$ and $I_n = 0$, the abstained class A will necessarily contain healthy people only.

We now show that the total population declines to zero if the non-reproductive rates ν_1 , ν_2 , α_1 and α_2 are too large when compared with the natural growth rate.

In our previous paper [3] that considers only the sexually abstained class A , we found that the extinction equilibrium is globally stable if

$$\beta - \mu < \min\{\nu_1, \nu_2\},$$

and it is locally asymptotically stable if

$$\beta - \mu < \nu_1.$$

A similar result holds for the model analyzed in this paper:

Proposition 2.1:

The extinction equilibrium $(0, 0, 0, 0, 0)$ satisfies:

- *If $\beta - \mu < \min\{\alpha_1 + \nu_1, \alpha_2 + \nu_2\}$ the extinction equilibrium is globally asymptotically stable,*
- *If $\beta - \mu > \alpha_1 + \nu_1$ the extinction equilibrium is unstable and*
- *If $\beta - \mu < \alpha_1 + \nu_1$ the extinction equilibrium is locally asymptotically stable and the total population $P(t)$ declines to zero or stays bounded away from zero depending on the initial conditions in (1).*

Proof: To prove the global stability condition, we use a suitable bounding equation for $S_r + I_r$:

$$(S_r + I_r)' \leq \beta(S_r + I_r) - \min\{\alpha_1 + \nu_1, \alpha_2 + \nu_2\}(S_r + I_r) - (\mu + b(S_r + I_r))(S_r + I_r).$$

This is in fact a logistic equation in $S_r + I_r$ that declines to zero whenever

$$\beta - \mu < \min\{\alpha_1 + \nu_1, \alpha_2 + \nu_2\}.$$

Summing up all the equations of (1) we obtain the following ODE for the total population P :

$$P' = \beta(S_r + I_r) - (\mu + bP)P.$$

Since

$$\lim_{t \rightarrow \infty} (S_r(t) + I_r(t)) = 0,$$

for any $\epsilon > 0$ we can choose a time t_0 such that

$$S_r(t) + I_r(t) < \epsilon \quad \text{for every } t > t_0.$$

It follows that

$$P' \leq \beta\epsilon - (\mu + bP)P \leq \beta\epsilon - \mu P \quad \text{whenever } t > t_0.$$

Integrating this inequality, we obtain the following upper bound for $P(t)$:

$$P(t) \leq \frac{\beta\epsilon}{\mu} + \left[P(t_0) - \frac{\beta\epsilon}{\mu} \right] e^{-\mu(t-t_0)}, \quad \text{whenever } t > t_0.$$

We now have two cases: If

$$P(t_0) - \frac{\beta\epsilon}{\mu} \leq 0 \quad \text{then} \quad P(t) \leq \frac{\beta\epsilon}{\mu},$$

otherwise we can choose t big enough so that $e^{-\mu(t-t_0)} < \epsilon$, i.e.

$$t^* = \max \left\{ t_0, t_0 - \frac{\ln \epsilon}{\mu} \right\},$$

and the upper bound computed above becomes

$$P(t) \leq \left[\frac{\beta}{\mu} + P(t_0) - \frac{\beta\epsilon}{\mu} \right] \epsilon \quad \text{whenever } t > t^*.$$

In both cases, since ϵ can be chosen arbitrarily small, this proves the global stability of the extinction equilibrium in the conditions stated in the proposition.

The local stability threshold comes from analyzing the Jacobian of (1) evaluated at the origin:

$$J[0, 0, 0, 0, 0] = \begin{pmatrix} \beta - \mu - \alpha_1 - \nu_1 & 0 & \beta & 0 & 0 \\ \alpha_1 & -\mu - \gamma_1 & 0 & 0 & 0 \\ 0 & 0 & -\mu - \alpha_2 - \nu_2 & 0 & 0 \\ 0 & 0 & \alpha_2 & -\mu - \gamma_2 & 0 \\ \nu_1 & \gamma_1 & \nu_2 & \gamma_2 & -\mu. \end{pmatrix}.$$

All its eigenvalues are negative provided that

$$\beta - \mu - \alpha_1 - \nu_1 < 0.$$

□

Remark 1: Note that the global stability condition in this proposition is only sufficient but not necessary as shown in the example below.

If $\alpha_2 + \nu_2 < \beta - \mu < \alpha_1 + \nu_1$ the total population declines to zero only if $P(t)$ starts close enough to the origin. Furthermore, there exists two positive steady states which are difficult to compute in general. One of them is unstable and another one is locally asymptotically stable. In the next figure we illustrate this by plotting the total population $P(t)$ for several values of the initial population size $P(0)$ in order to emphasize the basins of attraction of the extinction equilibrium and that of the stable interior steady state.

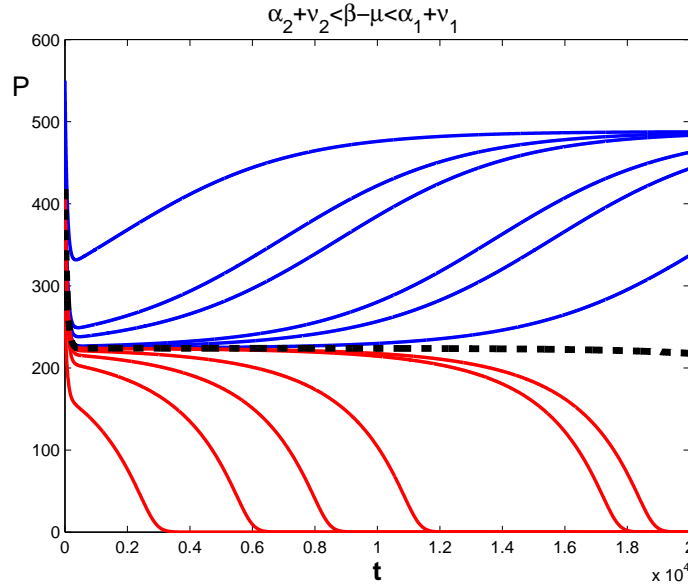


Figure 1. Case of two positive steady states.

$$\beta = 0.01442 \quad \mu = 0.01303 \quad \lambda = 0.00075 \quad \nu_1 = 0.008 \quad \nu_2 = 0.0002 \quad \alpha_1 = \alpha_2 = 0.0002$$

It follows from this proposition that the net reproductive number for a population that includes isolation from reproduction is $\mathcal{R} = \frac{\beta}{\mu + \alpha_1 + \nu_1}$ which represents the average offspring production from susceptibles during their expected *reproductive* life-time. Under normal conditions the total population remains bounded away from zero unless catastrophic changes occur in the natural mortality and fertility. Consequently, throughout this paper, we will assume

$$\mathcal{R} > 1 \quad \text{or} \quad \beta - \mu - \alpha_1 - \nu_1 > 0.$$

3. The disease-free equilibrium and the epidemic reproductive number

In this section we establish a threshold condition on the vital parameters that separates the disease-free steady state from an endemic situation. The system (1) admits the following disease-free equilibrium (DFE):

$$\bar{S}_r = \frac{\mu^*}{\beta} \bar{P}, \quad \bar{S}_n = \frac{\alpha_1 \mu^*}{\beta(\mu^* + \gamma_1)} \bar{P},$$

$$\bar{I}_r = 0, \quad \bar{I}_n = 0, \quad \text{and} \quad \bar{A} = \left[\frac{\nu_1}{\beta} + \frac{\alpha_1 \gamma_1}{\beta(\mu^* + \gamma_1)} \right] \bar{P},$$

where

$$\bar{P} = \bar{S}_r + \bar{S}_n + \bar{A} = \frac{\beta - \mu - \alpha_1 - \nu_1}{b} \quad \text{and} \quad \mu^* = \beta - \alpha_1 - \nu_1.$$

To study the stability of the DFE we are going to use a method provided by van den Driessche and Watmough in [1] based on the next generation matrix. This method is considerably easier than computing the eigenvalues of $J[\bar{S}_r, \bar{S}_n, \bar{I}_r, \bar{I}_n, \bar{A}]$ because it restricts the analysis to the equation corresponding to the infected classes, which reduces the dimension of the problem. We first need to write our system in the form

$$x_i' = \mathcal{F}_i(x) - \mathcal{V}_i(x),$$

where \mathcal{F} denotes the rate of appearance of new infections, which in our case is represented by the terms

$$\lambda_1 S_r I_r + \lambda_2 S_r I_n \quad \text{and} \quad \lambda_1 S_n I_r + \lambda_2 S_n I_n.$$

In general we need only consider the rate of appearance of *new* infections in the expression of \mathcal{F} , so we do not include $\alpha_2 I_r$, $\nu_2 I_r$ and $\gamma_2 I_n$ in \mathcal{F} because they represent a transfer of already infected individuals from one compartment to another.

Remark 1: Note that a “disease-free” equilibrium in our model neglects the infected people that are part of the abstained class A . In other words, we do not consider A as being an infected compartment although A does contain infected and healthy abstained people. However, the abstained people do not have any epidemiological effect on the population since there is no transfer back from the abstained people into the sexually active groups. As soon as I_r and I_n approach

zero, the remaining infected people in the abstained class A will eventually disappear through natural mortality. Consequently, at the disease-free steady state, the abstained class A contains healthy people only.

In order to apply the method in [1] we need to verify that the DFE is stable in the absence of the disease. Without the infected groups, the system (1) becomes

$$\begin{cases} S'_r = \beta S_r - (\alpha_1 + \nu_1)S_r - \bar{\mu}(P)S_r, \\ S'_n = \alpha_1 S_r - \gamma_1 S_n - \bar{\mu}(P)S_n, \\ A' = \nu_1 S_r + \gamma_1 S_n - \bar{\mu}(P)A, \end{cases} \quad (2)$$

where $P(t) = S_r(t) + S_n(t) + A(t)$.

Proposition 3.1: *In the absence of the disease, the disease-free equilibrium, $(\bar{S}_r, \bar{S}_n, \bar{A})$, is locally asymptotically stable.*

Proof: The Jacobian of (2) is

$$J(S_r, S_n, A) = \begin{pmatrix} \beta - \alpha_1 - \nu_1 - \bar{\mu}(P) - bS_r & -bS_r & -bS_r \\ \alpha_1 - bS_n & -\gamma_1 - \bar{\mu}(P) - bS_n & -bS_n \\ \nu_1 - bA & \gamma_1 - bA & -\bar{\mu}(P) - bA \end{pmatrix}.$$

At the DFE, we have

$$\bar{P} = \bar{S}_r + \bar{S}_n + \bar{A} = \frac{\beta - \mu - \alpha_1 - \nu_1}{b} \quad \text{and} \quad \bar{\mu}(\bar{P}) = \beta - \alpha_1 - \nu_1.$$

Therefore,

$$J(\bar{S}_r, \bar{S}_n, \bar{A}) = \begin{pmatrix} -b\bar{S}_r & -b\bar{S}_r & -b\bar{S}_r \\ \alpha_1 - b\bar{S}_n - \gamma_1 - \bar{\mu}(\bar{P}) - b\bar{S}_n & -b\bar{S}_n & -b\bar{S}_n \\ \nu_1 - b\bar{A} & \gamma_1 - b\bar{A} & -\bar{\mu}(\bar{P}) - b\bar{A} \end{pmatrix}.$$

The eigenvalues are the zeros of the characteristic polynomial of $J(\bar{S}_r, \bar{S}_n, \bar{A})$ which is denoted by

$$f(x) = \begin{vmatrix} -b\bar{S}_r - x & -b\bar{S}_r & -b\bar{S}_r \\ \alpha_1 - b\bar{S}_n - \gamma_1 - \bar{\mu}(\bar{P}) - b\bar{S}_n - x & -b\bar{S}_n & -b\bar{S}_n \\ \nu_1 - b\bar{A} & \gamma_1 - b\bar{A} & -\bar{\mu}(\bar{P}) - b\bar{A} - x \end{vmatrix}.$$

Subtracting the third column from the first and the second one, and then, adding the first two rows to the third one, we obtain the following simpler form for $f(x)$:

$$f(x) = \begin{vmatrix} -x & 0 & -b\bar{S}_r \\ \alpha_1 & -\gamma_1 - \bar{\mu}(\bar{P}) - x & -b\bar{S}_n \\ \alpha_1 + \nu_1 + \bar{\mu}(\bar{P}) & 0 & -\bar{\mu}(\bar{P}) - b\bar{P} - x \end{vmatrix}.$$

It is clear that one eigenvalue is $x_1 = -\gamma_1 - \bar{\mu}(\bar{P})$ and the other two, x_2 and x_3 , are the roots of

$$x^2 + [\bar{\mu}(\bar{P}) + b\bar{P}]x + b\bar{S}_r[\alpha_1 + \nu_1 + \bar{\mu}(\bar{P})] = 0.$$

However,

$$x_2 + x_3 = -\bar{\mu}(\bar{P}) - b\bar{P} < 0 \quad \text{and} \quad x_2x_3 = b\bar{S}_r[\alpha_1 + \nu_1 + \bar{\mu}(\bar{P})] > 0,$$

hence all eigenvalues have negative real parts and the DFE is locally asymptotically stable as claimed. \square

We now arrange the original system of equations in the following order: I_r, I_n, S_r, S_n and A and compute \mathcal{F} and \mathcal{V} :

$$\mathcal{F} = \begin{pmatrix} \lambda_1 S_r I_r + \lambda_2 S_r I_n \\ \lambda_1 S_n I_r + \lambda_2 S_n I_n \\ 0 \\ 0 \\ 0 \end{pmatrix},$$

and

$$\mathcal{V} = \begin{pmatrix} \alpha_2 I_r + \nu_2 I_r + \bar{\mu}(P) I_r \\ -\alpha_2 I_r + \bar{\mu}(P) I_n + \gamma_2 I_n \\ -\beta(S_r + I_r) + \bar{\mu}(P) S_r + \nu_1 S_r + \alpha_1 S_r + \lambda_1 S_r I_r + \lambda_2 S_r I_n \\ -\alpha_1 S_r + \bar{\mu}(P) S_n + \lambda_1 S_n I_r + \lambda_2 S_n I_n + \gamma_1 S_n \\ -\nu_1 S_r - \nu_2 I_r + \bar{\mu}(P) A - \gamma_1 S_n - \gamma_2 I_n. \end{pmatrix}.$$

Evaluating the Jacobians $D\mathcal{F}$ and $D\mathcal{V}$ at the DFE we obtain the following block matrices:

$$D\mathcal{F} = \begin{pmatrix} F & 0 \\ 0 & 0 \end{pmatrix}, \quad D\mathcal{V} = \begin{pmatrix} V & 0 \\ J_1 & J_2 \end{pmatrix}.$$

where

$$F = \begin{pmatrix} \lambda_1 \bar{S}_r & \lambda_2 \bar{S}_r \\ \lambda_1 \bar{S}_n & \lambda_2 \bar{S}_n \end{pmatrix},$$

and

$$V = \begin{pmatrix} \bar{\mu}(\bar{P}) + \alpha_2 + \nu_2 & 0 \\ -\alpha_2 & \bar{\mu}(\bar{P}) + \gamma_2 \end{pmatrix}.$$

Cf. [1], the eigenvalues of J_2 have positive real part and V is non-singular. The epidemic reproductive number, \mathcal{R}_0 , is then defined as the spectral radius of FV^{-1} . Denoting $\mu^* = \bar{\mu}(\bar{P})$ we obtain

$$V^{-1} = \begin{pmatrix} \frac{1}{\mu^* + \alpha_2 + \nu_2} & 0 \\ \frac{\alpha_2}{(\mu^* + \gamma_2)(\mu^* + \alpha_2 + \nu_2)} & \frac{1}{\mu^* + \gamma_2} \end{pmatrix}$$

and

$$FV^{-1} = \begin{pmatrix} \frac{\lambda_1 \bar{S}_r}{\mu^* + \alpha_2 + \nu_2} + \frac{\alpha_2 \lambda_2 \bar{S}_r}{(\mu^* + \gamma_2)(\mu^* + \alpha_2 + \nu_2)} & \frac{\lambda_2 \bar{S}_r}{\mu^* + \gamma_2} \\ \frac{\lambda_1 \bar{S}_n}{\mu^* + \alpha_2 + \nu_2} + \frac{\alpha_2 \lambda_2 \bar{S}_n}{(\mu^* + \gamma_2)(\mu^* + \alpha_2 + \nu_2)} & \frac{\lambda_2 \bar{S}_n}{\mu^* + \gamma_2} \end{pmatrix}.$$

The biological interpretation of the entries of V^{-1} is as follows:

- $\frac{1}{\mu^* + \alpha_2 + \nu_2}$ represents the average time spent by a newly introduced infected reproductive individual in the I_r class during his life time,
- $\frac{\alpha_2}{\mu^* + \alpha_2 + \nu_2}$ is the fraction of infected reproductive people that move into the isolated class I_n , therefore the (2, 1) entry represents the average time spent by an infected reproductive individual into the I_n class during his life time,
- the (1, 2) and (2, 2) entries simply mean that a non-reproductive infected individual will stay forever in either I_n or A class during his lifetime, i.e. there is no transition back from I_n and A to I_r .

A straightforward computation shows that $\det(FV^{-1}) = 0$, hence

$$\mathcal{R}_0 = \text{Tr}(FV^{-1}) = \left[\frac{\lambda_1}{\mu^* + \alpha_2 + \nu_2} + \frac{\alpha_2 \lambda_2}{(\mu^* + \gamma_2)(\mu^* + \alpha_2 + \nu_2)} \right] \bar{S}_r + \frac{\lambda_2 \bar{S}_n}{\mu^* + \gamma_2}.$$

The first two terms of \mathcal{R}_0 represent the secondary infections of susceptible reproductive individuals who either remain reproductive life-long or eventually become non-reproductive and the last term represents the secondary infections of non-reproductive susceptible individuals. Hence the DFE is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.

In [3] we showed that the group isolated from sexual contact can eliminate the infected group while keeping the healthy group bounded away from zero: if λ is within the range indicated below,

$$\frac{\beta}{K} < \lambda < \frac{\beta - \nu_1 + \nu_2}{\left(K - \frac{\nu_1}{b}\right) \left(1 - \frac{\nu_1}{\beta}\right)}, \quad (3)$$

where

$$K = \frac{\beta - \mu}{b},$$

then the DFE is stable.

Using the notation in this paper, (3) can be written as

$$\frac{\beta(\mu^* - \mu)}{\bar{P}(\mu^* - \mu + \alpha_1 + \nu_1)} < \lambda < \frac{\beta(\mu^* - \mu)(\mu^* + \alpha_1 + \nu_2)}{\bar{P}(\mu^* + \alpha_1)(\mu^* - \mu + \alpha_1)}. \quad (4)$$

The interpretation of (3) is the following:

If λ satisfies the left-hand side bound, then the disease is endemic in the absence of the isolated group; whereas if λ also satisfies the right-hand side bound, then the DFE is stable. The question now arises whether a similar result can be derived in the case of the presence of both non-reproductive but sexually active groups represented by S_n and I_n together with the isolated-from-sexual-contact group A . We will show that not only a similar result holds but also that, under certain

conditions, the range of DFE-stability for λ is even larger than before. This is a counter-intuitive effect because one would expect that the sexually active group can contribute only to the disease spread since there is no contribution to the healthy class from S_n and I_n .

We first substitute S_r and S_n in \mathcal{R}_0 with $\frac{\mu^* \bar{P}}{\beta}$ and $\frac{\alpha_1 \mu^* \bar{P}}{\beta(\mu^* + \gamma_1)}$ respectively. After rearranging the terms, we observe that the DFE stability condition, i.e. $\mathcal{R}_0 < 1$, together with the assumption that the disease is endemic in the absence of the non-reproductive groups can be written as

$$\frac{\beta}{K} < \lambda_1 \text{ and } \lambda_1 + \left[\frac{\alpha_1(\mu^* + \alpha_2 + \nu_2) + \alpha_2(\mu^* + \gamma_1)}{(\mu^* + \gamma_1)(\mu^* + \gamma_2)} \right] \lambda_2 < \frac{\beta(\mu^* + \alpha_2 + \nu_2)}{\mu^* \bar{P}}. \quad (5)$$

This condition reconciles with (3) because if we keep in the model only the groups isolated from sexual contact then, after we replace $\alpha_1 = \alpha_2 = 0$, λ_2 becomes irrelevant, and one obtains precisely (3).

In order to compare the range of the infection rate in (5) with the one in (3) or (4) we will assume that incidence of infection is the same for both reproductive and non-reproductive people, i.e. $\lambda_1 = \lambda_2 = \lambda$. Then (5) becomes

$$\frac{\beta}{K} < \lambda < \frac{\beta(\mu^* + \gamma_1)(\mu^* + \gamma_2)(\mu^* + \alpha_2 + \nu_2)}{\mu^* \bar{P} [(\mu^* + \gamma_1)(\mu^* + \alpha_2 + \gamma_2) + \alpha_1(\mu^* + \alpha_2 + \nu_2)]}. \quad (6)$$

If the range of λ in (6) is greater than the range of λ in (4) we can conclude that there is a threshold for λ which shows that in the presence of the sexually active non-reproductive class the DFE is stable whereas in the absence of it the disease is endemic. In other words we would like to verify whether it is possible for λ to satisfy

$$\frac{\beta(\mu^* - \mu)(\mu^* + \alpha_1 + \nu_2)}{\bar{P}(\mu^* + \alpha_1)(\mu^* - \mu + \alpha_1)} < \lambda < \frac{\beta(\mu^* + \gamma_1)(\mu^* + \gamma_2)(\mu^* + \alpha_2 + \nu_2)}{\mu^* \bar{P} [(\mu^* + \gamma_1)(\mu^* + \alpha_2 + \gamma_2) + \alpha_1(\mu^* + \alpha_2 + \nu_2)]}. \quad (7)$$

This is equivalent to

$$\mathcal{A} := \frac{(\mu^* + \gamma_1)(\mu^* + \gamma_2)(\mu^* + \alpha_2 + \nu_2)(\mu^* + \alpha_1)(\mu^* + \alpha_1 - \mu)}{\mu^*(\mu^* - \mu)(\mu^* + \alpha_1 + \nu_2) [(\mu^* + \gamma_1)(\mu^* + \alpha_2 + \gamma_2) + \alpha_1(\mu^* + \alpha_2 + \nu_2)]} > 1. \quad (8)$$

To simplify the interpretation of the above threshold, let us assume that the transition rates into the sexually active but non-reproductive classes are equal, i.e.

$$\alpha_1 = \alpha_2 = \alpha.$$

Then (8) becomes

$$\mathcal{A} = \frac{(\mu^* + \gamma_1)(\mu^* + \gamma_2)(\mu^* + \alpha)(\mu^* + \alpha - \mu)}{\mu^*(\mu^* - \mu) [(\mu^* + \gamma_1)(\mu^* + \alpha + \gamma_2) + \alpha(\mu^* + \alpha + \nu_2)]} > 1. \quad (9)$$

Remark 2: Notice that the left-hand side of the inequality (9) is decreasing in ν_2 and convergent to zero as $\nu_2 \rightarrow \infty$. Thus, (9) has an interesting interpretation. If we analyze the range of λ in (3) we notice that a bigger ν_2 will extend the stability range of the infection rate for the disease-free equilibrium in the model that only includes the abstained classes analyzed in [3]. This is because abstained infected individuals quarantine themselves through isolation. On the other hand, the above inequality suggests, to the contrary, that, if the infected abstinence rate ν_2 increases above a certain threshold, then $\mathcal{A} < 1$ and the stability range of

λ for the disease-free steady state will not become larger. This shows that the ability of the non-reproductive sexually active group to decrease the size of the epidemic reproductive number can be stronger than the similar effect created by the quarantine through abstinence of reproductive infected individuals.

Data from the U.S. Census 2000 shows that the *per capita* birth rate is approximately $\beta = 0.01442$ and the mortality rate $\mu = 0.01303$. Assuming equal isolation rates for both healthy and infected reproductive people, we have the following restriction on these rates that ensures the population will not decline to zero:

$$\alpha + \nu_1 < \beta - \mu = 0.00139.$$

For simplicity, we will also assume in this example that the transition rates into the abstained class A are independent of the reproductive status of any given individual, i.e.

$$\gamma_1 = \nu_1 \quad \text{and} \quad \gamma_2 = \nu_2$$

The ratio between the two bounds of λ in (7) becomes

$$\mathcal{A} = \frac{(\mu^* + \nu_1)(\mu^* + \nu_2)(\mu^* + \alpha)(\mu^* + \alpha - \mu)}{\mu^*(\mu^* + \alpha + \nu_1)(\mu^* + \alpha + \nu_2)(\mu^* - \mu)}. \quad (10)$$

It is difficult to estimate the transition rates α , ν_1 and ν_2 from real data since the reasons for being non-reproductive, whether sexually active or abstained, are not part of the usual census collection of information. Furthermore, being childless by personal choice or life style reasons is evidently a private undertaking unlikely to be accurately reflected in statistical data. However, one can reasonably assume that $\alpha > \nu_1$ and $\alpha > \nu_2$ since there are, in general, more sexually active non-reproductive individuals than those who are abstained from sexual contact. If we choose, for reference, $\alpha = 0.001$, $\nu_1 = 0.0002$ and $\nu_2 = 0.0008$ then $\mu^* = 0.01322$ and

$$\mathcal{A} \approx 5.5,$$

meaning that, with the presence of the sexually active non-reproductive groups, the infection rate λ could be more than 5 times higher and still be within the range of DFE-stability.

Below we provide a numerical example to illustrate this result. All graphs presented below use logarithmic scale for better clarity. Fig.2 shows an example when the disease is eliminated by the abstained group A in the absence of the sexually active non-reproductive groups, i.e. $\alpha_1 = \alpha_2 = 0$. By increasing 5 times the infection rate λ and still in the absence of the sexually active non-reproductive groups, we have an endemic situation as seen in Fig.3. Finally, in Fig.4 we see the role of the sexually active non-reproductive groups in causing the stability of the disease-free equilibrium. We chose the transition rate into the isolated class $\alpha = 0.001$, and the same common infection rate as in Fig.3, $\lambda_1 = \lambda_2 = 0.000075$, which satisfies condition (7).

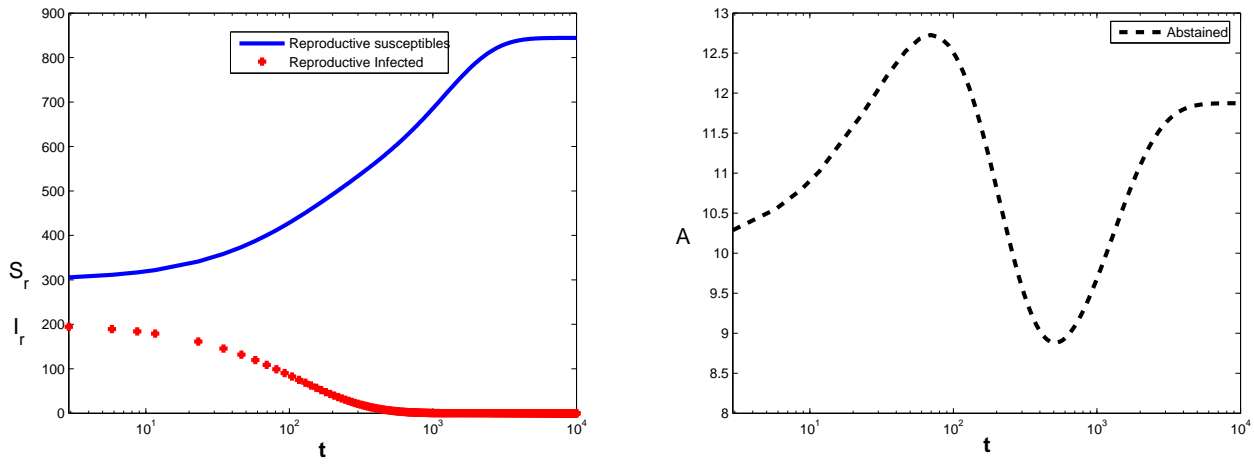


Figure 2. Case $\frac{\beta}{K} < \lambda < \frac{\beta - \nu_1 + \nu_2}{(K - \frac{\nu_1}{b})(1 - \frac{\nu_1}{\beta})}$.
 $\beta = 0.01442$ $\mu = 0.01303$ $\lambda = 0.000015$ $\nu_1 = 0.0002$ $\nu_2 = 0.0008$ $\alpha = 0$

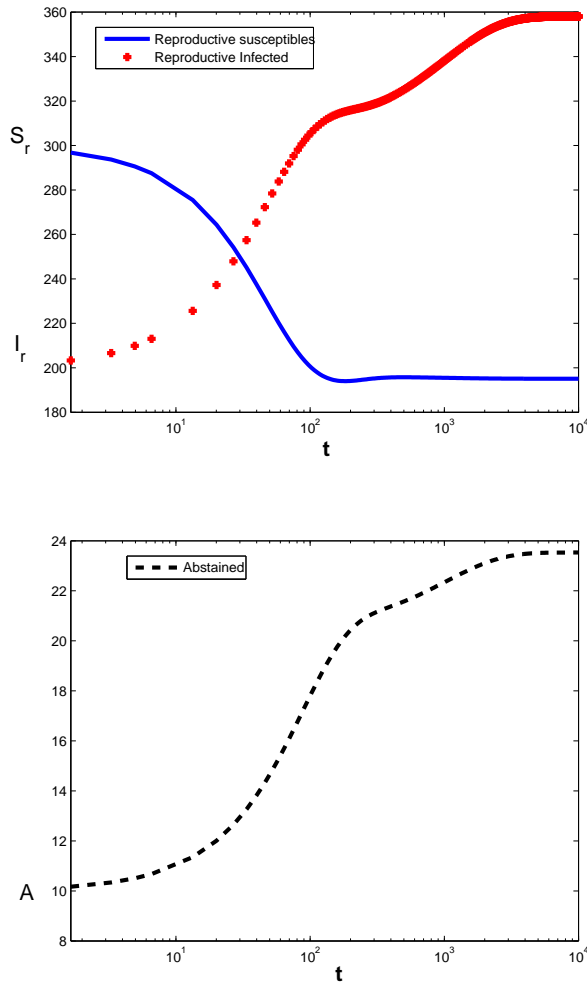


Figure 3. Case $\lambda > \frac{\beta - \nu_1 + \nu_2}{(K - \frac{\nu_1}{b})(1 - \frac{\nu_1}{\beta})}$.
 $\beta = 0.01442$ $\mu = 0.01303$ $\lambda = 0.000075$ $\nu_1 = 0.0002$ $\nu_2 = 0.0008$ $\alpha = 0$

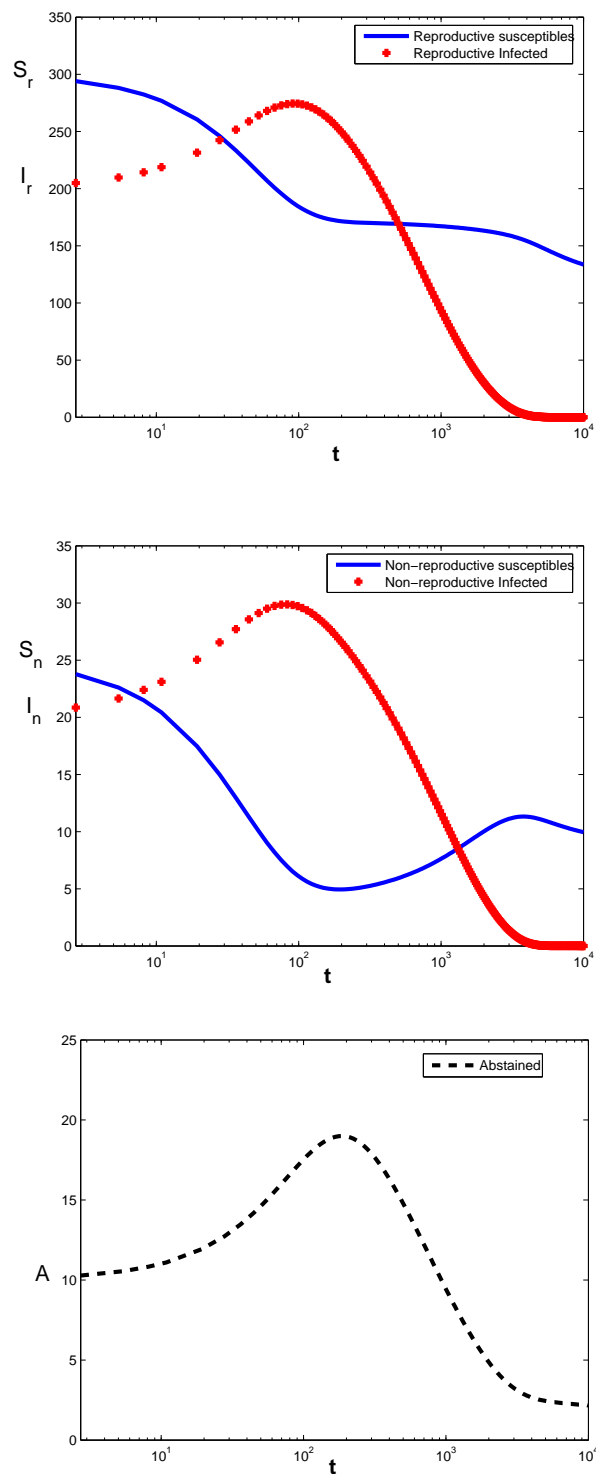


Figure 4. Case $\frac{\beta - \nu_1 + \nu_2}{(K - \frac{\nu_1}{b})(1 - \frac{\nu_1}{\beta})} < \lambda < \frac{\beta(\mu^* + \gamma_1)(\mu^* + \gamma_2)(\mu^* + \alpha_2 + \nu_2)}{\mu^* P[(\mu^* + \gamma_1)(\mu^* + \alpha_2 + \gamma_2) + \alpha_1(\mu^* + \alpha_2 + \nu_2)]}$.
 $\beta = 0.01442$ $\mu = 0.01303$ $\lambda = 0.000075$ $\nu_1 = 0.0002$ $\nu_2 = 0.0008$ $\alpha = 0.001$

4. The interior steady state in the absence of the abstained group

In this section we study the stability of the interior equilibrium in the particular case $\lambda_1 = \lambda_2 = \lambda$, $\alpha_1 = \alpha_2 = \alpha$ and in the absence of the abstained group A , i.e. $\nu_1 = \nu_2 = 0$ and $\gamma_1 = \gamma_2 = 0$. The abstained groups represent people

who not only do not reproduce but also quarantine themselves from the infection through abstinence. If they did reproduce, the effect of the isolation rate ν_1 would be identical to a quarantine effect and the rate ν_2 would be identical to a recovery with immunity effect, as pointed out in [3]. Based on the result from the previous section, we are now motivated to analyze in more detail the original model having non-reproductive and sexually active groups only that are not quarantined from the disease. Another reason for this approach is given by the fact that, in general, the majority of the people isolated from reproduction are sexually active and unaware of whether they are infected or not. With these assumptions, the population's behavior is completely independent of the presence of the disease.

The model becomes

$$\begin{cases} S'_r = \beta(S_r + I_r) - \lambda S_r I_r - \lambda S_r I_n - \alpha S_r - \bar{\mu}(P)S_r, \\ S'_n = \alpha S_r - \lambda S_n I_r - \lambda S_n I_n - \bar{\mu}(P)S_n, \\ I'_r = \lambda S_r I_r + \lambda S_r I_n - \alpha I_r - \bar{\mu}(P)I_r, \\ I'_n = \alpha I_r + \lambda S_n I_r + \lambda S_n I_n - \bar{\mu}(P)I_n. \end{cases} \quad (11)$$

This system can be reduced to a two-dimensional one that follows the dynamics of the total reproductive and non-reproductive populations,

$$R = S_r + I_r \quad \text{and} \quad N = S_n + I_n.$$

Notice that if we add the first to the third equation and the second to the fourth we obtain the following reduced system:

$$\begin{cases} R' = [\beta - \alpha - \bar{\mu}(P)]R, \\ N' = \alpha R - \bar{\mu}(P)N, \end{cases} \quad (12)$$

where

$$P(t) = R(t) + N(t).$$

Below we are going to use the Poincaré-Bendixson theory in \mathbf{R}^2 to establish the asymptotic sizes of $R(t)$ and $N(t)$:

Proposition 4.1: *The system (12) has an interior steady state which is globally stable, i.e.*

$$\lim_{t \rightarrow \infty} R(t) = \left(1 - \frac{\alpha}{\beta}\right) \left(K - \frac{\alpha}{b}\right) \quad \text{and} \quad \lim_{t \rightarrow \infty} N(t) = \frac{\alpha}{\beta} \left(K - \frac{\alpha}{b}\right)$$

where

$$K = \frac{\beta - \mu}{b}.$$

Proof: From

$$R' < (\beta - \alpha - \mu - bR)R \quad \text{and} \quad N' < \alpha R - \mu N$$

any solution of (12) is bounded in the open set

$$\mathcal{O} = \left(0, \max\left\{R_0, K - \frac{\alpha}{b}\right\}\right) \times \left(0, \frac{\alpha}{\mu} \max\left\{R_0, K - \frac{\alpha}{b}\right\}\right),$$

where $R(0) = R_0$. Notice that in this set there exists a unique equilibrium of (12):

$$R^* = \left(1 - \frac{\alpha}{\beta}\right) \left(K - \frac{\alpha}{b}\right) \quad \text{and} \quad N^* = \frac{\alpha}{\beta} \left(K - \frac{\alpha}{b}\right).$$

Furthermore, the trivial state $(0, 0)$ is a saddle since the Jacobian evaluated at the origin is

$$\begin{pmatrix} \beta - \mu - \alpha & 0 \\ \alpha & -\mu \end{pmatrix},$$

and we know that $\beta - \mu > \alpha$ by our original assumption. Therefore, any orbit that starts arbitrarily close to the origin will move away from the origin, except the ones starting on the nullcline $R = 0$. Furthermore, the interior equilibrium (R^*, N^*) is locally asymptotically stable. The Jacobian of (12) evaluated at (R^*, N^*) is

$$\begin{pmatrix} \beta - \alpha - \bar{\mu}(P^*) - bR^* & -bR^* \\ \alpha - bN^* & -\mu(P^*) - bN^* \end{pmatrix},$$

where $P^* = R^* + N^* = \frac{\beta - \mu - \alpha}{b}$. Replacing $\bar{\mu}(P^*)$ by $\beta - \alpha$, the Jacobian becomes

$$J = \begin{pmatrix} -bR^* & -bR^* \\ \alpha - bN^* & -\beta + \alpha - bN^* \end{pmatrix},$$

with

$$\text{Tr } J = -(\beta - \alpha) - bP^* < 0 \quad \text{and} \quad \det J = b\beta R^* > 0,$$

showing that both eigenvalues have negative real part.

Also notice that (12) satisfies Dulac's criterion

$$\begin{aligned} \frac{\partial}{\partial R} \left[\frac{1}{RN} (\beta - \alpha - \bar{\mu}) R \right] + \frac{\partial}{\partial N} \left[\frac{1}{RN} (\alpha R - \bar{\mu} N) \right] = \\ = -\frac{1}{RN} \left(bR + bN + \alpha \frac{R}{N} \right) < 0, \end{aligned}$$

which eliminates the possibility of periodic solutions or of separatrix cycles and graphics in \mathcal{O} . From these the global stability of the interior steady state follows. \square

We can now use the theory of asymptotically autonomous systems established by Thieme and Castillo-Chavez in [5] and [6] to study the behavior of the original system based on the reduced one in R and N . Using the previous proposition we can return to our original system (11) to write the last two equations as an asymptotically autonomous system in I_r and I_n .

$$\begin{cases} I_r' = [\lambda R - \alpha - \bar{\mu}(P)]I_r + \lambda R I_n - \lambda I_r I_n - \lambda I_r^2 & := f(t, I_r, I_n) \\ I_n' = (\alpha + \lambda N)I_r + [\lambda N - \bar{\mu}(P)]I_n - \lambda I_r I_n - \lambda I_n^2 & := g(t, I_r, I_n). \end{cases} \quad (13)$$

This is an asymptotically autonomous system whose limiting system is

$$\begin{cases} I_r' = (\lambda R^* - \beta)I_r + \lambda R^* I_n - \lambda I_r I_n - \lambda I_r^2 & := \bar{f}(I_r, I_n) \\ I_n' = (\alpha + \lambda N^*)I_r + (\lambda N^* - \beta + \alpha)I_n - \lambda I_r I_n - \lambda I_n^2 & := \bar{g}(I_r, I_n). \end{cases} \quad (14)$$

The limiting system (14) has two steady states: the trivial $(0, 0)$ corresponding to a disease-free equilibrium in the original system and an interior one:

$$I_r^* = \left(1 - \frac{\alpha}{\beta}\right) \left(K - \frac{\alpha}{b}\right) \left[1 - \frac{\beta}{\lambda \left(K - \frac{\alpha}{b}\right) + \alpha}\right],$$

$$I_n^* = \frac{\alpha}{\beta} \left[\left(K - \frac{\alpha}{b}\right) - \frac{\beta(\beta - \alpha)}{\lambda^2 \left(K - \frac{\alpha}{b}\right) + \lambda \alpha} \right].$$

If

$$\lambda < \frac{\beta - \alpha}{K - \frac{\alpha}{b}},$$

both I_r^* and I_n^* are negative and the only steady state of (14) is $(0, 0)$, whereas if

$$\lambda > \frac{\beta - \alpha}{K - \frac{\alpha}{b}},$$

then $(0, 0)$ is unstable and (I_r^*, I_n^*) is the only interior steady state. Notice that this is consistent with the epidemic reproductive number computed in the previous section. If we replace $\alpha_1 = \alpha_2 = \alpha$, $\gamma_1 = \gamma_2 = 0$ and $\nu_1 = \nu_2 = 0$ in \mathcal{R}_0 then,

$$\mathcal{R}_0 = \frac{\lambda \left(K - \frac{\alpha}{b}\right)}{\beta - \alpha}.$$

Proposition 4.2: *Every solution of (13) asymptotically approaches the trivial equilibrium $(0, 0)$ if $\lambda < \frac{\beta - \alpha}{K - \frac{\alpha}{b}}$ and (I_r^*, I_n^*) otherwise.*

Proof: Notice that if we sum the equations of the limiting system (14) we obtain the following equation for the sum of the two unknowns, $I(t) = I_r(t) + I_n(t)$:

$$I' = \left[\lambda \left(K - \frac{\alpha}{b}\right) - \beta + \alpha \right] I - \lambda I^2.$$

This is a logistic equation and we thus have

$$I(t) \rightarrow 0 \quad \text{if} \quad \lambda < \frac{\beta - \alpha}{K - \frac{\alpha}{b}},$$

and

$$I(t) \rightarrow K - \frac{\alpha}{b} - \frac{\beta - \alpha}{\lambda} \quad \text{otherwise.}$$

We already know that every solution of (13) is bounded in \mathcal{O} . According to [5] and [6], in order to use the Poincaré-Bendixson-type trichotomy for (13) we must show that the closure of any possible periodic orbit or graphic of (14) is included in the open set \mathcal{O} . Notice that there is no orbit tangent to either $I_r = 0$ or $I_n = 0$. For example it is impossible for (I_r, I_n) to be arbitrarily close to a point $(0, a)$ with $a > 0$ because $I_r' \approx \lambda R^* a > 0$. Similarly, (I_r, I_n) cannot approach a point $(a, 0)$ with $a > 0$ since $I_n' \approx (\alpha + \lambda N^*) a > 0$. Furthermore, there can be no separatrix cycle including the origin because this would imply that there is a solution which starts from a positive initial datum and declines to zero and a solution that moves away from zero if it starts close to the origin. This is impossible for two reasons: If $\lambda < \frac{\beta - \alpha}{K - \frac{\alpha}{b}}$, then $(0, 0)$ is a stable node; hence, starting close enough to the origin all solutions must decline to zero from any direction. On the other hand, if $\lambda > \frac{\beta - \alpha}{K - \frac{\alpha}{b}}$, then we know that $I(t)$ approaches a positive limit so there is no solution that declines to zero.

In other words neither I_r nor I_n can be arbitrarily close to $I_r = 0$ or $I_n = 0$. Hence, any possible periodic orbit or graphic and their closure is included in the open set mentioned above. However, the existence of these types of solutions is ruled out by Dulac's criterion applied on \mathcal{O} :

$$\begin{aligned} & \frac{\partial}{\partial I_r} \left[\frac{1}{I_r I_n} \bar{f}(I_r, I_n) \right] + \frac{\partial}{\partial I_n} \left[\frac{1}{I_r I_n} \bar{g}(I_r, I_n) \right] = \\ & = -\frac{1}{I_r I_n} \left[\lambda I_r + \lambda I_n + \lambda R^* \frac{I_n}{I_r} + (\alpha + \lambda N^*) \frac{I_r}{I_n} \right] < 0. \end{aligned}$$

Thus our system satisfies the Poincaré-Bendixson-type trichotomy established by Thieme in [5] for asymptotically autonomous systems. Therefore, as shown in [6], the only possibility is that any solution of (13) will converge to an equilibrium of the limiting system (14). □

One might be tempted to conjecture that the global behavior of the system in the case $\alpha_1 \neq \alpha_2$ is similar. We believe that global stability of the endemic state holds as well in this case, although we did not find a Lyapunov function for the most general system. On the other hand, an analysis of the Center Manifold of the disease-free steady state when $\mathcal{R}_0 = 1$ reveals that for different isolation rates we may obtain unstable endemic steady states for $\mathcal{R}_0 < 1$. This will also give an interpretation of the role of α_2 in the nature of this bifurcation. One possible explanation of why α_2 is not present in the epidemic reproductive number is given by the fact that if a single infected individual is introduced in a population of susceptibles, it is irrelevant in the beginning whether he reproduces or not since, by being sexually active, his force of infection remains unchanged.

5. The existence of sub-threshold endemic equilibria near $\mathcal{R}_0 = 1$

As pointed out by van den Driessche et al. in [1], the analysis of the center manifold near the bifurcation point $\mathcal{R}_0 = 1$ may show the existence of sub-threshold equilibria. The epidemiological interpretation is that although $\mathcal{R}_0 < 1$ and the DFE is locally asymptotically stable, non-trivial endemic steady states may still exist, meaning that even small perturbations could lead to an epidemic.

The purpose of this section is to show that having different isolation rates for the healthy and the infected group may change the dynamics in the sense that if α_2 is too low, sub-threshold endemic equilibria can occur.

We consider the original model (1) without the abstained class A :

$$\begin{cases} S'_r = \beta(S_r + I_r) - \lambda S_r I_r - \lambda S_r I_n - \alpha_1 S_r - \bar{\mu}(P)S_r, \\ S'_n = \alpha_1 S_r - \lambda S_n I_r - \lambda S_n I_n - \bar{\mu}(P)S_n, \\ I'_r = \lambda S_r I_r + \lambda S_r I_n - \alpha_2 I_r - \bar{\mu}(P)I_r, \\ I'_n = \alpha_2 I_r + \lambda S_n I_r + \lambda S_n I_n - \bar{\mu}(P)I_n. \end{cases} \quad (15)$$

Van den Driessche and Watmough proved in [1] that, when $\mathcal{R}_0 = 1$, the Jacobian of (15) evaluated at the DFE has exactly one zero eigenvalue while the other eigenvalues have negative real part. Therefore they were able to analyze the corresponding one-dimensional center manifold and provide a useful characterization of the nature of this bifurcation. We state their result below:

Consider the following system that satisfies all the hypotheses in [1]

$$x' = f(x, \epsilon),$$

where ϵ is a bifurcation parameter chosen so that $\epsilon > 0$ if $\mathcal{R}_0 > 1$ and $\epsilon < 0$ if $\mathcal{R}_0 < 1$. Denoting the DFE by x_0 , we define the following constants:

$$\bar{a} = \frac{v}{2} D_{xx} f(x_0, 0) w^2, \quad (16)$$

$$\bar{b} = v D_{x\epsilon} f(x_0, 0) w, \quad (17)$$

where v and w are the left and right null vectors corresponding to the zero eigenvalue, chosen so that $vw = 1$. Theorem 4 of [1] states that, if the zero eigenvalue of $D_x f(x_0, 0)$ is simple and if $b \neq 0$, then there exists $\delta > 0$ such that:

- if $\bar{a} < 0$, then there are locally asymptotically stable endemic equilibria near x_0 for $0 < \epsilon < \delta$,
- if $\bar{a} > 0$, then there are unstable endemic equilibria near x_0 for $-\delta < \epsilon < 0$.

To apply this result to our model we consider first the Jacobian of (15):

$$\begin{pmatrix} \beta - \bar{\mu}(P) - \alpha_1 - \lambda(I_r + I_n) - bS_r & -bS_r & \beta - (\lambda + b)S_r & -(\lambda + b)S_r \\ \alpha_1 - bS_n & -\bar{\mu}(P) - \lambda(I_r + I_n) - bS_n & -(\lambda + b)S_n & -(\lambda + b)S_n \\ \lambda(I_r + I_n) - bI_r & -bI_r & -\bar{\mu}(P) - \alpha_2 + \lambda S_r - bI_r & \lambda S_r - bI_r \\ -bI_n & \lambda(I_r + I_n) - bI_n & \alpha_2 + \lambda S_n - bI_n & -\bar{\mu}(P) + \lambda S_n - bI_n \end{pmatrix}.$$

The disease-free equilibrium is

$$(\bar{S}_r, \bar{S}_n, \bar{I}_r, \bar{I}_n) = \left[\left(1 - \frac{\alpha_1}{\beta} \right) \left(K - \frac{\alpha_1}{b} \right), \frac{\alpha_1}{\beta} \left(K - \frac{\alpha_1}{b} \right), 0, 0 \right].$$

Setting $\nu_1 = \nu_2 = 0$ in (7), we find that the DFE is asymptotically stable provided that

$$\frac{\beta}{K} < \lambda < \frac{\beta - \alpha_1}{K - \frac{\alpha_1}{b}}.$$

Thus, the epidemic reproductive number can be written as

$$\mathcal{R}_0 = \frac{\lambda}{\lambda^*} \quad \text{where} \quad \lambda^* = \frac{b(\beta - \alpha_1)}{(\beta - \mu - \alpha_1)}.$$

We have that $\mathcal{R}_0 < 1$ if $\lambda < \lambda^*$ and $\mathcal{R}_0 > 1$ if $\lambda > \lambda^*$. In conclusion, we can take as bifurcation parameter

$$\epsilon = \lambda - \lambda^*$$

and apply the result from [1] described above.

We substitute λ with $\epsilon + \lambda^*$ in (15) and compute the Jacobian evaluated at the DFE with $\epsilon = 0$:

$$\begin{pmatrix} \beta - \mu - \alpha_1 - 2b\bar{S}_r - b\bar{S}_n & -b\bar{S}_r & \beta - (\lambda^* + b)\bar{S}_r & -(\lambda^* + b)\bar{S}_r \\ \alpha_1 - b\bar{S}_n & -\mu - b\bar{S}_r - 2b\bar{S}_n & -(\lambda^* + b)\bar{S}_n & -(\lambda^* + b)\bar{S}_n \\ 0 & 0 & -\mu - \alpha_2 - b\bar{S}_r - b\bar{S}_n + \lambda^*\bar{S}_r & \lambda^*\bar{S}_r \\ 0 & 0 & \alpha_2 + \lambda^*\bar{S}_n & -\mu - b\bar{S}_r - b\bar{S}_n + \lambda^*\bar{S}_n \end{pmatrix}.$$

The eigenvalues of the above matrix are:

$$0, \alpha_1 - \beta, \alpha_1 - \alpha_2 - \beta, \alpha_1 - \beta + \mu.$$

After a lengthy but straightforward computation we find the following pair of left and right null vectors corresponding to the zero eigenvalue, denoted by v and w and chosen such that $vw = 1$:

$$v = (v_1, v_2, v_3, v_4), \quad \text{and} \quad w = (w_1, w_2, w_3, w_4)$$

where

$$\begin{aligned} v_1 = v_2 = 0, \quad v_3 = v_4 &= \frac{\alpha_1(\beta - \alpha_1) + \beta\alpha_2}{\beta(\beta - \alpha_1 + \alpha_2)}, \\ w_1 &= \frac{(\beta - \alpha_1)[3\alpha_1^2 + 2\alpha_2\beta + \beta^2 - 2\alpha_1(\alpha_2 + 2\beta - \mu) - \mu\alpha_2 - \beta\mu]}{(\beta - \mu - \alpha_1)[\alpha_1^2 - \beta(\alpha_1 + \alpha_2)]}, \\ w_2 &= \frac{\alpha_1[3\alpha_1^2 + 2\beta(\beta - \mu) + \alpha_2(2\beta - \mu) + \alpha_1(-2\alpha_2 - 5\beta + 2\mu)]}{(\beta - \mu - \alpha_1)[\alpha_1^2 - \beta(\alpha_1 + \alpha_2)]}, \\ w_3 &= \frac{(\beta - \alpha_1)^2}{\beta(\alpha_1 + \alpha_2) - \alpha_1^2}, \quad \text{and} \quad w_4 = 1. \end{aligned}$$

The constant \bar{b} defined as in (17) becomes:

$$\bar{b} = K - \frac{\alpha_1}{b},$$

which is clearly positive due to our assumption that $\beta > \mu + \alpha_1$. According to the Center Manifold Theorem and [1], the nature of the bifurcation is characterized by the sign of \bar{a} defined in (16), which in our case is

$$\bar{a} = \frac{b\beta(\beta - \alpha_1)[\beta\mu - (\beta - \alpha_1)^2 - \alpha_2(\beta - \alpha_1)]}{[\beta\alpha_2 + \alpha_1(\beta - \alpha_1)](\beta - \mu - \alpha_1)^2}.$$

Since $\beta > \mu + \alpha_1$ the sign of \bar{a} is determined by the following expression:

$$\mathcal{L}(\alpha_1, \alpha_2) = \beta\mu - (\beta - \alpha_1)^2 - \alpha_2(\beta - \alpha_1).$$

Notice that, if $\alpha_1 = \alpha_2 = \alpha$, this expression becomes:

$$\mathcal{L}(\alpha, \alpha) = -\beta(\beta - \mu - \alpha) < 0,$$

and, according to the theorem from [1] stated above, there exists a stable endemic equilibrium for $\mathcal{R}_0 > 1$. This is no surprise, since we have proved in the previous section that the endemic steady state is in fact globally stable if $\mathcal{R}_0 > 1$.

However, if

$$\alpha_1 \neq \alpha_2 \quad \text{and} \quad \mathcal{L}(\alpha_1, \alpha_2) > 0,$$

then $\bar{a} > 0$ implying, by the same theorem, that there exist a neighborhood of the bifurcation point λ^* where the system admits an unstable endemic steady state

when $\mathcal{R}_0 < 1$. This condition is equivalent to

$$\alpha_1 > \sqrt{\beta}(\sqrt{\beta} - \sqrt{\mu}) \quad \text{and} \quad \alpha_2 < \frac{\beta\mu - (\beta - \alpha_1)^2}{\beta - \alpha_1}. \quad (18)$$

In the next figure we show a numerical example based again on the 2000 U.S. Census data, with $\beta = 0.01442$ and $\mu = 0.01303$. The dark region corresponds to $\beta - \mu - \alpha_1 > 0$ and $\mathcal{L}(\alpha_1, \alpha_2) > 0$.

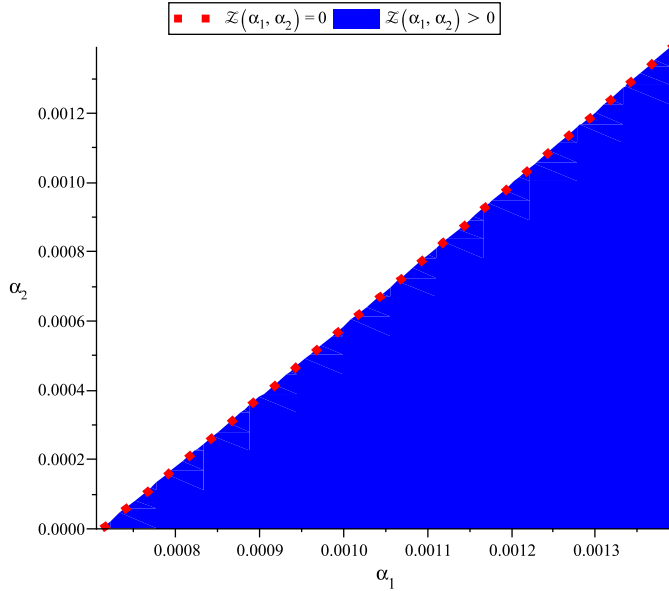


Figure 5. $\beta = 0.01442$, $\mu = 0.01303$

Remark 1 :

One possible interpretation of (18) is the following: if an infective individual is introduced in a susceptible population and there is no epidemic, i.e. the DFE is stable, then the infected class remains small while the evolution of the susceptibles is dominated by β , μ and α_1 . A higher value of α_1 than the above threshold implies that the contribution from the reproductive infected people to the susceptible class becomes important and a lower isolation rate α_2 maintains a critical number of susceptibles exposed to the infection, enough to allow the existence of a sub-threshold endemic steady state.

6. Conclusions

We have extended models developed in [3] to include a more general class of sexually active but non-procreating people and derived similar thresholds for the infection rate that lead to the stability of the disease-free equilibrium. The sexually active class may extend the range of stability for the DFE under certain conditions, which suggests that the depletion of susceptibles alone could be enough to eradicate the disease from a population that otherwise would reach an endemic steady state. Another related result is that the quarantine role provided by the abstinence of the infected in achieving a disease-free steady state can be less significant than the isolation from reproduction of the sexually active population. In the case of equal infection rates for both reproductive and non-reproductive people we managed to prove global stability of both the DFE and the endemic steady state when $\mathcal{R}_0 < 1$

and $\mathcal{R}_0 > 1$, respectively. We also found that different isolation rates play an important role in the dynamics of the disease. While the isolation rate of infected people does not impact the epidemic reproductive number \mathcal{R}_0 , it may lead to the existence of sub-threshold endemic steady states, thus emphasizing the importance of differences in the reproduction behavior between the infected and the healthy people.

A more realistic method of analyzing the problem can be achieved by using demographic gender structured models that include couple formation between infected and healthy individuals who, by separation or death of a partner, provide a source for newly infected single persons. This is the focus of an upcoming paper that uses a modified logistic two-sex model with non-linear mortality and including abstained classes only. In this research we will attempt to establish a similar result as in [3]. The more general analysis of a gender structured model that also includes non-reproductive sexually active groups is currently underway and will be reported later.

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